Matters of the Athletic Heart

Clifton P. Titcomb, Jr, MD

Frequently an underwriter or medical director will question whether an increase in left ventricular mass represents pathologic left ventricular hypertrophy (LVH) or physiologic changes related to exercise. The LVH condition reflects end-organ damage related to abnormal hemodynamic stresses and confers an increased morbidity and mortality risk. When left ventricular mass is increased because of exercise, measured changes represent a normal, healthy cardiovascular system responding to the demands of that exercise. This article summarizes medical findings that distinguish pathologic LVH from an “athletic heart.”

During exercise, muscles demand more oxygen and nutrients. Normally the heart can increase its output 4 to 5 times to accommodate this need. Cardiac output increases as a result of an increase in heart rate and stroke volume, and the amount of blood pumped with each heartbeat. An individual’s maximum heart rate is age dependent and does not increase with training. Because there is an age-dependent limit to the maximum achievable heart rate, an increase in stroke volume is the primary way for the heart to adapt to the demands of chronic exercise training. More efficient use of oxygen by peripheral tissues is another important result of training.¹

TYPES OF TRAINING AND THE EFFECTS

There are 2 major forms of exercise: isotonic and isometric. Isotonic exercise changes skeletal muscle length and generally occurs when muscle contraction works against a low level of resistance. Examples of isotonic exercise include bicycling, running and other forms of “endurance” training. Isometric exercise occurs when muscles contract against a fixed resistance. Many forms of exercise consist of a mixture of isotonic and isometric components. Weight training is a good example. Weight training is primarily isotonic exercise if it consists of many repetitions using relatively light weights. Conversely, lifting very heavy weights with relatively few repetitions is primarily isometric exercise.

Isotonic and isometric exercise have different effects on the heart. Isotonic exercise reduces peripheral vascular resistance and increases venous return to the heart. Increased blood return to the heart causes increased diastolic filling of the left ventricle preceding the next contraction. This “diastolic volume load” increases the vigor with which the left ventricle contracts resulting in increased systolic blood pressure and either unchanged or
reduced diastolic pressure during exercise. Isometric exercise requires sustained contraction of the active muscle groups, which increases peripheral resistance to blood flow. During systole, the left ventricle must eject its output against this increased resistance, and the net effect is an increased pressure load on the left ventricle. The left ventricle responds to these volume and pressure loads by dilating and increasing its muscle mass, resulting in an overall increase in heart size (cardiomegaly).

ATHLETIC HEART FINDINGS

Athletic heart syndrome consists of the normal adaptive changes to the heart resulting from regular vigorous exercise.

Physical Findings

The physical findings associated with athletic heart syndrome include slow resting pulse, exaggerated heart rate variability with respiration, and cardiomegaly. In 30% of well-trained athletes, a systolic murmur is present due to augmented stroke volume and resultant increased blood flow. S3 and S4 gallop rhythms are also commonly heard. All of these findings are normal and of no consequence if they result from athletic heart.

ECG Findings

There are certain electrocardiographic findings typically found in well-trained athletes, as well. Sinus bradycardia and sinus arrhythmia are very common. Sinus pauses, often lasting more than 2 seconds, are frequent and are found in more than one-third of well-trained individuals. However, pauses of more than 4 seconds are uncommon and a potential cause for concern.

First-degree atrioventricular block is found in up to 20% of individuals and Mobitz I second-degree heart block is also frequently encountered. Both these findings are due to reduced sympathetic and increased vagal nerve tone. Mobitz II second-degree and third-degree AV block may occur but are unusual, and a pathologic cause should be ruled out.

Although an incomplete right bundle branch block (IRBBB) is frequently seen, a complete right or left bundle branch block (CRBBB or CLBBB) is not. Increased QRS voltage is common and correlates with the degree of training. In addition, ST-T changes may also occur. ST elevation compatible with early repolarization is seen regularly. T-wave inversions are encountered in up to 30% of athletes and will regress with the cessation of training. The combination of increased voltage and T-wave inversion may simulate the pattern seen with left ventricular strain. However, it should be noted that ST depression is not a normal finding with training.

Chest X-ray Findings

A chest x-ray of an athlete typically exhibits cardiomegaly with a cardiothoracic ratio of 0.5–0.6 and a globular-shaped heart. There may also be an increase in pulmonary vascularity probably due to the adaptation of the pulmonary vessels to increased blood flow, that may mimic a left to right shunt.

Echocardiographic Findings

Changes may also be seen on echocardiography. Some athletes show a significant increase in left ventricular mass. This increase in mass is reflected by an increase in LV diastolic diameter and an increase in ventricular wall thickness. Enlargement of the left atrium and right ventricle may also be seen. These changes occur rapidly (within weeks to months) with the institution of vigorous training and disappear just as rapidly when training is stopped.

In the past, it was thought that endurance athletes, such as runners, would develop eccentric hypertrophy, (ie, increased mass with a normal ratio of wall thickness to diameter) and strength training would lead to concentric hypertrophy with a relative increase in wall thickness. However, it is now known that most athletes have a blend of the 2 patterns.
The largest increases in ventricular mass occur in those sports that demand a combination of endurance and strength training such as rowing or cycling.

**ATHLETIC HEART AND HYPERTROPHIC CARDIOMYOPATHY**

Occasionally, the changes related to the athletic heart may be confused with hypertrophic cardiomyopathy or pathologic LVH. Asymmetric left ventricular hypertrophy, a common finding in hypertrophic cardiomyopathy, may occur in athletes, but it is unusual. There are also other factors that differentiate athletic heart from the other 2 conditions. One is that changes in the athletic heart are reversible when training stops. Further, diastolic function, ie, filling of the ventricle during diastole, remains normal in athletes, even with large increases of LV mass. Finally, ventricular wall thickness in excess of 13 mm is rare with training, even for those individuals at the world-class level.\(^1,5\)

**UNDERWRITING SUMMARY**

Thus, pathologic left ventricular hypertrophy is differentiated from the changes seen in athletic hearts by the lack of diastolic dysfunction, a lesser degree of wall thickness, and the reversibility that occurs with the cessation of training. Nevertheless, physiologic changes related to regular strenuous exercise may lead to alterations of the usual tests of cardiac function. The following bullets summarize findings in athletic hearts.

**COMMON FINDINGS IN ATHLETIC HEARTS**

- Physical findings include a systolic ejection murmur and an audible S3 and S4 gallop.
- Cardiomegaly on a chest x-ray with a cardiothoracic ratio of 0.5–0.6 is not unusual.
- Common ECG findings of no prognostic significance in athletic hearts:
  - First-degree and Mobitz I second-degree AV block
  - Incomplete RBBB
  - Increased LV voltage
  - ST elevation due to early repolarization
- Common echocardiographic findings of no prognostic significance in athletic hearts:
  - Increased LV mass
  - Increased LV wall thickness (≤12 mm in women and ≤13 mm in men)
  - Increase in LV diastolic dimension

**PATTERN OF CARDIAC ENLARGEMENT IN ELITE ATHLETES**

In one study of elite athletes, 9% of the men and 7% of the women exceeded the upper limit of normal for LV mass index (LV mass corrected for body surface area). Most of this increase was the result of an increased LV cavity dimension, reflecting the increase in stroke volume that occurs with training. LV diastolic diameter exceeded the upper limit of normal (54 mm) in 45% of individuals and was markedly elevated (>60 mm) in 14%. However, overall wall thickness was increased in only 1.2% and relative wall thickness in only 1.8%.\(^7\) In fact, wall thickness exceeded 13 mm in only 1.7% of 947 world-class Italian athletes. This degree of thickening was confined to individuals involved in rowing or cycling activities.\(^8\)

Female athletes also have an increase in ventricular cavity dimension, wall thickness and LV mass relative to the sedentary group. These changes were similar to, but less extensive than, the same measurements seen in comparably trained males. In addition, while 9% of female athletes exceeded the upper limit of normal for ventricular diastolic diameter, none exceeded 12 mm for wall thickness.\(^9\)

**UNCOMMON FINDINGS IN ATHLETIC HEARTS**

- Mobitz II second-degree and third-degree AV block may occur but are unusual and warrant investigation.
- ST depression, CRBBB, CLBBB or ventric-
ular arrhythmias on ECG are not due to athletic heart. Although major T wave inversions can occur with significant (not casual) athletic training, these changes are reversible if exercise is stopped. Generally, major T wave changes require some form of investigation to rule out a pathologic condition.

- LV wall thickness >12 mm is rare in women. In men, a LV wall thickness >13 mm is uncommon and is seen only in well-trained rowers and cyclists.
- Asymmetric hypertrophy of the left ventricular wall may occur, but it is unusual.
- Left ventricular diastolic dysfunction does not occur. Diastolic dysfunction is a pathologic sign.

**REFERENCES**