Athletes and their Hearts: What the Primary Care Physician Should Recognize

ABSTRACT

Physicians will undoubtedly follow athletic patients in their practice, and must therefore be aware of the cardiac adaptations that occur in these patients. Athletic heart syndrome (AHS) is a term used to describe the physiologic adaptation (leading to cardiac hypertrophy and/or dilation) that the heart undergoes in response to intense physical activity. Although these are adaptive responses, physicians need to ensure that these changes are not due to pathological causes such as hypertrophic cardiomyopathy, other genetic or congenital disorders, etc. To do so, physicians must take a through history from the athlete (including family history), conduct a physical exam, and order investigations (such as ECGs, an echocardiograph, etc.) as appropriate. If a pathologic cause is not identified and AHS is noted to be the sole cause of these changes, the athlete should still be counselled on how to safely participate in physical activity.

KEYWORDS: Athletes, cardiovascular care, sports medicine, primary care, screening

CME Pre-test Quiz

Primary care physicians will see patients in clinic who are elite or recreational athletes.[1] Some of these patients will be engaging in intense physical activities that stimulate the heart to enlarge in mass or chamber size (i.e. hypertrophy or dilation) in order to cope with increased workload. This physiologic adaptation allows the heart to increase the stroke volume, the amount of blood ejected per heartbeat. Because of this change, the heart is able to eject more blood in fewer beats, which results in a lower heart rate (i.e. 45 times per minute, as opposed to the normal average of 70-80 beats per minute). These changes, termed 'athletic heart syndrome' (AHS)[2] allow for increased oxygen transport and blood flow, permitting the athlete to engage in their physical activities.

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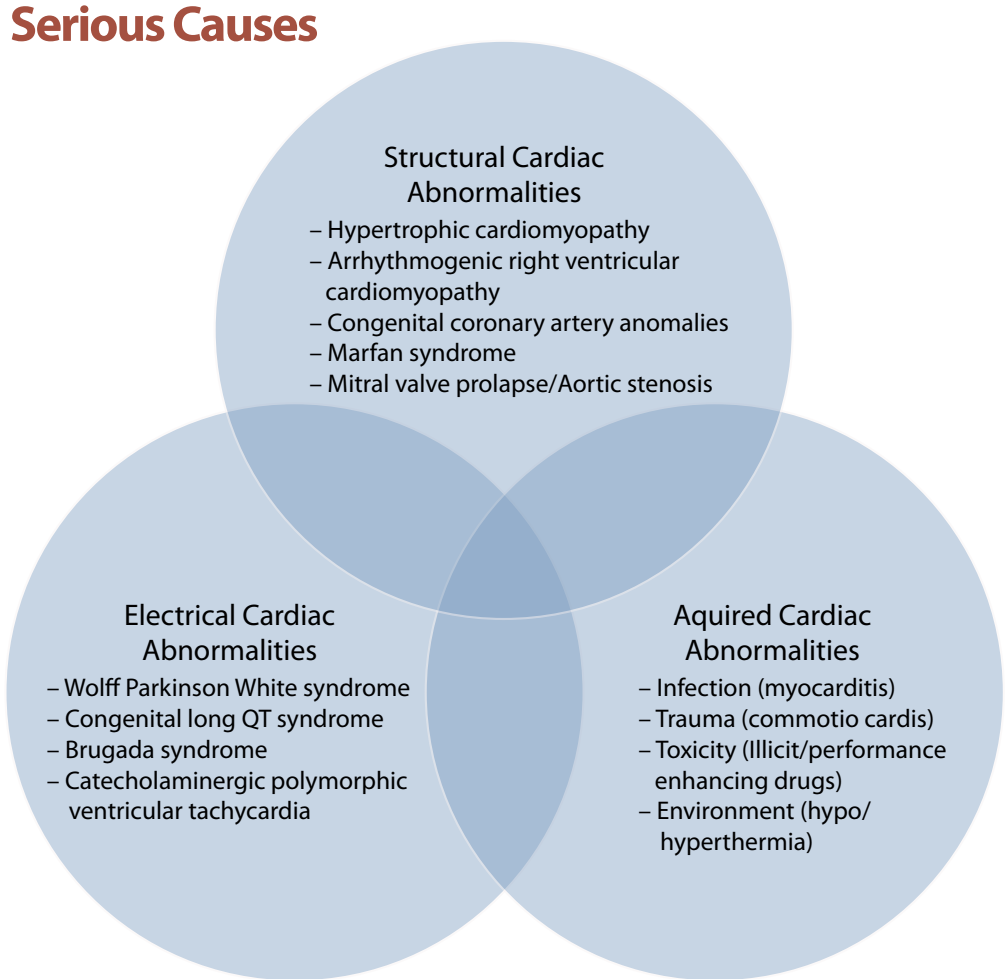
Figure 1: Sudden Cardiac Death (SCD) in Athletes: Serious Causes



Taken from http://heartfailure.onlinejacc.org/content/6/1/30

The changes seen in AHS can mimic a variety of cardiac illnesses, such as hypertrophic cardiomyopathy (HOCM), dilated cardiomyopathy, Marfan syndrome, etc. Sudden cardiac death (SCD) in athletes can be caused by various serious conditions (see Figure 1). Therefore, the physician must ensure that these changes are indeed a response to the athlete's activity routine (i.e. 'AHS'), and not a cause for concern.[4] This can be done by taking a thorough history, conducting a physical exam, and ordering investigations as appropriate.

This article will not explore all the conditions that may lead to hypertrophic or dilated cardiac changes, nor will it discuss the management of these conditions. Rather, it will briefly highlight common mimickers of AHS, and simple clues that the physician cause use to try to determine whether the cause is truly AHS, or a condition that must be managed appropriately.[3] If no other causes are found and it is determined that the changes are, indeed, due to AHS, patients should nonetheless be counselled on warming up prior to exercise, pacing activity, and ensuring they maintain adequate hydration and rest.

Hypertrophic cardiomyopathy (HOCM) is an autosomal dominant condition with variable penetration. It is responsible for 50% of all sudden cardiac deaths (SCD) in young athletes.[5] As the name suggests, HOCM is characterized by a hypertrophied left ventricle, with or without obstruction. Usually, the condition manifests in adolescence, with the sudden onset of chest pain or syncope during exercise. Definitive diagnosis is made by echocardiography, but often, a history of SCD in the family can give physicians an additional clue. Genetic testing may also be performed by a specialist. On physical examination, the physician may note a systolic murmur, much like that of aortic stenosis. However, the murmur of HOCM increases with the Valsalva maneuver or activities that decrease preload (as opposed to aortic stenosis, in

which the murmur decreases with decreased preload). Another clue may be incidental or otherwise unexplained cardiomegaly noted on a chest x-ray. Left ventricular hypertrophy may also be noted on an ECG.[6] Patients with HOCM must be instructed to avoid strenuous physical activity, and are usually followed by a cardiologist.

Of note, ECG changes may also be found in AHS, which include sinus bradycardia, and increased QRS voltage5. Pathological Q waves, left axis deviation, T wave inversion, and left ventricular hypertrophy are more indicative of HOCM, as discussed above. Other clues that differentiate AHS from HOCM can be found in Figure 2.

Dilated cardiomyopathy (DCM) can also mimic AHS if the heart is noted to be dilated on echocardiography. However, patients with DCM often present with an acute episode of congestive heart failure and shortness of breath.[6] DCM can be caused by a variety of conditions, including alcohol, cocaine, pregnancy, infection, hyperthyroidism, and more. Thus, the physician should ask the patient about symptoms such as fever, cough, chest pain, weight loss, fast heart rate, etc. An abnormal ECG, along with other changes such as ejection fraction, can also help to differentiate the two diagnoses.

Marfan syndrome is a congenital syndrome characterized

Figure 2: Features Distinguishing Athlete's Heart From Cardiomyopathy

Feature	Athlete's Heart	Cardiomyopathy
Left ventricular septal thickness*	In men, < 13 mm	In men, > 15 mm
Left ventricular end-diastolic diameter†	< 60 mm	> 70 mm
Diastolic function	Normal (E:A ratio >1)	Abnormal (E:A ratio < 1)
Septal hypertrophy	Symmetric	Asymmetric (in hypertrophic cardiomyopathy)
Family history	None	May be present
Blood pressure response to exercise	Normal	Normal or reduced systolic blood pressure response
Deconditioning	Left ventricular hypertrophy regression	No left ventricular hypertrophy regression

*A value of 13 to 15 mm in men and 11 to 13 mm in women is indeterminate.

†A value of 60 to 70 mm is indeterminate.

E:A ratio = ratio of early to late atrial transmitral flow velocity.

HOCM vs AHS. Taken from https://www.merckmanuals.com/en-ca/professional/cardiovascular-disorders/sports-and-the-heart/athlete's-heart

SUMMARY OF KEY POINTS

Athletic heart syndrome (AHS) is a physiologic adaptation hypertrophy and/or dilation of the heart that allows for increased stroke volume, decreased heart rate, and increased blood flow and oxygen delivery

The hypertrophy and/or dilation that occurs in AHS can mimic serious illnesses that must be ruled out

To differentiate between AHS and pathological causes of AHS, the physician should take a history and conduct a physical exam. Echocardiography and an ECG are also important

by connective tissue changes. Pathological cardiovascular conditions noted in Marfan syndrome include aortic dilatation and dissection, mitral valve prolapse, and arrythmias. The physician should look for other clues of Marfan syndrome, such as those highlighted in the Ghent nosology (ex. ectopic lens dislocation, criteria related to aortic dilatation, arachnodactyly, etc.).[1] Genetic testing can also be pursued.

Arrythmias can also cause SCD. Long QT syndrome is a congenital disease that leads to a prolonged QT interval, and may result in fatal ventricular tachyarrhythmia such as torsades de pointes. Genetic testing, along with appropriate investigations such as ECG monitoring and a family history, are clues to this disease. Acquired prolonged QT syndrome can also be caused by electrolyte imbalances, many medications, etc. Appropriate questioning and investigations can help to determine if the prolonged QT seen on ECG is precipitated by certain factors.[7]

When assessing patients with suspected AHS, physicians must ask questions that will help to guide further investigations and appropriate management. As noted above, ECG changes can provide clues to other pathological causes of hypertrophy or dilation, as can a family history of SCD. Family history of other diseases, such as prolonged QT syndrome or Marfan, is also important to inquire about. Clinical clues on physical examination may increase the suspicion of HOCM or Marfan syndrome. The presence of infectious symptoms, use of certain medications, or other medical diagnoses may lead to prolonged QT. The routine use of an ECG and other investigations are not recommended by expert opinion, but rather, are done on a case-by-case basis. The American Heart Society (AHA) recommends that physicians approach athletes with a personal and family history, and a physical examination (Figure 3). Depending on these results, further action may or may

Figure 3: Pre-participation Screening in Athletes

	AHA
Personal history	1. Chest pain/discomfort/tightness/pressure related to exertion
	2. Unexplained syncope/near syncope (judged not to be vasovagal; particularly concerning when occurring during exercise)
	3. Excessive and unexplained dyspnea/fatigue or palpitations, associated with exercise
	4. Prior recognition of a heart murmur
	5. Elevated systemic blood pressure
	6. Prior restriction from participation in sports
	7. Prior testing for the heart, ordered by a physician
Family history	8. Premature death (sudden and unexpected, or otherwise) before 50 years of age attributable to heart disease in ≥ 1 relative
	9. Disability from heart disease in close relative < 50 years of age
	10. Hypertrophic or dilated cardiomyopathy, long QT syndrome, or other ion channelopathies, Marfan syndrome, or clinically significant arrhythmias; specific knowledge of genetic cardiac conditions in family members
Physical examination	11. Heart murmur (likely to be organic and unlikely to be innocent; auscultation should be performed with the patient in both the supine and standing positions or with Valsalva maneuver specifically to identify murmurs of dynamic left ventricular outflow tract obstruction)
	12. Femoral pulses to exclude coarctation
	13. Physical stigmata of Marfan syndrome
	14. Brachial artery blood pressure (sitting position), preferably taken in both arms

Adapted from: https://www.bcmj.org/sites/default/files/public/BCMJ_Vol58_No_3_pre-participation_screening-table1.JPG

not be indicated. An echocardiogram may be ordered to assess cardiac structure and function. If a pathologic cause is identified, patients should be referred to a cardiologist and/or other relevant specialists.

Although AHS is, in itself, not dangerous (but rather, an adaptive change in the heart), general counselling about physical activity must still be offered to all athletes. This includes advising patients to warm-up prior to exercise, ensuring that they maintain adequate nutrition and hydration, training under supervision, pacing activity, etc. Patients should also know when to be concerned and seek medical attention—for example, if they experience extreme fatigue or heat exhaustion, chest pain, shortness of breath, dizziness, and so forth.[2,7] Regular discussions, monitoring, and follow-up is essential.

References

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CLINICAL PEARLS

A family history of sudden cardiac death (SCD) is a 'red flag' that must be investigated further

Inquire and investigate for symptoms such as syncope, shortness of breath, connective tissue changes, lab abnormalities, etc. It is important to keep the differential diagnosis broad to ensure a serious cardiovascular condition isn't missed

An echocardiogram should be ordered to assess cardiac function and look for structural changes in the heart

When other causes have been ruled out, AHS may be diagnosed. Although this is not inherently dangerous in itself, all athletes engaging in strenuous activity require counselling and advice around warming up, pacing activity, etc.