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The Pathophysiology of Athlete's Heart

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What is Athlete's Heart?

Athlete's heart is a pathologic cardiac hypertrophy, closely resembling hypertrophic cardiomyopathy (HCM), seen in those who undergo regular, intense exercise (Pagourelias et al., 2014). Per Pagourelias et al., (2014), athlete's heart is characterized by two main findings on echocardiography:

- Left ventricular hypertrophy (LVH)
- Intraventricular septum thickness (IVST)

Other findings may be present in some cases, but are not necessary for diagnosis:

- Left atrial dilation and dysfunction (Gabrielli et al., 2012)
- Enlarged right and left ventricles (O'Keefe et al., 2012)

These findings take time to occur, taking roughly two years of vigorous exercise for at least five hours per week to cause the changes seen with athlete's heart (Martinez & Nair, 2014). These adaptations occur so that the heart can more readily meet the body's demand.

The author first saw this phenomenon on an echocardiogram (echo) of a 17-year-old female patient. The patient came into the cardiologist's office with complaints of near syncope and dizziness. The patient was a high school athlete who played sports year-round and was on her high school basketball, cross-country, and track teams. The patient exercised vigorously for over 2-3 hours per day for the past 3 years. Her echo showed LVH, IVST and some left atrial dilation. The patient was bradycardic with an ejection fraction (EF) in the high normal range. The author became interested in understanding the pathophysiology of how the heart developed these functional changes and how they affect the athlete in both the short and long terms.

Signs & Symptoms

Most athletes are asymptomatic (McKelvie, 2017). However, according to McKelvie (2017), a variety of signs may be present:

- Bradycardia
- Left ventricular impulse that is out of place, large, and with increased amplitude
- Systolic flow murmur
- S_3 and S_4 heart sounds
- Hyperdynamic carotid pulses

Underlying Pathophysiology

Athlete's heart is the result of reversible physiological remodeling resulting in increased cardiac mass, but normal cardiac function (Weeks & McMullen, 2011). There are slight differences in the extent of remodeling present based on type of exercise:

- In endurance athletes, such as runners and swimmers, the left ventricle's walls thicken and left ventricle dilates.
- In strength athletes, such as weightlifters and wrestlers, the left ventricle's walls thicken and only mild ventricular dilation is present.
- In combination exercise, exhibited by rowers, cyclists and canoeists, the left ventricular walls are grossly thickened and left dilation is present (Weeks & McMullen, 2011).

There are two types of athlete's heart. These types are differentiated based on the pathologic changes seen in the heart anatomy:

- Eccentric myocardial hypertrophy due to volume overload
- Concentric hypertrophy due to resistance load (Galanti, Stefani, Mascherini, Di Tante, & Toncelli, 2016).

The eccentric form is defined by the presence of left ventricular wall thickness and left ventricular dilation (Galanti et al., 2016). Left ventricular wall thickness is present in the concentric form, but no left ventricular dilation is seen (Galanti et al., 2016). The eccentric form is more commonly seen in endurance athletes, while the concentric form is seen more in strength trainers (Weeks & McMullen, 2011).

Cardiac formation of insulin-like growth factor 1 (IFG1) plays a large role in stimulating the physiologic changes necessary for the development of athlete's heart (Weeks & McMullen, 2011). When IGF1 levels are elevated, the heart has the ability to increase in size due to intense exercise (Weeks & McMullen, 2011). Elevated IGF1 leads to the overexpression of IGF1 receptor (IGF1R) triggering downstream signaling of phosphoinositide 3-kinase p110α [PI3K(p110α)] and protein kinase B (Akt1) activation (Weeks & McMullen, 2011). PI3K(p110α) uses plasma membrane lipids to form phosphatidylinositol 3,4,5-triphosphate (PIP_3) (Weeks & McMullen, 2011). PIP_3 is a second messenger used to activate Akt (Weeks & McMullen, 2011). Akt, particularly the Akt1 isoform of Akt, affects protein synthesis and apoptosis, allowing cardiac remodeling to occur (Weeks & McMullen, 2011).

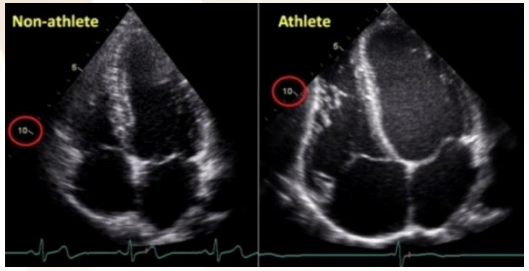

Figure 1. Echocardiographic difference between the thickness of the intraventricular septum of a normal heart versus that of one with pathophysiologic athlete's heart.
Image retrieved from http://medifitbiologicals.com/athletes-heart/

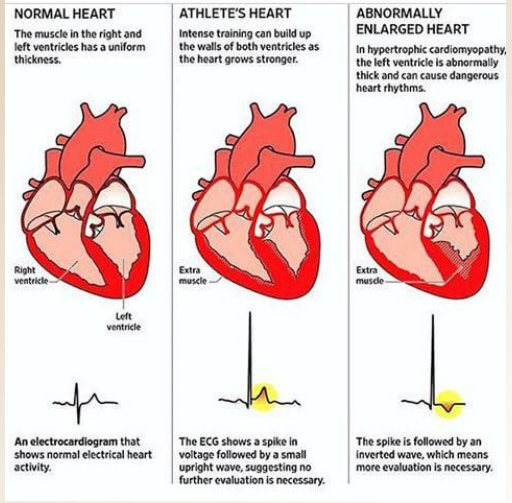

Figure 2. A comparison of the anatomy of the normal heart, the athlete's heart and the heart with hypertrophic cardiomyopathy.
Image retrieved from http://www.imgrum.org/user/doctordconline/1591812152/952597801020270435_1591812152

Significance of Pathophysiology

The diagnosis of athlete's heart is typically done through clinical evaluation, EKG, echo, and, rarely, stress testing (McKelvie, 2017).

On EKG, the patient may exhibit various arrhythmias:

- Sinus arrhythmia
- Sinus bradycardia at rest that can lead to atrial tachycardia, non-sustained ventricular tachycardia, or premature ventricular contractions
- Atrioventricular (AV) blocks
  - 1st degree AV block is most common
  - 2nd degree type 1 AV block that is present at rest and disappears with exercise
  - Rarely 3rd degree AV block
- High-voltage QRS with inferolateral T-wave changes that show LVH on the EKG
- Deep anterolateral T-wave inversion
- Incomplete right bundle branch block (RBBB) (McKelvie, 2017)

Echocardiography is an important imaging modality in the diagnosis and assessment. An echo can show:

- Left ventricular septal thickness between 13-15 mm in men and 11-13 mm in women (McKelvie, 2017)
- Left ventricular mass seen as LVH
- Left ventricular dilation (McDiarmid et al., 2016)

The twisting methods of apical rotation (Arot) and apical circumference left ventricular strain (AVCS) support the increased cardiovascular performance seen on an echo in athlete's heart during maximal intensity exercise (Santoro et al., 2015). During exercise, the left ventricular end-diastolic volume decreases and subpericardial fibers have a mechanical advantage, allowing the left ventricle to eject a greater volume of blood during systole (Santoro et al., 2015). Reduced end-diastolic volume paired with increased heart rate during exercise is thought to cause myofiber alignment to change in a manner that increases left ventricular twisting, making it more effective (Santoro et al., 2015). In athlete's heart, the torsional reserve is an important factor in the systolic-diastolic coupling seen during high-intensity exercise, improving stroke volume (Santoro et al., 2015).

While stress testing is rarely used to diagnose athlete's heart, it can be used to help distinguish physiologic changes from pathologic diseases (McKelvie, 2017). Stress testing shows:

- Submaximal heart rate response to maximal stress with a normal blood pressure response
- Resting EKG abnormalities may disappear with exercise (McKelvie, 2017).

Implications for Nursing Care

It is important for the provider to distinguish athlete's heart from the pathologic remodeling seen with hypertrophic cardiomyopathy (HCM). HCM can result in sports-related sudden cardiac death (SCD), making distinguishability vital to proper patient care (Pagourelias et al., 2014). Several different factors are used to distinguish between HCM and athlete's heart:

- Left ventricular end-diastolic diameter
- Relative wall thickness
- Deceleration time
- Isovolumic relaxation time
- Tricuspid E/A
- Septal E'
- QRS-T angle
- Brain natriuretic peptides (Pagourelias et al., 2014)

A relative septal thickness of greater than 0.54 and spatial QRS-T angle of greater than 45 were found to be most useful in the diagnosis of HCM in studies that looked at both the patient's echo and EKG to distinguish between the two conditions (Pagourelias et al., 2014).

Athlete's heart is traditionally considered reversible condition. New studies are beginning to show that elite athletes who undergo chronic high-intensity, high endurance exercise training may exhibit cardiac remodeling that is not completely benign (O'Keefe et al., 2012). Studies of retired athletes show that even years into retirement, the heart does not completely regress to normal physiology (O'Keefe et al., 2012). The most evident changes found during exercise are:

- Acute dilation of the right atrium
- Acute dilation of the right ventricle
- Sudden decrease in right ventricular EF
- Elevated troponin and B-type natriuretic peptides
- Myocardial fibrosis
- Aortic valve stiffness
- Higher than expected levels of coronary artery calcium and calcified coronary plaque volume

Elite endurance athletes have a higher prevalence of atrial fibrillation, which is likely due to a variety of mechanisms (O'Keefe et al., 2012). Athletes exhibit increased vagal and sympathetic tones, bradycardia, inflammatory changes, myocardial fibrosis, and atrial dilation (O'Keefe et al., 2012). It's possible that left atrial dilation could predict the likelihood of developing atrial fibrillation, however this is all still under investigation (O'Keefe et al., 2012).

Conclusion

Athlete's heart is the result of long-term high-intensity exercise causing physiologic hypertrophy of the heart (Weeks & McMullen, 2011). The heart adapts to the increases in demand needed for high-intensity, high-endurance exercise by increasing in size, resulting in hypertrophy of the left ventricle and intraventricular septum (Pagourelias et al., 2014). Differentiating between athlete's heart and HCM is important as they are treated quite differently since HCM can result in SCD (Pagourelias et al., 2014). While athlete's heart is considered a reversible condition, new research shows that in career athletes the heart may never return to normal cardiovascular function (O'Keefe et al., 2012). The provider needs to be aware of the pathophysiology of athlete's heart and its long-term effects on the cardiovascular system in order to care for athletic patients effectively.

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