Andy is a 40-year-old Masters swimmer who trains six days a week, incorporating a variety of other activities into his exercise regimen as well. Last winter he developed a respiratory tract infection and went to the ER, where a chest X-ray was taken. The ER physician told Andy that his heart was enlarged and, because this could mean serious heart disease, Andy should see his doctor as soon as possible.

The heart, a hollow, muscular organ, is located behind the sternum and consists of four chambers: two atria and two ventricles. The heart’s job is to make the blood flow properly throughout the body, with the atria receiving the blood coming to the heart and the ventricles pushing blood to the rest of the body. Muscle cells within the heart contract rhythmically and pump blood to the skeletal muscle and organs, delivering oxygen and nutrients and removing waste products such as carbon dioxide and lactic acid.

The muscle of the heart responds to exercise, with changes in structure and function to make it work more efficiently. Athletes who work out regularly may develop athlete’s heart, a condition that represents desirable changes that are part of getting into shape. Unfortunately, the changes also can resemble diseases of the heart causing concern on the part of the physician and the athlete.

Just like skeletal muscle, the heart and its muscle “grow” with exercise. When the athlete is engaged in aerobic exercise, the heart must pump a larger quantity of blood to the exercising muscles. As a result, heart cavity size increases, meaning the heart chambers dilate, to accommodate the larger volume of blood being pumped through the body. Because the heart must also pump the larger volume of blood forward, the heart muscle—like skeletal muscles asked to lift progressively heavier weights—gets larger and thicker (hypertrophy). Increased size of the heart cavity or thickening of the heart muscle can mimic the appearance of an enlarged heart.

The changes that occur in the heart muscle as a result of exercise happen gradually over the first four to six weeks of consistent training, usually when the athlete engages in more than one hour of vigorous exercise five or more days per week. Conversely, the changes will regress over four to six weeks of less intense or less frequent training. The degree of changes seen may also reflect differences in individual genetics.

Andy went to see his primary care doctor who examined him and reviewed the X-ray and expressed concern about the enlarged heart. After performing an electrocardiogram (EKG), the physician told Andy the results showed a slow heart rate and some changes in the electrical forces suggestive of heart injury or disease and recommended that Andy see a cardiologist.

For the heart to pump well, it must beat with an adequate heart rate and with the correct rhythm. Specialized heart cells that act as electrical wires control the heart rate and the rhythm. The “wires” conduct the impulses, ensuring that the electrical current reaches every part of the heart and results in nearly simultaneous contraction of the heart muscle. It is this contraction that a swimmer counts as a heart beat (with the number of heartbeats per minute making up the pulse).

With exercise, the heart rate must increase to supply blood to the skeletal muscles. But unlike skeletal muscle where contraction is under voluntary control, the heart is under involuntary control and is directed by signals from the brain. These signals tell the heart to speed up or slow down. The messages are passed from the brain by direct nerve pathways or by the release of chemicals, such as adrenaline. Sympathetic and parasympathetic pathways provide connections from the brain to the heart. The sympathetic messages tend to speed up the heart rate while the parasympathetic messages slow it down. It is the balance of input from these two pathways that determine the rate of heart contraction.

When an athlete trains, initiation of the exercise will first cause withdrawal of the parasympathetic (slowing) messages until the heart rate

Jody Welborn, M.D., is a USMS member and a contributing editor for SWIMMER. She is a cardiologist in private practice at the Oregon Clinic in Portland.
is about 100 beats per minute. At that point, the sympathetic (speeding) stimulation increases until the athlete reaches maximal heart rate.

Changes in heart rate or heart rhythm are characteristic in the athlete’s heart, and the most common means of detection is the EKG, a paper tracing that records the heart’s electrical activity. With this tool, it's possible to see changes in the heart’s electrical activity caused by physical training.

The most common medical finding related to athlete's heart is resting sinus bradycardia (slow heart rate), which can sometimes occur in association with sinus arrhythmia (a variation in the heart rate associated with breathing). The resting pulse of a trained ath-

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In athletes younger than 35, exercise-related sudden death is usually a result of congenital heart disease (heart disease present at birth).

In athletes over 35, exercise-related sudden death usually is due to coronary artery disease.

Consult your doctor before exercising if:

- you have a history of fainting for no apparent reason, particularly if the fainting occurs during or just after exercise
- you have symptoms of chest discomfort or shortness of breath
- you have symptoms of an irregular heartbeat at rest or with exercise
- you have a family history of sudden death, particularly if the family member was younger than 55 years old
- you are a male older than 40, or a female older than 50.

Evaluation of athlete’s heart depends on the symptoms and the specific findings that the athlete exhibits, but usually involves an echocardiogram and a stress test.

The athlete may be asked to stop exercising for a period of weeks. If the findings are due to athlete’s heart, the muscle thickness and chamber sizes will return to normal.

Stress testing typically shows the patient’s excellent capacity for exercise, with the heart rate rapidly returning to normal after the test is done. Athletes with bradycardia will have a normal increase in their heart rate to the maximal heart rate and, for some of those with EKG findings suggesting previous heart damage, the abnormalities will disappear at these higher heart rates. For athletes with palpitations, slow heart rates or other symptoms, continuous EKG monitoring may be done to evaluate whether serious rhythm abnormalities occur.

These tests sometimes are unable to distinguish normal cardiac response to exercise from heart disease, and additional testing, including nuclear medicine stress testing, cardiac catheterization, or electrophysiologic evaluation, may be recommended.

To Andy’s great relief, the echocardiogram and stress test were normal. The cardiologist told him that he had athlete’s heart. He also reassured Andy that the condition was a result of his vigorous exercise program and did not portend future problems.

Although aerobic training offers many cardiac benefits, it does not make anyone immune to heart problems, no matter their age. The risk of heart disease such as hypertension and coronary artery disease is lower in those who exercise, but the risk is not abolished. But it’s also important to remember that sudden death due to exercise is rare and is not caused by athlete’s heart. Swimmers who have any symptoms associated with exercise should consult their physician for further evaluation.

Maximal heart rate is the fastest rate at which an individual’s heart can beat. Everyone’s maximal heart rate differs and has been observed to decline with age. This decline is not specifically related to the preferred exercise but is more likely related to changes in the conduction system of the heart as well as decreases in the number of receptors on the heart cells that respond to catecholamines (e.g., adrenaline).