

THE UNIVERSITY OF TOLEDO MEDICAL CENTER

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ORTHOPAEDIC MONTHLY

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Heat-Related Illness in Athletes

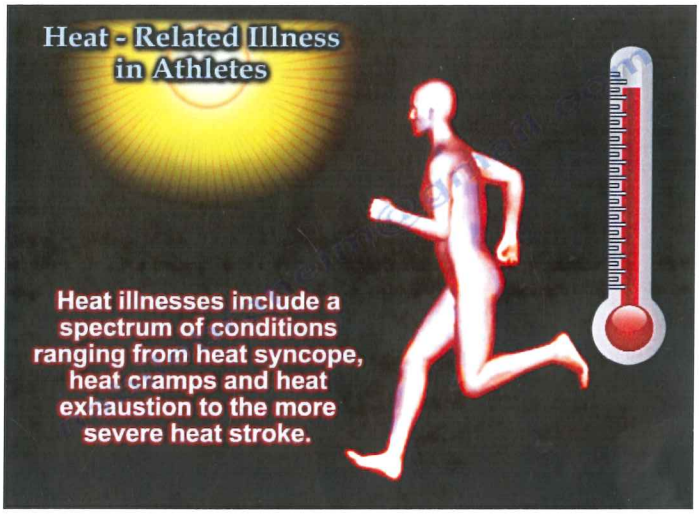
Heat illnesses include a spectrum of conditions ranging from heat syncope, heat cramps and heat exhaustion to the more severe heat stroke.

Heat Syncope (fainting) is a form of orthostatic hypotension that is related to dehydration. It occurs due to inadequate cardiac output and hypotension. It occurs with standing quickly after sitting or lying down for prolonged durations in the heat. Symptoms include fainting, dizziness and light-headedness. Treatment includes oral rehydration (water, juice or sports drinks) and placing the patient flat on the ground in a cool area with slight elevation of the legs to push the blood back to the vital organs such as the brain.

Heat cramps, painful muscle cramps, occur due to decreased sodium concentration in the blood. The patient's core temperature is usually not elevated. Sodium may decrease when salts are lost in sweat or with excessive water intake that does not include electrolytes leading to a situation called dilutional hyponatremia. Symptoms include painful muscle cramps occurring commonly in the abdominal muscles, arms, legs and thighs. Treatment includes rest, cooling and IV fluids or oral rehydration with fluids rich in electrolytes (sports drinks and juices) to replenish the sodium stores. Heat cramps could be prevented by consumption of fluids high in electrolytes before strenuous activities.

Heat exhaustion is the most common heat illness. The body temperature is elevated but less than 40 C. The core body temperature is best measured rectally. Signs and Symptoms include: profuse sweating; a core body temperature lower than 40 C; weakness and fatigue; cramping; headaches, nausea and vomiting; fainting; hypotension; increased heart rate; and fast shallow breathing. Treatment includes rest; IV fluids or oral rehydration; rapid cooling by whole-body immersion in an ice bath.

Heat Stroke is the most severe form of heat illness. It is a medical emergency that needs immediate attention. The patient should be transported to the hospital as soon as possible. Heat strokes occur due to failure of the body's normal thermo-regulatory mechanism. If treatment is not started promptly, end-organ failure and ultimately death may occur. Heat strokes have a high mortality rate and require quick reduction of the patient's temperature. The three characteristic features of this condition include: 1) Lack of sweating, 2) A core body temperature above 40 C (best measured rectally), and 4) Altered mental status. Additional signs and symptoms include: hot, dry skin; disorientation, confusion and hallucinations; headache; slurred speech. Treatment: one must remember this is a serious medical emergency that requires rapid core body temperature reduction, and close monitoring of Airway, Breathing and Circulation. Basic life and advanced life support protocols should be implemented promptly. Rapid cooling is achieved by immersing the whole body in an ice bath; additionally, IV fluids should be administered.



Triceps Tendon Rupture

The triceps muscle is a large muscle on the back of the upper arm. The triceps muscle is a powerful extensor of the elbow joint. There are three heads to the tricep muscle: 1) Long Head, 2) Lateral Head, and 3) the Medial Head. All three heads of the triceps muscle share a common tendon that inserts into the olecranon process at the elbow.

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Triceps Tendon Rupture continued

A triceps tendon rupture is an injury that occurs to the triceps tendon in the back of the elbow. This injury can be missed in many patients. Ruptures of the triceps muscle typically occur in male athletes such as body builders, football players, and in athletes who lift heavy weights.

A triceps tendon rupture is a tear of the tendon that attaches the triceps muscle to the ulna. Rupture of the tendon can be complete or incomplete.

The common mechanisms of injury include 1) Stress from a sudden increase in intensity of training, 2) Direct trauma to the tendon, and 3) Laceration of the tendon.

Rupture of the tendon may also occur due to local steroid injection or it may be due to a history of anabolic steroid use. Rupture may also occur due to previous elbow surgery or systemic diseases such as renal disease and gout. There will be a painful limitation of range of motion and the patient will be unable to extend the elbow, especially against resistance. The patient may hear a "pop" and the tendon may retract upwards. A gap may be felt in the back of the elbow where the rupture occurs. X-rays may show a small bony avulsion. The "Flake" sign identifies the avulsion on lateral x-ray. X-rays are helpful for diagnosing triceps tendon rupture, however, an MRI is the best study for visualizing the tear.

Treatment of Triceps Tendon Rupture:

It is important to establish the correct diagnosis early. 1) Early repair of the tendon in acute cases is also very important. If a delay in diagnosis occurs, primary repair of the tendon may not be possible and the patient may require tendon reconstruction with a less favorable outcome.

2) Reconstruction of the tendon by a graft may be needed in chronic neglected cases, especially if the patient has a major disability in elbow extension.

Management of the Unconscious Athlete

Athletes falling unconscious are not uncommon, especially in contact sports such as football, hockey or rugby.

The first thing to do when assessing an unconscious athlete is to assess his ABC: Airway, Breathing and Circulation. In an unconscious patient always assume a cervical spine injury and handle the patient with extreme caution. If the individual is found to be face down, proceed to slowly bring him on his back by using the log-rolling technique. This maneuver should be directed by the individual maintaining the patient's airway and cervical spine alignment. Remember that skilled personnel with adequate training should be involved in this step. Remove face masks to allow airway access. Also, leave helmets and shoulder pads in place. The helmet should be removed if it is not stabilizing the head and cervical spine or if it is obstructing the airway.

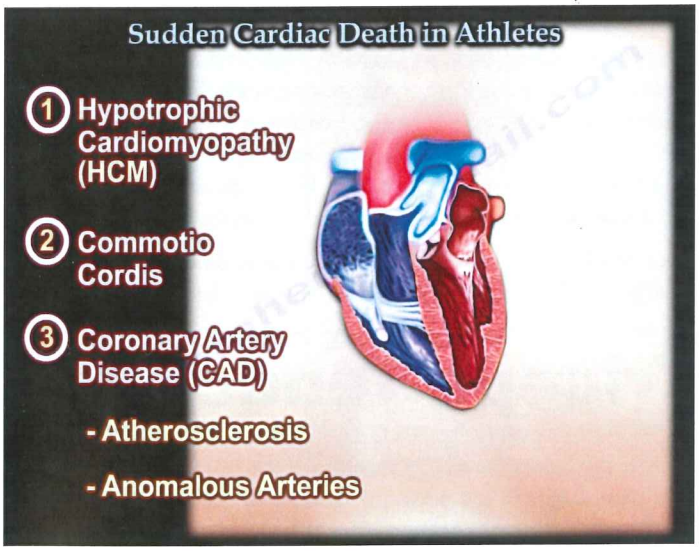
Transporting the patient onto the spine board is achieved by either the log-rolling or five-man lift technique. Do not forget to securely strap the patient onto the spine board before moving him. Advanced trauma and cardiac life support protocols should be performed promptly.

Sudden Cardiac Death in Athletes

The heart is a muscular organ about the size of a closed fist that functions as the body's circulatory pump.

First, we would like to describe the normal anatomy of the heart. The heart is divided into four chambers. The two upper chambers are called the atria and the two bottom chambers are the ventricles. The interventricular septum separates the left ventricle from the right ventricle. The four heart chambers are separated by four valves: the Tricuspid valve, Pulmonary valve, Aortic valve and the Mitral valve.

Deoxygenated blood returning from the entire body enters the heart through the right atrium. It then passes to the right ventricle where it is pumped through the pulmonary artery to the lungs to become loaded with oxygen. Oxygenated blood returns to the left atrium and then passes down into the left ventricle where it is pumped back into the circulation through the aorta.



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The following conditions may lead to sudden cardiac death: 1) Hypotrophic Cardiomyopathy (HCM), 2) Commotio Cordis, 3) Coronary Artery Disease (CAD) such as Atherosclerosis and Anomalous Arteries, 4) Wolff-Parkinson White Syndrome (WPWS), 5) Myocarditis, 6) Arrhythmogenic Right Ventricular Dysplasia (ARVD), and 7) Long QT Syndrome.

Hypotrophic Cardiomyopathy (HCM) is a disease of the heart muscle that leads to its abnormal thickening. This abnormal thickening of the heart muscle occurs due to an autosomal dominant genetic abnormality of the muscle cell proteins. It is considered the most common genetic heart malformation in athletes affecting approximately 1/500 individuals. Asymmetrical thickening of the interventricular septum may lead to a condition known as Hypertrophic Obstructive Cardiomyopathy (HOCM) which may lead to intermittent cardiac outflow obstruction, which may ultimately cause sudden cardiac death. Abnormal Systolic Anterior Motion (SAM) of the mitral valve leaflet exacerbated by exercise may lead to aortic obstruction and sudden death. The mitral valve leaflet may come in contact with the abnormal septum leading to obstruction of blood going through the aorta. Increased heart rate during exercise leads to decreased filling of the left ventricle with blood. This leads to a narrower left ventricular chamber that may increase the chances of aortic obstruction. Therefore, Hypotrophic Cardiomyopathy (HOCM) is an absolute contraindication to vigorous exercise.

PRESENTATION: Most patients are asymptomatic and HCM is found incidentally during regular physical examinations. Thorough history taking is one of the most important parts of the examination. Some patients may present with one or more of the following symptoms: Dyspnea, Angina, Palpations, Syncope, or Sudden Cardiac Death. Cardiac auscultation may reveal an ejection systolic murmur that is best heard at the left parasternal edge and it increases in intensity with maneuvers that decrease left ventricular venous return such as standing abruptly or performing the Valsalva maneuver. Investigative studies include EKG and Echocardiography. PROGNOSIS: Fortunately, the majority of patients have a normal life expectancy. However, risk assessment for the development of sudden cardiac death should be performed. Patients with a high risk of developing sudden cardiac death may benefit from the implantation of a cardioverter defibrillator. TREATMENT: It is important to remember that vigorous exercises should be avoided in all patients with HCM. Genetic testing and physical screening for family members could be helpful for early detection.. Symptomatic patients are treated medically first in order to control their symptoms. Surgical interventions including alcohol septal ablation and surgical myomectomy are indicated only after failure of all drug therapies to control the patient's symptoms.

Commotio Cordia is the sudden death of a healthy individual with no underlying cardiac disease due to ventricular fibrillation following a blunt, nonpenetrating blow to the precordial area of the chest. Sports with a higher risk of commotio cordis include baseball, hockey, lacrosse, cricket, rugby, boxing, karate and other martial arts. The chances of developing commotio cordis are influenced by the following factors: high energy impact, site of impact, anterior chest wall over the heart, the timing of the impact relative to the cardiac cycle. The risk of commotio cordis increases when the impact coincides with the first 10-30 milliseconds of the ascending phase of the T wave. TREATMENT: Unfortunately, most cases are fatal. Defibrillation should be started as soon as possible, preferably within the first 3 minutes. Players should be advised to wear proper protective gear and to avoid blocking balls or pucks with their chest. Furthermore, the presence of automated external defibrillators at sporting events and training grounds has been shown to decrease mortality rates with commotio cordis.

Coronary Artery Disease (CAD). Atherosclerosis may lead to sudden heart attacks and death due to blockage of a coronary artery with plaque. Plaque builds up in the arteries over time and leads to narrowing of the vessels. Risk factors associated with Atherosclerosis: advanced age, a positive family history of heart disease, smoking, obesity, high cholesterol levels, hypertension and diabetes.

Anomalous arteries: An anomalous coronary artery is an artery that has an abnormal anatomical orientation that may impede sufficient blood flow to the heart muscle. This condition is more common in young female athletes and may lead to sudden cardiac death.

Wolff-Parkinson-White Syndrome (WPWS): The normal heart is wired in such a way to prevent extra beats from occurring. Patients with WPWS have an abnormal extra electrical pathway that allows the heart to beat prematurely leading to tachycardia. A heart rate exceeding 240 beats per minute may lead to sudden cardiac death. Symptoms may include chest pain, syncope and palpitations. Continuous ambulatory EKG monitoring (Holter monitor) can help in diagnosis. The heart rate may be controlled with medical treatment; however, in some cases electrical cardioversion may be required. Radiofrequency ablation may be required for long-term control.

Myocarditis: Myocarditis is inflammation of the heart muscle that may lead to its damage, occurring due to an infection. It is usually caused by Coxsakie virus. It may also occur with Adenovirus or Parvovirus. Myocarditis is believed to be responsible for up to 20% of sudden cardiac death cases. Unfortunately, less than 50% of cases with myocarditis demonstrate anti-mortem symptoms.

Arrhythmogenic Right Ventricular Dyplasia (ARVD): In ARVD the muscles of the right ventricle are replaced with fat and fibrous non-contractile tissue. This impedes the heart's normal blood pumping capability. Furthermore, this condition increases the patient's risk of developing a fatal heart arrhythmia and sudden cardiac arrest.

Long QT Syndrome: The QT interval on an EKG represents the duration it takes for the heart to recharge after each heartbeat. A longer than normal QT interval increases the risk of developing a potentially fatal arrhythmia called Torsade de Pointes which may cause sudden cardiac death.

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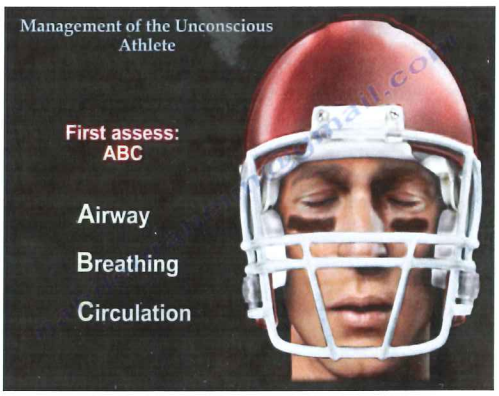
Management of the Unconscious Athlete

see Dr. Nabil Ebraheim's YouTube video:
https://www.youtube.com/watch?v=It_Pm8RiIEk



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Dr. Ebraheim, Amanda Ferguson and Suzanne Payne do not have any Relationships with industry to disclose.

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