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SUDDEN DEATH in the Young

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Your EMS crew is dispatched to the local neigborhood park for an injured person. Upon arrival, you see kids frantically waving from the basketball court. On arrival, you find a male patient in his late teens who is unresponsive and pulseless, and CPR is in progress.

As you start resuscitation, you ask the friends to tell you what happened. They explain they had been playing basketball for about 30 minutes when the patient was running down the court and collapsed. You ask about medications, medical history and allergies as friends try frantically to reach the boy's parents. The friends say they don't know his medical history, and they absolutely deny any drugs or alcohol.

CPR is ongoing, the patient is defibrillated and ALS care is continued en route. Despite your aggressive treatment and the hospital's efforts, he's pronounced dead by the emergency department physician. As you clean up the truck and write your report, you tell yourself, "We deal with older patients in cardiac arrest frequently, but not a young, healthy appearing kid like this. His friends have to be lying. There has to be more to this story."

In the United States, it is estimated that 200–300 young people die each year from cardiovascular events during physical exercise.[1] [http://] While this number is relatively low compared with other causes of death in similar age groups and considering how many athletic events take place annually, several times a year, television media report an athlete's death. These deaths are always high profile and generate community attention. But what causes a healthy appearing child, teen or young adult to suffer fatal cardiac arrest?

The generally accepted definition of sudden death is a sudden loss of consciousness from a cardiovascular event within an hour of the onset of symptoms. Sadly, diagnosis is often made upon autopsy.[2] [http://] In the U.S., 90% of deaths occur during competitive sports like basketball or football. Soccer has the highest death rate in Europe. Males are nine times more likely to die than females.[3] [http://]

Many paramedics have responded to a child with chest pain. While the majority are non-cardiac, a 2007 study in the *Journal of Pediatrics* should raise awareness that it is possible for coronary artery disease to be present even in the young. Over an 11-year period, nine children ages 12 to 20 were diagnosed with myocardial infarction after arriving at an Ohio hospital with chest pain radiating to the left arm and jaw. Interestingly, there was no drug use involved and no other risk factors.[4] [http://] In 2002, 33-year-old St. Louis Cardinals pitcher Darryl Kile was found dead in his hotel room after complaining of left shoulder pain during dinner with his brother the previous evening. Upon autopsy, he was found to have 90% occlusion of three heart vessels. San Francisco 49ers offensive lineman Thomas Herrion, age 23, died of coronary artery disease after suffering cardiac arrest following a preseason game in Denver.[5] [http://]

HYPERTROPHIC CARDIOMYOPATHY

There are structural changes in the heart that are considered normal in athletes. Because the heart is a muscle, it functions as any other muscle. When it is worked, it grows. This increases the size and mass of the muscle to allow for increased stroke volume and cardiac output. The question is, what are normal physiologic changes compared to changes in a diseased heart? The typical answer is rest. When the athlete is noncompetitive for a period upwards of three months, the nondiseased "athletic heart" will remold and reshape itself, whereas the diseased heart will remain enlarged.[6] [http://]

The normal heart has a ventricular wall measurement of 12 millimeters. Hypertrophic cardiomyopathy (HCM) is diagnosed in patients with a myocardial wall thickness of greater than 15 millimeters. In general, the thicker the wall the less effective the heart is and the harder it has to work. A study of 387 U.S. deaths found that HCM was the most common cause of death in the young. Twenty-six percent, equating to 102 deaths, were diagnosed post-mortem with HCM. Although it can be caused by drug use, it is primarily an inherited disease that develops during conception when an overgrowth in the muscle interferes with the heart's ability to contract.[7] [http://] In 1998, Italian researchers examined 49 competitive athletes who died from HCM and found that 44 were males with an average age of 23. Many times the first symptom a patient experiences is cardiac arrest. Studies have found that only 21% of patients had symptoms prior to their event. These symptoms include shortness of breath, chest pain, syncope or an abnormal blood pressure response during exercise. While shortness of breath is the most commonly seen symptom, syncope identifies patients who are at greater risk of sudden death and is a huge red flag for EMS providers.[8] [http://] Palpitations are encountered in up to 70% of athletes, depending upon their age and the sport. These palpitations are described in different ways and are most commonly benign.[9] [http://]

Although clinical appearance and electrocardiogram can raise suspicion for HCM, the main diagnostic test is echocardiography, which allows visualization and measurement of the heart walls. Over 200 genes have been associated with HCM. Genetic testing is available for about $4,500. There is no specific cure. Treatment includes medication, automatic pacemaker/defibrillator, and, in some cases, surgical remodeling. Patients with this disease are generally restricted from participating in competitive sports, and the court system has upheld schools' decisions to deny student participation in athletic events if they have HCM.[10] [http://]

COMMOTIO CORDIS

As the second leading cause of sudden death in athletes, *commotio cordis* is Latin for commotion of the heart. It is caused from blunt, non-penetrating trauma to the chest in the immediate area of the heart during the period of vulnerability (T wave). While 62% suffered sudden death during competitive sports, 32% died during daily activities like falls from a jungle gym. Deaths have occurred during hockey, karate and lacrosse, but baseball is the sport attributed to the most deaths. A study of 63 deaths found that all but one occurred during baseball: 35 playing in the field and 14 while at bat. It appears that a strike to the chest with a hard object is to blame. Athletes died less frequently in sports using air-filled balls, such as basketball.[11] [http://] Of the deaths studied, males were involved 95% of the time; 87% were Caucasian. The mean age was 13, and 80% of deaths occurred in athletes younger than age 18. Patients are most commonly found in ventricular fibrillation; sadly, fewer than 15% are successfully resuscitated.[12] [http://] It is often the case that after the blow to the chest, the athlete has a period of several seconds where they continue playing before becoming unconscious. There are commercial chest protectors on the market to protect the sternum from inadvertent chest blows; however, it should be noted that a 2006 study found 28% of kids suffering commotio cordis were wearing protective devices, questioning their effectiveness and potentially giving a false sense of security.[13] [http://]

Electrophysiological studies conducted in a controlled setting using swine found that a 30 mph impact during a 20 ms window of ventricular repolarization (t wave) will generate ventricular fibrillation 30% of the time. It is estimated that the ball in Little League pitching travels at approximately 50 mph. As velocity of the impact increases, so does the rate of ventricular fibrillation. Compare that to chest strikes outside that 20 ms window where no incidents of ventricular fibrillation were noted. Again, strikes outside the immediate silhouette of the heart generated no case of ventricular fibrillation. There is no documented evidence that these events are caused by ischemia of the myocardium. Rather, it is believed that this is an electrical conduction event.[14] [http://]

CORONARY ANOMALIES

"Pistol" Pete Maravich is the leading scorer in NCAA history with an average 44 points a game. He played in the NBA for 10 years and retired in 1980. Eight years later, at age 41, he was playing in a pick-up game when he suffered sudden cardiac arrest and died. Upon autopsy, it was determined that only one coronary artery was supplying blood flow to his heart.[15] [http://]

Cardiac anomalies like these pose challenges to both athletes and EMS providers. For example, John, a healthy 20-year-old, grew up playing competitive sports. After high school he continued working out, but became frustrated at his fatigue and some occasional chest pain during exertional activity. While playing soccer with friends, he dropped to one knee when he became lightheaded and dizzy, and woke up in a cardiac care unit hearing how his heart had stopped. A nurse watching the game started CPR, and there was return of spontaneous circulation after three shocks by EMS. He was transported to the local hospital, where hypothermia was initiated, and then transferred to a tertiary care facility. He told his mom that he had been having these spells of pain and dizziness for several months. While his initial EKG was suspicious for LVH, his echocardiogram was negative for an enlarged myocardium, effectively ruling it out. Further examination determined that a coronary artery anomaly was present and was caused by his right coronary artery tunneling under the pulmonary artery. Upon exertion, his pulmonary artery dilated to accommodate the increased demand for oxygen, compressing his coronary artery and restricting blood flow to the inferior wall of his heart at a time when demand was greatest. This caused a myocardial infarction, which led to cardiac arrest. After stabilization, he was taken to the operating room, where the coronary artery was re-routed to prevent future complications.

INFECTIOUS CAUSES

A 13-year-old female playing softball for her school team was rounding first base on her way to second when she collapsed. Those rushing to her side found her without pulses or respiration. When EMS arrived, CPR was in progress and she was in ventricular fibrillation. After being successfully resuscitated by EMS, she was transported to the local pediatric medical facility. Her only significant history was an upper respiratory infection approximately two weeks earlier. She was eventually diagnosed with myocarditis.

Myocarditis is an infectious disease that causes inflammation of the myocardium, which affects the heart's ability to pump and makes it irritable. Myocarditis is generally caused by the coxsackievirus, although it can also be from drug use. Frequently, patients are initially misdiagnosed. Symptoms such as fatigue, chest pain, fever, EKG changes and exertional shortness of breath mimic other conditions and make diagnosis challenging. Treatment includes rest, pain medication, antibiotics and steroids as needed. These patients will receive an echocardiogram to look at the structures of the heart. Seventy-five percent of patients will respond to treatment within two weeks, although approximately 10% will continue to have long-term difficulties; some will require transplant.[16] [http://]

LONG QT SYNDROME AND BRUGADA

Long QT syndrome is a disorder that affects the sodium-potassium and calcium pump and places people at risk for sudden death. Although generally inherited, there are currently more than 70 medications that have been known to cause prolongation of the QT interval (QTI). QTI greater than 450 ms in females and 440 ms in males is diagnostic for long QT syndrome, which is estimated to be present in one in every 5,000 people. When long QT syndrome is suspected, the entire immediate family is screened. The most common arrhythmia causing death is torsade de pointes.[17] [http://]

The Brugada brothers are three electrophysiologists who first described a genetically inherited problem with the sodium pump. When the sodium pump closes early, it pushes potassium out, causing early repolarization. First described in 1986, Brugada syndrome is characterized by EKG changes that include a prolonged PR interval, a right bundle branch block EKG pattern and ST segment elevation in V1-V3. Initial symptoms generally include syncope. It is interesting to note that fever can unmask Brugada. Experts speculate that the increase in heart rate caused by fever causes the sodium channel to work faster. An estimated 50% of sudden cardiac death patients with structurally normal hearts suffer from Brugada syndrome.[18] [http://] It is unknown why Asian men are at highest risk.

BRONCHOSPASM

There have been 61 reported deaths over a seven-year period from exercise-induced bronchospasm (EIB). Eighty-one percent of these deaths occurred in kids younger than 21 years of age and 57% in athletes at college

level. While most had a history of asthma, 10% had no known medical history. This prompted Ohio State University researchers to look within their own athletic department. They initially tested 100 student athletes and found 39% with EIB; 88% had no known history of wheezing.[19] [http://]

In April 2007, the FDA approved Singulair for use in prevention of EIB after three studies looked at 160 patients age 15 or older with EIB and found a statistically significant protective benefit when used two hours prior to exercise.[20] [http://] While most physicians recommend this practice, others contend that premedication was found to be effective only 50% of the time and more important is an adequate warm up period. Thirty minutes into exercise, epinephrine and norepinephrine release is highest. During this refractory period, epinephrine kicks in and functions as a bronchodilator.[21] [http://] It should be noted that premedication does not substitute the need for a rescue device, such as an albuterol inhaler.

Another situation that can cause wheezing in the athlete is vocal cord dysfunction. Most common in females in their early 20s, the vocal cords actually have been found to narrow during exertion, which decreases airflow to the lungs at a time when increased flow is in demand. This causes anxiety, shortness of breath and wheezing. Upon rest, the symptoms generally resolve. By the time EMS arrives, the patient may appear asymptomatic and is oftentimes not transported due to normal assessment findings.

OTHER CAUSES

Other less common causes involve Marfan's syndrome, which is an autosomal disorder that predisposes people for aortic dissection. In Marfan's, athletes are generally tall and slim with an arm span wider than their height. Weightlifters have been found to be prone to aneurysm due to the excessive pressure that is applied to the vessels during lifting. Systolic blood pressure may get as high as 400mmHg. It appears that a valsalva occurs, which increases blood pressure during lifts that exceed 80% of maximum voluntary contraction.[22] [http://]

Certain drugs like ephedrine, a weight loss supplement, have been banned from many professional sports. When Baltimore Orioles pitcher Steve Bechler died during spring training, his death was attributed to ephedrine use. Cocaine took the life of basketball star Len Bias of Maryland in 1986.[23] [http://]

Heat illness has been linked to multiple deaths. Minnesota Viking all pro lineman Corey Stringer became sick in excessive heat during training camp. At 300 pounds, his body had difficulty shedding heat, and his core temperature rose to 108.8°. He died August 1, 2001, at age 27 from multiple organ failure related to heat stroke. Ironically, four high school athletes died the same week from heat-related illness. Despite media attention to heat illness and changes in dealing with athletes and heat, five athletes ages 11 to 17 died in 2006, and a high school coach in Kentucky was recently indicted in the death of a student athlete who died from heat illness. Prevention involves proper hydration, adequate rest periods and limiting exposure to the heat. Temperatures of 90° with 70% humidity have been identified as increasing heat risks. Immediate cooling and hydration is the priority treatment.[24] [http://]

SCREENING

In the United States, athletes must go through a medical screening process prior to playing competitive sports. The American Heart Association encourages routine pre-screening physicals based on assessment questions filled out by parents and a physical examination, but stop short of recommending electrocardiograms. The questionnaire asks about previous medical conditions, medical history of family members, other risk factors and symptoms. These documents are generally poor at catching disease and have only a 2.5% chance of finding a problem.[25] [http://] In order for them to screen appropriately, both athletes and parents have to be honest when filling them out and appreciate how minor complaints can be linked to serious issues. Any suspicion during the pre-participation physical leads to further evaluation and testing.

Routine use of a 12-lead EKG as part of the physical exam has been debated. Knowing that 90% of hypertrophic cardiomyopathy can be picked up on EKG and conditions like long QT or Brugada syndrome are typically found on EKG, it seems reasonable to consider. Italian researchers found that a simple 12-lead EKG can help prevent episodes of cardiac arrest. They believed the research was conclusive enough that law changes in parts of Italy now require a 12-lead EKG for all athletes prior to competitive sports.[26] [http://] The United States has not embraced this concept. A study in the *New England Journal of Medicine* reviewed 12,550

electrocardiograms and found t wave abnormalities in 81 athletes. Further assessment revealed 6% with HCM.[27] [http://] The medical community believes that with 10 million kids playing sports, the concept would add $2 billion annually to the cost of healthcare and that there are not enough physicians to review all the test results. There is also the concern that a high false-positive EKG could add additional costs for unnecessary additional screening tests, as well as liability in the event of misinterpretation.[28] [http://]

Understanding that sudden death can occur in even the healthiest appearing athletes, it is important to educate schools, coaches, parents, sports clubs and athletes themselves about appropriate screening, having an AED at sporting events, good bystander CPR and early access to advanced care. Athletes experiencing symptoms like fatigue, shortness of breath and syncope during activity need further assessment to determine their risk for serious problems. Athletes should be encouraged to report problems without fear of penalty from coaches and parents. EMS providers need to appreciate that syncope in athletes is a true concern that can be an ominous sign prior to sudden cardiac death.

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