Pediatric

Case Studies in Cardiology

Aurora Burlington-Walworth Patient Service Market
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More than 5000 children experience a non-traumatic sudden cardiac arrest (SCA) each year in the United States. Critical factors that influence survival include the environment in which the arrest occurs, the child’s preexisting condition, the duration of no cardiovascular blood flow before resuscitation, the initial ECG rhythm detected, coupled with the quality of the basic and advanced life support interventions. Although most adult SCAs are caused by primary cardiac disease, pediatric SCAs are more than twice as likely to be attributable to non-cardiac causes than as to primary cardiac disease.

Outcomes after SCAs are often better among children more so than adults. Although SCA occurs even at young ages and at rest, the likelihood of child to young adult SCA for those with underlying cardiovascular disease is increased by participation in athletic events.

Cardiac arrest is the leading cause of death in young athletes, but how common is sudden cardiac arrest in adolescents and young adults? Pediatric sudden cardiac death occurs in fewer than 3 out of every 100,000 children, with approximately 25 percent of cases occurring during sporting events. In comparison, sudden cardiac death occurs in about 135 of every 100,000 adults.

Check it out…  https://fast.wistia.net/embed/iframe/9sr10t28qq?popover=true
SCA in young people is rare, and when it does happen it often leads to death if it’s not treated within minutes. Once EMS has been activated the best chance of survival for SCA is bystander CPR and rapid access to an AED. Every second after a cardiac arrest is critical, and the chances of survival diminish exponentially after 4 to 6 minutes without CPR. In fact, for each minute a patient goes without bystander initiated CPR, survival rates decrease by 7 to 10 percent. When CPR is quickly initiated, the rate of survival improves significantly.

What leads to sudden cardiac arrest in adolescents and young adults? There are several underlying causes of sudden cardiac arrest in young people. Most commonly it is due to underlying abnormalities of the heart, such as unrecognized congenital heart disease, heart rhythm abnormalities, or heart muscle abnormalities (such as hypertrophic\textsuperscript{1} cardiomyopathy\textsuperscript{2}, coronary artery abnormalities, and long QT syndrome).

**Hypertrophic cardiomyopathy (HCM).**

In this usually inherited condition, the walls of the heart muscle thicken. The thickened muscle can disrupt the heart's electrical system, leading to fast or irregular heartbeats (arrhythmias), which can lead to sudden cardiac death. Although not usually fatal, it is the most common cause of heart-related sudden death in people under 30. It's the most common identifiable cause of sudden death in athletes. HCM often goes undetected. Most often diagnosed during infancy or adolescence, hypertrophic cardiomyopathy (HCM) is the second most common form of heart muscle disease, is usually genetically transmitted, and comprises about 35 to 40% of cardiomyopathies in children. “Hypertrophic” refers to an abnormal growth of muscle fibers in

\textbf{Further reading for the EMT-P}

Although HCM can involve both lower chambers, it usually affects the main pumping chamber (left ventricle) with thickening of the septum (wall separating the pumping chambers), posterior wall or both. With hypertrophic obstructive cardiomyopathy (HOCM), the muscle thickening restricts the flow of blood out of the heart. Often, leakage of the mitral valve causes the blood in the lower chamber (left ventricle) to leak back into the upper chamber (left atrium). In less than 10% of patients, the disease may progress to a point where the heart muscle thins and the left ventricle dilates resulting in reduced heart function similar to that seen in dilated cardiomyopathy (DCM).

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\begin{itemize}
\item \textsuperscript{1} abnormal enlargement of a part or organ; excessive growth.
\item \textsuperscript{2} any disease of the heart muscle, leading to decreased function: usually of unknown cause.
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the heart. In HCM, the thick heart muscle is stiff, making it difficult for the heart to relax and for blood to fill the heart chambers. While the heart squeezes normally, the limited filling prevents the heart from pumping enough blood, especially during exercise.

HCM is most often diagnosed during infancy or adolescence. Gene defects can be familial, and it is estimated that 50–60% of children with HCM have a relative with the disease, although they may not have been diagnosed or have symptoms.

**Signs and Symptoms of HCM**

There is tremendous variation in how HCM presents and progresses. While some children have no or mild symptoms, others may have more severe symptoms including heart failure. Some patients develop abnormal heart rhythms (arrhythmias) that may put them at increased risk for sudden cardiac death. Children under 1 year of age often have symptoms of congestive heart failure whereas older children may be symptom free and therefore unaware that HCM is present. The onset of symptoms often coincide with the rapid growth and development stages of late childhood and early adolescence. Strenuous exercise and stimulation inherent of competitive sports has also been known to make symptoms of HCM more apparent. Disease severity and symptoms is related to the extent and location of the hypertrophic area of the heart, and whether there is an obstruction to blood leaving the heart or valve leakage from the left ventricle into the left atrium. The first
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Beta-blocking medications are used to slow the heartbeat and allow the heart to fill more completely when the thick muscle in the ventricular septum narrows the outflow of blood from the heart. These medications can cause excessive slowing of the heart rate, low blood pressure, dizziness, and in some cases, fluid retention, fatigue, impaired school performance and depression. Atenolol and propranolol are examples of beta-blocking medications.

Calcium channel blockers improve the filling of the heart by reducing the stiffness of the heart muscle, and are used in patients with chest pain or breathlessness. Side effects can include excessive slowing of the heart rate and lower blood pressure. Common calcium channel blockers are verapamil and diltiazem.

sign of HCM may simply be a heart murmur, with other symptoms manifesting such as dyspnea on exertion (shortness of breath during exercise), angina (chest pain), presyncope (light-headedness or dizziness), syncope (fainting), exercise intolerance, or palpitations/arrhythmias (irregular heartbeats). Symptoms in infants may be more difficult to detect but include difficulty breathing, poor growth, diaphoresis (excessive sweating), or crying and agitation during feeding thought to be due to chest pain. Children with severe HCM may have symptoms of heart failure such as difficulty breathing, swelling around the eyes and legs (edema), tiredness or weakness, coughing, abdominal pain and vomiting. Mild symptoms of heart failure can also resemble asthma. Children with HCM may also develop an abnormal heartbeat (arrhythmia), either beating too fast (tachycardia) or too slow (bradycardia). Symptoms resulting from arrhythmias can appear without a child showing signs and symptoms of congestive heart failure or other more obvious symptoms of HCM. The risk of sudden death from arrhythmia is higher with this form of cardiomyopathy compared with other forms of pediatric cardiomyopathy, especially among adolescent patients.

Treatment Modalities for HCM

Currently, there are no therapies that can “cure” HCM; however many treatments are available that can improve symptoms and potentially decrease risk in children with HCM. The choice of a specific therapy depends on the clinical condition of the child, the risk of dangerous events, and the ability of the child to tolerate the therapy. In the following sections, treatments for HCM are summarized.

Medical Therapies - Medications are used to treat children with HCM who have symptoms such as difficulty breathing, chest pain, decreased activity tolerance or fatigue and generally include beta-blocking and calcium channel-blocking medicines.

ICD - Another option for some is an implantable cardioverter-defibrillator (ICD). This pager-sized device implanted in your chest like a pacemaker continuously monitors your
heartbeat. If a life-threatening arrhythmia occurs, the ICD delivers electrical shocks to restore a normal heart rhythm.

**Surgery for HCM** - Septal myomectomy is a surgery that is performed in patients with hypertrophic cardiomyopathy when symptoms of heart failure have developed. The surgery involves removing the muscle that has obstructed the blood flow. While septal myomectomy is effective in controlling symptoms, it does not stop hypertrophy from progressing, nor does it treat life-threatening abnormal rhythms.

**Heart Transplantation** - Currently, transplantation for HCM is not routinely performed. Two exceptions include medically refractory ventricular arrhythmias, and HCM which has developed features of DCM not responsive to standard DCM therapy. A heart transplant will offer the child the chance to return to a normal lifestyle. While a donor heart can cure the symptoms of heart failure and greatly improve survival, it is a major operation with considerable risks and long-term complications. Once a transplant is done, other concerns arise, such as infection, organ rejection, coronary artery disease, and the side effects of medications.

**Dilated cardiomyopathy (DCM)**

Although less common than HCM, dilated cardiomyopathy (DCM) is also a known cardiac risk factor for sudden cardiac arrest. It affects the heart's ventricles and atria, the lower and upper chambers of the heart, respectively.

Frequently the disease starts in the left ventricle, the heart's main pumping chamber. The heart muscle begins to dilate, meaning it stretches and becomes thinner. Consequently, the inside of the chamber enlarges. The problem often spreads to the right ventricle and then to the atria. As the heart chambers dilate, the heart muscle doesn't contract normally and cannot pump blood very well. As the heart becomes weaker heart failure can occur.

Common symptoms of heart failure include shortness of breath, fatigue and swelling of the ankles, feet, legs, abdomen and veins in the neck. Dilated cardiomyopathy also can lead to heart valve problems, arrhythmias (irregular heartbeats) and blood clots in the heart.

**Foot Notes:**

3 Septal myectomy is a cardiac surgery treatment for hypertrophic cardiomyopathy (HCM). The open-heart surgery entails removing a portion of the septum that is obstructing the flow of blood from the left ventricle to the aorta. Septal myectomies have been successfully performed since the 1960s.

4 A stubborn or unmanageable abnormal heart rhythm in at least one of the two main chambers of the heart, left or right, that does not respond to medical treatments.
Other Names for Dilated Cardiomyopathy include:

- Alcoholic cardiomyopathy (A term used when overuse of alcohol causes the disease)
- Congestive cardiomyopathy
- Diabetic cardiomyopathy
- Familial dilated cardiomyopathy (A genetic form of heart disease)
- Idiopathic cardiomyopathy (The exact cause remains initially unknown)
- Ischemic cardiomyopathy (A term used when coronary heart disease, also called coronary artery disease or heart attack cause the disease. Not all forms of DCM are ischemic in origin.)
- Peripartum cardiomyopathy. (A term used when the disease develops in a woman shortly before or after she gives birth.)
- Primary cardiomyopathy (In some cases, cardiomyopathies are inherited and may be passed down to other family members. Secondary cardiomyopathy - caused by a medical condition such as hypertension, valve disease, congenital heart disease, coronary artery disease, toxins, or medications)

What Causes Dilated Cardiomyopathy:

Often, cause of dilated cardiomyopathy isn't known. Up to one-third of the people of those who have it inherit it from their parents. Some diseases, conditions and substances also can cause the disease, such as:

- Coronary heart disease, heart attack, high blood pressure, diabetes, thyroid disease, viral hepatitis and HIV
- Infections, especially viral infections that inflame the heart muscle
- Alcohol, especially if you also have a poor diet
- Complications during the last month of pregnancy or within 5 months of birth
- Certain toxins such as cobalt
- Certain drugs (such as cocaine and amphetamines) and two medicines used to treat cancer (doxorubicin and daunorubicin)

Case Study Two…

You are standing by at a high school soccer game when in the 2nd period you are requested to respond to the field for the report a 14 year old male was the ball carrier preparing for a center pass when he stumbles forward, collapses, and goes into sudden cardiac arrest.
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**Congenital long-QT syndrome (LQTS)**

The congenital long-QT syndrome (LQTS) is a life-threatening cardiac arrhythmia syndrome that represents a leading cause of sudden death in the young. LQTS is typically characterized by a prolongation of the QT interval on the ECG and by the occurrence of syncope or cardiac arrest, mainly precipitated by emotional or physical stress. Several reasons make LQTS an important disease. It can often be a lethal disorder, and symptomatic patients left without therapy have a high mortality rate, 21% within 1 year from the first syncope. However, with proper treatment, mortality is now ≈1% during a 15-year follow-up. LQTS is without doubt the cardiac disease in which molecular biology and genetics have made the greatest progress and unquestionably is the best example of genotype-phenotype correlation. In this regard, it represents a paradigm for sudden cardiac death, and its progressive unraveling helps to better understand the mechanisms underlying sudden death in more complex disorders, such as ischemic heart disease and heart failure.

**Further reading for the EMT-P**

The electrocardiographic QT interval represents the depolarization and the repolarization phases of the cardiac action potential. The interplay of several ion channels determines the action potential duration. Decreases in repolarizing outward K+ currents or increases in depolarizing inward sodium or calcium currents can lead to prolongation of the QT interval, thus representing a pathophysiological substrate for LQTS. The ventricular tachyarrhythmia that underlies the cardiac events of LQTS is Torsades-de-Pointes, a curious type of ventricular tachycardia that most of the time is self-limiting and produces transient syncope but that can also degenerate into ventricular fibrillation and cause cardiac arrest or sudden death. It would be extremely important to know what causes Torsades-de-Pointes to stop after a limited number of seconds or to continue, with devastating consequences, but we do not. The morphology of the T wave is often useful for the diagnosis, and the precordial leads are especially informative when they reveal biphasic or notched T waves. T-wave alternans in polarity or amplitude, when observed, is diagnostic. T-wave alternans is a marker of major electric instability and identifies patients at particularly high risk; its presence in a patient already undergoing treatment should alert the physician to persistent high risk and warrants an immediate reassessment of therapy. Sinus pauses, unrelated to sinus arrhythmia, are an additional warning signal especially in patients with SCN5A mutations.
This inherited heart rhythm disorder can cause fast, chaotic heartbeats, often leading to fainting. Young people with long QT syndrome have an increased risk of sudden death. Long QT syndrome (LQTS) is a genetically transmitted cardiac arrhythmia caused by ion channel protein abnormalities. It can be mistaken for palpitations, neurocardiogenic syncope, and epilepsy. It has been estimated that 1 in 2500 people in the United States have LQTS. LQTS-precipitated sudden deaths continue to claim otherwise healthy infants, children, adolescents, and adults at an unacceptably high rate. However, with increased awareness, genetic testing, and effective treatment options, LQTS can be diagnosed early and sudden death prevented. Still, this condition is often undetected prior to death and not recognized as the cause of death. Family members of individuals with unexplained death should be tested for LQTS and other genetic arrhythmias. LQTS is a treatable disorder and, with correct diagnosis and common treatments, most deaths are preventable.

**Major Signs and Symptoms:**

If you have long QT syndrome (LQTS), you can have sudden and dangerous arrhythmias (abnormal heart rhythms). Signs and symptoms of LQTS-related arrhythmias often first occur during childhood and include:

- **Unexplained fainting.** This happens because the heart isn't pumping enough blood to the brain. Fainting may occur during physical or emotional stress. Fluttering feelings in the chest may occur before fainting.
- **Unexplained drowning or near drowning.** This may be due to fainting while swimming.
- **Unexplained sudden cardiac arrest (SCA) or death.** SCA is a condition in which the heart suddenly stops beating for no obvious reason. People who have SCA die within minutes unless they receive treatment. In about 1 out of 10 people who have LQTS, SCA or sudden death is the first sign of the disorder.
- **Often, people who have LQTS develop an abnormal heart rhythm during sleep.** This may cause noisy gasping while sleeping.

**Specific Types of Treatment:**

- **Lifestyle Changes** - If possible, try to avoid things that can trigger abnormal heart rhythms. For example, people who have LQTS should avoid medicines that lengthen the QT interval or lower potassium blood levels. Many people who have LQTS also benefit from adding more potassium to their diets.
**Medicines** - Beta blockers are medicines that prevent the heart from beating faster in response to physical or emotional stress. Most people who have LQTS are treated with beta blockers. Doctors may suggest that people who have LQTS take sodium channel blockers, such as mexiletine. These medicines make sodium ion channels less active.

**Medical Devices** - Pacemakers and ICDs are small devices that help control abnormal heart rhythms. Both devices use electrical currents to prompt the heart to beat normally. Surgeons implant pacemakers and chest or belly with a minor procedure. The use of these devices is similar in children and adults. However, because children are still growing, other issues may arise. For example, as children grow, they may need to have their devices replaced.

**Surgery** - People who are at high risk of death from LQTS sometimes are treated with surgery. During surgery, the nerves that prompt the heart to beat faster in response to physical or emotional stress are cut. This type of surgery keeps the heart beating at a steady pace and lowers the risk of dangerous heart rhythms in response to stress or exercise.

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**Case Study Three…**

You are standing by at a high school baseball game when in the 7th inning you are requested to respond to the field for the report a 15 year old male who was hit in the chest with a baseball and then stumbled forward, collapsed, and went into sudden cardiac arrest.

**Commotio cordis**

Commotio cordis is a phenomenon in which a sudden blunt impact to the chest causes sudden death in the absence of cardiac damage. This condition was first described in the middle of the 18th century in the context of chest trauma among workers. Through most of the 20th century, it was only sporadically reported. In the last 2 or 3 decades, commotio cordis events have primarily occurred in sports, and thus, this phenomenon has become more well known to the sports communities and physicians. Commotio cordis is to be differentiated from cardiac contusion (contusio cordis), a situation in which blunt chest trauma causes structural cardiac damage, such as observed in motor vehicular accidents. Another rare cause of sudden cardiac death that can occur in anyone, occurs as the result of a blunt blow to the chest, such as being hit by a hockey puck or another player. The blow to the chest can trigger ventricular fibrillation if the blow strikes at exactly the wrong time in the heart's electrical cycle. Based on Registry cases, the survival rate was 24% with 95% of those cases affecting males. Commotio cordis most frequently occurs in those aged between 10 and 18 years, however cases have been documented between the age of 7 weeks to 51 years-old. Statistics indicate that 50% of episodes occur during
competitive sports, a further 25% occur during recreational sports, and the other 25% occurs during other activities that involve blunt force trauma to the chest wall, e.g. kick by a horse, violence act. Baseball retains the highest incidence of commotio cordis, followed by softball, hockey, and lastly football.

**Resuscitation**

Once thought to be nearly universally unsuccessful, has now been demonstrated to be successful in up to 35% of commotio cordis victims. The initial rhythm is ventricular fibrillation in those in whom a defibrillator is used relatively early after the event. Currently, the outcome of resuscitation in commotio cordis appears to be very similar to that for resuscitation in other forms of sudden cardiac death. Whether syncope associated with chest wall impact is an aborted commotio cordis event secondary to nonsustained ventricular fibrillation or transient complete heart block has not yet been determined.
**Further reading for the EMT-P**

**Mechanism** - What is commonly known as ventricular fibrillation is a surface tracing that results from multiple wavelets in the myocardium at the same time. This leads to a surface ECG of the summation of the wavelets and thus complete disorganization. However, internally and focally, ventricular fibrillation is much more organized, especially in the initial stages. It is hypothesized that ventricular fibrillation can be the result of a mother rotor, either stationary or roaming throughout the myocardium, or multiple random wandering wavelets. In fact, the mechanism of ventricular fibrillation in commotio cordis may be similar to or different from conditions such as acute ischemia, electric shock on T-wave, long-QT syndrome, and Brugada syndrome. By surface morphology, ventricular fibrillation in commotio cordis appears as it does in other situations: initially coarse and undulating (like torsade de pointes/polymorphic ventricular tachycardia). Over a relatively short time, this coarse polymorphic ventricular tachycardia will degenerate into a lower-amplitude and higher-frequency (“finer”) ventricular fibrillation. This ventricular fibrillation will continue for up to 30 minutes if the animal is not defibrillated.

**Alterations in Repolarization** - It is suspected that the ventricular fibrillation in commotio cordis is an acquired form of abnormal repolarization. Abnormalities in repolarization are caused by impact and likely increase the heterogeneity of repolarization, which makes the myocardial substrate ripe for a reentrant arrhythmia. However, temporal alteration of the myocardial substrate alone is not sufficient. There also must be a trigger, an initial ventricular arrhythmic depolarization. This trigger could be an afterdepolarization, such as is present in long-QT situations, or a premature ventricular depolarization (induced by the blow), as is present in the R-on-T phenomenon in acute ischemia. Support for this dual-abnormality hypothesis lies in the fact that in impacts that do not induce ventricular fibrillation, 2 predominant abnormalities are observed: that of alteration of repolarization as manifested by ST-segment elevation and that of a premature ventricular contraction. Support for changes in repolarization is also observed in an elegant Langendorff preparation in which a rabbit heart is exposed to acute left ventricular pressure pulses. In this model, acute pressure changes mediated via a fluid-filled balloon in the left ventricle caused a dispersion of repolarization, and if the timing of the balloon inflation occurred during a narrow window of repolarization, ventricular fibrillation could result.

**Immediate Trigger** - The initial depolarization trigger is likely a focal phenomenon from direct impact, similar to a premature ventricular contraction induced by a catheter, as seen in electrophysiology and cardiac catheterization laboratories. Alternatively, it could be an afterdepolarization induced by changes in current flow. This 2-step process is similar to the R-on-T phenomenon, in which a premature ventricular contraction that occurs on the upslope of the T wave will cause ventricular fibrillation in acute ischemic conditions but not in nonischemic situations. This 2-step process is also observed in the long-QT syndrome, in which an afterdepolarization is necessary to initiate the torsade de pointes seen in this condition. Ongoing
mapping studies using real-time 3-dimensional mapping in our laboratory should help elucidate the mechanism of ventricular fibrillation in commotio cordis.

**Ion Channels Involved in Altered Repolarization** - The candidate ion channels for the altered repolarization are those whose conduction is known to be altered by stretch or pressure changes. These channels include the calcium stretch-activated channel, the KATP channel, certain other known potassium and sodium channels, and possibly even some unknown channels. The first candidate channel evaluated was the KATP channel. We chose this channel because of the similarities of ventricular fibrillation and ST-segment elevation between our experimental model and acute ischemia. In addition, this channel is known to be activated by stretch. Indeed, in our animal model, blocking the KATP channel with glibenclamide nearly eliminated ventricular fibrillation. In this study, 20 swine were randomized to glibenclamide or a control vehicle, and with T-wave impacts, glibenclamide animals had significantly fewer occurrences of ventricular fibrillation (1 episode in 27 impacts [4%]) than controls (6 episodes in 18 impacts [33%]; \( P=0.01 \)).

Another stretch-activated channel, the nonspecific calcium stretch-activated channel, was also evaluated in our model; however, the infusion of streptomycin, an agent that blocks this channel, did not alter the incidence of ventricular fibrillation. In another series, blocking the calcium channel with verapamil did not affect the incidence of ventricular fibrillation.

The treatment of commotio cordis is not different from any other cardiopulmonary emergency associated with a non-perfusing cardiac rhythm. For victims of witnessed ventricular fibrillation arrest, as occurs in commotio cordis, early cardiopulmonary resuscitation (CPR) and rapid defibrillation can significantly increase the chances of survival.

The 2010 AHA Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care recommend early CPR that emphasizes chest compressions immediately after the emergency response system has been activated. The guidelines deemphasize the importance of rescue breaths and pulse checks. Although a chest compression to ventilation ratio of 30:2 and a compression rate of at least 100 per minute are still recommended for adults and children (above age 1 y), bystander "hands-only" CPR (compression only) also significantly improves survival compared with no bystander CPR. Rapid defibrillation significantly increases the chances for survival to hospital discharge. CPR, beginning with chest compressions, should resume immediately after a shock and should continue for 2 minutes before a rhythm or pulse check is conducted.

Performing CPR while the AED is readied for use is strongly recommended. A shorter time interval between the last chest compression and the shock is directly correlated with the success of defibrillation.
The relatively low rate of survival from commotio cordis is probably caused by the delay in instituting effective CPR measures because bystanders frequently fail to appreciate the severity of the event, lack knowledge of commotio cordis, or mistakenly believe that the trauma was insignificant. Many observers have commented that they believed that the wind was knocked out of the person. Experience suggests that survival is associated with resuscitation efforts begun within 1-3 minutes of collapse.

**Electrical defibrillation**

The hallmark of effective resuscitation is rapid, direct current defibrillation. Time to defibrillation is probably the single most important determinant of survival in cardiac arrest. The likelihood of successful defibrillation decreases rapidly over time, in part because ventricular fibrillation generally evolves to asystole within a few minutes. Experimental data gathered using the commotio cordis swine model suggest that defibrillation within 1 minute of ventricular fibrillation onset results in a 100% survival rate and that defibrillation after 2 minutes results in an 80% survival rate. In animals in which ventricular fibrillation was present for more than 4 minutes, the survival rate was 0% unless CPR was instituted after defibrillation (in which case, the likelihood of survival increased to 65%). In humans, every 1-minute delay in defibrillation beyond the first 3 minutes decreases the likelihood of survival by approximately 10%.

Because emergency paramedical technicians cannot be expected to arrive at the scene of a cardiac arrest in less than 5 minutes, the expanded use of public-access automated external defibrillators (AEDs) may save the lives of countless young people who experience cardiac arrest due to blunt trauma to the precordium. AEDs, even when used by persons with minimal training, can recognize and automatically terminate fatal arrhythmias. AEDs are now approved for use in children as young as 1 year. Ideally, AEDs should have a specific "low-output" setting or a special pediatric pad and cable set which attenuates the charge for use in children aged 1-8 years. Even without these, all AEDs can be used in children of any age older than 1 year.

**Precordial thump**

Use of the precordial thump during CPR is controversial. No prospective studies have evaluated the efficacy of precordial thump in resuscitation. Recently, limited studies in animals and humans have shown precordial thumps to be ineffective in terminating ventricular fibrillation. The 2010 AHA Guidelines for adult ACLS mention that one immediate precordial thump may be considered after a witnessed cardiac arrest if an AED or defibrillator is not immediately available. Precordial thump is not mentioned at all as an option in pediatric CPR or pediatric advanced life support (PALS).

**Conclusions**

Commotio cordis is a significant cause of morbidity and mortality on the playing field. The epidemiology of commotio cordis has been well established to include young male athletes participating in sports with a solid small ball. Resuscitation is possible. A reduction in the risk of commotio cordis is possible with age-appropriate safety baseballs.