A 17-Year-Old Athlete in Cardiac Arrest

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Editor's Note:
The Case Challenge series includes difficult-to-diagnose conditions, some of which are not frequently encountered by most clinicians but are nonetheless important to recognize accurately. Test your diagnostic and treatment skills using the following patient scenario and corresponding questions. If you have a case that you would like to suggest for a future Case Challenge, please contact us.

Background

The emergency department (ED) receives a paramedic call reporting a 10-minute estimated time of arrival for a 17-year-old boy who was found in cardiac arrest after a blow to the chest. The patient has regained spontaneous circulation and is currently awake and alert. A rhythm strip faxed to the ED before the patient's arrival shows ventricular fibrillation, with subsequent conversion to a normal sinus rhythm after defibrillation with 200 J (Figure 1).

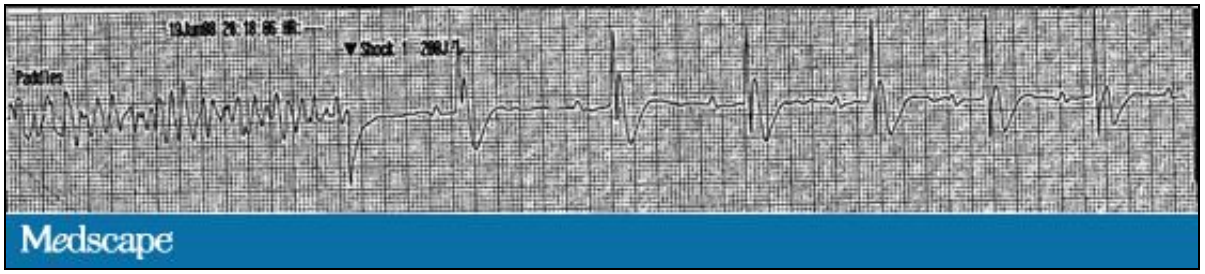

Figure 1.

The prehospital personnel further report that the patient, a center outfielder for a local baseball team, was trying to catch a baseball when one of his teammates accidentally ran into him, elbowing him in the middle of his chest. The patient immediately dropped to the ground, was unresponsive, and showed no spontaneous movement.

His coach immediately initiated cardiopulmonary resuscitation (CPR) after no pulses were palpated. The paramedics arrived 5 minutes later and, as noted on the rhythm strip, found the patient to be in ventricular fibrillation. One 200-J countershock was administered converting the ventricular fibrillation to a normal sinus rhythm. The patient slowly regained consciousness. He was confused initially and was amnestic to the event.

Upon arrival at the ED, the patient reports mild anterior chest-wall pain but no shortness of breath, palpitations, weakness, or feelings of confusion. He states that he has never before fainted. The patient and his mother report no significant past medical or family history, including any arrhythmias, unexplained sudden deaths, or cardiac structural diseases. He states that he does not have a lower exercise tolerance than his teammates and also denies any smoking, drinking, use of medications, illicit substance abuse, or doping practices.

Physical Examination and Workup

Upon physical examination, the primary survey of his airway, breathing, and circulation is unremarkable. The patient has a blood pressure of 130/71 mm Hg and a heart rate of 106 beats/min, with a normal rhythm. His respirations are 28-30 breaths/min. The initial oxygen saturation is 83% while the patient is breathing room air, but it corrects to 98% with a non-rebreather mask. Soon after, his saturation improves to 99% with a 2-L nasal cannula.

His mentation is normal and he is alert, with a Glasgow coma scale rating of 15. The skin examination reveals mild ecchymosis just anterior to his sternum. The lungs are clear to auscultation bilaterally, and the cardiac examination

reveals a regular rate, with normal S1 and S2 heart sounds and no clicks, gallops, rubs, or murmurs. The abdominal and neurologic examinations are unremarkable. No hyperflexibility or marfanoid appearance is noted.

The patient is placed on a cardiac monitor upon arrival at the ED. A 12-lead electrocardiogram (ECG) reveals sinus tachycardia at a rate of 110 beats/min, with mild right-axis deviation. The QRS complex, QT interval, ST/T waves, and P waves are all normal (Figure 2). A portable, upright chest x-ray shows no signs of fractures, widening of the mediastinum, cardiomegaly, effusion, or pneumothorax (Figure 3).

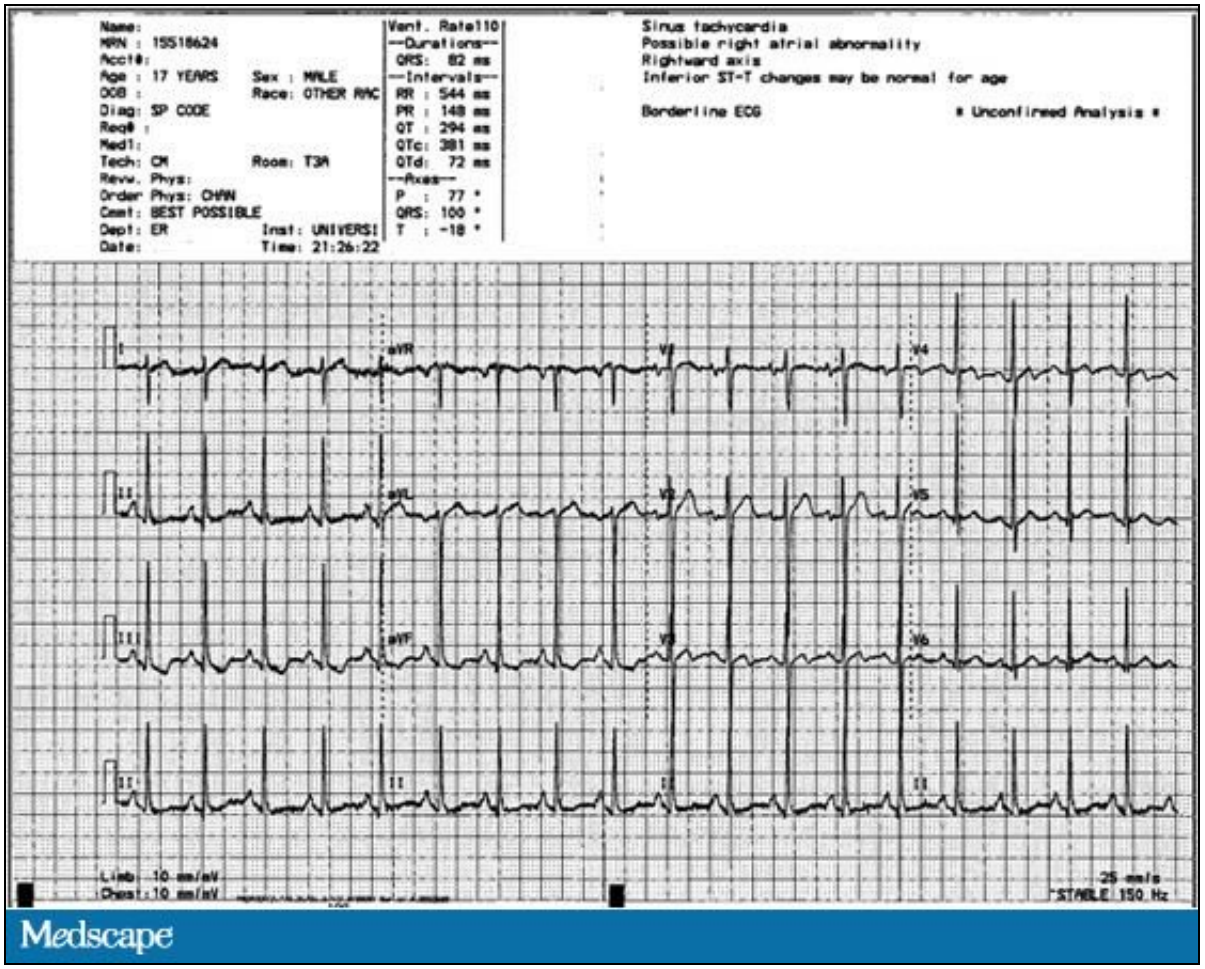

Figure 2.

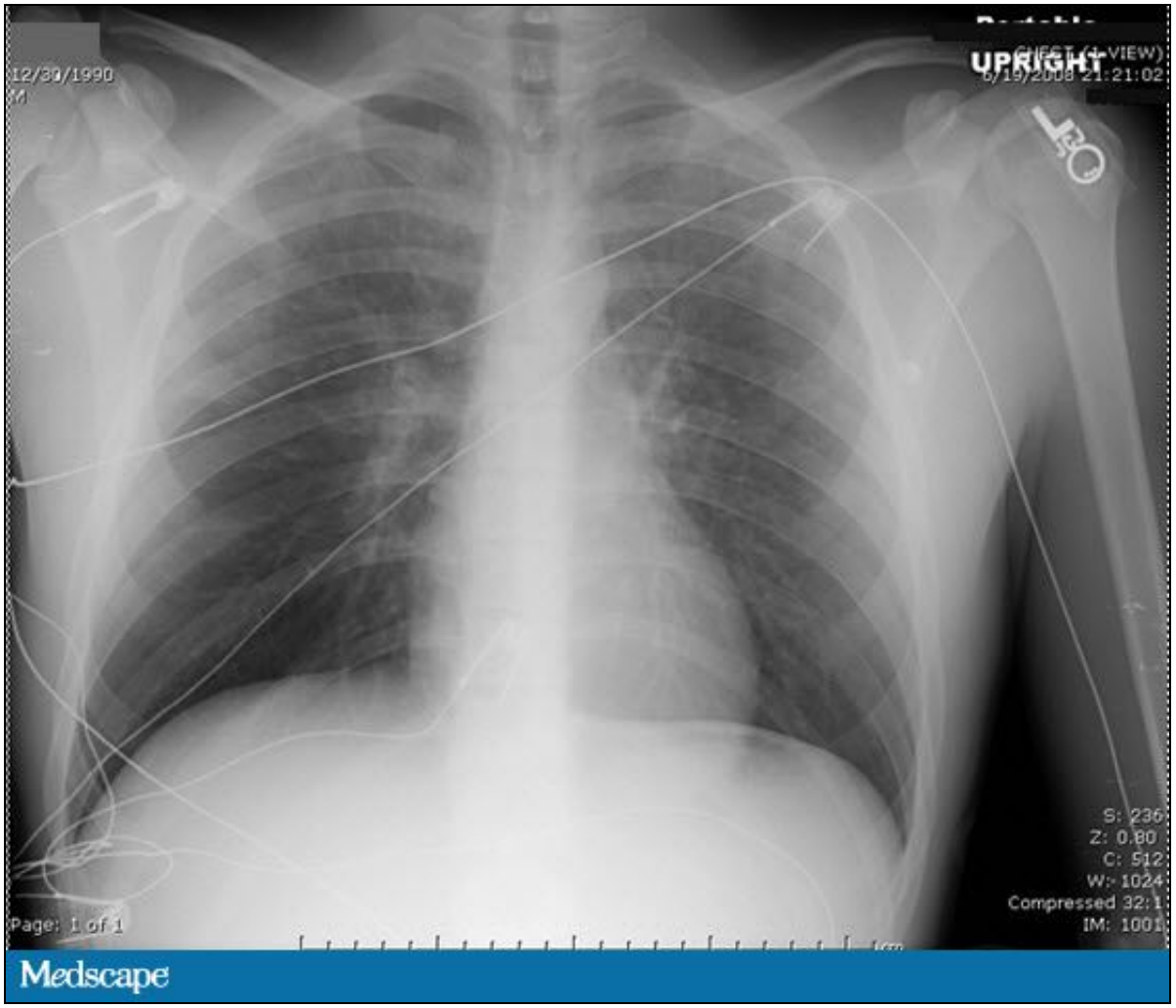

Figure 3.

A complete blood count is normal except for a mildly elevated white blood cell count of 13.6 ×10^3/µL (13.6 ×10^9/L). A metabolic panel is normal, including normal potassium and magnesium findings. The initial troponin I level is 0.04 ng/mL (0.04 µg/L; normal range, 0.02-0.04 ng/mL; indeterminate range, 0.05-0.30 ng/mL). A urine drug screen is negative. Computed tomography of the chest (Figure 4) is remarkable only for mild pulmonary and periportal edema.

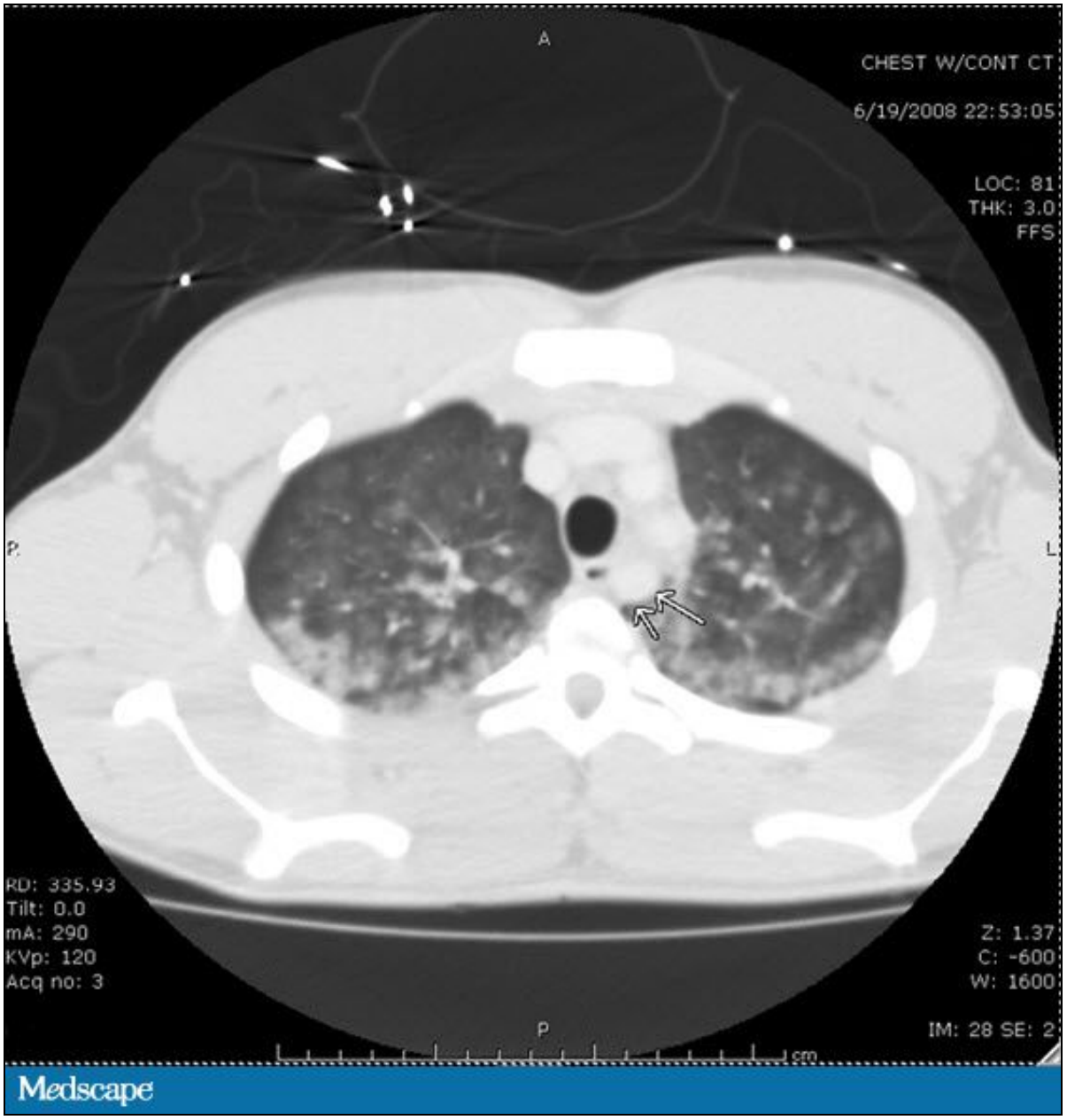

Figure 4.

The patient is admitted to the pediatric intensive care unit (PICU) for continuous cardiac monitoring and cardiology consultation. An echocardiogram ordered in the ED is to be done in the PICU.

Which of the following is the likely pathophysiology that led to this patient's cardiac arrest?

Your Peers Chose:

- Hypertrophic cardiomyopathy 21%
- Myocardial infarction 2%
- Commotio cordis 62%

Long-QT syndrome 15%

Discussion

Commotio cordis (which is Latin for "disturbance of the heart") is, in essence, a concussion of the heart. Initially described as early as 1857, it is defined as an instantaneous cardiac arrest produced by a witnessed, nonpenetrating blow to the chest, in the absence of preexisting heart disease or identifiable morphologic injury to the sternum, ribs, chest wall, or heart. Commotio cordis is a diagnosis of exclusion. Other causes, such as myocardial infarction, electrolyte abnormality, long-QT syndrome, and hypertrophic obstructive cardiomyopathy (HOCM), must first be ruled out with examinations such as serial assessment of cardiac biomarkers and electrocardiography, electrolyte level testing, and echocardiography.[1,2]

Commotio cordis is believed to be underreported and underrecognized. Less than 30 cases are reported each year.[2] The vast majority of cases (95%) occur in boys. The mean age is 15 years, with few cases reported in individuals older than 20 years. This may be due to the fact that protection of the heart by subcutaneous fat, muscle bulk, and fully ossified ribs all become more common in adulthood.[2,3]

A review of data from the US Commotio Cordis Registry (USCCR), in Minneapolis, Minnesota revealed that most cases were caused by a blow to the chest from an object used during an organized youth sporting event.[2] A baseball accounted for 53 of the cases, with a softball and a hockey puck the next most frequent, at 14 and 10 cases, respectively. Other documented sporting cases have been caused by blows delivered by body parts, such as an elbow, knee, foot, or fist hitting the anterior chest wall (5-6 cases of each).

Daily activities, including parent-child discipline (5 cases), and even a fall from monkey bars (1 case), can also lead to commotio cordis. Regardless of the mechanism, impacts resulting in commotio cordis are typically of low energy and velocity.[1,4] The victim may collapse immediately after the blow, but in as many as 50% of cases, a short delay occurs between the impact and collapse.

In 1930, George Schlomka was the first to describe the factors that can lead to arrhythmia after a moderate precordial impact. He believed that the force, location, and type of object causing the impact determined the type of injury and the subsequent risk for arrhythmia.[2] The force transmitted to the heart is directly related to the hardness of the striking object. Madias and colleagues[4] reported that the threshold speed of impact at which a standard baseball can cause ventricular fibrillation is between 25 and 30 miles per hour. However, when the speed is over 50 miles per hour, the likelihood of ventricular fibrillation actually decreases, although the possibility of myocardial contusion becomes greater. Furthermore, the authors stated that the impact must be directly over the heart near or just to the left of the sternum in order to instigate ventricular fibrillation. Impact on the center of the heart induced ventricular fibrillation in 30% of reported cases, compared with 13% and 4% at the left ventricular base and apex, respectively.[4]

The use of a standard baseball leads to the incidence rate reported above, but if the core of the ball is softer, then the rate for ventricular fibrillation drops. Link and colleagues[5] reported that changes to the cores of baseballs to make them softer led to a decrease in the rate of ventricular fibrillation with commotio cordis from 70% to 19%. As such, the use of safety baseballs with rubber cores of different degrees of hardness has been advocated to reduce the risk for such traumatic injury in young athletes.[3,5]

Not all impacts to the anterior chest lead to the ventricular fibrillation observed in commotio cordis. The impact must be delivered 10-30 milliseconds before the peak of the T wave in the cardiac cycle (Figure 5) in order to induce ventricular fibrillation.

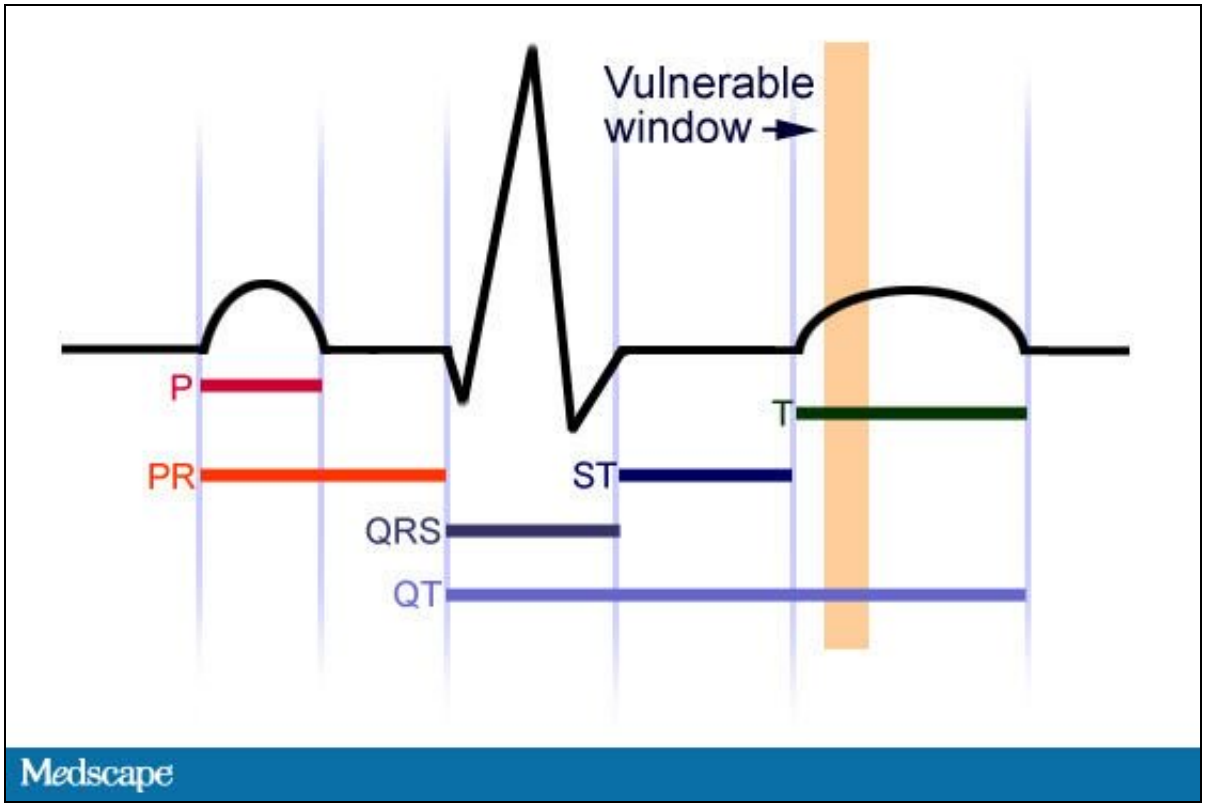

Figure 5.

Induction is probably secondary to the activation of potassium-carrying ion channels via mechanoelectric coupling. The activation of these ion channels generates an inward current, thus locally augmenting repolarization and resulting in premature ventricular depolarization and the initiation of unstable ventricular arrhythmias. If impact occurs during other portions of the cardiac cycle, different conduction disturbances, such as heart block, bundle branch block, or transient ST segment elevation, may be induced.[2,4,5]

Regardless of the cause, if a young athlete goes into sudden cardiac arrest, CPR should be implemented immediately. Among sports-related cases of commotio cordis documented in the USCCR, 15% of patients survived. When CPR was instituted within 3 minutes of the impact, 68% of patients survived; however, if CPR was delayed by more than 3 minutes, only 3% of patients survived. Animal studies have shown that CPR instituted within the first 3 minutes of injury can increase survival rate by up to 25%. Concomitantly, early use of an automatic external defibrillator (AED) device has been proven to increase survival rates. With an AED recognizing ventricular fibrillation at a sensitivity of 98% and a specificity of 100%, defibrillation within the first 3 minutes can increase the survival rate by an additional 50% or more in animal models, yielding a survival rate of 46% at 4 minutes and 25% at 6 minutes. The USCCR has recommended that all athletic venues have an accessible AED. Preventive measures for commotio cordis include parental education, softer baseballs, and protective padding of an athlete's precordium.[1,4] Secondary prevention may involve avoidance of certain sports until at least age 18 years.

While the patient in this case was in the PICU, he was placed on continuous cardiac monitoring for 24 hours, and no incident of arrhythmia was noted. An echocardiogram revealed a normal left ventricular systolic ejection fraction, with no structural abnormalities, valvular disease, or hypertrophy. A repeat 12-lead ECG showed no changes from the previous one, and subsequently, the serial troponin measurements decreased from 0.25 to 0.04 ng/mL. A pediatric cardiologist consultation was unrevealing. The patient was transferred to the pediatric floor the next day, and he was discharged 2 days after initial admission, with no signs of postarrest sequelae. His final diagnosis was commotio cordis.

Question 1 of 2

A 15-year-old boy collapses while practicing with his school baseball team. The boy was attempting to catch a ball thrown at him; he missed, and the ball struck him in the chest. He collapsed after the blow and was unresponsive. Which of the following actions will greatly improve the chances of survival in this patient?

Your Peers Chose:

- Performance of an immediate precordial thump 6%
- Use of an automated external defibrillator (AED) within 3 minutes of impact 83%
- Initiation of cardiopulmonary resuscitation (CPR) within 10 minutes of impact 11%
- Intubation and mechanical ventilation 0%

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Question 2 of 2

The patient above is resuscitated with immediate bystander CPR and defibrillation from an AED at the baseball field. Which of the following will most likely be found during his subsequent hospitalization?

Your Peers Chose:

- A small elevation in his cardiac enzymes, but otherwise normal studies 91%
- Abnormal echocardiography revealing an enlarged heart 1%
- A QT interval of 520 ms on his electrocardiogram 6%
- A chest x-ray demonstrating a R-sided pneumothorax 2%

Commotio cordis is a diagnosis of exclusion in that other causes, such as substance abuse, myocardial infarction, electrolyte abnormality, prolonged QT syndrome, and hypertrophic obstructive cardiomyopathy, must first be ruled out with examinations such as urine drug screens, serial assessment of cardiac biomarkers and ECGs, electrolyte level testing, and echocardiography.

References

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