APPROXIMATELY 350,000 cases of sudden cardiac death (SCD) occur in the United States each year. The incidence of SCD during various types of athletic activity varies. Maron et al. estimated that 1 in 200,000 high school athletes die of SCD each year. Although the incidence of cardiac arrest among athletes is low, such an event demands an immediate response. The purpose of this report is to review the etiology of commotio cordis (CC) and to present possible prevention strategies.

Case reports of CC have been documented over the past 130 years. Reports were often cited as SCD, with no structural damage, during performance of routine manual labor. Theories about the etiology of CC include alteration in coronary blood flow and mechano-electrical feedback. Currently, the general consensus among experts is that CC is primarily an electrical event that results in instantaneous ventricular fibrillation (VF).

SCD in athletes is most often attributable to a congenital or acquired cardiac disease (e.g., hypertrophic cardiomyopathy) but may also result from a seemingly innocuous chest blow from body contact with an opponent or the impact of a projectile (i.e., a baseball). A “cardiac concussion” can induce cardiac arrest in the absence of any premorbid cardiac disease or structural abnormality.

Although CC appears to be a random event, there have been a number of cases that have had legal ramifications. In 1993, an Italian hockey player was charged with unintentional manslaughter when his opponent died suddenly after having been hit in the chest. The prevalence of CC in sport has not been clearly established, because many cases are not reported or are simply reported as accidental deaths. A National Commotio Cordis Registry was established at the Minneapolis Heart Institute Foundation in 1995 to track cases of CC, with a focus on sport events.

Clinical Presentation

The creation of the National Commotio Cordis Registry has provided a more accurate description of the circumstances of CC cases. Most cases have occurred during participation in competitive youth baseball (Figure 1), but participation in hockey, lacrosse, soccer, softball, and karate has also been reported. The events usually involve a blow to the chest from a projectile thrown at normal velocity, with most victims being adolescent males between 5 and 15 years of age who had no preexisting heart condition (Table 1).

Immediately after chest impact, collapse usually occurs, and resuscitation efforts are rarely successful. Cases have been reported, however, that involved the victim briefly continuing to perform an activity in a conscious state (e.g., attempting to stand, speak, cry, walk, or throw a baseball) before subsequently collapsing in a state of cardiac arrest.
This suggests that VF may be tolerated for a brief period, before cardiac arrest occurs.\(^7\) Although lesions are evident in only a minority of cases, there may be bruising on the left side of the sternum at the level of the nipples.\(^9\) Maron et al.\(^9\) found that VF only occurs when an athlete is hit during the repolarization phase of the heart cycle (diastole), specifically during the 15 to 30 msec period preceding the T-wave peak.\(^9\) Chest blows received outside this defined interval can produce a heart block that is generally not lethal. Thus, the incidence of CC “near-miss” cases may be great.

The force of chest impact is a third factor that is directly related to the risk for cardiac arrest. An animal study of chest blows with baseballs demonstrated that the softest baseballs triggered VF in fewer impacts than those of intermediate hardness or the hardness of a standard baseball.\(^10\) Moreover, the velocity at which baseballs are thrown in the 15-30 msec window can affect risk for cardiac arrest. Occurrence of VF in juvenile swine was greatest at 40 mph, and the occurrence was lower at velocities that were either less than or greater than 40 mph. The high-velocity impacts may have created structural damage that precluded the occurrence of VF.\(^11\) Thus, the risk of VF appears to be linked to a specific velocity of chest wall impact.

The susceptibility of young athletes to CC may be attributable to the amount and rate of chest wall compression that results from the external blow. A more compliant chest wall transmits a greater amount of impact energy to the heart.\(^12\) A defect in the function of sodium channels, or irregular activation of potassium-ATP channels, may also play a role in sudden death that is attributable to CC.\(^3\)

**Pathophysiology**

The three major determinants of CC occurrence appear to be body location, timing, and force of impact.\(^9\) Cardiac arrest that is attributable to CC is precipitated by impact directly over the heart. Although lesions are evident in only a minority of cases, there may be bruising on the left side of the sternum at the level of the nipples.\(^9\)

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**Prevention and Immediate Care**

The survival rate of individuals who experience VF is 15%.\(^6\) Athletic trainers and therapists (ATs) must recognize and react to CC immediately. The recommendations of the National Athletic Trainers’ Association for prevention of CC include training athletic personnel in the recognition of the signs and symptoms of CC and teaching athletes how to avoid being hit in the chest.\(^13\)

Utilization of protective athletic equipment may seem to be an obvious method of prevention; however, while some experts have advocated the use of sport-specific chest protectors, it may not protect young athletes from deadly blows.\(^5\) Chest padding can shift during play, thereby allowing a ball to hit the unprotected chest wall, and protective gear has not been found to adequately dissipate the pressure generated by chest wall impact to prevent CC.\(^12\) Further research is needed to establish the characteristics of protective gear that will provide optimum effectiveness.

Age-appropriate safety balls should be used to decrease incidence of CC in youth baseball. The