[Primary Care]

Commotio Cordis

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Content: Commotio cordis is blunt, nonpenetrating trauma to the chest resulting in irregular heart rhythm and often leading to sudden death. This article presents the epidemiology, variables leading to commotio cordis, theories on predisposing factors, diagnosis, treatment, treatment outcomes, and return-to-play recommendations.

Evidence Acquisition: A PubMed (MEDLINE) search for *commotio cordis* was conducted on July 1, 2008, and it yielded 106 results, of which 26 were used for this review, including experimental models, simulation studies, case analysis studies, case reports, general recommendation, review articles, and editorials.

Results: There are more than 190 reported cases of commotio cordis in the United States. Forty-seven percent of reported cases occurred during athletic participation. Commotio cordis is the second-most common cause of sudden cardiac death in athletes. Occurrence of commotio cordis is related to time of impact during the cardiac cycle, direct impact over the heart, the hardness and speed of the projectile, and the ineffectiveness of chest barriers. As a result, the US Consumer Product Safety Commission recommends that softer "safety" baseballs be used for youth baseball. Resuscitation using defibrillation was effective in only 15% of cases. Resuscitation within 3 minutes resulted in a survival rate of 25% (17 of 68 cases). Survival drops to 3% when resuscitation is delayed beyond 3 minutes. Survival of commotio cordis has risen from 10% to 15% since 2001. Reduced ventricular ejection fraction has been identified in some commotio cordis survivors.

Conclusion: Preventive measures, such as using soft "safety" balls and making automated external defibrillators available at sporting venues, can reduce commotio cordis morbidity and mortality. Chest protector designs can be improved to enhance protection. Return to play is best left to clinical judgment given that data are lacking with regard to susceptibility for reoccurrence.

Keywords: athlete; commotio cordis; sudden cardiac death

Commotio cordis (CC) is blunt, nonpenetrating trauma to the chest resulting in irregular heart rhythm and often leading to sudden death. Although CC was once viewed as being exceedingly rare, the number of reported cases is rising.[7,21,22] Cases demonstrate no structural damage to myocardial tissue at autopsy, and victims are typically young and otherwise healthy. Ventricular fibrillation (VF) is the most common arrhythmia, with early defibrillation improving survival rates.

A study analyzing a swine model brought to light many factors that play a role in CC. Timing and location[10] of the blow, as well as speed[12] and hardness of the projectile,[15] determine whether CC will cause sudden death. The underlying mechanism is thought to be an increase in ventricular pressure leading to activation of stretch channels.[2]

Protection has improved through the use of softer baseballs in youth leagues. Commercially available chest protectors, however, are not effective.[4]

EPIDEMIOLOGY

Commotio cordis was described as early as 1763, and it was first systematically evaluated in 1932 by Schlomka and Schmitz.[8,18] More than 190 cases have been described in the United States alone. Twenty percent of reported sudden-death cases in young athletes is attributed to Commotio cordis (77 of 387 sudden death cases), second only to hypertrophic

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cardiomyopathy.[19] There has been an increase in reported CC cases; of 25 cases reviewed in 1995,[22] 20 occurred after 1986 and only 5 before 1983. Today, there are more than 180 reported cases in the CC registry. Cases are usually missed because of lack of recognition and consequent underreporting.[17]

Almost half (47%) of all CC cases occur during competitive sports.[4] The age range of the victim is from 7 to 42 years (mean age 15.6 ± 6.8; Figure 1). Young males, ages 4 to 18 years old, are at greatest risk.[7,14,22] Vulnerability in this age group has been attributed to increased chest wall pliability,[12] but it is unclear why there is a male predilection.

Approximately 60% of reported CC events involved sports (Figure 2).[11] Although common projectiles responsible for CC cases include hockey pucks, softballs, soccer balls, and extremities (during martial arts competition),[7] occurrence of CC is most common in baseball—particularly, youth baseball. Twenty-five percent of reported cases of CC in youth baseball were from a pitch that averaged 30 to 50 mph.

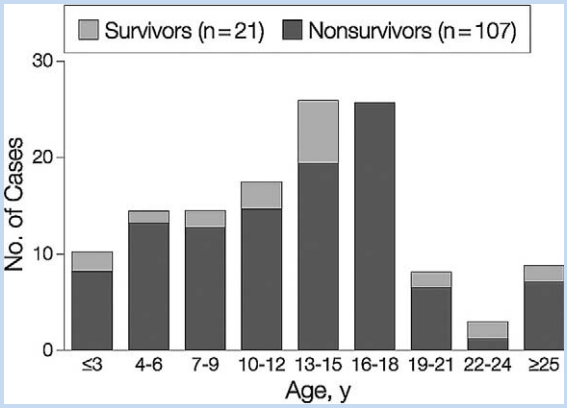

Figure 1. Ages at which commotio cordis has been reported. Commotio cordis victims are predominantly preadolescents and adolescents. (Reprinted with permission from *JAMA*.[21])

VARIABLES IMPORTANT IN CC

VF is the most common initial rhythm in CC.[22] Link et al studied CC in swine model and reported that VF occurs with a precordial impact during a vulnerable portion of the repolarization upslope of the T wave (10-30 msec before the T-wave peak).[15] Another study by Link et al demonstrated that a low-energy impact must occur directly over the heart and that the center of the left ventricle is the most vulnerable.[10] In this analysis, left ventricular impacts were more likely to produce VF (7 of 23 strikes, or 30%) than were impacts at other precordial sites (5 of 55, or 9%; $P = .02$); blows to noncardiac sites did not generate VF (n = 26).

Projectile structure also plays a role—that is, harder projectiles are more likely to cause CC (Figure 3).[3,15] As a result, the US Consumer Product Safety Commission recommends softer "safety" baseballs to decrease the incidence of youth injury.[6]

Projectile speed is also a factor; velocities of 20 mph do not cause VF, whereas speeds between 30 and 50 mph pose the greatest risk for CC.[12] Link et al reported that projectiles traveling 40 mph were most likely to cause VF in swine. Incidents of VF actually decrease when the velocity of the impact is between 50 and 60 mph.[12]

CURRENT THEORIES

Potassium Adenosine Triphosphate and Stretch Channel Theory

Causative theories on a predisposition to VF include that of mechanical electrical coupling. Studies have also shown that a rapid rise in left ventricular pressure to forces between 250 and 450 mm Hg may mediate electrophysiological consequences.[2,7,10,12] Stretch channels may be activated by a specific degree of myocardial stretch.[12] Potassium adenosine triphosphate channel activation has also been implicated.[16] Glibenclamide was used in a swine model to inhibit these channels, causing attenuation of ST-segment elevation, as well as a reduction of VF incidence. Streptomycin, a blocker of the calcium stretch-sensitive ion channel, did not reduce the frequency of CC, but there was a noted reduction in ST elevation after impact.[5]

Critical Mass Theory

The critical mass theory suggests that higher-velocity impacts (≥50 mph) are less likely to cause CC, because they lead to myocardial tissue damage, and that viable myocardial tissue is vital in sustaining VF. Higher velocities often produce contusio cordis or myocardial contusion with tissue damage.

Autonomic Nervous System

Hypothetically, sympathetic activation that occurs in sports may increase the likelihood of CC. However, a recent study did not substantiate this hypothesis.[23]

DIAGNOSIS AND TREATMENT

The differential diagnosis of sudden cardiac death includes the following: hypertrophic cardiomyopathy, coronary artery abnormalities, arrhythmogenic right ventricular dysplasia, long QT syndrome, Brugada syndrome, Wolf-Parkinson-White syndrome, dilated cardiomyopathy, Marfan syndrome, aortic valve

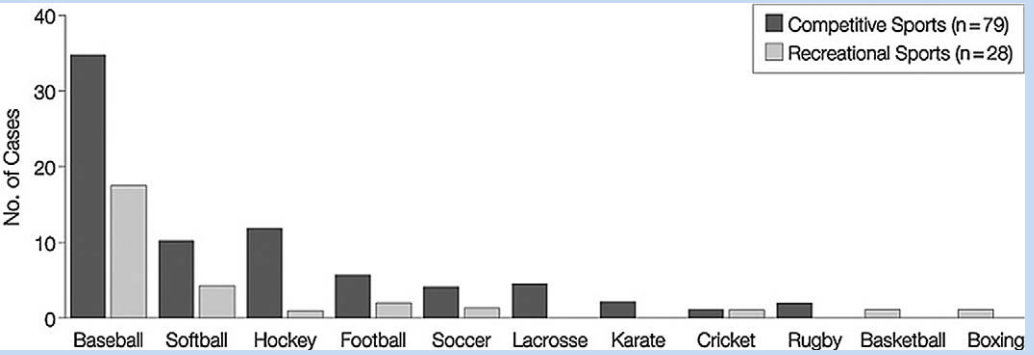

Figure 2. Sports during which a commotio cordis event occurred. Most cases occur in sports with a hard compact ball, and the event occurs because the ball strikes the chest. (Reprinted with permission from *JAMA*.[21])

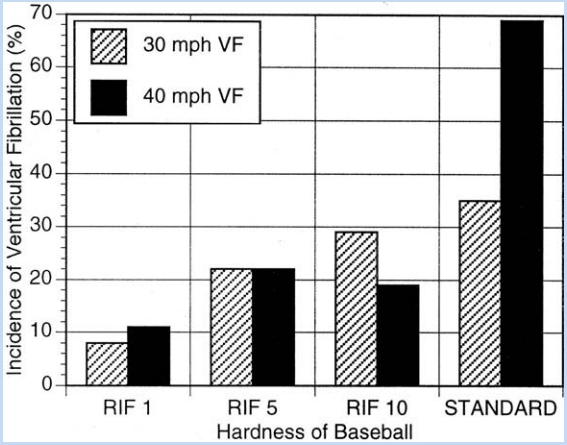

Figure 3. Influence of ball hardness on the likelihood of ventricular fibrillation caused by a chest blow. Softer balls are less likely to cause commotio cordis. (Reprinted with permission from *Pediatrics*.[11])

stenosis, mitral valve prolapse, coronary artery disease, myocarditis, asthma, heat stroke, drug abuse, and a ruptured cerebral artery. These conditions and injuries must be ruled out before a definitive diagnosis of CC can be made. However, a history of witnessed trauma to the chest should immediately raise suspicion of CC. Immediate initiation of cardiopulmonary resuscitation and automated external defibrillator (AED) protocol is paramount during a witnessed CC event.

Acute Treatment and the Role of AED

Early defibrillation is critical for survival of CC. Unfortunately, resuscitation after CC is often unsuccessful.[14,21,22] In a Link et al swine model, animals that were defibrillated in less than 2 minutes generally survived, whereas among those that were defibrillated in more than 4 minutes, fewer than half survived.[9] AED use in animals within 1 to 2 minutes resulted in 100% and 92% success, respectively. Only 46% of shocks were successful after 4 minutes; after 6 minutes, success rates dropped to 25% ($P < .0001$).[9] In human cases, resuscitation within 3 minutes resulted in a survival rate of 25% (17 of 68 cases); when resuscitation was prolonged beyond 3 minutes, the survival rate dropped to 3% (1 of 38 cases).[7,21] The total survival rate from CC is approximately 15%,[7,21] which has improved from 10% in 2001.[13]

RETURN TO PLAY

There is currently only 1 known case of CC recurrence. Maron and colleagues' 2001 series[21] demonstrated that 71% (15 of 21) achieved complete physical recovery and 29% (6 of 21) had mild to moderate residual neurologic disability or cardiac impairment (as manifested by reduced left ventricular ejection fraction) at follow-up between 1 and 20 years.

CC survivors should undergo a thorough cardiac evaluation, including a 12-lead electrocardiogram, ambulatory Holter monitoring, exercise stress testing, and echocardiogram.[20] Electrophysiologic testing and implantable cardioverter defibrillator are not generally recommended.[20] Return-to-play decisions should be made on a case-to-case basis.

There is concern regarding individual susceptibility to chest wall-induced CC, based on data from the swine model[1]; thus, return to play in contact sports must be balanced against a possibly increased risk of recurrence.

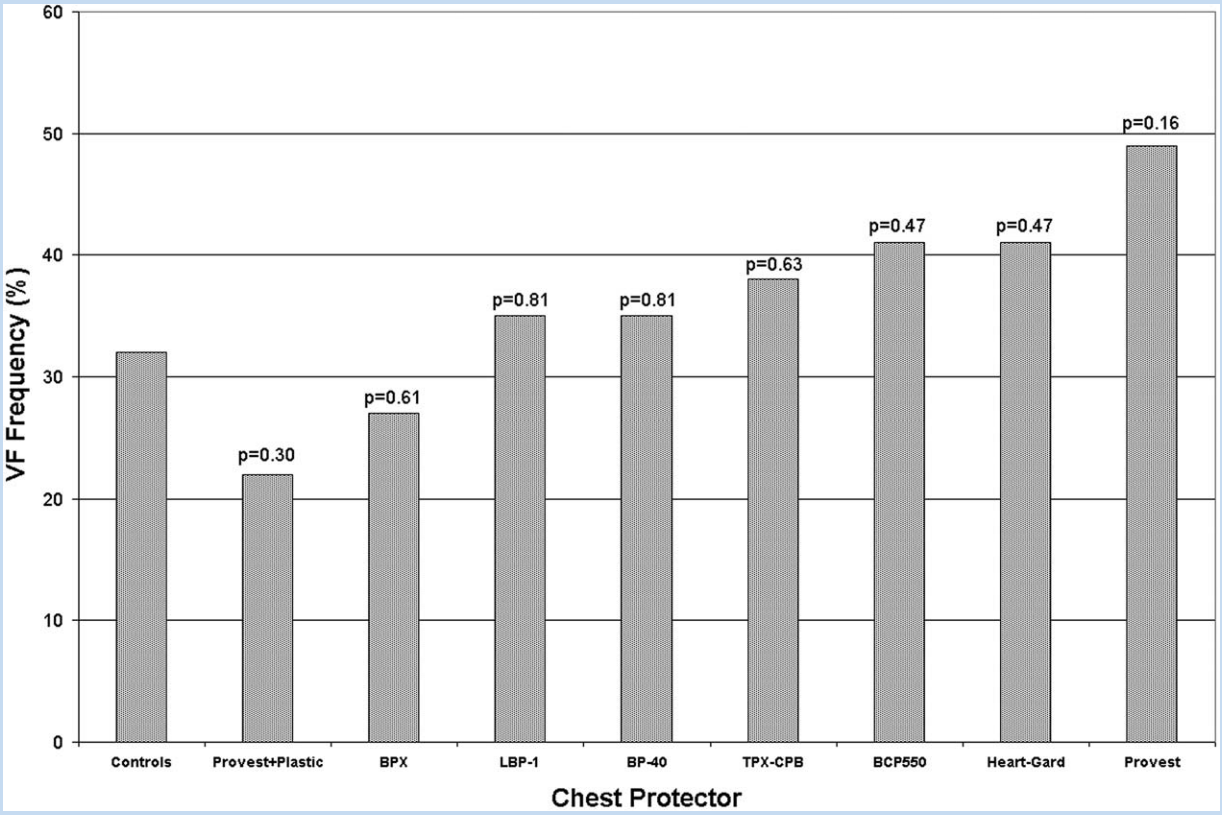

Figure 4. Evaluation of chest protectors marketed for use in baseball; the control impacts are on the left. (Reprinted with permission from *Pediatrics*.[24])

PREVENTION

As mentioned, the US Consumer Product Safety Division recommends soft "safety" baseballs to reduce the risk of soft tissue trauma.[15] Soft "safety" baseballs did reduce the risk for VF in the swine model,[11] and there is a linear correlation between CC events and the hardness of a baseball. Unfortunately, safety baseballs will probably not eliminate CC.

Doerer et al and Weinstock et al have demonstrated the shortcomings of chest protectors.[4,24] Nearly half of CC cases (85 of 182, or 47%) occurred during organized competition, and 38% (32 of 85) of these victims were wearing chest protectors. In 78% of the chest protector incidents (25 of 32), the device was not covering the precordium at the time of impact. This malpositioning can happen if a player raises his or her hands over his or her head. Of the 7 athletes hit directly on the chest protector, 3 were lacrosse goalies, 2 were baseball catchers, and 2 were hockey goalies.[21] Only 13% of the victims (4 of 32) who were wearing chest protectors survived the CC episode. Consequently, protective athletic equipment must be evaluated and tested carefully.

A Link et al study (using a swine model) revealed that commercially available chest protectors—specifically, those marketed for use in baseball and lacrosse—did not prevent CC. They had roughly the same likelihood of inducing VF as that of control impacts, in which animals had no chest protection (Figures 4 and 5).[24]

Also having the potential to reduce CC events are changes in rules (eg, eliminating chest blocking of shots by defenders in lacrosse) and in coaching (eg, teaching techniques to shield the chest).

SUMMARY

CC is a devastating cause of sudden death in young and healthy individuals, where VF is the most common arrhythmia.[18] Subsequent survival rates are dismal. Increased awareness of this phenomenon is imperative, especially among those who may be first responders, such as parents, coaches, game officials,

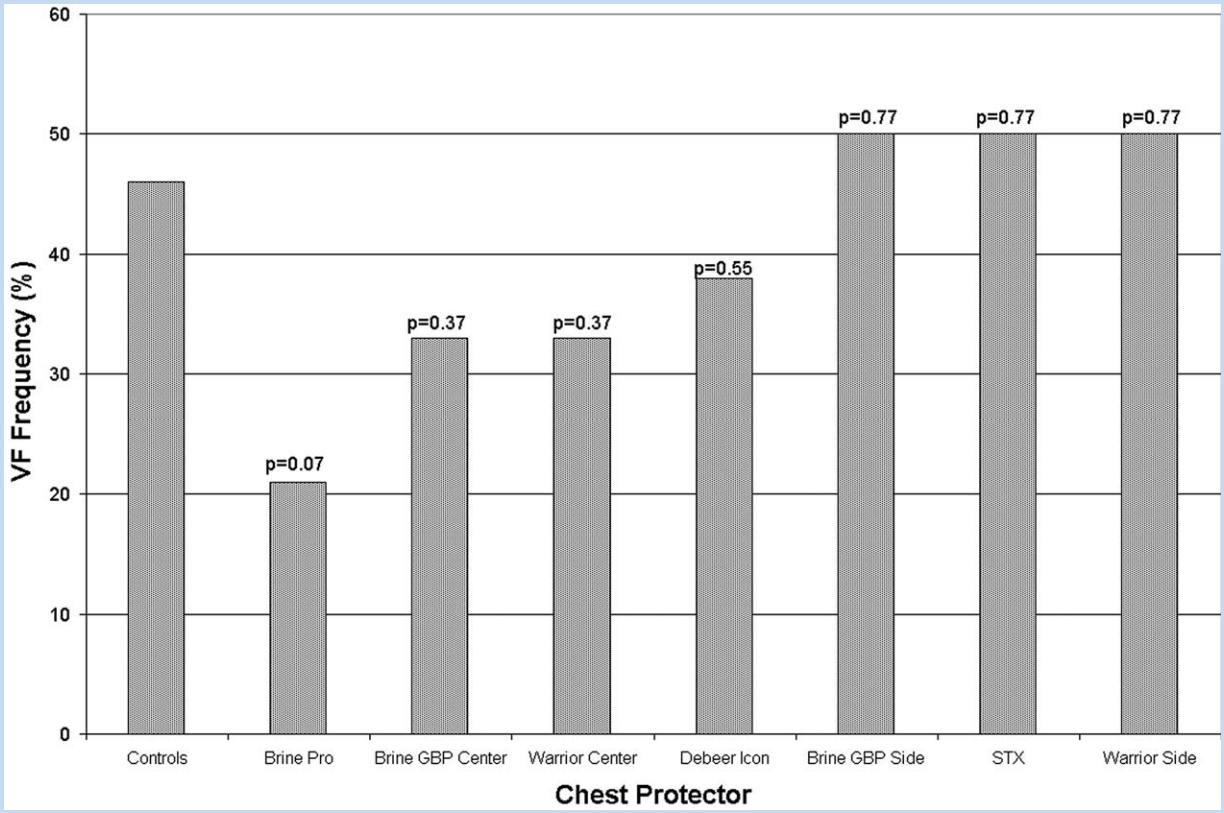

Figure 5. Evaluation of chest protectors marketed for use in lacrosse; the control impacts are on the left. (Reprinted with permission from *Pediatrics*.[24])

Clinical Recommendations

SORT: Strength of Recommendation Taxonomy

A: consistent, good-quality patient-oriented evidence

B: inconsistent or limited-quality patient-oriented evidence

C: consensus, disease-oriented evidence, usual practice, expert opinion, or case series

Clinical Recommendation	SORT Evidence Rating
Use of age-appropriate soft "safety" balls (ie, up to age 13), to reduce commotio cordis events.[20]	B
Use of chest protectors in high-risk positions (eg, catchers and goalies).[20]	C
Thorough cardiac evaluation for survivors of commotio cordis with ventricular fibrillation.[20]	C
Return to play left to clinical judgment, on a case-by-case basis.[11]	C
Automated external defibrillator availability at sporting venues, as well as first-responder education in cardiopulmonary resuscitation and automated external defibrillators.[20]	C

For more information about the SORT evidence rating system, see www.aafp.org/afpsort.xml and Ebell MH, Siwek J, Weiss BD, et al. Strength of Recommendation Taxonomy (SORT): a patient-centered approach to grading evidence in the medical literature. *Am Fam Physician.* 2004;69:549-557.

and medical personnel. Prevention may be enhanced through the use of soft "safety" baseballs and through the improved design of chest protectors. Availability of AEDs at youth sport venues may also improve survival of commotion cordis rates, given that early defibrillation improves outcomes. Cardiopulmonary resuscitation education and first-responder awareness are also important.

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