Commotio cordis: an important cause of sudden cardiac death in young athletes

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Although a rare occurrence, commotio cordis is the second most common cause of sudden cardiac death in young, healthy athletes. A slow-moving projectile (often a baseball or some other sports equipment) strikes the compliant precordium of an affected patient immediately before the T wave of the cardiac cycle, causing a premature ventricular contraction that induces ventricular fibrillation and cardiac arrest. Resuscitation attempts are often unsuccessful and survival rates are only 15%. This review outlines the pathophysiology and epidemiology associated with commotio cordis, and addresses strategies that may help prevent this life-threatening occurrence.

Introduction
Commotio cordis (CC), also termed “cardiac concussion”, is a rare but devastating event characterized by a seemingly innocuous blow to the chest followed by sudden death. 170 cases have been reported in the U.S. Commotio Cordis Registry (USCCR) since 1996, almost all of whom are young male athletes with no prior history of heart abnormalities or dysfunction.1,2 Although it is relatively rare, the seriousness of CC warrants investigation into its pathophysiology and subsequent indications for treatment and prevention.

Case Report
The patient described by Maron et. al. (2005) illustrates many key features present in patients affected by CC. A 22-year-old male who was not wearing any protective chest gear was struck directly over the precordium by a lacrosse ball. He was extremely athletic, had no previously reported health problems, and weighed 185 lbs. After being struck, he stumbled two steps and fell to the ground. A trainer immediately ran onto the field, determined that a pulse was absent, and initiated cardiopulmonary resuscitation (CPR). A sports medicine physician quickly entered the scene with an automated external defibrillator (AED) which detected ventricular fibrillation (VF). The AED delivered an appropriate defibrillation shock of 200J within 2 minutes of the athlete’s collapse. This successfully terminated the VF and advised no further shocks, however the man’s pulse did not return, and CPR was continued by bystanders and 5 minutes later by paramedics. The man was later pronounced dead in the emergency department, approximately 1 hour after collapse. A chest x-ray revealed no rib fractures or other injuries, and an echocardiogram revealed no structural abnormalities of the heart.3

As in the preceding case, most cases of CC involve a young male being struck in the chest during an athletic event, immediately initiating VF. Resuscitation is unsuccessful in the vast majority of cases.4

A case of commotio cordis was seen in London over 10 years ago. Attending physician Dr. Andrew Krahn recounts, “a young healthy trucker, father of 2, [was] tightening his load with a cheater bar (big steel ratchet like bar for the strap winch on a transport truck)... at Wellington and the 401. [He suffered a] minor blow to the chest [with a] long delay to the ambulance.” Unfortunately, this man ultimately suffered the same fate as many commotio cordis patients and died before the ambulance reached him.5

Pathophysiology
The pathophysiology of CC is not entirely understood, although Madias et al. (2006) have devised a possible model to explain its occurrence. It has been widely observed that key factors must align in order to cause CC: a sufficient but not excessive force must strike the precordium of a relatively compliant chest wall just prior to the peak of the T-wave of the cardiac cycle1,2,6 (see Figure 1). The force striking the compliant chest wall causes a rapid
increase in pressure within the left ventricle. This stretches the myocardium and activates mechano-sensitive ion channels, resulting in a rapid influx of potassium ions and consequently a premature depolarization of the myocardium. This alone would cause depolarization of a single beat, but would not be sufficient to cause a sustained arrhythmia. However, within a period a few milliseconds (15-30ms) before the peak of the T-wave, some of the myocytes of the left ventricle can be depolarized, while others are still refractory and are therefore unable to be depolarized. Depolarization of only a portion of the left ventricular myocardium results in non-simultaneous excitation, and thus fibrillation, providing a 15ms window of vulnerability within the cardiac cycle.\(^7\)

When one is struck directly over the heart, the force is transmitted significantly within the chest cavity due to mechanical compliance of their chest wall.\(^7\) If the impact is at exactly the right time within the cardiac cycle, non-uniform depolarization of the myocardium occurs, resulting in sustained VF and consequently sudden death. It is important to note that structural damage to the heart and surrounding tissues is not present in cases of CC due to the low velocity of impact\(^8\); the pressure wave that is transmitted to the left ventricle is the basis of the pathology.

CC may occur in any healthy heart.\(^9\) Athletes are more commonly affected because they are at greater risk than the average person of sustaining a blow to the chest wall. Young people are more commonly affected due to increased compliance of their chest wall, transmitting the force to the left ventricle more easily than adults. Other than these two indirect risk factors, CC requires no predisposing features. It can theoretically happen to anyone, but it is rare because very specific factors must align within an extremely small window of opportunity.

### Population at Risk
The specific factors that coalesce to trigger CC place a fairly narrow demographic at risk: commonly, young male athletes appear to be the most susceptible. Indeed, an analysis of 128 cases in the USCCR performed by Maron et al. (1999) reported that 62% of the events occurred in male athletes of median age 14 years during sporting competitions. This finding may be due in part to sport’s increased risk of projectiles, especially those that are rigid and/or possess a dense core such as baseballs and hockey pucks, hitting one’s chest as well as the increased chest wall compliance of young athletes described above.\(^9\)

It is important to note that CC can occur in non-athletes as well. The USCCR has recorded cases in which CC was induced following chest contact during seemingly harmless events as horse-play, being struck with a snow saucer, and a family pet colliding with a child’s chest.\(^8\) While these instances are thought to be uncommon, they emphasize the point that CC can affect almost anyone if all the etiological factors are present.

### Strategies for Prevention
Though cases of CC are considered to be rare overall, there has been an increased incidence of reports over the past two decades as recorded by the USCCR.\(^8\) Survival rates are dismal at approximately 15%. In athletes this condition has become the second most common cause of
instant death.\textsuperscript{8,11} Since several factors are involved in the etiology of CC, several measures may be taken to reduce the incidence of this tragic occurrence. Both emergency medical techniques and sporting apparatus must be examined and improved upon to reduce the risks of deleterious effects for athletes. Medical professionals present during sporting events should not only be well trained in cardiopulmonary resuscitation and defibrillation technique, but should also be educated regarding the necessity of quick response when CC is suspected. It is thought that assistance within 3 minutes or less of collapse may improve the outcome for some patients\textsuperscript{8} although it is not a guarantee of success, as evidenced by the athlete in the case study.

Methods of altering sporting equipment and protective gear to reduce the risk of CC for athletes have been suggested, most prominently for baseball and sports requiring chest protectors.\textsuperscript{8} The implementation of softer baseballs was proposed, but met with opposition following trials that demonstrated altered bounce and velocity. Baseballs of an intermediate density have been developed and are currently being evaluated as a compromise between safety and the integrity of the game. Chest protectors have fallen under scrutiny for both their composition and their functional capacity. In a study conducted by Doerer et al. (2007) examining chest protectors, 38\% of the afflicted athletes were wearing approved protective chest padding. It has thus been suggested that the density and its distribution within the protective gear be altered to provide additional defense.\textsuperscript{8} It is also relevant to note that of the athletes wearing chest protectors, 25 of 32 experienced displacement of the chest protector during the course of game play.\textsuperscript{12} A more effective means of keeping the protective gear over the cardiac silhouette should be devised.

Conclusion
Commotio cordis, though rare, is a particularly devastating cause of fatality that predominantly affects healthy young male athletes. It is caused by a projectile striking one’s compliant precordium moments before the T wave and causes ventricular fibrillation that may not respond to resuscitation. The risk of physical and projectile contact inherent to sporting competition enhances the likelihood of susceptibility in athletes, but non-athletes may also be affected if the etiological factors converge. Although CC is considered to be a relatively uncommon event, its rising incidence and tragic consequences necessitate increased awareness and preventive efforts on several levels.

References