Sudden death in sport

Commotio cordis

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Instantaneous cardiac arrest caused by a blow to the chest depends on the timing of the blow relative to the cardiac cycle

Sudden death following a sharp but seemingly inconsequential blow to the chest is a frightening occurrence known as “commotio cordis” or “concussion of the heart.” Although commotio cordis is considered rare by some authors, it represents one of the most common mechanisms of sudden death in sport seen in young athletes. Commotio cordis is generally understood to mean “instantaneous cardiac arrest produced by non-penetrating chest blows in the absence of heart disease or identifiable morphologic injury to the chest wall or heart.” Most cases report accidental death of otherwise healthy children or adolescents after chest impact during recreational or competitive sport or, less commonly, during road traffic accidents.

Such fatalities receive extensive media coverage, provoke legal debate, and may stimulate research into the public health aspects of this condition—for example, the capacity of protective gear to prevent commotio cordis or the possibility of developing safer sporting equipment.

HISTORY

The current concept of commotio cordis is often ascribed to a review of cases by Maron et al. Their report portrays commotio cordis as a rare but dangerous condition in which the chest wall is a poor response to resuscitation measures. Most of those affected were young (mean age 12 years, male [all but one]) and, at the time of accident, engaged in sport (>90% of the event leading to sudden death is a precordial impact, most commonly by projectiles such as baseballs, softballs, or hockey pucks). A similar occurrence is seen in a victim who was vulnerably positioned; the cardiac cycle was interrupted. Interestingly, the term commotio cordis was in use as early as 1857. A review from 1896 shows that the term was associated with various forms (both lethal and non-lethal) of cardiovascular disorder caused by mechanical impact to the chest (both in the presence and absence of minor cardiac bruising).

Commotio cordis underwent a conceptual modification at the turn of the century whereby a distinction between non-penetrating precordial impact in the presence (contusion) or absence (compression) of cardiac bruising was established. In many ways, the concept of commotio cordis paralleled that of commotio cerebri (brain concussion) for which the issue of structural injury has been controversial since the early 1900s and terminological inexactitude has plagued the medical literature up to the present day.

PATHOPHYSIOLOGY

The most comprehensive early experimental physiological studies of this condition were performed by Georg Schomka at Bonn University in the early 1950s. On the basis of more than 300 experiments on anaesthetised animals, he identified three factors that determined the induction of arrhythmias by cardiac precordial impact: type of impact, location of impact, and force of impact. Schomka disproved the “resplendent theory” of arrhythmias and instead proposed the “direct impact” concept of mechanically induced rapid vasospasms to explain commotio cordis.

The risk factors identified by Schomka in the 1950s are still relevant, whereas the identification of a fourth factor (time of impact) had to wait for technological advances. Contemporary experimental investigations into commotio cordis with anaesthetised pigs confirmed the existence of such a vulnerable period during early ventricular repolarization and showed the involvement of ATP-dependent potassium channels in the electrophysiological genesis of this condition.

When the precordial impacts were delivered within a narrow temporal window between 30 and 15 milliseconds before the peak of the T wave, ventricular fibrillation was reproducibly induced. The vulnerable period of the cardiac cycle amounted to just over 1/100th of a second. Remarkably, ventricular fibrillation was immediate, occurring on the very next heart beat. The arrhythmia was not produced by impacts at any other time during the cardiac cycle, although transient complete heart block was sometimes observed with impacts during the QRS complex. Occasionally, with impacts delivered just outside the 15 millisecond period of vulnerability, sustained polymorphic ventricular tachycardia was seen.

The observation that transient rhythm disturbances may occur with chest impact raises the possibility that there may be “near miss” cases of commotio cordis. This may have happened in 1998 to St Louis Blues hockey captain Chris Pronger, when he collapsed briefly, then spontaneously regained consciousness, after being struck on the left side of his chest by a puck during a playoff game. It is possible that other near miss cases have gone undetected, and in some instances the arrhythmias were too brief to cause loss of consciousness.

In another part of their study, Link et al. examined whether the use of safety baseballs, which are softer than regulation baseballs, could reduce the risk of arrhythmia in the animal model. They found that the ball was proportional to the hardness of the ball. This finding may have implications for the prevention of commotio cordis in young baseball players, as specially designed safety baseballs are required for use in recreational baseball and Little League. Another approach to prevention is the use of chest protectors specifically designed to protect the precordium. As not all cases will be preventable, it is important to emphasise that rapid cardiopulmonary resuscitation, including a precordial thump and immediate defibrillation when possible, may be lifesaving.

Both early and contemporary research into commotio cordis appears to have been motivated by case reports of sudden death. It is sobering that a seemingly minor chest impact at an instant when the heart is suspended in diastole can have such devastating consequences.

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REFERENCES

19 Link MS, Wang PJ, VanderBlink BA, et al. Selective activation of the K+(ATP) channel is a mechanism by which sudden death is produced by low-energy chest wall impact (Commotio cordis). Circulation 1999;100:413–18.
Retraction: Commotio cordis


This article has been retracted due to plagiarism of the following material:


We would like to acknowledge the preliminary work of Nick Brown in investigating publications by Dr Paul McCrory and thank him for bringing these concerns to our attention.

During 2021 and 2022 there was an investigation by British Journal of Sports Medicine and BMJ which found that some of McCrory’s work was the product of publication misconduct. British Journal of Sports Medicine published a summary of the investigation.1

References

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