Editorials
Sudden cardiac death caused by innocent chest wall blows

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Commotio cordis is ventricular fibrillation (VF) induced by relatively innocent chest wall blows in the absence of structural cardiac damage[1]. Commotio cordis is presently the second leading cause of sudden death in competitive youth sports[2]. Although formerly regarded as a particularly rare event, sudden deaths due to chest wall blows are being reported with increasing frequency[1,3,4]. It is unresolved as to whether this increased number of recognized cases is due solely to an enhanced awareness of the condition (and thus the probability of reporting), or alternatively, whether the incidence of commotio cordis is truly increasing.

Historical context and clinical profile

Commotio cordis was first described in the European medical literature during the 19th century[5-7]. Initially, these events were generally described in association with workplace accidents, such as falls from heights. However, since 1980, commotio cordis has been reported with increasing frequency associated with sporting activities[1,8,9].

The US Commotio Cordis Registry (Minneapolis, MN), established in 1996, has documented in detail more than 170 cases, with a mean age of 14 years (80% ≤ 18 years)[1]. It is believed that young athletes are at particular risk for commotio cordis because of their pliable chest wall; however, other factors such as the increased exposure to chest blows in young people may also be determinants. Males comprise 95% of the victims, a proportion seemingly too high to be accounted for solely by the predominance of males in sports.

In the United States, the most common sporting activity involved in commotio cordis is baseball, comprising almost one-half of the recognized cases. Other organized sports in which these events commonly occur include softball, ice hockey, and lacrosse; in each of these sports the impact object is a solid projectile. Often, the energy of impact does not appear unusual for the sport. Commotio cordis is rarely reported due to blows from air-filled balls such as used in European soccer and American football. In these sports, commotio cordis is usually caused by chest blows with a body part such as a knee, elbow, head, or fist (as in karate).

While organized competitive sports account for about 60% of all commotio cordis cases, 20% occur in non-competitive recreational athletic activities often during play with peers. The remainder of these events take place during ordinary and routine daily activities – e.g. innocent and playful boxing, parental disciplining, gang rituals, blows by plastic projectiles or playground swings. Energy of impact does not seem unusual for the sport in most instances, and indeed most events occur during play with peers.

Approximately one-half the victims of commotio cordis experience instantaneous collapse following chest wall impact while others may demonstrate a few brief seconds of physical activity prior to collapse[1]. Although overall survival is low (only about 15%) from a commotio cordis event, up to one-third survive when cardiopulmonary resuscitation and defibrillation is prompt, while less than 5% survive when resuscitation is delayed by more than 3 min. In a re-

cent case in Chicago, complete recovery was achieved in a 14-year-old boy with commotio cordis due to a chest blow with a baseball who had received an automated external defibrillator shock within 3 min[10].

Experimental model

An experimental swine model developed over the last 8 years has provided a large measure of clarification with regard to the mechanism of commotio cordis[11]. Probably the most important determinant of VF is the timing of the chest impact in the cardiac cycle. If a 49 km/hour (30 miles/hour) projectile impact occurs during a 20 ms window on the up-slope of the T wave, approximately 30% of strikes will initiate VF. Impacts occurring outside of this narrow window but still on the up-slope of the T wave triggered VF only 5% of the time, while blows at all other times in the cardiac cycle (including the down-slope of the T wave) did not result in VF[12]. Of note, the onset of VF immediately follows the chest impact, and is not preceded by ischemic ST-segment changes or premature ventricular beats or tachycardias. Also, of note, nonsustained bursts of VF are occasionally induced, as well as transient complete heart block, and these may account for cases of commotio cordis in which collapse following the chest blow is transient and recovery occurs spontaneously within 10 to 20 s[1].

Other important variables predisposing to commotio cordis include the site and velocity of the chest blow. For example, VF will not occur with a chest blow outside of the cardiac silhouette (i.e., on the right chest wall or back). Furthermore, impacts at the center of the cardiac silhouette are most likely to provoke VF, while those at the base and the apex only occasionally trigger VF; at all other sites on the chest, including the right and left thorax and back, blows will not induce VF[13]. Commotio cordis events occur at a wide range of velocities in humans, paradoxically with innocent appearing blows, but also with hockey pucks and lacrosse balls, at impact velocities of up to 160 km/hour (100 miles/hour). Of note, in our experimental model, as the velocity of impact increased to 65 km/hour (40 miles/hour), the frequency of VF increased dramatically up to 70%, but with much higher velocities of 81 to 113 km/hour (50 to 70 miles/hour), VF was less frequent[14]. Therefore, it is indeed a paradox that particularly forceful blows may not cause commotio cordis (even when timed to the vulnerable phase of repolarization), and very soft and low velocity impacts may counterintuitively result in a catastrophe.

Considerations for prevention

Given this degree of understanding the clinical profile and mechanism of commotio cordis, most of the recent focus has turned to the critical issue of prevention. Safety baseballs (i.e., softer-than-standard) decreased, but did not abolish, the likelihood of VF in the experimental commotio cordis model with blows at 49 km/hour (30 miles/hour)[11], or 65 km/hour (40 miles/hour)[15]. At present, such balls are frequently employed in organized play for children of 6 years of age and younger, although under-utilized for participants of 7 to 13 years[9,16]. Commercially available chest wall protectors, often marketed with the explicit or implied claim that they will prevent sudden death have not at present been shown to abolish the risk of commotio cordis[17]. Indeed, in the Commotio Cordis Registry nearly one-third of those individuals struck in the chest during competitive sports were wearing some type of chest barrier, i.e., catchers in baseball, and goalies in hockey and lacrosse[1]. Such chest protectors did not provide absolute protection against sudden cardiac death, either because they did not cover the precordium during all likely body movements, or did not attenuate the force of the blow when the projectile struck the chest barrier.

Conclusion

Our novel observations regarding commotio cordis over the last 10 years have provided considerable insights into this new risk to young people on the athletic field as well as in recreational sporting and daily activities. Fundamentally, it is inadvisable to strike the chest under any circumstances. While commotio cordis has been increasingly recognized in North American sports activities, this syndrome has been reported less commonly in Europe. It is possible that this reflects a true geographic difference in incidence, possibly explained by disproportionately less participation in sports involving hard solid projectiles (such as baseballs) in Europe. However, greater recognition and awareness of commotio cordis and its risks in European countries, including Italy, will undoubtedly promote the identification of greater numbers of cases, as well as measures directed toward the prevention of these tragic events.

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