COMMOTIO CORDIS:
An under-appreciated cause of sudden cardiac death in young athletes

Commotio cordis (Latin, “agitatio of the heart”) is a primary arrhythmic event that occurs when mechanical energy from a blunt, non-penetrating force is transferred to a small area of the praecordium at a critical time during the cardiac cycle, which alters the electrical stability of an otherwise healthy myocardium, resulting in a life threatening ventricular fibrillation (VF).1

In most reported cases, sudden death following this form of sports trauma, is believed to have been of insufficient force to have caused a major injury and out of proportion to the outcome; with many having believed that the player had “had the wind knocked out of him/her”.

Commotio cordis is considered as a diagnosis of exclusion; as previously diagnosed or undiagnosed structural or primary electric cardiac abnormalities are more often associated with unexpected sudden cardiac death (SCD) in young athletes during sports participation.1, 4, 5, 38 Hypertrophic cardiomyopathy (HOCM), arrhythmogenic right ventricular cardiomyopathy, congenital prolongation of the QTc interval, polymorphic ventricular tachycardia, anomalous coronary artery syndromes, myocardial infarction in childhood and viral myocarditis account for the majority of these unfortunate instances.1, 4, 5, 38

The worldwide incidence of commotio cordis is probably more common than previously considered due to poor recognition and under-reporting of past occurrences;7 and is now recognised as one of the more frequent causes of SCD (up to 20%)4, 46, 47 in young athletes after HOCM and congenital coronary artery anomalies.4, 5 The American National Commotio Cordis Registry (Minneapolis, Minnesota) is the most comprehensive database on this condition and has had almost 250 reported cases over the past 15 years.1, 3, 6, 15, 35

Epidemiology
International studies have shown that commotio cordis occurs primarily in young, healthy and active individuals most frequently between 10 – 18 years of age. 95% of affected individuals were male with the mean age being around 15 years of age; participating in a diverse spectrum of competitive (50% of episodes) or recreational (25% of episodes) sporting activities e.g. baseball, softball, ice hockey, American football, lacrosse, polo, soccer, boxing, cricket, rugby, martial arts and even basketball.1, 35

In the South African setting, examples of the most commonly involved sporting activities and the events triggering commotio cordis would include:

- Rugby: bodily collision with opponent’s forearm, shoulder, knee, or head.
- Soccer: player being kicked by opponent in chest; player colliding with another player/goalpost; goalie being struck by shot on goal.
- Cricket: batsman being hit by bowled cricket ball; bowler/fielder being hit by ball.
- Hockey: player being struck by stick during shot; player being struck by ball during short corner.

The individual is hit in the chest (with or without chest protection) usually by a pitched, thrown or batted projectile at high or low velocity (58% of occurrences) or by the force of bodily contact (42% of occurrences); with the majority collapsing immediately.1, 38

10-20% of reported cases have displayed a brief period of purposeful activity, movement or behaviour (e.g. taking a few steps and continuing to walk or run, opening his or her eyes, or even speaking) prior to collapse, suggesting a brief period of individual tolerance for the ventricular arrhythmia (ventricular tachycardia (VT) or VF)1 before cardiac arrest.

Following collapse, patients are unresponsive, apneic and pulseless. Convulsions may be present initially, which could easily be misdiagnosed as epileptic instead of a manifestation of cardiac arrest.47 Localised oval or circular contusions or abrasions at the site of impact are noted in one-third of patients.2, 3

Laboratory Findings
Post mortems show an absence of congenital and acquired structural entities that are known to predispose young athletes to SCD; cardiac enzymes are within normal haematological reference ranges with no evidence of myocardial necrosis; toxicology screenings have been universally negative; and there has been no evidence of infarction, infection or inflammation histologically.34, 38

Cardiac weight, wall thickness and chamber dimensions were normal; as were the coronary arteries with no evidence of damage or thrombosis. There have been no reports of active or healed myocarditis or arrhythmogenic right ventricular cardiomyopathy. Isolated reports of haemorrhage in the left ventricular wall, arteriovenous node and specialised conduction system have been noted, however, the significance of these rare occurrences has yet to be determined.38

The absence of any structural cardiac injury, haemopericardium, external myocardial contusion and no underlying fracture(s) to the sternum or ribs, distinguishes commotio cordis from contusion cordis,1, 38 where high impact trauma results in morbidity and mortality due to direct trauma to myocardial tissue and the overlying thorax.

The most common ECG finding of non-survivors and survivors has been found to be VF and asystole. The timing of this ECG reading, following impact, is the determining factor to which rhythm is present i.e. an ECG reading recorded late during a resuscitation attempt.
usually shows asystole, which in all likelihood was preceded by VF. This has been demonstrated repeatedly on anaesthetised animal test models.8,9,11-13,15-17,19-24,34,38

Some patients have shown impressive ST-segment elevation in precordial leads V1-V3, however, the significance of this finding remains unclear as myocardial ischaemia has yet to be demonstrated from commotio cordis in human or anaesthetised animal models.8,9,11-13,15-17,19-24,34,38

Determinants
This precise synchronisation of numerous and relevant variables during the smallest of windows of vulnerability, helps to explain why this is such a rare event; and a wide variation in individual vulnerability to VF from appropriately timed strikes has been noted during anaesthetised animal studies.8,9,11-13,15-17,19-24,34,38

Three primary determinants have been identified using various biomechanical and biologic experimental models.7-12,15-24
1. A relatively low-energy impact precisely over the centre of the left ventricle.15,27 Impacts over other precordial sites resulted in VF less often; and impacts that did not overlie the heart failed to produce VF or any other ECG abnormalities.

Non-sustained polymorphic VT’s, ST segment elevation, transient complete heart block, left bundle branch block and left ventricular wall motion abnormalities transiently occurred in the absence of VF following impacts over the cardiac silhouette in anaesthetised animal models.7,9,11,15-24 No permanent ECG findings have yet to be described in any survivors.

2. The electro-physiologic events, following impact, were determined to be critically dependent on the precise timing of the impact during the repolarisation stage of the cardiac cycle (a narrow 10-30ms portion of the ascending phase just before the T wave peak).15,24,38

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Therefore, the probability of commotio cordis is about 4% in a cardiac cycle of 500ms (heart rate of 120b/min).38 It has been suggested that exercise induced hypoxia and acceleration of the cardiac conductive system also allows for the heart to be more susceptible to stretch induced VF.38

In vivo studies suggest that following impact, a rapid rise in left ventricular pressure (250 – 450mmHg)1,9,16,19,24 causes activation of normally inactive mechano-sensitive ion channels (particularly ATP dependant K+ channels) via mechano-electric coupling and inward current formation.9,11,16,20,21,34 These cellular mechanisms augment non-uniform myocardial activation and cause premature ventricular depolarisations that are the final triggers of VF in commotio cordis.1,34 VF was not preceded by VT, conduction abnormalities or ischaemic ST changes in various studies; suggesting that the primary mechanism is electrical.

Coronary artery vasospasm1,6 excessive autonomic vagal reflex1,6 and varying degrees of myocardial contusion22 may have a pathophysiological role in commotio cordis but these have yet to be confirmed. Susceptibility to VF from commotio cordis has been found in anaesthetised animal models with longer QRS and QTc durations at baseline. This potential relevance to human subjects still needs further research for verification.

3. A narrow and compliant chest wall. The predisposition of commotio cordis to the younger population may be directly related to their physical characteristics.24,25 A relatively underdeveloped chest cage with immature intercostal musculature may be less capable of blunting the arrhythmogenic consequences of precordial forces.2,3 Adults gain a measure of protection from a fully developed and mature chest cage, which may help to explain the low incidence of commotio cordis in sports such as boxing or martial arts in that age group.

Other factors that have been shown to increase the risk of commotio cordis include: the size, shape and density of the object; where solid, hard, small and spherical projectiles have been shown to be directly related to VF.15,18 Anaesthetised swine models have also shown a bell shaped curve relationship between ball strike velocity and the induction of VF, with the highest incidences occurring at 64km/h (70% of strikes).1,3,17 This is the optimum speed needed to generate a minimal impact energy of 50 joules to cause commotio cordis.

Prevention
Public education on the importance of avoiding and preventing precordial blows, safety interventions to make impacts less forceful and improved automated external defibrillator (AED) availability, represent the most important primary and secondary intervention strategies that have been instituted to date, as pre-participation screening in young athletes for this presentation of SCD is simply not possible.
Correct techniques for avoiding contact with the chest have now been incorporated into many sports in the United States (e.g. teaching batters to turn away from errant baseball pitches; avoidance of the chest to block the ball or puck in lacrosse or ice hockey). 22,26

The use of more suitable materials for various age groups have been debated if the nature of the sport remains unchanged e.g. softer-than-standard safety baseballs 1 were proposed at the 36th Bethesda Conference on Eligibility Recommendations for Competitive Athletes with Cardiovascular Abnormalities for recreational and Little League baseball. 18,36 The softer projectiles are presumed to be safer as their propensity to collapse would allow for greater dissipation of energy on impact.

Equipment design of various chest guards have come under much investigation and scrutiny. 1,25,27-29 Chest protectors are designed more for greater movement and performance and not specifically with commotio cordis in mind. The use of inadequate materials (closed-foam) for padding against blunt bodily trauma has been proven to be easily penetrable by projectiles and does not dissipate forces adequately enough. This has been a documented occurrence for 32% of commotio cordis victims in competitive American football, baseball, lacrosse and hockey. 2,7,10,28,30

The most important secondary preventative measure is the increased availability and familiarity of AEDs at all youth sporting events and recreational settings. 1,3,17 As emergency services in our setting cannot possibly be expected to arrive at a scene in the necessary time period (within 5 minutes of the cardiac arrest), the expanded availability and use of AEDs even by persons with minimal training, may save a life by recognising and automatically terminating a fatal arrhythmia. 5,26,31,33,47

Medical Care
Commotio cordis is managed as with any cardiopulmonary emergency associated with a non-perfusing cardiac rhythm. The 2010 American Heart Association (AHA) Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care, recommend immediate implementation of cardiopulmonary resuscitation (CPR) emphasising chest compressions after the emergency response system has been activated. Compression to ventilation ratio of 30:2 or initial external chest compression only CPR (no ventilation) at a compression rate of at least 100 per minute ("push hard, push fast"), are recommended for adults and children above 1 year until modalities for defibrillation are available. 37

The success rate of external chest compression only CPR has been found to be at least similar to that of traditional compression-ventilation CPR. 47-53 However, it should only be considered in circumstances where rescuers are unwilling or unable to ventilate i.e. infectious or aesthetic concerns. 47 Many arrests in children will be hypoxic in origin and ventilations are crucial for survival in these instances.

An adequate emergency action plan together with compulsory defibrillator or AED on the field-side is an absolute necessity as SCD in athletes is usually fatal if not managed expeditiously and effectively. 47 Trained medical responders (team physician, physiotherapist, emergency medical service providers or first aiders) or other witnesses of the collapse (the referee, coaches or players) must respond appropriately when SCD is recognised and must all be made aware of the availability and location of the defibrillator or AED; and also be well versed in the use of the device. This basic understanding is vital, as every individual and component is invaluable in the contribution to a successful outcome.

Performing CPR while the AED or defibrillator is readyed for use is strongly recommended, as a shorter time interval between the last chest compression and the shock directly correlates with the success of defibrillation. Chest compressions should resume immediately after a shock and should continue for 2 minutes before a rhythm or pulse check is conducted.

High quality CPR, early recognition of a shockable rhythm and prompt defibrillation with an AED are the key interventions that have helped increase out-of-hospital cardiac arrest survival rates.

A controversial issue that is still debated, is the use of the precordial thump during resuscitative efforts. There are no prospective studies that have evaluated its efficacy in resuscitation attempts and recent studies have also shown the precordial thump to be ineffective in terminating VF. 38-45 The 2010 AHA Guidelines for adult advanced cardiac life support (ACLS) do mention that a single, immediate precordial thump may be considered after a witnessed cardiac arrest if a defibrillator is not present, but it is not mentioned as an option for paediatric CPR or paediatric advanced life support (PALS). 38-45 It should not delay prompt defibrillation if available.

Survival after a commotio cordis event is still the exception, as the severity of the injury is often underestimated and the aggressive activation of the chain of survival (early CPR, early defibrillation, followed by advanced life support measures) is therefore delayed. 4,46

In the South African setting, examples of the most commonly involved sporting activities and the events triggering commotio cordis would include: rugby, soccer, cricket and hockey.
The duration and intensity of exercise prior to a commotio cordis arrest may allow for higher than normal endogenous catecholamine levels and a decreased systemic vascular resistance, which may also play a role in limiting the success rate of resuscitation.

Time to defibrillation is the single most important determinant of survival in cardiac arrest, as VF generally evolves into asystole with a matter of minutes.56 The probability of a successful defibrillation declines 7-10% per minute for every minute that defibrillation is delayed without CPR and 3-4% per minute with bystander CPR.1,37,56 Following induction of VF in anaesthetised swine models via blunt chest trauma, defibrillation after 1, 2, 4 and 6 minutes, showed survival rates of 100%, 92%, 46% and 25% respectively.17,56 Survival rates were only 3% if resuscitation efforts were delayed longer than 3 minutes and overall, survival trends following exercise-related sudden cardiac arrest from all causes in young athletes continues to be disappointing.1,37,56 According to the American National Commotio Cordis Registry,1,4 survival rates for commotio cordis remain between 24-35% at present; which represents a low percentage, given the absence of any structural cardiac abnormalities.

Patients with return of spontaneous circulation should be transferred to the nearest appropriate facility capable of providing post resuscitation care e.g. therapeutic hypothermia and ventilatory support. A complete cardiac evaluation, including a 12-lead ECG, ambulatory Holter monitoring, and echocardiography, is advised for all survivors. Echocardiography in survivors, have shown normal anatomy and functioning post event. There is no evidence of HOCM, anomalous coronary artery syndromes or aortic root dissection; and cardiac valves have also been shown to be normal in anatomy and functioning. There have been documented isolated incidences in some survivors of mildly diminished global left ventricular systolic function or limited areas of hypokinesis, however, these were all of short duration and resolved spontaneously within a few days.17,38

Eligibility for return to competitive sporting activity is based on sound clinical judgment for that individual. There is, at present, no evidence to suggest that survivors of commotio cordis have a greater risk for any future arrhythmic events and accordingly, survivors cannot be disqualified from competitive action solely on the basis of a previous occurrence.1

Conclusion
It is recommended that South African communities and schools re-examine their awareness of commotio cordis and all other possible causes of SCD in athletes. As these tragic events cannot be completely prevented; the need for accessible AEDs, continued CPR training and emergency action plan updates and rehearsals for as many individuals involved as possible (team physician, physiotherapist, emergency medical service responders, first aiders, referees, coaches, parents, teachers and players), are some of the most important and easily available forms of management for these types of occurrences.

Most schools in South Africa (private and government) and recreational sporting venues are surprisingly not equipped even with a single AED device on their entire premises. If they are present, the location, availability, accessibility and proper use of the device also needs to be known.

Emergency medical services and the presence of a defibrillator are usually only present at organised events, if they have been part of the school’s medical preparation plan and if they are affordable for those few hours.

AEDs are widely dispersed at airports, shopping centres, large sporting arenas and at most gym facilities; in most part due to regulations for the FIFA 2010 World Cup and some private initiatives that have allowed for this increased distribution. If this can be done for the larger population in case of cardiac arrest from any number of causes, I see no reason why other areas where these devices are still needed cannot be equipped accordingly.

Even though these events are rare, all sporting venues for practices and matches should have access to an AED as the necessity for their presence cannot be underestimated and critical reasons for minimal delays in prompt defibrillation have been noted in this review article.

Combining continued education with these strategies and efforts will help to provide safer environments for all our young individuals participating in all sporting activities.

References on request