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REVIEW ARTICLE

Commotio cordis as a cause of sudden cardiac death

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Commotio cordis is responsible for the sudden cardiac death of an individual with an anatomically normal heart after moderate precordial impact with a blunt object. Victims are usually young and typically players of contact sports, although cases have also occurred during normal daily activities. The pathogenesis seems to be related to occurrence of the R-on-T phenomenon at a moment when the heart is particularly vulnerable to repolarization; ventricular fibrillation then triggers cardiorespiratory arrest. Recently, patient registries have been established to allow study of the epidemiologic characteristics and test physical medicine approaches to preventing commotio cordis. However, even if basic life support and chain-of-survival measures have taken place promptly, the rates of recovery from cardiorespiratory arrest due to commotio cordis are markedly lower than might be expected from the age and health status of the victims. The condition is generally fatal. [Emergencias 2011;23:471-478]

Key words: Commotio Cordis. Ventricular fibrillation. Sudden cardiac death. Sports medicine. Heart arrest.

Historical notes

Sudden cardiac death (SCD) due to commotio cordis (CC) is triggered by an apparently trivial, low-energy impact to the precordium, which produces ventricular fibrillation (VF) and cardiac arrest[1-4]. The term CC first appeared in medical literature during the nineteenth century, but the condition was described previously, both in Chinese martial arts (the Dim Mak or "touch of death") and eighteenth century Western reports of SCD after minimal chest impact, mostly in the context of industrial accidents[1,5].

Around 1870, the Italian Felice Meola experimented with chest blows to rabbits and attributed the SCD produced in some of these animals to an extreme vagal reaction[6]. In Germany at the end of the nineteenth century, the terms commotio cordis (without structural lesions) and contusio cordis (with structural lesions) were differentiated by the German investigators Rieninger and Reinebroth, whose interest derived from the existence of labor insurance and the study of accidental deaths[2,4].

In the 1930s, George Schlomka of the University of Bonn conducted a more detailed investigation, using feline and canine models. He monitored electrocardiographic and hemodynamic parameters and found they were altered by impacts applied with different energies and locations[7]. Schlomka was the first to describe conduction anomalies and arrhythmia resulting from impact on the chest wall, including ST segment abnormalities, ventricular extrasystole, atrioventricular and bundle branch block, ventricular tachycardia (VT) and VF, and identified three factors that influenced the arrhythmias: the type of projectile, the force and location of impact[2]. He refuted the Meola vagal crisis concept by producing SCD in vagotomized animals, and attributed the arrhythmogenesis to intense coronary vasospasm. After World War II and during the next 50 years, CC received little attention in the scientific literature and few cases were reported until 1995 when Maron et al. published 25 cases of CC where the victims were aged 3 to 19 years[8]. Although the term CC is currently restricted to SCD after an impact to the precordial area (and not to all mechanically induced arrhythmias), it generally refers to the same concept as that described more than a century ago[2].

The absence of macroscopic lesions due to impact distinguishes CC from contusio cordis or car-

diac contusion; the latter involves high-energy impact (as in chest trauma patients) producing myocardial lesions and damage to the overlying rib cage[1,9]. Until the 1990s, the term CC was hardly accepted outside the community of coroners (as a cause of SCD in the absence of findings of chest injury, and a structurally normal heart at necropsy, with no known history of harmful impact) and medical literature only contained isolated case reports of SCD due to CC[2,4].

However, since then, both the general public and the medical community have become more aware of the importance of CC as a cause of SCD in healthy people, often children, adolescents or young adults involved in sporting activities mainly (competitive or recreational), but also routine activities[2,8-10] (Table 1). It is likely that CC as a cause of death in athletes has been underestimated in the past. However, the most common cause of SCD during sporting activity remains underlying structural disorders of the heart, particularly hypertrophic obstructive cardiomyopathy (HOCM) and congenital coronary anomalies[11].

Table 1. Some situations that have triggered sudden cardiac death by commotio cordis, recorded in the U.S. National Commotio Cordis Registry[1,10-11]

Baseball, softball, cricket, lacrosse[1,10-11,26]:
Batter struck by a ball thrown by the pitcher[26].
Pitcher struck by a ball thrown by hand or batted[29].
Player struck by batted ball[26].
Catcher, umpire or spectator struck by ball.
Catcher struck by a bat[38].
Body collisions on the bases.
Player fell on a softball after catching it[45].

Football[8,11]:
Impact of a kick in the chest.
Ball impacting goalkeeper's chest.
Player collision with goalpost.

Hockey[10,11]:
Goalkeepers or other defensive players hit by a shot on goal[26].
Multiple collisions between players.
Players hit by sticks.

Fights and assaults:
Warden struck by a psychiatric patient[10].
Professor struck after a intervening in a fight between adolescents[10].
Different impacts during games.
Impact of fist fighter (with gloves) sparring[11].
Children struck by parents or babysitters (punishment)[34].
Child struck by snowballs struck[10].
Adolescents and young people involved in fistfights[33].
Protestors dispersed with rubber bullets[32].
Baby struck during diaper change[35].
Adult struck in a prision ritual[33].
Karate kick on the chest of a child[51].

Other circumstances[1,10]:
Child struck by a horse kick.
Youth struck by recoil of a hunting rifle.
Youth struck by low-energy car wheel in accident.
Child struck by a swing.
Child fell from a slide head-height[50].
Youth struck by a tennis ball filled with coins.
Child struck by the head of a 20 kg dog.
Child struck by a frisbee.
Adolescents who received a slap on the chest to stop hiccups[10].
Child struck by the handlebars on falling off a bicycle.

Epidemiology

Since it was launched in 1996, the United States Commotio Cordis Registry of the Minneapolis Heart Institute Foundation has recorded over 220 cases of SCD, and this registry has allowed the publication of several epidemiological and laboratory studies[1,9,12-17]. Growing interest in CC is mainly directed at ways to increase effective protection in different sports[18-20] and, above all, the dissemination and teaching of basic life support (BLS) in the general population as well as the presence of semi-automatic external defibrillators (SAED) at sports events[21-24], especially those held at schools and universities[25-27].

Given the wide variety of circumstances in which CC may occur (Table 1), it seems reasonable to suppose it is probably under-reported and more common than was previously thought[1,27]. Although clinical contexts vary widely, CC is usually produced by impact of a ball used in sports like baseball, softball and lacrosse, a hockey puck, or impact with a another player. The US registry also includes rare cases of CC produced playing football, a sport which is played far more in our setting. The registry has collected 224 cases since its inception 15 years ago, with an annual incidence of 5-15 new cases[1,17,27]. CC occurs predominantly in children and adolescents, aged 15 ± 9 years; 26% of the victims are under 10 years of age and only 9% are 25 years or older (range: between 6 weeks and 50 years). The victims are predominantly males (95%) and Caucasian (78%)[1]. This susceptibility in young people has been attributed to the narrower chest structure (with a greater cardiac outline surface area relative to the ribcage), and more deformable cage wall, which favor greater transmission of kinetic energy to the myocardium[16,24,28]. The broader, stronger and more developed ribcage of adults explains the relatively low rate of CC in older people, even in those involved in violent contact sports[1,11,26]. The gender distribution can be attributed to sex-different sports played in childhood and adolescence (the impact of projectiles or other athletes are the most common causes of CC in children under 15), but there is no easy explanation for the apparent predominance of white-race victims[1,8-11].

Approximately 50% of cases are related with organized competitive sports (baseball, softball, lacrosse, ice-hockey etc) and chest impact by a projectile (balls or pucks,) or by collision with other players (as often occurs in rugby, American football, soccer, karate rtc.). The goal-keepers in sports like lacrosse and hockey, despite wearing protective equipment, are at increased risk of CC due to projectile chest impact than other players since they sometimes use their chests to stop shots on goal[26,29]. Regarding the type of projectile, both hockey pucks and the balls used in baseball, softball or lacrosse (and pelota vasca in our country) are characterized by their solid cores, so the transfer of kinetic energy on impact is greater and linear. In contrast, the balls used in tennis, paddle or handball etc, are hollow and have a more flexible surface, so they tend to be deformed on impact and their kinetic energy is distributed and lost; thus cases of CC in players of these sports are exceptional[8,17,20,28].

Approximately 25% of CC occurs apparently innocuous activities such as recreational sport games at home, in family gatherings, children's games, etc. Most CC in children under 10 years occurred in these circumstances. Finally, another 25% of cases is not related to sports of any kind, but triggered by everyday activities. Thus, for example, episodes of CC have occurred after trivial impact with a frisbee, snowballs, a horse kick, bicycle handlebars, playing with pets or children wrestling (Table 1)[1,30-31]. The use of rubber bullets by riot police has also been described as a cause of CC death after chest impact[32]. Occasionally, some accidents of this type (especially in cases of allegedly corrective aggression or involving very young child victims) have led to criminal charges for manslaughter or murder[33-35].

Pathogenic mechanisms

CC is an arrhythmic event triggered when the kinetic energy of an impact affects a circumscribed area of the precordium and profoundly disturbs the electrical stability of myocardium during repolarization, leading to functional re-entry and VF[15-17] (Figure 1).

Although cardiovascular collapse in CC is virtually instantaneous, up to 20% of victims remain physically active and preserve their level of consciousness after impact (they can walk, return the ball, and even talk), which is attributed to individual tolerability (usually higher in healthy adolescents) to ventricular tachycardia sustained before degeneration into VF[1,24]. The rhythm initially registered by AED or monitor is generally VF (the appearance of asystole is related to delay until the first ECG recording, and seems not to be the initial rhythm, but late degeneration to VF), suggesting that survival is feasible, assuming defibrillation can be administered immediately[1,21].

Animal models (with dogs, pigs and rabbits) have been used in attempts to elucidate the pathophysiology of this phenomenon. The evidence currently available suggests a multi-factorial mechanism that requires the simultaneous coincidence of various circumstances[4,28].

One experimental model involved launching projectiles at different speeds at anesthetized pigs, in synchrony with the cardiac cycle, and this study demonstrated two determining circumstances for CC to occur[36]. The first was impact location, directly over the center of the heart (left parasternal), which is consistent with observations in human victims[16]. The second concerned the prcise moment of impact, which must occur within a narrow range of 10 to 20 msec prior to T wave peak (approximately 1% of the total duration of the cardiac cycle) (Figure 2)[1,16,36]. Such a concurrence of a cluster of circumstances explains the rarity of CC[28].

When impact occurs in this vulnerable window period of 10-20 msec, in which there is dispersion of repolarization, this increases and results in electrical heterogeneity, leading to "direct" VF, without being preceded by premature ventricular contractions or VT (configuring a "R on T phenomenon") (Figure 2)[14]. In pigs, when impacts occur at other times in the cardiac cycle, such as the QRS complex, the observed effects (usually

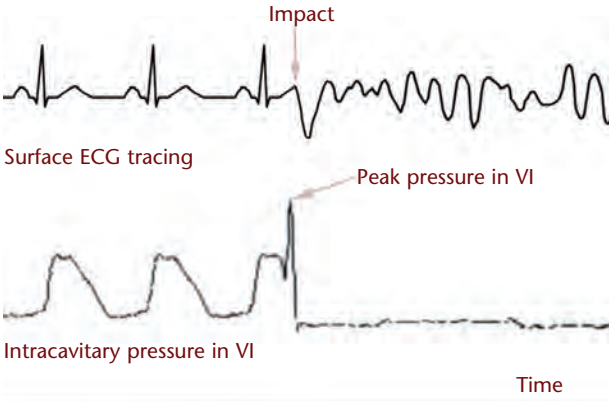

Figure 1. Commotio cordis episode with electrocardiographic recording (top) and pulse wave (lower) after impact by a blunt object. VF is triggered immediately after the impact when it occurs in the vulnerable period of repolarization (10-20 ms before the peak of the T wave)[7,17]. Authors.

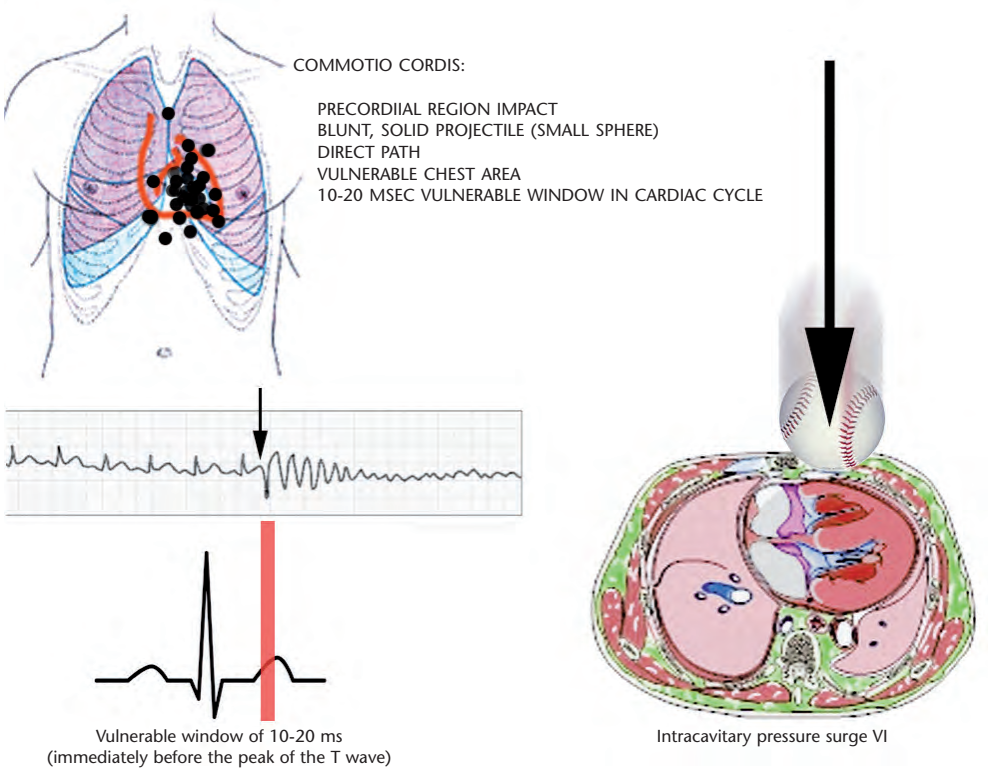

Figure 2. The exact location of impact and the time it occurs determine the occurrence of ventricular fibrillation by commotio cordis. Other factors influencing the event are the type of projectile (baseball, lacrosse, fists, other objects), its mass, shape and hardness, its trajectory and orientation of the morphology and deformability of the rib cage. In the image of the rib cage can be seen superimposed in black the points of impact previously published that caused commotio cordis. Note that the predominant points are parasternal that lie over the cardiac silhouette[1,10,16].

transient) are not FV but complete AV block, branch blocks, ST segment elevation or non-sustained VT, which could be considered an "aborted" CC (Figure 3)[10,14,36]. Angiography performed immediately after impact revealed no evidence of stenosis or spasm in the epicardial coronary arteries[13,36], despite human cases with transient ST-segment elevation post-resuscitation[37] (a type of injury also attributable to the resuscitation procedure itself)[37,38].

The kinetic energy at impact varies greatly according to speed, shape, size, weight and hardness of the projectile[20,38,39]. The projectiles mentioned in the Minneapolis registry range from hockey pucks travelling at speeds close to 140 km/h to seemingly innocuous objects travelling at low speed (frisbees or slapping to cure hiccups)[10]. Under experimental conditions, the velocity of the projectile (in the case of a baseball) required to trigger VF on impact in the critical window period was 30-120 km/h[17,38]. The incidence of VF after impact increased progressively to a maximum of 65 km/h (a speed usually attainable for baseball pitchers aged 10-12 years). However, it subsequently decreased: at higher speeds myocardial contusion and structural damageof the ribcage is more likely to occur than CC[36].

The importance of hardness, impact location, internal structure and speed of the projectile in inducing VF is related to the peak instantaneous pressure in the left ventricle (LV). As with the impact speeds, the relationship between peak LV pressure and the probability of triggering VF follows a Gaussian distribution (Figure 4), with the highest incidence coinciding with intracavitary pressure of 250-450 mmHg[17,38]. This suggests the existence of upper and lower limits of myocardial vulnerability to mechanically induced VF[15,38].

Understanding the underlying cellular and subcellular mechanisms of CC is still incomplete, but it appears to be multi-factorial[12-17]. The induction of electrical events in the myocardium after a mechanical stimulus is described in numerous circumstances, including catheter-mediated extrasystole or precordial blows as immediate treatment for VT[14,39]. This phenomenon, called electromechanical coupling, has been attributed to the

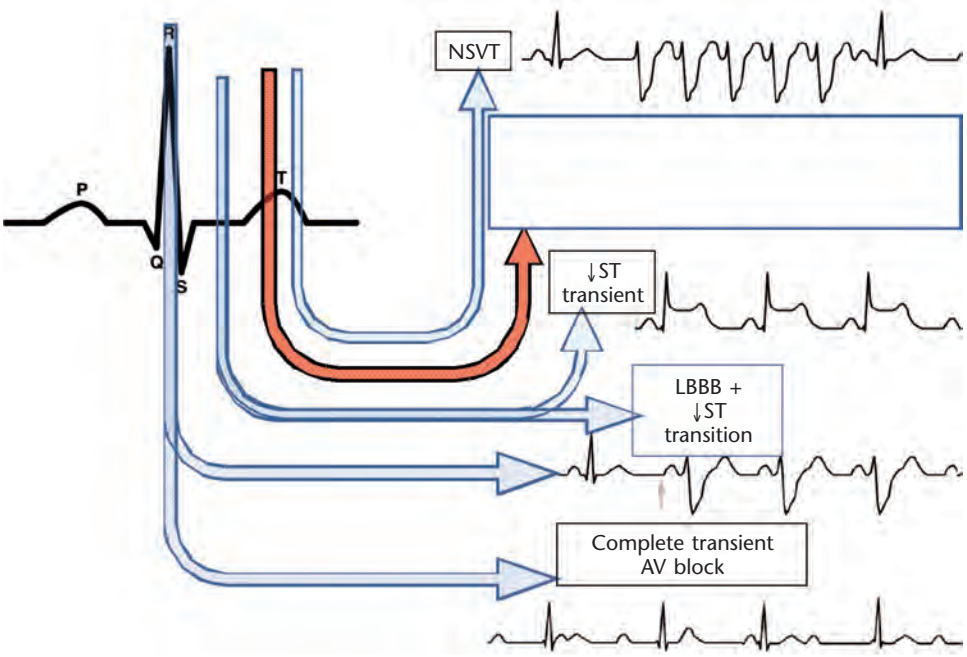

Figure 3. Schematic representation of the various rhythms and repolarization abnormalities obtained after projectile impact on porcine models in different phases of the cardiac cycle. The critical period 10-20 ms preceding the peak of the T wave is the window of susceptibility for producing ventricular fibrillation (VF) and sudden death from commotio cordis. Impact during ST itself resulted in a transient elevation of this or aberrant conduction (LBBB: left bundle branch block), while if it occurred at the time of QRS, it also produced transient third-degree blocks, with an escape rhythm that was usually suprahissian. If the impact occurred at the end of repolarization, this resulted in non-sustained ventricular tachycardia (NSVT)[14,17,36].

existence of mechano-sensitive ion channels that are activated by deformation and sudden stretching of the myocardiocyte membrane that expresses them[15]. In CC, the currently accepted hypothesis (Figure 5) implies that the peak intracavitary pressure induced on impact generates activation of ion channels, which in turn leads to a dispersion of repolarization and myocardial electrical heterogeneity which may generate the VF[12,15-16]. Thus, CC may involve an electro-physiological mechanism in common with other arrhythmogenic channelopathies such as long QT syndrome or Brugada syndrome[39-41]. The selective activation of ATP-mediated potassium channels (which contribute to the onset of VF in myocardial ischemia) is now considered the most probable pathogenic scenario in CC[12,40].

Prognosis and treatment

SCD due to CC is generally though not invariably fatal[1,10,28,42]. The data on historical series indicate median survival of 15%, but survival reaches 25% if cardiopulmonary resuscitation (CPR) is performed in the first three minutos[42]. This survival rate is very low, considering that the victims are young and, by definition, lack structural heart disease. This poor prognosis has often been attributed to the circumstances in which CC occurred, as witnesses of the CPR procedure can be slow to recognize the severity of the situation[10,11,27].

However, registry data also indicate that CC survival rates are increasing with time, up to 35% in the last decade, and above 50% in the period 2006-2009[1]. This has been attributed to increased public awareness, greater availability of AED, early activation of the survival chain and Emergency Medical Services (EMS) with implementation of the ILCOR 2005 protocols[1,22,42,43].

Thus, treatment of CC is closely related with prevention and prognosis: it is the same as all out-of-hospital CPR treatment and requires the immediate availability of AEDs. Basic Life Support (BLS) involves early recognition of the arrest, EMS activation and the immediate start of chest-ventilation compressions until an AED is provided (the first three links in the chain of survival)[43]. These

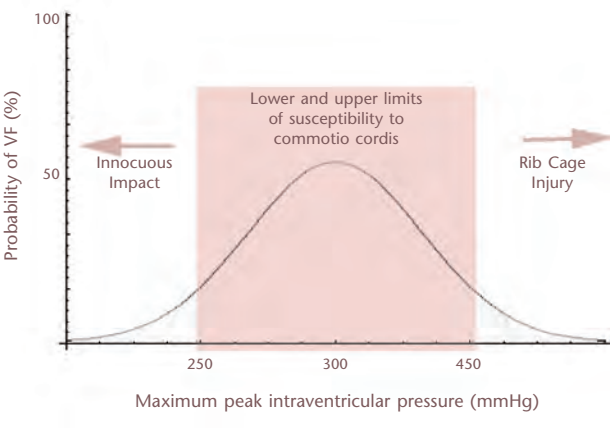

Figure 4. The probability of ventricular fibrillation (VF) in relation to the maximum pressure reached in the left ventricle in an animal model after impact with a baseball follows a Gaussian distribution. The increased frequency of VF occurred with peak pressure between 250 and 450 mmHg[17,36,38].

are the only effective methods available to prevent a probable death, and one that is particularly tragic because of the type of victims in this case.

Survivors of CC should receive standard post-resucitation care; and thorough study (ECG, echocardiography, Holter, including coronary angiography) to completely rule out the existence of underlying structural heart disease. Electrophysiological studies or implantation of an automatic defibrillator are not recommended in the absence of heart disease[1,28]. The possibility that changes in QT interval length can modify individual susceptibility to CC has been studied but not yet clarified[1].

Due to the absence of data allowing us to assess susceptibility to recurrent episodes of CC, the decision to allow a return to sports competition in CC survivors should be, at present, an individual clinical decision. However, in the absence of any heart disease, there is no evidence to support an individual ban on competitive sport for that reason alone[1,28].

Preventive considerations

Different strategies have been considered to prevent CC. Innovations in sports equipment for use by adolescents in the U.S. include softer baseballs (called safety baseballs), and chest protection[26,30]. Safety baseballs in laboratory conditions have been shown to decrease the risk of VF, but are not absolutely safe in the field (several episodes of CC with them have been described[10]) but seem a sensible recommendation for use by

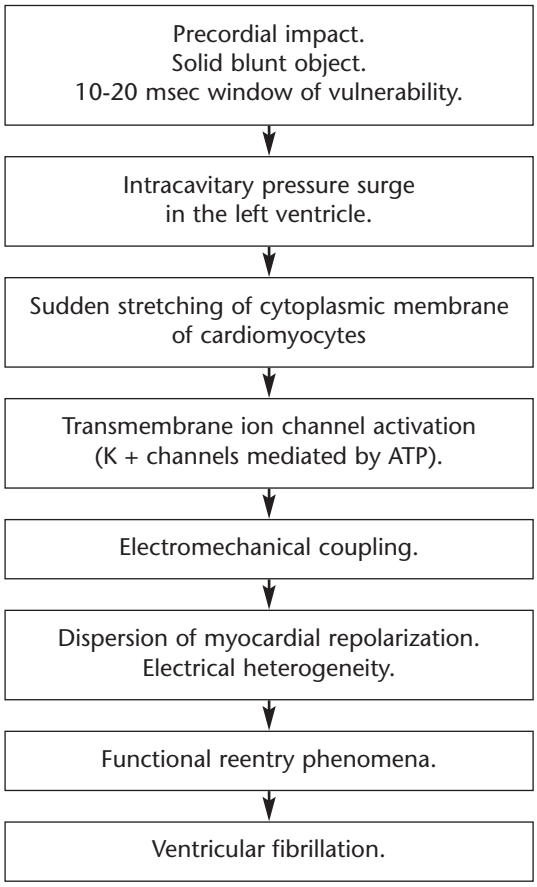

Figure 5. Schematic representation of sub-cellular pathophysiological sequence responsible for commotio cordis (CC). The phenomena of "R on T", acute coronary ischemia and certain channelopathies such as long QT syndrome or Brugada syndrome share with CC the same final arrhythmogenic pathway on producing dispersion of repolarization and functional reentry; which can cause sudden cardiac death[12,14,17,36].

children up to the age of 13 years in baseball[28]. With respect to chest protectors on the market, they are designed to prevent chest trauma rather than CC, and do not offer full protection against arrhythmia induced by impact[10,18]. In fact, in the series of cases on the U.S. National Commotio Cordis Registry, almost one third of competitive sport players dying of CC were wearing chest protectors at the time of the event[18,26]. No existing precordial protector has proven to be effective against CC[1,44].

Reports of sport-related CC emphasize the importance of educating the public in recognizing life-threatening situations and acting effectively with BLS measures[21-22,45-46]. AEDs can save lives and should be made available for immediate use in places where CPR may be expected for any reason (or in mobile emergency units, both basic and advanced), forming part of public access de-

fibrillation programs, although in Spain there is still no uniform legislation in this regard, and their development depends on autonomous regional health authorities[46-48]. At this time there are no reliable data on cases of CC in European territories[49]. A health strategy that seeks to provide the population with greater availability of AEDs and gradual training in BLS by ordinary citizens can result in higher survival rates in cases of CC[10,22,48]. The effectiveness of AEDs in treating SCD due to CC has also been demonstrated in animal models[13]. According to available data, early CPR and early defibrillation using an AED have been shown to be the only successful way of treating CC, but fatal cases have also occurred in spite best efforts[23]. However, data obtained in recent years (when implementation of ILCOR 2005 protocols have managed to increase overall survival) allow some degree of optimism in the secondary prevention and prognosis of CC[1,2,22,50].

References

1 Maron BJ, Estes NAM III. Commotio cordis N Engl J Med. 2010;362:917-27.
2 Nesbitt AD, Cooper PJ, Kohl P. Rediscovering commotio cordis. Lancet. 2001;357:1195-7.
3 Pinto, DS, Josephson ME. Sudden cardiac arrest in the absence of apparent structural disease. Uptodate [revista electronica] 2010. (Consultado 14 Abril 2010). Disponible en: http://www.uptodate.com/online/content/topic.do?topicKey=carrhyth/47754&view=print
4 Geddes LA, Roeder RA. Evolution of our knowledge of sudden death due to *commotio cordis*. Am J Emerg Med. 2005;23:67-75.
5 Maron BJ, Doerer JJ, Haas TS, Estes NAM III, Link MS. A historical observation on commotio cordis. Heart Rhythm. 2006;3:605-7.
6 Meola F. La commozione toracica. Gior Internaz Sci Med. 1879;1:923-37.
7 Schlomka G. Commotio cordis und ihre folgen. Die einwirkung stumpfer brustwandtraumen auf das herz. Ergeb Inn Med Kinderheilk. 1934;47:1-91.
8 Maron BJ, Poliac LC, Kaplan J A, Mueller FO. Blunt impact to the chest leading to sudden death from cardiac arrest during sports activities. N Engl J Med. 1995;333:337-42.
9 Legome E, Blunt Cardiac Injury in adult Trauma. Uptodate, 2010. (Consultado 3 Mayo 2010). Disponible en: http://www.uptodate.com/online/content/topic.do?topicKey=ad_traum/9443&view=print
10 Maron BJ, Gohman TE, Kyle SB, Estes NAM III, Link MS. Clinical profile and spectrum of commotio cordis. JAMA. 2002;287:1142-6.
11 Maron BJ, Doerer JJ, Haas TS, Tierney DM, Mueller FO. Sudden deaths in young competitive athletes: analysis of 1866 deaths in the United States, 1980-2006. Circulation. 2009;119:1085-92.
12 Link MS, Wang PJ, Van der Brink BA, Avelar E, Pandian NG, Maron BJ, 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;100:413-8.
13 Link MS, Maron BJ, Stickney RE, Vanderbrink BA, Zhu W, Pandian NG, et al. Automated external defibrillator arrhythmia detection in a model of cardiac arrest due to commotio cordis. J Cardiovasc Electrophysiol. 2003;14:83-7.
14 Kohl P, Nesbitt AD, Cooper PJ, Lei M. Sudden cardiac death by *commotio cordis:* role of mechano-electric feedback. Cardiovasc Res. 2001;50:280-9.
15 Bode F, Franz MR, Wilke I, Bonnemeier H, Schunkert H, Wiegand UK. Ventricular fibrillation induced by stretch pulse: implications for sudden death due to commotio cordis. J Cardiovasc Electrophysiol. 2006;17:1011-7.
16 Link MS, Maron BJ, Van der Brink BA, Takeuchi M, Pandian NG. Impact directly over the cardiac silhouette is necessary to produce ventricular fibrillation in an experimental model of commotio cordis. J Am Coll Cardiol. 2001;37:649-54.
17 Madias C, Maron, BJ, Winstock, J, Mark Estes A, Link MS. Commotion cordis—sudden cardiac death with chest wall impact. J Cardiovasc Electrophysiol 2007;18:115-25.
18 Weinstock J, Maron BJ, Song C, Mane PP, Estes NAM III, Link MS. Failure of commercially available chest wall protectors to prevent sudden cardiac death induced by chest wall blows in an experimental model of commotio cordis. Pediatrics. 2006;117:656-62.
19 Doerer JJ, Haas TS, Estes NAM III, Link MS, Maron BJ. Evaluation of chest barriers for protection against sudden death due to commotio cordis. Am J Cardiol. 2007;99:857-9.
20 Link MS, Maron BJ, Wang PJ, Pandian NG, VanderBrink BA, Estes NAM III. Reduced risk of sudden death from chest wall blows (commotio cordis) with safety baseballs. Pediatrics. 2002;109:873-7.
21 Myerburg RJ, Estes NAM III, Fontaine JM, Link MS, Zipes DP. Task Force 10: automated external defibrillators. J Am Coll Cardiol. 2005;45:1369-71.
22 Salib EA, Cyran SE, Cilley RE, Maron BJ, Thomas NJ. Efficacy of bystander cardiopulmonary resuscitation and out-of hospital automated external defibrillation as life-saving therapy in commotio cordis. J Pediatr. 2005;147:863-6.
23 Maron BJ, Wentzel DC, Zenovich AG, Estes NAM III, Link MS. Death in a young athlete due to commotio cordis despite prompt external defibrillation. Heart Rhythm. 2005;2:991-3.
24 Boraita A. Muerte súbita y deporte. ¿Hay alguna manera de prevenirla en los deportistas? Rev Esp Cardiol. 2002;55:333-6.
25 Zangwill SD, Strasburger JF. Commotio cordis. Pediatr Clin North Am. 2004;51:1347-54.
26 Boden BP, Tacchetti R, Mueller FO. Catastrophic injuries in high school and college baseball players. Am J Sports Med. 2004;32:1189-96.
27 Maron BJ. Sudden death in young athletes. N Engl J Med. 2003;349:1064-75.
28 Maron BJ, Estes NAM III, Link MS. Task Force 11. Commotio Cordis. J Am Coll Cardiol 2005;45:1369-71.
29 Maron BJ, Doerer JJ, Haas TS, Estes NAM III, Hodges JS, Link MS. Commotio cordis and the epidemiology of sudden death in competitive lacrosse. Pediatrics. 2009;124:966-71.
30 Hamilton SJ, Sunter JP, Cooper PN. Commotio cordis- a report of three cases. Int J Legal Med. 2005;119:88-90.
31 Tibballs J, Thiruchelvam T. A case of commotio cordis in a young child caused by a fall. Resuscitation. 2008;77:139-41.
32 Ritchie AJ, Gibbons JR. Life threatening injuries to the chest caused by plastic bullets. BMJ. 1990;301:1027.
33 Maron BJ, Mitten MJ, Greene Burnett C. Criminal consequences of commotion cordis. Am J Cardiol. 2002;89:210-3.
34 Denton JS, Kalelkar MB. Homicidal commotio cordis in two children. J Forensic Sci. 2000;45:734-5.
35 Boglioli LR, Taff ML, Harleman G. Child homicide caused by commotion cordis. Pediatr Cardiol. 1998;19:436-8.
36 Link MS, Wang PJ, Pandian NG, Bharati S, Udelson JE, Lee MY, et al. An experimental model of sudden death due to low-energy chest-wall impact (commotio cordis). N Engl J Med. 1998;338:1805-11.
37 Link MS , Estes NAM III, Paul J. Wang PJ, Charles I. Berul CI,Kirchhoffer JB, Ginsburg SH, et al. Commotio Cordis: Cardiovascular Manifestations of a Rare Survivor. Chest. 1998;114;326-8.
38 Link MS, Maron BJ, Wang PJ, VanderBrink BA, Zhu W, Estes NAM III. Upper and lower limits of vulnerability to sudden arrhythmic death with chest-wall impact (commotio cordis). J Am Coll Cardiol. 2003;41:99-104.
39 Viswanathan PC, Rudy Y. Cellular arrhythmogenic effects of congenital and acquired long QT syndromes in the heterogeneous myocardium. Circulation. 2000;101:1192-8.
40 Yan GX, Antzelevitch C. Cellular basis for the Brugada syndrome and other mechanisms of arrhythmogenesis associated with ST-segment elevation. Circulation. 1999;100:1660-6.
41 Antzelevitch C, Oliva A. Amplification of spatial dispersion of repolarization underlies sudden cardiac death associated with catecholaminergic polymorphic VT, long QT, short QT and Brugada syndromes. J Intern Med. 2006;259:48-58.
42 Soar J, Deakin CD, Jerry P, Nolan JP, Gamal Abbas G, Alfonzo A, et al. European Resuscitation Council Guidelines for Resuscitation 2005: Section 7. Cardiac arrest in special circumstances. Resuscitation. 2005;(Supl. 1):156.
43 Resumen de los aspectos más destacados de las guías 2005 para la RCP de la American Heart Association. Currents in Emergency Cardiovascular Care, vol 16, nº 4. Invierno 2005-2006. Traducción revisada por SEMES.
44 Link MS, Bir C, Dau N, Madias C, Estes NAM III, Maron BJ. Protecting our children from the consequences of chest blows on the playing field: a time for science over marketing. Pediatrics. 2008;122:437-9.
45 Strasburger JF, Maron BJ. Commotio cordis. N Engl J Med. 2002;347:1248.
46 Ayuso Baptista F, Jiménez Moral G, Fonseca del Pozo FJ, Ruiz Madruga M, Garijo Pérez A, Jiménez Corona J, et al. Nuevos horizontes

frente a la muerte súbita cardiaca: la desfibrilación externa semiautomática. Emergencias. 2003;15:36-48.
47 Handley AJ, Koster R, Monsieurs K, Perkins GD, Davies S, Bossaert L. European Resuscitation Council Guidelines for Resuscitation 2005 Section 2. Adult basic life support and use of automated external defibrillators. Resuscitation. 2005;67(Supl. 1):S7-S23.
48 Dirección General de la Agencia de Calidad del SNS. Oficina de Planificación Sanitaria y Calidad. Desfibrilación Semiautomática en España: Informe. Ministerio de Sanidad y Política Social, 2007. (Consultado 3 Mayo 2010). Disponible en: http://www.msc.es/organizacion/sns/planCalidadSNS/docs/Informe_uso_Desfibriladores_sep_07.pdf
49 Suárez-Mier MP, Aguilera B. Causas de muerte súbita en deportistas en España. Rev Esp Cardiol. 2002;55:347-58.
50 Lee C-C, Chang W-T, Chen S-C, Yen Z-S, Chen W-J. Successful resuscitation after sudden death in a one year old infant who sustained a blunt chest injury from a fall from 10 m. Resuscitation. 2005;64:241-3.
51 Caso real en video de un episodio fatal de *commotio cordis.* (Consultado 15 Junio 2010). Disponible en: http://www.youtube.com/watch?v=LLtzT2bXVGI

Muerte súbita cardiaca causada por *commotio cordis*

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Los eventos de muerte súbita cardiaca (MSC) en corazones estructuralmente normales causados por impactos de escasa entidad en la región precordial se denominan *commotio cordis.* Las víctimas suelen ser muy jóvenes, habitualmente se produce en el contexto de prácticas deportivas, aunque también se ha descrito su aparición durante actividades cotidianas. La patogenia parece estar relacionada con un fenómeno de "R sobre T" en un instante particularmente vulnerable de la repolarización cardiaca, que desencadena una parada cardiorrespiratoria (PCR) por fibrilación ventricular. Recientemente, la aparición de registros específicos de casos ha permitido indagar sobre las características epidemiológicas y ensayar medios físicos de prevención de la *commotio cordis.* Sin embargo, aunque se produzca una instauración precoz del soporte vital básico y la activación de la cadena de supervivencia, las tasas de recuperación de PCR causadas por *commotio cordis* son bastante inferiores a lo esperable por la edad y el estado de salud de las víctimas; y con un desenlace, generalmente fatal. [Emergencias 2011;23:471-478]

Palabras clave: *Commotio cordis.* Fibrilación ventricular. Muerte súbita cardiaca. Medicina deportiva. Parada cardiaca.