

Commotio Cordis

Ventricular Fibrillation Triggered by Chest Impact–Induced Abnormalities in Repolarization

Mark S. Link

Commotio cordis is a phenomenon in which a sudden blunt impact to the chest causes sudden death in the absence of cardiac damage. This condition was first described in the middle of the 18th century in the context of chest trauma among workers.¹ Through most of the 20th century, it was only sporadically reported. In the last 2 or 3 decades, commotio cordis events have primarily occurred in sports, and thus, this phenomenon has become more well known to the sports communities and physicians.^{2–4} Commotio cordis is to be differentiated from cardiac contusion (contusio cordis), a situation in which blunt chest trauma causes structural cardiac damage, such as observed in motor vehicular accidents.

Commotio Cordis in Humans

Epidemiology

Approximately 10 to 20 cases are added to the Commotio Cordis Registry yearly.^{3,4} Until the late 1990s, commotio cordis was only rarely reported. It is thought that this increase in the number of cases is not due to an increase in incidence but rather to a greater awareness based on the 1995 *New England Journal of Medicine* report on commotio cordis.² Many more cases of commotio cordis are now recognized as such. Indeed, what was thought to be a uniquely North American phenomenon is increasingly being reported in countries outside the United States.⁵

Commotio cordis primarily affects young individuals, generally in adolescence. In the Registry, the mean age is 15 years⁴; there have been very few commotio cordis victims over the age of 20 years. It traditionally has been thought that the stiffening of the chest wall contributes to this decrease in incidence in older individuals; however, this decreased incidence in those over 20 years of age is likely also influenced by the reduced ball-related sports participation by older individuals. Victims are overwhelmingly male. A partial explanation for the overwhelming predominance of males is that they populate the majority of sports in which commotio occurs, but it appears unlikely that the 95% predilection for males reflects a 95% incidence of chest wall impact in sports and activities of daily living. I suspect that there may also be some gender-related biological susceptibility to chest wall impact induced sudden cardiac death. Indeed, other arrhyth-

mic conditions demonstrate a gender predilection for arrhythmia, including females with long-QT syndrome^{6,7} and males with Brugada syndrome.⁸ Genetic differences in ion channels between the sexes or biological modification of these channels by sex hormones may be involved in the male susceptibility to commotio cordis.

Sports

Baseball is the most common sport in which commotio occurs. Nearly all commotio events are caused by direct baseball strikes to the left chest wall over the cardiac silhouette. Pitchers, catchers, and batters have the highest incidence of commotio cordis, likely because of the frequency of chest wall strikes; however, all players can be affected by this phenomenon. There is evidence from early baseball reports that commotio cordis occurred in the early 20th century, even though it was not recognized as such. In a 1900 to 1910 archival baseball report, there were 19 reported deaths caused by ball strikes over the heart.⁹ Lacrosse, with its increased popularity, is reporting a rising incidence of commotio cordis events.¹⁰ Other common sports in which commotio cordis occur are hockey and softball, yet commotio cordis has been described in nearly all sports. In those sports in which there is no solid hard ball, commotio occurs secondary to impacts with elbows, fists, and helmets. Commotio is also reported in activities of daily living in which impact to the chest wall occurs with fists or other hard compact objects.

Resuscitation

Resuscitation, once thought to be nearly universally unsuccessful, has now been demonstrated to be successful in up to 35% of commotio cordis victims.⁴ The initial rhythm is ventricular fibrillation in those in whom a defibrillator is used relatively early after the event. Currently, the outcome of resuscitation in commotio cordis appears to be very similar to that for resuscitation in other forms of sudden cardiac death. Whether syncope associated with chest wall impact is an aborted commotio cordis event secondary to nonsustained ventricular fibrillation or transient complete heart block has not yet been determined.

From the Cardiac Arrhythmia Service, Department of Medicine, Tufts Medical School, Tufts University School of Medicine, Boston, MA. Correspondence to Mark S. Link, MD, Tufts Medical Center, 800 Washington St, Box 197, Boston, MA 02111. E-mailmlink@tuftsmedicalcenter.org (*Circ Arrhythm Electrophysiol*. 2012;5:425-432.)
© 2012 American Heart Association, Inc.

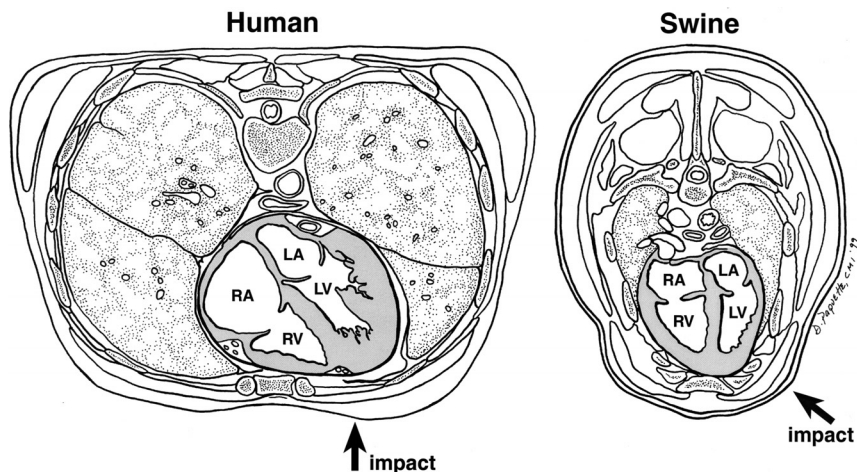


Figure 1. Cross-sectional view of a swine and human torso. Although the swine torso is ovoid compared with the human torso, there are similarities in the position of the heart relative to the chest wall. In both, there is no lung tissue between the ventricles and the chest wall. In human commotio cordis, the ball strikes the chest directly over the ventricles, and in the swine, the ball strikes the chest directly over the left ventricle. Thus, ball strikes relative to the heart are similar in humans and in our swine model. RA indicates right atrium; LA, left atrium; RV, right ventricle; and LV, left ventricle.

Individual Susceptibility

There may be a component of individual susceptibility to commotio cordis, similar to that of individual susceptibility to a prolonged QT interval and torsade de pointes with drugs that affect cardiac repolarization channels.^{11–13} This concept of repolarization reserve or subclinical long-QT syndrome has been evaluated more formally in drug-induced QT prolongation but may also be present in commotio cordis. I am personally aware of 1 adolescent who experienced 2 nonfatal episodes of commotio cordis. One event was caused by a fist blow to the chest, after which he collapsed, became unresponsive, and turned blue for 1 minute, with spontaneous awakening. The second occurred 1 year later when he was struck in the left chest with a ball during baseball practice. His symptoms and time course were identical to the first episode.

Experimental Models of Commotio Cordis

An early animal model of commotio cordis was developed by Georg Schlomka at Bonn University using hammer blows to the chest wall of rabbits, cats, and dogs.¹⁴ Schlomka identified 3 characteristics of the impact necessary for induction of cardiac arrhythmias (type, force, and location) and surmised that a mechanically induced coronary vasospasm might be responsible for the various arrhythmias encountered with chest impact.

Tufts Medical Center Model

An animal model more relevant to sports was developed at Tufts Medical Center shortly after the 1995 article by Maron et al was published.² This animal model sought to recreate the clinical characteristics of commotio as it was described in sporting events. This model has demonstrated that the arrhythmia that causes commotio cordis is ventricular fibrillation. It has also allowed the investigation of variables involved in the creation of the ventricular fibrillation and offered a rudimentary understanding of the mechanism(s) of commotio cordis. In this model, a juvenile male swine is anesthetized and placed feet down in a sling to approximate physiological conditions. The swine's heart lies opposed to the left chest wall, which is ovoid in shape (Figure 1). A baseball or lacrosse ball strikes the left chest wall directly

over the cardiac silhouette, where there is no interposed lung tissue (as in humans). The distance from the external skin of a 20-kg swine to the myocardium is approximately 2 cm. High-speed video has shown penetration of a 40-mph lacrosse ball into the thoracic cavity averages 3.7 ± 0.7 cm in a 20-kg swine and can reach depths of up to 6 cm (unpublished data). In the early model in which the ball strikes were given throughout the cardiac cycle, ventricular fibrillation was induced by impact number 3 in swine number 9 (Figure 2). This impact occurred 27 ms before the T-wave peak. Impacts in subsequent animals demonstrated that ventricular fibrillation was reliably induced with impacts in a vulnerable time period of the cardiac cycle. Other series evaluated the importance of additional critical variables such as location, energy, and shape and hardness of the object as determinants of ventricular fibrillation. With impacts <50 mph, rib fractures, hemothorax, hemopericardium, or evidence of myocardial contusion are not observed. Histological examination of the left and right ventricles in early animals showed a moderate degree of hemorrhage in the anterior left ventricle. With specialized examination of the conduction system, 1 of the 2 swine studied with transient induced heart block had marked hemorrhage of the atrioventricular bundle and bundle branches, and 1 of 2 animals with ventricular fibrillation had mild hemorrhage in the periphery of the left bundle branch.

Variables Critical to the Induction of Ventricular Fibrillation

The confluence of several critical variables is necessary to induce ventricular fibrillation with a ball impact (Figure 3).¹⁵ If all variables currently known are maximized, approximately 30% of 30-mph impacts and 50% of 40-mph impacts will cause ventricular fibrillation in a 20-kg swine.¹⁶ Perhaps the most important variable is the timing of impact relative to the cardiac cycle (Figure 4). Only impacts on a narrow region on the upslope of the T wave (40 ms before the peak of the T-wave peak to the peak of the T wave) will cause ventricular fibrillation, with a markedly increased likelihood with impacts from 30 to 10 ms before the peak of the T wave. Although polymorphic ventricular tachycardia was seen more frequently during impacts at these time periods, it was also occasionally induced by impacts at other times in the cardiac

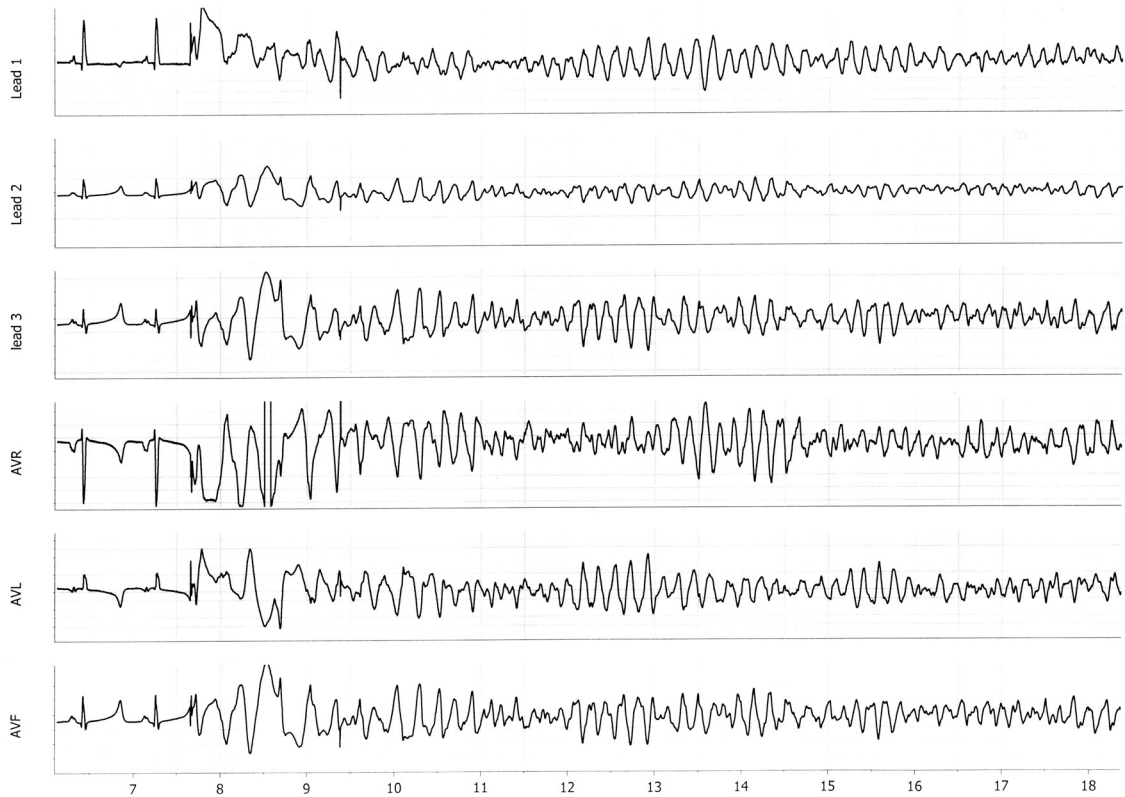


Figure 2. The first ventricular fibrillation produced in the Tufts laboratory. A 7.5-kg swine was struck by a 30-mph wooden sphere directly over the cardiac silhouette 27 ms before the T-wave peak. Ventricular fibrillation was immediate. Note how the initial few seconds were consistent with polymorphic ventricular tachycardia before the arrhythmia broke down and became an arrhythmia of lower amplitude and higher frequency.

cycle. This polymorphic ventricular tachycardia was identical in morphology to the initiation of ventricular fibrillation; however, it only continued for up to 10 beats. If the polymorphic ventricular tachycardia continued beyond 10 beats, it degenerated into ventricular fibrillation. Heart block is rarely seen and is more common with smaller animals, with baseball compared with lacrosse ball impacts, and with a higher velocity of impact (unpublished data). Impacts throughout the cardiac cycle may cause ST-segment elevation and left bundle-branch block in those impacts in which ventricular fibrillation is not induced. Premature ventricular beats are nearly always observed.

Impacts are given perpendicular to the chest wall (the occasional glancing blow will never cause ventricular fibrillation). Impacts must occur directly over the center of the left ventricle in order for ventricular fibrillation to regularly occur, although strikes still over the left ventricle but more peripheral can occasionally cause ventricular fibrillation.¹⁷ Strikes not over the cardiac silhouette have never caused ventricular fibrillation. Not surprisingly, ventricular fibrillation is more easily induced in a smaller swine than in a larger animal. Indeed, there appears to be a significant drop-off in ventricular fibrillation inducibility with animal weights >40 kg. Velocity of the impact object is also critical. Impacts at 40 mph were the most likely to cause ventricular fibrillation in a 20-kg swine.¹⁸ At velocities of 20 mph, ventricular fibrillation was never induced, and at velocities of 50 to 70 mph, the occurrence of ventricular fibrillation dropped and the inci-

dence of cardiac rupture and major trauma increased. Thus, at these velocities, our model becomes more of a cardiac contusion model. An important concept for prevention is that softer, more pliable objects are less likely to cause ventricular fibrillation.^{19,20} More recently, the shape of the impact object was assessed. Spheres with smaller radii were more likely to induce ventricular fibrillation, whereas ventricular fibrillation never occurred with flat objects.²¹ Smaller spheres were also more likely to cause ST-segment elevation and left bundle-branch block.

Mechanism

What is commonly known as ventricular fibrillation is a surface tracing that results from multiple wavelets in the myocardium at the same time. This leads to a surface ECG of the summation of the wavelets and thus complete disorganization. However, internally and focally, ventricular fibrillation is much more organized, especially in the initial stages. It is hypothesized that ventricular fibrillation can be the result of a mother rotor, either stationary or roaming throughout the myocardium, or multiple random wandering wavelets.²² In fact, the mechanism of ventricular fibrillation in commotio cordis may be similar to or different from conditions such as acute ischemia, electric shock on T-wave, long-QT syndrome, and Brugada syndrome. By surface morphology, ventricular fibrillation in commotio cordis appears as it does in other situations: initially coarse and undulating (like torsade de pointes/polymorphic ventricular tachycardia).²³

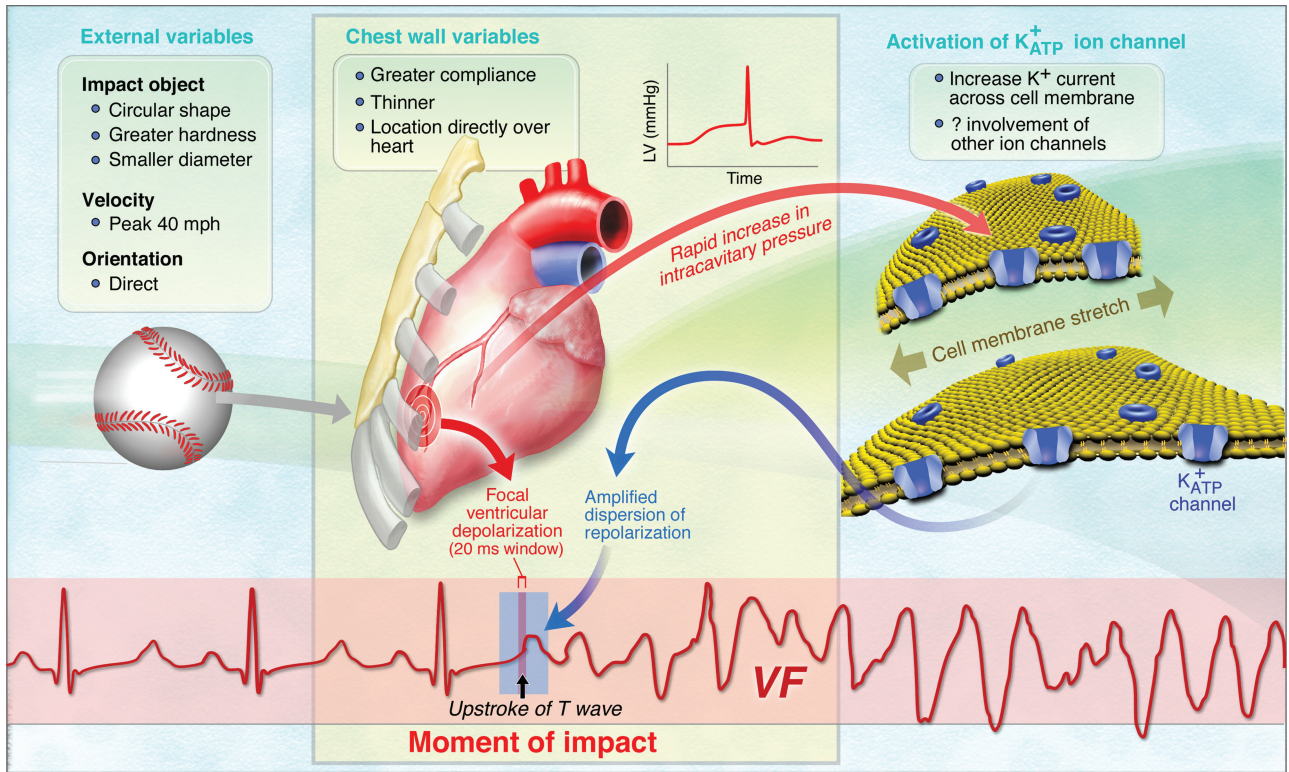


Figure 3. The confluence of variables and a proposed mechanism necessary for commotio cordis to occur. Important impact-object variables are shape, hardness, diameter, and velocity. Human characteristics are the pliability of the chest wall, impact timing, location and orientation of blow, and individual susceptibility, likely carried in ion channels involved in repolarization. LV indicates left ventricle. Reprinted from the *Journal of Cardiovascular Electrophysiology*, with permission.¹⁵

Over a relatively short time, this coarse polymorphic ventricular tachycardia will degenerate into a lower-amplitude and higher-frequency (“finer”) ventricular fibrillation. This ventricular fibrillation will continue for up to 30 minutes if the animal is not defibrillated.

Alterations in Repolarization

I suspect that the ventricular fibrillation in commotio cordis is an acquired form of abnormal repolarization. Abnormalities in repolarization are caused by impact and likely increase the heterogeneity of repolarization, which makes the myocardial

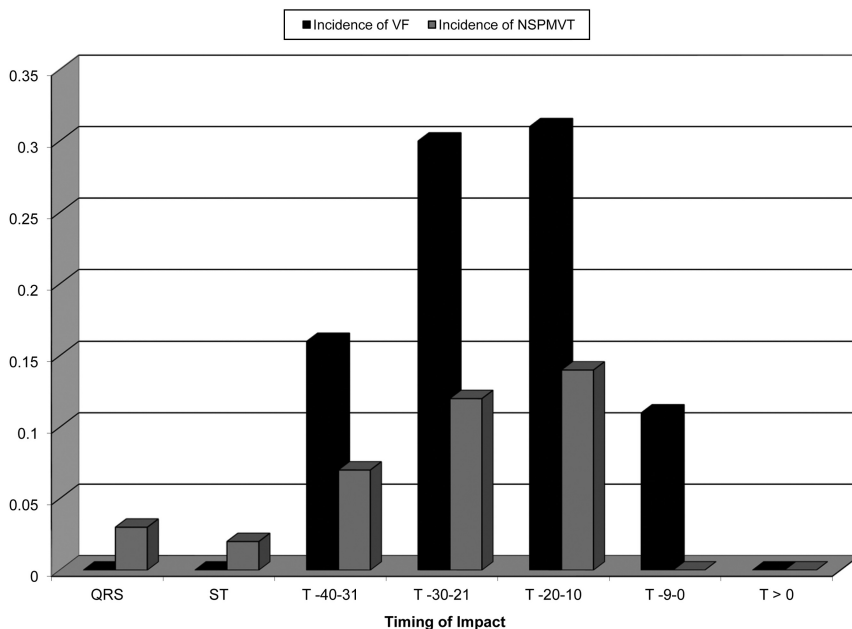


Figure 4. Incidence of ventricular fibrillation (VF) and nonsustained polymorphic ventricular tachycardia (NSPMVT) relative to the timing of the cardiac cycle. Impacts were at 30 and 40 mph. VF was observed in approximately 30% of impacts that occurred in cardiac repolarization 30 to 10 ms before the T-wave peak. Nonsustained polymorphic ventricular tachycardia was predominantly observed during this time window but occasionally was seen with strikes during the QRS and ST segments. Reprinted from *Heart Rhythm*, with permission.¹⁶

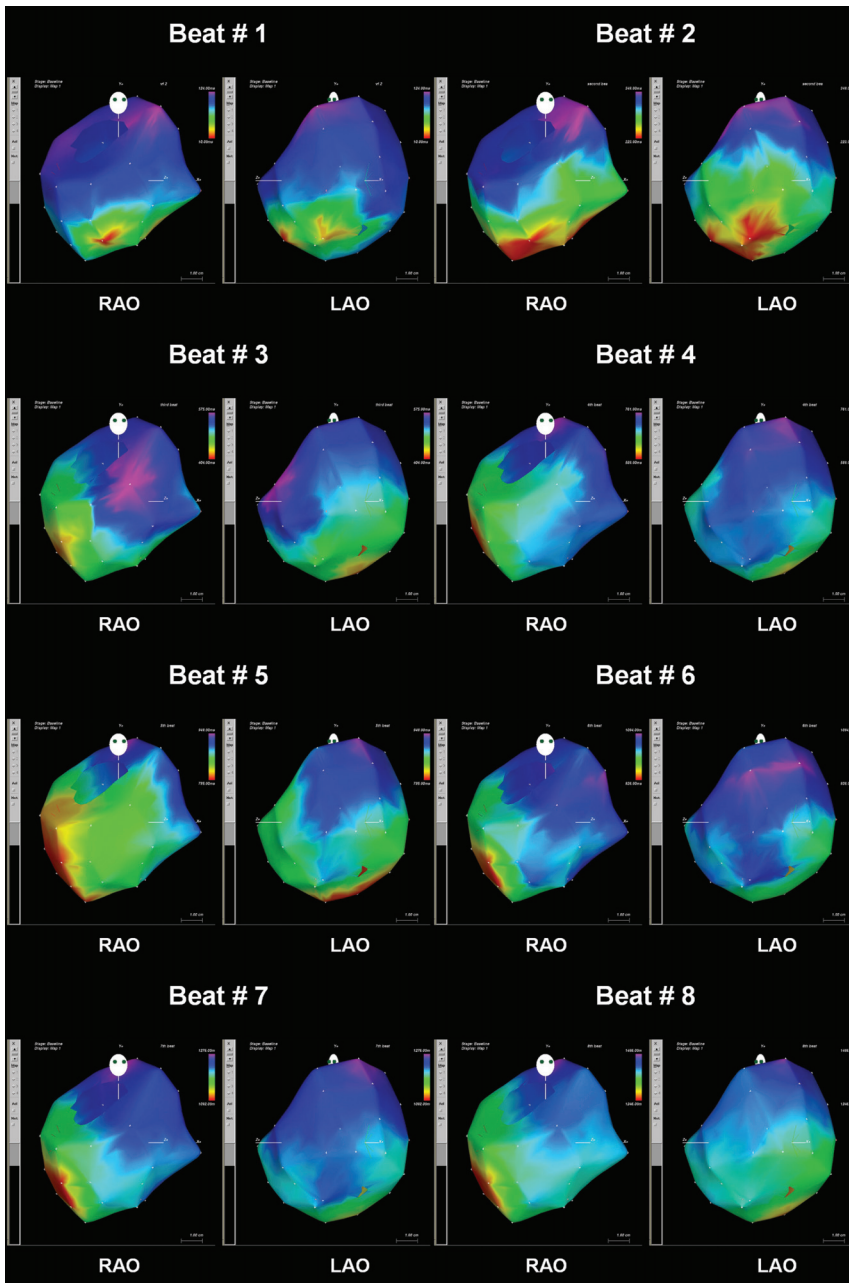


Figure 5. Three-dimensional endocardial mapping of the first 8 beats of ventricular fibrillation in commotio cordis. Note how it appears that a mother rotor is centered in the septum of the myocardium. RAO indicates right anterior oblique; LAO, left anterior oblique. Adapted from *Heart Rhythm*, with permission.²⁴

substrate ripe for a reentrant arrhythmia (Figure 5).²⁴ However, temporal alteration of the myocardial substrate alone is not sufficient. There also must be a trigger, an initial ventricular arrhythmic depolarization. This trigger could be an afterdepolarization, such as is present in long-QT situations, or a premature ventricular depolarization (induced by the blow), as is present in the R-on-T phenomenon in acute ischemia. Support for this dual-abnormality hypothesis lies in the fact that in impacts that do not induce ventricular fibrillation, 2 predominant abnormalities are observed: that of alteration of repolarization as manifested by ST-segment elevation and that of a premature ventricular contraction. Support for changes in repolarization is also observed in an elegant Langendorff preparation in which a rabbit heart is exposed to acute left ventricular pressure pulses.²⁵ In this

model, acute pressure changes mediated via a fluid-filled balloon in the left ventricle caused a dispersion of repolarization, and if the timing of the balloon inflation occurred during a narrow window of repolarization, ventricular fibrillation could result.

Altered repolarization is caused by current flows, predominantly mediated via increases in left ventricular pressure. In all experimental protocols in which left ventricular pressure was measured, the increase in left ventricular pressure caused by the ball blow correlated with the risk of ventricular fibrillation occurring with the blow. The minimum pressure necessary to induce ventricular fibrillation appears to be 250 mm Hg, and the optimal increase is 400 to 500 mm Hg (Figure 6). Higher-velocity impacts that cause pressure increases of >600 mm Hg typically cause structural damage

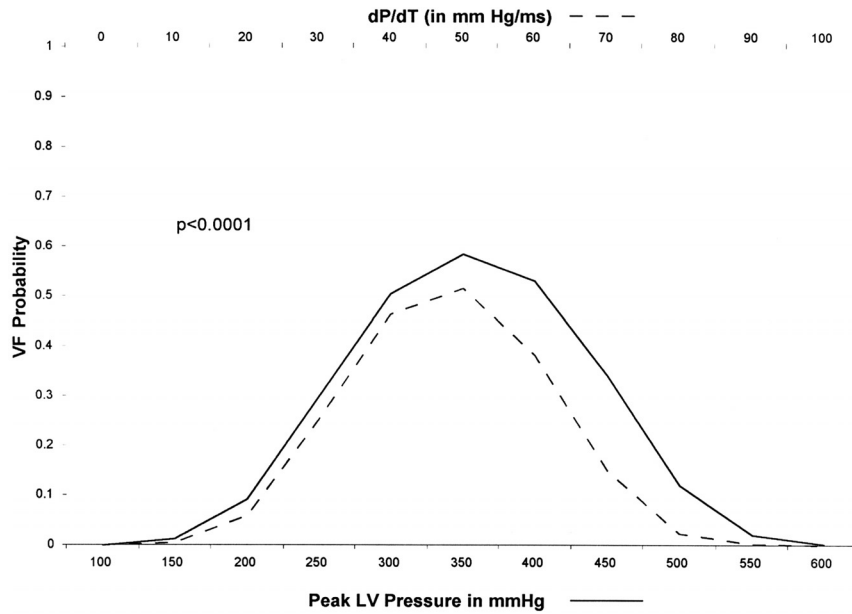


Figure 6. The association of peak left ventricular (LV) pressure induced by the chest blow and the incidence of ventricular fibrillation (VF). The occurrence of VF was highest with peak left ventricular pressure rises from 250 to 450 mm Hg. Reprinted from the *Journal of the American College of Cardiology*, with permission.¹⁸

such as myocardial rupture and acute mitral valve insufficiency, and thus, at these left ventricular pressures, our model becomes one of cardiac contusion.

Immediate Trigger

The initial depolarization trigger is likely a focal phenomenon from direct impact, similar to a premature ventricular contraction induced by a catheter, as seen in electrophysiology and cardiac catheterization laboratories. Alternatively, it could be an afterdepolarization induced by changes in current flow. This 2-step process is similar to the R-on-T phenomenon, in which a premature ventricular contraction that occurs on the upslope of the T wave will cause ventricular fibrillation in acute ischemic conditions but not in nonischemic situations. This 2-step process is also observed in the long-QT syndrome, in which an afterdepolarization is necessary to initiate the torsade de pointes seen in this condition. Ongoing mapping studies using real-time 3-dimensional mapping in our laboratory should help elucidate the mechanism of ventricular fibrillation in commotio cordis.

Ion Channels Involved in Altered Repolarization

The candidate ion channels for the altered repolarization are those whose conduction is known to be altered by stretch or pressure changes. These channels include the calcium stretch-activated channel, the K_{ATP} channel, certain other known potassium and sodium channels, and possibly even some unknown channels. The first candidate channel evaluated was the K_{ATP} channel. We chose this channel because of the similarities of ventricular fibrillation and ST-segment elevation between our experimental model and acute ischemia.^{26,27} In addition, this channel is known to be activated by stretch.²⁸ Indeed, in our animal model, blocking the K_{ATP} channel with glibenclamide nearly eliminated ventricular fibrillation.²⁹ In this study, 20 swine were randomized to glibenclamide or a control vehicle, and with T-wave impacts, glibenclamide animals had significantly fewer occurrences of ventricular

fibrillation (1 episode in 27 impacts [4%]) than controls (6 episodes in 18 impacts [33%]; $P=0.01$).

Another stretch-activated channel, the nonspecific calcium stretch-activated channel, was also evaluated in our model; however, the infusion of streptomycin, an agent that blocks this channel, did not alter the incidence of ventricular fibrillation.³⁰ In another series, blocking the calcium channel with verapamil did not affect the incidence of ventricular fibrillation.

Genetic Susceptibility

Genetic susceptibility to commotio cordis is likely carried in genotypes of repolarization channels. In our animal model, there is evidence of individual susceptibility to ball strikes in that some animals are uniquely susceptible to chest wall-induced ventricular fibrillation, whereas others are quite resistant (Figure 7).¹² In this study of 1274 total impacts in 139 swine, 360 impacts (28%) resulted in ventricular fibril-

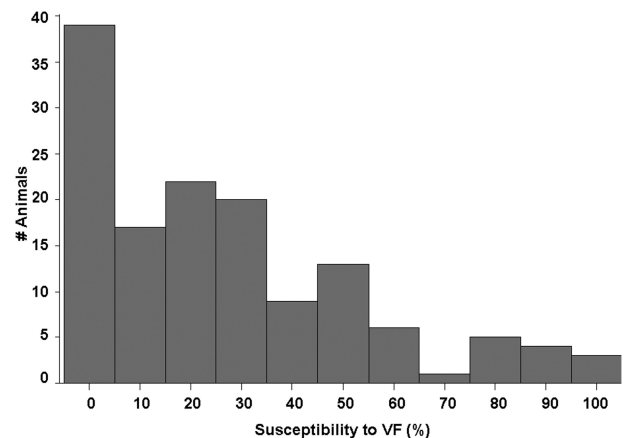


Figure 7. Marked variability to the induction of ventricular fibrillation (VF) with ball impacts with all variables optimized. In this analysis of 139 swine with 1274 impacts, a small minority of swine were uniquely susceptible to ball-induced VF. Reprinted from *Circulation*, with permission.¹²

lation; however, in 38 animals, none of the impacts resulted in ventricular fibrillation, and only 7 swine (5%) had >80% occurrence of ventricular fibrillation with chest impacts. On the basis of our swine study, I believe that the animals that exhibit unique susceptibility to ventricular fibrillation with chest impact may carry indolent forms of long-QT syndrome. This subclinical long QT would be similar to what is seen in humans with a normal QT at baseline but who are susceptible to a prolonged QT interval with QT-prolonging drugs, hypokalemia, or other stressors of repolarization. Individual susceptibility is also likely present for the male sex. Although we have no animal data to support this hypothesis (we include only males in our studies), commotio cordis predominantly affects human males. It may be that males possess less repolarization reserve or possess ion channels that are more susceptible to wall stretch or increases in left ventricular pressures.

Resuscitation in the Experimental Model

At a time when the human reports purported a dismal response to resuscitation, our experimental model demonstrated that resuscitation in commotio cordis is possible, especially if early defibrillation is performed.³¹ In our study,³¹ all episodes of ventricular fibrillation were recognized and terminated by automated external defibrillators, and if defibrillation occurred within 2 minutes, the majority of animals had an immediate return of spontaneous circulation.

Prevention of Commotio Cordis

Commotio cordis can be prevented in many circumstances, but it is likely the risk cannot be eliminated entirely. One avenue of prevention is safety baseballs. These safety baseballs are available in different degrees of hardness. Safety baseballs marketed for T-ball are quite pliable and elastic; however, there are more playable balls for older age groups that are intermediate in pliability between the T-balls and standard baseballs. In our animal model, we found that T-balls were associated with the lowest risk of ventricular fibrillation with ball impact, but there was also decreased risk with the intermediate grades of pliability.^{19,20} In 30-mph baseball impacts, ventricular fibrillation occurred with 7% of impacts with the T-balls compared with 22% and 29% with intermediate grades of baseballs and 35% with a regulation baseball ($P<0.0001$).¹⁹ At 40 mph, the softest safety baseballs triggered ventricular fibrillation in only 11% of impacts compared with 19% and 22% with safety baseballs of intermediate hardness and 69% with standard baseballs ($P<0.01$).²⁰ The Consumer Product Safety Commission has also recommended use of age-appropriate baseballs to decrease the risk of structural injury in baseball.³²

Chest protectors are another potential means to prevent commotio cordis; however, their benefit has not yet been shown. In commotio events in competitive athletics, approximately one third of individuals were wearing chest wall protection at the time of their cardiac arrest. In some instances, such as in hockey, these chest wall protectors were lifted from the cardiac silhouette by lifting the arms³³; however, in sports such as baseball and lacrosse, the ball struck the chest protector while it lay directly over the

heart.^{10,33} In our animal model, we found that commercially available baseball and lacrosse chest wall protectors did not lower the risk of commotio cordis.³⁴ High-speed cinematography has shown that baseball penetration into the chest is not inhibited by the use of these chest protectors. These observations point to the need for the development of more effective chest wall protectors.³⁵

Conclusions

Commotio cordis is a significant cause of morbidity and mortality on the playing field. The epidemiology of commotio cordis has been well established to include young male athletes participating in sports with a solid small ball. Resuscitation is possible. Experimental models point to the importance of timing, location, velocity, and shape and hardness of the object as determinants of ventricular fibrillation. Biological characteristics such as sex, pliability of the chest wall, and genetic susceptibility also play a role. The mechanism of ventricular fibrillation in commotio cordis is likely a combination of impact-initiated heterogeneity of repolarization and a premature ventricular depolarization trigger. A reduction in the risk of commotio cordis is possible with age-appropriate safety baseballs.

Acknowledgments

The author thanks Darisse A. Paquette, CMI, for her drawing of Figure 1.

Disclosures

Dr Link has a patent on a chest protector that is designed to reduce the risk of sudden death with chest wall impact. This chest protector is licensed to Cascade Sports and is not discussed in this article.

References

1. Nesbitt AD, Cooper PJ, Kohl P. Rediscovering commotio cordis. *Lancet*. 2001;357:1195–1197.
2. Maron BJ, Poliac LC, Kaplan JA, Mueller FO. Blunt impact to the chest leading to sudden death from cardiac arrest during sports activities. *N Engl J Med*. 1995;333:337–342.
3. Madias C, Maron BJ, Weinstock J, Estes NA 3rd, Link MS. Commotio cordis: sudden cardiac death with chest wall impact. *J Cardiovasc Electrophysiol*. 2007;18:115–122.
4. Maron BJ, Estes NA 3rd. Commotio cordis. *N Engl J Med*. 2010;362:917–927.
5. Maron BJ, Ahluwalia A, Haas TS, Semsarian C, Link MS, Mark Estes NA 3rd. Global epidemiology and demographics of commotio cordis. *Heart Rhythm*. 2011;8:1969–1971.
6. Zareba W, Moss AJ, Locati EH, Lehmann MH, Peterson DR, Hall WJ, Schwartz PJ, Vincent GM, Priori SG, Benhorin J, Towbin JA, Robinson JL, Andrews ML, Napolitano C, Timothy K, Zhang L, Medina A. Modulating effects of age and gender on the clinical course of long QT syndrome by genotype. *J Am Coll Cardiol*. 2003;42:103–109.
7. Goldenberg I, Moss AJ, Bradley J, Polonsky S, Peterson DR, McNitt S, Zareba W, Andrews ML, Robinson JL, Ackerman MJ, Benhorin J, Kaufman ES, Locati EH, Napolitano C, Priori SG, Qi M, Schwartz PJ, Towbin JA, Vincent GM, Zhang L. Long-QT syndrome after age 40. *Circulation*. 2008;117:2192–2201.
8. Gehi AK, Duong TD, Metz LD, Gomes JA, Mehta D. Risk stratification of individuals with the Brugada electrocardiogram: a meta-analysis. *J Cardiovasc Electrophysiol*. 2006;17:577–583.
9. Maron BA, Boren SD, Estes NAM 3rd. Early descriptions of sudden cardiac death due to commotio cordis occurring in baseball. *Heart Rhythm*. 2010;7:992–993.
10. Maron BJ, Doerer JJ, Haas TS, Estes NA, Hodges JS, Link MS. Commotio cordis and the epidemiology of sudden death in competitive lacrosse. *Pediatrics*. 2009;124:966–971.

11. Knollmann BC, Roden DM. A genetic framework for improving arrhythmia therapy. *Nature*. 2008;451:929–936.
12. Alsheikh-Ali AA, Madias C, Supran S, Link MS. Marked variability in susceptibility to ventricular fibrillation in an experimental commotio cordis model. *Circulation*. 2010;122:2499–2504.
13. Roden DM. Drug-induced prolongation of the QT interval. *N Engl J Med*. 2004;350:1013–1022.
14. Schlomka G. Commotio cordis und ihre folgen: die einwirkung stumpfer brustwandtraumen auf das herz. *Ergebn Inn Med Kinderheilk*. 1934;47:1–91.
15. Link MS, Estes NA. Athletes and arrhythmias. *J Cardiovasc Electrophysiol*. 2010;21:1184–1189.
16. Link MS, Estes NA 3rd. Mechanically induced ventricular fibrillation (commotio cordis). *Heart Rhythm*. 2007;4:529–532.
17. Link MS, Maron BJ, VanderBrink BA, Takeuchi M, Pandian NG, Wang PJ, Estes NA 3rd. Impact directly over the cardiac silhouette is necessary to produce ventricular fibrillation in an experimental model of commotio cordis. *J Am Coll Cardiol*. 2001;37:649–654.
18. Link MS, Maron BJ, Wang PJ, VanderBrink BA, Zhu W, Estes NA 3rd. Upper and lower limits of vulnerability to sudden arrhythmic death with chest-wall impact (commotio cordis). *J Am Coll Cardiol*. 2003;41:99–104.
19. Link MS, Wang PJ, Pandian NG, Bharati S, Udelson JE, Lee MY, Vecchiotti MA, VanderBrink BA, Mirra G, Maron BJ, Estes NA 3rd. An experimental model of sudden death due to low-energy chest-wall impact (commotio cordis). *N Engl J Med*. 1998;338:1805–1811.
20. Link MS, Maron BJ, Wang PJ, Pandian NG, VanderBrink BA, Estes NA 3rd. Reduced risk of sudden death from chest wall blows (commotio cordis) with safety baseballs. *Pediatrics*. 2002;109:873–877.
21. Kalin J, Madias C, Alsheikh-Ali AA, Link MS. Reduced diameter spheres increases the risk of chest blow-induced ventricular fibrillation (commotio cordis). *Heart Rhythm*. 2011;8:1578–1581.
22. Vaquero M, Calvo D, Jalife J. Cardiac fibrillation: from ion channels to rotors in the human heart. *Heart Rhythm*. 2008;5:872–879.
23. Wiggers CJ. The mechanism and nature of ventricular fibrillation. *Am Heart J*. 1940;20:399–412.
24. Alsheikh-Ali AA, Akelman C, Madias C, Link MS. Endocardial mapping of ventricular fibrillation in commotio cordis. *Heart Rhythm*. 2008;5:1355–1356.
25. 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;17:1011–1017.
26. Kondo T, Kubota I, Tachibana H, Yamaki M, Tomoike H. Glibenclamide attenuates peaked T wave in early phase of myocardial ischemia. *Cardiovasc Res*. 1996;31:683–687.
27. Kubota I, Yamaki M, Shibata T, Ikeno E, Hosoya Y, Tomoike H. Role of ATP-sensitive K⁺ channel on ECG ST segment elevation during a bout of myocardial ischemia. *Circulation*. 1993;88:1845–1851.
28. Van Wagoner DR. Mechanosensitive gating of atrial ATP-sensitive potassium channels. *Circ Res*. 1993;72:973–983.
29. Link MS, Wang PJ, VanderBrink BA, Avelar E, Pandian NG, Maron BJ, Estes NA 3rd. 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;100:413–418.
30. Garan AR, Maron BJ, Wang PJ, Estes NA 3rd, Link MS. Role of streptomycin-sensitive stretch-activated channel in chest wall impact induced sudden death (commotio cordis). *J Cardiovasc Electrophysiol*. 2005;16:433–438.
31. Link MS, Maron BJ, Stickney RE, Vanderbrink BA, Zhu W, Pandian NG, Wang PJ, Estes NA 3rd. Automated external defibrillator arrhythmia detection in a model of cardiac arrest due to commotio cordis. *J Cardiovasc Electrophysiol*. 2003;14:83–87.
32. Adler P, Monticone RCJ. Injuries and deaths related to baseball. In: Kyle SB, ed. *Youth Baseball Protective Equipment Project Final Report*. Washington, DC: US Consumer Product Safety Commission; 1996:1–43.
33. Doerer JJ, Haas TS, Estes NA 3rd, Link MS, Maron BJ. Evaluation of chest barriers for protection against sudden death due to commotio cordis. *Am J Cardiol*. 2007;99:857–859.
34. Weinstock J, Maron BJ, Song C, Mane PP, Estes NA 3rd, 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:e656–e662.
35. Link MS, Bir C, Dau N, Madias C, Estes NA 3rd, Maron BJ. Protecting our children from the consequences of chest blows on the playing field: a time for science over marketing. *Pediatrics*. 2008;122:437–439.

KEY WORDS: long-QT syndrome ■ cardiac arrest, sudden ■ death, sudden ■ ventricular fibrillation ■ athletes