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Review

## Mechanically induced sudden death in chest wall impact (commotio cordis)

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### Abstract

Sudden death due to nonpenetrating chest wall impact in the absence of injury to the ribs, sternum and heart is known as commotio cordis. Although once thought rare, an increasing number of these events have been reported. Indeed, a significant percentage of deaths on the athletic field are due to chest wall impact. Commotio cordis is most frequently observed in young individuals (age 4–18 years), but may also occur in adults. Sudden death is instantaneous or preceded by several seconds of lightheadedness after the chest wall blow. Victims are most often found in ventricular fibrillation, and successful resuscitation is more difficult than expected given the young age, excellent health of the victims, and the absence of structural heart disease. Autopsy examination is notable for the lack of any significant cardiac or thoracic abnormalities.

In an experimental model of commotio cordis utilizing anesthetized juvenile swine, ventricular fibrillation can be produced by a 30 mph baseball strike if the strike occurred during the vulnerable period of repolarization, on the upslope of the T-wave. Energy of the impact object was also found to be a critical variable with 40 mph baseballs more likely to cause ventricular fibrillation than velocities less or greater than 40 mph. In addition, more rigid impact objects and blows directly over the center of the chest were more likely to cause ventricular fibrillation. Peak left ventricular pressure generated by the chest wall blow correlated with the risk of ventricular fibrillation. Activation of the  $K_{ATP}^+$  channel is a likely cause of the ventricular fibrillation produced by chest wall blows. Successful resuscitation is attainable with early defibrillation.

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*Keywords:* Commotio cordis; Ventricular fibrillation; Athletes; Sudden death

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**1. Introduction**

Sudden death due to nonpenetrating chest wall impact in the absence of injury to the ribs, sternum and heart is increasingly reported. Although once considered to be a particularly rare event, it has become apparent that important proportions of deaths on the athletic field are due to chest wall impact. The incidence of this event has been thought to be 2–4 deaths per annum in the United States, but underreporting and misclassification of deaths undoubtedly occur and the true number of deaths due to relatively mild chest wall impacts is unknown. The Commotio Cordis Registry, in its 5 year existence, has documented > 140 cases of commotio cordis (Maron et al., 2002) and is accruing 5–10 cases/year. Indeed, examples of sudden deaths initially thought of as unexplained sudden deaths are now more properly reclassified as commotio cordis (Haq, 1998; Link, 1999).

**2. History**

Commotio cordis, or cardiac concussion, was first described in the 19th century medical literature to describe cardiac death secondary to chest wall blows (Nelaton, 1876; Meola, 1879; Kohl et al., 2001). Initial reports were confined to adults in workplace environments, with trauma of significant magnitude such as stone throws to the chest or falls from buildings. More recently, sudden death due to low-energy chest wall trauma in the absence of cardiac damage has been described in youth baseball and other sports (Maron et al., 1995, 2002). In addition, an increasing number of syncopal events secondary to chest wall impact (aborted sudden deaths) have been described and are likely due to nonsustained arrhythmic events (Nesbitt et al., 2001; Maron et al., 2002).

### 3. Human observations

*Demographics:* Commotio cordis predominantly affects young male individuals. In the Commotio Cordis Registry, the mean age is 14 years, with 78% <18 years of age (Maron et al., 1999, 2002). Young athletes may be particularly at risk because of a more pliable chest wall that facilitates the transmission of chest impact energy to the myocardium.

Baseball ( $n = 53$ ), softball ( $n = 14$ ), ice hockey ( $n = 12$ ), football ( $n = 8$ ) and lacrosse ( $n = 5$ ) are the common sports in which commotio cordis occurs. In addition, cases have been reported in a variety of other sports including karate, soccer, as well as nonsports related contact with a body part, usually hand, foot, or elbow (Maron et al., 2002).

*Circumstances of sudden death:* Instantaneous collapse following the chest blow occurs in approximately one-half of the victims. In the others, collapse follows a brief period of consciousness often marked by extreme lightheadedness (Maron et al., 1995). In the 82 cases from the Commotio Cordis Registry in which a rhythm was documented, 33 had VF. After prolonged resuscitation, asystole is commonly found (Maron et al., 2002). In the few survivors, in whom a 12 lead ECG was performed, the ECG showed marked ST segment elevation, especially in the anterior leads, which resolve over time (Link et al., 1998b).

*Implements:* In most commotio victims, the impact object is an implement of the game, i.e., a relatively hard object such as a baseball, hockey puck or lacrosse ball. With only two exceptions (a soccer ball and a plastic bat) all impact objects had a solid and not an air filled core (Maron et al., 2002).

*Force of blows:* Although it is difficult to quantify the energy or force of chest blows in victims of commotio cordis, in the vast majority of cases it appears that the velocity of the impact object is not unusual for the sport or activity involved. In youth baseball, the most common sport in which commotio cordis has been reported, a pitched baseball is generally at 30–50 mph (Seefeldt et al., 1993). In baseball, 25% of the deaths were secondary to pitched baseballs; the others occurred with batted balls or balls thrown from one player to another. In these cases the velocity of the baseball is more difficult to determine.

*Location of blows:* In commotio cordis victims, the chest blows strike the left chest. Most of these blows reportedly occur directly over the cardiac silhouette; however, the exact location of the chest wall strike cannot always be determined with precision (Maron et al., 1995).

*Survival:* Although initially reported to be almost invariably fatal, survival now appears to approach 15%, including some cases of spontaneous resolution of collapse after chest wall impact (Maron et al., 2002). Indeed, it is also possible that many spontaneously resolved events are never recognized as forme fruste commotio cordis and thus not reported to the Registry. As with other causes of VF, the most important determinant of survival is early resuscitation. Of 68 Registry events in which resuscitation was begun within 3 min 25% survived, compared to only 3% in which resuscitation was delayed for more than 3 min.

*Prevention:* Standard, commercially available chest wall protection was worn by 22 of 79 (28%) of the commotio cordis victims in organized sports (Maron et al., 2002). However, in 13 of these individuals the chest wall barriers did not adequately cover the left chest wall and precordium; these included at least 8 of 13 hockey players in which direct chest impact occurred due to the angulation of the shot or by the raising of arms causing displacement of the protector, and 5 football players in which standard shoulder/chest pads did not cover the precordium. However, in

others (including 3 lacrosse goalies, 2 baseball catchers, and 2 hockey goalies) the chest wall protector covered the heart and the projectile struck the chest protector; nonetheless, a commotio cordis event occurred (Maron et al., 2002).

Softer than standard or “safety” baseballs accounted for 2 of the 53 baseball induced commotio cordis events (Maron et al., 2002). However, there is no data on the relative frequency of safety baseball use relative to standard baseball use, and thus real comparisons in safety profile cannot be made with regard to safety baseballs in human impact.

#### 4. Experimental models of chest wall trauma

##### 4.1. Models prior to 1995

Early experimental efforts on chest wall trauma focused on more severe trauma, such as that typically seen with manual laborers, victims of motor vehicular accidents, falls from heights, and bomb blasts (Meola, 1879; Riedinger and Kummell, 1903). Because of the frequent cardiac damage produced in these experiments they may not be relevant to commotio cordis, in which myocardial damage is not observed. Riedinger distinguished between “contusion” in which morphologic damage was present and “commotion” in which morphologic damage was absent, although he commented on observing only two cases of true “commotion” (Riedinger and Kummell, 1903). In these early models sudden death was occasionally described and ascribed to heightened vagal stimulation.

In Schlomka’s experiments with hammer blows to the chest of various animals, frequent ECG changes (S-T segment abnormalities) were reported and VF was occasionally seen (Schlomka, 1934). The force of the blow was quantified only subjectively; most blows were of sufficient strength to cause readily evident cardiac damage. Severing or blocking the vagus nerve had no effect on the ECG or survival. Schlomka and colleagues (Schlomka, 1934) proposed that the immediate ECG and arrhythmic changes, and long-term pathologic changes (nearly 60% of animals followed for > 80 days had permanent heart damage), were due to either coronary spasm or trauma-related myocardial damage. Similarly, Bright in his experiments with direct blows in an open chest canine model, produced ECG changes of ST elevation (Bright and Beck, 1935). He replicated the ECG changes with blood injected into the myocardium, and thus ascribed the ECG changes to myocardial contusion and hemorrhage.

In the first experiments using measured force, ventricular arrhythmias were produced by impacts with high velocity or high-energy objects (Liedtke et al., 1974; Cooper et al., 1982). However, most animals were found to have severe cardiac damage that could account for their death and thus these experiments were more relevant to cardiac contusion than to commotio cordis. Only one experiment gated the chest impact to the cardiac cycle, and although VF was more commonly seen with T-wave impacts, the substantial energies of the blows (on average, equivalent to a 123 mph baseball) caused severe chest wall trauma in most swine (Cooper et al., 1982). In the early 1990s a swine model of chest wall impact was developed utilizing baseballs propelled at 95 mph (Viano et al., 1992). In this experiment, bradyarrhythmias and ventricular tachyarrhythmias were observed, but no correlation to the cardiac cycle was noted, and nearly all animals suffered cardiac contusions.

## 5. Our experimental model of commotio cordis

Because the aforementioned models utilized high-energy projectiles with resultant chest wall and cardiac damage, we were concerned that they did not necessarily evaluate the mechanisms of commotio cordis. Therefore, we developed an experimental model in swine which attempted to replicate the clinical scenario of commotio cordis. In this model, baseballs in free flight at velocities relevant to youth baseball were propelled to the chest wall of juvenile swine (8–22 kg) anesthetized with ketamine and isoflurane. The impact object was designed to strike the swine perpendicular to the chest wall, directly overlying the anatomic position of the left ventricle (Link et al., 1998a). The release of the impact object was timed to the cardiac cycle using a commercially available cardiac stimulator triggering from a surface ECG.

*Importance of timing of impact.* We found the electrophysiologic consequences of chest wall blows to be largely dependent on the timing of the cardiac cycle in which the impact occurred (Table 1). In these studies, impacts during the vulnerable portion of the T-wave (10–30 ms prior to the T-peak) triggered VF instantaneously and reproducibly (Fig. 1) (Link et al., 1998a, 1999b). In 30 mph impacts during this vulnerable period, approximately 30% of the impacts resulted in VF while another 10% caused nonsustained polymorphic ventricular tachycardia.

Blows during other portions of the cardiac cycle did not produce VF, but could cause ST-segment elevations and transient complete heart block. Heart block was never permanent, and usually lasted <5 beats.

*Morphologic changes.* In early studies utilizing immediate echocardiography and sestamibi injection, segmental, transient wall motion abnormalities were observed in the apex of the heart, a region distant from the area of precordial impact. Immediate (within 60 s) coronary angiography did not reveal any epicardial coronary artery abnormalities (Link et al., 1998a). Myocardial bruises were rarely seen with impacts <50 mph, but increased with higher velocity impacts to nearly 100% of those impacts at 70 mph. Similarly, structural cardiac abnormalities that could cause arrhythmias or death were not observed with impacts <50 mph.

Table 1

Incidence of ventricular fibrillation (VF), nonsustained ventricular fibrillation (NSVF) (excluding impacts resulting in ventricular fibrillation), and transient heart block (HB) (excluding impacts resulting in ventricular fibrillation), with 30 and 40 mph impacts grouped according to the timing of impact in our experimental model

	QRS	ST	–40 to –31 ms to T-peak	–30 to –21 ms to T-peak	–20 to –10 ms to T-peak	–9 to –1 ms to T-peak	T downslope
Total impacts (no)	58	93	74	516	384	31	47
VF induced	0	0	12	148	113	1	0
% VF	<b>0%</b>	<b>0%</b>	<b>16%</b>	<b>29%</b>	<b>29%</b>	<b>3%</b>	<b>0%</b>
Total impacts (no)	58	93	62	368	271	30	47
NSVF induced	1	1	4	39	28	0	0
% NSVF	<b>2%</b>	<b>1%</b>	<b>6%</b>	<b>11%</b>	<b>10%</b>	<b>0%</b>	<b>0%</b>
Total impacts (no)	58	93	62	368	271	30	47
HB induced	9	10	6	14	11	3	3
% HB	<b>16%</b>	<b>11%</b>	<b>10%</b>	<b>4%</b>	<b>4%</b>	<b>10%</b>	<b>6%</b>

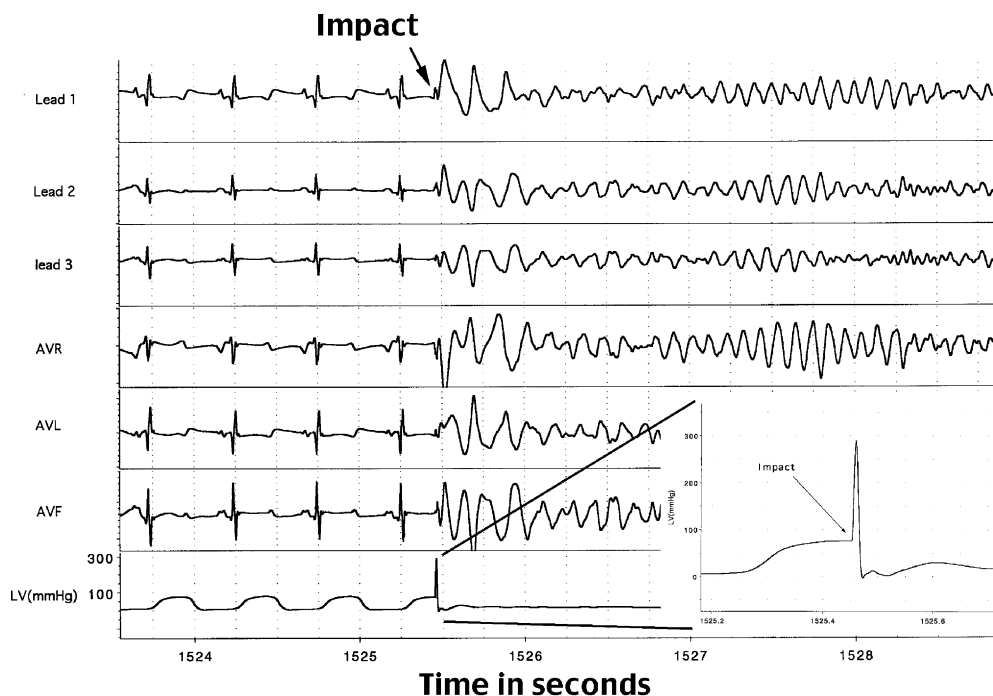


Fig. 1. Six lead electrocardiogram from a 11 kg swine undergoing chest wall impact with a 30 mph object the shape and weight of a standard baseball. Following chest impact within the vulnerable zone of repolarization (10–30 ms prior to the T-peak) ventricular fibrillation is immediately produced. From Link et al. (2001) with permission from The Journal of the American College of Cardiology.

*Hardness of impact object.* The incidence of VF directly correlated with the hardness of the impact object (Fig. 2), with firmer objects more likely to trigger VF (Link et al., 1998a, b). Impact object hardness correlated with the induction of VF not only at 30 mph, but also at 40 mph, a velocity more relevant to that causing commotio cordis in youth baseball (Link et al., 2002).

*Location of impact.* VF was most commonly observed with impacts directly over the center of the cardiac silhouette (30%) compared to those over the base of left ventricle (13%) or over the apex (4%) (Fig. 3) (Link et al., 2001). Impacts at sites that did not overly the cardiac silhouette did not cause VF.

*Speed of impact.* In experiments with the velocity of baseball impact varying from 20 to 70 mph, blows at 20 mph did not cause VF (Link et al., 2003b). As impact velocity increases, the risk of VF rises, to nearly 70% at 40 mph. At velocities  $\geq 50$  mph, however, the likelihood of VF decreases, and cardiac structural damage more commonly occurred (left ventricular rupture and papillary muscle tears), suggesting that at these velocities the electrophysiologic abnormalities can be due to structural damage (contusio cordis). The mechanism for decreased vulnerability of commotio cordis at higher impact velocities is unresolved, but may relate to the critical mass of myocardial tissue needed to sustain reentrant arrhythmias or to critical left ventricular pressure changes produced by the chest wall blow.

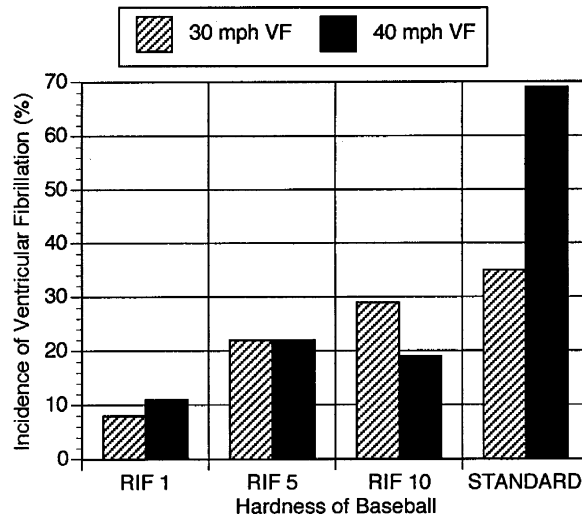


Fig. 2. Incidence of ventricular fibrillation with chest wall impacts at 30 and 40mph with a regulation baseball compared to softer-than-standard (safety) baseballs of three different grades of hardness. From Link et al. (2002); with permission from Pediatrics.

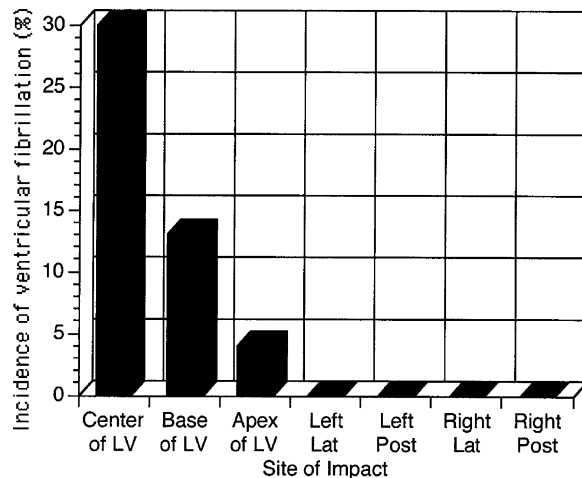


Fig. 3. Bar graph demonstrating the occurrence of ventricular fibrillation (VF) with 30-mph baseball impacts with respect to different sites on the chest wall in our experimental swine model of commotio cordis. Ventricular fibrillation was produced only by impacts directly over the cardiac silhouette with the highest incidence evident at the center of the left ventricle (LV). Ventricular fibrillation did not occur with blows to the right and left lateral (Lat) or the right and left posterior (Post) chest wall sites. From Link et al. (2001) with permission from The Journal of the American College of Cardiology.

## 6. Mechanisms of commotio cordis

Based on the findings in humans, as well as the experimental data described above, VF initiated by chest wall blows appears to be the cause of death in commotio cordis. Since human victims

collapse either virtually instantaneously or immediately after impact and the VF produced in our model is also instantaneous, commotio cordis is surely a primary electrical event rather than secondary to heart block, or myocardial ischemia or hemorrhage.

We suspect that the initiation of VF is multifactorial and likely requires at least two necessary features, one involving a premature ventricular depolarization (trigger) and another involving an altered myocardial substrate, both of which are produced by the chest wall blow. Although the initiation of VF is always preceded by depolarization of the ventricle, depolarization of a single beat is not sufficient to cause a reentrant arrhythmia. Indeed, in the experiments of impact velocity, 287 impacts did not cause VF. Yet, premature ventricular depolarizations were observed in 199 (69%) of these impacts including 36 of 80 (45%) at 20 mph, 63 of 86 (73%) at 25 mph, 51 of 68 (75%) at 30 mph, 16 of 17 (94%) at 40 mph and all impacts  $\geq 50$  mph.

The other necessary alteration is likely the activation of specific ionic channels in the myocardial substrate. There is a correlate to this hypothesis in our experimental model in the R on T phenomenon. In this phenomenon, premature ventricular contractions fall on the vulnerable portion of the T-wave and produce VF. However, R on T producing VF is present primarily in ischemic myocardial conditions (El-Sherif et al., 1976; Naito et al., 1982). In the setting of an acute myocardial infarction or coronary ischemia an appropriately timed premature depolarization can cause VF. However, in nonischemic situations (such as continuous ventricular pacing, VOO) premature depolarizations during the vulnerable portion of the T-wave do not generally cause VF.

*Autonomic nervous system:* One of the proposed mechanisms of commotio cordis includes hypervagatonia or activation of the sympathetic nervous system. In our model, there was no difference in the incidence of VF (40% vs. 35%, respectively), nonsustained polymorphic ventricular tachycardia (13% vs. 23%, respectively), ST segment elevation or bundle branch block between animals given control agent or complete sympathetic and parasympathetic blockade (0.4 mg IV atropine and 2 mg IV propranolol) (Link et al., 2000).

*$K_{ATP}^+$  channel.* Because of the electrical similarities between commotio cordis and myocardial ischemia, including ST segment elevation and R on T causing VF, we hypothesized that the specific ion channel activation's may be comparable. A likely candidate is the  $K_{ATP}^+$  channel, given that it is primarily responsible for the ST-segment elevation and contributes to the risk of VF in the presence of myocardial ischemia (Kubota et al., 1993; Kondo et al., 1996). Indeed, in our experimental model, we found that glibenclamide, a specific blocker of the  $K_{ATP}^+$  channel reduced the magnitude of ST segment elevation and the incidence of VF following experimental chest blows (Fig. 4) (Link et al., 1999a), suggesting that activation of this channel by chest wall impact is critical in the initiation and maintenance of VF. However, despite the reduction in VF, premature ventricular contractions caused by the chest wall blow were not eliminated (23 of 27 impacts, 85% in glibenclamide animals vs. 15 of 18, 83% in control) again suggesting that a single ventricular depolarization is not sufficient to cause VF.

*Left ventricular pressure rise.* Data from our experimental model also suggest that instantaneous left ventricular pressure rise produced by the chest blow may mediate the electrophysiologic consequences of commotio cordis. In experiments designed to define both the site of impact (Link et al., 2001), and the velocity of impact (Link et al., 2003b), the left ventricular pressure rise created by the chest wall blow correlated with the risk of VF. In both of these studies the peak probability of VF was seen with peak left ventricular pressures between 250 and 450 mmHg



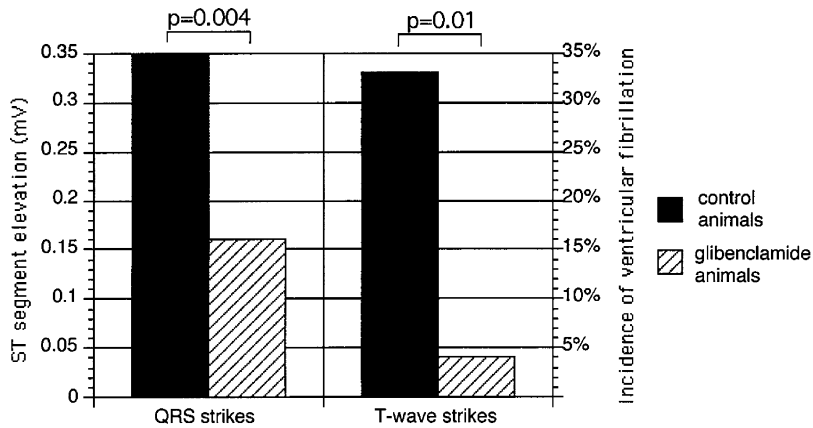


Fig. 4. Magnitude of ST segment elevation and the incidence of ventricular fibrillation induced by 30 mph chest wall impacts by a spherical object the size and weight of a regulation baseball in experimental model of commotio cordis. Significant differences between the control animals and the animals given glibenclamide (a selective blocker of the  $K_{ATP}^+$  channel) were observed with respect to the magnitude of ST segment elevation with QRS strikes and the incidence of ventricular fibrillation with T-wave strikes. From Link et al. (1999b) with permission from Circulation.

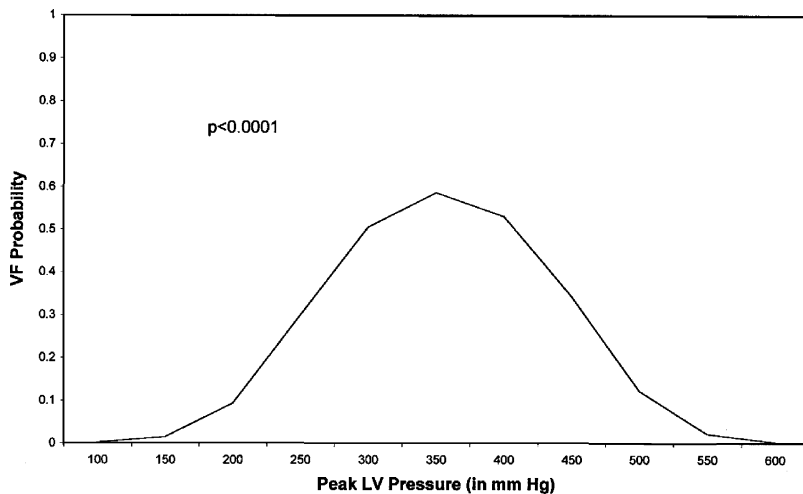


Fig. 5. Probability of ventricular fibrillation relative to the peak left ventricular pressure produced by a 30 mph baseball in our sites of impact experiment. From Link et al. (2003b) with permission of The Journal of the American College of Cardiology.

(Fig. 5). These observations suggest that it may be the rapid pressure rise immediately following the chest blow that causes the VF, possibly mediated by stretch activated channels. The  $K_{ATP}^+$  channel has been shown to be activated by myocardial stretch in a rat atrial model (Van Wagoner, 1993), and our data of the importance of the  $K_{ATP}^+$  channel in the initiation of VF is consistent with stretch activation of this channel.

Other channels (Hu and Sachs, 1997; Kohl et al., 1999) may be activated by myocardial stretch, and in this way underlie the genesis of ventricular arrhythmias following chest blows. The

stretch-activated-channel (SAC), a nonselective cation channel activated by myocardial stretch is a likely candidate for the initiation of VF with chest wall blows; however, preliminary data in our model do not confirm the importance of this channel in commotio cordis.

## 7. Treatment of commotio cordis

Of the human commotio cordis victims only about 15% survive (Maron et al., 2002). In the Commotio Cordis Registry the major determinant of survival was early defibrillation. In our commotio cordis model, early defibrillation was also found to be critical for survival. After 1 and 2 min of VF, 96% of animals survived after defibrillation. However, after 4 min of VF, only 46% survived, and at 6 min just 25% ( $p < 0.0001$ ) (Link et al., 2003a).

Currently the automated external defibrillator (AED) is only approved for use in individuals  $\geq 8$  years old because of concerns for appropriate arrhythmia recognition and potential adverse consequences emanating from adult defibrillation energy levels. In our model, the sensitivity of the AED for the recognition of VF was 98%, and specificity for nonshockable rhythms was 100% (Link et al., 2003a).

## 8. Prevention of commotio cordis

Softer-than-standard or safety baseballs have been proposed to reduce the risk of injury and death in baseball. Based on experimental data, these safety baseballs appear to decrease the risk of commotio cordis. In a laboratory utilizing 95 mph baseball impact with swine, minor reductions in fatalities were reported when a softer-than-standard baseball was used in combination with commercially available chest wall protectors (Janda et al., 1992). In a more recent publication by this same group evaluating the risk of cardiac injury with 40–60 mph impacts in a dummy model, 78% of the safety balls tested had significantly lower impact force than the standard baseball at least one of the velocities tested (Janda et al., 1998).

In our experimental model, a statistically significant reduction in the risk of fatal ventricular arrhythmias was observed with safety baseballs at both 30 and 40 mph (Fig. 5) (Link et al., 1998a, 2002). The softest ball (Reduced injury factor (RIF) 1<sup>®</sup>, Worth Inc., Tullahoma, Tennessee), marketed for T-ball use in youths aged 5–7, had an incidence of VF of 8% at both 30 and 40 mph. The incidence of VF increased linearly with the hardness of the ball and reached a peak of almost 70% with standard baseballs propelled at 40 mph.

In a 3 rib dummy model, only 20% of commercially available chest protectors tested were found to statistically lower the impact force with baseballs at 40 mph; at velocities of 50–70 mph, 80% of the chest wall protectors decreased the impact force (Viano et al., 2000).

## 9. Conclusions

Commotio cordis is an unusual, but devastating event occurring in young people during sports-related or routine daily activities. Its prevalence is likely underestimated, and self-limited cases due

to nonsustained arrhythmias may be more common than is commonly appreciated. Critical variables that determine the likelihood of VF occurring from a chest blow include the precise timing of the impact to a 20 ms window on the upslope of the T-wave, greater hardness of the impact object, precordial impact location of the blow, and the speed of the impact. The initiation of VF is related to the left ventricular pressure rise produced by the chest blow and may be a prime mediator of the arrhythmia. Activation of specific cardiac ion channels, especially the  $K_{ATP}^+$  channel, likely occurs with chest wall impact and produces the VF and ST segment elevation seen in this disorder. The experimental model described herein simulates the human scenario of commotio cordis, offers specific insights into its mechanism and helps to explain why commotio cordis is a relatively rare event.

## 10. Editor's note

Please see also related communications in this volume by [Janse et al. \(2003\)](#) and [Taggart et al. \(2003\)](#).

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