

# Impact Directly Over the Cardiac Silhouette Is Necessary to Produce Ventricular Fibrillation in an Experimental Model of *Commotio Cordis*

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<b>OBJECTIVES</b>	In an experimental model of sudden death from chest wall impact ( <i>commotio cordis</i> ), we sought to define the chest wall areas important in the initiation of ventricular fibrillation (VF).
<b>BACKGROUND</b>	Sudden death can result from an innocent chest blow by a baseball or other projectile. Observations in humans suggest that these lethal blows occur over the precordium. However, the precise location of impact relative to the risk of sudden death is unknown.
<b>METHODS</b>	Fifteen swine received 178 chest impacts with a regulation baseball delivered at 30 mph at three sites over the cardiac silhouette (i.e., directly over the center, base or apex of the left ventricle [LV]) and four noncardiac sites on the left and right chest wall. Chest blows were gated to the vulnerable portion of the cardiac cycle for the induction of VF.
<b>RESULTS</b>	Only chest impacts directly over the heart triggered VF (12 of 78: 15% vs. 0 of 100 for noncardiac sites: $p < 0.0001$ ). Blows over the center of the heart (7 of 23; 30%) were more likely to initiate VF than impacts at other precordial sites (5 of 55; 9%, $p = 0.02$ ). Peak LV pressures generated instantaneously by the chest impact were directly related to the risk of VF ( $p < 0.0006$ ).
<b>CONCLUSIONS</b>	For nonpenetrating, low-energy chest blows to cause sudden death, impact must occur directly over the heart. Initiation of VF may be mediated by an abrupt and substantial increase in intracardiac pressure. Prevention of sudden death from chest blows during sports requires that protective equipment be designed to cover all portions of the chest wall that overlie the heart, even during body movements and positional changes that may occur with athletic activities. ( <i>J Am Coll Cardiol</i> 2001;37:649–54) © 2001 by the American College of Cardiology

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*Commotio cordis*, or sudden death due to relatively low-energy chest wall impact without structural injury to the sternum, ribs or the heart itself, has been reported with increasing frequency in young individuals participating in sporting activities (1–4). These tragic events predominantly occur in youths age 4 to 16 years. While most commonly due to chest blows from baseballs, *commotio cordis* has also been described in hockey, softball, lacrosse, karate, fist fights or in any situation in which a relatively hard projectile or body part strikes the unprotected chest wall. In a series of 70 victims of *commotio cordis*, all impacts appeared to be over or to the left of the sternum (1,3) although the exact impact site was difficult to assess with precision. Victims' collapse immediately after the blow, and the most common initial recorded rhythm is ventricular fibrillation (VF) (1,5–10). Some form of chest wall protection had been present in a minority of the victims; however, there is evidence in several cases that critical areas of the precordium nevertheless became exposed and struck during sporting play (1,3).

In an experimental model of *commotio cordis* employing a baseball propelled at 30 mph, we have demonstrated the importance that the precise timing of the blow plays in the initiation of VF (11) in which impact is required during a narrow window of repolarization (10 to 30 ms before the T wave peak). However, the precise areas of the chest wall most vulnerable to sudden death from impact, as well as the hemodynamic and cellular determinants of the VF, are not completely understood. Such information is crucial to the optimal design of protective devices for young people in organized sports and the prevention of sudden death from chest wall blows.

## METHODS

**Animals.** Juvenile domesticated swine, four to eight weeks old and weighing 8 to 12 kg, were used in this study. The research protocol was approved by the Animal Research Committee of the New England Medical Center in conformity with the regulations of the Association for Assessment and Accreditation of Laboratory Animal Care. Animals were anesthetized with ketamine and isoflurane and intubated; anesthesia was maintained with 1% to 2% isoflurane mixed with oxygen and nitrous oxide. Millar Mikrotip (Houston, Texas) pressure catheters were placed in the left ventricle (LV), and a transesophageal echocardiographic

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**Abbreviations and Acronyms**

ECG	= electrocardiogram
LV	= left ventricle, left ventricular
VF	= ventricular fibrillation

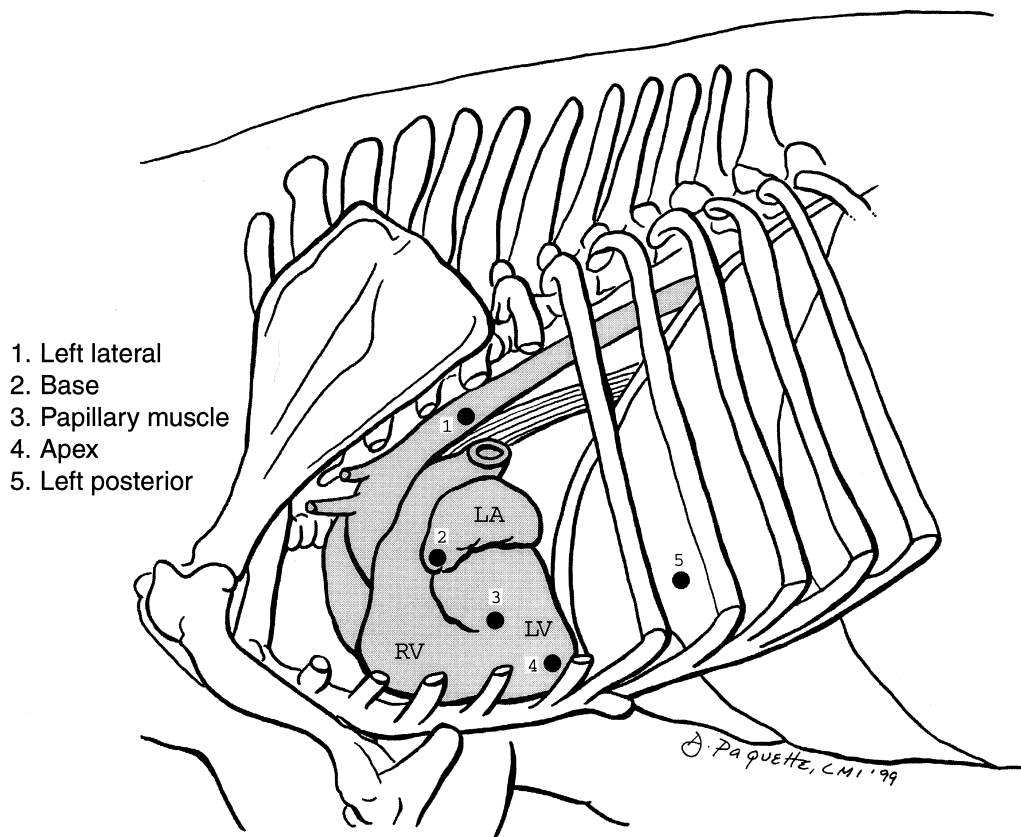
probe was placed. A standard 6-lead electrocardiogram (ECG) was attached, and the animals were placed prone in a sling (11,12). Transthoracic echocardiography was used to guide the chest blows, and distances from the impact site to the center of the LV were measured.

**Experimental protocol.** Impacts were delivered to seven chest wall sites, including three directly over the heart, two additional sites on the left chest wall removed from the area of the heart and two sites on the right chest wall (Fig. 1). Impacts occurred first on the left chest wall; the order of impacts was randomly assigned to the five left chest wall sites in each animal. In previous experiments with our swine model of commotio cordis, we chose the center of the LV (i.e., over the anterolateral papillary muscle) as the site of impact (11,12). In the current investigation, in addition to impact over the center of the LV (the anterolateral papillary muscle was used as the anatomic reference point), other left chest

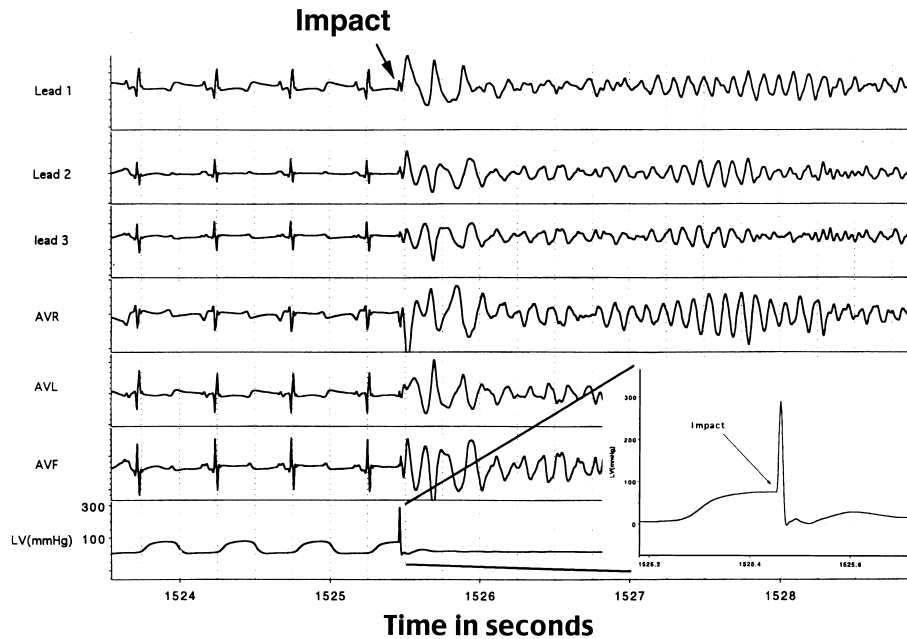
wall impact sites were: over the base of the LV ( $28 \pm 8$  mm from the papillary muscle), over the LV apex ( $26 \pm 7$  mm from the papillary muscle), the high left lateral chest wall ( $53 \pm 11$  mm dorsal from the papillary muscle), left posterior chest wall ( $70 \pm 11$  mm caudal to the papillary muscle) and the right lateral and right posterior chest wall.

Each chest wall site received two impacts (separated by at least 2 min) with a regulation baseball (propelled at 30 mph) during the known vulnerable period of the cardiac cycle for the induction of VF, 10 to 30 ms before the peak of the T wave (11,12). Impacts occurring outside of this time window were excluded from this analysis. If VF resulted from a chest blow, the animal was immediately defibrillated. The experiment was terminated prematurely if the ECG pattern and LV wall motion did not return to normal after defibrillation (as occurred in 2 of 15 animals).

Impact was created by the delivery of a baseball mounted on an aluminum shaft propelled by a spring (11,12). The baseball was designed to strike the swine perpendicular to the chest wall at a speed of 30 mph, as measured by a chronograph (Oehler Research, Austin, Texas) modified for low velocity. All chest strikes were delivered at a distance of one meter from the point of



**Figure 1.** Impact sites used in this experimental model of commotio cordis. All chest blows were delivered with a regulation baseball at 30 mph. The LV anterolateral papillary muscle and base and apex of the LV were defined by direct anatomic visualization with transthoracic echocardiography. Distances to the other chest wall sites (unrelated to the heart) were defined using the papillary muscle as a reference point. Not shown here are the right chest wall sites. LA = left atrium; LV = left ventricle; RV = right ventricle.



**Figure 2.** Six-lead electrocardiogram demonstrating VF produced by a 30-mph baseball impact to the chest wall directly over the anterolateral papillary muscle, timed to the upstroke of the T wave in an 11-kg juvenile swine. Ventricular fibrillation occurs immediately and is not preceded by ischemic electrocardiographic changes, ventricular tachycardia or heart block. **Inset (lower right).** Magnification of the LV pressure tracing obtained at the precise moment of impact; note the marked and immediate pressure rise within the LV. Left ventricular pressure increases abruptly to 290 mm Hg over a time period of 8 ms and falls to 0 mm Hg 12 ms later. LV = left ventricular; VF = ventricular fibrillation.

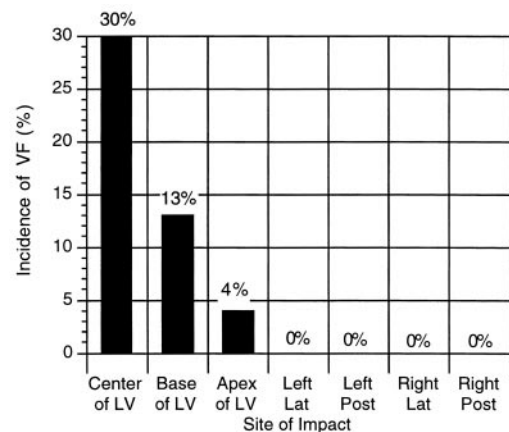
release of the ball, allowing a constant and reproducible velocity of impact.

**Data acquisition and analysis.** Continuous six-lead ECGs were collected using MacLab Chart software and ADInstruments (Mountain View, California) analogue to digital converter. Left ventricular pressure monitoring was performed with Millar catheters and converted from analogue to digital with the ADInstrument converter. Electrocardiograms and pressure recordings were sampled at 1,000 Hz and recorded on a computer (MacIntosh G3). Ventricular fibrillation was defined as a rapid polymorphic ventricular arrhythmia that required defibrillation, while nonsustained polymorphic ventricular tachycardia was defined as a rapid polymorphic ventricular arrhythmia that spontaneously terminated. Criteria for ST segment elevation were  $\geq 1$ -mm elevation after impact. Criteria for bundle branch block included the characteristic bundle branch block morphology and doubling of QRS duration after impact. Transesophageal echocardiograms were recorded in real time and later analyzed for wall motion abnormalities and LV function by an observer blinded to the site of impact.

**Statistical analysis.** Continuous data were analyzed with the Student *t* test; categorical data were assessed with the chi-square or Fisher exact test. Univariate logistic regression was performed for the comparison of LV pressure and risk of VF. A *p* value of  $\leq 0.05$  was considered statistically significant. Data are presented as mean  $\pm$  standard deviation.

## RESULTS

**Electrophysiological results.** Of 178 chest impacts in 15 animals, 78 were delivered over the cardiac silhouette and 100 to portions of the chest wall not directly overlying the heart. Ventricular fibrillation was triggered only by impacts directly over the cardiac silhouette (12 of 78; 15% compared with 0 of 100 for noncardiac sites;  $p < 0.0001$ ) (Fig. 2 and 3, Table 1). Furthermore, impacts over the center of the LV



**Figure 3.** Bar graph demonstrating the occurrence of VF as a result of 30-mph baseball impacts with respect to different sites on the chest wall of swine in our experimental 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 LV. Ventricular fibrillation was not produced with impacts at the right and left lateral (Lat) or the right and left posterior (Post) chest wall sites. LV = left ventricle; VF = ventricular fibrillation.

**Table 1.** Risk of Inducing Electrophysiologic Abnormalities With a Baseball Propelled at 30 mph and Impacting the Chest Wall at Different Anatomic Sites During the Vulnerable Time Period for Induction of Ventricular Fibrillation (10 to 30 ms Before the T wave Peak)

	Sites of Chest Impacts						
	Cardiac			Noncardiac			
	Center of LV	Base of LV	Apex of LV	Left Lateral Chest	Left Posterior Chest	Right Lateral Chest	Right Posterior Chest
Induction of VF	7/23 (30%)*	4/30 (13%)	1/25 (4%)	0/26	0/25	0/25	0/24
Induction of PMVT†	3/16 (19%)	2/26 (8%)	1/24 (4%)	0/26	0/25	0/25	0/24
ST elevation†	7/16 (44%)	5/26 (19%)	5/24 (21%)	0/26	0/25	0/25	0/24
Transient HB†	1/16 (6%)	2/26 (8%)	1/24 (4%)	0/27	0/25	0/25	0/24
LBBB†	8/16 (50%)	5/26 (19%)	5/24 (21%)	0/26	0/25	0/25	0/24
Peak LV pressure (in mm Hg)‡	280 ± 36*	258 ± 60 $\alpha$	224 ± 48 $\beta$	160 ± 47	118 ± 21	110 ± 23	98 ± 16

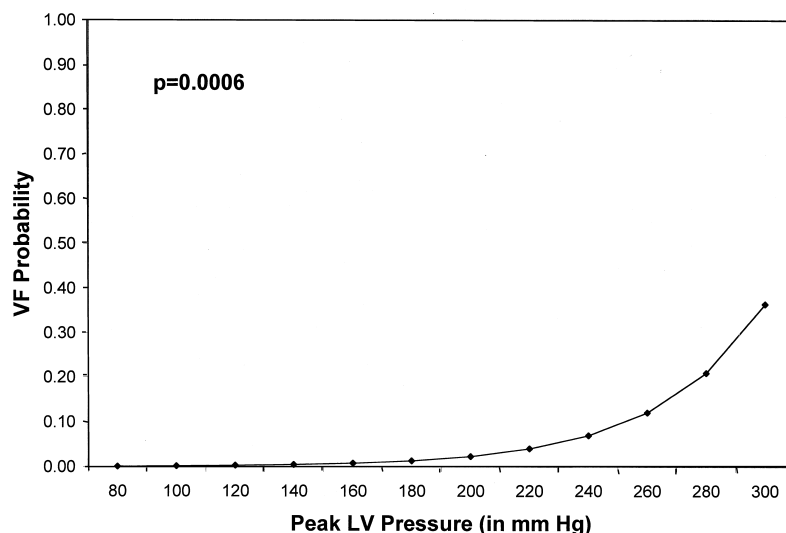
\*Significantly different than all impact sites except for the base of the LV; †Expressed only for those impacts that did not produce VF; ‡Measured immediately after chest impact.  $\alpha$ , significantly different than impacts at all other sites except for the center of the LV;  $\beta$ , significantly different than impacts at all sites.  
 HB = heart block; LBBB = left bundle branch block; LV = left ventricle; PMVT = nonsustained polymorphic ventricular tachycardia; VF = ventricular fibrillation.

were more likely to produce VF (7 of 23; 30%) than impacts to other precordial sites (5 of 55; 9%,  $p = 0.02$ ), including the apex of the LV (1 of 25; 4%,  $p = 0.02$ ) and the base of the LV (4 of 30; 13%,  $p = 0.17$ ). None of the blows to noncardiac sites including the left lateral ( $n = 26$ ), left posterior ( $n = 25$ ), right posterior ( $n = 24$ ) and right lateral chest wall ( $n = 25$ ) generated VF. In addition, nonsustained polymorphic ventricular tachycardia (mean of  $3 \pm 1$  beats) occurred only with impacts over the cardiac silhouette: 3 of 16 at the center (19%), 2 of 26 at the base (8%) and 1 of 24 at the apex of the LV (4%).

Of those 166 impacts that did not cause VF, ST segment elevation ( $\geq 1$  mm) occurred in 17 (10%), all with blows directly over the cardiac silhouette ( $p < 0.0001$  when

compared with nonprecordial impact sites) (Table 1). Transient heart block (mean duration of 2 s) was observed with 4 of the 166 impacts not causing VF and only with impacts over the heart ( $p = 0.02$  when compared with noncardiac impact sites) (Table 1). Left bundle branch blocks occurred in 20 of 166 of the impacts not causing VF and were also limited to impacts over the cardiac silhouette ( $p < 0.0001$  when compared with noncardiac impact sites) (Table 1).

**LV pressure.** Peak LV pressures became markedly elevated as an immediate consequence of chest impacts directly over the center ( $280 \pm 36$  mm Hg), base ( $258 \pm 60$  mm Hg) and apex ( $224 \pm 48$  mm Hg) of the LV (Fig. 2). A univariate logistic regression model revealed these peak pressures to be highly predictive of the initiation of VF ( $p = 0.0006$ , odds



**Figure 4.** Logistic regression plot showing the significant correlation between the risk of VF induction with baseball impact to the chest wall at 30 mph and the peak LV pressure resulting immediately after the blow. LV = left ventricular; VF = ventricular fibrillation.

ratio = 1.028 per mm Hg increase in pressure) (Fig. 4). In noncardiac sites on the chest wall, the peak LV pressures were significantly less than they were in cardiac sites in accord with increasing distance from the anterolateral papillary muscle (left lateral =  $160 \pm 47$  mm Hg; left posterior =  $118 \pm 21$  mm Hg; right lateral =  $110 \pm 23$  mm Hg and right posterior =  $98 \pm 16$  mm Hg) (all  $p$  values  $<0.001$  when compared with peak LV pressures with impacts over the cardiac silhouette).

A univariate logistic regression model also revealed that temporal change in LV pressure ( $dP/dT$  in mm Hg/ms) occurring as a result of a chest wall blow to be highly predictive of the initiation of VF ( $p = 0.0005$ , odds ratio = 1.149 per mm Hg/ms increase in temporal change).  $DP/dT$  was highest at the center of the LV ( $38.3 \pm 7.0$  mm Hg/ms) and decreased relative to the distance of the impact from the center of the LV (base,  $36.0 \pm 11.5$  mm Hg/ms; apex,  $27.3 \pm 9.6$  mm Hg/ms; left lateral,  $13.9 \pm 8.3$  mm Hg/ms; left posterior,  $6.1 \pm 3.1$  mm Hg/ms; right lateral,  $6.4 \pm 3.1$  mm Hg/ms and right posterior,  $3.4 \pm 1.7$  mm Hg/ms).

**LV wall motion abnormalities.** Left ventricular wall motion abnormalities resulted only from blows directly over the cardiac silhouette ( $p = 0.0008$ ). Eight impacts caused a transient ( $< 2$  min) decrease in global LV ejection fraction (mean  $8.3 \pm 5\%$ ), including 5 of 25 apical, 2 of 30 basal and 1 of 23 papillary muscles impacts. Segmental wall motion abnormalities were observed with seven impacts and involved apical hypokinesia in each and hypokinesia of the anterior wall in two, lateral wall in three and septum in three. None of the blows to the right or left posterior and lateral chest walls was associated with alterations in LV function.

## DISCUSSION

**Importance of chest impact site.** In the experimental model of sudden death due to low-energy, nonpenetrating chest wall blows (commotio cordis) reported here, VF was limited to chest wall impacts directly over the anatomic position of the heart. Furthermore, blows directly over the center of the heart (i.e., the LV anterolateral papillary muscle) were more frequently lethal than were those at the periphery of the heart. Of note, chest wall impacts that did not directly overlie the heart produced neither VF nor other electrophysiological abnormalities, such as nonsustained polymorphic ventricular tachycardia, transient heart block, bundle branch block, ST segment elevation nor wall motion abnormalities identified by echocardiography.

**Implications for chest wall safety equipment.** Our experimental data regarding the importance of the impact site for a commotio cordis event will be crucial for the appropriate design of safety equipment for youth sports. Protective gear that portends to provide protection against sudden death due to chest wall impact during sports activities must cover the entire cardiac silhouette regardless of body movement or position. Indeed, in some hockey-related commotio cordis

fatalities, there has been evidence that the shoulder-chest protector became displaced just before the chest blow, allowing the puck to have direct access to the unprotected precordium (1). Based on the experimental data presented here, as well as clinical observations of commotio cordis (1), chest wall protection need not extend to the right chest wall, the back or to other areas of the chest distant from the heart.

**Potential mechanisms of commotio cordis.** Previous experiments with our commotio cordis model have shown that the critical variables that must conspire for the initiation of VF with chest impact include the timing of impact (11) and the hardness of the impact object (11). Therefore, the present experiment expands on those prior observations by demonstrating that the anatomic location of the chest wall impact must be rather precise in order for the blow to produce VF. It is likely that the number of variables required to produce commotio cordis largely explains the low incidence with which such sudden deaths appear to occur.

In addition to the practical implications of the data reported here for prevention of sudden death associated with commotio cordis, the present experiment also provides potentially important information regarding the mechanism by which chest wall blows produce VF. In our animal model, with blows over the heart, peak LV pressures increased instantaneously, and the magnitude and rate of this rise correlated with the risk for VF. This association of VF and acute LV pressure rise suggests that the marked and immediate increase in LV pressure may play a key role in the genesis of VF, possibly by activating ion channels of the myocardial cell membrane (13-17). Stretch-sensitive ion channels, such as  $K^+_{ATP}$  (13,18),  $K^+_{ACh}$  (19) calcium (20,21) and other potassium, sodium and calcium channels (15,22) have been described and could be involved in commotio cordis. In our experimental model we have previously reported the importance of  $K^+_{ATP}$  channel activation in the occurrence of experimental commotio cordis events (12). The relationship of the  $K^+_{ATP}$  channel to other channels in experimental and clinical commotio cordis is not known, nor is it confirmed that the  $K^+_{ATP}$  channel is activated by stretch in our model. The exact role of LV pressure rise, ion channel activation and other possible variables in the initiation of VF after chest wall blows will need to be established in future experiments.

**Justification for the swine model.** Although certain differences in cardiac physiology and chest anatomy exist between humans and swine, we do not believe these issues have a measurable effect on either our animal model of commotio cordis or the conclusions drawn from the present experiment. For example, it has been suggested that swine may be more susceptible to VF under ischemic conditions (23), but we have found little evidence of ischemia in our experimental commotio cordis model (11,12). In addition, the QTc may normally be longer in swine than it is in humans (24,25). However, we have previously shown that a critical factor governing the initiation of VF in experimentally produced commotio cordis is the timing of the blow to

a narrow time window of vulnerability on the upslope of the T wave, a variable independent of the absolute length of the QT interval (whether or not corrected for heart rate) (11). Furthermore, the use of swine to study the mechanisms of VF and resuscitation is well established (23,26,27).

Although differences in the geometry of the human and porcine thoracic cages could conceivably affect the transmission of impact energy to the myocardium, nevertheless, in both circumstances chest blows occur perpendicular to the chest wall, directly over the heart and at a point where the heart is in close proximity to the chest wall without intervening pulmonary tissue. Therefore, we would expect the kinetic energy transmitted by nonpenetrating chest blows to the heart to be similar in the precipitation of commotio cordis events in both humans and our experimental animal model.

**Conclusions.** The precise location of chest wall impact is a critical determinant of commotio cordis events. To result in VF, the chest blow must occur directly over the anatomic position of the heart and, in fact, is more likely to be lethal when impact is at the center of the cardiac silhouette. Incorporation of these observations into the design of chest wall protectors may prevent sudden deaths and make the athletic field safer for youthful participants in sports activities.

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