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Upper and Lower Limits of Vulnerability to Sudden Arrhythmic Death With Chest-Wall Impact (Commotio Cordis)

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OBJECTIVES In an animal model of commotio cordis, sudden death with chest-wall impact, we sought to systematically evaluate the importance of impact velocity in the generation of ventricular fibrillation (VF) with baseball chest-wall impact.

BACKGROUND Sudden cardiac death can occur with chest-wall blows in recreational and competitive sports (commotio cordis). Analyses of clinical events suggest that the energy of impact is often not of unusual force, although this has been difficult to quantify.

METHODS Juvenile swine (8 to 25 kg) were anesthetized, placed prone in a sling to receive chest-wall strikes during the vulnerable time window during repolarization for initiation of VF with a baseball propelled at 20 to 70 mph.

RESULTS Impacts at 20 mph did not induce VF; incidence of VF increased incrementally from 7% with 25 mph impacts, to 68% with chest impact at 40 mph, and then diminished at ≥50 mph ($p < 0.0001$). Peak left ventricular pressure generated by the chest blow was related to the incidence of VF in a similar Gaussian relationship ($p < 0.0001$).

CONCLUSIONS The energy of impact is an important variable in the generation of VF with chest-wall impacts. Impacts at 40 mph were more likely to produce VF than impacts with greater or lesser velocities, suggesting that the predilection for commotio cordis is related in a complex manner to the precise velocity of chest-wall impact. (J Am Coll Cardiol 2003;41:99–104)

Sudden death caused by blows to the chest with baseballs and other objects (commotio cordis) has been reported with increasing frequency, and may account for a significant proportion of sudden cardiac deaths occurring in young athletes (1–3). While the velocity and energy of impact causing these tragedies have not been assessed in precise terms, they appear to comprise a wide range, and often are not unusual for the sport and age group involved (1–3).

We have recently developed an experimental model of commotio cordis in which chest blows to juvenile swine by baseballs propelled at 30 mph under controlled conditions immediately triggered ventricular fibrillation (VF) when the impact was timed to a narrow vulnerable 20-ms window on the upstroke of the T-wave (4–6).

Because the limits of vulnerability for electrically induced VF are known to include not only the timing of the shock but also the energy of the shock (7–14), we hypothesized that, in commotio cordis, impact energy also may be an important variable in generating VF.

METHODS

Animal model. A total of 116 domesticated swine four to eight weeks old weighing between 8 and 25 kg were used in our study. The research protocol was approved by the Animal Research Committee of the New England Medical Center in accordance with the regulations of the Association for Assessment and Accreditation of Laboratory Animal Care. Animals were sedated with 12 mg/kg intramuscular ketamine and then anesthetized with inhaled 1% to 2% isoflurane mixed with oxygen and nitrous oxide. Continuous six-lead coronal electrocardiograms (ECG) were acquired and filtered from 0.3 to 100 Hz. Left ventricular (LV) pressure catheters (Millar mikrotip, Houston, Texas) were placed and signals acquired with a low-pass filter at 50 Hz. Animals were then placed prone in a sling in order to approximate physiologic anatomy and function of the cardiovascular system.

The chest-wall impact was created by the delivery of a regulation baseball (Rawlings Little League, LLB-1, St. Louis, Missouri) mounted on an aluminum shaft. At the time of impact, the baseball and shaft were in free flight and, thus, mimicked the field conditions of chest impact with a baseball. The baseball was propelled by a mechanism that adjusted the velocity in a range of 20 to 70 mph. A chronograph (Oehler Research, Austin, Texas), modified for low velocity, assessed the speed of the baseball.

Baseballs, with guidance by transthoracic echocardiography, struck each swine at a right angle to the chest wall over the center of the LV (6). All chest impacts were timed to the

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Abbreviations and Acronyms
dP/dt = left ventricular pressure rise over time
ECG = electrocardiogram
LV = left ventricle or left ventricular
VF = ventricular fibrillation

vulnerable portion of the cardiac cycle for the induction of VF, on the upslope of the T wave (4,8). In prior experiments with 30-mph baseball impact, the time window of vulnerability for VF was limited to impacts between 10 and 30 ms before the T-wave peak. However, because of the possibility that the time window of vulnerability could be longer with higher velocity impacts, in this experiment we included chest blows over a wider time range, from 40 ms before the T peak to the T peak; 348 of 373 total blows (94%) fell within that window and, therefore, constitute the subsequent analyses (Table 1).

Experimental protocol. Two groups of juvenile swine with different body weight were studied: 1) 8 to 12 kg; mean, 10.2 ± 1.6 kg (n = 86); and 2) 18 to 25 kg; mean, 21.7 ± 1.7 kg (n = 30). Each of the smaller 8 to 12 kg animals received six impacts at either 20 mph or 25 mph, and three blows at 30 and 40 mph. To limit cardiac and thoracic damage, 8 to 12 kg animals received only one impact at each of the higher velocities, that is, 50, 60, and 70 mph. Three 8 to 12 kg animals received impacts at both 20 and 25 mph velocities, whereas the remaining 83 animals received impacts at a single velocity.

In the larger 18 to 25 kg swine, eight impacts were delivered to the animals at 20 or 25 mph, five impacts at 30 to 50 mph, three impacts at 60 mph, and one impact at 70 mph. Four animals (18 to 25 kg) received impacts at both 30 and 40 mph, whereas the remaining 26 animals received impacts at a single velocity.

Electrocardiograms (ECGs) were recorded, and LV ejection fractions assessed before and after each chest impact. If VF occurred, animals were immediately defibrillated. If permanent ECG or echocardiographic abnormalities were evident after chest impact or defibrillation, no further blows were delivered. Animals were euthanized with an intravenous potassium chloride solution. Gross autopsy examination was performed.

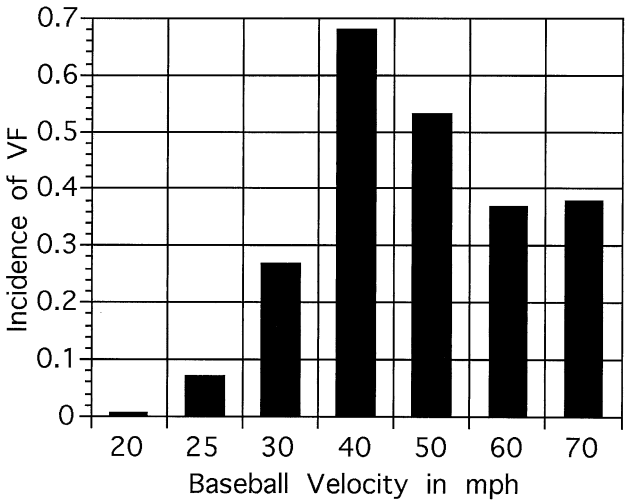

Figure 1. Incidence of ventricular fibrillation (VF) induced by chest-wall impact at the vulnerable period of repolarization, with a regulation baseball propelled at a range of velocities under controlled conditions (logistic regression model $p < 0.0001$ predicting VF from mph and mph^2).

Statistical analysis. Continuous data are reported as mean ± SD. Differences between the various impact velocities were analyzed by the Fisher exact test, linear regression for continuous outcome variables, and logistic regression for dichotomous outcome variables. P values ≤0.05 were regarded as statistically significant. Animals weighing 8 to 12 kg and 18 to 25 kg were analyzed separately, and the data were then combined for further analysis. Analysis was performed in SAS statistical software (version 8, Cary, North Carolina).

RESULTS

Energy limits of vulnerability to VF. Incidence of VF relative to the velocity of chest impact exhibited a Gaussian curve with the peak incidence of VF at 40 mph ($p < 0.0001$ by logistic regression) (Fig. 1, Tables 1 and 2). There were no significant differences in the incidence of induced VF between the smaller and larger animals at any impact velocity.

Table 1. Incidence of VF Relative to the Timing and Energy of Chest-Wall Impact*

	Timing of Chest Impact						
Impact Velocity (mph)	**QRS**	**ST**	**40 to 31 ms pre-T Peak**	**30 to 21 ms pre-T Peak**	**20 to 10 ms pre-T Peak**	**9 to 0 ms pre-T Peak**	**Post-T Peak**
70	0/1	0/0	0/0	2/7 (29%)	2/5 (40%)	0/1	0/0
60	0/0	0/1	0/2	4/9 (44%)	3/8 (38%)	0/0	0/0
50	0/1	0/0	1/3 (33%)	6/17 (35%)	9/10 (90%)	0/0	0/0
40	0/0	0/5	0/2	8/13 (62%)	18/23 (78%)	0/0	0/0
30	0/0	0/2	0/11	15/48 (31%)	10/31 (32%)	0/1	0/0
25	0/2	0/3	0/4	2/45 (4%)	4/36 (11%)	0/2	0/0
20	0/2	0/0	0/2	0/41	0/30	0/1	0/4

*Shown as number of occurrences of VF relative to the number of chest-wall blows delivered in each category. All impacts were directed to the vulnerable time window (40 ms before T peak to T peak) for triggering VF, and 348/373 (94%) fell within that time period.
VF = ventricular fibriliation.

Table 2. Consequences of Chest-Wall Impact at a Variety of Velocities in the Experimental Commotio Cordis Model

Impact Velocity* (mph)	No. of Animals	No. of Impacts	No. of Impacts Causing VF (%)	No. With ST-Segment Elevation (%)*	No. With Bundle Branch Block (%)*	No. With Transient Heart Block (%)*	Papillary Muscle Tear and/or Myocardial Rupture†
20	14	74	0 (0%)	6 (8%)	1 (1%)	0 (0%)	‡
25	11	87	6 (7%)	16 (20%)	10 (12%)	0 (0%)	‡
30	37	91	25 (27%)	30 (45%)	22 (36%)	9 (14%)	‡
40	14	38	26 (68%)	6 (50%)	7 (58%)	1 (8%)	‡
50	14	30	16 (53%)	9 (64%)	12 (86%)	7 (14%)	2 (7%)§
60	13	19	7 (37%)	12 (100%)	12 (100%)	11 (91%)	4 (21%)‖
70	13	13	5 (38%)	8 (100%)	8 (100%)	6 (75%)	10 (77%)¶
Total	116	352	75 (21%)	87 (31%)	72 (26%)	34 (12%)	16 (5%)

*Excludes animals with induced VF; †Expressed as a proportion of the number of impacts at each velocity; ‡No animal had papillary muscle tear or myocardial rupture; §Both animals also had myocardial contusions; ‖Two animals also had myocardial contusions; ¶Eight animals also had myocardial contusions.
VF = ventricular fibriliation.

With blows at 20 mph, VF was not induced in any animal. Chest impacts at 25 mph caused VF in 7% of the animals, whereas 30-mph impacts produced VF in 27%. Ventricular fibrillation incidence with chest-wall impacts increased to a maximum of 68% at 40 mph (Fig. 1, Tables 1 and 2), and then descended with blows at 50 mph, 60 mph, and 70 mph to 53%, 37%, and 38%, respectively. All episodes of VF were successfully defibrillated with immediate restoration of sinus rhythm.

LV pressure. Chest-wall impact velocities correlated with the generated peak instantaneous LV pressures ($p < 0.001$) (Fig. 2). However, when the incidence of VF was related to the peak LV pressure, the data best fit a Gaussian curve, rather than a linear curve ($p < 0.0001$ by logistic regression) (Fig. 3). The highest incidence of VF was evident with peak LV pressures between 250 mm Hg to 450 mm Hg, whereas VF incidence decreased with pressures above and below this range. The rate of LV pressure rise (dP/dt) correlated linearly with the velocity of impact, whereas the incidence of VF relative to dP/dt best fit a Gaussian curve ($p < 0.0001$ by logistic regression) (Fig. 3).

Electrophysiologic results: heart block, bundle branch block, and ST-segment elevation. In contrast with VF, the incidence of transient complete heart block, bundle branch block, and ST-segment elevation increased incrementally relative to the velocities of impact and best fit a

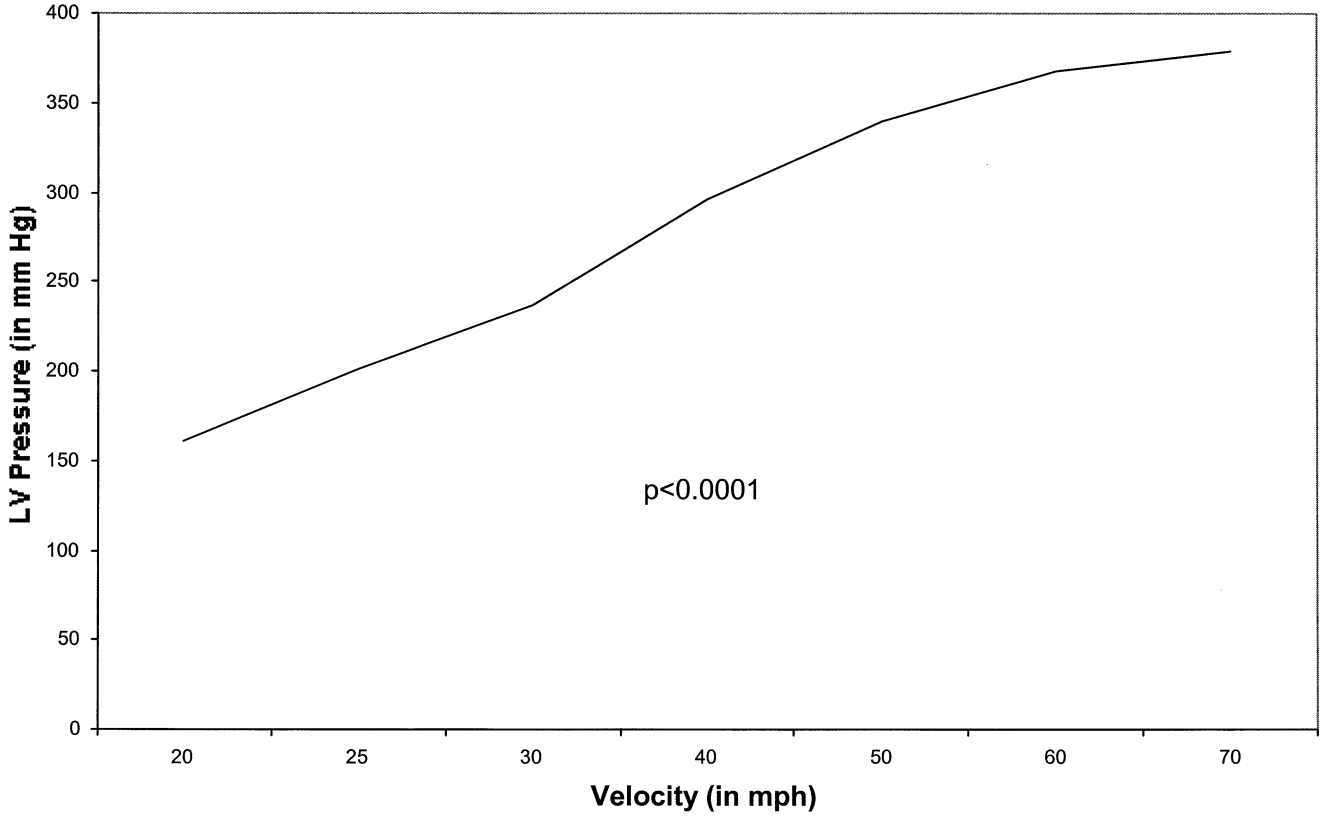

Figure 2. Peak instantaneous left ventricular (LV) pressures generated by chest-wall blows correlate linearly with the velocity of impact. P value derived from logistic regression procedure predicting peak LV pressure from velocity and velocity^2.

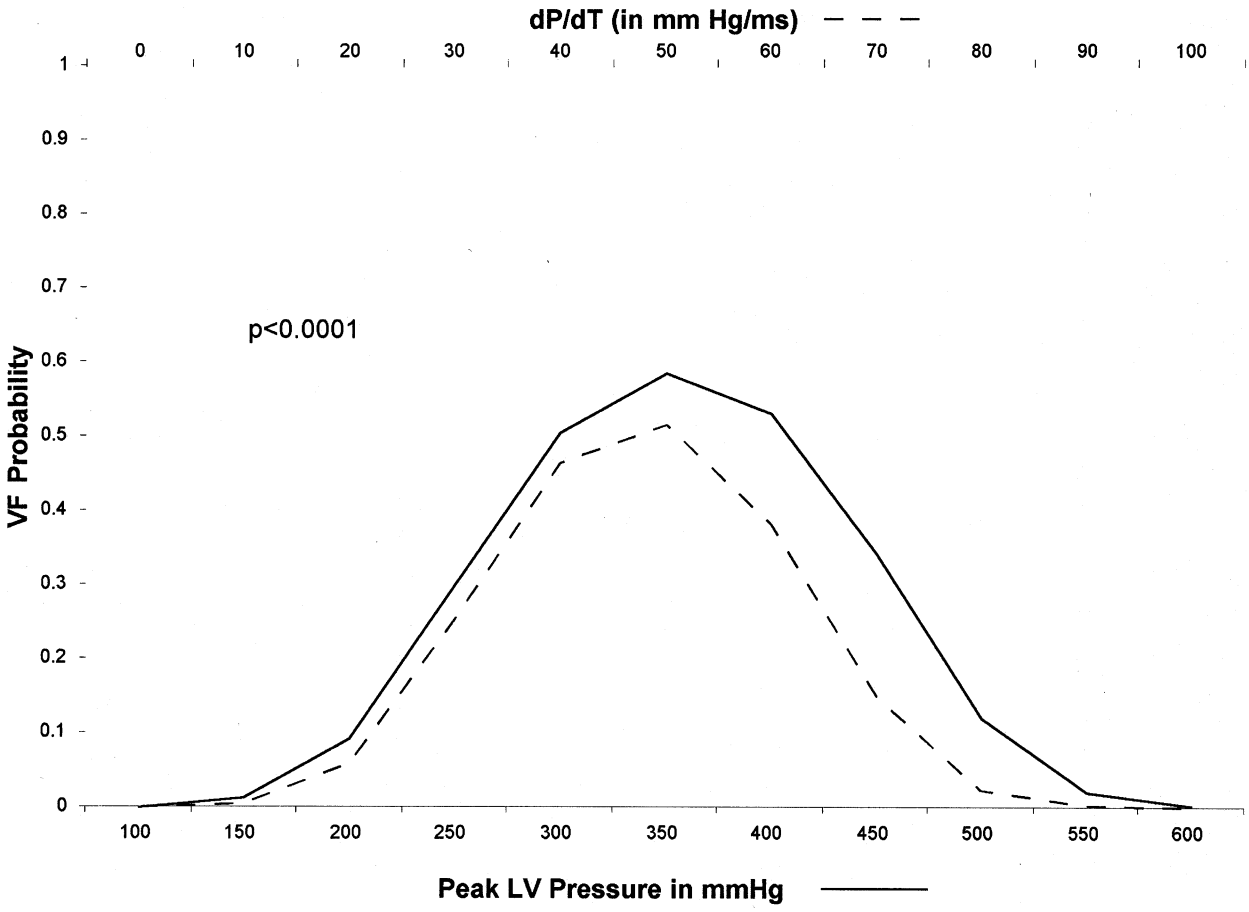

Figure 3. Incidence of ventricular fibrillation (VF) with chest-wall impact plotted against the magnitude of left ventricular (LV) pressure rise and the magnitude of LV pressure over time (dP/dt) demonstrating a highly significant Gaussian curve. P value derived from logistic regression models predicting the probability of VF from the peak LV pressure and dP/dt.

linear curve (only those impacts not resulting in VF were included in this analysis). Heart block was always transient and was not produced until 30 mph impacts (nine of 66, 14%), and increased to nearly all animals (86%) in the 60 and 70 mph impacts (chi-squared for trend analysis, $p < 0.0001$) (Table 2). Likewise, bundle branch block rarely occurred at lower velocities of impact (1% at 20 mph), but increased to 100% of impacts at 60 mph and 70 mph (when assessed during sinus rhythm after resumption of atrioventricular conduction); chi-squared for trend analysis, $p < 0.0001$. The incidence of ST-segment elevation also increased with the higher velocity impacts to 100% of impacts (chi-squared for trend analysis, $p < 0.0001$).

Morphologic analysis. All animals showed mild superficial skin bruising in the area of chest impact, but no animal incurred sternal or thoracic rib fractures. Myocardial contusions did not occur at impact velocities ≤25 mph, but were present in one of 30 of animals (3%) with strikes at 30 mph, eight of 28 (29%) at 40 to 50 mph, 12 of 26 (46%) at 60 to 70 mph. However, severe structural damage (with LV myocardial tears or papillary muscle ruptures) was limited to strikes at 50 mph (2/30; 7%), 60 mph (4/19; 21%), or at 70 mph (10/13; 77%) (Table 2). Nine animals died acutely from such severe cardiac injury, including three of papillary muscle rupture (one each at 50 mph, 60 mph, and 70 mph) and six of myocardial rupture (including one at 60 mph and five at 70 mph).

When the precordial strikes that caused severe structural cardiac damage were excluded from this analysis, the proportion of chest strikes resulting in VF were similar to that in the overall group of animals at 50 mph (14 of 28 impacts, 50% vs. 16 of 30, 54%; $p = 0.98$), 60 mph (4 of 15 impacts, 27% vs. 7 of 19, 37%; $p = 0.52$), and 70 mph (3 of 3 impacts, 100% vs. 5 of 13, 38%; $p = 0.20$).

DISCUSSION

The results of the present experiment show that commotio cordis events occur with a wide range of velocities, but are more likely in the presence of low-energy blows, consistent with the clinical experience with these catastrophic events occurring during youth sports (1–3). Under experimental conditions we found that baseball-inflicted chest blows were most likely to produce VF with 40-mph impacts, that is, in almost 70% of such blows. Impacts of 25, 30, 50, 60, and 70 mph triggered VF less frequently, and blows of 20 mph did

not produce VF. Thus, the threshold velocity to cause VF in our model (lower limit of vulnerability to commotio cordis) is 25 to 30 mph, and the greatest risk was associated with baseballs propelled at 40 mph. The predilection for commotio cordis to occur in younger athletes has been viewed in large measure as the consequence of greater chest-wall pliability in children. However, our experimental data suggest that chest impact velocities also appear to have a major role. In youth baseball where sudden death due to chest blows is most common, baseball velocities are estimated to commonly range between 30 and 50 mph (15), suggesting that the vulnerability of children to commotio cordis may, in fact, be linked to the velocities most commonly encountered in play.

Data from the present experiment may be valuable in the design of safety equipment to protect young athletes from sudden death on the playing field. For example, we can infer that, should a chest-wall protective device effectively lower the energy (and resultant intracardiac pressure rise) of the projectile to the equivalent of a 20-mph velocity baseball, then the risk for VF can probably be neutralized. However, if a chest-wall protector lowers the energy (and blunts the intracardiac pressure rise) delivered by a 60-mph ball to that at 40 mph, such a barrier may not only be ineffective, but could conceivably *increase* the likelihood of VF and a fatal impact. Therefore, our experimental commotio cordis model has the potential to define the safety limits of chest-wall protectors with respect to the velocity of impact, and may ultimately trigger the development and testing of materials to be utilized in the future design of chest protectors effective against commotio cordis.

While it is easily understood that particularly low-velocity impacts failed to cause VF, it is not immediately apparent why higher-velocity impacts of 50 to 70 mph caused VF less frequently than the 40-mph blows in our experimental model of commotio cordis. However, this apparent paradox may be explained, in part, by our peak instantaneous LV pressure data. For example, if the initiation of VF is mediated via specific ion channel activation, there may well be an optimal LV pressure rise that evokes channel activation (16–19). Our experimental data would suggest that this peak pressure rise lies in the range of 250 mm Hg to 450 mm Hg, which was, in fact, most frequently generated by 40-mph chest impacts. We have previously shown that chest-wall blows activate the K^+_{ATP} channel and that blocking this channel prevents the adverse electrophysiologic consequences of chest-wall impact (5). Therefore, it is possible that this channel or others are activated by ventricular pressure increases and resultant myocardial stretch.

A second hypothesis to explain why higher-velocity impacts are less likely to cause commotio-cordis-related VF is the critical mass theory in which a minimum amount of viable myocardial tissue must be present in order to sustain VF (20,21). Therefore, at high-impact velocities ≥50 mph, extreme cardiac structural damage is produced, and the minimum tissue mass sufficient to sustain VF may not be present. In fact, in those animals subjected to such particularly high-velocity impacts in the present experiment and others (22–24), sudden death was often due to LV myocardial damage and was not a manifestation of true commotio cordis. Indeed, such high-energy chest-wall trauma, in which extreme cardiac structural disruption occurred (e.g., papillary muscle or myocardial rupture, as in some human victims of motor vehicle accidents), not uncommonly accounts for sudden death (25). In our model, papillary muscle tears and LV wall rupture first occurred at 50 mph (7%) and increased to 77% in animals experiencing 70 mph blows, indicating that at such high velocities our model often created contusio cordis (i.e., myocardial contusion) (26). However, of note, the relationship between velocity of the chest blows and the probability of induced VF was not significantly different when the analysis was limited to those animals without structural cardiac injury. A correlate for this hypothesis can be found in the initiation of VF with electrical T-wave shocks, which not only exhibit a Gaussian curve for the initiation of VF, but also show that VF can be produced with very high energy shocks that cause myocardial tissue damage (9,11,27–29).

In conclusion, similar to the electrical induction of VF, mechanically triggered VF (i.e., commotio cordis) exhibits a Gaussian curve relative to the energy of impact. The frequency with which commotio cordis occurs in young athletes in whom baseballs frequently achieve estimated velocities of about 40 mph may be partially explained by our experimental model in which VF was most commonly induced at this velocity. As in electrically induced VF, the lower and upper limits of vulnerability for the mechanically induced VF of commotio cordis provide important insights into this phenomenon. The observation that energy of impact is an important variable in the generation of VF with chest-wall blows may partially explain the rarity of these events. Also, armed with the knowledge that the energy of chest impact is safest at ≤20 mph, use of this model will allow assessment of whether safety equipment, including softer than standard baseballs and protective chest-wall barriers, will offer a large measure of protection against commotio-cordis-related sudden death in young athletes.

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