

## AN EXPERIMENTAL MODEL OF SUDDEN DEATH DUE TO LOW-ENERGY CHEST-WALL IMPACT (COMMOTIO CORDIS)

MARK S. LINK, M.D., PAUL J. WANG, M.D., NATESA G. PANDIAN, M.D., SAROJA BHARATI, M.D.,  
JAMES E. UDELSON, M.D., MAN-YOUNG LEE, M.D., MARK A. VECCHIOTTI, B.S., BRIAN A. VANDERBRINK, B.S.,  
GIANLUCA MIRRA, M.D., BARRY J. MARON, M.D., AND N.A. MARK ESTES III, M.D.

### ABSTRACT

**Background** The syndrome of sudden death due to low-energy trauma to the chest wall (commotio cordis) has been described in young sports participants, but the mechanism is unknown.

**Methods** We developed a swine model of commotio cordis in which a low-energy impact to the chest wall was produced by a wooden object the size and weight of a regulation baseball. This projectile was thrust at a velocity of 30 miles (48 km) per hour and was timed to the cardiac cycle.

**Results** We first studied 18 young pigs, 6 subjected to multiple chest impacts and 12 to single impacts. Of the 10 impacts occurring within the window from 30 to 15 msec before the peak of the T wave on the electrocardiogram, 9 produced ventricular fibrillation. Ventricular fibrillation was not produced by impacts at any other time during the cardiac cycle. Of the 10 impacts sustained during the QRS complex, 4 resulted in transient complete heart block.

We also studied whether the use of safety baseballs, which are softer than standard ones, would reduce the risk of arrhythmia. A total of 48 additional animals sustained up to three impacts during the T-wave window of vulnerability to ventricular fibrillation with a regulation baseball and safety baseballs of three degrees of hardness. We found that the likelihood of ventricular fibrillation was proportional to the hardness of the ball, with the softest balls associated with the lowest risk (two instances of ventricular fibrillation after 26 impacts, as compared with eight instances after 23 impacts with regulation baseballs).

**Conclusions** This experimental model of commotio cordis closely resembles the clinical profile of this catastrophic event. Whether ventricular fibrillation occurred depended on the precise timing of the impact. Safety baseballs, as compared with regulation balls, may reduce the risk of commotio cordis. (N Engl J Med 1998;338:1805-11.)

©1998, Massachusetts Medical Society.

**S**UDDEN death may occur in young sports participants when a baseball or other projectile strikes the victim in the precordium. This phenomenon is termed commotio cordis and predominantly affects children and adolescents 5 to 15 years of age without preexisting heart disease.<sup>1</sup> Characteristically, there is no structural damage to the chest wall, thoracic cavity, or heart. In 1996 the Consumer Product Safety Commission described 38

deaths from baseball blows to the chest between 1973 and 1995.<sup>2</sup> In addition, commotio cordis has been reported in ice hockey, lacrosse, softball, and as a consequence of fistfights<sup>1,3-6</sup> and may, indeed, be more common than was initially believed.<sup>7</sup>

In most cases of commotio cordis the victim does not survive. In the few cases in which a cardiac rhythm was documented after collapse, the most common rhythm was ventricular fibrillation.<sup>1,3-6,8</sup> Complete heart block<sup>3</sup> and idioventricular rhythm<sup>9</sup> have also been described. Among the few reported survivors, marked precordial ST-segment elevation has been seen.<sup>4,8,10</sup>

Previous efforts to replicate commotio cordis experimentally have been limited by the considerable chest-wall trauma inflicted, which caused severe cardiac and thoracic injuries.<sup>11-13</sup> Our goal was to develop an animal model of low-energy impact to the chest that replicated the clinical presentation of commotio cordis. With this model we then evaluated softer-than-standard (safety) baseballs to assess the relative risk of their producing sudden cardiac death with nonpenetrating chest-wall impact.

### METHODS

#### Laboratory Animals

Young domesticated pigs, four to eight weeks old and weighing 8 to 12 kg, were used in this study. Pigs were chosen because their anatomy is similar to that of humans and because the previous work on chest-wall trauma also used pigs.<sup>12-14</sup> The research protocol was approved by the Animal Research Committee of the New England Medical Center and conducted in accordance with the regulations of the Association for Assessment and Accreditation of Laboratory Animal Care.

In the development of the model, a total of 22 animals were studied. One group of six animals sustained a total of 20 impacts (range, 1 to 6) at various times throughout the cardiac cycle. Subsequently, 12 animals were subjected to only a single impact, delivered during either the QRS complex or the T wave. Four other animals that underwent all the experimental procedures except the chest-wall impact served as controls.

To evaluate the safety baseballs, we subjected 48 additional animals to as many as three impacts with one of four balls. These impacts were sustained on the upslope of the T wave, at a time in the cardiac cycle when we had demonstrated that the first 22

From the Cardiac Arrhythmia Service, Tufts–New England Medical Center, Boston (M.S.L., P.J.W., N.G.P., J.E.U., M.-Y.L., M.A.V., B.A.V., G.M., N.A.M.E.); the Maurice Lev Congenital Heart and Conduction Center, the Heart Institute for Children, Christ Hospital and Medical Center, Oak Lawn, Ill. (S.B.); and the Cardiovascular Research Division, Minneapolis Heart Institute Foundation, Minneapolis (B.J.M.). Address reprint requests to Dr. Link at the New England Medical Center, Box 197, 750 Washington St., Boston, MA 02111.

animals were vulnerable to the induction of ventricular fibrillation. The four balls differed with respect to hardness and consisted of a very soft safety ball (Reduced Injury Factor [RIF] 1; Worth, Tullahoma, Tenn.), marketed for use by children 5 to 7 years old; a medium-soft ball (RIF 5), for use by children 8 to 10 years old; a least-soft ball (RIF 10), for use by children 11 and older; and a standard regulation Little League baseball (Rawlings Little League LLB-1, Rawlings Sporting Goods, St. Louis). All the balls had the same mass (150 g) and were propelled at the same velocity (30 miles [48 km] per hour).

### Chest-Wall Impact

In the first 18 animals, the impact to the chest wall was caused by striking them with a wooden object, similar in size, shape (convex), and weight (150 g) to a regulation baseball. The remaining animals sustained impacts with one of four baseballs of various degrees of hardness. The wooden object and the baseballs were mounted on the end of a light-weight aluminum shaft. They were designed to strike the animal perpendicularly to the chest wall, directly over the left ventricle, at a speed of 30 miles per hour, measured by a chronograph (Oehler Research, Austin, Tex.) modified for low velocity. The release of the object was timed to the cardiac cycle with the use of a commercially available cardiac stimulator (EP-2, EP Medical, Budd Lake, N.J.) triggered by surface electrocardiographic R waves from the pigs. There was a known and consistent delay of 130 msec between the release of the object and the subsequent impact. Thus, the chest impact could be timed throughout the cardiac cycle with an accuracy of  $\pm 5$  msec.

### Experimental Protocol

The animals were sedated with 12 mg of intramuscular ketamine per kilogram of body weight and with inhaled isoflurane and then intubated. Anesthesia was maintained with 1.0 to 2.0 percent isoflurane mixed with oxygen and nitrous oxide. Six-lead coronal electrocardiograms were monitored continuously. Pigtail catheters were placed in the left ventricular apex and the ascending aortic arch. A base-line myocardial perfusion scan was performed with the injection of 2 to 3 mCi of technetium-99m sestamibi. Twenty minutes later, base-line nuclear images were obtained with planar images in the anterior, left anterior oblique, and left lateral projections. All the pigs were then placed prone in a sling. Base-line left ventriculography, coronary angiography (with an aortic-root flush), and echocardiography were then performed.

The blows to the chest were delivered at various times during the cardiac cycle. If ventricular fibrillation occurred, the animals were electrically defibrillated into sinus rhythm. Within 60 seconds of defibrillation, 7 to 9 mCi of technetium-99m sestamibi was injected, and aortic-root injection and left ventriculography were performed. Once the animals had ventricular fibrillation, they were not struck again. If ST elevations or heart block occurred, technetium-99m sestamibi was injected immediately and coronary angiograms were obtained. After each impact, echocardiography was performed after angiography. All the animals were euthanized with an intravenous potassium chloride solution. Another set of angiograms obtained with technetium-99m sestamibi, matched with the preimpact views, was taken. Because of the theoretical concern that the left ventricular and aortic-arch catheters could induce arrhythmias, the last five animals in the group that sustained a single chest impact did not have catheters placed.

Once the period of vulnerability to ventricular fibrillation had been determined, 48 additional animals were exposed to as many as three impacts during this period with one of the three safety baseballs or the regulation baseball. If ventricular fibrillation was produced, the animals were exposed to no further impacts.

### Data Analysis

Perfusion images obtained with technetium-99m sestamibi, left ventricular angiograms, and coronary arteriograms were inter-

preted by one observer who was unaware of which animals were controls and which were subjected to impact. Echocardiograms were analyzed by another observer who was likewise blinded to the study protocol. Echocardiograms were assessed for the presence, location, and severity of regional wall-motion abnormalities according to the 16-segment scoring system of the American Society of Echocardiography.<sup>15</sup>

Gross pathological evaluation of the chest wall, heart, pericardium, and great vessels was performed after the animals were euthanized. The hearts were preserved in a solution of 10 percent formalin. Histologic examination was performed on specimens of myocardium from the anterior and posterior walls of the left and right ventricles of 18 animals. More specialized pathological evaluation was performed by a third observer, also blinded to the experimental design. In six animals (two with ventricular fibrillation, two with heart block and ST elevations, one with ST elevations, and one control), specialized histologic analysis of the conduction system was performed and compared with previous observations in pigs.<sup>16</sup> This assessment included serial-section examinations of the sinus node and its approaches, the atrioventricular node and its approaches, the atrioventricular bundle, and the bundle branches. The sections were stained alternately by hematoxylin and eosin and Weigert-van Gieson stains.

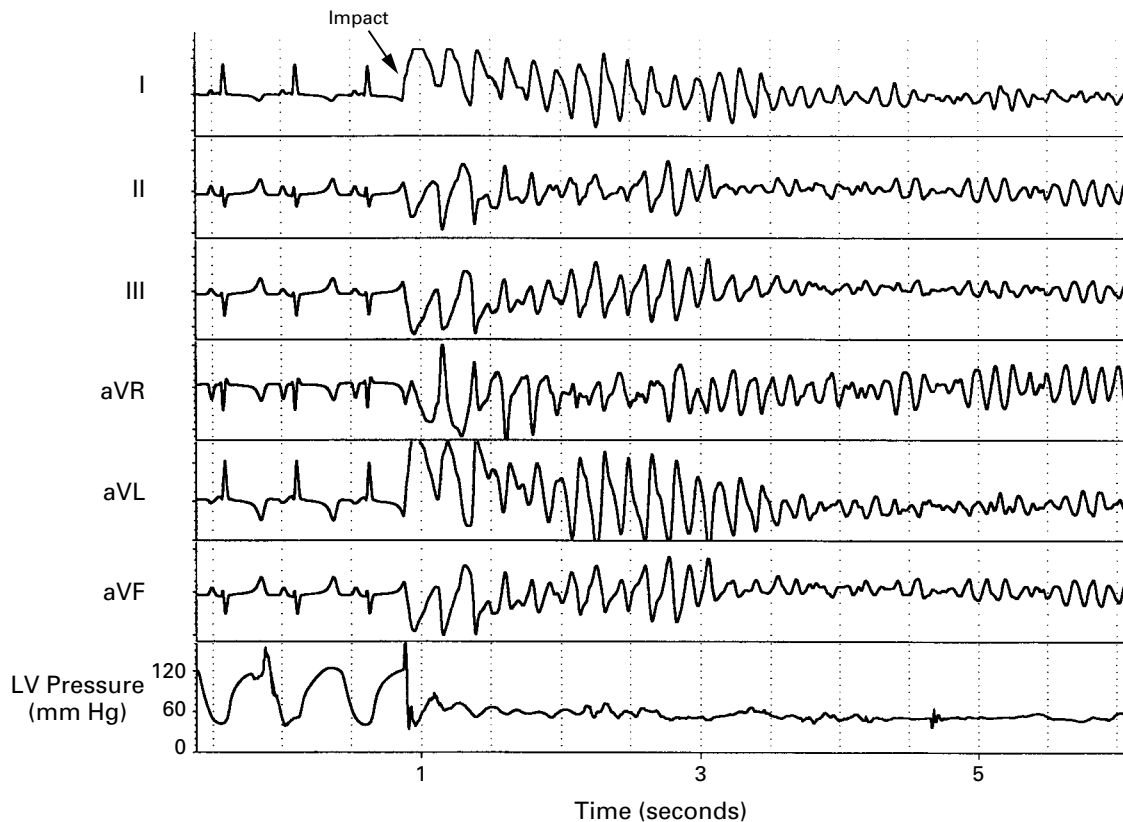
The statistical significance of the differences between groups was analyzed with a two-sided Fisher's exact test and a chi-square test for trend.

## RESULTS

### Electrophysiologic Consequences of Chest-Wall Impact

Chest-wall impact during the T wave produced nine episodes of ventricular fibrillation with 17 impacts (six episodes with the first chest impact and one each with the second, third, and sixth impacts). Ventricular fibrillation occurred immediately on impact, commencing on the next heartbeat (Fig. 1). Each of the nine impacts that produced ventricular fibrillation occurred from 30 to 15 msec (mean,  $22 \pm 5$ ) before the peak of the T wave (Table 1). Of a total of 10 blows during this 15-msec window, 9 produced ventricular fibrillation. The occurrence of ventricular fibrillation due to chest-wall impact during this period of vulnerability on the T-wave upstroke (9 of 10 impacts) was statistically significant as compared with the occurrence of ventricular fibrillation induced by impact at other times during the cardiac cycle (0 of 22 impacts) ( $P < 0.001$ ). Seven impacts during the T wave but outside the 30-to-15-msec window failed to produce ventricular fibrillation; however, brief bursts of polymorphic ventricular tachycardia occurred with two blows at 13 msec (four beats) and 33 msec (five beats) before the T-wave peak.

Of the 10 impacts during the QRS complex, 4 were followed by transient and instantaneous complete heart block (Fig. 2 and Table 1), which persisted for up to seven sinus beats. When sinus rhythm returned, ST-segment elevation and left bundle-branch block were present and persisted for up to 120 seconds. The occurrence of heart block with impacts during the QRS complex (4 of 10 impacts) was significant as compared with the occurrence of heart block with impacts during other portions of



**Figure 1.** Six-Lead Electrocardiogram Showing the Electrophysiologic and Hemodynamic Consequences of an Impact to the Chest by a Wooden Object at 30 Miles per Hour, Timed to Occur 16 msec before the Peak of the T Wave in a 9-kg Pig.

Ventricular fibrillation began immediately (within one cardiac cycle) after the chest impact, which was associated with instant loss of effective left ventricular (LV) pressure.

the cardiac cycle (0 of 22) ( $P=0.006$ ). The six impacts during the QRS complex that did not produce complete heart block elicited immediate ST-segment elevation and left bundle-branch block. With the 10 QRS impacts, ST-segment elevation occurred inferiorly in 4, anterolaterally in 4, and diffusely in 2 and was most marked on the first conducted beat following impact, gradually decreasing over the subsequent 30 to 120 seconds (Fig. 2).

Impacts during the ST segment (five impacts) caused left bundle-branch block and associated ST-segment elevation twice and isolated ST-segment elevation twice.

#### Myocardial Perfusion and Wall-Motion Abnormalities

Coronary angiography performed immediately after impact in 13 pigs subjected to impact and in the 4 controls showed no epicardial abnormalities of the coronary artery, such as spasm, dissection, or stenosis. Myocardial-perfusion imaging with technetium-99m sestamibi was performed after impact in 12 animals as well as in the 4 controls. Only 3 of the 12 animals that sustained chest impact had new myo-

cardial-perfusion defects (including 2 of 5 animals resuscitated from ventricular fibrillation and 1 of 7 animals with ST-segment elevation). The perfusion defects were small, apical, and mild. No control animal had a perfusion defect.

Left ventriculograms and echocardiograms revealed apical or distal septal hypokinesis in nine animals after impact (4 of 9 impacts with ventricular fibrillation and 5 of 10 impacts during the QRS complex that produced ST elevations). No control animal had a wall-motion abnormality.

#### Pathological Findings

No animal had rib fractures, hemothorax, hemo-pericardium, or evidence of myocardial contusion. Mild hyperemia of the myocardium was observed in all the animals, including the controls. Of the 18 hearts (including those of 4 controls) in which we performed histologic examination of the left and right ventricles, 3 showed a moderate degree of hemorrhage in the anterior left ventricle (1 with induced ventricular fibrillation, 1 with ST elevation, and 1 from a control).

**TABLE 1.** TIMES OF IMPACT AND ELECTROPHYSIOLOGIC RESULTS OF IMPACTS DELIVERED AT 30 MILES PER HOUR.\*

ANIMAL NO.	T-WAVE IMPACTS		QRS IMPACTS	ST-SEGMENT IMPACTS
	30 TO 15 msec BEFORE PEAK	DURING OTHER PARTS OF T WAVE		
	no. of impacts: electrophysiologic result			
1	1: ST elevation		1: CHB for 3 beats, BBB, ST elevation	1: ST elevation
2	1: VF		2: BBB, ST elevation after both	
3	1: VF			
4	1: VF	1: BBB, ST elevation	1: BBB, ST elevation	3: ST elevation after 2 impacts, BBB after 1 impact
5	1: VF	1: No electrocardiographic effects		
6		3: PMVT for 4 beats after 1 impact	1: BBB, ST elevation	1: BBB, ST elevation
7	1: VF			
8			1: CHB for 4 beats, BBB, ST elevation	
9	1: VF			
10		1: ST elevation		
11	1: VF			
12			1: BBB, ST elevation	
13			1: BBB, ST elevation	
14		1: PMVT for 2 beats		
15	1: VF			
16			1: CHB for 7 beats, BBB, ST elevation	
17	1: VF			
18			1: CHB for 2 beats, BBB, ST elevation	
Total	10 total impacts: 9 episodes of VF	7 total impacts: 2 brief episodes of PMVT, 1 episode of BBB, and 2 of ST elevation	10 total impacts: 4 episodes of combined CHB, BBB, and ST elevation; BBB and ST elevation in 6 others	5 total impacts: 2 episodes of BBB and 4 of ST elevation

\*CHB denotes complete heart block, BBB bundle-branch block, VF ventricular fibrillation, and PMVT polymorphic ventricular tachycardia.

In 6 of these 18 animals, specialized examination of the conduction system was undertaken. In one of the two pigs with heart block induced by impact during the QRS complex, marked hemorrhage of the atrioventricular bundle and bundle branches was present. In each of the animals with ventricular fibrillation, mild hemorrhage was present in the approaches to the sinus node; one of these animals also had mild hemorrhage in the periphery of the left bundle branch.

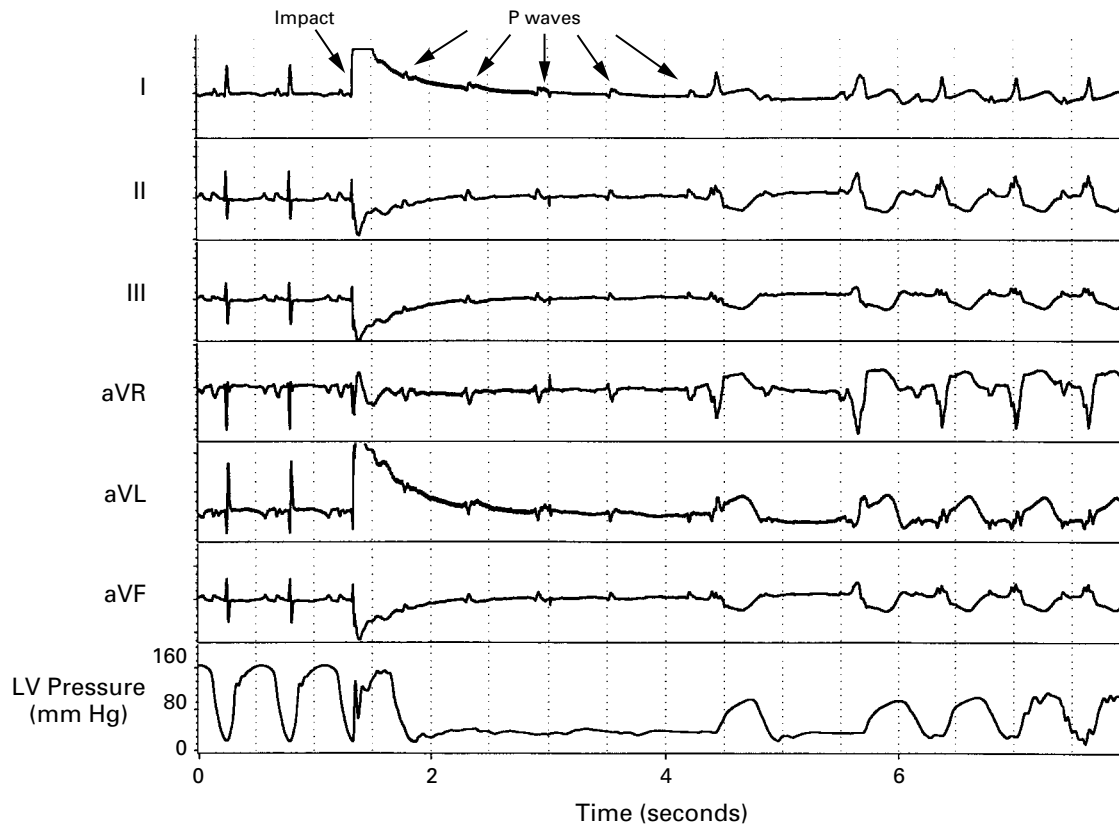
**Evaluation of Regulation and Safety Baseballs**

Significant differences in the occurrence of ventricular fibrillation were observed among the baseballs tested (P for trend <0.001) (Fig. 3). In addition, there were significant differences in the induction of ventricular fibrillation between the softest safety ball and the regulation ball (P=0.03) and between the wooden object and each of the baseballs (P<0.01 for all comparisons). No significant differences were evident between the regulation baseball and the me-

dium-soft and the least-soft safety baseballs. With the softest ball, ventricular fibrillation was produced twice in 26 impacts (8 percent) in 12 animals (Fig. 3). With the medium-soft ball, 27 impacts produced six episodes of ventricular fibrillation (22 percent) in 12 animals. Twenty-one chest impacts with the least-soft safety ball resulted in six episodes of ventricular fibrillation (29 percent) in 12 other animals. In the remaining 12 animals, 23 impacts with a regulation baseball resulted in eight episodes of ventricular fibrillation (35 percent).

**DISCUSSION**

This report describes an animal model of commotio cordis that mimics the syndrome of sudden death due to low-energy impact to the chest wall that has been described in young sports participants.<sup>1</sup> With this experimental model, we were able to induce ventricular fibrillation, complete heart block, and ST-segment elevation reproducibly with low-energy blows to the chest wall. Our model for chest-wall



**Figure 2.** Six-Lead Electrocardiogram Showing the Electrophysiologic and Hemodynamic Consequences of an Impact to the Chest by a Wooden Object at 30 Miles per Hour during the QRS Complex in an 8-kg Pig.

Complete heart block began immediately and persisted for seven beats, after which atrioventricular conduction resumed in association with marked ST-segment elevation and left bundle-branch block. A tracing of the left ventricular (LV) catheter pressure shows the immediate loss of effective intraventricular pressure on impact.

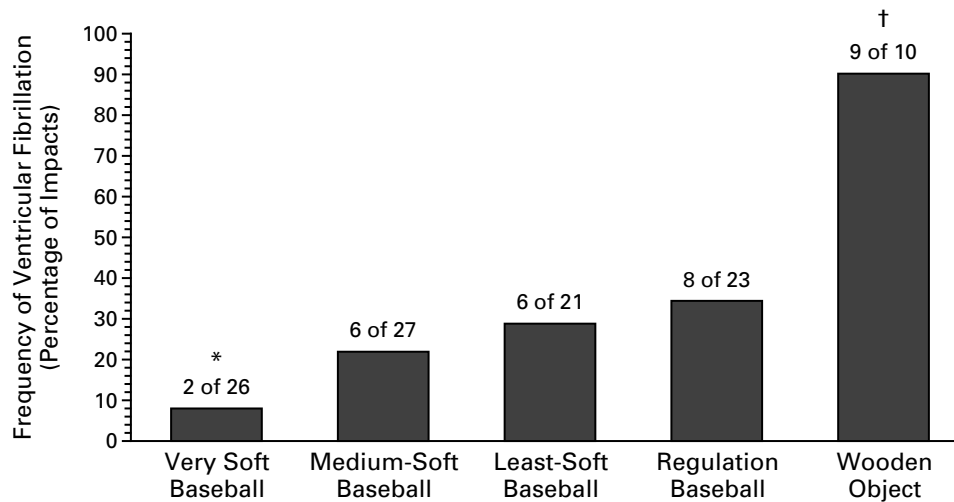
impact differs from previous models<sup>11-13</sup> not only because the force used was markedly less, but also because the impact was timed to various points in the cardiac cycle.

We found that the electrophysiologic consequences of chest-wall impacts were critically dependent on the precise timing of the impact during the cardiac cycle. When the impact occurred in a narrow 15-msec window during cardiac repolarization (just before the peak of the T wave), ventricular fibrillation was consistently produced. In this circumstance, ventricular fibrillation was induced instantaneously and was not preceded by premature ventricular contractions, ischemic ST-segment changes, or heart block.

In contrast, chest-wall impact during ventricular depolarization (the QRS complex) did not produce ventricular fibrillation but, rather, transient complete heart block followed by ST-segment elevation and, in some pigs, left bundle-branch block. It is possible that blunt chest-wall impact in such animals produced trauma to critical areas of the conduction sys-

tem directly. The observed ST-segment elevation was marked and immediate, occurring on the first conducted beat after impact. We did not, however, find evidence on perfusion imaging and coronary arteriography to support the presence of myocardial ischemia in these animals. Previous investigators have shown that ST-segment elevation following acute epicardial coronary occlusion does not occur immediately but only after one to three minutes of ischemia.<sup>17</sup> Thus, it is unlikely that the ST-segment elevation observed in our model is due to epicardial coronary-artery spasm, dissection, or thrombosis.

The profound electrophysiologic findings reported in this animal model are similar to those reported in victims of commotio cordis.<sup>1,3-6,8,9</sup> The cardiac rhythms initially identified in these victims were predominantly ventricular fibrillation,<sup>1,3-6,8</sup> but complete heart block<sup>1,3</sup> and idioventricular rhythm<sup>9</sup> have also been reported. Moreover, ST-segment elevation, sometimes associated with segmental wall-motion abnormalities, has been described in a few survivors of commotio cordis.<sup>4,8,10</sup> The energy and location of



**Figure 3.** Differences in the Frequency with Which Ventricular Fibrillation Resulted from Chest-Wall Impact at 30 Miles per Hour during the Period of the Cardiac Cycle Vulnerable to Induction of Ventricular Fibrillation (from 30 to 15 msec before the T-Wave Peak).

Safety baseballs of different hardnesses (Reduced Injury Factor 1, 5, and 10) were compared with a regulation Little League baseball and the wooden object used to simulate a baseball in this model. Shown above each bar is the number of impacts that resulted in ventricular fibrillation with respect to the total number of impacts during the period of vulnerability to the induction of ventricular fibrillation. Significant differences in the occurrence of ventricular fibrillation were observed among the baseballs tested ( $P$  for trend  $<0.001$  by the chi-square test). The asterisk denotes  $P < 0.03$  for the differences between the regulation baseball and the very soft baseball. The dagger denotes  $P < 0.01$  for the differences between the wooden object and each type of baseball.

the precordial impact in our model also appear to be similar to the clinical profile of commotio cordis. Maron et al.<sup>1</sup> found that precordial blows by baseballs were responsible for death in 19 of 25 victims. Estimates of the impact velocity of these baseballs ranged from 20 to 50 miles (32 to 80 km) per hour (average, 30 miles [48 km] per hour), the rate used in our experiment. In the series of Maron et al.,<sup>1</sup> there were no pathological abnormalities more severe than skin contusions (seen in 12 of 22 victims) and minor myocardial and pulmonary contusions (seen in 11 of 22 victims), nor were any observed in our pigs.

The use of safety baseballs has been recommended by the Consumer Product Safety Commission as a way to decrease the incidence of injuries in youth baseball.<sup>18</sup> Nevertheless, there has been substantial controversy about whether softer baseballs truly decrease the risk of commotio cordis.<sup>19</sup> Some investigators have even suggested that the risk of commotio cordis may be increased by a softer baseball as compared with a regulation ball.<sup>19</sup> However, using our model of commotio cordis, we found a relation between the hardness of the baseball and the likelihood of ventricular fibrillation. Impact with the softest baseball was associated with the lowest risk of producing ventricular fibrillation. The medium-soft baseballs and the least-soft baseballs had lower incidences of ventricular fibrillation than the regulation baseball, but these differences did not achieve statis-

tical significance. Although the suitability of the softest safety ball for competitive play by older youths is limited because of its pliability, its use in T-ball or recreational play may be feasible. The medium-soft and least-soft balls may be suitable for play in the older age groups; however, further research is needed to determine whether these balls will reduce the risk of commotio cordis in actual play.

Our animal model, although it closely resembles the clinical syndrome of commotio cordis, has some potential limitations. For example, it is possible that the general anesthesia we used may have lowered the threshold for induced arrhythmias. The young pigs used in this model were smaller than the children and adolescents who have experienced commotio cordis in the community. Thus, the force of the chest-wall impacts may have been relatively greater in the pigs than in humans. Also, because the geometry of the chest of pigs differs from that of humans, the transmission of energy from the chest wall to the heart may be different. Because of concern about the potential proarrhythmic effects of the left ventricular catheter, we studied the last five animals without catheters placed. Nevertheless, in these pigs, two of three impacts during the T wave produced ventricular fibrillation, and one of two impacts during the QRS complex caused complete heart block.

We have described an experimental model of commotio cordis that closely simulates the clinical

profile of that catastrophic event. With our swine model we have shown that a precisely timed low-energy impact can have devastating and immediate electrophysiologic sequelae, even when induced by only modest force. Ventricular fibrillation was reproducibly and instantaneously initiated with impacts during a narrow window on the upstroke of the T wave. Impacts during the QRS complex immediately and reproducibly induced complete heart block as well as associated ST-segment elevation and left bundle-branch block. Furthermore, in this model, the use of safety baseballs decreased the incidence of ventricular fibrillation, suggesting that their use in the community may reduce the risk of commotio cordis. Ultimately, this model could be used to evaluate devices for chest-wall protection and resuscitative measures that could lead to a decreased risk of sudden death due to chest-wall impact among young athletes.

Supported by a grant from the National Operating Committee on Standards for Athletic Equipment, Overland Park, Kans. The opinions expressed herein are those of the authors and do not necessarily reflect the opinions of the committee.

*We are indebted to Dr. Bonnie L. Bermas for her careful review of the manuscript; to Debra Kinan for her assistance with the technetium-99m sestamibi perfusion imaging; to Robert Doucette for his engineering assistance; to Stacey E. Supran of the Division of Clinical Care Research at the New England Medical Center for her assistance with the statistical analysis; and to Darisse A. Paquette, C.M.I., for her assistance with the figures.*

## REFERENCES

1. 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;333:337-42.
2. Adler P, Monticone RC. Injuries and deaths related to baseball. In: Kyle SB, ed. Youth baseball protective equipment project final report. Washington, D.C.: Consumer Product Safety Commission, 1996:1-43.
3. Kaplan JA, Karofsky PS, Volturo GA. Commotio cordis in two amateur ice hockey players despite the use of commercial chest protectors: case reports. *J Trauma* 1993;34:151-3.
4. Abrunzo TJ. Commotio cordis: the single, most common cause of traumatic death in youth baseball. *Am J Dis Child* 1991;145:1279-82.
5. Dickman GL, Hassan A, Luckstead EF. Ventricular fibrillation following baseball injury. *Physician Sportsmed* 1978;6(7):85-6.
6. Maron BJ, Strasburger JF, Kugler JD, Bell BM, Brodkey FD, Poliac LC. Survival following blunt chest impact-induced cardiac arrest during sports activities in young athletes. *Am J Cardiol* 1997;79:840-1.
7. Maron BJ, Poliac LC, Kyle SB. Clinical profile of commotio cordis: an underappreciated cause of sudden cardiac death in the young during sporting activities. *Circulation* 1997;96:Suppl I:I-755. abstract.
8. Link MS, Ginsburg SH, Wang PJ, Kirchhoffer JB, Estes NAM III, Paris YM. Commotio cordis: cardiovascular manifestations of a rare survivor. *Chest* (in press).
9. Green ED, Simson LR Jr, Kellerman HH, Horowitz RN, Sturmer WQ. Cardiac concussion following softball blow to the chest. *Ann Emerg Med* 1980;9:155-7.
10. Morikawa M, Hirose K, Mori T, Kusakawa J, Tomioka N, Watanabe Y. Myocardial contusion caused by a baseball. *Clin Cardiol* 1996;19:831-3.
11. Liedtke AJ, Gault JH, Demuth WE. Electrocardiographic and hemodynamic changes following nonpenetrating chest trauma in the experimental animal. *Am J Physiol* 1974;226:377-82.
12. Cooper GJ, Pearce BP, Stainer MC, Maynard RL. The biomechanical response of the thorax to nonpenetrating impact with particular reference to cardiac injuries. *J Trauma* 1982;22:994-1008.
13. Viano DC, Andrzejak DV, Polley TZ, King AI. Mechanism of fatal chest injury by baseball impact: development of an experimental model. *Clin J Sport Med* 1992;2:166-71.
14. Howe B, Fehn PA, Pensinger RR. Comparative anatomical studies of the coronary arteries of canine and porcine hearts. I. Free ventricular walls. *Acta Anat (Basel)* 1968;71:13-21.
15. Schiller NB, Shah PM, Crawford M, et al. Recommendations for quantitation of the left ventricle by two-dimensional echocardiography. *J Am Soc Echocardiogr* 1989;2:358-67.
16. Bharati S, Levine M, Huang SKS, et al. The conduction system of the swine heart. *Chest* 1991;100:207-12.
17. Kleber AG, Janse MJ, van Capelle FJL, Durrer D. Mechanism and time course of S-T and T-Q segment changes during acute regional myocardial ischemia in the pig heart determined by extracellular and intracellular recordings. *Circ Res* 1978;42:603-13.
18. Kyle SB. Youth baseball protective equipment project final report. Washington, D.C.: Consumer Product Safety Commission, 1996.
19. Janda DH, Viano DC, Andrzejak DV, Hensinger RN. An analysis of preventive methods for baseball-induced chest impact injuries. *Clin J Sport Med* 1992;2:172-9.