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Development of a Chest Wall Protector Effective in Preventing Sudden Cardiac Death by Chest Wall Impact (Commotio Cordis)

Kartik Kumar, MBBS, Swati N. Mandleywala, MBBS, Michael P. Gannon, MD, Nathan Anthony Mark Estes III, MD, Jonathan Weinstock, MD, and Mark S. Link, MD

Objective: Commotio cordis, sudden death with chest impact, occurs clinically despite chest wall protectors worn in sports. In an experimental model of commotio cordis, commercially available chest wall protectors failed to prevent ventricular fibrillation (VF). The goal of the current investigation was to develop a chest wall protector effective in the prevention of commotio cordis.

Design: In the Tufts experimental model of commotio cordis the ability of chest protectors to prevent VF was assessed. Impacts were delivered with a 40-mph lacrosse ball, timed to the vulnerable period for VF.

Intervention: A chest wall protector or no chest wall protector (control) was randomly assigned to be placed over the chest. Four iterative series of 2 to 4 different chest wall material combinations were assessed. Materials included 3 different foams (Accelleron [Unequal Technologies, Glen Mills, PA], closed cell high density foam; Airilon [Unequal Technologies, Glen Mills, PA], closed cell low density soft foam; and an open cell memory foam) that were adhered to a layer of TriDur (Unequal Technologies, Glen Mills, PA), a flexible elastomeric coated aramid that was bonded to a semirigid polypropylene polymer (ImpacShield, Unequal Technologies, Glen Mills, PA).

Main Outcome Measure: Induction of VF by chest wall impact was the primary outcome.

Results: Of 80 impacts without chest protectors, 43 (54%) resulted in VF. Ventricular fibrillation with chest protectors ranged from a high of 60% to a low of 5%. Of 12 chest protectors assessed, only 3 significantly lowered the risk of VF compared with impacts without chest protectors. These 3 chest protectors were combinations of Accelleron, Airilon, TriDur, and ImpacShield of different thicknesses. Protection increased linearly with the thicker combinations.

Conclusions: Effective protection against VF with chest wall protection can be achieved in an experimental model of commotio cordis.

Clinical Relevance: Chest protector designs incorporating these novel materials will likely be effective in the prevention of commotio cordis on the playing field.

Key Words: commotio cordis, sudden cardiac death, athlete, ventricular fibrillation, chest protectors

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INTRODUCTION

Sudden cardiac death due to chest impact with projectiles in sports (commotio cordis) is rare but devastating.[1–5] Baseball, lacrosse, and hockey are 3 common sports in which commotio cordis occurs. Although these events are not common, the clinical consequences are devastating. Prevention of these events is highly desirable; yet, sudden death has occurred in athletes with chest wall impact despite the use of a chest protector.[6] Indeed, in commotio events in competitive sports, more than one-third occurs in individuals wearing a chest protector.[6] In hockey, the chest protector is commonly lifted up when the arms are raised; thus, it no longer covers the precordial area. However, in lacrosse and baseball, the impact is directly over the chest protector; yet, sudden death has still occurred. In our experimental swine model of commotio cordis, we have replicated the human condition. In this model, commercially available chest protectors manufactured in 2002, similar if not identical to those worn in young athletes who died from chest impact, did not lower the risk of sudden death with ball impact.[7] The current project is designed to produce a chest protector that will lower the risk of sudden death with chest impact.

METHODS

Experimental Model

The experimental model has been extensively published.[8–11] The research protocol was approved by the Animal Research Committee of Tufts Medical Center in conformity with the regulations of the Association for Assessment and Accreditation of Laboratory Animal Care. Briefly, juvenile domesticated male swine, 12 to 16-weeks-old and weighing 15 to 25 kg (mean 19.7 ± 3.9 kg), are sedated with 12 mg/kg intramuscular ketamine and then anesthetized with inhaled

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Corresponding Author: Mark S. Link, MD, Tufts Medical Center Box # 197, 800 Washington St, Boston, MA 02111 (mlink@tuftsmedicalcenter.org).

1% to 2% isoflurane mixed with oxygen. Anesthesia was maintained with isoflurane. Left ventricular pressure catheters (Millar Mikrotip) were placed in the left ventricle. Animals were then positioned prone in a sling to approximate physiologic blood flow and cardiac hemodynamics.

Chest wall impact was produced by a lacrosse ball mounted on a lightweight (20 g) aluminum shaft. The impact object was directed, with echocardiographic guidance, to strike the animal perpendicular to the chest wall, directly over the center of the heart during the vulnerable time window for VF. Impacts occurring outside this time window were excluded from the analysis. All impacts were given at 40 mph; in previous experiments, 50-mph impacts caused myocardial and valvular rupture, features not consistent with commotio cordis.[11]

Chest Protector Protocol

The current investigation assessed the outcome of 4 sequential and iterative series of experiments grouped around sets of chest protectors. In the first series, 4 different material combinations were compared with impacts without chest protectors. In the second series, 3 different material combinations were compared with impacts without chest protectors. In the third series, 3 chest protector combinations were compared with impacts without chest protectors. And in the final series, 2 different materials were compared with impacts without chest protectors. Within each series the protocol was similar. The order of impacts with the individual material combinations and a control impact (impact without a chest protector) were randomized. Impacts were then given with at least a 2-minute window between impacts. After all material combinations and a control impact were completed, the materials and control impact were randomized again and impacts were then repeated. Up to 20 impacts were given in a single animal.

Materials Tested

Materials included 3 different foams (Accelleron [Unequal Technologies, Glen Mills, PA], closed cell high density foam; Airilon [Unequal Technologies, Glen Mills, PA] closed cell lighter density soft foam; and open cell memory foam), TriDur (Unequal Technologies, Glen Mills, PA), a layer of flexible elastomeric coated aramid and a multilayer semirigid polypropylene polymer (ImpacShield, Unequal Technologies, Glen Mills, PA) (Table) (Figures 1–4). All chest protectors were cut to a 4-inch by 4-inch square so as to be placed securely against the animal's torso.

Endpoints and Statistical Analysis

The primary endpoint was the incidence of VF with chest wall strikes. Secondary endpoints included a combined endpoint of VF and nonsustained VF, ST segment elevation, and peak LV pressure and LV dP/dT produced by the ball impact. Generalized estimating equations were used to estimate adjusted mean values and corresponding SEs for binary and continuous outcomes (using binomial and normal distributions, respectively), accounting for the multiple impacts and outcome evaluations per animal. Pairwise comparisons between the control group and each vest were performed, and adjusted *P* values from a step-down Dunnett test were used to control for multiple testing. These analyses were done with and without including animal weight as a covariate in the analysis. All analyses were performed using SAS for Windows, SAS 9.4 TS Level 1M1 Copyright 2002 to 2012 by SAS Institute Inc., Cary, North California.

RESULTS

Impacts without chest protectors caused VF in 43 of 80 impacts (54%). Four chest protectors (numbers 6, 7, 8, and 9) significantly decreased the incidence of VF with ball impacts, including number 9 (21-mm thickness), which reduced the incidence of VF down to 5% (Figure 5). Chest protectors 6 (19-mm thickness), 7 (12-mm thickness), and 8 (21-mm thickness) reduced the VF incidence to 8%, 20%, and 20%, respectively. All 4 chest protectors contained the same

TABLE. Composition of the Various Chest Protectors

Chest Protector	Accelleron, Closed Cell High Density Foam	Airilon, Closed Cell Low Density Soft Foam	Open Cell Memory Foam	TriDur	ImpacShield, Multilayer Semirigid Polypropylene Polymer	No of Impacts	No of VF	VF (%)
Control						80	43	54
1	6			0.35	0.33	33	11	33
3	6			0.35		33	18	54
6	10	8		0.35	0.33	25	2	8
7	6	6		0.35	0.33	25	5	20
8	10	10		0.35	0.33	20	4	20
9	10	10		0.35	0.67	20	1	5
10			12	0.35		20	10	50
11			12	0.35	0.33	15	9	60
12			12	0.35		15	6	40
13	6		8	0.35	0.33	15	9	60
14	10		10	0.35	0.33	12	4	33
15	10		10	0.35	0.67	12	5	42

Thicknesses of the different layers are measured in millimeter.

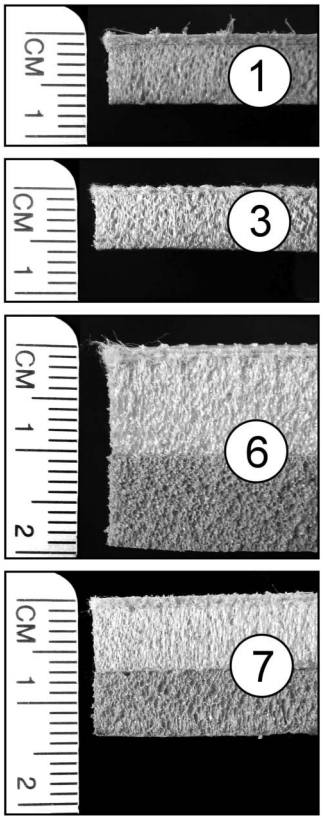

FIGURE 1. The first series of materials consisted of combinations of Accelleron, Airilon, Tridur and ImpacShield. Total thickness varied from 7 mm to 19 mm. Chest protector number 6 was the most efficacious in this series; only 8% of impacts caused ventricular fibrillation with this chest protector.

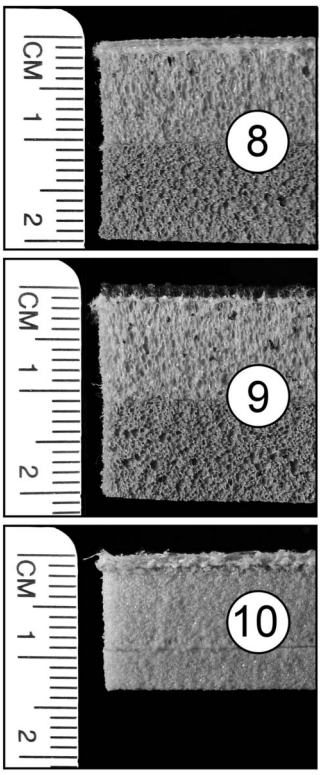

FIGURE 2. The second series of materials consisted of combinations of Accelleron, Airilon, Tridur and ImpacShield (numbers 8 and 9). Open celled memory foam (number 10) was also tested. Total thickness varied from 13 mm to 21 mm. Chest protector number 9 was the most efficacious in this series; only 5% of impacts caused ventricular fibrillation with this chest protector.

materials, but of different thicknesses. After adjustment for animal weight, only three chest protectors (numbers 6, 8, and 9) remained significant. Eight chest protectors did not lower the incidence of VF with ball impact.

For the combined endpoint of VF or nonsustained VF, the same 4 chest protectors (numbers 6, 7, 8, and 9) reduced this endpoint. All 4 remained significant after adjustment for weight. Again, chest protector 9 had the lowest incidence of 5%, compared with control impacts of 54%. In those impacts without VF, the magnitude of ST elevations was reduced for chest protectors 3, 6, and 9. The mean peak LV pressure induced by ball impact in impacts without chest protectors was 546 mm Hg. All chest protectors except numbers 1 and 3 lowered the peak LV pressure induced by the impact to 398 to 490 mm Hg (unadjusted P values from <0.0001 to 0.04). After adjustment for weight for chest protectors 6 to 8 and 11 to 15, the reduction remained significant. In control impacts, the change in pressure over time (dP/dT) was 365. All chest protectors except 1, 3, and 10 significantly reduced the dP/dT. After adjustment for weight, only chest protectors 9 and 11 to 15 remained significant.

DISCUSSION

The current experiment demonstrates that in an experimental model of commotio cordis, a more effective chest protector can be developed. The maximal tested thickness combination of Accelleron (high density elastomer) foam, Airilon (low density elastomer) foam, TriDur (flexible elastomer coated aramid), and ImpacShield (polypropylene polymer) reduced the incidence of VF from 54% to 5%. Two other thinner combinations of the same materials statistically reduced VF compared with no chest protector. However, not all new materials were effective in preventing commotio. In the current protocol, 9 protectors did not significantly reduce VF. These observations should be considered as chest wall protectors are developed for use on the athletic field. Chest protector equipment using materials efficacious in our experimental laboratory should reduce the risk of commotio cordis for our young athletes.

The successful chest protectors comprised 4 distinct layers (coated aramid, semirigid polypropylene polymer, high and low density elastomers). The outermost layer was a semirigid polypropylene polymer (of 0.35 and 0.67-mm thicknesses). In theory, this stiff layer both blocks and redirects energy laterally. The second layer is a coated TriDur, a flexible elastomeric coated aramid that absorbs and disperses high impact vibrational energy across the elongation of its fibers. The third layer is a closed cell, high density elastomeric foam that virtually maintains its structure

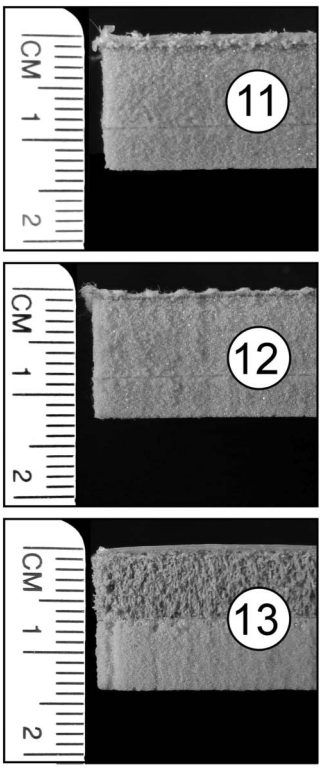

FIGURE 3. The third series of materials consisted of combinations of Accelleron, Tridur, open cell memory foam and ImpacShield. Total thickness varied from 13 mm to 14 mm. Chest protectors in this series did not decrease the risk of ventricular fibrillation.

and does not "bottom out." Finally, the fourth layer is a closed cell, low density soft elastomeric foam that likely attenuates most of the remaining energy.

In humans who have had clinical commotio cordis, as many as one-third were wearing chest protectors at the

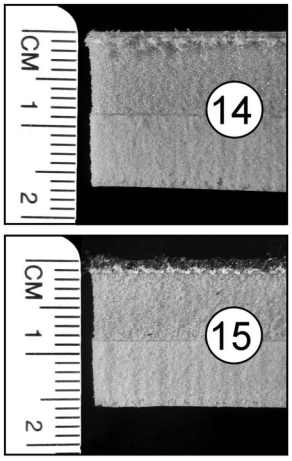

FIGURE 4. The fourth series of materials consisted of combinations of Accelleron, Tridur, open cell memory foam and ImpacShield. Total thickness varied from 16 mm to 17 mm. Chest protectors in this series did not decrease the risk of ventricular fibrillation.

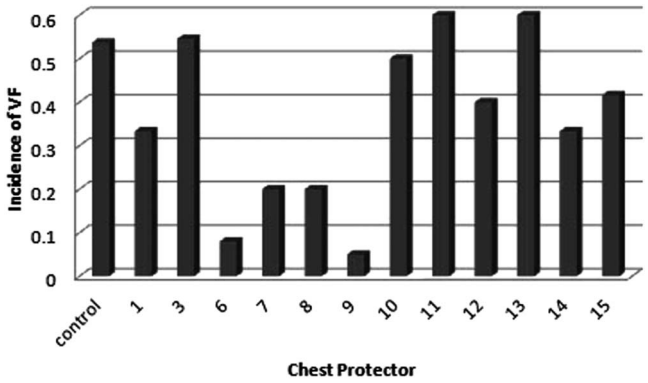

FIGURE 5. Incidence of ventricular fibrillation (VF) with ball impact to chest protectors assessed in the current experiment. Chest protectors 6, 7, 8 and 9 significantly reduced the risk of VF in the current experiments. After adjustment for weight, numbers 6, 8 and 9 remained significant.

time.[6] Although this human data alone do not prove that current chest protectors are inadequate in providing a margin of safety, it certainly raises concern about the efficacy of chest protectors to prevent commotio. It is notable that we have replicated this human scenario of the inadequacies of commercially available chest protectors in our experimental laboratory. Lacrosse and baseball chest protectors, available in 2002, did not reduce the incidence of VF in our experimental model.[7] Eight chest protectors marketed for baseball and 6 marketed for lacrosse were tested and none were found to significantly reduce the risk of VF with chest impact. Ventricular fibrillation occurred in a range of 22% to 49% in these chest protector impacts compared with impacts without chest protectors in which 32% caused VF. More recent chest protectors do not seem any safer. Commercially available chest protectors, available in 2012 and tested under the same conditions in our laboratory, fared no better (data not yet published). These chest protectors had an incidence of VF with ball strikes from 40% to 56%. Thus, we believe that our current data demonstrating a viable product, which reduces the risk of commotio cordis, can be extended to humans.

Critical to the development of an effective chest protector is the practical issue of thickness and comfort. Commercially available chest wall protectors that are deemed acceptable for use by sports participants range from 10 to 29 mm in thickness.[7] Chest protectors assessed in the current experiment ranged from 7 to 21 mm, well within the aforementioned range. Thus, introduction of these more effective chest protectors should not compromise sports participants' comfort.

A concern has been raised regarding the 40-mph impacts in this model in which the highest incidence of VF is seen.[11] If a chest protector decreases a 90-mph ball to the energy of a 40-mph ball, then, in theory, the ball could then be more likely to cause VF. Yet we feel that in our experimental model of 15 to 25 kg swine, 40-mph impacts are the ideal velocities to assess chest protectors. With 50-mph impacts, severe cardiac damage, including myocardial

rupture, valvular rupture, and cardiac tamponade, are observed.[11] Thus, 40-mph impacts in our model are the maximal energy that produces clinical commotio cordis. Since myocardial rupture, valvular rupture, and cardiac tamponade are not seen in sports, we feel that the ability of a chest protector to markedly reduce VF compared to control impacts in our experimental model will correlate with reduced risk in youth sports.

LIMITATIONS

There may be species differences in the risk of VF with chest impacts. Swine may be more susceptible than humans. Experimental animal models are not always directly applicable to humans. However, in our model, the control impacts were in the same animals that had chest protectors. Thus, even if swine are more susceptible, the *relative* reduction in VF should translate to humans. The materials tested in our laboratory were 4 in squares; larger diameter materials may provide increased protection from chest wall impact.

CONCLUSIONS

Effective protection against VF with chest wall protection of modest thickness can be achieved in an animal model of commotio cordis. It is reasonable to expect that chest protector designs incorporating these novel materials will be effective in the prevention of commotio cordis on the playing field.

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