

Protecting Children Against Commotio Cordis in Baseball by Understanding the Effects of Impact Locations Over the Heart

G. J. Dickey¹, K. Bian¹, H. R. Khan² and H. Mao^{1,3}

¹ Department of Mechanical and Materials Engineering, University of Western Ontario

² Department of Cardiology, London Health Sciences Center, University of Western Ontario

³ Department of Biomedical Engineering, University of Western Ontario

ABSTRACT

Commotio Cordis is the second leading cause of sudden cardiac death in young athletes. Currently available chest protectors on the market are shown ineffective in preventing cases of commotio cordis in young athletes that play baseball. The new NOCSAE (National Operating Committee on Standards for Athletic Equipment) regulations specify force limits at three loading cells over the chest, with one upper and one lower loading cell, and one over the heart. This study focused on understanding the effects of baseball impact locations to the heart. By understanding such effects and then identifying vulnerable impact locations, we may design and develop chest protectors that can effectively provide protection to prevent commotio cordis in young athletes. Simulation cases were run using the validated CHARM-10 model, a detailed finite element model representing an average 10-year-old child. A baseball model was developed and then used to impact the chest at different locations. An 8x7 impact location matrix was designed with 56 unique simulations. Eight locations in the transverse plane, seven locations in the sagittal plane. The baseball was moved by half of the ball's radius (18.75 mm) in the transverse and/or sagittal plane in each individual case. An initial velocity of 17.88 m/s was used for baseball impacts. The baseball was of regulation stiffness. Left ventricle strain and pressure, contact force between the baseball and chest and rib deformations were analyzed. Left ventricle strain was highest from baseball impacts directly over the left ventricle (0.34) as well as impacts slightly lateral and superior to the cardiac silhouette (0.34). Left ventricle pressure was highest with impacts directly over the left ventricle (82.9 kPa). For impacts close to the upper and lower positions as specified by NOCSAE, left ventricle strain and pressure were 0.24 and 29.5 kPa (upper loading cell) and 0.04 and 4.0 kPa (lower loading cell). This study systematically analyzed the effects of impact locations on left ventricle response. We have identified the most dangerous impact locations not only to the left ventricle but also to the upper, left side of the heart. Impacts to the NOCSAE lower-loading-cell position was of minimal concern in inducing left ventricle strain or pressure. This novel study provided evidence through computational modeling of where to emphasize protective materials for establishing effective chest protectors that will minimize instances of commotio cordis in young baseball athletes.

INTRODUCTION

Commotio cordis is a rare but lethal mechanism that is the second leading cause of sudden cardiac death in young athletes (Maron, 2003). Upwards of 75% of cases of commotio cordis occur in competitive and recreational youth sports (Maron & Estes, 2010). It is the result of the heart going into ventricular fibrillation from a non-penetrating impact such as a baseball over the chest (Link, 2012; Link et al., 1998; Maron & Estes, 2010). To induce commotio cordis, a combination of 3 specific factors must occur simultaneously: the impact must occur over the precordium, the impact must have a velocity of approximately 40 mph, and the impact must occur during the upslope of the T-wave during the cardiac cycle (Link et al., 1998). As of 2010, the commotio cordis registry has recorded 224 deaths since its inauguration in 1995 (Maron et al., 2009; Maron et al., 2002; Maron et al., 1995).

Despite the use of chest protectors in youth sports, sudden death in young athletes from impacts over the chest still occur (Doerer et al., 2007). Current chest protector brands on the market claim their chest protectors are safe for children, but these companies may give a false sense of safety for parents of young children. An experimental animal model commonly discussed in the literature analyzed popular baseball and lacrosse chest protectors and their ability to decrease ventricular fibrillation occurrence when compared to a control group (no chest protector). They found that none of the baseball chest protectors were able to significantly reduce the rate of ventricular fibrillation, with the frequency of ventricular fibrillation ranging between a low of 22% and a high of 49% (Weinstock et al., 2006). Furthermore, another study found that approximately 40% of commotio cordis instances occurred across various sports despite the athlete wearing a commercially available chest protector (Doerer et al., 2007).

As of 2017, NOCSAE (National Operating Committee on Standards for Athletic Equipment) introduced safety standards for commotio cordis prevention in baseball and lacrosse chest protectors (NOCSAE, Revised July, 2019). These standards are a significant step in developing safer chest protector designs for young athletes. Currently, the NOCSAE surrogate model uses an upper loading cell (ULC), lower loading cell (LLC) and a cardiac loading cell (CLC) to measure the amount of force (N) impacting the surrogate model. Reaction force is the only parameter that NOCSAE has chosen to determine whether a chest protector is able to prevent instances of commotio cordis. Mechanical responses of the heart remain unknown using this criterion for evaluation.

Commotio cordis is difficult to study due to the nature of the incident, and challenges arise due to the inability to test on live subjects or PMHSs. To date, predominately swine studies have been conducted in the literature (Link et al., 2001; Link et al., 2002; Link et al., 1998). Finite element modeling can help us to understand this sudden-death mechanism through the use of a validated finite element child model. In particular, it may allow us to analyze the left ventricle of the heart and any deformations that occur upon impact. This analysis is a much-needed alternative to the conventional swine studies, as the chest in swine is rounded and therefore impacts during testing make contact directly with the left ventricle. Meanwhile, in humans the heart is positioned so that the right ventricle is most superficial to the chest. These anatomical differences may play a key role in the results from these swine studies.

The current void in the literature, alongside the ineffectiveness of current chest protectors, inspired us to research and explore ways to identify the most dangerous impact locations over the chest wall. This study focused on using contour maps to identify these impact locations and create a visual representation that is legible to everyone. By identifying the most threatening impact locations, we may design and develop a chest protector that would provide maximum protection without sacrificing mobility and comfort.

METHODS

Finite element simulations and post processing

Finite element simulations were run using the CHARM-10 child model developed at Wayne State University (Shen et al., 2016), a detailed finite element (FE) model representing an average 10-year-old child. This detailed FE model contains all major anatomical structures based on detailed clinical scans of 10-year-old children (Mao et al., 2014) including 12 pairs of ribs, scapula, sternum, clavicle, humerus, the entire spinal column, cartilage and ligaments, all internal organs and major arteries (e.g., aorta). The model consists of 742,087 elements and 504,775 nodes and contains 8-node hexahedral elements. The model was developed with a multi-block approach; selectively reduced integration was used with hourglass control type 4 and a parameter of 0.1 for soft tissue.

The CHARM-10 model has been validated based on data collected through cardiopulmonary resuscitation on live subjects (Jiang et al., 2014) and impact data gathered from cadavers (Jiang et al., 2013). We placed an internal pressure of 9.3 kPa to the heart to replicate blood pressure. A baseball model with a radius of 37.5 mm and material properties based on the literature was developed to be used for impacts with the validated chest model (Vedula, 2004). All impacts to the chest were given an initial velocity of 17.88 m/s (40 mph). Previous research has shown that impact speeds any higher than this may cause severe cardiac damage, which is not uniform with commotio cordis instances (Link et al., 2003). Simulations were run using the Ls-Dyna solver. Once completed, simulations were analyzed and data was collected using Ls-PrePost2.4.

Matrix design

An 8x7 impact location matrix was designed with 56 unique simulations. Eight locations in the transverse plane, seven locations in the sagittal plane, and an initial baseball velocity of 17.88 m/s aimed towards the chest. The baseball moved by half of the ball's radius (18.75 mm) in the transverse and/or sagittal plane in each individual case.

Impact response

Impact responses were analyzed using the CHARM-10 computational model on Ls-PrePost2.4. Impact responses included: Left ventricle strain and pressure, reaction force between

the baseball and chest, max rib deformation, and rib deformation at the left ventricle which includes rib 3 (ULV), rib 4 (MLV) and rib 5 (LLV).

Left ventricle strain and pressure were analyzed and calculated by selecting all of the elements that make up the left ventricle. No filter was needed when analyzing strain; meanwhile, a CFC 600 filter was used when calculating left ventricle pressure.

Reaction force was measured by analyzing the force between the baseball and chest. This was calculated using *Contact_Automatic_Surface_to_Surface in Ls-Dyna, which is a penalty-based contact. A CFC 1000 filter was used to collect peak values of force from impacts.

Max rib deformation was calculated by measuring the displacement (mm) between the anterior rib that experienced direct contact from the baseball and the rib that sits directly posterior to it (e.g., anterior rib 3, posterior rib 5).

Contour maps

Contour maps were designed on Microsoft Excel. The data was collected and then input into an 8x7 matrix where it was color coded with a green-yellow-red color scale.

RESULTS

Left ventricle mechanical responses

Left ventricle strain was highest from impacts directly over the left ventricle and impacts slightly lateral and superior to the cardiac silhouette (0.34) (Figure 1. A). Left ventricle pressure (kPa) was highest when impacts were aimed directly over the left ventricle, reaching a peak internal pressure of 82.94 kPa (Figure 1. B).

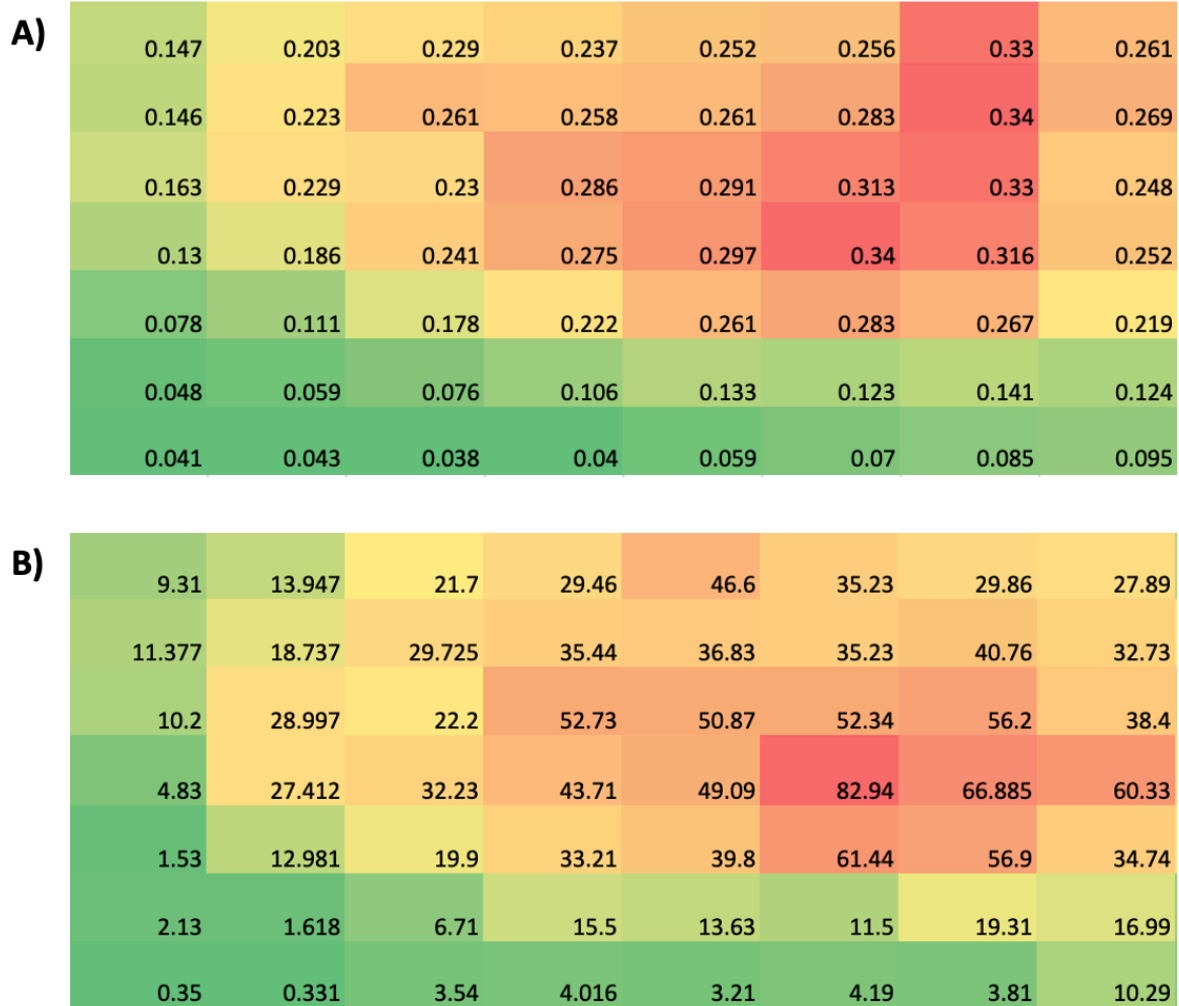


Figure 1. A) Left ventricle strain. Left ventricle strain ranges from 0.041 (green) to 0.34 (red).

B) Left ventricle pressure. Left ventricle pressure ranges from 0.35 kPa (green) to 82.94 kPa (red).

Chest external mechanical responses

Reaction force between the baseball and chest had no visible correlation between impact location and reaction force. Reaction force was highest when impacts were inferior to the sternum (1.1 kN) and lowest over some areas directly over the heart (0.86 kN) (Figure 2. A). Max rib deformation was highest with impacts directly over the left ventricle (36.2 mm) and slightly inferior (Figure 2. B).

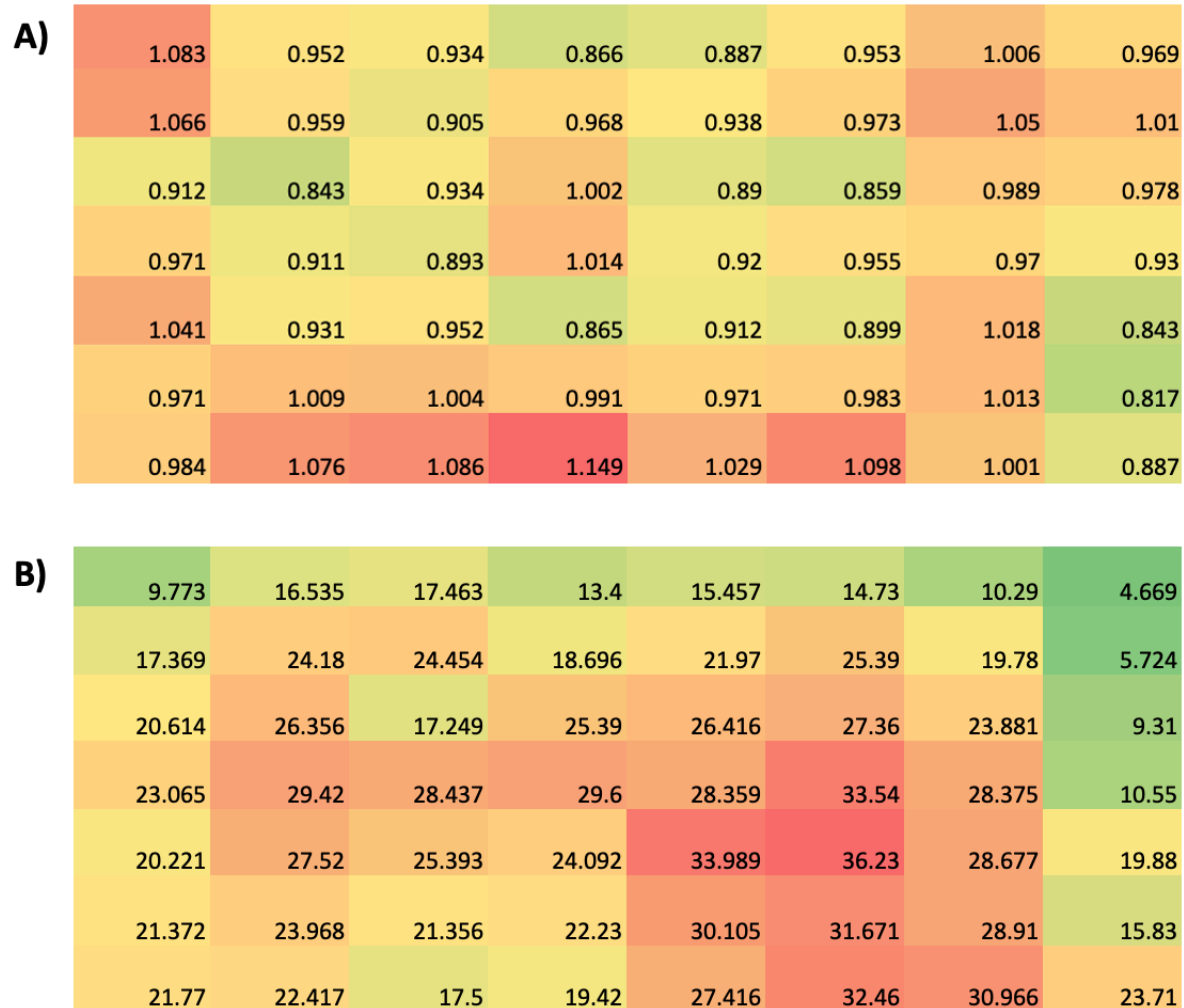


Figure 2. A) Reaction force between the baseball and chest. Reaction force ranges from 0.817 kN (green) to 1.149 kN (red). B) Max rib deformation ranged from 4.669 mm (green) to 36.23 mm (red).

Rib deformations at the left ventricle

ULV, MLV and LLV displayed large rib deformations when impacts were aimed directly over their respective rib. LLV had the highest rib deformations at 35.5 mm (Figure 3. A), while MLV had a peak deformation of 33.0 mm (Figure 3. B), and ULV at 26.9 mm (Figure 3. C).

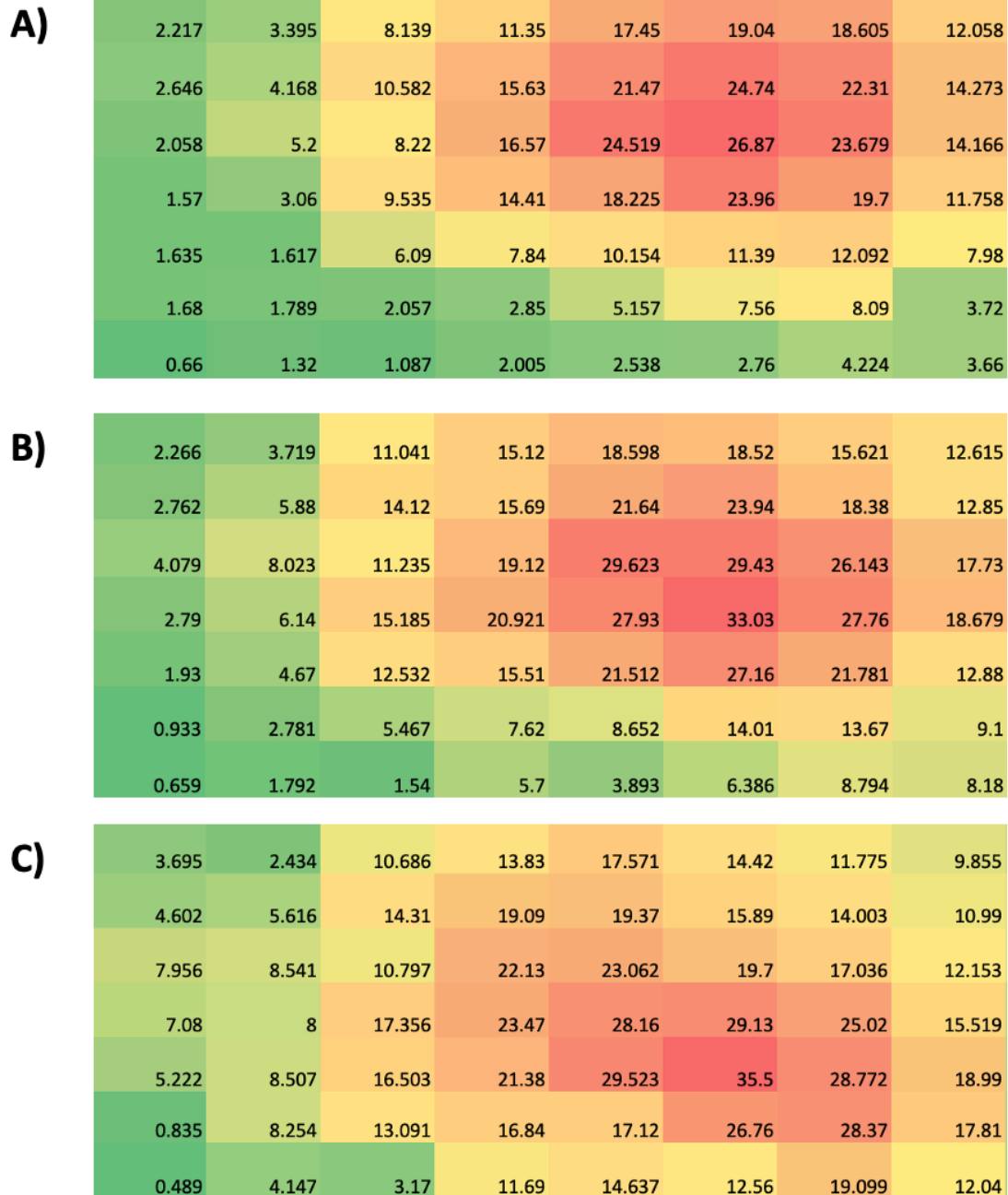


Figure 3. A) Rib deformation at ULV with a peak deformation of 26.87 mm (red). B) Rib deformation at the MLV with a peak deformation of 33.03 mm (red). C) Rib deformation at the LLV with a peak deformation of 35.5 mm (red).

DISCUSSION

This study used a detailed finite element model to analyze baseball impacts to the chest and understand the effect of impact locations over the heart with respect to left ventricle strain, left ventricle pressure, reaction force, max rib deformation, ULV, MLV and LLV. The left ventricle strain and pressure contour maps illustrate where to place more emphasis on chest protectors and may allow future researchers and baseball chest protector manufacturers to design safer products. Meanwhile, other contour maps, such as reaction force between the baseball and the chest illustrate why we believe reaction force is not the most effective commotio cordis injury metric, whereas contour maps of rib deformation over the left ventricle region support our recommendation of using it as an effective injury metric.

We have found that impacts aimed slightly lateral and superior to the left ventricle caused very high left ventricle strain and require more intensive protection in these regions. After careful examination of these cases producing higher strain, it was concluded that the baseball impacts the chest wall and produces a contact force on the rib cage, specifically ribs 3 (ULV) and 4 (MLV), increasing left ventricle strain as a result. Due to the chest cavity not being completely flat, this caused the baseball to have a unique impact on the ribs, resulting in the baseball creating a downward force pushing into the left ventricle.

Researchers in this field are commended for exploring new materials and thickness levels to be incorporated into the design of their commercially available chest protectors (Kumar et al., 2017). Meanwhile, based on our results, we believe that past and current designs may have overlooked areas that require more protection in order to mitigate incidences of commotio cordis. We believe that the combination of studies in which materials and thickness levels are analyzed, alongside our study determining where extra protection is needed, we can develop a chest protector capable of reducing instances of ventricular fibrillation.

Currently, the NOCSAE chest protector testing requires the chest protector to endure a 30-mph (13.41 m/s) and 50-mph (22.35 m/s) condition over the chest. For 30-mph conditions, the CLC must not exceed 400 N of force, while the ULC or LLC must not exceed 498 N of force. In the 50-mph condition, the CLC must not exceed 800 N of force, and the ULC and LLC shall not exceed 1001 N of force. Our data suggests that this model may be missing a potentially critical area superior and to the left of the cardiac silhouette (Figure 1. A). For impacts close the ULC and LLC, left ventricle strain was 0.24 (ULC) and 0.04 (LLC), while left ventricle pressure was 29.5 kPa (ULC) and 4.0 kPa (LLC). These findings suggest that impacts to the NOCSAE LLC location are of minor concern in terms of inducing left ventricle strain and pressure. Considering this surrogate model and our results, we believe the addition of the area we have identified in this study may improve the protective effect of a chest protector.

A limitation to our study is that the CHARM-10 finite element model does not contain blood flow throughout the heart. Due to our data being collected in 20 ms we do not believe that this limitation would influence our results. However, to compensate for this limitation, we placed an internal pressure of 9.3 kPa to the heart to replicate blood pressure.

CONCLUSIONS

This novel study used finite element modelling to help analyze the effects of baseball impacts locations over the heart in order to reduce the incidence of commotio cordis in young baseball athletes. Our results have identified the most dangerous baseball impact locations regarding left ventricle strain and pressure. Strain was highest when impacts were located directly over the left ventricle, as well as impacts slightly lateral and superior to the cardiac silhouette. Left ventricle pressure was highest with impacts directly over the left ventricle. Additionally, we addressed the reaction force between the baseball and chest wall, max rib deformation, and rib deformations at the left ventricle. Reaction force did not have a strong correlation with impact location, showing peak impact force when the impact was situated inferior to the sternum. Max rib deformation showed the largest deformations when impacts were placed directly over the left ventricle and impacts inferior to that position. Moreover, rib deformations of the ULV, MLV and LLV were highest when impacts were directly over the corresponding rib, as expected. Overall, we have described a computational approach that helps analyze the effects of baseball impact locations over the heart and provides unique evidence that identifies where to emphasize protective materials for establishing effective chest protectors that will minimize the risk of commotio cordis.

ACKNOWLEDGEMENTS

We acknowledge the support of NSERC and the Canada Research Chairs Program.

REFERENCES

- Doerer, J. J., Haas, T. S., Estes, N. A., Link, M. S., & Maron, B. J. (2007). Evaluation of chest barriers for protection against sudden death due to commotio cordis. *Am J Cardiol*, 99(6), 857-859. <https://doi.org/10.1016/j.amjcard.2006.10.053>
- Jiang, B., Mao, H., Cao, L., & Yang, K. (2013). Experimental validation of pediatric thorax finite element model under dynamic loading condition and analysis of injury. *SAE Technical Paper*, 2013-01-0456. <https://doi.org/https://doi.org/10.4271/2013-01-0456>
- Jiang, B., Mao, H., Cao, L., & Yang, K. (2014). Application of an anatomically-detailed finite element thorax model to investigate pediatric cardiopulmonary resuscitation techniques on hard bed. *Computers in Biology and Medicine*, 52, 28-34. <https://doi.org/10.1016/j.compbiomed.2014.05.014>
- Kumar, K., Mandleywala, S. N., Gannon, M. P., Estes, N. A., Weinstock, J., & Link, M. S. (2017). Development of a Chest Wall Protector Effective in Preventing Sudden Cardiac Death by Chest Wall Impact (Commotio Cordis). *Clin J Sport Med*, 27(1), 26-30. <https://doi.org/10.1097/JSM.0000000000000297>
- Link, M. S. (2012). Commotio cordis: ventricular fibrillation triggered by chest impact-induced abnormalities in repolarization. *Circulation: Arrhythmia and Electrophysiology*, 5(2), 425-432. <https://doi.org/10.1161/CIRCEP.111.962712>
- Link, M. S., Maron, B. J., VanderBrink, B. A., Takeuchi, M., Pandian, N. G., Wang, P. J., & Estes, N. M. (2001). Impact directly over the cardiac silhouette is necessary to produce ventricular fibrillation in an experimental model of commotio cordis. *Journal of the American College of Cardiology*, 37(2), 649-654. [https://doi.org/10.1016/s0735-1097\(00\)01142-6](https://doi.org/10.1016/s0735-1097(00)01142-6)
- Link, M. S., Maron, B. J., Wang, P. J., Pandian, N. G., VanderBrink, B. A., & Estes, N. M. (2002). Reduced risk of sudden death from chest wall blows (commotio cordis) with safety baseballs. *Pediatrics*, 109(5), 873-877.
- Link, M. S., Maron, B. J., Wang, P. J., VanderBrink, B. A., Zhu, W., & Estes, N. M. (2003). Upper and lower limits of vulnerability to sudden arrhythmic death with chest-wall impact (commotio cordis). *Journal of the American College of Cardiology*, 41(1), 99-104. [https://doi.org/https://doi.org/10.1016/s0735-1097\(02\)02669-4](https://doi.org/https://doi.org/10.1016/s0735-1097(02)02669-4)
- Link, M. S., Wang, P. J., Pandian, N. G., Bharati, S., Udelson, J. E., Lee, M.-Y., Vecchiotti, M. A., VanderBrink, B. A., Mirra, G., & Maron, B. J. (1998). An experimental model of sudden death due to low-energy chest-wall impact (commotio cordis). *New England Journal of Medicine*, 338(25), 1805-1811.
- Mao, H., Holcombe, S., Shen, M., Jin, X., Wagner, C. D., Wang, S. C., Yang, K. H., & King, A. I. (2014). Development of a 10-year-old full body geometric dataset for computational modeling. *Ann Biomed Eng*, 42(10), 2143-2155. <https://doi.org/10.1007/s10439-014-1078-5>
- Maron, B. J. (2003). Sudden death in young athletes. *N Engl J Med*, 349(11), 1064-1075. <https://doi.org/10.1056/NEJMra022783>
- Maron, B. J., Doerer, J. J., Haas, T. S., Estes, N. A., Hodges, J. S., & Link, M. S. (2009). Commotio cordis and the epidemiology of sudden death in competitive lacrosse. *Pediatrics*, 124(3), 966-971. <https://doi.org/10.1542/peds.2009-0167>

- Maron, B. J., & Estes, N. A. (2010). Commotio cordis. *N Engl J Med*, 362(10), 917-927. <https://doi.org/10.1056/NEJMra0910111>
- Maron, B. J., Gohman, T. E., Kyle, S. B., Estes, N. A., & Link, M. S. (2002). Clinical profile and spectrum of commotio cordis. *JAMA*, 287(9), 1142-1146. <https://doi.org/10.1001/jama.287.9.1142>
- Maron, B. J., Poliac, L. C., Kaplan, J. A., & Mueller, F. O. (1995). Blunt impact to the chest leading to sudden death from cardiac arrest during sports activities. *New England Journal of Medicine*, 333(6), 337-342. <https://doi.org/https://doi.org/10.1056/NEJM199508103330602>
- NOCSAE. (Revised July, 2019). *Standard Test Method and Performance Specification Used in Evaluating the Performance Characteristics of Protectors for Commotio Cordis*. N. O. C. O. S. F. A. Equipment. <https://nocsae.org/wp-content/uploads/2018/05/ND200-19-Commotio-Cordis-Test-Method-002-1.pdf>
- Shen, M., Mao, H., Jiang, B., Zhu, F., Jin, X., Dong, L., Ham, S., Palaniappan, P., Chou, C., & Yang, K. (2016). Introduction of two new paediatric finite element models for pedestrian and occupant protections., *SAE Technical Paper*, 2016-01-1492, 10. <https://doi.org/https://doi.org/10.4271/2016-01-1492>
- Vedula, G. (2004). *Experimental and finite element study of the design parameters of an aluminum baseball bat* [Doctoral dissertation, University of Massachusetts Lowell].
- Weinstock, J., Maron, B. J., Song, C., Mane, P. P., Estes, N. M., & Link, M. S. (2006). Failure of commercially available chest wall protectors to prevent sudden cardiac death induced by chest wall blows in an experimental model of commotio cordis. *Pediatrics*, 117(4), e656-e662. <https://doi.org/10.1542/peds.2005-1270>