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Biomechanical Analysis of Commotio Cordis in Children's Baseball - A Computational Approach

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A thesis submitted in partial fulfillment of the requirements for the Master of Engineering Science degree in Mechanical and Materials Engineering

Abstract

Commotio cordis is the second leading cause of cardiac death in young athletes. This rare sudden-death mechanism most commonly affects young children playing baseball. It is caused by impacts to the chest during the repolarization phase of the cardiac cycle, which causes the heart to go into ventricular fibrillation, often leading to death. This study adopted the detailed and validated CHARM-10 child finite element model to replicate commotio cordis instances by simulating baseball impacts to the chest. New commotio cordis injury metrics were developed to create a more accurate prediction for chest protector effectiveness. The conventionally used injury metrics for chest protectors did not correlate strongly with left ventricle strain and pressure. This study also identified potentially vulnerable impact locations slightly lateral and above the heart that may be ignored by current commotio cordis safety regulations and commercially available baseball chest protectors.

Keywords

Commotio Cordis, finite element analysis, impact biomechanics, ventricular fibrillation, left ventricle strain, left ventricle pressure, heart, baseball

Summary for Lay Audience

Commotio cordis is the result of a small object impacting the chest at speeds of approximately 40 mph during a vulnerable period of the cardiac cycle. In commotio cordis instances, the heart goes into ventricular fibrillation which means the heart stops pumping blood out to the body, often leading to death. These impacts typically come in the form of a baseball, lacrosse ball, or hockey puck, although it can happen in a more casual setting such as children playing around in the backyard. Commotio cordis most commonly occurs in youth males which can be explained by the abundance of males who play contact sports, as well as the weak and narrower chest walls found in children when compared to the chest walls of adults.

Currently, swine models dominate the commotio cordis research field as testing on live subjects and cadavers is not possible. Our research lab wanted to take a novel approach in analyzing commotio cordis; we used finite element modeling to replicate commotio cordis instances by simulating baseball impacts to the chest. Our study started by developing new injury metrics for commotio cordis, while also analyzing current injury metrics used by NOCSAE for chest protectors on the market today. We then identified vulnerable impact locations over the chest when considering commotio cordis. Our results from this study helped us to illustrate where future chest protectors should place protective material to reduce left ventricle strain and pressure, and as a result, mitigate commotio cordis in children. This thesis work finished with preliminary studies of a circular pad design in which we analyzed its ability to reduce strain and pressure in the left ventricle of the heart. We also identified preliminary results for alternative positions that youth baseball players can turn their bodies to prevent impacts over the chest if they are unable to catch the ball in time.

Co-Authorship Statement

Chapter 2 (‘Developing Commotio Cordis Injury Metrics for Baseball Safety: Unravelling the Connection Between Chest Force and Rib Deformation to Left Ventricle Strain and Pressure’) was co-authored by Dr. Haojie Mao, Kewei Bian, and Habib R. Khan MD

Chapter 3 (‘Identifying Vulnerable Impact Locations to Reduce the Occurrence of Deadly Commotio Cordis Events in Children’s Baseball – A computational Approach’) was co-authored by Dr. Haojie Mao, Kewei Bian, Xingyu Liu, and Habib R. Khan MD

All manuscripts were drafted by Grant James Dickey and reviewed by Dr. Haojie Mao.

Acknowledgments

I would like to thank my supervisor Dr. Haojie Mao for his consistent support, and my advisory committee member Habib R. Khan MD for his assistance. I acknowledge Kewei Bian for his assistance in my research with computational modeling, and Xingyu Liu in assisting with MATLAB code development.

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List of Acronyms

VF: Ventricular fibrillation

ULV: Upper left ventricle

MLV: Middle left ventricle

LLV: Lower left ventricle

FE: Finite element

MPS: Maximum principal strain

Chapter 1

1 Introduction

1.1 Research Rationale

Commotio cordis is a rare but lethal mechanism that is the second leading cause of sudden cardiac death in young athletes [1]. Upwards of 75% of cases of commotio cordis occur in competitive and recreational youth sports [2]. It is the result of the heart going into ventricular fibrillation (VF) from a non-penetrating impact such as a baseball over the chest [2-4]. To induce commotio cordis, a combination of 3 specific scenarios must occur simultaneously: the impact must occur over the precordium, the impact speed must have a velocity of approximately 40 mph, and the impact must occur during the upslope of the T-wave during the cardiac cycle [4]. As of 2010, the commotio cordis registry has recorded 224 deaths since it's inauguration in 1995 [5-7].

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 cadavers. Current chest protectors on the market are not efficient in preventing instances of commotio cordis cases in children [8-11]. To date, predominately swine studies have been conducted in the literature [4, 12, 13]. This thesis involves the first use of computational modelling to analyze commotio cordis in children's baseball events.

1.2 Cardiac and Chest Wall Anatomy and Functions

1.2.1 Cardiac Anatomy and Function

The heart is made up of various parts, each of which plays an important role in the process of the heart taking in de-oxygenated blood, sending it to the lungs to be oxygenated, and then pumping the newly oxygenated blood out to the body to be used. Four chambers make up the heart: right atrium, right ventricle, left atrium and left ventricle (Figure 1.1). The right chambers are responsible for taking in de-oxygenated blood from the superior and inferior vena cava and helping to transfer it to the lungs, while the left chambers are responsible for distributing the newly oxygenated blood from the lungs to the aorta and out to the rest of body.

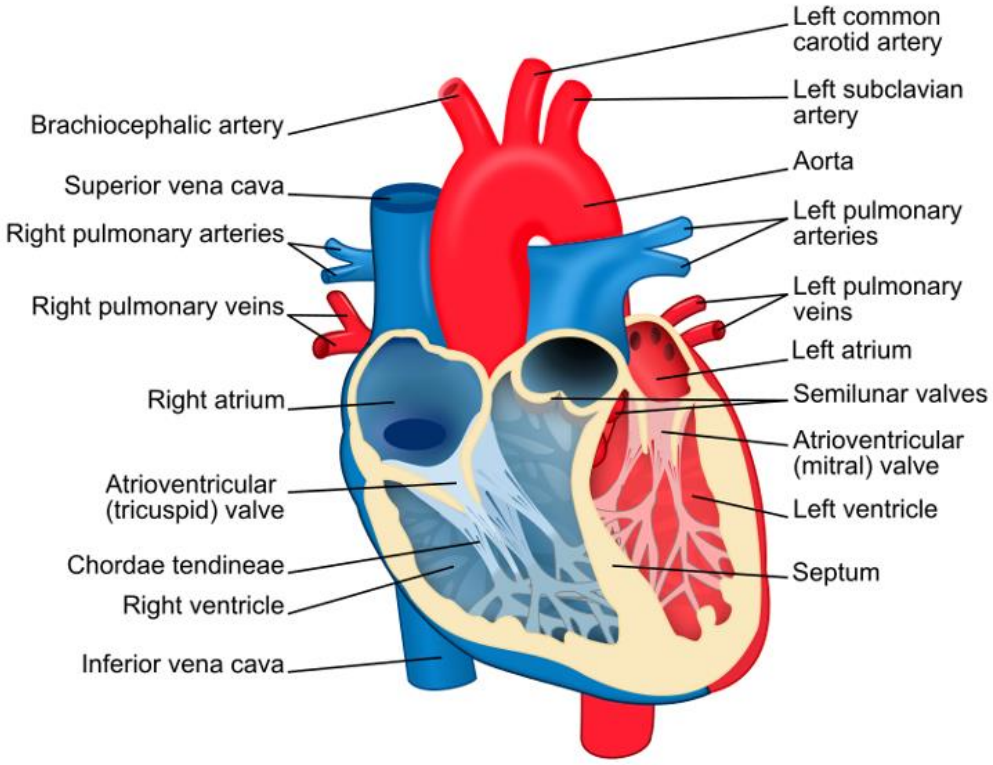

Figure 1.1 Sliced anatomical view of the heart labelled.(Courtesy of Wikimedia Commons) [14].

The cardiac cycle goes through a cyclic process in which it fluctuates through periods of systole (ventricular contraction) and diastole (ventricular relaxation). The Electrocardiogram (ECG) consists of a PQRST complex (Figure 1.2). Each alphabetical letter notions a specific action during the cardiac cycle that makes up the heartbeat. The P-wave indicates atrial depolarization, the QRS complex is responsible for ventricular depolarization, and the T-wave represents ventricular repolarization. Ventricular systole occurs during the period between the QRS complex and the end of the T-wave. Diastole occurs during the period between the end of the T-wave and the end of the PR interval.

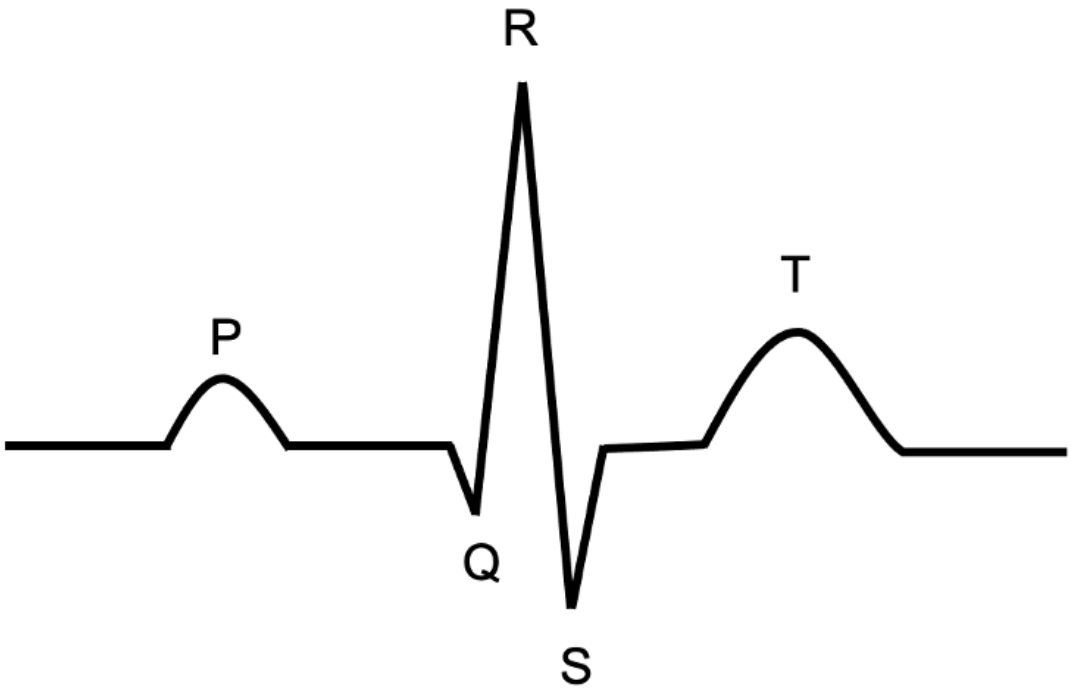

Figure 1.2 Electrocardiogram (ECG) containing the PQRST complex.

The cardiac cycle is split into separate phases that can be distinguished through systole and diastole and is commonly displayed via a Wiggers diagram (Figure 1.3). Systole phases include isovolumetric contraction, early and late ejection. In isovolumetric

contraction, the ventricles are contracting which increases the pressure in the ventricles; there is a fixed ventricular volume (130 mL) because all valves are closed during this phase. In the early ejection phase, the ventricles reach a high pressure (120 mmHg) that allows the aortic and pulmonary valves to open (the end of this phase signifies the beginning of the T-wave during the ECG). During late ejection, ventricular pressure begins to drop and the aortic and pulmonary valves close (end of this phase signifies peak of the T-wave during the ECG) [15].

Diastole phases include isovolumetric relaxation, rapid filling, slow filling, and atrial systole. Isovolumetric relaxation allows the ventricles to relax and the pressure to drop. On an ECG, this would be the end of the T-wave. During rapid inflow, the ventricular pressure is now lower than the atrial pressure, which allows them to fill rapidly with blood (end of the P-wave on an ECG). The rapid inflow slows down as the pressures in the ventricles and atriums become equal. During atrial systole, the right and left atrium contract which increases pressure in the ventricles. This process on an ECG starts at the P-wave, and finishes during the peak of the R-wave [15].

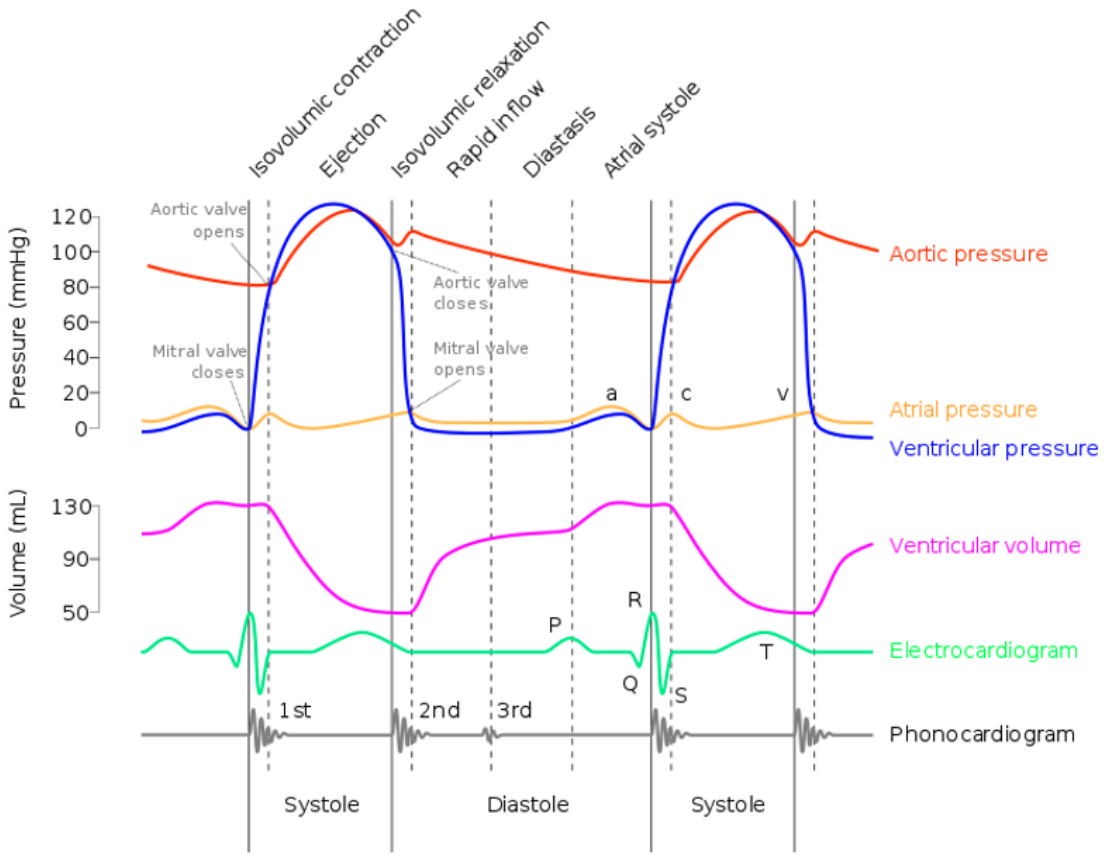

Figure 1.3 Wiggers Diagram (Courtesy of Wikimedia Commons) [16].

1.2.2 Thoracic Wall and Chest Anatomy and Function

The thoracic wall is made up of bones and muscles; the major bone landmarks are the thoracic spine, rib cage, and the sternum. The rib cage is made of 12 ribs per side and consists of true, false and floating ribs (Figure 1.4b). Ribs 1-7 are true ribs as they connect to the sternum, meanwhile, ribs 8-10 are false as they do not connect directly to the sternum and are shorter. Ribs 11-12 are considered floating ribs as they only connect to the spinal column [17]. The sternum is made up of 3 distinct parts: manubrium, body, and the xiphoid process (Figure 1.4a).

Alongside these bones, the major muscle landmarks include the external, internal and innermost intercostals, the subcostalis and the transversus thoracis (Figure 5). The pectoralis minor and major, subclavius and serratus all attach to the thoracic wall, but are not considered part of it [18].

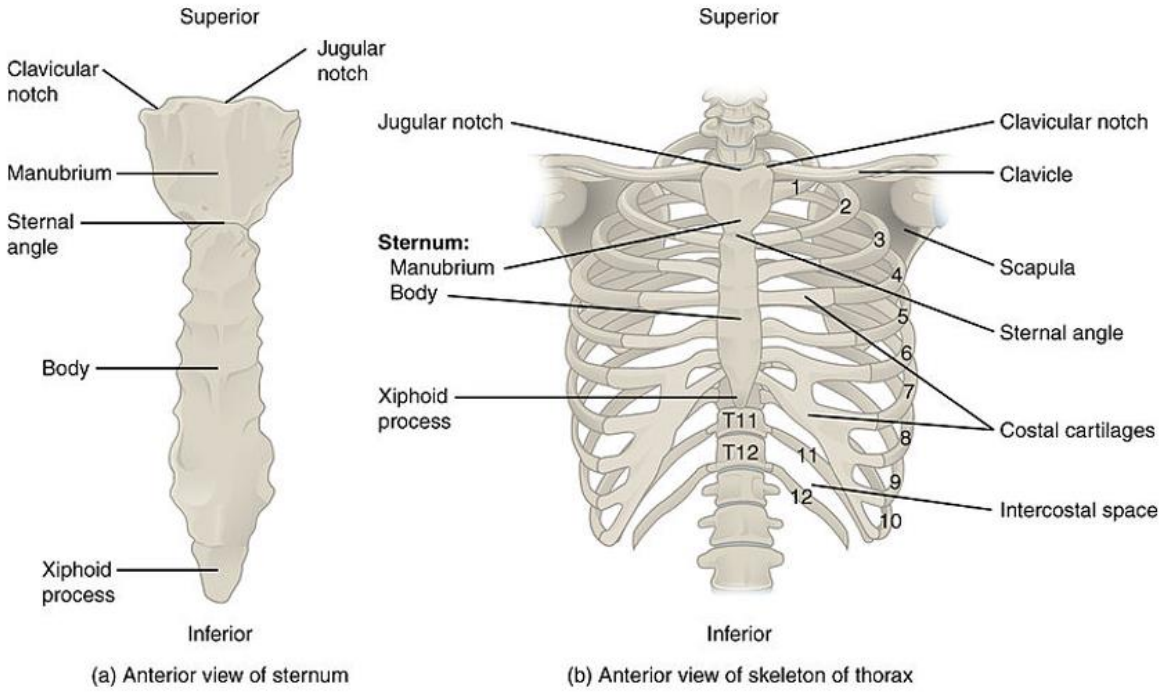

Figure 1.4 Anatomy of the rib cage and sternum(Courtesy of Wikimedia Commons) [19].

The thoracic wall's primary role is to protect the organs inside the body while also providing a foundation for muscles to connect to (Figure 1.5). The rib cage and thoracic cavity enclose and provide protection for the heart and the lungs, as well as allowing space for the mechanical functions of the respiratory cycle [20].

The intercostal muscles fill the gap between the rib cage, and provide a layer of protection for the organs [21]. The sternum is particularly important as it is positioned directly over the heart as a means of protection [17].

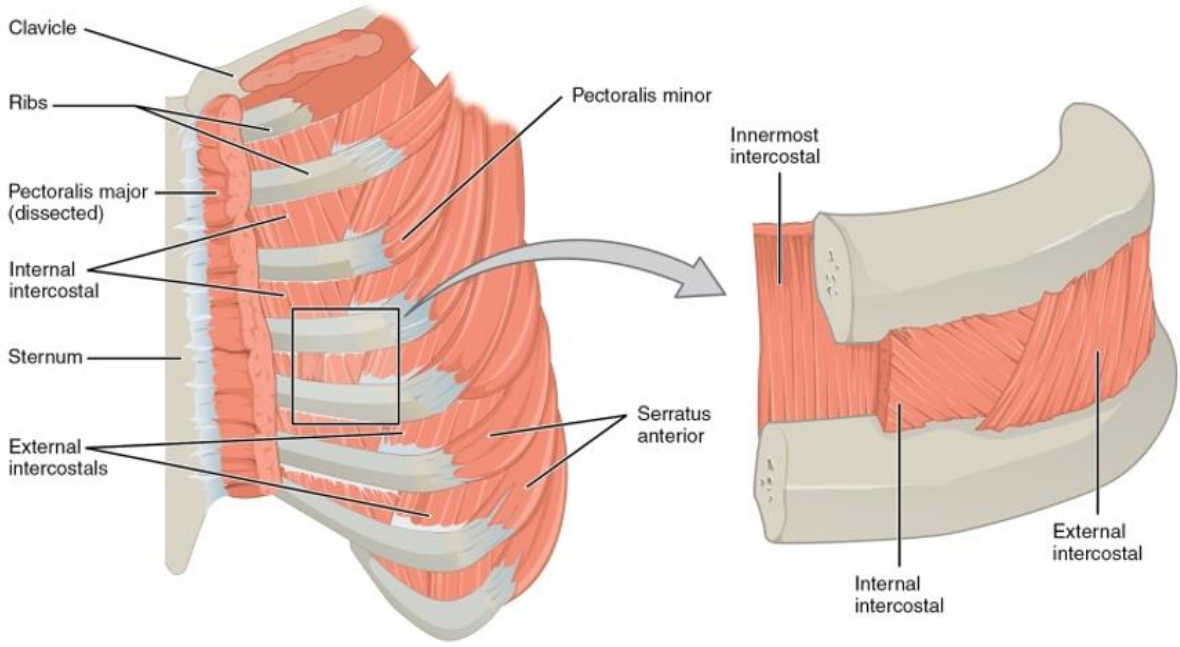

Figure 1.5 Muscles and bones of the thoracic walland the surrounding muscles that attach to it [22].

1.3 Commotio Cordis in Children's Sports

1.3.1 Sports Relevance

Commotio cordis cases have been reported in a variety of sports, but are most prominently reported in hockey, lacrosse, and baseball [7, 23-27]. These 3 sports all have one major thing in common: The sport involves a relatively small object travelling at speeds of approximately 40 mph (17.88 m/s) and the potential for impacts to make contact directly over the chest. Hockey and lacrosse involve the use of either a hockey stick, or lacrosse stick while baseball only involves a fast pitch.

Hockey has resulted in commotio cordis deaths most notably from pucks to the chest [25], although other deaths have occurred from impacts such as a hockey stick impacting a players chest during a routine hit [23]. Lacrosse uses a ball that is similar in shape to a baseball but consists purely of rubber. In an analysis of commotio cordis deaths in American lacrosse players, type of deaths from commotio cordis were similar to those found in hockey; the players were struck over the chest from the ball, or they were struck in the chest by an opponent's stick [7].

Baseball is the most common sport in which commotio cordis takes place [3]. Although all players on the field are susceptible to strikes over the chest from the baseball, the most at-risk players are the individual at bat and the catcher due to the ball being pitched towards them. Baseball catchers wear chest protectors, but players at bat typically do not.

1.3.2 Chest Protection

In both hockey and lacrosse, chest protection is mandatory for all players. Meanwhile, baseball only requires the catcher position to wear a chest protector while other players on the field are free to participate with no chest protection. Chest protectors vary in all 3 sports. Hockey goalies wear a much thicker, heavier, and full covering chest protector, whereas the players wear a thin and light shoulder/chest protector. In lacrosse the same is true, goalies typically wear large, thicker chest padding while the players wear little to no protection. The chest protector designs are not created with commotio cordis protection in mind, instead, they predominately focus on the prevention of external wounds that the player may experience during practice or a game. Novel chest protector designs have

come out in recent years that are targeted specifically to reduce instances of commotio cordis, these chest protectors have been proven efficient only in swine studies [28].

Commercial chest protection used in sports is not effective in preventing commotio cordis instances in children. There has been a substantial amount of commotio cordis cases occur with children wearing commercially available chest protectors [6, 9]. Recent studies have analyzed commercially available chest protectors and their ability to reduce ventricular fibrillation. Results were consistent that the protectors failed to do so, as some protectors resulted in a 50% chance of causing ventricular fibrillation when impacts were aimed directly over the heart [9].

1.3.3 NOCSAE Standards and Regulations

In 2017, the National Operating Committee on Standards for Athletic Equipment (NOCSAE) introduced standards and regulations for chest protectors regarding commotio cordis prevention. These standards were introduced for baseball and lacrosse [29]. Testing is based around the NOCSAE thoracic surrogate model which measures impact kinematics (Figure 1.6). There are 3 loading cells that measure impact force on the surrogate model. This includes an upper loading cell (ULC), lower loading cell (LLC) and a cardiac loading cell (CLC). Testing requires the chest protector to endure a 30-mph (13.41 m/s) and 50-mph (22.35 m/s) condition. 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. 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.

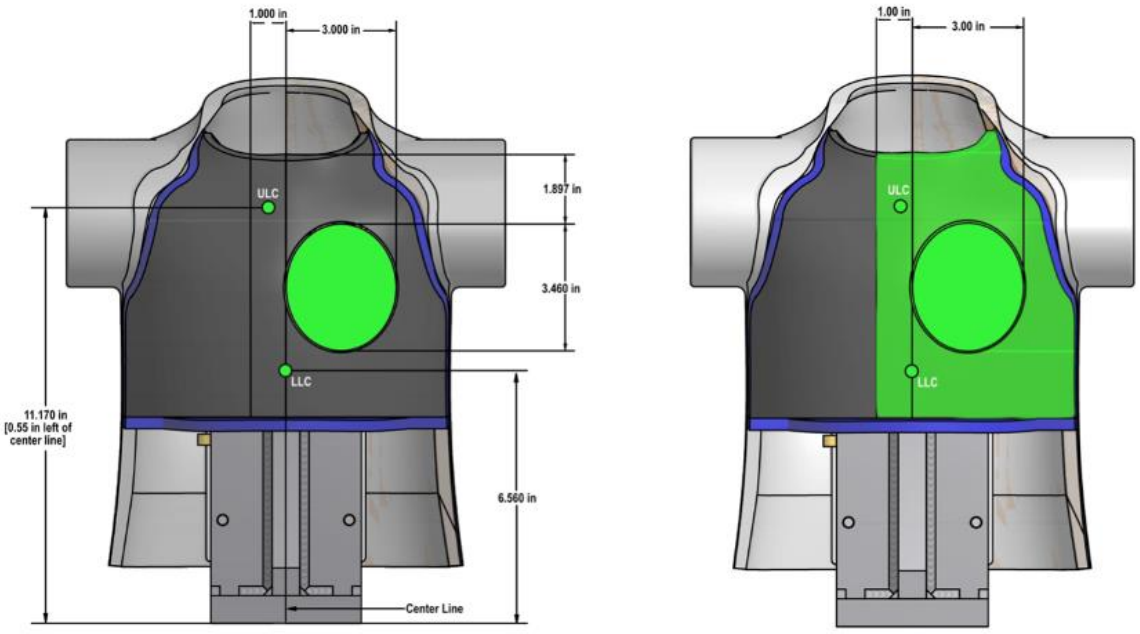

Figure 1.6 NOCSAE thoracic surrogate modelinclusive of the cardiac loading cell (large green circle on the right), upper and lower loading cell (small green circles in the middle) [29].

1.4 Commotio Cords Mechanisms

1.4.1 Animal Studies

Animal studies have allowed researchers to determine the mechanisms that lead to commotio cordis, which include the time during the cardiac cycle in which the impact occurs, the velocity of the impact, and the location of the impact. In terms of the literature, there are some classical studies that have been monumental in the development of determining commotio cordis mechanisms. A few swine studies have identified the

precise timing during the cardiac cycle in which an impact must occur to induce commotio cordis. Impacts during the upslope of the t-wave (approximately 40 ms) resulted in VF in ~20 kg swine [3, 4, 13, 30]. Specifically, the impacts need to be delivered during the upstroke, before the peak of T wave to replicate commotio cordis in lab animals [31].

Swine studies were also used to analyze and test impact speeds from 20 mph (8.94 m/s) to 70 mph (31.29 m/s) to determine which velocities were most likely to induce VF. Impacts with a velocity of 40 mph (17.88 m/s) induced VF approximately 70% of the time with impacts over the left ventricle of the heart during the up-slope of T-wave; impact velocities at 20 mph or less never induced VF during the study [3]. Impacts with velocities of higher than 50 mph (22.35 m/s) began to fail in replicating commotio cordis events as it led to structural damage such as papillary muscle tearing and/or myocardial rupture [32].

Other swine studies analyzed location of impact (30 mph, 13.41 m/s, with regulation baseball) and determined that impacts directly over the heart resulted in VF the most, while impacts that were not over the cardiac silhouette did not result in VF [5, 13]. Similar results from Link et al. determined that impacts that were not directly positioned over the cardiac silhouette never resulted in VF in their swine model [3]. These studies suggest that impact location is a very important factor to induce commotio cordis in swine.

In the swine literature, it is currently hypothesized that commotio cordis occurs as a result of ion channels (K^{+}_{ATP}) becoming increasingly activated post impact as the cell

membrane becomes stretched due to the rapid increase in pressure inside the heart. This ion activation is thought to result in an amplified dispersion of repolarization during the upstroke of the T-wave, resulting in ventricular fibrillation [30].

1.4.2 Humans vs. Swine Studies

Swine studies have dominated the commotio cordis field thus far due to the inability to test on humans or cadavers because a live heart needs to be involved. Although a human and swine torso are similar anatomically, swine have a rounded chest, while humans have a much flatter chest (Figure 1.7). The geometrical difference between the torso may have a negative effect on studies in which swine are meant to replicate a human model. In the swine torso we can identify clearly that the left ventricle is directly exposed to the impact, while all other chambers of the heart are not directly targeted. In the human torso, we can see that the heart's position in the chest is slightly different than that of the swine. The left ventricle in the swine model appears to be more exposed than in the human torso, which may result in differences throughout the model.

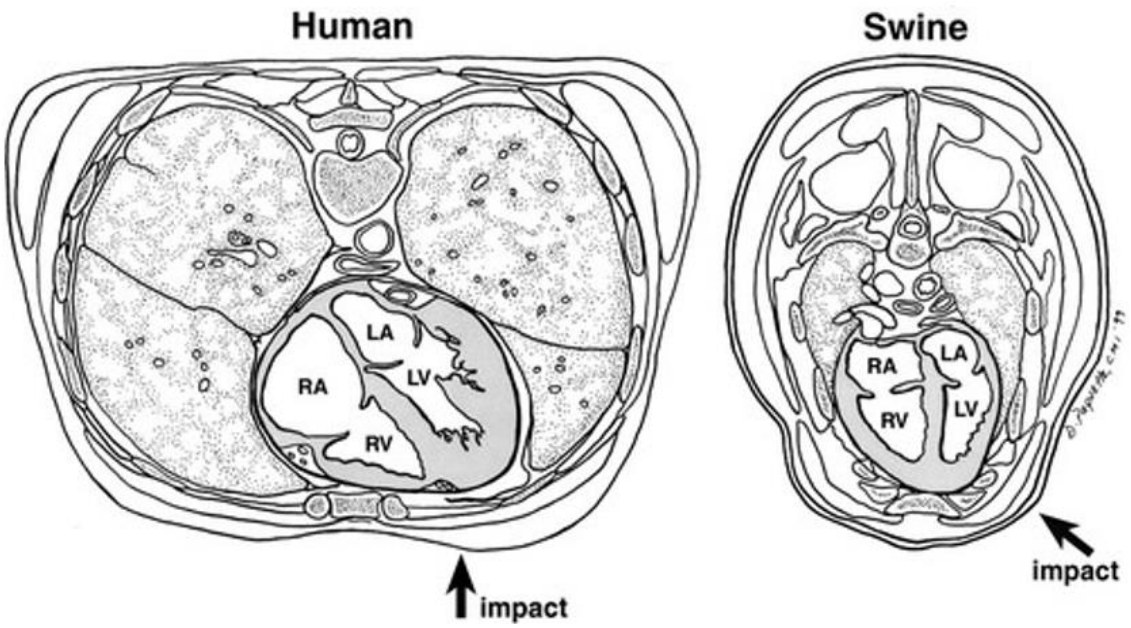

Figure 1.7 Swine vs. human torso: Cross-sectional anatomical view of a human torso (left) and swine torso (right) [3].

1.5 Finite Element Child Model

This thesis involved the use of the CHARM-10 finite element child model (developed at Wayne State University) which resembles an average 10-year-old child (Figure 1.8A) [33]. The model weighs 35 kg, contains all major anatomical features (Figure 1.8B) and biomechanical properties based on clinical scans of 10-year-old children (Table 1.1) and is highly complex with 742,087 elements and 504,775 nodes [34, 35]. This model used 8-node hexahedral elements (Figure 1.8C) and a multi-block approach. Selectively reduced integration was used with hourglass control type 4 and a parameter of 0.1. As a testament to the accuracy of the model, it has been validated based on both cardiopulmonary

resuscitation on live subjects as well as impact data from cadavers (Figure 1.9) [36, 37].

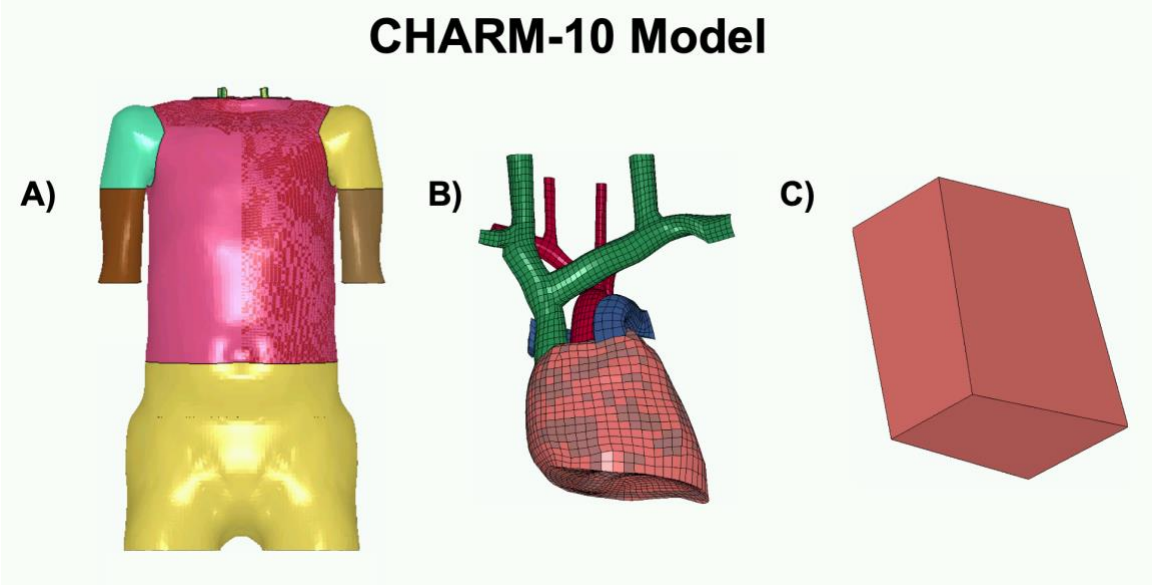

Figure 1.8 CHARM-10 Model: A) CHARM-10 finite element model frontal view; B) discretized heart from the model, found inside the chest made up of 8-node hexahedral elements; C) single hexahedral element from the heart.

Table 1. Material properties of CHARM-10 finite element model thoracic cavity.

Component	**Density**	**Material coefficient (C)**	**B1**	**B2**	**B3**	**Pressure**
Heart tissue	1.000e-006	1.085e-006	24.26	40.52	1.63	2.4825001
Component	**Density**	**Bulk modulus**	**Material coefficient (C)**	**Delta**	**Alpha**	**Beta**
Lung	2.880e-007	0.00266	1.115e-006	0.1	0.213	-0.343
Component	**Density**	**Young's modulus**	**Poisson's ratio**	**Axial damping**	**Bending damping**	**Material type**

Costal cartilage	1.000e-006	0.0051	0.4	0.0	0.0	Elastic
Intercostal muscle	1.000e-006	0.0021	0.4	0.0	0.0	Elastic
Thoracic flesh	1.100e-006	0.005	0.45	0.0	0.0	Elastic
Component	**Density**	**Young's modulus**	**Poisson's ratio**	**Material type**		
Clavicle (end)	1.830e-006	6.48	0.29	Rigid		
Sternum (end)	1.830e-006	6.48	0.29	Rigid		
Rib (end)	1.830e-006	6.48	0.29	Rigid		
Component	**Density**	**Young's modulus**	**Poisson's ratio**	**Yield stress**	**Tangent modulus**	**Material type**
Clavicle (trabecular)	1.100e-006	0.3	0.3	0.003	0.0	Piecewise linear plastic
Sternum (trabecular)	8.620e-007	0.2524	0.3	0.001408	0.01	Piecewise linear plastic
Rib (trabecular)	8.620e-007	0.2524	0.3	0.001408	0.01	Piecewise linear plastic
Component	**Density**	**Young's modulus**	**Poisson's ratio**	**Yield stress**	**Tangent modulus**	**Material type**
Clavicle (cortical)	1.800e-006	8.6400003	0.0	0.0641	0.0	Piecewise linear plastic
Sternum (cortical)	1.830e-006	6.48	0.29	0.0548	0.5	Piecewise linear

						plastic
Rib (cortical)	1.830e-006	6.48	0.29	0.0548	0.5	Piecewise linear plastic

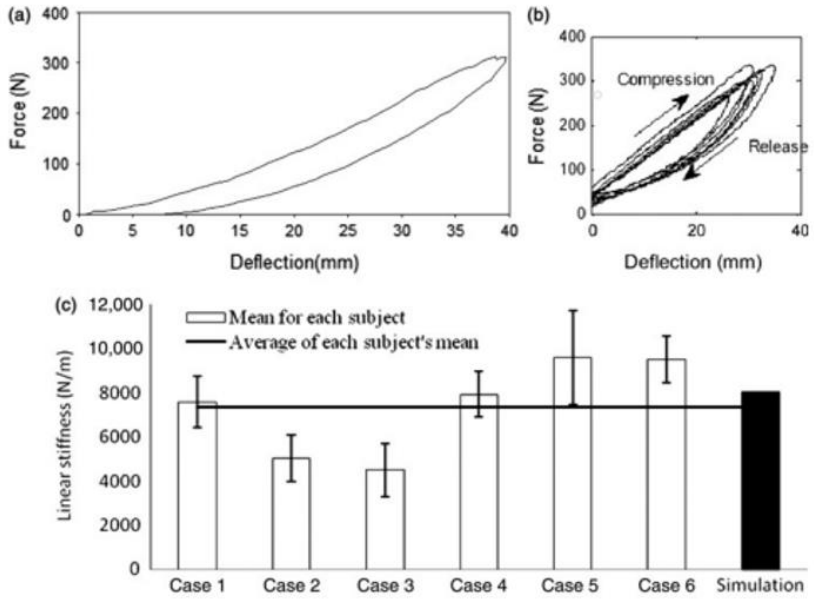

Figure 1.9 Simulation and CPR results.(a) Baseline FE model predicted force-deflection response; (b) result from CPR case showing linear force-deflection relationship [Adapted from Maltese et al. (2008)]; (c) comparison of the linear stiffness obtained from simulation and CPR.

An alternative comparison model we considered was the THUMS model, which was created for automotive testing. The THUMS model is validated through automotive testing (Figure 1.10) and uses tetrahedral elements.

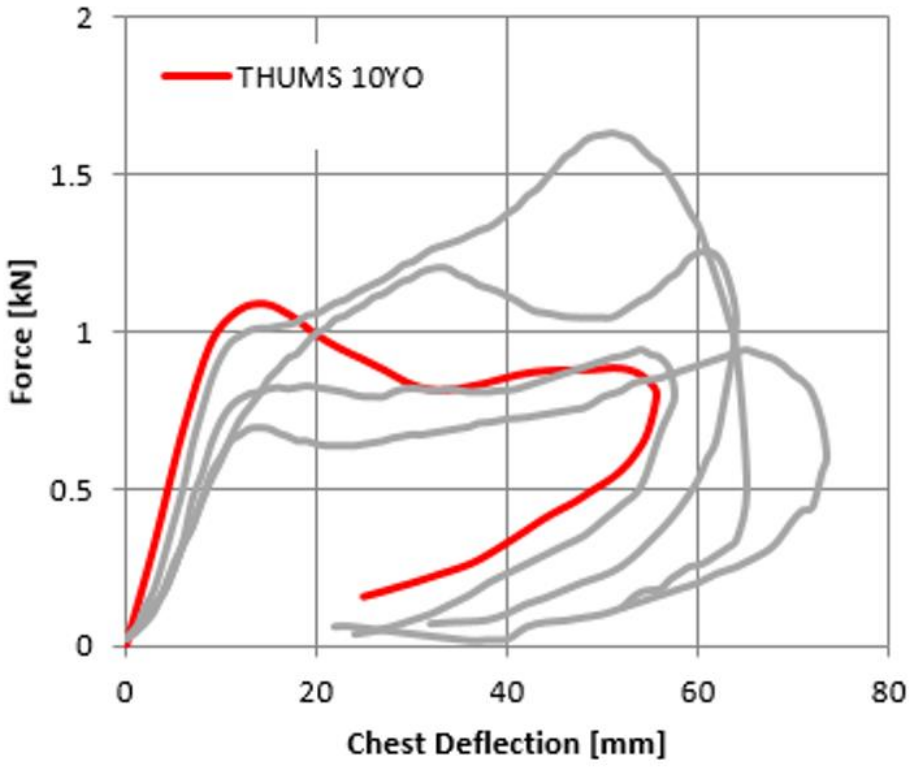

Figure 1.10 THUMS validation: Comparison of child cadaver (grey) to THUMS 10YO chest model (red) in frontal impact validation testing [38].

We believe the CHARM-10 model was better suited for our study because it was validated not only through frontal impact testing, but also through CPR, which is more relevant when testing baseball impacts to the chest that affect the heart.

1.6 Research Objectives

To improve and expand on the quality of research regarding commotio cordis, this thesis focused on the following main objectives: developing new injury metrics for commotio cordis in baseball, identifying the most vulnerable impact locations over the precordium, and analyzing the current NOCSAE regulations to determine their efficiency, including

any ways we may improve the current standards. Furthermore, this study introduced two new mechanical parameters to the topic: left ventricle strain and left ventricle pressure, which are thought to directly load heart tissues, causing VF.

This thesis approached the topic of commotio cordis in a novel way, as we used a highly detailed finite element child model to identify this sudden-death mechanism from a different perspective. To the best of my knowledge, no other research groups have analyzed commotio cordis using computational human thorax modelling. As highlighted previously in this chapter, commotio cordis testing and analysis can be difficult due to the limitations and constraints regarding testing on humans or cadavers, as neither of these are viable options. Current swine model testing has allowed for breakthroughs in the literature, and although swine and human anatomy have major similarities, they are ultimately different, particularly in the shape of the thorax.

Chapter 2

2 Developing Commotio Cordis Injury Metrics for Baseball Safety - Investigating the Relationship Between Chest Force and Rib Deformation to Left Ventricle Strain and Pressure

This chapter was co-authored by Dr Haojie Mao, Kewei Bian and Habib R Khan MD and is under peer review with the journal, “Computer methods in biomechanics and biomedical engineering”

2.1 Abstract

Commotio cordis is a sudden death mechanism that occurs when the heart is impacted during the repolarization phase of the cardiac cycle. This study aimed to investigate commotio cordis injury metrics by correlating chest force and rib deformation to left ventricle strain and pressure. We simulated 128 chest impacts using a simulation matrix which included two initial velocities, 16 impact locations spread across the transverse and sagittal plane, and four baseball stiffness levels. Results showed that an initial velocity of 17.88 m/s and an impact location over the left ventricle was the most damaging setting across all possible settings, causing the most considerable left ventricle strain and pressure increases. The impact force metric did not correlate with left ventricle strain and pressure, while rib deformations located over the left ventricle were strongly correlated to left ventricle strain and pressure. These results lead us to the recommendation of exploring new injury metrics such as the rib deformations we have highlighted for future commotio cordis safety regulations.

Keywords Commotio cordis, finite element, heart strain, heart pressure, baseball injury

2.2 Introduction

Commotio cordis (CC) refers to sudden death from low-energy non-penetrating chest impacts over the cardiac silhouette in the absence of structural heart disease. Defined as a cardiac concussion, commotio cordis shows no signs of structural damage to the heart post-impact [5, 23]. According to the US Commotio Cordis Registry (USCCR) in Minneapolis, there are currently over 200 confirmed cases worldwide [2, 3]. Although the occurrence rate of commotio cordis is low, most cases are fatal. [39]. Commotio cordis can happen in a wide variety of circumstances; ranging from casual play in a backyard or playground, to competitive hockey, lacrosse, or baseball games [2, 27]. Meanwhile, the statistics in cases are believed to be strongly influenced by the lack of awareness towards commotio cordis, suggesting that there are many more cases of the sudden death mechanism that have gone unreported [2].

Prevention of commotio cordis has been investigated in the literature and focuses on the use of safety chest protectors [8, 9, 28, 39, 40]. Although the use of chest protectors in contact sports are common, they are not designed with the prevention of CC in mind. One recent study found that a combination of high- and low-density foam, flexible elastomer and a polypropylene polymer in a chest protector reduced the incidence of ventricular fibrillation (VF) by 49% in swine models [41]. On the other hand, various studies in the literature have explained how commercially available chest protectors fail to reduce the incidence of VF in commotio cordis events [8-11]. Meanwhile, there is a very specific time window in which an impact to the cardiac silhouette must occur to induce commotio cordis in a subject [4, 30, 42]. The limited-time window makes laboratory investigations

challenging, with only a handful of swine experiments with impacts being aimed at the left ventricle (LV) successfully causing commotio cordis [4, 43].

Currently, the National Operating Committee on Standards for Athletic Equipment (NOCSAE) has standard test methods for evaluating chest protectors in their ability to prevent commotio cordis. The evaluation measures peak force over the chest cavity of the NOCSAE Thoracic Surrogate (NTS) through two impact velocity tests, 30-mph (13.4 m/s) and 50-mph (22.4 m/s), with an upper and lower load cell, as well as a cardiac load cell used to measure the impact force. Impact force was measured in newtons (N). For baseballs, peak force from impact in the 30-mph case must not exceed 400 N by the cardiac load cell, and 498 N for the upper or lower load cell. In the 50-mph case, the cardiac load cell must not exceed 800 N, while the upper and lower load cell shall not exceed 1001 N [29]. Currently, only force over the chest cavity is included in the testing criteria. Moreover, impact-induced cardiac responses, especially mechanical responses of the LV such as strain and pressure that directly affect the heart remain unknown. Therefore, it is necessary to understand the correlation between external parameters such as chest force and rib deformations to internal heart responses.

This study adopted a detailed finite element (FE) model representing a 10-year-old child chest, which was validated under higher-energy blunt impacts on post-mortem human subjects (PMHSs) [37] and was exercised under lower-energy cardiopulmonary resuscitation on live subjects [33, 36]. We simulated a total of 128 baseball to chest impacts covering a wide range of real world-relevant events, including various impact velocities, impact locations, and different baseballs. The focus of this study was to

understand external forces/deformations to LV strain and pressure. Meanwhile, how different impact settings could affect LV responses were also investigated.

2.3 Methods

2.3.1 Finite Element Simulation of Baseball to Chest Impact and Post Processing

Impact responses were analyzed using the chest model of the CHARM-10 developed at Wayne State University [34], which represents an average 10-year-old child. This detailed FE model includes 742,087 elements and 504,775 nodes. This model used 8-node hexahedral elements and a multi-block approach. Selectively reduced integration was used with hourglass control type 4, which is a Flanagan and Belytschko stiffness control [44], and a parameter of 0.1 for soft tissues. The model contains all major anatomical structures based on detailed clinical scans of 10-year-old children [45], including 12 pairs of ribs, the spinal column from T1-T12 and L1-L5, scapula, sternum, clavicle, humerus, cartilage and ligaments, lungs, heart, kidney, liver, spleen, stomach, gallbladder, intestines, diaphragm, all major arteries (e.g. Aorta), costal cartilage, glenoid cartilage, intercostal muscles, coracoclavicular ligament, and coracoacromial ligaments. Another advantage of the chest model is that the model has been validated based on both data collected through cardiopulmonary resuscitation on live subjects [36] and impact data collected on cadavers [37]. Alongside the validated chest model, a baseball model with a radius of 37.5 mm was created with the material property being defined based on the literature [46].

Each simulation had a run time of 20 ms, with an output frequency of 10,000 Hz for force. 2,000 Hz for strain and pressure, and 1,000 Hz for deformation. Simulations were

run on Ls-Dyna (LSTC, Livermore, Ca) [47]. After the simulations were completed, LS-PrePost2.4 [48], an advanced pre- and post-processor, was used for data collection and analysis for the impact response.

2.3.2 Design of Experiments

Impact Velocity

The baseball had two initial impact velocities of 13.41 m/s and 17.88 m/s, positioned 1.0 m from the chest cavity (Figure 2.1. A). These initial velocities are consistent with the literature which reports this as the most susceptible velocity range [32].

Impact Location

Sixteen impact locations were simulated with the standard direction aiming directly over the heart (Figure 2.1. B) The baseball moved by the radius of the ball (37.5 mm) medial to lateral (transverse) and/or inferior to superior (sagittal). Together this created four locations in the transverse direction and four in the sagittal direction, for a total of sixteen impact locations.

Baseball Stiffness

Four baseball stiffness values were used (Figure 2.1. C), representing the reduced injury factor (RIF). RIF 1, RIF 5, RIF 10, and standard were simulated. RIF 1 represents a stiffness of 213 N/cm, RIF 5 represents 353 N/cm, RIF 10 represents 1114 N/cm and standard represents a standard baseball stiffness of 2533 N/cm [10].

In total, 128 simulations were conducted, with two impact velocities, sixteen impact locations, and four baseball stiffness values (Figure 2.1).

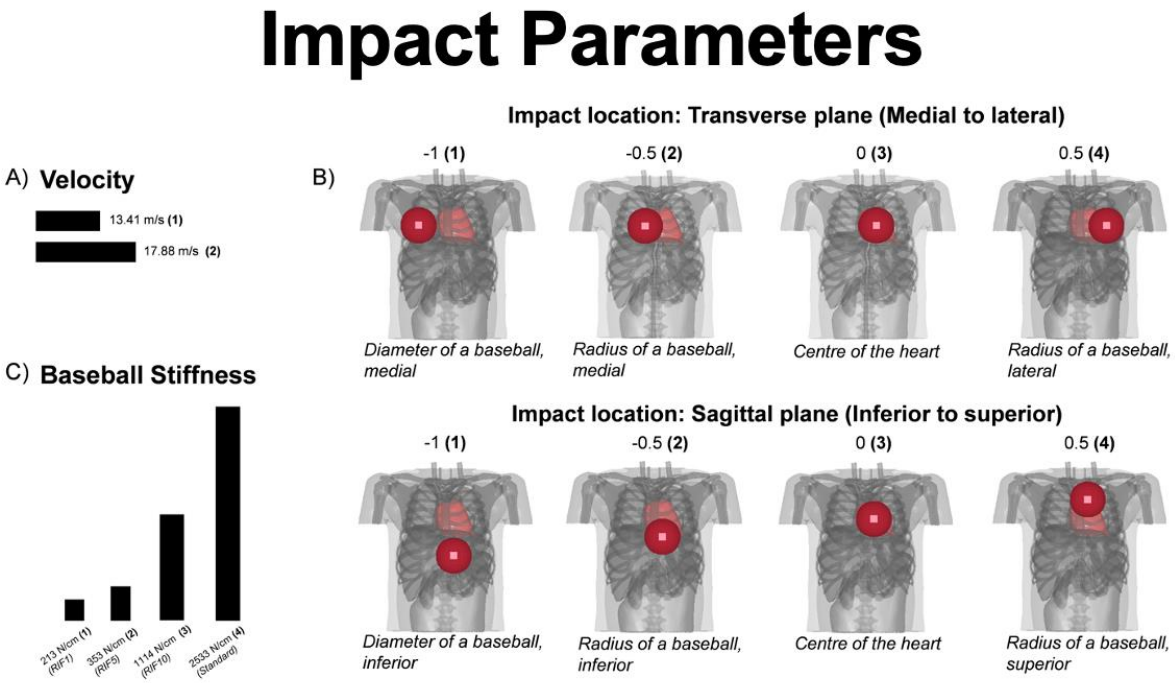

Figure 2.1 Impact parameters for simulation matrix: A) Impact velocity speeds (m/s). B) Impact locations in transverse and sagittal plane. C) Baseball stiffness levels (N/cm) ranging from soft to standard baseball stiffness.

Boundary Conditions

The boundary and loading conditions for the CHARM-10 were set so that the chest model could move freely upon impacts and during simulation (Figure 2.2). The contact

force was defined between the baseball and chest, and the baseball was set with an initial velocity for impacts.

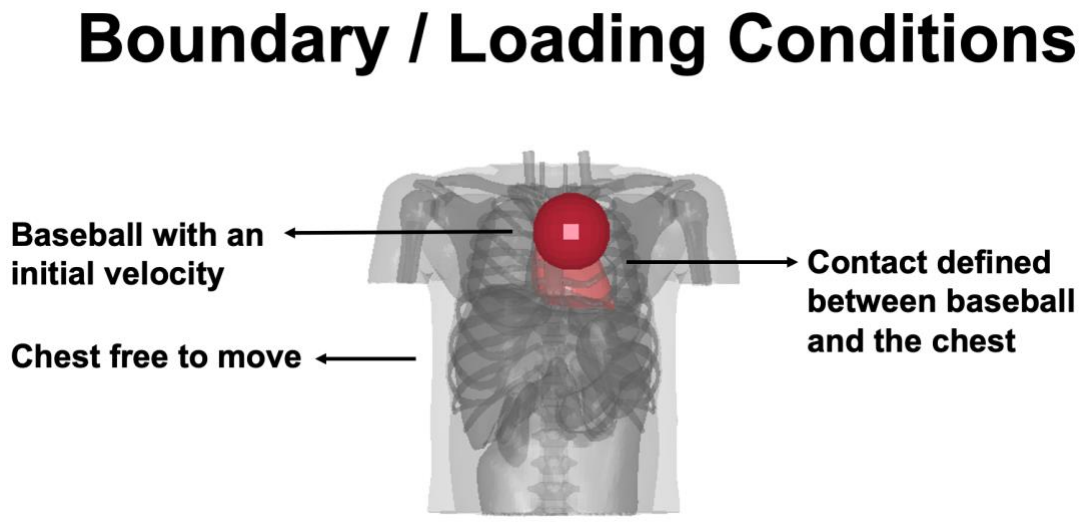

Figure 2.2 Boundary / loading conditions of the CHARM-10 model. The chest was free to move and included a baseball with an initial velocity. The contact force was defined between the baseball and the chest.

Impact Responses

Using the CHARM-10 computational model, we analyzed the following impact responses: Force between baseball and chest (the contact force between the baseball and chest), max rib deformation, LV strain, which was calculated by analyzing maximum principal logarithmic strain from elements specific to the LV of the heart, LV pressure, and rib deformation at the LV (Figure 2.3). The highest strain was obtained by calculating strain for all the elements that make up the left ventricle of the heart and determining the highest value during the entire time history. The time histories for all LV

element maximum principal strain were output, and then averaged. The peak value from the averaged curve was selected as the highest strain. The left ventricle pressure was obtained by calculating hydrostatic pressure for all the elements that make up the left ventricle and identifying the highest value during the entire time history. Similar, an average was conducted, and peak value of the entire time history was used. Commotio cordis is an electrophysiological failure involving the entire LV tissue rather than tear failure at a specific region, therefore, we used the average over all LV elements to provide a description of the loading to LV during impacts. Rib deformation was calculated by measuring the displacement (mm) between the anterior rib receiving the baseball impact, and the corresponding posterior rib. Regarding rib deformation at the LV region, we further examined the deformation of rib 3, 4, and 5 as marked in Figure 2.3.

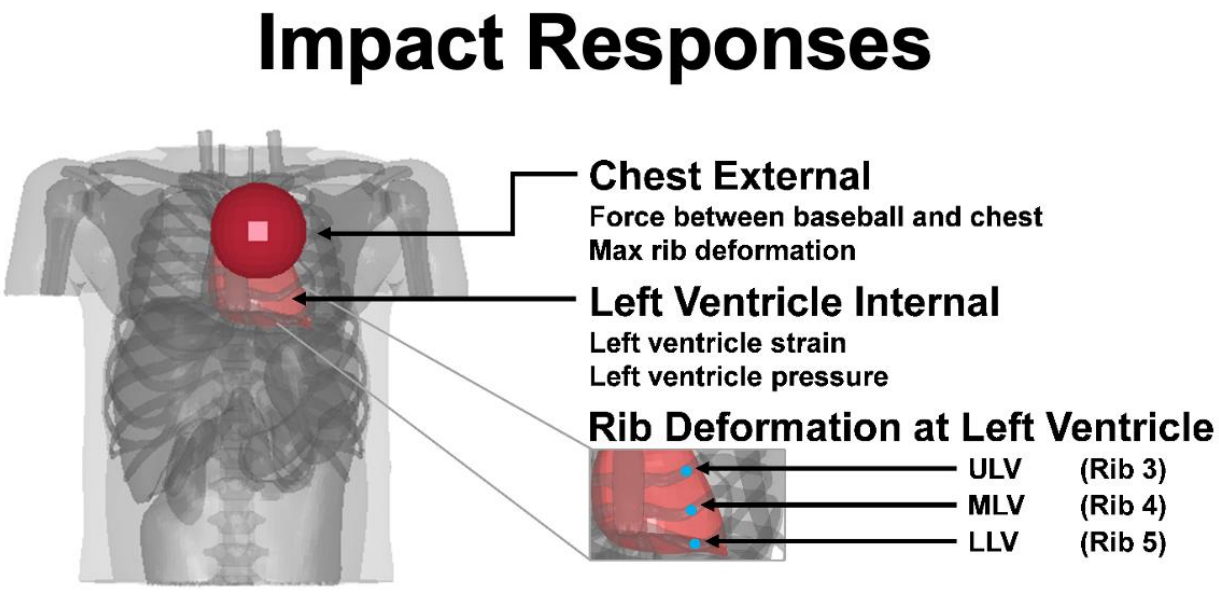

Figure 2.3 Impact responses analyzed from the computational model. Chest internal responses, left ventricle internal responses and detailed chest external responses as rib deformations at the left ventricle region. ULV: Upper left ventricle, MLV: Middle left ventricle and LLV: Lower left ventricle.

Impact Parameter Analysis

Impact parameter results were managed through the use spreadsheets and Minitab (Minitab, LLC, State College, Pennsylvania, USA). The correlation between external response and internal response was conducted through spreadsheet with R^2 values. Statistically, R^2 indicates the proportion of one variable such as strain, which is predictable from another variable, such as rib deformation. Mathematically, the two variables may switch and still result in the same R^2 value. Minitab was used to analyze the contribution of each impact parameter by creating a Pareto chart. Main effect charts were created to determine how influential each parameter was in affecting LV strain and pressure.

2.4 Results

2.4.1 Impact Responses vs. Strain Correlations

Both maximum rib deformation and reaction force did not correlate with LV strain with R^2 values less than 0.01 (Figure 2.4. A&B). Meanwhile, rib deformation near the upper left ventricle (ULV) and middle left ventricle (MLV) regions did have a strong correlation, with R^2 of 0.77 and R^2 of 0.75, respectively (Figure 2.4. C&D). Rib deformation at the lower left ventricle (LLV) region showed a positive correlation with strain as well, but with R^2 of 0.34 (Figure 2.4. E). Overall, ULV and MLV rib deformation correlated with LV strain best (Figure 2.4. F).

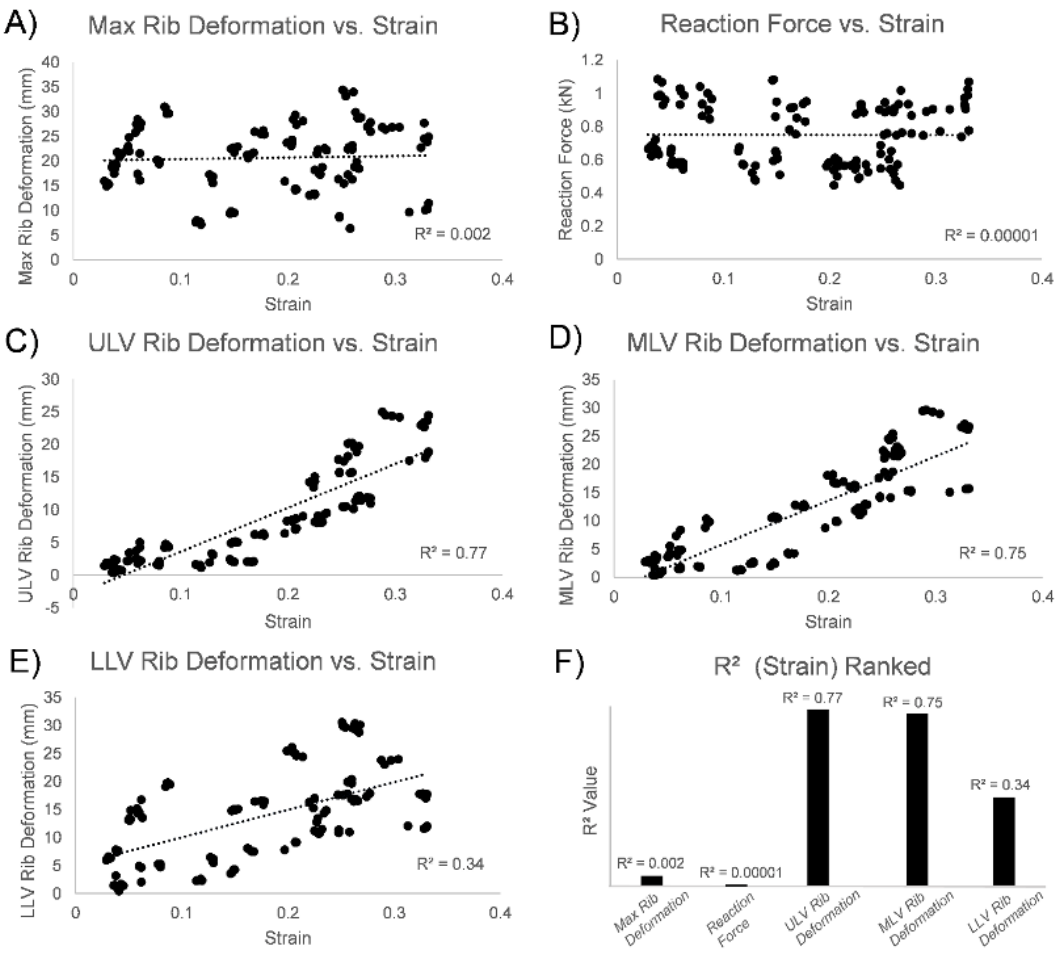

Figure 2.4 Impact response vs. strain correlation. A) Max rib deformation vs. Strain (P = 0.617) B) Reaction force vs. Strain (P = 0.969) C) ULV rib deformation vs. Strain (P ≤ 0.001) D) MLV rib deformation vs. Strain (P ≤ 0.001) E) LLV rib deformation vs. Strain (P ≤ 0.001) F) R^2 (strain) ranked. ULV and MLV rib deformation have the strongest R^2 correlation, with reaction force showing the weakest correlation.

2.4.2 Impact Responses vs. Pressure Correlations

Similar to LV strain, pressure had a very weak correlation to max rib deformation, and more notably, reaction force (Figure 2.5. A&B). Reaction force between the baseball and

chest had a very low correlation to pressure, with R^2 values less than 0.1. MLV rib deformation stood out as the strongest correlation with an R^2 of 0.83 (Figure 2.5. D), while ULV rib deformation had a strong correlation as well, with an R^2 value of 0.71 (Figure 2.5. C). LLV had a moderate correlation with an R^2 value of 0.52 (Figure 2.5. E). Overall, MLV and ULV rib deformation correlated with pressure best.

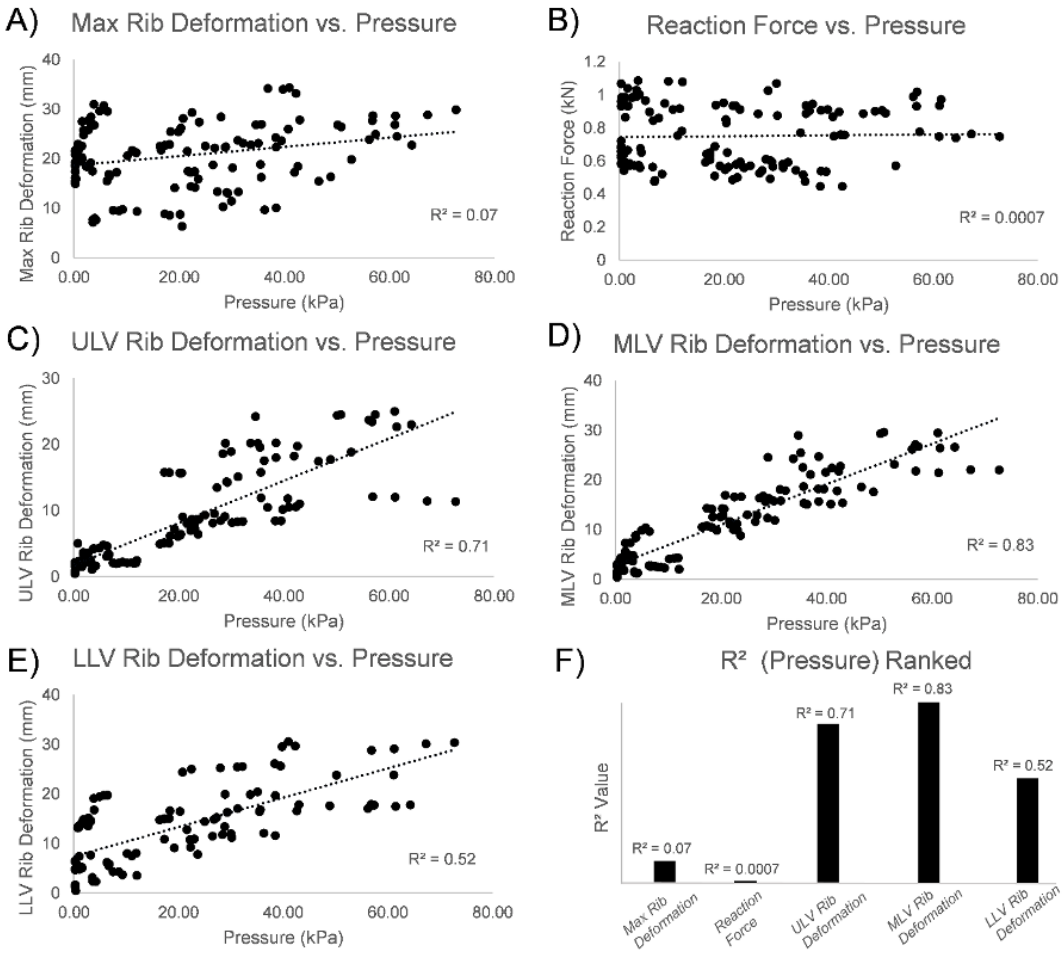

Figure 2.5 Impact responses vs. pressure correlation.. A) Max rib deformation vs. Pressure (P = 0.002) B) Reaction force vs. Pressure (P = 0.771) C) ULV rib deformation vs. Pressure (P ≤ 0.001) D) MLV rib deformation vs. Pressure (P ≤ 0.001) E) LLV rib deformation vs. pressure (P ≤ 0.001) F) R^2 (pressure) ranked.

ULV and MLV rib deformation have the strongest correlation, with reaction force showing the weakest correlation

2.4.3 Parameters Affecting Left Ventricle Strain and Pressure

Regarding LV strain, the Pareto chart highlighted velocity as the most influential factor (Figure 2.6. A). Regarding LV pressure, velocity, and impact position (transverse and sagittal) are the most important factors (Figure 2.6. A). Baseball stiffness was found to be an insignificant factor as changing the baseball from soft to hard stiffness levels did not affect LV strain or pressure (Figure 2.6. A).

2.4.4 Most Damaging Setting

After analyzing the pareto and main effect charts (Figure 2.6. A), it was concluded that the most damaging setting in all 128 simulations was the combination of a baseball with an initial velocity of 17.88 m/s and an impact location in the transverse (position 4) and sagittal (position 3) direction (Figure 2.6. B). The most damaging setting produced the highest strain and pressure in the LV.

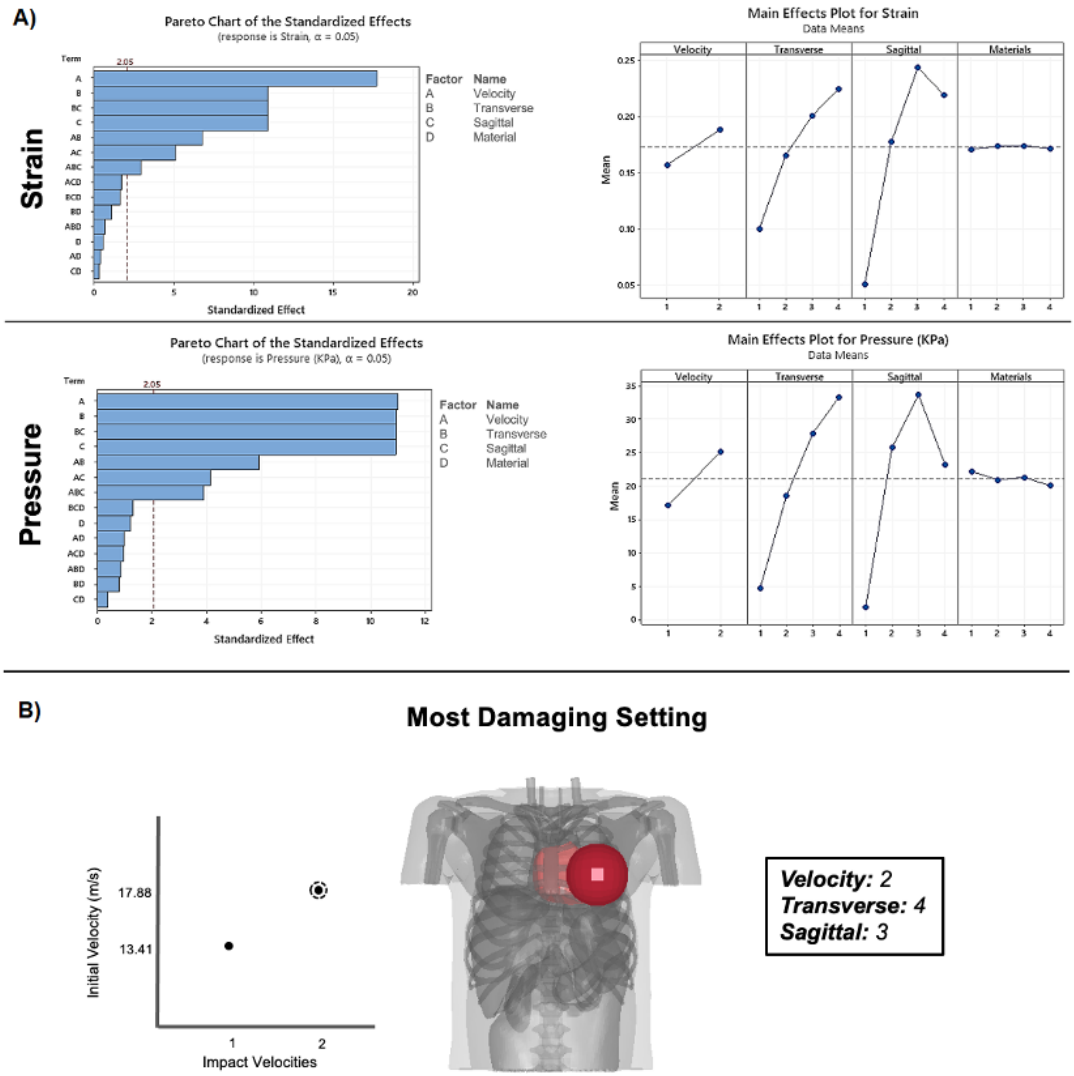

Figure 2.6 Most damaging setting: A) Pareto and main effect charts for strain and pressure of the left ventricle. B) Most damaging setting according to the main effect charts. Velocity (2), transverse (4), sagittal (3).

2.4.5 Left Ventricle Strain and Pressure and MLV Rib Deformation Time History

Comparisons were made between two representative high and low velocity cases for LV strain, pressure and MLV rib deformation (Figure 2.7). Peak strain occurred

approximately 5 ms after initial impact, while peak pressure occurred at moment of impact. MLV rib deformation peaked around 5 to 10 ms after initial contact, consistent with strain development.

2.4.6 Reaction Force Time History with Filter Comparison

A reaction force time history graph shows the effects of different force filters (Figure 2.7). The different filter options were selected to further investigate the ability for the NOCSAE accepted low-pass channel frequency class (CFC) 120 filter to collect peak values when looking at reaction force from impacts. We included a no-filter option, as well as a high-pass filter of CFC 1000 for comparisons. Based on the comparison, the filter CFC 1000 was deemed as acceptable and used in this study. Other predictions including strain, pressure, and deformations did not show noise, therefore no filter was applied.

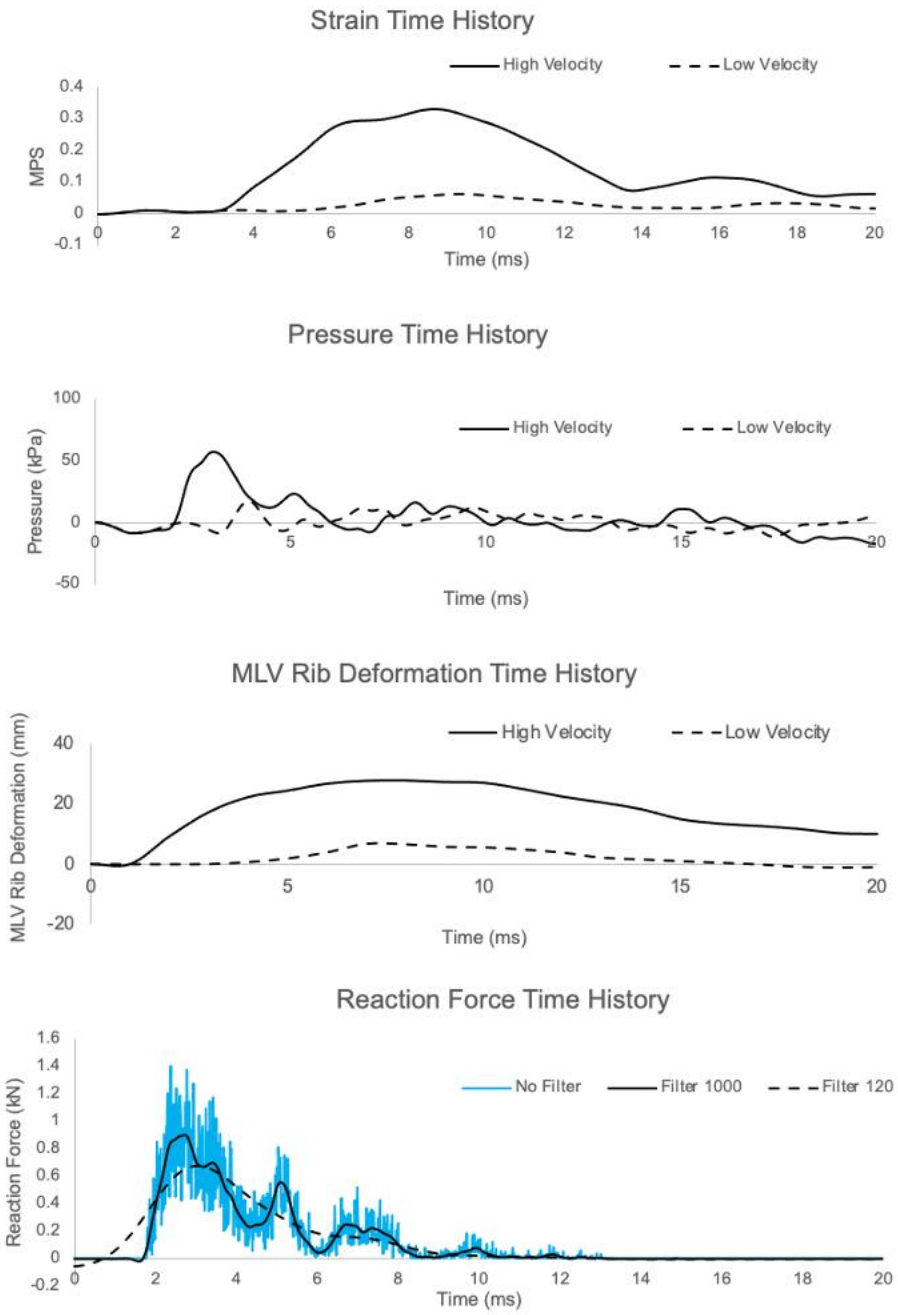

Figure 2.7 Time history graphs: Strain, pressure, MLV rib deformation and reaction force time history graphs comparing either high velocity and low velocity cases or different force filters (MPS = maximum principal strain).

2.5 Discussion

We used a validated child chest model to systematically understand how various chest impacts affected the heart, especially the LV that has been the target in studying commotio cordis. Our data suggested that rib deformation at the upper and middle portion of the LV correlated strongest to LV strain and pressure acutely developed during impacts. However, the impact force or the maximum rib deformation did not correlate with LV strain or pressure, mostly due to varying impact locations. Pareto chart analysis further demonstrated how both impact velocity and impact locations could affect LV strain and pressure. Interestingly, the use of softer baseballs did not reduce LV strain and only slightly reduced LV pressure. To the best of our knowledge, our detailed computational study is the first of its kind to provide data correlating impact parameters and external chest responses to the biomechanical responses of LV strain and pressure using a validated child chest model.

The injury criteria suggested by NOCSAE, as well as those found in current literature, use reaction force to test impact safety [29, 49]. Our data suggests that impact force does not correlate with LV strain and pressure (Figure 2.4, 2.5). It should be noted that the force sensor specified by NOCSAE was at fixed locations such as the heart position which allows the measurement of force directly applied to the heart region, while the maximum force was measured at the location of impact which could be off from the heart in this study. Considering impacts delivered at various locations, we recommend using ULV and MLV rib deformation as a means of injury metrics for commotio cordis safety, as they both have strong correlations with strain and pressure of the LV.

Initial velocity and impact location are heavily favored parameters in the pareto and main effect charts. Specifically, transverse (4), sagittal (3) and velocity (2) were the highest for LV strain and pressure. Our results from initial velocity match the current literature, a study from 2007 analyzed the effects from different impact velocities on the incidence of VF, finding that a velocity of 40 mph (17.88 m/s) induced VF at a rate of approximately 70%, the highest rate between all impact velocities tested [50].

With reference to impact location, our most damaging case depicts an impact over the cardiac silhouette, specifically over the center and base of the LV (Figure 2.6. B). Previous studies have identified in swine models that baseball impacts over the LV induce VF during the repolarization phase of the cardiac cycle [3, 13, 51]. Most interestingly, the same study that identified which velocities were most likely to induce VF, reported that it occurred mostly with blows directly over the center of the cardiac silhouette (30% of impacts) compare to those over the LV base (13%) or apex (4%) from a swine model [50]. It should also be acknowledged that the baseball was moved at 37.5 mm of increments during simulations.

Literature suggests that softer baseballs mitigate commotio cordis events in swine studies [12]. However, our results indicate that the effect of baseball stiffness levels on LV strain and pressure was not significant, especially when comparing to impact velocities and impact locations. When identifying the effects of varying stiffness levels, we found that for LV strain there was almost no change, whereas for LV pressure there was a mean difference of approximately 1-2 kPa across the 4 stiffness levels. With respect to the Pareto charts, baseball stiffness levels were the clear cut least important factor, showing 95% less significance than initial velocity, and 91% less significant than impact location

for left ventricle strain. When considering LV pressure, baseball stiffness was found to be 91% less significant of a factor than initial velocity and impact location. Softer baseballs did slightly reduce LV pressure, which aligns with the recommendation to mitigate commotio cordis events [40]. However, it should be emphasized that chest protection is greatly needed even with using softer baseballs.

We used the 1000 Hz filter rather than the 120 Hz filter specified in the NOCSAE standard test methods for commotio cordis protectors [29]. The reason we chose this filter was because the lowpass filter 120 may have been too strong for our data, therefore missing higher peak values of data and ultimately missing high reaction force values. The low-pass 120 filter had an 18% decrease in its' ability to measure peak force values when compared to a low-pass 1000 filter. The 120 filter measured a peak of 0.65 kN, while the 1000 filter measured 0.90 kN. Meanwhile, it should be acknowledged that the filter 1000 was deemed as acceptable for out computational prediction which is different from the experimental force sensors.

While our model is exceedingly detailed, one of our limitations is that the model does not include fluid structures within the heart, such as blood. Further studies could attempt to incorporate blood flow through the heart to simulate a more realistic simulation with more computational power being available to solve the fluid-structure interaction with deformable boundaries. Nevertheless, we have applied an internal pressure of 9.3 kPa to the heart wall mimicking blood pressure and focused on collecting acute strain and pressure raised in milliseconds during the impact, which was expected to not be affected by the flow change in this short period of duration.

Literature has suggested that for commotio cordis to occur, the impact must strike when the cardiac cycle is repolarizing (upslope of the T-wave) [50] (Figure 2.8. C). Our model used an internal heart pressure of 9.3 kPa, equivalent to 70 mmHg. Ventricular pressure of the heart peaks at the start of the T-wave and begins to dramatically drop after the peak of the T-wave. We performed a parametric study to calculate the difference between 0 mmHg to 120 mmHg of internal heart pressure when calculating left ventricle strain and pressure. Our results found that there was a difference of 4 % between 70 mmHg and 120 mmHg for left ventricle pressure (Figure 2.8. B), and a 2 % difference between 70 mmHg and 120 mmHg for left ventricle strain (Figure 2.8. A). It is our understanding that this small of a difference did not affect our overall results. We also acknowledge that there was no validation of left ventricle pressure and strain conducted in this study. Nevertheless, with the chest model being validated by force-deflection data from cadavers and live subjects during CPR, we justified that it is acceptable to use the predicted pressure and strain to analyze overall effects of baseball-related chest impacts on left ventricle responses.

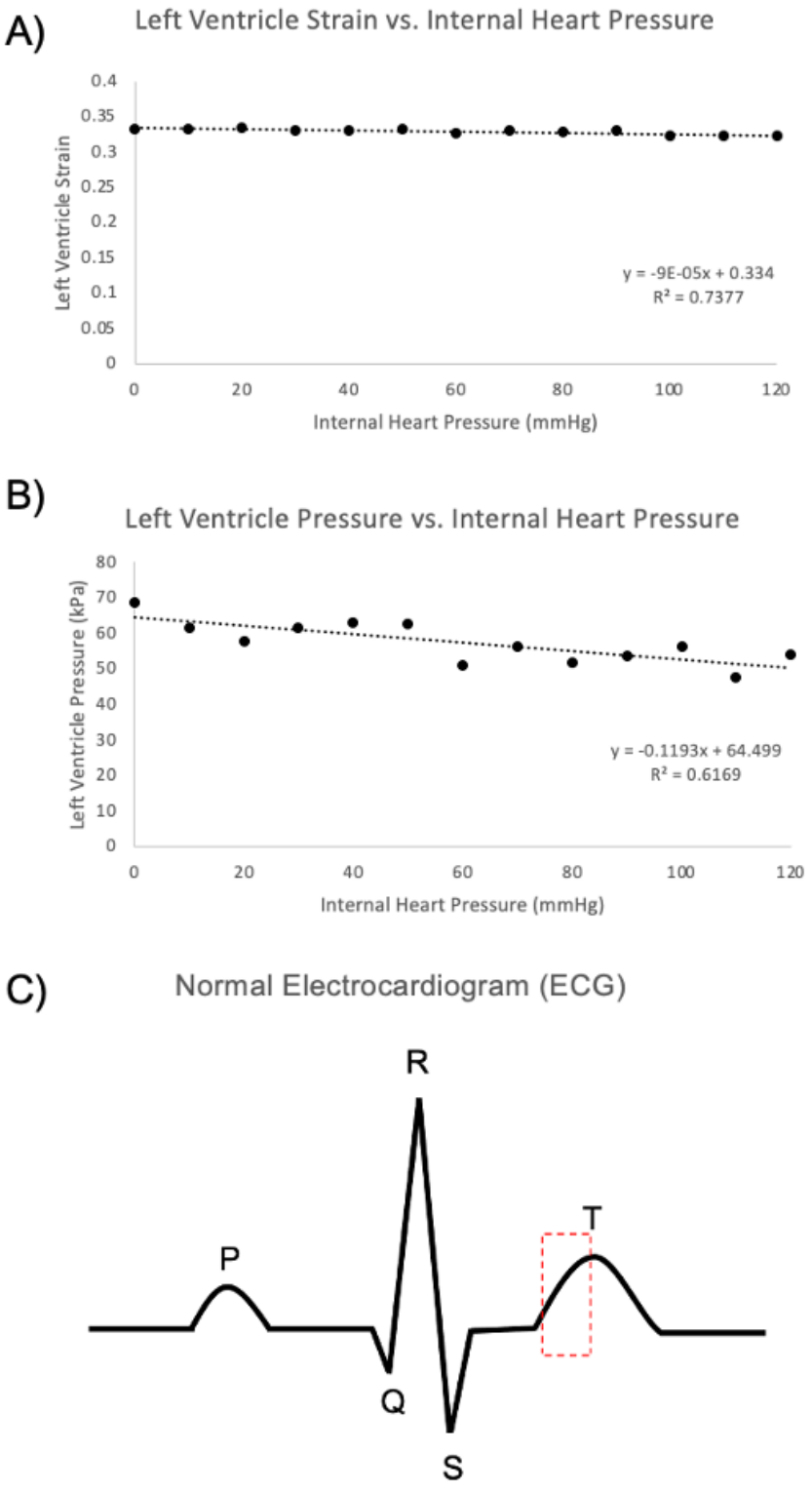

Figure 2.8 Parametric study results: A) Left ventricle strain vs. internal heart pressure. B) Left ventricle pressure vs. internal heart pressure. C) Normal electrocardiogram (ECG) identifying in the dotted red square the up-slope of the T-

wave (repolarization phase) where researchers have identified as the most vulnerable portion of the cardiac cycle.

2.6 Conclusion

This detailed computational study helps to address the tissue-level biomechanical mechanisms of commotio cordis. Initial velocity and impact location in the transverse and sagittal plane over the chest cavity played important roles in LV strain and pressure, whereas baseball stiffness made almost no difference in this regard. Left ventricle strain and pressure correlated strongly with ULV and MLV rib deformation, while they did not correlate with maximum reaction force from the baseball to chest. From our understanding of the current literature and through this study, we should place a more considerable emphasis on the heart's response during and after initial impact and develop injury metrics based on this knowledge. Impact responses such as strain and pressure of the LV and deformation of the ribs located at ULV and MLV could prove to be appropriate measurements in the evaluation of chest protectors through commotio cordis testing methods.

Chapter 3

3 Identifying Vulnerable Impact Locations to Reduce the Occurrence of Deadly Commotio Cordis Events in Children's Baseball – A Computational Approach

This chapter was co-authored by Dr. Haojie Mao, Kewei Bian, Xingyu Liu and MD Habib R. Khan and is under peer review from the journal of biomechanical engineering

3.1 Abstract

Commotio cordis is the second leading cause of sudden cardiac death in young athletes. Currently available chest protectors on the market are ineffective in preventing cases of commotio cordis in young athletes that play baseball. This study focused on using contour maps to identify specific baseball impact locations to the chest that may result in instances of commotio cordis to children during baseball games. By identifying these vulnerable locations, we may design and develop chest protectors that can provide maximum protection to prevent commotio cordis in young athletes. Simulation cases were run using the validated CHARM-10 chest model, a detailed finite element model representing an average 10-year-old child's chest. A baseball model was developed in company with the chest model, and then used to impact the chest at different locations. An 8x7 impact location matrix was designed with 56 unique baseball impact simulations. Left ventricle strain and pressure, reaction 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 (82.94 kPa). We have identified the most dangerous impact

locations resulting in high left ventricle strain and pressure. This novel study provided evidence of where to emphasize protective materials for establishing effective chest protectors that will minimize instances of commotio cordis in young athletes.

3.2 Introduction

Despite the use of chest protectors in youth sports, sudden death in young athletes from impacts over the precordium known as commotio cordis still occur [52]. Low-energy, non-penetrating impacts to the chest, such as those found in children's baseball games may lead to incidences of commotio cordis [31]. Identified as the second leading cause of sudden cardiac death in youth athletes [1], this rare sudden death mechanism is a result of the heart going into ventricular fibrillation due to the combination of multiple variables: impact speed (30 to 50 mph), impact location (impacts over the precordium), and timing of impact relative to the cardiac cycle (repolarization phase during the upslope of the T-wave) [1, 3].

Commotio cordis is most commonly found in children, as it is believed that children are susceptible due to their narrow and underdeveloped chest cages, which deform on impact. Baseball, hockey, and lacrosse all involve small, fast traveling projectiles that can come in contact directly over the chest at any given moment. To combat this alongside other injuries, athletes often wear chest protectors during play. When considering popular sports such as hockey and lacrosse, all players must wear chest protectors, whereas baseball requires only the catcher position to wear a chest protector. Moreover, the problem with current chest protectors on the market is that they are designed to protect the chest cage from structural damage, and not necessarily designed to protect against commotio cordis instances. As of 2010, the U.S. Commotio Cordis Registry (USCCR)

contained records of 224 fatal cases of commotio cordis with almost half of these cases occurring in competitive sports, and 40 of those competitive athletes wearing commercially available chest protectors [31]. The registry identifies baseball as the leader of all sports in the frequency of commotio cordis cases with 80 [31].

Current brands on the market claim their chest protectors are safe for children, but these companies may give a false sense of safety for the parents of young athletes. Link et al. found that current commercially available chest protectors failed to mitigate commotio cordis instances as the chest protectors were either found to be composed of material incapable of absorbing impacts and/or the design was unable to maintain coverage over the chest wall during athletic movements [11]. One recent study showed some positive results in reducing ventricular fibrillation occurrence by combining different density foam alongside a flexible elastomer and polypropylene polymer [28]. Contrarily, an abundance of studies from the last two decades have outlined the ineffectiveness of commercially available chest protectors. 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 % [9]. All other protectors in the study landed between this range of occurrence. Furthermore, another study found that an astonishing amount of commotio cordis events (40 %) occurred across various sports in the presence of the athletes wearing commercially available chest protectors [52].

Although baseball is the most common sport involving commotio cordis cases, it is not the only sport with this risk. Research has found that lacrosse balls launched directly over the chest protector resulted in the death of 4 lacrosse goalies [7]. Additionally, a young hockey player died after being struck in the chest by a puck while wearing a chest protector, just one of many instances of commotio cordis in the presence of a chest protector found in youth hockey [25]. It should also be stressed that in addition to considering the capacity of commercially available chest protectors to reduce ventricular fibrillation, consideration must also be extended to the development of products that can adapt to sports with a high level of athletic movements. Movement or raising of the athlete's arms during athletic movement results in the chest protector sliding in different directions, which can allow projectiles to impact the chest wall without any impedance in areas specifically susceptible to commotio cordis [53].

Commotio cordis has provoked the demand for substantial innovation and design of chest protectors. The progression of the chest protector includes the implementation of new fabrics, material properties and varying degrees of thickness. Despite these improvements, there is a lack of emphasis towards a calculated approach regarding impact locations on the chest wall resulting in ventricular fibrillation. It is well known that impacts over the precordium, especially impacts directly over the cardiac silhouette may result in ventricular fibrillation [13]. However, current literature on the analysis of protection positioning on commercially available chest protectors is insufficient to create a capable design that defends these young athletes.

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.

3.3 Methods

3.3.1 Finite Element Simulations and Post Processing

Simulation cases were run using the CHARM-10 chest model developed at Wayne State University [54], a detailed finite element (FE) model representing an average 10-year-old child's chest. This detailed FE model contains all major anatomical structures based on detailed clinical scans of 10-year-old-children [35]. The model is comprised 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 parameter of 0.1 for soft tissue. This chest model has been validated based on data collected through cardiopulmonary resuscitation on live subjects [36] and impact data gathered on cadavers [37]. A baseball model with a radius of 37.5 mm and material properties based on the literature was developed in company with the validated chest model [49]. All baseball impacts to the chest were given an initial velocity of 17.88 m/s (40 mph). Previous research has shown that impact speeds greater than this cause severe cardiac damage, which is not uniform with commotio cordis instances [32]. Simulations were run using Ls-Dyna (LSTC, Livermore, Ca). Once completed, LS-PrePost2.4 was used for collecting and analyzing data from impacts.

3.3.2 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 directly towards the chest. 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. The baseball was placed 1 meter from the chest model to start.

3.3.3 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 included rib 3 (ULV), rib 4 (MLV) and rib 5 (LLV).

Left ventricle strain and pressure were analyzed and calculated by selecting all the elements that make up the left ventricle using the elout* output, which was then processed using Ls-PrePost2.4. 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 by using *Contact_Automatic_Surface_to_Surface in LS-DYNA, which is a penalty-based contact. The contact segments from the CHARM-10 model were

defined to be in contact with the solid baseball model. 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). ULV, MLV, and LLV rib deformations were calculated using approximately the same method as max rib deformation. Alternatively, when measuring rib deformations over the left ventricle, the location of measurement did not change when the impact location changed.

3.3.4 Contour Maps

Contour maps were designed using MATLAB R2018. The main code, Surfc, was used to generate a contour plot under the surface plot, allowing us to turn our 8x7 matrix design into a smooth contoured image to place over the CHARM-10 model. Jet was chosen as the contour map colour pattern.

3.4 Results

3.4.1 Left Ventricle Internal 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 3.1. A). Left ventricle pressure (kPa) was highest when impacts were directly over the left ventricle, reaching a peak internal pressure of 82.9 kPa (Figure 3.1. B). While left ventricular impacts (bottom left of the heart) generated high strain and pressure, it can be observed that impacts superior and to the left of the left ventricle also generated high strains (Figure 3.1. A).

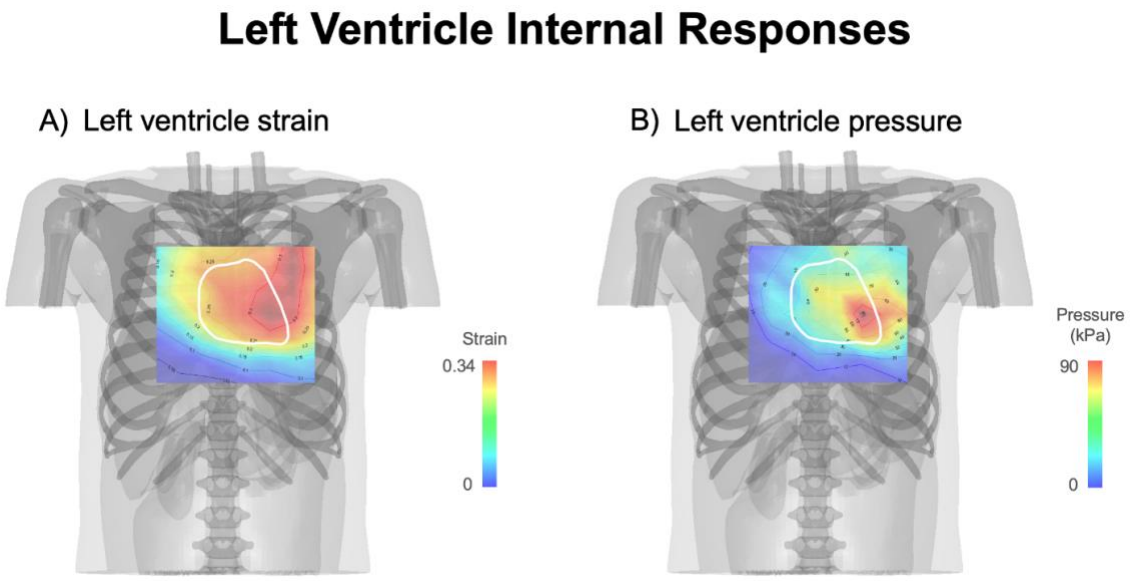

Figure 3.1 Left ventricle internal responses in relation to impact location: A) Left ventricle strain. B) Left ventricle pressure. Left ventricle strain ranges from 0 (blue) to 0.34 (red). Left ventricle pressure ranges from 0 kPa (blue) to 90 kPa (red).

3.4.2 Chest External Responses

Reaction force between the baseball and chest resulted in a scattered contour map, with no identifiable 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 3.2. A). Max rib deformation was highest with impacts directly over the left ventricle (36.2 mm) and slightly inferior (Figure 3.2. B).

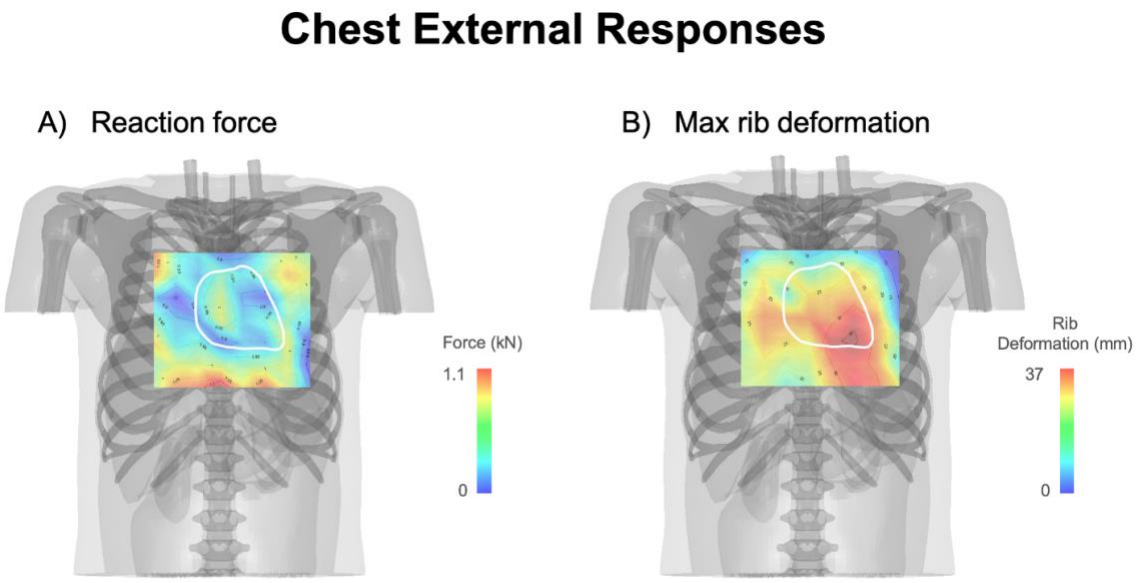

Figure 3.2 Chest external responses include A) Reaction force between the baseball and chest. B) Max rib deformation. Reaction force ranges from 0 kN (blue) to 1.1 kN (red). Max rib deformation ranged from 0 mm (blue) to 37 mm (red).

3.4.3 Rib Deformation at Left Ventricle

ULV (Figure 3.3. A), MLV (Figure 3.3. B) and LLV (Figure 3.3. C) displayed large rib deformation when impacts were aimed directly over their respective rib. LLV had the highest rib deformation at 35.5 mm, while MLV had a peak deformation of 33.0 mm, and ULV at 26.9 mm.

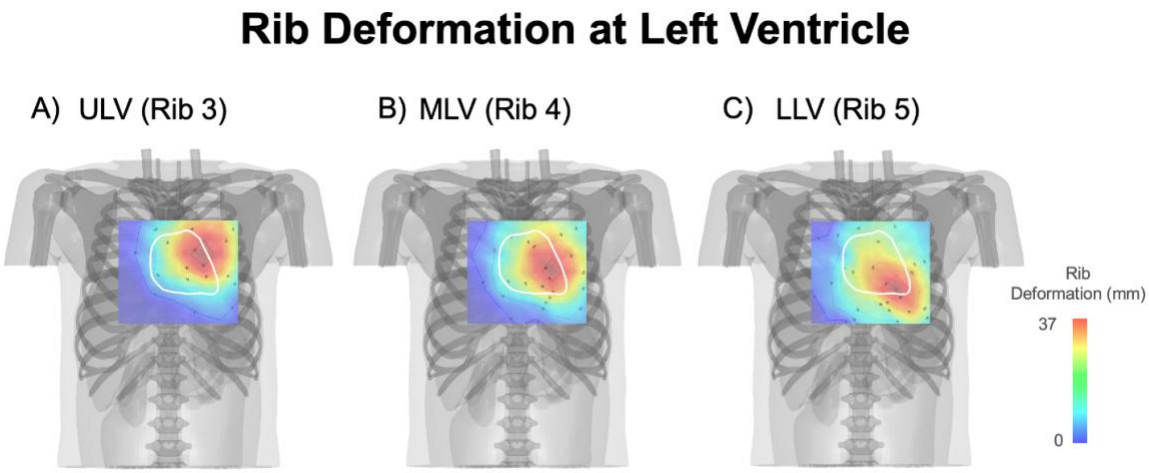

Figure 3.3 Rib deformation at the left ventricle. Rib deformation ranges from 0 mm (blue) to 37 mm (red). A) ULV (rib 3), B) MLV (rib 4), LLV (rib 5) represent all the ribs covering the left ventricle of the heart.

3.5 Discussion

This study used a computational approach to identify the hazardous baseball impact locations over the precordium to better understand how chest protectors can be designed to protect against commotio cordis. Through MATLAB, we created a visual representation from our 8x7 matrix, which was then developed into a contour map and accurately placed over our chest model. The left ventricle strain and pressure contour maps illustrate where to place more emphasis on chest protectors in future designs. While other contour maps such as reaction force between the baseball and chest wall illustrate why we believe reaction force as a means of commotio cordis injury metrics is not the most effective, contour maps of rib deformation over the left ventricle region support our suggestion of using it as an effective injury metric.

Our results display which impact locations generate the highest left ventricle strain and pressure. The introduction of analyzing left ventricle strain is specific to our studies, whereas other researchers have focused specifically on left ventricle pressure. We believe that looking at both injury metrics will help determine their influence towards inducing commotio cordis based not only on our current model, but from other studies alike [30, 50, 51, 53, 55].

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, as shown in Figure 3.1. A. 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 (Figure 3.4. A&B). Two small arrows pointing away from the baseball were used to identify the contact force direction upon impact (Figure 3.4. A). Due to the chest cavity not being completely flat, this caused the ball to have a unique impact on the ribs, resulting in the baseball creating a downward and inward force pushing into the left ventricle. The velocity arrow (Figure 3.4. B) displays the initial direction in which the baseball was traveling before contact, during a lateral view of the impact. These figures were taken 6 ms into the simulation, as this was when peak left ventricle strain was recorded.

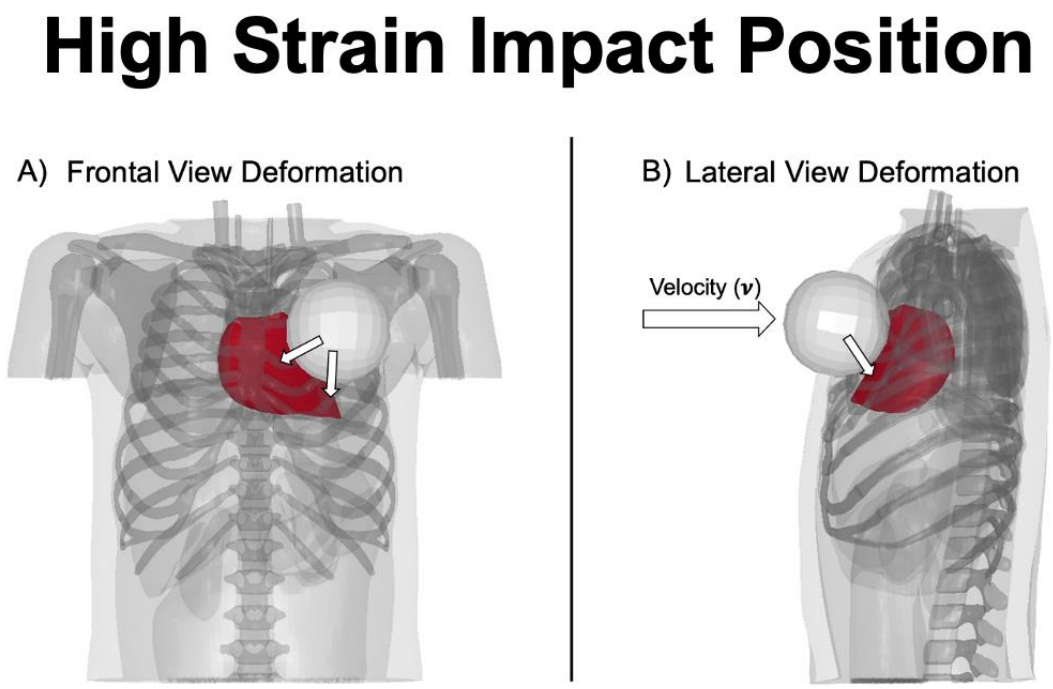

Figure 3.4 High strain impact position: How impact position 7x6, lateral and superior to the left ventricle, 6 ms into the baseball impact caused high strain of the left ventricle (0.34). A) Frontal view deformation of impact. White arrows illustrate the direction of contact force placed against ribs 3 (ULV) and 4 (MLV). B) Lateral view deformation of impact, white velocity arrow identifies the direction of initial velocity, small white arrow illustrates the direction of contact force placed against ribs (ULV & MLV).

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. [28]. Meanwhile, based on our results, we believe that past and current designs may have overlooked areas that require more protection 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 incidences of ventricular fibrillation.

As of 2017, NOCSAE introduced safety standards for commotio cordis prevention in chest protectors [29]. These standards are a significant step in developing safer designs, seeing as all chest protectors in baseball and lacrosse were required to adhere to the injury metrics. Currently, the NOCSAE surrogate model uses an upper load cell (ULC), lower load cell (LLC) and a cardiac load cell to measure the amount of force (N) impacting the surrogate model. Our data suggest that this model may be missing a potentially critical area superior and to the left of the cardiac silhouette (Figure 3.1. A). 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.

For the future design of chest protectors, the area that we suggest focusing on is superior and left of the cardiac silhouette, as our contour map shows that this region generates substantial left ventricle strain. Regarding material properties, the conventional closed-cell foams have been shown to easily deform upon impacts [5-7]. Meanwhile, creating a chest protector design that can spread the impact force across the chest, rather than a confined amount of force in one spot over the precordium, may be found to be helpful [56]. Drewniak et al. described pressure distribution to be an essential factor when testing chest protectors' ability to reduce ventricular fibrillation occurrences; there was a significant decrease when the pressure distribution increased [39]. Specifically, a chest protector from the Drewniak et al. study that had the largest area of pressure distribution, including the make-up of polypropylene beads instead of the traditional closed-cell foam, demonstrated the lowest ventricular fibrillation occurrence out of all other chest

protectors in the study. Therefore, integrating the knowledge of how impact positions influenced left ventricle strain/pressure with material studies and pressure distribution analysis, could help to create better chest protectors in the future.

Our study's limitation is that the CHARM-10 finite element model does not contain blood flow throughout the heart. Including blood flow would require extensive computational power but is a limitation that we intend to explore in the future to construct a more realistic simulation model. Due to our data being collected in 20 milliseconds 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.

3.6 Conclusion

We have described a computational approach that identifies vulnerable impact locations to reduce the incidence of death in commotio cordis cases in young baseball athletes. Our results have identified the most dangerous impact locations regarding left ventricle strain and pressure. Strain was found to be highest when impacts were located 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, maximum rib deformation, and rib deformations at the left ventricle. Reaction force did not seem to have a strong correlation with impact location, showing a peak 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, this study provided unique evidence that identifies where to emphasize protective materials for establishing effective chest protectors that will minimize the risk of commotio cordis.

Chapter 4

4 Conclusion and Future Work

4.1 Conclusion

Commotio cordis has already been responsible for over 200 deaths [5-7]. Although it is increasingly becoming more aware to the public, it continues to take the lives of children through sports and recreational activities. If we as scientists, cardiologists, and engineers, want to mitigate commotio cordis, we will need to conduct further research while building on the groundwork that Dr. Link and Dr. Maron have laid for us, including this innovative project that we believe should be included in the discussion. This final chapter will conclude the work that has been conducted over a two-year period of the M.E.Sc. program, while also providing preliminary results for future studies from our lab relating to commotio cordis.

4.1.1 Literature Review

The first chapter of this thesis took an in-depth literature review to fundamentally understand the topic of commotio cordis. This chapter established that commotio cordis is a tissue-level biomechanical issue involving the left ventricle of the heart that predominately affects children playing baseball. We explored how it is caused by a small object striking the chest while traveling approximately 40 mph (17.88 m/s) which must occur during the repolarization phase of the cardiac cycle, and how children might be more vulnerable to experiencing commotio cordis due to a weaker and narrower chest wall than that of a fully grown adult.

4.1.2 Development of Commotio Cordis Injury Metrics

The development of new commotio cordis injury metrics were described in chapter 2. This study found that left ventricle strain and pressure strongly correlated with ULV and MLV rib deformation, while they did not correlate with reaction force between the baseball and chest. Results from this chapter illustrated that we should place a more considerable emphasis on the heart's response during, and after initial impact while developing injury metrics based on this knowledge. Impact responses such as strain and pressure of the LV and deformation of the ribs located at ULV and MLV could prove to be appropriate measurements in the evaluation of chest protectors through commotio cordis testing methods.

4.1.3 Identification of Vulnerable Impact Locations

The third chapter identified the most vulnerable baseball impact locations over the chest using contour maps. Our findings revealed that the most dangerous impact positions for left ventricle strain were impacts over the left ventricle, as well as impacts slightly lateral and superior to the cardiac silhouette. Left ventricle pressure was highest when impacts were aimed directly over the left ventricle. The contour maps helped to illustrate that reaction force did not have a strong correlation with impact location, while also proving that rib deformations of the ULV, MLV and LLV were highest when impacts were directly over the corresponding rib, as expected. This chapter provided unique evidence

that identified where to prioritize protective materials to design effective chest protectors that will reduce the risk of commotio cordis.

4.2 Limitations

The CHARM-10 model, although validated through CPR and frontal pendulum impact testing [37], does not include blood flow or a beating heart. We understand this limitation and have identified it in previous chapters, but also believe that the time limit of each simulation in this study allows us to avoid this limitation as our analysis is a 20 ms simulation. Due to the short-lived nature of these simulations, the change in volume and pressure of the heart during relaxation, or in this case contraction, would not cause a noticeable change in the results of strain or pressure of the left ventricle – this was acknowledged in a parametric study in chapter two.

4.3 Future Research, Novelty and Significance

4.3.1 Expansion of Analysis to Other Sports

This thesis focused specifically on commotio cordis in children's baseball, while future studies from our lab will expand to other sports by analyzing commotio cordis in hockey and lacrosse. This expansion is justified due to the number of commotio cordis events that have occurred in these sports [6, 7, 25, 27]. Our hypothesis is that lacrosse ball impacts will be similar to baseball impacts due to the similarities in shape, size and weight. Meanwhile, hockey pucks can be manipulated to impact the chest while the puck is positioned at different angles which may significantly affect simulation results regarding left ventricle strain and pressure. This is because the dimension of a hockey

puck differs depending on which side of the puck impacts the chest (E.g., length side of puck vs. width side of puck).

4.3.2 Analysis and Optimization of Current Chest Protector Designs

The next step in our future research is to analyze current chest protectors available on the market that claim to prevent instances of commotio cordis. Not only will we test them with our new injury metrics, but we will also strive to optimize them by improving on the current design if we find that they are ineffective, or not as effective as they should be. We have successfully started the preliminary research for this next phase.

We ran a parametric study to analyze the effectiveness of a circular pad that attempts to cover the heart during impacts, based loosely off of the HART Pad by Unequal Technologies [41]. This chest pad claims to protect against commotio cordis instances and passes the NOCSAE commotio cordis impact testing. The circular pad is 14 cm in diameter, and 21 mm in thickness and made of numerous materials. As this was a preliminary study, material property was not the focus. We assigned a material property to the circular pad the same as the material property of the skin of the finite element model (Table 2).

Table 2. Chest protector material ID used in preliminary study.

Component	**Mass density**	**Elastic bulk modulus**	**Short-time shear modulus**	**Long-time shear modulus**	**Decay constant**
Chest protector	1.100e-006	0.00133	1.400e-004	4.400e-005	0.1

Once the circular pad was set up, it was placed over the heart, and a baseball with a regulation stiffness (2533 N/cm) and initial velocity of 40 mph (17.88 m/s) impacted the chest over the potentially vulnerable area (transverse 7 sagittal 6) which we identified in chapter three (3.1).

According to our preliminary findings, the circular pad, while covering much of the heart, still exposes vulnerable areas resulting in a left ventricle strain of 0.305 (~ 9 % decrease in left ventricle strain). Specifically, the area superior and lateral to the left ventricle remained exposed and/or was not fully covered during these preliminary impacts. Ideally, using the results we collect from this future study we plan to develop a finite element model of a chest pad that will completely protect the heart by reducing strain and pressure found in the left ventricle during impacts to the chest. Novel materials will also be explored.

4.3.3 Alternative Safer Positions to Protect from Impacts to the Chest

The aim of this future research chapter is to investigate alternative safer impact locations for children to prevent baseball impacts to the chest. The goal is to identify which positions children should turn or rotate their body when they do not have enough time to catch the baseball safely. We chose 3 alternative impact locations to test: far lateral, near lateral and posterior (Figure 4.1). These positions were chosen because they seemed to be practical alternatives for children to rotate their bodies to avoid direct impacts to the

chest. Once impact locations were picked, we ran the simulations and analyzed left ventricle strain and pressure.

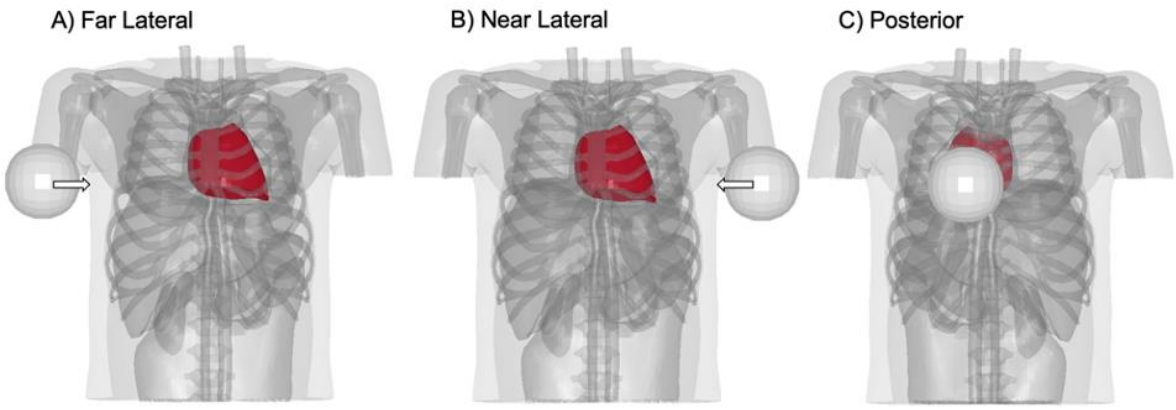

Figure 4.1 Alternative impact positions tested in preliminary study. A) Far lateral impacts in which the baseball (white) impacts the far lateral portion of the chest, away from the heart (red). B) Near lateral impacts where the baseball impacts the lateral portion of the chest closest to the left ventricle. C) Posterior impacts where the baseball was positioned at the vulnerable transverse t sagittal 6 location, now from the back.

Our preliminary results suggest that impacts far lateral to the heart are safe, as well as posterior impacts to the back as they generate very low left ventricle strain and pressure (Figure 4.2 & 4.3). Meanwhile, impacts near lateral to the heart generated a strain of 0.18 and a pressure of 30.99 kPa which may be considered dangerous when regarding our commotio cordis injury metrics.

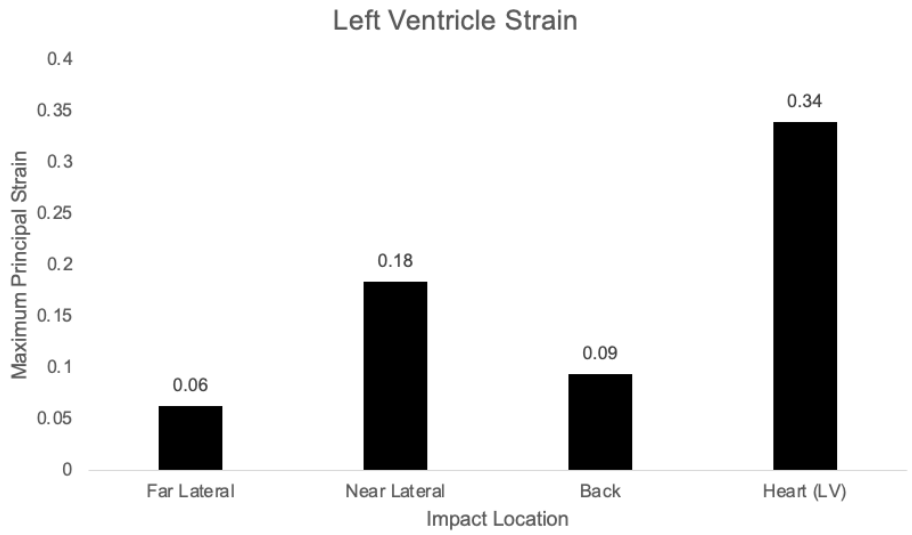

Figure 4.2 Peak left ventricle strain for alternative positionsas well as highest left ventricle strain case for comparison (right).

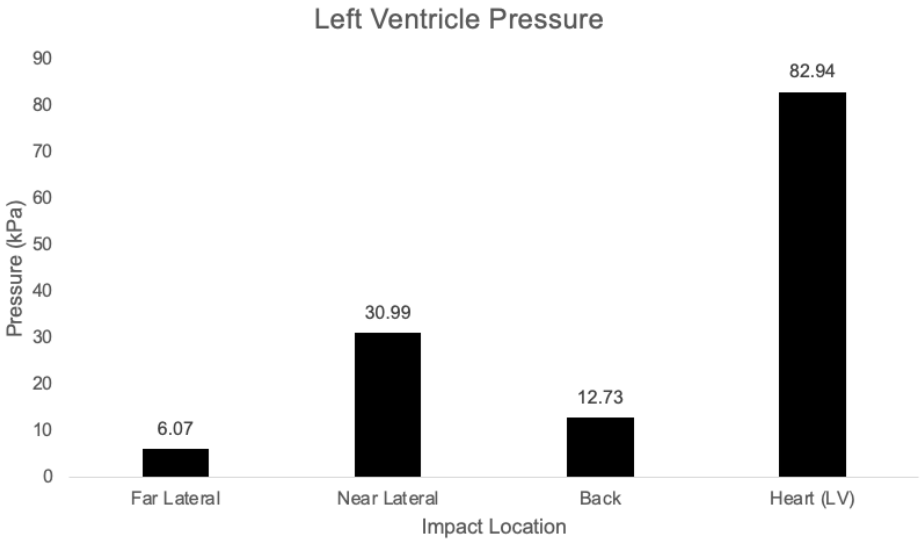

Figure 4.3 Peak left ventricle pressure for alternative positionsas well as highest left ventricle pressure case for comparison (right).

These alternative positions are strictly meant to prevent impacts directly over the chest which could result in instances of commotio cordis. We understand and acknowledge that these impacts could result in damage to other areas of the body where contact will be made, and that any impact from a baseball may result in injury to a child. We have identified these positions as a ‘last resort’ option specifically to prevent commotio cordis. Further research into this study will allow for a deeper understanding of these alternative positions, as the preliminary results only scratch the surface regarding which areas are considered safer to be impacted when considering commotio cordis.

4.3.4 Novelty and Significance

To the best of our knowledge, this serves as the first study to investigate left ventricle strain and pressure during commotio-cordis-relevant chest impacts in children. This thesis used the CHARM-10 model which as mentioned in previous chapters contains detailed anatomical regions and has been validated thoroughly, most notably through CPR validation.

Our major findings include developing commotio cordis injury metrics, as well as identifying the most vulnerable impact locations over the chest. The injury measurements included ULV and MLV rib deformation, which can be assessed outside the heart using a physical dummy, as well as left ventricle strain and pressure which are assessed inside the heart. Since the traditionally used reaction force injury metric did not always correlate with left ventricle pressure and strain, we strongly suggest rib deformation at the left ventricle for commotio cordis.

In our identification of most vulnerable impact locations over the chest, we used contour maps to create a visible representation of which areas are most dangerous when considering commotio cordis inducing impacts. We determined that impacts slightly lateral and superior to the left ventricle can generate high left ventricle strain (0.34). This can be explained by the way the baseball impacts the chest, which caused the ball to have a unique impact on the ribs, resulting in the baseball creating a downward force pushing the ribs into the left ventricle. Given that manufacturers may believe the protection is intended to protect the heart region, this study's analysis of vulnerable locations provides guidance for future chest protector design.

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