Understanding the Effects of Baseball Stiffness, Impact Location and Impact Velocity on the Risk of Commotio Cordis for Improved Protection

Kewei Bian, Jianan Bai and Haojie Mao

Abstract Commotio cordis occurs suddenly under blunt impacts to the chest without heart structural damage, causing sudden deaths in youth. We simulated 128 impacts including four different baseball stiffness levels (213, 353, 1114 and 2533 N/cm), 16 locations including aiming at the heart centre plus three offsets in the horizontal and three offsets in the vertical orientations, and two impact velocities (13.41 m/s and 17.88 m/s). Results demonstrated that peak pressures in the left ventricle happened quickly during 2 to 7 ms after the ball started to contact the thorax. Peak strains in the left ventricle happened during 5 to 10 ms. Impact location greatly affected left ventricle pressure and strain with the impact to the lower left of the heart (to the left ventricle) producing the highest strains. Increasing velocities increased both pressure and strain predictions. Interestingly, reducing baseball stiffness did not reduce heart pressure and strain. For example, a softer baseball (stiffness of 213 N/cm) only reduced strain and pressure by 0.4% and 1% when compared to a standard baseball (stiffness of 2533 N/cm), respectively, in a 13.41 m/s impact. Moreover, an overall softer baseball even generated slightly higher left ventricle strain and pressure.

Keywords Baseball stiffness, commotio cordis, finite element model, heart strain, heart stress.

I. INTRODUCTION

Commotio cordis refers to the disturbances of cardiac rhythm caused by a blow on the chest area without any penetration injury. Usually no sign of any structural damage were found on the heart but sometimes there were bruises on the impact area [1]. Although the occurrence rate of commotio cordis was low, it could happen in daily events like a backyard baseball play or an amateur hockey game [2]. In addition, the high fatality rate made the problem especially damaging to the society. It is reported that only 25% subjects survived when injured with the commotio cordis after resuscitation and defibrillation, given all 25 cases studied [1].

The specific time window required to deliver an impact to the chest for inducing commotio cordis made lab investigation challenging, with only a few swine experiments reported in the literature [3,4]. Studies reported that in general the impacts needed to be delivered at around 10 to 20 ms, or up to 30 ms, before the peak of T wave (Fig.2a and 2b) [3,5,6]. The biophysical mechanism of the specific time window could be explained by the abrupt mechanical activation of the K+ ATP channel [7]. However, how parameters of such abrupt mechanical activation including strain and pressure were developed in the heart remain to be further analysed. The PQR wave is when ventricles depolarize followed by the muscle contracts and T wave is where ventricles repolarization begins, and they start to relax. While the left ventricle starts to relax to its expanded shape, an external impact that compress the chest and contradicts to the relaxing expansion movement, and cause commotio cordis. Hence, strain and pressure responses of the left ventricle during the impact are of particular interests to the research community.

Prevention of commotio cordis has been investigated in the literature. Many chest protectors have been found to fail in reducing injury risk [8]. On the other hand, increasing the area of chest contact [9] and using softer baseball [6,10] were reported to reduce injury risk. However, it is still unclear how the heart responded differently with the changes of impact parameters and usage of softer baseballs. Moreover, the various impact environments with different impact locations and impact speeds added up to the difficulty in protecting the heart.

In this study, we adopted a 10 years old chest finite element (FE) model developed by Wayne State University,

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US, and validated it under higher-energy blunt impacts on post-mortem human subjects (PMHSs) [11] and exercised it under lower-energy cardiopulmonary resuscitation on live subjects [12,13]. We used the FE model to simulate baseball to chest impacts with different baseball stiffness, impact locations and impact speeds. We investigated strain and pressure response of the left ventricle, whose contraction is associated with the rising period of T wave. Using a full factorial design, we comprehensively analysed how different impact parameters affected heart biomechanical responses.

II. METHODS

Finite Element Simulation of Ball to Chest Impact and Post Processing

A detailed 10 years old FE chest model with 742,087 elements and 504,775 nodes was used. The model included anatomy structures such as the heart, lung, chest and kidney. A baseball with the radius of 37.5 mm was created. The *Automatic_Surface_To_Surface contact in Ls-Dyna (LSTC, Livermore, CA) was used to define the contact property between the baseball and the body. The whole termination duration of each simulation was 20 ms, with an output frequency of 1000 Hz.

After the simulation, we used LS-PREPOST to look into the pressure and strain in the left ventricle. We calculated the average of six elements experiencing highest strain and pressure. We used CFC 1000 Hz filter to process all the strain and pressure data.

Design of Experiments

Baseball Stiffness

Four baseball stiffness values defined using Reduced Injury Factor (RIF) 1, RIF 5, RIF 10, and standard [14] were simulated. RIF 1 stands for the stiffness of 213 N/cm, RIF 5 stands for the stiffness of 353 N/cm, RIF 10 stands for the stiffness of 1114 N/cm, and standard stands for the stiffness of 2533 N/cm.

Impact Location

Sixteen impact locations were simulated with the baseline direction aiming at the heart with location shown in Fig. 1 c & g. The baseball was moved to the median of the body for half-ball distance and full-ball distance, and moved to the lateral side of the body at half-ball distance, creating four parameters along the horizontal plane. The baseball was moved to the bottom of the body at half-ball distance and full-ball distance, and moved to the upper of the body at half-ball distance, creating four parameters along the vertical plane. Together, four times four, a total 16 impact locations were simulated.

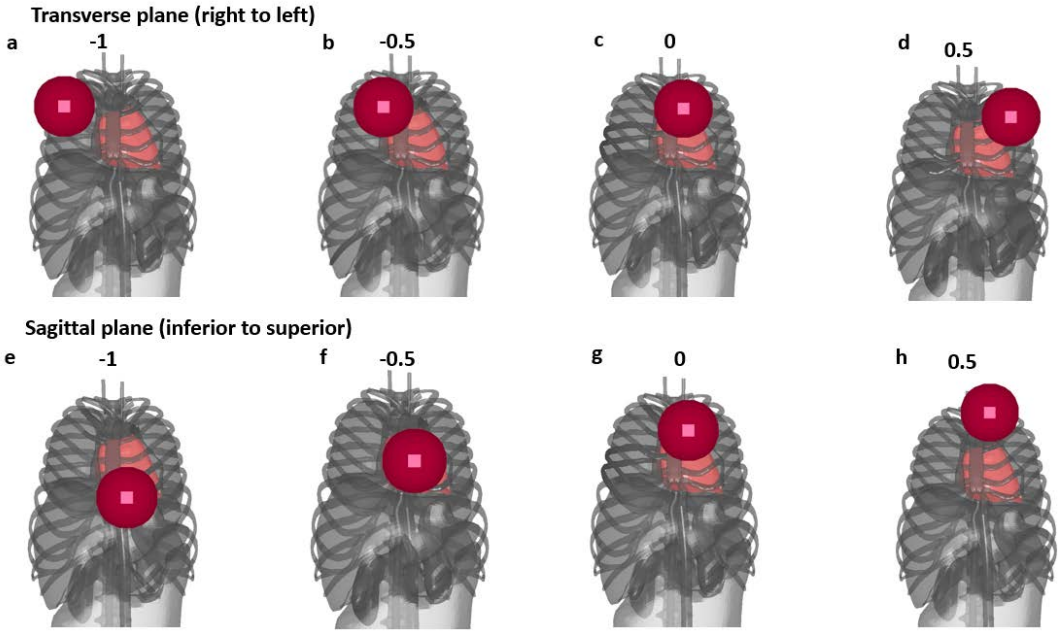

Fig.1 Impact locations. (a-d) Impact locations in transverse plane; (e-h) Impact locations in sagittal plane.

Impact Velocity

The based was triggered with impact velocities of 13.41m/s and 17.88m/s respectively to impact the chest. These velocity settings were consistent with the literature reporting the most damaging velocity range [6].

Simulation Matrix

With four baseball stiffness values, 16 impact locations, and two impact velocities, a total of 128 simulations (4x16x2) were conducted (Table 1), formulating a full factorial design.

TABLE I
FULL FACTORIAL DESIGN OF A TOTAL OF 128 IMPACT SIMULATIONS

A - Velocity (m/s)	13.41 (1)	17.88 (2)		
B - Distance from the heart (transverse plane)	-1 (1) Diameter of a baseball Medial	-0.5 (2) Radius of a baseball Medial	0 (3) Center of the heart	0.5 (4) Radius of a baseball Lateral
C - Distance from the heart (sagittal plane)	-1 (1) Diameter of a baseball Inferior	-0.5 (2) Radius of a baseball Inferior	0 (3) Center of the heart	0.5 (4) Radius of a baseball Superior
D. Stiffness (N/cm)	213 (RIF1) (1)	353 (RIF5) (2)	1114 (RIF10) (3)	2533 (Standard) (4)

We focused on peak strain and pressure of the left ventricle. We used Minitab (Minitab, LLC, State College, Pennsylvania, USA) to analyze the contribution of each factor by creating a Pareto chart. We also analyzsd major effects of each parameter and their interaction effect.

III. RESULTS

Left Ventricle Strain and Pressure Time Histories

For the impact aimed at the left ventricle, under impact velocity of 17.88 m/s, the highest left ventricle strain (0.37) and pressure (82.77 kPa) were produced at the lower left and upper right heart, respectively. The alignment between strain/pressure time histories of two representative cases and heart T wave are shown in Fig.2. Overall, peak pressures in the left ventricle happened quickly during 2 to 7 ms after the ball started to contact the thorax. Peak strains in the left ventricle happened during 5 to 10 ms after impact started.

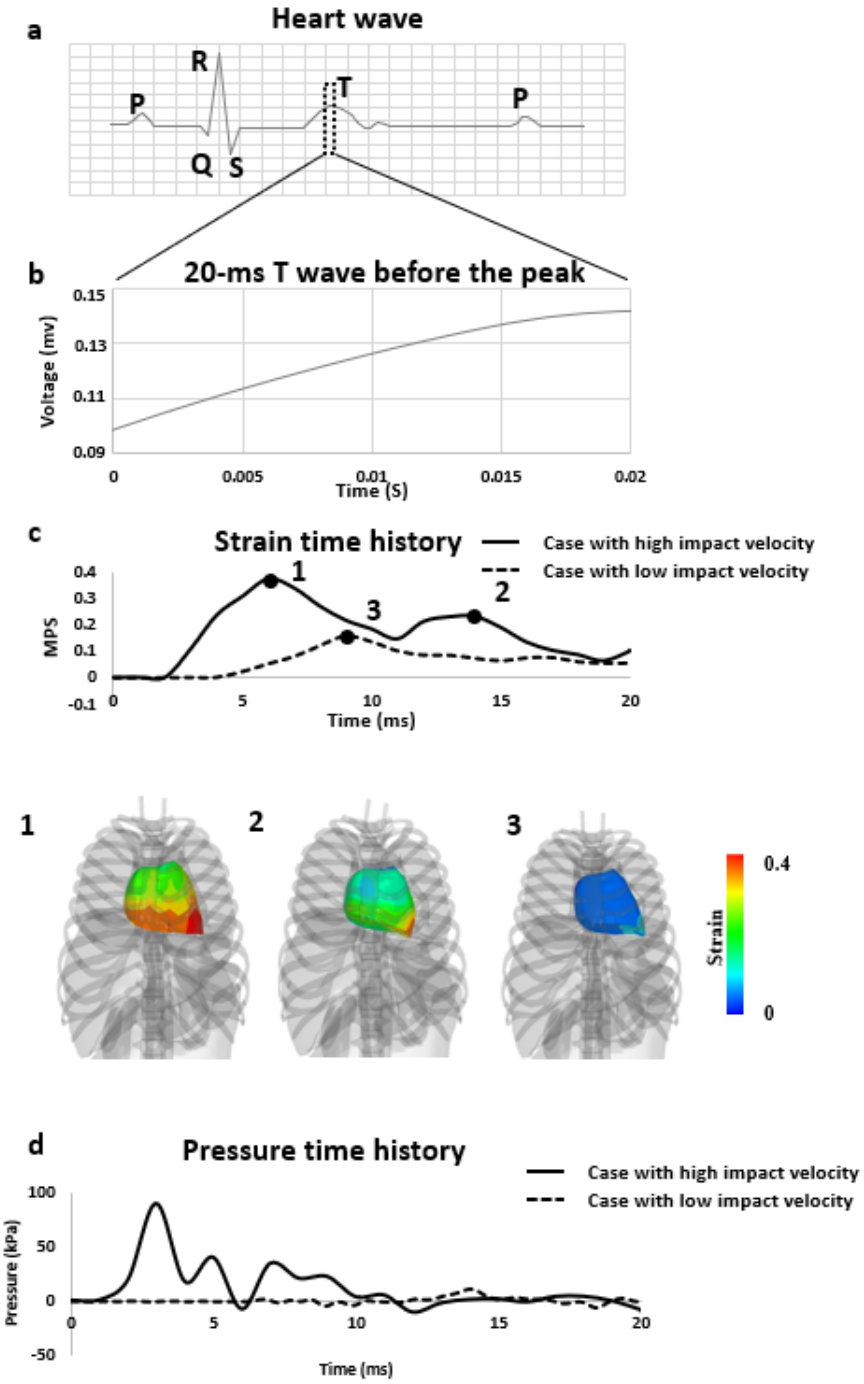

Fig.2. Heart wave/T wave and comparison to strain/pressure responses. (a) A typical heart wave, (b) The 20-ms duration of T wave before its peak, (c) Left ventricle strain time history, and (d) Left ventricle pressure time history.

Factors Affecting Left Ventricle Strain

The Pareto chart shows that the velocity played the most important role to the strain development in the left ventricle (Fig. 3). The location on the transverse plane (left to right) and the sagittal plane (top to bottom) had very similar effect to the strain, followed by the interaction effect of impact locations. Baseball stiffness had some effect on strain development but ranked only 5th.

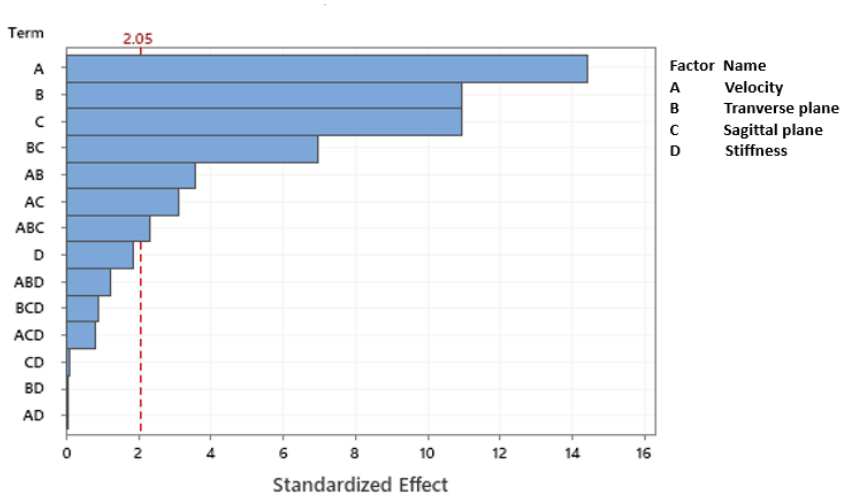

Fig.3. Pareto chart of factors on left ventricle strain.

The main effect plot shows that with the velocity increasing, left ventricle strain increased (Fig. 4). Moving the baseball from the median to lateral (right to left) increased left ventricle strains. Moving the baseball from the inferior to superior side (bottom to top) in generally increased left ventricle strain. Changing baseball stiffness almost did not affect left ventricle strain.

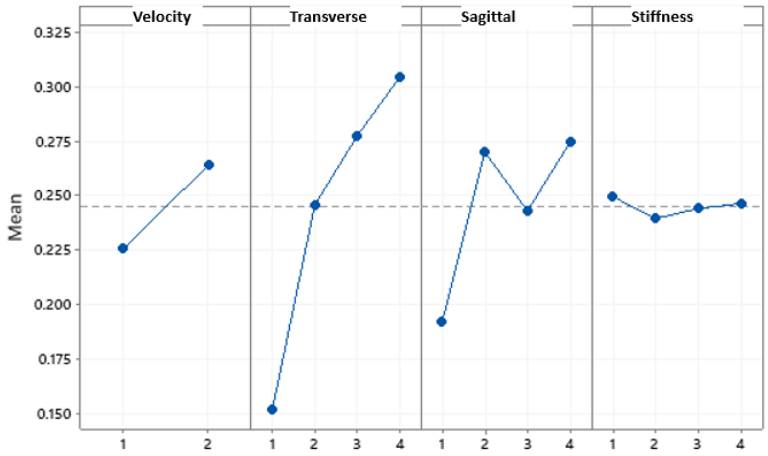

Fig.4. Main effect of each factor on left ventricle strain.

Factors affecting Left Ventricle Pressure

The Pareto chart shows that the location was the most important factor affecting left ventricle pressure (Fig. 5). The location of the transverse plane (left to right) was ranked as the #1 factor, followed by the location in the sagittal plane (bottom to top), and then the interaction effect of these two location planes. Compared to the location, the velocity was the fourth factor. The stiffness of baseball was found as a non-significant factor in affecting left ventricle pressure.

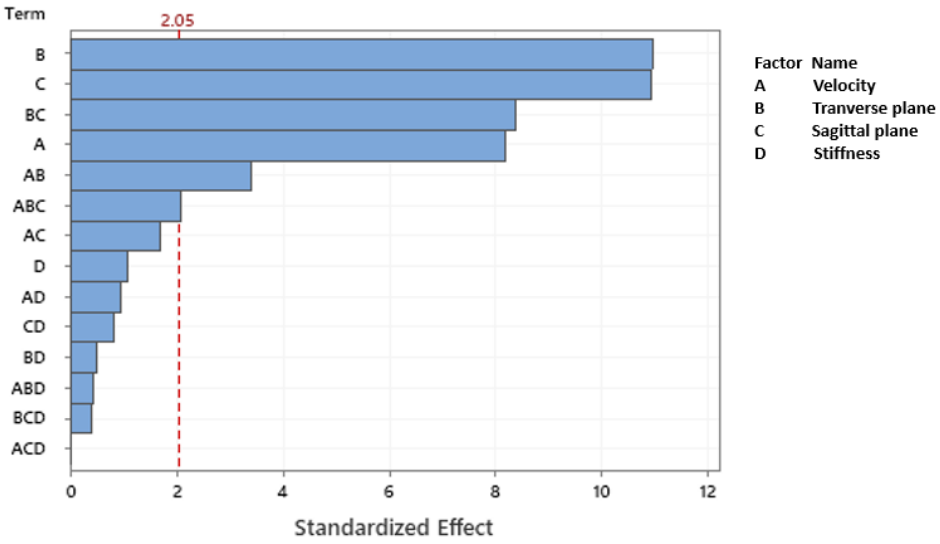

Fig.5. Pareto chart of factors on left ventricle pressure.

With velocity increasing, left ventricle pressure largely increased (Fig. 6). With the baseball moving from the medial to the lateral (right to left), pressure increased with the largest pressure being produced while the baseball facing the centre of the heart. The pressure slightly dropped while the baseball moved from the centre of the heart to the side for one radius of distance. Along the sagittal plane, the highest pressure was produced while the baseball was aimed at one radius of distance down to the centre. Regarding baseball stiffness, reduction of baseball stiffened increased left ventricle pressure, but with a small magnitude compared to the effects from other three factors.

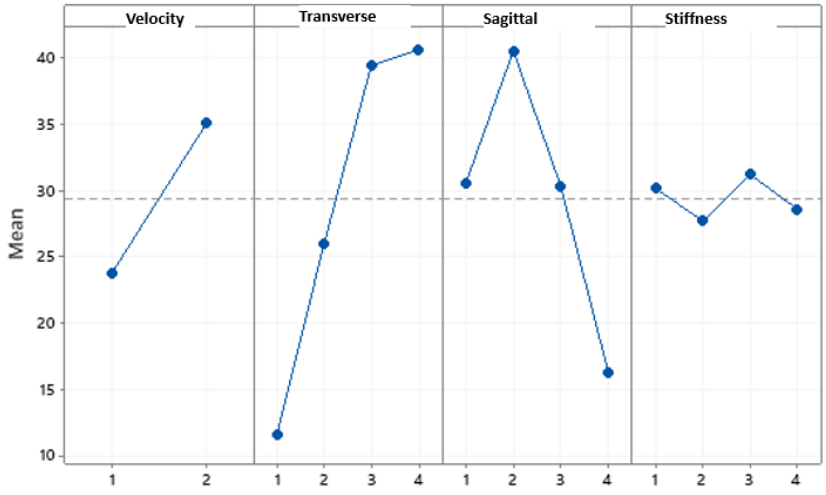

Fig.6. Main effect of each factor on left ventricle pressure.

Chest deflection analysis

Correlation between maximum chest deflection and pressure & strain response of left ventricle were investigated. There existed some correlation between maximum chest wall deflection with r square of 0.56 (Fig.7a), while the correlation between left ventricle and strain was weak with t square of 0.03 (Fig.7b).

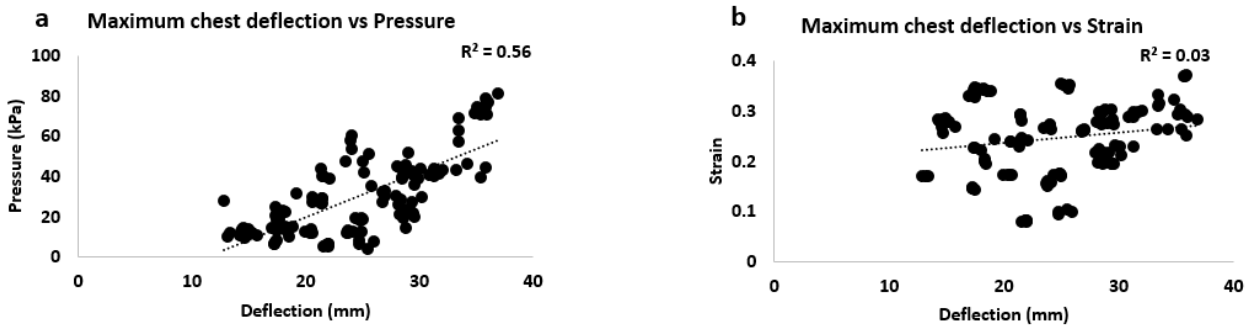

Fig.7. Correlation between maximum chest deflection and pressure & strain

IV. DISCUSSION

We used a detailed FE chest model and full factorial method to systematically evaluate the effects of different impact factors on the biomechanical response of the left ventricle. With the impact factors (velocity, location and stiffness of baseball) being comprehensively considered, both the strain and pressure response of the left ventricle were investigated. Among 128 simulations, we found that both the strain and pressure peaked within 10 ms after the baseball started to touch the chest, while pressure peaked earlier during 2 to 7 ms. In the literature, researchers have found that commotio cordis occurred in animal models when the impact was delivered in a narrow time window before the peak of T wave. Maron et. al [5] indicated that it could be dangerous when an impact occurred 10-20 ms before the peak of T wave. Link et. al. [3] demonstrated that nine out of 10 pigs got ventricle damage with impacts delivered 15-30 ms before the peak of T wave. Considering that there could be a couple of or tens of milliseconds from the impactor to be triggered and to reaching the chest, our results corroborate literature findings as strain and pressure peaked in the time when the left ventricle contracts the most. Moreover, our study demonstrates that strain lasted longer and could play an important role related to the specific time window (Fig.2c). For the cases with high impact velocity, when first peak occurred with the ball impacting the chest, the left ventricle got deformed and showed high strain as 0.4. After such a high-velocity impact, the heart got slight away from the intruding ribs. At the time of second impact (around 13 ms), although the ball slight separated from the chest, part of the left ventricle experienced the strain of 0.4, as the rib kept moving forward and further deformed the heart a little bit (Fig 2c). Taking the median 20 ms before T wave peak, we found that peak strains happened during a time that 80-90% of peak T wave have been reached, while peak pressures happened during a time that 75%-90% of peak T wave has been reached. Experimental measurement of heart strain has not been conducted and is recommended for future study.

The effects of impact velocity, location, and baseball stiffness were investigated. Based on the Pareto charts, impact velocity had the most important effect on left ventricle strain. Madias et. al. [15] studied the effect of different impact velocities on the incidence of ventricular fibrillation, and found that when velocity reached 40 mph (17.88 m/s), the incidence of ventricular fibrillation reached to the highest (around 70%). Also, our study demonstrates that impact location played an important role in left ventricle strain. We found that when a baseball was impacted at the centre or the bottom of the heart, the strains were high. Link et. al. [16] reported high incidence of ventricular fibrillation when the ball impacted the centre and base of the left ventricle. Lastly, one traditional conclusion in the field was that a baseball with low stiffness reduced the risk of ventricular fibrillation [3]. However, we found that the effect of baseball stiffness on left ventricle strain and pressure was limited. Rather, a softer baseball slightly increased pressure and strain responses. Overall, our findings on baseball stiffness, location and velocity support protection measures in absorbing energy and covering the heart, but not reducing baseball stiffness. There existed a correlation between chest deflection and pressure (Fig.7a), as the chest was squeezed by the ball to increase the left ventricle pressure. However, the correlation between chest wall deflection and left ventricle strain was weak (Fig.7b), as the strain was sensitive to the impact locations of the ball.

One of the limitations of this study is that the model used in this study did not include the fluid structures. An improvement could be to apply blood pressure to the interior surface of the heart during simulations. Overall, as we simulated high-rate, large deformation blunt impacts and investigated strain/pressure responses in the

time window of 20 ms, the effect of physiological pressure from the blood on mechanical strain and pressure of left ventricle wall was postulated as limited.

V. CONCLUSIONS

Both left ventricle strain and pressure peaked within a time window of 10 ms after touching the chest, corroborating the 10-30 ms specific window before the peak of T wave for delivering impacts that can induce commotio cordis in animals. This detailed FE study helped to address the tissue-level biomechanical mechanisms of commotio cordis. As impact location in transverse and sagittal plane played an important role in left ventricle strain and pressure, future studies on how the whole heart should be covered by protective gear during games are needed. Lastly, the conventionally used soft baseball was found to have a limited effect on reducing left ventricle strain and pressure response.

VI. ACKNOWLEDGEMENT

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