Analysing Peak Strain in the Right Ventricle during Blunt Thoracic Impacts: Revisiting Right Ventricular Involvement in *Commotio cordis*

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I. INTRODUCTION

Blunt trauma to the thorax resulting in cardiac pump failure, known as *Commotio cordis* (CC), is a fatal occurrence mostly seen in sports such as baseball [1]. This trauma induces rapid myocardial tissue stretch during a critical 20 ms of cardiac repolarisation, resulting in arrhythmias that can lead to death without immediate defibrillation [1]. These impacts to the chest alter crucial electrophysiological mechanisms of the heart, including action potential duration and amplitude [2], conduction velocity [3], and effective refractory period [4]. While CC predominately affects children, its significance extends to teenagers and adults. Studies using computational modeling [5-7] and youth swine [8-10] have advanced our understanding of CC, highlighting impact-induced strain in cardiac tissue and the potential for ventricular arrhythmogenesis. Notably, juvenile swine studies have emphasised the left ventricle's role in CC induction [9], while research on lambs has provided evidence for mechanoelectric feedback (MEF) in the right ventricle [11], and has further suggested that the right ventricle likely plays an important role in the development of CC [12]. When considering swine studies, it is important to compare their anatomy to that of a human. The human myocardium has the right ventricle positioned most anterior to the chest wall, with the left ventricle slightly posterior. Therefore, in CC cases in humans, the right ventricle is directly in line for the absorption of mechanical impacts. In swine anatomy, the ventricles are positioned more adjacent and a strike to the left ventricle will make contact with only the left ventricle [13]. This study aims to investigate the role of the right ventricle in CC using computational modeling of both ventricles during baseball impacts. This approach attempts to bridge the gap in current CC research by providing a preliminary analysis of the strain response from both ventricles from impacts, underscoring the potential involvement of the right ventricle in CC induction.

II. METHODS

The TOYOTA THUMS v7 (Total Human Model for Safety) Adult Male 50th Percentile (AM50) computational model was used in this study to analyse right and left ventricular average peak maximum principal strain values during 20 ms CC-inducing impacts. Our simulation recreated a real-life incident from a baseball practice where a player was struck directly over the heart by a baseball travelling an estimated 90 mph, based on eyewitness accounts and bruising. To increase accuracy, we scaled the THUMS model up from its standard dimensions (5'9" 172 lbs) to match the individual's stature (6'0" 190 lbs). Baseball material properties were matched to regulation balls. This study focused on understanding how different angles of impact – frontal, medial, and lateral (with variations based on the baseball's radius [36.8 mm] or diameter [73.6 mm]) – affect the ventricles. All five impacts were targeted towards the centre of the most anterior portion of the heart and can be seen in Fig. 1. Diameter medial (DM), radius medial (RM), radius lateral (RL) and diameter lateral (DL) were designated as abbreviations for angles of impact. Simulation environments were set up and analysed on LS-PrePost v4.8 and solved using LS-DYNA Version 971 MPP R11.2.2 Single Precision. Strain time histories of the right and left ventricles were averaged, and then the peak value of these averaged time histories was reported on, which is consistent with previous approaches [5-7].

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III. INITIAL FINDINGS

Both right and left ventricles experienced average curve peak strain values above 0.15 (Table I). The impact directions affected ventricular strain response, with medial impacts producing 0.019 (DM) to 0.028 (RM) higher strain in the right ventricle than the left ventricle. The highest right ventricular peak strain was seen in the RM case (0.189). In the lateral angles, the right ventricular strain response was once again higher than the left ventricular response by 0.028 (RL) and 0.021 (DL), respectively.

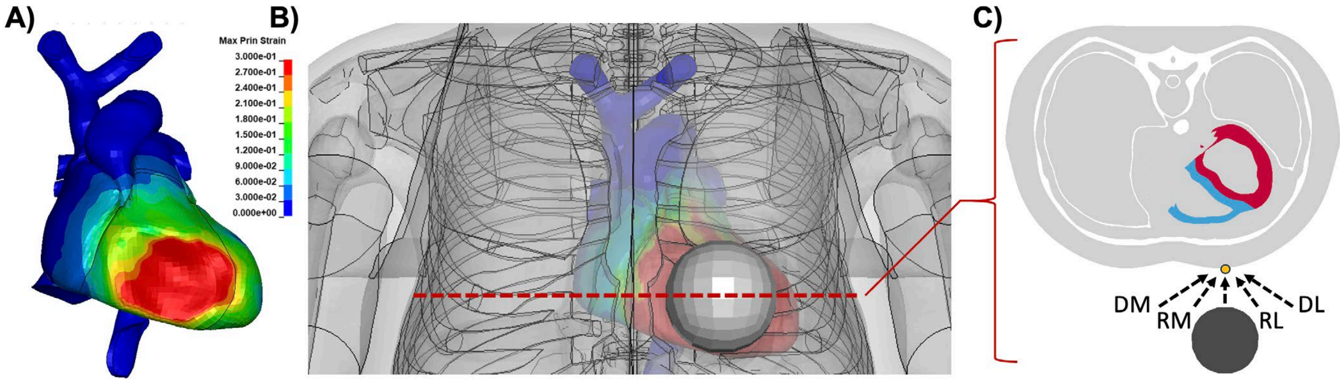

Fig. 1. A) Human heart 2 ms post-impact illustrating ventricular deformation and peak strain levels. B) Frontal view of the thorax with baseball (grey) impacting the chest. C) Top sliced view at the ventricular level marking the impact point (yellow dot) and baseball (black) with angular locations DM, RM, Frontal, RL, DL. Right ventricle is blue, left ventricle is red.

TABLE I

RIGHT AND LEFT VENTRICULAR AVERAGE PEAK STRAIN VALUES FROM EACH IMPACT LOCATION

Impact Location Direction	DM	RM	Frontal	RL	DL
Right Ventricular Peak Strain	0.182	0.189	0.184	0.183	0.176
Left Ventricular Peak Strain	0.163	0.161	0.158	0.155	0.155

IV. DISCUSSION

At this specific impact location, peak right ventricular strain was higher than peak left ventricular strain at all impact location directions, suggesting that impacts targeted to the most anterior portion of the human heart will result in a stronger mechanical response from the right ventricle than the left ventricle. Unexpectedly, both medial impacts caused the highest peak left ventricular strain, at 0.161 (RM) and 0.163 (DM), while first contacting the right ventricle. This was further understood by the angular effect of the baseball: while travelling from medial to lateral across the chest and striking the heart, the strain dispersion across the right ventricle spilled over to the left ventricle during deformation from impact, thus resulting in high ventricular peak strain for both.

While the findings from this study are novel, they are limited thus far and specific to one impact location based on a real-life CC case report. However, they provide a solid foundation for future investigation of right ventricular involvement in CC. Future work will continue to expand this study to reconstruct more real-life CC case reports and to further investigate ventricular differences based on impact directions with impact points over the apex of the left ventricle. This will allow for further comparison with previous swine studies, in which impact points from those studies were directly in line with the left ventricle.

V. REFERENCES

[1] Maron, B. J., *et al.*, *JAMA,* 2002.
[2] Franz, M. R., *et al.*, *Cardiovasc Res,* 1989.
[3] Reiter, M. J., *et al.*, *Circ Res,* 1988.
[4] Lerman, B. B., *et al.*, *J Clin Invest,* 1985.
[5] Dickey, G. J., *et al.*, *Comput Methods Biomech Biomed Eng,* 2021.
[6] Dickey, G. J., *et al.*, *J Biomech Eng,* 2022.
[7] Dickey, G. J., *et al.*, *Ann Biomed Eng,* 2023.
[8] Link, M. S., *et al.*, *New England J Med,* 1998.
[9] Link, M. S., et al., *J Am Coll Cardiol,* 2001.

[10] Link, M. S., *J Am Coll Cardiol,* 2003.
[11] Greve, G., *et al*., *Exp Physiol,* 2001.
[12] Chen, R. L., *et al.*, *Am J Physiol Heart Circ Physiol,* 2004.
[13] Link, M. S., *et al*., *Circulation: Arrhythmia and Electrophysiology,* 2012.

VI. LIMITATIONS

The strain measurement used in this short communication is maximum principal strain, which was used to correlate to the rapid myocardial tissue stretch that is seen in the heart during cases of CC. While traditionally the THUMS family of human body models have been used for injury prediction in traffic accidents, in this study we used the models for higher velocity impacts to the chest. We acknowledge that these material properties, the heart, and the human body model are yet to be further validated at these higher-rate loading conditions.