Analysis and Modeling of the Biomechanics of Brain Injury under Impact

DISSERTATION

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ABSTRACT

To better understand brain injury mechanisms and better predict brain injuries under impact, this work focuses on the analysis of experimental brain motion data and the development of brain injury models. An analytical method is used to separate the measured brain motion into rigid body displacement and brain deformation with a minimum total squared error. Under mild impact, it is found that the whole brain has nearly pure rigid body displacement, having a magnitude of 4 to 5 mm in translation and ±5 degrees in rotation. As the impact becomes more severe, the rigid body displacement is limited in magnitude for both translation and rotation, while the increased brain motion primarily is due to brain deformation, which is largest in the superior region of the brain.

Validated against the experimental brain motion data under low-severity impacts, new lumped-parameter brain injury models are developed to bridge the gap between simplified models that predict brain injuries based only on linear or angular accelerations and more complex finite element models that require complete knowledge of material properties and interface conditions. With proposed metrics for brain injury prediction, the new models are applied to more severe frontal and side impact tests and real-world car-pedestrian accidents. The results show that the new models are capable of predicting various brain injuries due to impact. Verified using a high-fidelity finite element model, sensitivity analysis indicates that the brain injury prediction is most sensitive to the brain moment of inertia, followed by the brain mass.
To my family
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Studies in:

<table>
<thead>
<tr>
<th>Field</th>
<th>Professors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dynamic systems</td>
<td>E.O. Doebelin, R. Singh, and R.G. Parker</td>
</tr>
<tr>
<td>Design</td>
<td>J.P. Schmiedeler, H.R. Busby, and C.H. Menq</td>
</tr>
<tr>
<td>Mathematics</td>
<td>M.R. Foster, J. Humpherys, and F.R. Tian</td>
</tr>
</tbody>
</table>
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abstract</td>
<td>ii</td>
</tr>
<tr>
<td>Dedication</td>
<td>iii</td>
</tr>
<tr>
<td>Acknowledgments</td>
<td>iv</td>
</tr>
<tr>
<td>Vita</td>
<td>vi</td>
</tr>
<tr>
<td>List of Tables</td>
<td>xiii</td>
</tr>
<tr>
<td>List of Figures</td>
<td>xv</td>
</tr>
<tr>
<td>Chapters:</td>
<td></td>
</tr>
<tr>
<td>1. Introduction</td>
<td>1</td>
</tr>
<tr>
<td>1.1 Motivation</td>
<td>1</td>
</tr>
<tr>
<td>1.2 Basic Head Anatomy</td>
<td>4</td>
</tr>
<tr>
<td>1.3 Traumatic Brain Injury</td>
<td>5</td>
</tr>
<tr>
<td>1.3.1 Diffuse Axonal Injury</td>
<td>6</td>
</tr>
<tr>
<td>1.3.2 Cerebral Contusion</td>
<td>6</td>
</tr>
<tr>
<td>1.3.3 Acute Subdural Hematoma</td>
<td>7</td>
</tr>
<tr>
<td>1.4 Background</td>
<td>7</td>
</tr>
<tr>
<td>1.4.1 Head Injury Criterion</td>
<td>7</td>
</tr>
<tr>
<td>1.4.2 Head Kinematics</td>
<td>8</td>
</tr>
<tr>
<td>1.4.3 Relative Brain Motion</td>
<td>9</td>
</tr>
<tr>
<td>1.4.4 Strain Deformation</td>
<td>10</td>
</tr>
<tr>
<td>1.4.5 Mathematical Models</td>
<td>11</td>
</tr>
<tr>
<td>1.5 Objectives</td>
<td>11</td>
</tr>
<tr>
<td>1.6 Organization</td>
<td>12</td>
</tr>
</tbody>
</table>
# 4. Background . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 111

4.3 Model Development . . . . . . . . . . . . . . . . . . . . . . . . . . . 113
4.3.1 Strain Measure for Sagittal Plane Model . . . . . . . . . . . . . 113
4.3.2 Strain Measure for Coronal Plane Model . . . . . . . . . . . . . 116

4.4 Material Properties of Brain Tissue . . . . . . . . . . . . . . . . . . . 117
4.4.1 Linear Elastic Brain Material . . . . . . . . . . . . . . . . . . . . 118
4.4.2 Linear Viscoelastic Brain Material . . . . . . . . . . . . . . . . . . 119

4.5 Application to Low-Severity Tests . . . . . . . . . . . . . . . . . . . . 120
4.5.1 Parameter Selection . . . . . . . . . . . . . . . . . . . . . . . . . . 120
4.5.2 Application of Sagittal Plane Model . . . . . . . . . . . . . . . . . 121
4.5.3 Application of Coronal Plane Model . . . . . . . . . . . . . . . . . 123

4.6 Application to More Severe Tests . . . . . . . . . . . . . . . . . . . . 126
4.6.1 Background . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 126
4.6.2 Application to Frontal Crash Tests . . . . . . . . . . . . . . . . . 127
4.6.3 Application to Side Impact Tests . . . . . . . . . . . . . . . . . . . 133

4.7 Discussion . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 144
4.8 Conclusions . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 146

5. Three-Dimensional Model and Its Application . . . . . . . . . . . . . . . . . . 147

5.1 Introduction . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 147
5.2 Background . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 148
5.3 Model Development . . . . . . . . . . . . . . . . . . . . . . . . . . . . 149
5.4 Strain Transformation . . . . . . . . . . . . . . . . . . . . . . . . . . . 151
5.5 Application to Low-Severity Impacts . . . . . . . . . . . . . . . . . . . 152
5.6 Application to NHTSA’s Crash Tests . . . . . . . . . . . . . . . . . . . 153
5.7 Car-Pedestrian Case Study . . . . . . . . . . . . . . . . . . . . . . . . . 154
5.7.1 Case Selection . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 154
5.7.2 Crash Simulation . . . . . . . . . . . . . . . . . . . . . . . . . . . 155
5.7.3 Parameter Modification . . . . . . . . . . . . . . . . . . . . . . . . 157
5.7.4 Reconstruction Results . . . . . . . . . . . . . . . . . . . . . . . . 160
5.8 Discussion . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 166
5.9 Conclusions . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 167

6. Sensitivity Analysis . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 169

6.1 Introduction . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 169
6.2 Background . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 170
6.3 Equations of Motion in State Space . . . . . . . . . . . . . . . . . . . . 174
6.4 Solution of State Sensitivity Equations . . . . . . . . . . . . . . . . . . . 176
6.4.1 Solution of Nominal State Equations . . . . . . . . . . . . . . . . 176
6.4.2 Solution of Trajectory Sensitivity Equations . . . . . . . . . . . . 178
6.4.3  Solution of Output Sensitivity Equations  . . . . . . . . . . . . . 180
6.5  Results of Sensitivity Analysis  . . . . . . . . . . . . . . . . . . . . . 180
  6.5.1  Results using Impulse Functions  . . . . . . . . . . . . . . . . . 181
  6.5.2  Results using Sinusoidal Functions  . . . . . . . . . . . . . . . . 185
6.6  Finite Element Model  . . . . . . . . . . . . . . . . . . . . . . . . . . 189
6.7  Results of Comparison  . . . . . . . . . . . . . . . . . . . . . . . . . . 191
6.8  Discussion  . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 194
6.9  Conclusions  . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 195

7.  Summary and Future Work  . . . . . . . . . . . . . . . . . . . . . . . . . . . 197
  7.1  Summary  . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 197
  7.2  Recommendations for Future Work  . . . . . . . . . . . . . . . . . . . 200
      7.2.1  Recommendations for Experimental Work  . . . . . . . . . . . . 200
      7.2.2  Recommendations for Modeling Work  . . . . . . . . . . . . . . 202
  7.3  Conclusions  . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 204

Appendices:

A.  Linear Viscoelastic Brain Tissue  . . . . . . . . . . . . . . . . . . . . . . . 205
     A.1  Kelvin Model  . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 205
        A.1.1  Constitutive Law  . . . . . . . . . . . . . . . . . . . . . . . . . 205
        A.1.2  Experimental Measurements  . . . . . . . . . . . . . . . . . . 206
     A.2  Standard Linear Solid Model  . . . . . . . . . . . . . . . . . . . . . 208
        A.2.1  Constitutive Law  . . . . . . . . . . . . . . . . . . . . . . . . . 208
        A.2.2  Stress Relaxation  . . . . . . . . . . . . . . . . . . . . . . . . . 209

B.  Nine Accelerometer Package  . . . . . . . . . . . . . . . . . . . . . . . . . 211

C.  Injury Risk Functions  . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 215
     C.1  Risk Functions of Skull Fracture  . . . . . . . . . . . . . . . . . . . 215
     C.2  Risk Functions in SIMon  . . . . . . . . . . . . . . . . . . . . . . . . 216

D.  Application of Three-Dimensional Model to NHTSA’s Crash Tests  . . . . . . 217

Bibliography  . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 221
## LIST OF TABLES

<table>
<thead>
<tr>
<th>Table</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.1 Configuration of low-severity impact tests.</td>
<td>19</td>
</tr>
<tr>
<td>2.2 Peak magnitudes of rigid body displacement for the whole brain.</td>
<td>30</td>
</tr>
<tr>
<td>2.3 Peak magnitudes of rigid body displacement for each column</td>
<td>46</td>
</tr>
<tr>
<td>2.4 Combination of NDTs for locating the brain deformation.</td>
<td>53</td>
</tr>
<tr>
<td>2.5 The averaged-normalized error of matching</td>
<td>56</td>
</tr>
<tr>
<td>2.6 The averaged-normalized error of matching various number of markers</td>
<td>56</td>
</tr>
<tr>
<td>3.1 Principle moments of inertia of the human head in the literature.</td>
<td>70</td>
</tr>
<tr>
<td>3.2 Parameters of the full brain injury model in the sagittal plane.</td>
<td>74</td>
</tr>
<tr>
<td>3.3 Parameters of the simplified brain injury model in the coronal plane.</td>
<td>102</td>
</tr>
<tr>
<td>4.1 Material properties of linear elastic brain tissue in the literature.</td>
<td>118</td>
</tr>
<tr>
<td>4.2 Summary of linear viscoelastic brain material in the literature.</td>
<td>120</td>
</tr>
<tr>
<td>4.3 Selected material properties of linear elastic and linear viscoelastic brain tissues.</td>
<td>121</td>
</tr>
<tr>
<td>4.4 Test configuration and results of strain measurement in the sagittal plane.</td>
<td>123</td>
</tr>
<tr>
<td>4.5 Test configuration and results of strain measurement in the coronal plane.</td>
<td>125</td>
</tr>
<tr>
<td>4.6 Measured head kinematics in the coronal plane for all six tests.</td>
<td>125</td>
</tr>
</tbody>
</table>
4.7 Configuration of NCAP frontal barrier crash tests. . . . . . . . . . . . . . . 128
4.8 Peak head angular velocities and accelerations in frontal crash tests. . . . . 129
4.9 Peak head linear velocities and accelerations in frontal crash tests. . . . . 130
4.10 Configuration of NHTSA’s side impact tests. . . . . . . . . . . . . . . . . . 137
4.11 Peak head angular velocities and accelerations in side impact tests. . . . . . 138
4.12 Peak head linear velocities and accelerations in side impact tests. . . . . 139
5.1 Test configuration and results of 3D strain measurement. . . . . . . . . . . 153
5.2 Selected cases from PCDS database for reconstructions. . . . . . . . . . . . 155
5.3 Profiles of nonlinear springs and dampers for the 3D model. . . . . . . . . 160
5.4 Comparing reported skull fractures with HIC values. . . . . . . . . . . . . 161
5.5 ASDH prediction with the simple 3D model. . . . . . . . . . . . . . . . . . 163
5.6 DAI prediction using the simple 3D model. . . . . . . . . . . . . . . . . . 164
5.7 Cerebral contusion prediction using DDM values in SIMon FE model. . . . . 165
6.1 Material properties used in the FE head model. . . . . . . . . . . . . . . . 190
6.2 Simulated cases with decreased brain size for the FE and planar models. . . 192
A.1 Experimental measurements of brain material properties. . . . . . . . . . . 207
# LIST OF FIGURES

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.1</td>
<td>Overview of the basic components of the human head.</td>
<td>4</td>
</tr>
<tr>
<td>2.1</td>
<td>Schematic of NDT and CG locations in an inverted head.</td>
<td>18</td>
</tr>
<tr>
<td>2.2</td>
<td>Schematic of two NDT position arrays.</td>
<td>21</td>
</tr>
<tr>
<td>2.3</td>
<td>Separated rigid body displacement and deformation for test C755-T2.</td>
<td>27</td>
</tr>
<tr>
<td>2.4</td>
<td>Separated rigid body displacement and deformation for test C755-T3.</td>
<td>28</td>
</tr>
<tr>
<td>2.5</td>
<td>Separated rigid body displacement and deformation for test C755-T5.</td>
<td>29</td>
</tr>
<tr>
<td>2.6</td>
<td>Separated rigid body displacement and deformation for test C383-T1.</td>
<td>31</td>
</tr>
<tr>
<td>2.7</td>
<td>Separated rigid body displacement and deformation for test C383-T3.</td>
<td>32</td>
</tr>
<tr>
<td>2.8</td>
<td>Separated rigid body displacement and deformation for test C383-T4.</td>
<td>33</td>
</tr>
<tr>
<td>2.9</td>
<td>Measured motion and rigid body displacement for test C755-T2.</td>
<td>34</td>
</tr>
<tr>
<td>2.10</td>
<td>Measured motion and rigid body displacement for test C755-T3.</td>
<td>35</td>
</tr>
<tr>
<td>2.11</td>
<td>Measured motion and rigid body displacement for test C755-T5.</td>
<td>36</td>
</tr>
<tr>
<td>2.12</td>
<td>Measured motion and rigid body displacement for test C383-T1.</td>
<td>37</td>
</tr>
<tr>
<td>2.13</td>
<td>Measured motion and rigid body displacement for test C383-T3.</td>
<td>38</td>
</tr>
<tr>
<td>2.14</td>
<td>Measured motion and rigid body displacement for test C383-T4.</td>
<td>39</td>
</tr>
</tbody>
</table>
3.3 The human brain with a full-ellipsoidal shape. . . . . . . . . . . . . . . . . 71
3.4 The human brain with a half-ellipsoidal shape. . . . . . . . . . . . . . . . . 72
3.5 Comparison of measured and simulated brain motions for test C755-T2. . . 76
3.6 Comparison of measured and simulated brain motions for test C755-T3. . . 77
3.7 Comparison of measured and simulated brain motions for test C755-T5. . . 78
3.8 Comparison of measured and simulated brain motions for test C383-T1. . . 79
3.9 Comparison of measured and simulated brain motions for test C383-T3. . . 80
3.10 Comparison of measured and simulated brain motions for test C383-T4. . . 81
3.11 Peak displacements in the sagittal plane in test C755-T2. . . . . . . . . . . 82
3.12 Peak displacements in the sagittal plane in test C755-T3. . . . . . . . . . . 82
3.13 Peak displacements in the sagittal plane in test C755-T5. . . . . . . . . . . 83
3.14 Peak displacements in the sagittal plane in test C383-T1. . . . . . . . . . . 83
3.15 Peak displacements in the sagittal plane in test C383-T3. . . . . . . . . . . 84
3.16 Peak displacements in the sagittal plane in test C383-T4. . . . . . . . . . . 84
3.17 Relative brain rotation in the sagittal plane for all six tests. . . . . . . . . . 85
3.18 Schematic of a simplified brain injury model in the sagittal plane. . . . . . 86
3.19 Schematic of relative brain displacement along the $X$ axis. . . . . . . . . . 87
3.20 Schematic of relative brain rotation about the $Y$ axis. . . . . . . . . . . . 88
3.21 Free body diagram of the brain for the simplified sagittal plane model. . . 90
3.22 Comparison of simplified and full sagittal plane models for test C755-T5. . 95
3.23 Comparison of simplified and full sagittal plane models for test C383-T4. 95
3.24 Schematic of the simplified brain injury model in the coronal plane. 98
3.25 Peak displacement in the coronal plane in test C755-T2. 104
3.26 Peak displacement in the coronal plane in test C755-T3. 104
3.27 Peak displacement in the coronal plane in test C755-T5. 105
3.28 Peak displacement in the coronal plane in test C383-T1. 105
3.29 Peak displacement in the coronal plane in test C383-T3. 106
3.30 Peak displacement in the coronal plane in test C383-T4. 106
3.31 Relative brain rotation in the coronal plane for all six tests. 107
4.1 Free body diagram for the upper hemispherical brain. 113
4.2 Free body diagram for the back hemispherical brain. 115
4.3 Free body diagram for the right hemispherical brain. 117
4.4 Shear strains under low-severity impacts using sagittal plane model. 122
4.5 Shear strains under low-severity impacts using coronal plane model. 124
4.6 Comparison of HIC15 and the maximum shear strain from the sagittal plane model with linear elastic brain tissue. 134
4.7 Comparison of CSDM (0.15) from SIMon and the maximum shear strain from the sagittal plane model with linear elastic brain tissue. 134
4.8 Comparison of HIC15 and the maximum shear strain from the sagittal plane model with linear viscoelastic brain tissue. 135
4.9 Comparison of CSDM (0.15) from SIMon and the maximum shear strain from the sagittal model with linear viscoelastic brain tissue. 135

xviii
D.1 Comparison of HIC15 and maximum shear strain from the 3D model in frontal crash tests. 218

D.2 Comparison of CSDM (0.15) from SIMon and maximum shear strain from the 3D model in frontal crash tests. 218

D.3 Comparison of HIC15 and maximum shear strain from the 3D model in side impact tests. 219

D.4 Comparison of CSDM (0.15) from SIMon and maximum shear strain from the 3D model in side impact tests. 219
CHAPTER 1

INTRODUCTION

1.1 Motivation

Injury is recognized as a major public health problem in the United States, as well as in the other countries. Injuries resulting from transportation-related accidents are the most frequent type of personal injuries, whether the accident occurs from travel on the ground, in the air, or on water. The majority of deaths and injuries result from ground vehicle transportation. According to the CDC Injury Research Agenda [21], automobile crashes resulted in 40,965 deaths in 1999 and were the leading cause of deaths in the United States among people ages 1 to 34. Each year, an additional 3.5 million people suffer nonfatal transportation-related injuries, causing about 4 million emergency department visits and 500,000 hospitalizations. The economic cost of motor vehicle crashes was more than $150 billion in property damage, lost productivity, and medical expenses in 1994 [21], which does not include those unquantifiable costs, such as suffering or the value of lives lost.

Among all types of injuries, brain injury is the most likely to result in death or permanent disability. An estimated 1.4 million people sustain a traumatic brain injury (TBI) each year in the United States. Of those, approximately 1.1 million are treated in an emergency department, about 235,000 are hospitalized and survive, and 50,000 die, which accounts for
one-third of all injury deaths [13]. However, many of those who are not hospitalized may experience long-term problems with headache, vision or hearing problems, sleep disorders, and mood changes. At least 5.3 million Americans - 2% of the U.S. population - currently live with disabilities resulting from TBI [112]. The cost of TBI totaled an estimated $56.3 billion in the U.S. in 1995 [112], reflecting substantial impacts on individuals, families, and society. These data present a critical need for more effective ways to understand the mechanisms of TBI and prevent brain injuries in automobile crashes.

Research on human brain injury resulting from automobile crashes has employed a variety of experiments involving animals, dummies, post-mortem human subjects (PMHS), and physical models to develop a better understanding of the mechanisms of brain injury. Recent studies have shown that brain injuries may be explained by the relative brain motion within the skull [119, 6], particularly for closed brain injuries without skull fractures. Therefore, understanding brain motion patterns under impacts is critical to understand the basics of brain injury mechanisms. Although relative brain motion has been directly observed and studied for decades, the patterns of relative brain motion with respect to the skull are not yet thoroughly understood. This study further investigates brain motion patterns using experimental brain motion data collected during low-severity impacts.

Mathematical models have also been widely used to study brain injury resulting from impacts, and recent studies are dominated by finite element (FE) models. Although offering detailed modeling of anatomical structures, these FE models have not been fully validated due to the lack of complete knowledge of the complex material properties and interface conditions of the human head. Simple models with analytical solutions for brain injury study have potential advantages such as the simplicity of parameter selection and the reduction of execution time. However, existing simple models are inadequate since none of
them has a validated measure for brain injury prediction. Therefore, this requires the development of a new simple model that can capture the key characteristics of brain responses and predict brain injuries.

The primary results of experimental tests and mathematical models have been proposed as indices of human head/brain injury that quantify the severity of damage due to impact. However, one element of both the experimental and mathematical modeling work that has not been adequately investigated to date is the sensitivity of the resulting injury predictions to small parameter changes in the experiment or the model. Clearly, it is not feasible to blindly execute a large number of physical tests or even to run a large number of high-fidelity FE models to determine to which parameters the injury predictions are most sensitive. Therefore, some fundamental understanding of the problem is needed beforehand to make the sensitivity analysis feasible.

Simple models with analytical solutions have the potential to provide insight into the influence of small parameter changes on the system response. However, existing simple lumped-parameter models are inadequate because they allow only linear or angular accelerations individually as the input [107, 66, 67]. Therefore, this work proposes a new lumped-parameter model to evaluate the sensitivity of injury prediction to small changes in a few parameters. The identified critical parameters through the sensitivity analysis will guide both experimental tests and mathematical modeling work, as well as standard establishment in injury biomechanics.

This work will bridge the gap between simplified models that predict injuries based only on linear accelerations of the head, such as in Stalnaker et al’s work [107, 108, 109], and more complex FE models whose predictions are dominated by angular accelerations, such as in Takhounts et al’s work [111]. Planar brain injury models will be developed
first based on the findings of experimental data analysis and applied to study brain injuries under impacts having dominant planar head kinematics. To accept three-dimensional (3D) head kinematics under more general impact scenarios, such as in car-pedestrian crashes, a simple 3D model will be developed for better injury prediction.

1.2 Basic Head Anatomy

To help explain and discuss the mechanics of brain injuries, the basic anatomical components of the human head are introduced in this section. The outer surface of the head is covered by the scalp, which is a soft tissue layer with a typical thickness of 5-7 mm [26]. As shown in Figure 1.1, the bony case that houses the brain is the skull, which is relatively rigid and protects the brain from injury.

![Figure 1.1: Overview of the basic components of the human head (duplicated from [10]). Left: sagittal section of the brain. Right: components of the meninges.](image)

Between the skull and brain are the meninges, consisting of three layers of soft tissue protecting the brain. As shown on the right of Figure 1.1, the three layers of the meninges,
from outermost layer inward, are the dura mater, the arachnoid, and the pia mater. The dura mater covers the inside of the skull and creates two special folds, the falx and tentorium, which separate the brain into left and right halves and upper and lower parts, respectively. The second layer of the meninges is the arachnoid, which is a thin layer of membrane made up of delicate tissue and blood vessels of varying sizes. The space between the dura mater and the arachnoid is called the subdural space. The layer of the meninges closest to the brain surface is the pia mater, which has blood vessels connecting to the brain tissue. The space that separates the arachnoid and the pia mater is called the subarachnoid space, wherein the cerebrospinal fluid (CSF) is located. The CSF acts as a cushion to protect the brain by absorbing shocks under impacts [91]. The human brain consists of three primary parts: the brainstem, the cerebellum, and the cerebrum. The cerebrum is the major part of the brain, and it is divided by the falx into two halves, the left and right cerebral hemispheres. According to the location, each hemisphere consists of the frontal, temporal, parietal and occipital lobes, and each lobe controls different functions of the brain.

1.3 Traumatic Brain Injury

Brain injury is used to indicate acute traumatic damage to the central nervous system (CNS). The broader term head injury is often used to also include skull fractures and soft tissue damage to the head. Head injury is classified into two categories: open and closed head injury [115]. Open head injuries occur when both the scalp and skull are penetrated due to serious skull fractures. Closed head injuries are often referred to as brain injuries when the skull remains undamaged. The severity of the injuries depends on the severity of impacts, which determines the resultant forces acting on the head. This work primarily focuses on closed head injuries without significant damage to the skull. Three main
brain injury mechanisms are recognized in modern research: diffuse axonal injury (DAI), cerebral contusion, and acute subdural hematoma (ASDH).

### 1.3.1 Diffuse Axonal Injury

DAI is one of the most common types of TBI, and it accounts for almost 50% of total TBI admissions [89], which most commonly are the result of vehicle accidents. DAI typically results in the loss of consciousness (LOC) and is one of the major causes of persistent vegetative state after severe head trauma [120]. Due to the shear forces associated with large accelerations experienced by the head, the brain tissue undergoes movement and deformation inside the skull. The movement and strain deformation of brain tissue produces either stretching or tearing of axons. Overstrained axons lose their ability to transport the signal in the brain. Small blood vessels deep in the brain are also likely damaged causing bleeding.

### 1.3.2 Cerebral Contusion

Cerebral contusion is a bruise of the brain tissue. It results from the bouncing of the brain against the rigid bone of the skull due to the incompressibility of brain tissue [101]. Contusions are generally formed at two different locations in two different ways: direct trauma and deceleration. Direct trauma occurs at the site of the impact to the brain, called coup injury. Deceleration causes injury, called contre-coup injury, at the opposite site when the brain bounces off the skull as it has moved away from the site of the impact. Contusions are more likely to result in hemorrhage than is DAI, with microhemorrhage often observed on the surface of the brain. Swelling within the brain may occur in this case due to the stress state. Frequently associated with edema, contusions are most likely to cause increases in intracranial pressure.
1.3.3 Acute Subdural Hematoma

Intracranial hemorrhaging is a potential life-threatening consequence of a head injury and forms a hematoma, which is a mass caused by the collection of leaked blood that may continue to enlarge after injury [33]. By their locations within the skull, hematomas are classified into three different categories: subdural hematoma, epidural hematoma, and intracerebral hematoma. Subdural hematoma occurs when the bridging veins between the surface of the brain and its outer layer dura mater are stretched and torn because of the relative motion of the brain with respect to its covering. An epidural hematoma develops between the skull and dura, and an intracerebral hematoma is located within the brain tissue itself. For closed brain injury during automobile crashes, ASDH is the type that frequently occurs.

1.4 Background

This section provides a brief overview of the development of various head injury criteria. The historical literature studying head and brain injuries is also briefly discussed. This background information is general, while detailed literature review is given in each chapter.

1.4.1 Head Injury Criterion

Using animal models, Gurdjian and Webster’s work [39] is one of the first studies related to the mechanics of head injury. Since then, head injury biomechanics has been studied for decades using various methods. However, the only injury criterion widely used today is the Head Injury Criterion (HIC), which was first introduced as a curve fit to the Wayne State Tolerance Curve (WSTC), relating linear head acceleration to skull fracture.
First generated by Lissner [63] based on experimental data using PMHS, the WSTC curve correlates head acceleration and time duration of impacts with skull fracture. This curve was finalized by Patrick et al. [90] with more experimental data involving animals, PMHS, and clinical research with various impact durations. Gadd [30] developed the Gadd Severity Index (GSI) by fitting the WSTC curve with additional long pulse duration data obtained by Eiband [24]. Versace [116] first introduced the HIC criterion by proposing different formulations to calculate GSI using the average acceleration pulse. The HIC criterion was then modified by the National Highway Traffic Safety Administration (NHTSA) to provide a better comparison to long-duration human volunteer tests [78]. In 1972, the Federal Motor Vehicle Safety Standards (FMVSS) No.208 for occupant crash protection adopted HIC to replace GSI. The HIC criterion has the following expression,

\[
HIC = \max \left\{ \frac{1}{t_2 - t_1} \int_{t_1}^{t_2} a(t) \, dt \right\}^{2.5},
\]

where \( a(t) \) is the time history of the resultant linear acceleration (in g) at the center of gravity (CG) of the head. \( t_1 \) and \( t_2 \) are any two arbitrary times (in seconds) during the acceleration pulse. In 1986, the time interval was reduced to 30 msec. The latest regulation uses 15 msec as the time interval, and the critical HIC value is 700 for the 50th percentile male and 5th percentile female [19].

1.4.2 Head Kinematics

Head input kinematics have been used as the head injury predictor for decades since the introduction of HIC, which only relates linear head acceleration to skull fracture. However, closed head injuries without skull fractures have been the research focus in more recent studies. Currently, linear and angular head accelerations have been proposed as the two major causes of brain injury under impacts. Ono et al. [86] produced the human head
impact tolerance curve called JHTC, indicating the threshold of linear head acceleration and its duration resulting in concussion. Margulies and Thibault [68] proposed a threshold of angular kinematics for TBI as the combination of 7,000 rad/s$^2$ of peak acceleration and 70 rad/s of peak change in velocity. Regarding the role of linear and angular accelerations in head injury, some early research contends that linear accelerations are a main cause of brain injury [38, 84, 86], while other work argues that angular accelerations are more injurious [32, 33, 68]. A number of recent studies, however, suggest that both linear and angular accelerations are significant causes of brain injury [81, 52, 54, 4, 128].

1.4.3 Relative Brain Motion

To understand brain injury mechanisms, relative brain motion with respect to the skull has been studied for decades. Relative brain motion was first directly observed by Pudenz and Shelden [94] through replacing the upper portion of Macaque monkey skulls with Lucite calvaria. Shatsky et al. [100] and Stalnaker et al. [110] both observed significant relative brain motion with respect to the skull using high speed cineradiographic films. More recently, neutral density targets (NDTs) were implanted in the brain tissue, and the paths of the NDTs were tracked during impacts by a high-speed x-ray system [1, 41, 53]. The relative brain motion was found to have a magnitude of 5 mm under low-severity impacts. Careful analysis of the experimental results in these studies indicates that the linear and angular movements of the brain with respect to the skull are coupled. Magnetic resonance imaging (MRI) has also been used to study brain motion. Ji et al. [50] showed that in vivo human brain motion with respect to the skull is about 1-2 mm as the head rotates voluntarily from modest extension to full flexion. They also concluded that the cerebellum rotates 2.7-4.3 degrees with respect to the skull. Although the patterns of relative brain

9
motion with respect to the skull have been studied by many researchers, they are not yet thoroughly understood.

1.4.4 Strain Deformation

Strains in the brain tissue have been recognized by many researchers as one of the most important factors causing TBI during impacts [68, 5, 76, 111]. Strain rate and the product of strain and strain rate were also proposed to predict mild TBI by reconstructing some NFL football collisions [129]. Various experiments have been conducted to investigate the tissue-level response of brain tissue, trying to understand brain injury mechanisms. Based on experimental data from primates, Margulies and Thibault [68] concluded that strains ranging from 0.05 to 0.10 correspond to moderate-to-severe DAI. Bain and Meaney [5] conducted experiments by stretching multiple right optic nerves from an adult guinea pig to produce axonal injuries. They found that functional impairment occurs at a threshold of 0.18 Lagrangian strain. Morrison III et al. [76] studied the relationship between mechanical stimuli and the resultant biological response of living brain tissues using a hippocampal slice of a Wistar rat brain. They concluded that mechanical deformations less than 0.10 Lagrangian strain are not injurious at various strain rates and that mechanical deformations greater than 0.20 Lagrangian strain induce significant levels of cell injury depending on strain rates, which probably lie between 10 and 50 s\(^{-1}\). Maxwell et al. [69] concluded that a 0.20-0.30 stretching of a neuron’s membrane leads to a chemical poisoning that causes neuronal death after around twelve hours. Under mild occipital deceleration using human volunteers, Bayly et al. [7] measured brain deformation of typically 0.02-0.05 during the impacts. In general, these studies suggest that the threshold of strain causing brain injury is close to 0.20, depending on strain rate.
1.4.5 Mathematical Models

Mathematical models have been widely used to study brain injuries in recent years. Early modeling work focused on lumped-parameter models, such as Slattenschek and Taufkirchen’s [104] translational model consisting of two masses connected by a spring and a damper in parallel. This model was used and modified by other researchers to study head injuries [11, 107]. Other studies focused on head/brain responses under pure rotational loads using simple mathematical models [27, 12, 64, 65]. More complicated lumped-parameter models include Alem’s [3] twelve-degree-of-freedom model, consisting of five lumped masses, ten linear springs, and three torsional springs. More recently, Young [124] developed an analytical model for predicting the responses of a fluid-filled shell impacting a solid sphere. More modern approaches are dominated by FE models. Zhou et al’s [130] 3D FE head model with detailed features was used for accident reconstruction to investigate injury mechanisms. Several other 3D FE head models [126, 58, 111, 43] were developed recently and validated using the experimental brain motion data reported by Hardy et al. [41]. Although these FE models offer detailed anatomical structures, further validation requires better knowledge of the complex material properties and interface conditions of the human head, as well as more experimental data of brain motion and strain deformation.

1.5 Objectives

The objectives in this study are: 1) to analyze experimental data for a better understanding of brain motion patterns and therefore, brain injury mechanisms under impact, 2) to develop and validate planar brain injury models that can be dealt with analytically for sensitivity analysis, 3) to determine the parameters to which injury prediction is most
sensitive and provide guidelines for the development of computational models and experimental tests, 4) to verify the findings of the sensitivity analysis by running a series of FE simulations with changing parameters, 5) to employ the planar models with a simple strain measure for brain injury prediction in NHTSA’s frontal and side impact tests, 6) to combine the planar models into a 3D model that accepts 3D head accelerations for brain injury prediction, and 7) to reconstruct real world car-pedestrian accidents using the 3D model and compare with the SIMon FE head model.

1.6 Organization

Chapter 2 analyzes the experimental brain motion data collected by Hardy et al. [41] during low-severity impact tests using PMHS. An analytical method is used to separate the measured brain motion into rigid body displacement and brain deformation. The brain motion patterns for the whole brain are analyzed. This chapter also investigates the regional brain motion patterns and identifies the regions where brain deformation is dominant under impacts.

Chapter 3 presents relatively simple brain injury models to bridge the gap between the very simple HIC criterion and more complex FE models. This chapter introduces new planar models in the sagittal and coronal planes that are used to study brain injury. The model development is based on the results of the experimental data analysis. The model is validated using the experimental brain motion data introduced in Chapter 2.

Chapter 4 proposes a simple measure of strain for brain injury prediction based on the planar brain injury models developed in Chapter 3. The proposed strain measure is validated under low-severity impacts and applied to more severe, realistic frontal and side
impact tests for brain injury prediction. The results from the simple models are compared with the HIC values and the results from the SIMon FE head model.

Chapter 5 develops a simple 3D brain injury model through the combination of the planar models developed in Chapter 3 so that 3D head kinematics can be used as inputs to have a more comprehensive study of brain injury. Injury metrics are proposed for predicting various brain injuries under more general impact scenarios. The simple 3D model is applied to reconstruct selected car-pedestrian accidents. The results are compared with the HIC values and the reconstruction results using the SIMon FE model.

Chapter 6 performs sensitivity analysis to determine the parameters to which injury prediction is most sensitive. It provides guidelines for the development of experimental tests and computational models. The sensitivity of injury prediction is obtained analytically with simple inputs using the simple sagittal plane model developed in Chapter 3. A high-fidelity FE head model is used to verify the findings by running a series of simulations with changing parameters.

Chapter 7 summarizes the findings and contributions of this work. It also recommends future work.
CHAPTER 2

BRAIN MOTION DATA ANALYSIS

2.1 Introduction

To better understand the mechanisms of human brain injury, research has employed a variety of experimental tests and mathematical models. The primary results have been proposed as indices of human brain injury that quantify the severity of damage due to impact. Three mechanisms of closed brain injury are widely accepted: DAI, ASDH, and cerebral contusion. These mechanisms may be explained by relative brain motion within the skull [119, 6]. Although the patterns of relative brain motion with respect to the skull have been studied for decades, they are not yet thoroughly understood.

This chapter investigates the relative brain motion patterns under low-severity impacts. An analytical method is used to separate the measured brain motion into rigid body displacement and brain deformation. The significant findings of this chapter are the relative rotation and translation of the whole brain with respect to the skull. Regional brain motion is also studied to identify local motion patterns of the brain under impact. The results of this chapter form the foundation of the model development and validation for the brain injury study in the rest of this work.
2.2 Background

Relative brain motion was first directly observed about sixty years ago using translucent calvaria. Pudenz and Shelden [94] observed relative brain motion through replacing the upper portion of Macaque monkey skulls with Lucite calvaria and recording the patterns of brain motion using high-speed cinematography. At a later time, Gosch et al. [37] used Lexan calvaria on Rhesus monkeys.

In the following years, radiographs have been used to observe relative brain motion. Shatsky et al. [100] conducted high speed cinefluorographic studies on anesthetized primates with blunt impacts to the head in the sagittal plane. Their results showed that the brain underwent significant displacements within the first few milliseconds after the impacts and that these transient motions were correlated with brain injuries. Stalnaker et al. [110] conducted a series of head impacts on fifteen PMHS. The 3D motion analysis was studied using an accelerometry technique and high speed cineradiography to understand the head injury mechanism. The brain motion with respect to the skull was observed from the acceleration data and confirmed by high speed cineradiographic films. Nusholtz et al. [82] investigated the response of the head to impact using live anesthetized and post-mortem monkeys and pressurized PMHS. The head motion was recorded with a nine-accelerometer system and high speed x-ray. The epidural pressure was measured, and the interval displacement of the brain was observed as well.

Relative brain motion was also studied through observing the motion of markers that were implanted in the brain tissue. Trosseille et al. [113] used pressurized and fully instrumented PMHS to measure 3D head dynamics and then to validate their FE model against the experimental data. However, accelerometers with higher density than the brain tissue were implanted in the brain of PMHS to study the dynamic motion of the transducers.
in the brain. With technical enhancement in recent years, more studies have investigated brain motion. Hardy et al. [40] employed neutral density accelerometers implanted in the brain tissue and an accelerometer array mounted on the head for the measurement of relative brain motion during occipital impacts by integrating the relative accelerations. Their results showed that the relative brain motion with respect to the skull was 3-5 mm in magnitude during low-speed impacts. Using the same method, Al-Bsharat et al. [1] mounted radio opaque markers on the head and implanted NDTs in the brain. In addition to integrating the relative accelerations, the relative brain motion was measured by tracking the paths of the markers during occipital impacts. They found that the peak magnitude of the relative motion between the targets and the skull was about 3.5 mm. With high frame rate and high resolution camera systems, Hardy et al. [41] conducted further experiments by tracking the paths of the NDTs during both occipital and frontal impacts. The relative motion of the brain with respect to the skull was found to be 5 mm under low-severity impacts.

MRI has also been used to study brain motion. Ji et al. [50] studied in vivo human brain motion relative to the skull using MRI. Their kinematic analysis showed that brain motion with respect to the skull was on the order of 1-2 mm as the head rotates voluntarily from modest extension to full flexion. In [50], Ji et al. also concluded that the cerebellum rotated about 2.7-4.3 degrees with respect to the skull as the head rotates voluntarily through the imaging process. Bayly et al. [7] measured brain deformation in human volunteers under mild occipital deceleration. The strains were typically 0.02-0.05 during these mild impacts.

Although brain rotation was discussed in Ji et al’s [50] work, it was limited to the cerebellum region and is inadequate to describe the motion pattern of the whole brain. The rotation of a relatively larger brain region has not yet been studied. With the relative brain motion data from Hardy et al’s [41] experiments, this study focuses on the analysis of
brain motion, which is composed of rigid body displacement and deformation. The brain motions due to rigid body displacement and deformation are separated in this work.

2.3 Experimental Data

In this work, Hardy et al.’s [41] experimental NDT data from six tests on two specimens are used to analyze brain motion patterns. Allowed to rotate and translate freely, two PMHS heads prepared quickly after death were inverted in a suspension fixture that facilitated either acceleration of a stationary head or deceleration of a moving head. At either the frontal or the occipital area, stationary specimen C755 was struck by a moving impactor, while moving specimen C383 struck a fixed block at low impact speeds between 2.5 and 3.5 m/s.

With tin granules inserted inside, the NDTs were thin-walled polystryrene cylinders having finished dimensions of 5 mm in length and 2.5 mm in diameter. The density of the targets was close to that of brain tissue so that they would move together with the brain tissue without lacerating it during impacts. With five or six in each column, the NDTs were implanted into the brain in two columns, anterior and posterior, located in the occipitoparietal and temporoparietal regions with approximately 50-70 mm spacing between them, as shown in Figure 2.1. The spacing of the adjacent NDTs ranged from 7 to 12 mm. The locations of the targets spread across a large area so that the brain motion analyzed closely represents whole brain motion.

The specimens were perfused using a recirculated artificial cerebrospinal fluid (aCSF) that was maintained at a constant pressure of 10.5 kPa throughout the experiments. High-speed bi-planar x-ray with a sampling rate of 3000 Hz and video camera systems with
Figure 2.1: Schematic of NDT and CG locations in an inverted head (duplicated from Hardy et al’s [41] work). “a” and “p” stand for the anterior and posterior columns, respectively.

Frame rates of 250 and 1000 fps were used to track the locations of the NDTs. The relative brain motion at each NDT location was obtained by eliminating the rigid body skull displacement from the NDT motion data. The head kinematics were measured using a 3-2-2-2 accelerometer array mounted on the apex of the head. Further information about the specimen preparation and testing procedure is described in detail in Hardy et al’s [41] work.

Due to the nature of the impacts, the relative brain motion was primarily in the sagittal plane, which was the focus in Hardy et al’s [41] work. However, the full 3D head and brain motions were measured throughout each impact. Although the out-of-sagittal-plane motions of the specimen were found to be smaller than those in the sagittal plane, the relative brain motion patterns in both the sagittal and coronal planes are analyzed in this study. As listed in Table 2.1, the HIC15 and maximum resultant accelerations in tests on
C383 are all larger than those on C755. In this work, the severity of impact specifically refers to these values.

<table>
<thead>
<tr>
<th>Test No.</th>
<th>Impact Type</th>
<th>HIC15</th>
<th>Maximum Resultant Acceleration</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Linear (g)</td>
</tr>
<tr>
<td>C755-T2</td>
<td>Occipital acceleration</td>
<td>16.9</td>
<td>21.8</td>
</tr>
<tr>
<td>C755-T3</td>
<td>Occipital acceleration</td>
<td>21.3</td>
<td>24.4</td>
</tr>
<tr>
<td>C755-T5</td>
<td>Frontal acceleration</td>
<td>5.2</td>
<td>12.1</td>
</tr>
<tr>
<td>C383-T1</td>
<td>Frontal deceleration</td>
<td>46.6</td>
<td>62.2</td>
</tr>
<tr>
<td>C383-T3</td>
<td>Frontal deceleration</td>
<td>67.6</td>
<td>62.4</td>
</tr>
<tr>
<td>C383-T4</td>
<td>Occipital deceleration</td>
<td>163.7</td>
<td>107.7</td>
</tr>
</tbody>
</table>

Table 2.1: Test configuration of Hardy et al’s [41] tests under low-severity impacts.

2.4 Method

2.4.1 Rigid Body Transformation

In Hardy et al’s [41] tests, the NDTs were located in two columns in the brain with five or six targets in each column. The motion of each NDT with respect to the skull was found by comparing its location with three targets fixed to the skull that defined an arbitrary local body-fixed frame. The initial positions of the NDTs were expressed in this local body-fixed frame and referred to as an initial array. At each time step during impact, the positions of the NDTs were obtained in the same frame, yielding another position array, referred to as a measured array.

In order to separate brain motion during impacts into rigid body displacement and deformation, the measured array needs to be compared with the initial array to quantify their individual contributions. As a rigid body, the initial array is transformed into a new position, referred to as the transformed array, to match the measured position array as closely
as possible at each time step. Introduced by brain deformation at each NDT location, strain energy is used as the measure to determine this matching. The strain energy is defined and described in detail in Section 2.4.2. For each NDT, the difference of the transformed position and the measured position at each time step is a measure of brain deformation at that location. Brain deformation is therefore obtained by subtracting the rigid body displacement of the initial array from the brain motion measured at each time step. Through this analysis, the brain motion can be separated into rigid body displacement and deformation. The knowledge of the brain motion patterns will improve the understanding of brain injuries and guide the development of computational models and experiments.

In Figure 2.2(a), the initial array of the NDT positions in the brain is represented by the gray dots $R_i$, where $i$ is from 1 to $n$ and $n$ is the number of NDTs. The NDTs at the initial positions are in two columns according to the experimental setup, as shown in the figure. The measured position array is represented by the stars $S_i$, which experience rigid body displacement as an entire array and deformation at each individual NDT location. Expressed as a vector, the positions of the NDTs are $R_i$ and $S_i$ for the initial and measured locations, respectively. The initial position array $R_i$ is transformed as a rigid body into a new position array $S_i^*$ by translating a distance $d$ and rotating through an angle $\theta$, as shown in Figure 2.2(b). The transformed array $S_i^*$ now closely matches the measured array $S_i$. Note that the empty dashed circles in Figure 2.2(b) are the initial positions of the NDTs. It leads to the expression,

$$S_i^* = AR_i + d, \quad (2.1)$$

where the transformation matrix $A$ is given by,

$$A = \begin{bmatrix} \cos \theta & -\sin \theta \\ \sin \theta & \cos \theta \end{bmatrix}. \quad (2.2)$$

20
2.4.2 Pseudo-Strain Energy

Introduced by brain deformation, a pseudo-strain energy is used as the measure to determine the matching between the transformed position array and the measured position array. To include strain energy induced by the brain deformation during impact, the brain tissue is modeled with linear springs connecting each NDT to the other NDTs. Each spring is independent and has a neutral length when the NDTs are all at the initial positions. A single constant $K$ is taken for all springs, assuming isotropic linear elastic brain material. The compression and extension of the springs will store strain energy that increases quadratically with spring deflection. When one NDT moves away from its initial position, its position relative to other NDTs will change. During impact, all of the NDTs tend to move away from their initial positions. As a result, their relative positions and the lengths...
of the springs will change, storing strain energy in the springs. Each NDT’s motion relative to others will be dealt with separately to calculate the resultant pseudo-strain energy, and the total pseudo-strain energy is obtained through superposition. The pseudo-strain energy in each spring introduced by the relative motion of the \( i \)th NDT is defined as,

\[
E_i = \frac{nK}{2} |S_i^* - S_i|^2 ,
\]

(2.3)

where \( |S_i^* - S_i| \) is the distance between the transformed position and the measured position for the \( i \)th NDT, and it is equivalent to the change of the spring length between the \( i \)th NDT and the other NDTs. The total pseudo-strain energy stored in the springs due to the motion of all the NDTs is given by,

\[
E_n = \sum_{i=1}^{n} E_i = \frac{nK}{2} \sum_{i=1}^{n} |S_i^* - S_i|^2 .
\]

(2.4)

### 2.4.3 Minimizing Total Pseudo-Strain Energy

As shown in Figure 2.2(b), the initial array of NDT positions has been transformed as a rigid body into a new position, which closely matches the measured position array. The total pseudo-strain energy \( E_n \) can be calculated using the motion of each NDT relative to the others, as defined in Equation 2.4. The initial positions are represented by hollow dashed circles after being transformed. The values of \( d \) and \( \theta \) are to be found such that the pseudo-strain energy \( E_n \) is minimal. Substituting Equation 2.1 into Equation 2.4 yields,

\[
E_n = \frac{nK}{2} \sum \left( R_i^T R_i + d_i^T d_i + S_i^T S_i + 2d_i^T A R_i - 2S_i^T A R_i - 2d_i^T S_i \right) .
\]

(2.5)

The index “\( i \)” of the summation is dropped for simplicity. The minimum value of \( E_n \) occurs when,

\[
\frac{\partial E_n}{\partial \theta} = 0 \quad \text{and} \quad \frac{\partial E_n}{\partial d} = 0 .
\]

(2.6)
From Equations 2.6,

\[
\frac{\partial E_n}{\partial d} = \frac{nK}{2} \sum (2d + 2AR_i - 2S_i) = 0,
\]

\[
\frac{\partial E_n}{\partial \theta} = \frac{nK}{2} \sum \left(2d^T A\hat{i} R_i - 2S_i^T A\hat{i} R_i\right) = 0,
\]

(2.7)

since,

\[
\frac{dA}{d\theta} = \frac{d}{d\theta} \begin{bmatrix} \cos \theta & -\sin \theta \\ \sin \theta & \cos \theta \end{bmatrix} = A \begin{bmatrix} 0 & -1 \\ 1 & 0 \end{bmatrix} \equiv A\hat{I},
\]

(2.8)

where,

\[
\hat{I} = \begin{bmatrix} 0 & -1 \\ 1 & 0 \end{bmatrix}.
\]

(2.9)

Simplifying Equations 2.7 yields,

\[
nd + AR_\Sigma - S_\Sigma = 0,
\]

(2.10)

\[
d^T A\hat{i} R_\Sigma - \sum S_i^T A\hat{i} R_i = 0,
\]

(2.11)

where \(AR_\Sigma = \sum AR_i = A \sum R_i\) and \(S_\Sigma = \sum S_i\). Note that the spring constant \(K\) disappeared after taking partial derivatives, so it is not significant in the analysis. Rewriting Equation 2.10 yields,

\[
d = \frac{1}{n} (S_\Sigma - AR_\Sigma),
\]

(2.12)

which is the translation portion of the transformation. Substituting Equation 2.12 into Equation 2.11 and expanding yields,

\[
\frac{1}{n} S_\Sigma^T A\hat{i} R_\Sigma - \frac{1}{n} R_\Sigma^T A^T A\hat{i} R_\Sigma - \sum S_i^T A\hat{i} R_i = 0.
\]

(2.13)

For the orthogonal matrix \(A\), note that \(A^T A = I\), where \(I\) is the identity matrix. Equation 2.13 now becomes,

\[
\frac{1}{n} S_\Sigma^T A\hat{i} R_\Sigma - \frac{1}{n} R_\Sigma^T \hat{i} R_\Sigma - \sum S_i^T A\hat{i} R_i = 0.
\]

(2.14)
For any $2 \times 1$ vector $\mathbf{R}_\Sigma$, $\mathbf{R}_\Sigma^T \mathbf{I} \mathbf{R}_\Sigma \equiv 0$. Therefore, Equation 2.14 now becomes,

$$\frac{1}{n} \mathbf{S}_\Sigma^T \mathbf{I} \mathbf{R}_\Sigma - \sum \mathbf{S}_i^T \mathbf{I} \mathbf{R}_i = 0. \tag{2.15}$$

For any $2 \times 1$ vectors $\mathbf{R}$ and $\mathbf{S}$,

$$\mathbf{S}^T \mathbf{I} \mathbf{R} = \begin{bmatrix} s_x & s_z \end{bmatrix} \begin{bmatrix} \cos \theta & -\sin \theta \\ \sin \theta & \cos \theta \end{bmatrix} \begin{bmatrix} 0 \\ 1 \\ 0 \end{bmatrix} \begin{bmatrix} r_x \\ r_z \end{bmatrix} = -(s_x r_x + s_z r_z) \sin \theta - (s_x r_z - s_z r_x) \cos \theta = -(\mathbf{S} \cdot \mathbf{R}) \sin \theta - (\mathbf{S} \times \mathbf{R}) \cos \theta, \tag{2.16}$$

since in 2D, the analog of the cross product between vectors $\mathbf{S}$ and $\mathbf{R}$ is,

$$\mathbf{S} \times \mathbf{R} = \det [\mathbf{SR}] = \det \begin{bmatrix} s_x & r_x \\ s_z & r_z \end{bmatrix} = s_x r_z - s_z r_x. \tag{2.17}$$

Rearranging Equation 2.15,

$$\frac{1}{n} \mathbf{S}_\Sigma^T \mathbf{I} \mathbf{R}_\Sigma = \sum \mathbf{S}_i^T \mathbf{I} \mathbf{R}_i. \tag{2.18}$$

Substituting the results from Equation 2.16 into Equation 2.18 gives,

$$\sin \theta \left( \frac{1}{n} \mathbf{S}_\Sigma \cdot \mathbf{R}_\Sigma \right) + \cos \theta \left( \frac{1}{n} \mathbf{S}_\Sigma \times \mathbf{R}_\Sigma \right) = \sin \theta \sum (\mathbf{S}_i \cdot \mathbf{R}_i) + \cos \theta \sum (\mathbf{S}_i \times \mathbf{R}_i). \tag{2.19}$$

Collecting the trigonometric terms yields,

$$\tan \theta = \frac{\sin \theta}{\cos \theta} = \frac{\frac{1}{n} \mathbf{S}_\Sigma \times \mathbf{R}_\Sigma - \sum (\mathbf{S}_i \times \mathbf{R}_i)}{\sum (\mathbf{S}_i \cdot \mathbf{R}_i) - \frac{1}{n} \mathbf{S}_\Sigma \cdot \mathbf{R}_\Sigma}. \tag{2.20}$$

Therefore, the rotation angle $\theta$ of the rigid body is given by,

$$\theta = \tan^{-1} \left( \frac{\frac{1}{n} \mathbf{S}_\Sigma \times \mathbf{R}_\Sigma - \sum (\mathbf{S}_i \times \mathbf{R}_i)}{\sum (\mathbf{S}_i \cdot \mathbf{R}_i) - \frac{1}{n} \mathbf{S}_\Sigma \cdot \mathbf{R}_\Sigma} \right). \tag{2.21}$$

The transformed positions $\mathbf{S}_i^*$ of the NDTs now can be found with the results of Equations 2.12 and 2.21 for any known initial positions $\mathbf{R}_i$ and measured positions $\mathbf{S}_i$. Applying
these results, the total strain energy stored in the springs as defined in Equation 2.4 has a minimum value. The difference between the transformed and measured positions for each NDT is a measure of the elastic brain deformation at that location. The measured brain motion at each time step is therefore separated into rigid body brain displacement and brain deformation.

2.5 Motion Patterns of the Whole Brain

A total of six tests were performed using two specimens, C755 and C383. Among these tests, three accelerating impacts were conducted on specimen C755 and three decelerating impacts on specimen C383 in either the frontal area or the occipital area for both specimens. Equations 2.12 and 2.21 were used to analyze the experimental brain motion data. Although these two specimens were subject to low-severity impacts, the severity of these tests are still within a large range, having HIC15 values from as low as 5.2 for test C755-T5 to as high as 163.7 for test C383-T4. The HIC15 threshold value of injury is 700 for a 50th percentile male and a 5th percentile female according to the frontal crash regulation 49CFR571.208 [19].

Figures 2.3-2.5 show the relative brain movement patterns at the NDT locations. The subfigure at the top in each case contains measured relative brain motion patterns, and the one in the middle contains brain displacement generated using rigid body translation and rotation with a least square error. The difference between those two at each NDT location is a measure of brain deformation and is plotted in the subfigure at the bottom. As shown in the plots, the measured relative brain motion and the rigid body brain displacement generally coincide in terms of both directions and magnitudes at each NDT location. Therefore, the brain deformation in these tests is relatively small compared with the magnitude of
the brain motion. In other words, the brain motion in these tests is primarily due to rigid body displacement, which results in no strain in the brain tissue. The strain introduced by brain deformation is therefore also small in the tests. Although it was not indicated, DAI is unlikely to have occurred due to small strains in the brain in these tests. Having large rigid body displacement without significant strains under similar impacts, however, the brain may have other types of injury, such as ASDH, resulting from the relative brain-skull displacement. In this experiment, though, brain injuries cannot be studied because these injuries cannot be detected in PMHS.

For the three tests on specimen C383, as shown in Figures 2.6-2.8, the motion patterns and magnitudes of the rigid body brain displacement are also close to that of the measured relative brain motion. However, the differences between these two are larger than those in the tests on specimen C755. Therefore, the brain deformation-induced strain in these tests is larger. Referring to Table 2.1, the tests on specimen C383 have higher HIC15 values than those obtained in the tests on specimen C755. The resultant linear accelerations and angular accelerations both have relatively larger magnitudes than those in the tests on specimen C755. Under mild impacts, like the impacts on specimen C755, the brain motion is nearly a pure rigid body displacement, so only very small deformations result. When the impacts become more severe, the brain tissue experiences larger deformations at different locations. The figures suggest that the brain deformation increases as the resultant head accelerations are larger. However, the rigid body brain displacement in these low-severity tests on specimen C383 still accounts for a major portion of the brain motion.

In Figures 2.9-2.14, the time history of rigid body brain displacement at each NDT location is compared with the measured brain motion from tests on specimens C755 and C383. The symbol “Δ” indicates the relative motion with respect to the skull. In terms
Figure 2.3: Comparison of the measured relative brain motion at the NDT locations (top), the rigid body brain displacement with a minimum strain energy (middle), and the brain deformation due to the difference between the first two (bottom) for test C755-T2.
Figure 2.4: Comparison of the measured relative brain motion at the NDT locations (top), the rigid body brain displacement with a minimum strain energy (middle), and the brain deformation due to the difference between the first two (bottom) for test C755-T3.
Figure 2.5: Comparison of the measured relative brain motion at the NDT locations (top), the rigid body brain displacement with a minimum strain energy (middle), and the brain deformation due to the difference between the first two (bottom) for test C755-T5.
of both the direction and magnitude of the relative brain motion at each NDT location, the rigid body displacement and measured experimental data are very close, and they generally match more closely in tests on C755 than those on C383. Larger difference at each NDT location indicates larger brain deformation.

Figure 2.15 plots the magnitudes of translation and rotation of the rigid body brain displacement for all six tests. The peak magnitudes are listed in Table 2.2. The results of tests C755-T2 and C755-T3 are similar since they are both under occipital acceleration impact. Similar results are also found for tests C383-T1 and C383-T3, which are both under frontal deceleration impact. The results show that the magnitude of rigid body translation of the brain is about 4-5 mm along the $X$ axis and 2-3 mm along the $Z$ axis. The magnitude of the rigid body brain rotation is about $\pm 5$ degrees. Under more severe impacts, specimen C383 has larger relative brain motion than C755, but the magnitude of the rigid body brain displacement does not follow the same trend. For all six tests, the rigid body brain translation and rotation have similar magnitudes. The results indicate that the rigid body brain displacement may be limited to about 4-5 mm in translation and $\pm 5$ degrees in rotation regardless of the severity of the impacts.

<table>
<thead>
<tr>
<th>Test No.</th>
<th>Peak Displacement</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$\Delta x$ (mm)</td>
</tr>
<tr>
<td>C755-T2</td>
<td>3.2</td>
</tr>
<tr>
<td>C755-T3</td>
<td>4.0</td>
</tr>
<tr>
<td>C755-T5</td>
<td>2.3</td>
</tr>
<tr>
<td>C383-T1</td>
<td>4.1</td>
</tr>
<tr>
<td>C383-T3</td>
<td>2.6</td>
</tr>
<tr>
<td>C383-T4</td>
<td>5.0</td>
</tr>
</tbody>
</table>

Table 2.2: Peak magnitudes of rigid body translation and rotation of the whole brain in the sagittal plane for all six tests.
Figure 2.6: Comparison of the measured relative brain motion at the NDT locations (top), the rigid body brain displacement with a minimum strain energy (middle), and the brain deformation due to the difference between the first two (bottom) for test C383-T1.
Figure 2.7: Comparison of the measured relative brain motion at the NDT locations (top), the rigid body brain displacement with a minimum strain energy (middle), and the brain deformation due to the difference between the first two (bottom) for test C383-T3.
Figure 2.8: Comparison of the measured relative brain motion at the NDT locations (top), the rigid body brain displacement with a minimum strain energy (middle), and the brain deformation due to the difference between the first two (bottom) for test C383-T4.
Figure 2.9: Comparison of the measured brain motion and rigid brain displacement at each NDT location for test C755-T2. “a_i” and “p_i” indicate NDTs in the anterior and posterior columns, respectively.
Figure 2.10: Comparison of the measured brain motion and rigid brain displacement at each NDT location for test C755-T3. “a_i” and “p_i” indicate NDTs in the anterior and posterior columns, respectively.
Figure 2.11: Comparison of the measured brain motion and rigid brain displacement at each NDT location for test C755-T5. “a_i” and “p_i” indicate NDTs in the anterior and posterior columns, respectively.
Figure 2.12: Comparison of the measured brain motion and rigid brain displacement at each NDT location for test C383-T1. “a_i” and “p_i” indicate NDTs in the anterior and posterior columns, respectively.
Figure 2.13: Comparison of the measured brain motion and rigid brain displacement at each NDT location for test C383-T3. “a_i” and “p_i” indicate NDTs in the anterior and posterior columns, respectively.
Figure 2.14: Comparison of the measured brain motion and rigid brain displacement at each NDT location for test C383-T4. “a<sub>i</sub>” and “p<sub>i</sub>” indicate NDTs in the anterior and posterior columns, respectively.
Figure 2.15: Time history of the rigid body translation and rotation of the original NDT position array for all six tests on specimens C755 and C383. On the left, T2 and T3 are occipital acceleration tests, and T5 is a frontal acceleration test. On the right, T1 and T3 are frontal deceleration tests, and T4 is an occipital deceleration test.

2.6 Regional Motion Patterns

The whole brain was found to closely follow a rigid body displacement under mild impact. As the impact becomes more severe, brain deformation represents an increasing portion of the total brain motion. Since the anterior and posterior columns have approximately 50-70 mm spacing, while the spacing of the adjacent NDTs in each individual column ranged only from 7 to 12mm, analysis of the experimental data in each single column is performed to better understand the brain motion patterns.

Although the head and brain kinematics in the sagittal plane are dominant for all of the six tests due to either frontal or occipital impacts, the 3D motion for all NDTs was measured
throughout the tests. The anterior and posterior columns form the sagittal plane; therefore, the motion data in the coronal plane are analyzed for each individual column separately. The measured motion in both the sagittal and coronal planes for each individual column is separated into rigid body brain displacement and brain deformation using the same method as for the whole brain. Thus, the motion patterns of the two columns representing different regions of the brain individually can be obtained through this analysis. The patterns of relative brain motion due to impact can therefore be better understood for the whole brain and individual columns under impacts primarily in the sagittal plane. Since the whole brain moves as a rigid body under mild impacts on specimen C755, the motion of each individual columns therefore also follows closely a rigid body displacement. The focus in this section will be on the more severe impact tests on C383.

The brain motion patterns in the sagittal plane of the anterior and posterior columns for test C383-T1 are shown in Figures 2.16 and 2.17. The results for tests C383-T3 and C383-T4 are shown in Figures 2.18-2.21. The subfigure on the left contains the measured brain motion, and the one in the middle contains the brain displacement generated using rigid body translation and rotation. The difference between these two at each NDT location is a measure of brain deformation, as shown in the subfigure on the right. For all six tests, the magnitude of measured brain motion generally varies within ±5 mm under low accelerations and within ±8 mm under higher accelerations. The rigid body brain displacement generally coincides with the measured motion in terms of both direction and magnitude for each NDT in both the anterior and posterior columns, where the brain deformation in each individual column is therefore small, only 0.5-1.5 mm. Note that only five targets (p1-p5) in the anterior column of test C383-T1 are included. Further analysis indicates that the measurement for the excluded target (p6) is problematic. It is partially indicated in Figure
2.6, where the brain deformation at target p6 is unexpectedly large. However, inclusion of target p6 does not affect the results presented in this section.

In the coronal plane, similar patterns are obtained, as shown in Figures 2.22-2.27, but the rigid body displacements of both columns generally have smaller magnitudes than in the sagittal plane. Again, target p6 in the anterior column in test C383-T1 is not included in the analysis. Table 2.3 includes the peak magnitude of rigid body translation and rotation for anterior and posterior columns in both the sagittal and the coronal planes. In general, it shows that tests on C755 have smaller rigid body displacements for both columns than tests on C383, which were under more severe impacts. It also shows that the rigid body displacement in the coronal plane for both columns is of smaller magnitude than in the sagittal plane, wherein the impacts were conducted.

Compared with the whole brain, each individual column in tests on C755 has almost the same rigid body translation of 4-5 mm, while the rigid body rotation of ±5 to ±8 degrees is slightly larger. Overall, the rigid body displacement for the whole brain and the individual columns have close magnitudes in tests on C755, which were under low accelerations. This reinforces the conclusion that the whole brain moves as a rigid body under mild impacts. In conclusion, rigid body displacement in both the sagittal and the coronal planes is found for each individual column, as well as the whole brain, in tests on C755 having low accelerations.

As the impact becomes more severe on C383, each column still follows closely a rigid body displacement, but the relative motion between the two columns becomes larger. In test C383-T4, the anterior column maximally translates about 9.0 mm along the X axis, 5.3 mm along the Z axis, and rotates 13.1 degrees about the Y axis, while these values for the posterior column are about 10.1 mm, 8.4 mm, and 13.9 degrees, respectively. In
Figure 2.16: Motion pattern of anterior column in the sagittal plane in test C383-T1.

Figure 2.17: Motion pattern of posterior column in the sagittal plane in test C383-T1.
Figure 2.18: Motion pattern of anterior column in the sagittal plane in test C383-T3.

Figure 2.19: Motion pattern of posterior column in the sagittal plane in test C383-T3.
Figure 2.20: Motion pattern of anterior column in the sagittal plane in test C383-T4.

Figure 2.21: Motion pattern of posterior column in the sagittal plane in test C383-T4.
<table>
<thead>
<tr>
<th>Test No.</th>
<th>Column</th>
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</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>$\Delta x$ (mm)</td>
<td>$\Delta z$ (mm)</td>
<td>$\Delta \theta$ (deg)</td>
<td>$\Delta y$ (mm)</td>
</tr>
<tr>
<td>C755-T2</td>
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<td>1.8</td>
<td>7.9</td>
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</tr>
<tr>
<td></td>
<td>P</td>
<td>3.8</td>
<td>2.3</td>
<td>6.2</td>
<td>1.9</td>
</tr>
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<td>0.7</td>
<td>8.6</td>
<td>1.8</td>
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<td>9.2</td>
<td>1.9</td>
</tr>
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<td></td>
<td>P</td>
<td>10.1</td>
<td>8.4</td>
<td>13.9</td>
<td>1.1</td>
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</table>

Table 2.3: Peak magnitudes of rigid body translation and rotation for anterior and posterior columns in both the sagittal and coronal planes. “A” and “P” represent anterior and posterior columns, respectively.

In addition, the anterior and posterior columns translate 5.3 mm and 8.4 mm in the opposite directions along the $Z$ axis, as shown in Figures 2.28 and 2.29. Thus, relative motion of the two columns and therefore, brain deformation, exist in the region between these two columns in test C383-T4. Similar results were obtained for tests C383-T1 and C383-T3, and the peak rigid body translation and rotation are listed in Table 2.3.

From the results presented here, it is concluded that the two columns move individually as a rigid body in both the sagittal and the coronal planes. As the impact becomes more severe, the increased portion of brain motion is primarily due to the brain deformation, which is predominantly located somewhere between these two columns.
Figure 2.22: Motion pattern of anterior column in the coronal plane in test C383-T1.

Figure 2.23: Motion pattern of posterior column in the coronal plane in test C383-T1.
Figure 2.24: Motion pattern of anterior column in the coronal plane in test C383-T3.

Figure 2.25: Motion pattern of posterior column in the coronal plane in test C383-T3.
Figure 2.26: Motion pattern of anterior column in the coronal plane in test C383-T4.

Figure 2.27: Motion pattern of posterior column in the coronal plane in test C383-T4.
Figure 2.28: Rigid body displacement of the anterior column in the sagittal plane for all six tests.

Figure 2.29: Rigid body displacement of the posterior column in the sagittal plane for all six tests.
Figure 2.30: Rigid body displacement of the anterior column in the coronal plane for all six tests.

Figure 2.31: Rigid body displacement of the posterior column in the coronal plane for all six tests.
2.7 Location of Brain Deformation

The results have shown that the brain deformation lies predominantly somewhere in between the two columns, which are located in the occipitoparietal region and in the temporoparietal region. The experimental data are further analyzed in this section to better estimate the brain deformation in the region between the two columns. In other words, this study seeks to determine if the brain deformation is dominant in the superior or inferior portion of the brain. Since tests on C755 are all under mild impacts without significant deformation in the brain, tests on C383 under more severe impacts are used for this analysis to find the dominant location of brain deformation.

In tests on specimen C383, a total of six markers were implanted in each column, anterior and posterior, and referred to as “a1-a6” and “p1-p6”. In order to locate the brain deformation, the measured motion is separated into rigid body displacement and deformation. The motion of an equal number of NDTs in both columns is matched with a rigid body displacement from both the superior and inferior directions. Starting with two NDTs, (a1,p1) from the inferior direction and (a6,p6) from the superior direction, the matchings are compared. Then, the comparison is made between the matchings of four NDTs, (a1,a2,p1,p2) and (a5,a6,p5,p6). With an increasing number of NDTs, similar comparisons are made until all twelve NDTs are included. The combinations of the NDTs in each comparison are summarized in Table 2.4. Test C383-T3 is not used in this analysis since it only has two NDTs, p5 and p6, in the posterior column. Having six markers in each column, tests C383-T1 and C383-T4 are used for this analysis.

The comparisons are quantified with the error of the matchings between the measured brain motion and rigid body displacement for various numbers of NDTs. The error of the matching for each individual NDT at each time step is a measure of brain deformation and
<table>
<thead>
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<th>Number of NDTs</th>
<th>NDTs Included From Inferior</th>
<th>NDTs Included From Superior</th>
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</thead>
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<tr>
<td>2</td>
<td>a1, p1</td>
<td>a6, p6</td>
</tr>
<tr>
<td>4</td>
<td>a1-a2, p1-p2</td>
<td>a5-a6, p5-p6</td>
</tr>
<tr>
<td>6</td>
<td>a1-a3, p1-p3</td>
<td>a4-a6, p4-p6</td>
</tr>
<tr>
<td>8</td>
<td>a1-a4, p1-p4</td>
<td>a3-a6, p3-p6</td>
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<td>10</td>
<td>a1-a5, p1-p5</td>
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</tr>
<tr>
<td>12</td>
<td>a1-a6, p1-p6</td>
<td>a1-a6, p1-p6</td>
</tr>
</tbody>
</table>

Table 2.4: Combination of NDTs for locating the brain deformation.

is used to quantify the matchings. Since the magnitude of the measured brain motion for each NDT varies dramatically, for instance, about 8.8 mm for NDT p1 and 1.0 mm for p5 in test C383-T4, a normalized error of the matching is used for the comparison. For each NDT, the errors in the X axis and the Z axis throughout the matching are divided by the peak magnitudes of measured motion in the X axis and the Z axis, respectively. The normalized errors in both axes are squared and summed at each time step. The square root of the summation is taken as the measure of the matching. The results at all time steps are summed for all NDTs and then averaged by the total number of data points to get the averaged-normalized error. The equation for the averaged-normalized error $E_{an}$ is,

$$E_{an} = \frac{1}{pq} \sum_{i=0}^{p} \sum_{j=0}^{q} \sqrt{\left(\frac{\Delta x_{mij} - \Delta x_{rij}}{\Delta x_{maxi}}\right)^2 + \left(\frac{\Delta z_{mij} - \Delta z_{rij}}{\Delta z_{maxi}}\right)^2}, \quad (2.22)$$

where $p$ is the total number of markers for the matching, $q$ is the total number of data points measured for each marker, $\Delta x_{m}$ is the measured motion along the X axis for each time step, $\Delta x_{r}$ is the rigid body displacement, $\Delta x_{max}$ is the maximum measured motion at each marker location. $\Delta z_{m}$, $\Delta z_{r}$, and $\Delta z_{max}$ are the quantities in the Z direction. The averaged-normalized error is used as the measure of comparison to analyze the experimental data and locate the brain deformation under more severe impacts.
Figures 2.32 and 2.33 compare the averaged-normalized error of matchings from the inferior and superior for tests C383-T1 and C383-T4. The combinations of the NDTs on the horizontal axes are listed in Table 2.4. In general, it shows that the errors in C383-T4 are larger than those in C383-T1 for all the cases having different numbers of NDTs matched, because test C383-T4 was under higher accelerations and therefore, more severe impact. The matching in test C383-T4 is not as close as in C383-T1 because larger brain deformation was experienced. This finding is consistent with the severity of impact characterized by HIC15 and maximum linear and angular accelerations.

The results in Figures 2.32 and 2.33 also show that the averaged-normalized error increases nearly linearly with the number of NDTs matched. The error of matching from the superior direction has larger magnitude than from the inferior in both tests. With two NDTs in test C383-T1, the error of matching is 0.07 for (a1,p1) and 0.33 for (a6,p6). This suggests that the location of the brain deformation in these two tests is likely closer to the superior portion of the brain. In addition, the maximum brain deformation is most likely dominant in the region between these two columns from the study in Section 2.6. Thus, the brain deformation in these two tests can be located in the superior region of the brain between the two columns.

The same set of data in Figures 2.32 and 2.33 is summarized in Table 2.5. Table 2.6 lists the averaged-normalized error when different numbers of markers in each individual column are matched. Again, the error increases with the increased number of NDTs matched in the anterior or posterior column. With the same number of NDTs, the matching for markers in the same column has less error than the matching for markers in both columns. Having six NDTs, for instance, the matching in each column, anterior and posterior, yields
Figure 2.32: Comparison of averaged-normalized error $E_{an}$ for the matchings from the superior and inferior for test C383-T1. Equal number of NDTs in two columns are used.

Figure 2.33: Comparison of averaged-normalized error $E_{an}$ for the matchings from the superior and inferior for test C383-T4. Equal number of NDTs in two columns are used.
an error of 0.22 in test C383-T1. For the same test, however, the error is 0.16 with markers a1-a3 and p1-p3 in the inferior region and 0.34 with markers a4-a6 and p4-p6 in the superior region. This supports the conclusion that the brain deformation is dominant in the superior portion of the brain in these two tests. In Table 2.5, the matching of two markers (a6,p6) in the superior portion in C383-T4 gives an error of 0.21. However, the error of matching six markers in the anterior column in this test is only 0.20, as shown in Table 2.6. The results suggest that smaller error of matching does not result from fewer markers to match.

<table>
<thead>
<tr>
<th>Test No.</th>
<th>Direction</th>
<th>Number of NDTs Matched</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Inferior</td>
<td>2</td>
</tr>
<tr>
<td>C383-T1</td>
<td>Superior</td>
<td>0.07</td>
</tr>
<tr>
<td></td>
<td>Inferior</td>
<td>0.33</td>
</tr>
<tr>
<td>C383-T4</td>
<td>Superior</td>
<td>0.08</td>
</tr>
<tr>
<td></td>
<td>Inferior</td>
<td>0.21</td>
</tr>
</tbody>
</table>

Table 2.5: The averaged-normalized error of matching in tests C383-T1 and C383-T4. Equal number of NDTs are used in both anterior and posterior columns for all cases.

<table>
<thead>
<tr>
<th>Test No.</th>
<th>Column</th>
<th>Direction</th>
<th>Number of NDTs Matched</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Anterior</td>
<td>Inferior</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Superior</td>
<td>0</td>
<td>0.04</td>
</tr>
<tr>
<td></td>
<td>Inferior</td>
<td>0</td>
<td>0.19</td>
</tr>
<tr>
<td></td>
<td>Superior</td>
<td>0</td>
<td>0.13</td>
</tr>
<tr>
<td></td>
<td>Inferior</td>
<td>0</td>
<td>0.05</td>
</tr>
<tr>
<td></td>
<td>Superior</td>
<td>0</td>
<td>0.06</td>
</tr>
<tr>
<td>C383-T4</td>
<td>Anterior</td>
<td>Inferior</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Superior</td>
<td>0</td>
<td>0.06</td>
</tr>
<tr>
<td></td>
<td>Inferior</td>
<td>0</td>
<td>0.06</td>
</tr>
<tr>
<td></td>
<td>Superior</td>
<td>0</td>
<td>0.07</td>
</tr>
</tbody>
</table>

Table 2.6: The averaged-normalized error of matching different number of markers in each individual column in tests C383-T1 and C383-T4.
2.8 Discussion

Separating the relative brain motion into rigid body displacement and brain deformation is significant to help better understand the brain motion patterns during impacts and therefore, brain injury mechanisms. Using Equations 2.12 and 2.21, the experimental NDT data were matched with the initial NDT positions to minimize the total pseudo-strain energy. The brain deformation due to the difference between the rigid body brain displacement and the measured brain motion was obtained. Although multiple tests were conducted on each single specimen, the brain was found to return to its initial configuration after each low speed impact. Therefore, the mechanical properties of the brain appear to have remained the same during the multiple tests.

Hardy et al’s [41] experimental data clearly show a rigid body brain displacement inside the skull. The important finding of this work is that the brain motion during impact consists of both relative displacement and deformation. For low-severity impacts, the rigid body brain displacement has a magnitude of 4-5 mm in translation and ±5 degrees in rotation. Unlike Bayly et al’s [7] work, Hardy et al’s [41] tests were not designed for strain measurement. The relatively small number of targets did not give high enough spatial resolution to measure strain accurately, especially between the two columns. Therefore, local strain cannot be estimated through this study, although the rigid body displacement was extracted.

The rigid body displacement and the deformation of the brain discussed in this work only represent the motion pattern of the portion with implanted NDTs. In Hardy et al’s [41] experiments, the NDTs were implanted in two columns, one in the occipitoparietal region and the other in the temporoparietal region. The NDTs occupy a relatively large brain region including the occipital, temporal, and parietal lobes, which contain a significant
portion of the whole brain volume. This region better represents the whole brain motion than the cerebellum region used in Ji et al’s [50] study. The experimental data does not include the brain motion in the frontal region of the brain, but it is believed that the frontal region follows similar patterns. The NDTs are not near the interface of the brain and the skull; therefore, the pattern does not indicate in any way the brain motion at the boundary.

The out-of-plane brain motion is relatively small compared with the sagittal plane motion, and the sagittal plane head kinematics dominate after the impacts. Therefore, only brain motion patterns in the sagittal plane are of particular interest in this study. As a result, the finding of the rigid body brain displacement is only valid in the sagittal plane. Kleiven and Hardy [58] found that the relative brain motion in the coronal plane had a smaller magnitude than that in the sagittal plane from both experiments and FE simulations. This can be explained by the supportive nature of the falx cerebri that divides the brain into hemispheres.

These six tests on two specimens were all under relatively low-severity impacts, and brain injuries were unlikely to have occurred. In order to thoroughly investigate the brain motion patterns and verify the findings in this study, experimental data under high-speed impacts that could result in more severe brain injuries are needed. In addition, the NDTs should spread across a large region, especially in the frontal region of the brain, to better represent the motion of the whole brain.

2.9 Conclusions

The experimental relative brain motion data from Hardy et al’s [41] work were used in this study to separate the brain motion into rigid body displacement and local deformation. The NDT position array at each time step was matched with the initial position array, which
was transformed as a rigid body, to minimize the total pseudo-strain energy to study the brain motion patterns. It was found that the brain has nearly pure rigid body displacement at low impact speed. As the impact becomes more severe, the increased brain motion is primarily due to deformation, while the rigid body displacement is limited in magnitude for both translation and rotation. Under low-severity impacts in the sagittal plane, the rigid body brain translation has a magnitude of 4-5 mm, and the whole brain rotation is on the order of ±5 degrees.

Similar to the whole brain, the two columns, representing the temporoparietal region and occipitoparietal region, individually follow closely a rigid body displacement in both the sagittal and coronal planes under mild impact. As the impact becomes more severe, each column still closely follows a rigid body displacement, which has a magnitude of 6-8 mm in translation and ±10 degrees in rotation. However, relative motion becomes larger between these two columns, introducing strain deformation. The analysis also indicates that the brain deformation in these tests is dominant in the superior region of the brain.
CHAPTER 3

PLANAR BRAIN INJURY MODELS

3.1 Introduction

Mathematical models have been widely used to study brain injury resulting from impacts. Early modeling work focused on lumped-parameter models [104, 11, 107], and more modern approaches are dominated by FE models [126, 58, 111, 43]. FE head models can capture detailed geometric properties and provide tissue-level information, such as strain and stress distributions, under impact loading. However, FE modeling also has limitations since it requires accurate material properties and boundary conditions and is computationally intensive. In contrast, simple lumped-parameter models with less structural complexity can capture the overall mechanical responses using less computational capacity. In addition, simple models have the potential to provide insight into the influence of small parameter changes on the system response.

This chapter presents relatively simple brain injury models to bridge the gap between the very simple HIC criterion and more complex FE models. The model development is based on the results of the experimental data analysis in Chapter 2. The rest of this chapter introduces new planar models in the sagittal and coronal planes for brain injury study during automobile crashes. Like many FE models [126, 58, 111, 43], the validation
of the simple brain injury model is also based on the advanced experimental brain motion data in Hardy et al’s work [41].

### 3.2 Background

Some early research contends that linear accelerations experienced by the head are a main cause of brain injury [38, 84, 86], while other work argues that angular accelerations are more injurious [32, 33, 68]. A number of recent studies, however, suggest that both linear and angular accelerations are significant causes of brain injury. Newman et al. [81] conducted a series of experiments with the Hybrid III crash test dummy to reproduce incidents that occurred on football fields and investigate correlations between head injury and head kinematics. They found that the resultant peak angular acceleration has the least significance to predict concussion compared with several other indices, such as the resultant peak linear acceleration, GSI index, HIC criterion, and head impact power index (HIP). King et al. [54] argued that the relationship between angular accelerations of the head and brain injury may not be as strong as reported in earlier work that proposed the primacy of angular accelerations. Anderson et al. [4] conducted a series of impact experiments on sheep to study the relationship between axonal brain injury and the severity of the impact to the head. They concluded that the most reliable correlates for the extent of injury were both linear and angular kinematics of the head. Zhang et al. [128] also concluded that both linear and angular accelerations are significant causes of mild traumatic brain injury and that linear head acceleration has a greater influence on intracranial pressure responses than angular acceleration. In an FE model, Kleiven [57] found that the peak change in angular velocity best correlated with the strain level in brain tissue for a pure angular impulse, while HIC and HIP had the best correlation with the strains for a pure linear impulse.
Some experimental work has achieved separation of pure linear and pure angular accelerations of the head, but most experimental studies involve the combination of the two. Careful analysis of the experimental results indicates that linear and angular movements of the brain with respect to the skull are coupled. Hardy et al. [41] conducted low-severity impact experiments with PMHS heads and observed relative brain displacements on the order of $\pm 5$ mm. Further analysis of the experimental data indicated that relative rotation of the brain occurred in addition to the relative translation, as discussed in Chapter 2. In another set of PMHS head experiments, King et al. [53] compared the relative motion between the brain and the skull when the head rotated in the coronal and sagittal planes. They reported that the pattern of relative brain motion was similar to that found in Hardy et al. [41]. Bayly et al. [7] studied strain deformation in human volunteers under occipital deceleration impact using MRI. They concluded that angular acceleration of the skull was not required to cause angular acceleration of the brain due to the tangential components of tethering forces, which were transmitted by the vascular, neural, and dural elements, binding the brain to the base of the skull. The tangential forces changed the angular momentum of the brain, causing brain rotation. These findings provide strong evidence that linear and angular brain motions are coupled. Experimental data for the case of pure head translation during impact are not currently available, but relative brain rotation in such a case is anticipated for consistency with the existing data.

Existing simple lumped-parameter models are inadequate to accomplish sensitivity analysis because they allow only linear or angular accelerations individually as the input. Considering only linear accelerations, Stalnaker et al. [107] developed a one-dimensional, translational model of the human head having two masses connected by a spring and
damper in parallel, based on Slattenschek’s [104] model. Considering only angular accelerations, Low [66] developed a rotational model consisting of three masses connected by eight springs and dampers. Due to the lack of experimental data, this rotational model was not effectively validated. In contrast, other existing lumped-parameter models are too complex to allow for analytical solutions, so they are likewise inappropriate for sensitivity analysis. Alem’s [3] twelve-degree-of-freedom head model consisting of five lumped masses, ten linear springs, and three torsional springs is one such example of a complex model that does not have an analytical solution.

### 3.3 Full Sagittal Plane Model

This work proposes a new lumped-parameter human brain injury model that can be solved analytically to evaluate the sensitivity of injury prediction to small changes in a few parameters. Based upon knowledge from the literature, the present work operates from a fundamental assumption that the linear and angular movements of the brain with respect to the skull are coupled. This section develops a model in the sagittal plane to study brain responses under frontal and rear impacts since relative brain motion is primarily planar under these types of impacts [41].

#### 3.3.1 Model Description

A new planar lumped-parameter model that captures the motion of the human head in the sagittal plane is depicted in Figure 3.1. The positive $X$ axis is in the anterior direction, and the positive $Z$ axis is in the superior direction. Not explicitly plotted, the positive $Y$ axis is in the left lateral direction. Positive angular displacement of the head about the $Y$ axis corresponds to forward flexion of the neck. The global coordinate system is $XZ$, the local coordinate system $X_SZ_S$ is fixed on the skull, and the local coordinate system $X_BZ_B$
is fixed on the brain. The model consists of a circular skull and brain having masses, \( m_s \) and \( m_b \), and moments of inertia, \( I_s \) and \( I_b \), where the subscripts “s” and “b” indicate the skull and brain, respectively. The skull and brain are connected by four Kelvin elements, each having a spring constant \( k_i \) and a damping coefficient \( c_i \), where \( i = 1 \ldots 4 \). The centers of mass of the skull and the brain are assumed to be coincident at the geometric center. In the neutral position, each Kelvin element makes a 45-degree angle with the horizontal and is attached to the brain at a perpendicular offset distance of \( d \) with respect to the center of mass. The parameter \( r \) is the radius of the circular brain.

The values of the spring and damping coefficients within the four Kelvin elements were selected asymmetrically so as to couple the relative translation and rotation between the brain and skull. For example, when the brain translates along the \( X \) axis relative to the skull, Kelvin elements 2 and 3 are in tension, while elements 1 and 4 are in compression.
Since the coefficients within these elements are not equal, the net moment on the brain about the Y axis is in general not zero, so rotation occurs. By a similar argument, the net force on the brain in the Z direction is also non-zero, so translation in the Z direction likewise occurs. Thus, the three degrees of freedom in the plane are all coupled.

3.3.2 Equations of Motion

The equations of motion can be derived to describe these three types of brain motions: the translation along the X and Z axes and the rotation about the Y axis. For the planar model shown in Figure 3.1, the origins $O_S$ and $O_B$ of the local coordinate systems $X_SZ_S$ and $X_BZ_B$ coincide with the origin $O$ of the global coordinate system $XZ$ before impact. In addition, the initial positions of the origins coincide with the CG of the brain. The points $P_i$ and $Q_i$ are the connecting points on the skull and the brain, respectively. Using a transformation matrix, the coordinates of points $P_i$ can be expressed in the $XZ$ frame as,

$$
\begin{bmatrix}
P_{ix} \\
- \sin \theta_s \\
0
\end{bmatrix}
\begin{bmatrix}
\cos \theta_s & \sin \theta_s & x_s \\
- \sin \theta_s & \cos \theta_s & z_s \\
0 & 0 & 1
\end{bmatrix}
\begin{bmatrix}
P_{ixs} \\
P_{izs} \\
1
\end{bmatrix},
$$

(3.1)

where $(P_{ixs}, P_{izs})$ are the coordinates of point $P_i$ in the $X_SZ_S$ frame, $x_s$ and $z_s$ are the translations of the $X_SZ_S$ frame in the $XZ$ frame along the X axis and the Z axis, respectively, and $\theta_s$ is the rotation of the $X_SZ_S$ frame.

Similarly, the coordinates of points $Q_i$ can be expressed in the $XZ$ frame as well,

$$
\begin{bmatrix}
Q_{ix} \\
- \sin \theta_b \\
0
\end{bmatrix}
\begin{bmatrix}
\cos \theta_b & \sin \theta_b & x_b \\
- \sin \theta_b & \cos \theta_b & z_b \\
0 & 0 & 1
\end{bmatrix}
\begin{bmatrix}
Q_{ixb} \\
Q_{izb} \\
1
\end{bmatrix},
$$

(3.2)

where $(Q_{ixb}, Q_{izb})$ are the coordinates of point $Q_i$ in the $X_BZ_B$ frame, $x_b$ and $z_b$ are the translations of the $X_BZ_B$ frame in the $XZ$ frame along the X axis and the Z axis, respectively, and $\theta_b$ is the rotation of the $X_BZ_B$ frame. For this brain injury model, the coordinates of points $P_i$, $(P_{ixs}, P_{izs})$, are $(0, \sqrt{2}r)$, $(-\sqrt{2}r, 0)$, $(0, -\sqrt{2}r)$, and $(\sqrt{2}r, 0)$ in the
The coordinates of points $Q_i$, $(Q_{izb}, Q_{izb})$, are $\left(-\frac{\sqrt{2}}{2}r, \frac{\sqrt{2}}{2}r\right)$, $\left(-\frac{\sqrt{2}}{2}r, -\frac{\sqrt{2}}{2}r\right)$, $\left(\frac{\sqrt{2}}{2}r, -\frac{\sqrt{2}}{2}r\right)$, and $\left(\frac{\sqrt{2}}{2}r, \frac{\sqrt{2}}{2}r\right)$ in the $X_BZ_B$ frame. Therefore, each Kelvin element makes a 45-degree angle with the horizontal in the neutral position before impact.

During head impacts, the connecting points $P_i$ and $Q_i$ will all move in the $XZ$ frame. The distance between points $P_i$ and $Q_i$ is given by,

$$L_i = |P_iQ_i| = \sqrt{(P_{ix} - Q_{ix})^2 + (P_{iz} - Q_{iz})^2}.$$  \hfill{(3.3)}

The change in length of the Kelvin elements is,

$$\Delta L_i = L_i - L_0,$$  \hfill{(3.4)}

where $L_0$ is the free length of the Kelvin elements when the brain and the skull are both in the neutral position. The rate of change in length is given by,

$$\Delta \dot{L}_i = \frac{\Delta L_i}{dt}.$$  \hfill{(3.5)}

Using the chain rule, Equation 3.5 becomes,

$$\Delta \dot{L}_i = \frac{\Delta L_i}{dt} = \frac{\partial \Delta L_i}{\partial x_s} \dot{x}_s + \frac{\partial \Delta L_i}{\partial x_b} \dot{x}_b + \frac{\partial \Delta L_i}{\partial z_s} \dot{z}_s + \frac{\partial \Delta L_i}{\partial z_b} \dot{z}_b + \frac{\partial \Delta L_i}{\partial \theta_s} \dot{\theta}_s + \frac{\partial \Delta L_i}{\partial \theta_b} \dot{\theta}_b,$$  \hfill{(3.6)}

where $\dot{x}_s$, $\dot{z}_s$ and $\dot{\theta}_s$ are the velocities of the skull, and $\dot{x}_b$, $\dot{z}_b$ and $\dot{\theta}_b$ are the velocities of the brain, all with respect to the global system. Applying Equations 3.3 and 3.4 to Equation 3.6 yields the rate of change of length defined in Equation 3.5.

The force in the Kelvin elements includes the deformation-dependent linear spring force and the velocity-dependent linear damping force, and it can be expressed as,

$$F_i = k_i \Delta L_i + c_i \Delta \dot{L}_i,$$  \hfill{(3.7)}

where $k_i$ and $c_i$ are the spring and damping constants of the Kelvin elements.
In order to set up the full equations of motion using this model, the angle \( \alpha_i \) that the Kelvin elements make with the horizontal axis is defined as follows,

\[
\begin{align*}
\cos \alpha_i & = \frac{P_{ix} - Q_{ix}}{L_i}, \\
\sin \alpha_i & = \frac{P_{iz} - Q_{iz}}{L_i}.
\end{align*}
\]  

(3.8)

Note that Figure 3.2 only shows the angle \( \alpha_1 \).

![Figure 3.2: Relative position of the brain with respect to the skull. Note that only the angles \( \alpha_1, \beta_1, \) and \( \gamma_1 \) are shown.](image)

The cosine and sine functions in Equations 3.8 are used to calculate the force components projected on the \( X \) and \( Z \) axes. The relative positions of the two connecting points, \( P_i \) and \( Q_i \), determine the role of the forces. For example, a positive sign of \( Q_{1x} - P_{1x} \) means that the \( X \) coordinate of point \( Q_1 \) is larger than that of point \( P_1 \). The force \( F_1 \) will therefore tend to pull the brain back in the negative \( X \) direction, pull the brain up in the
positive $Z$ direction, and turn the brain counter-clockwise. Using the law of cosines, the angle $\gamma_i$ between the lines $P_iQ_i$ and $Q_iO_B$ is defined as,

$$\cos \gamma_i = \frac{|P_iO_B|^2 - L_i^2 - r^2}{2L_ir},$$  \hspace{1cm} (3.9)$$

where $O_B$ is the origin of the coordinate system $X_BZ_B$, and the distance between points $P_i$ and $O_B$ and is given by,

$$|P_iO_B| = \sqrt{(P_{ix} - O_{Bx})^2 + (P_{iz} - O_{Bz})^2}.  \hspace{1cm} (3.10)$$

The supplementary angle of $\gamma_i$ is $\beta_i$; therefore, $\gamma_i + \beta_i = \pi$. The angles $\beta_1$ and $\gamma_1$ are shown in Figure 3.2. The moment arm of $F_i$ about the mass center $O_B$ of the brain is,

$$d_i = r \sin \beta_i = r \sin \gamma_i.  \hspace{1cm} (3.11)$$

Using Newton’s second law and Euler’s equation, the full equations of motion of the brain are,

$$m_b\ddot{x}_b = \sum_{i=1}^{4} F_i \cos \alpha_i,$$

$$m_b\ddot{z}_b = \sum_{i=1}^{4} F_i \sin \alpha_i,$$

$$I_b\ddot{\theta}_b = \sum_{i=1}^{4} F_id_i,$$  \hspace{1cm} (3.12)$$

where the force $F_i$ can be obtained by substituting Equations 3.4 and 3.6 into Equation 3.7. The moment arm $d_i$ is defined in Equation 3.11.

The equations of motion in 3.12 are nonlinear because of the trigonometric and square root functions. The experimental data provide the head kinematics that can be used as inputs to the system. By integration, the translational and angular displacement of the skull are obtained. Using the transformation in Equation 3.1, the coordinates of points $P_i$ can
be expressed in the global frame. The only unknowns in Equations 3.12 are $x_b$, $z_b$, and $\theta_b$, which can be solved numerically for a given brain mass and moment of inertia. Once the kinematics of the brain is obtained, the relative brain displacement with respect to the skull can be found by comparing the measured skull kinematics and calculated brain kinematics.

### 3.4 Parameter Selection

To solve the equations of motion in 3.12, the model parameters including the inertia properties of the skull and brain and spring and damping coefficients need to be determined. This section explains the determination of different model parameters.

#### 3.4.1 Brain Mass

The brain has a mass of 1.40 kg because the average mass of the human brain is 1.30 to 1.50 kg [9, 80]. The total mass of the head is 4.5 kg according to the published data of the average mass of the human head [14, 125, 105]. Therefore, the skull mass is 3.10 kg, including all of the mass of the head except the brain.

#### 3.4.2 Brain Moment of Inertia

The principle moments of inertia of the human head have been measured and calculated by many researchers, and they are listed in Table 3.1. Unfortunately, experimental data for the human brain are not available. In his twelve degree-of-freedom lumped parameter head model, Alem [2] chose 0.011 kg·m$^2$ and 0.022 kg·m$^2$ as the moments of inertia for the skull and the brain, respectively. Compared with the values listed in Table 3.1, the moment of inertia of the whole head is excessively large in Alem’s work, particularly due to the moment of inertia of the brain. The moment of inertia of the human brain can be
approximately calculated by assuming the brain has the simple geometry of one half an ellipsoid or a full ellipsoid.

<table>
<thead>
<tr>
<th>Moment of Inertia (kg·m²)</th>
<th>Becker et al. [8]</th>
<th>Chandler et al. [14]</th>
<th>McConville et al. [70]</th>
<th>Zatsiorsky &amp; Seluyanov [125] a</th>
<th>Average</th>
</tr>
</thead>
<tbody>
<tr>
<td>$I_{xx}$</td>
<td>0.0199</td>
<td>0.0174</td>
<td>0.0204</td>
<td>0.0271</td>
<td>0.0212</td>
</tr>
<tr>
<td>$I_{yy}$</td>
<td>0.0221</td>
<td>0.0164</td>
<td>0.0233</td>
<td>0.0293</td>
<td>0.0228</td>
</tr>
<tr>
<td>$I_{zz}$</td>
<td>0.0134</td>
<td>0.0203</td>
<td>0.0151</td>
<td>0.0201</td>
<td>0.0172</td>
</tr>
</tbody>
</table>

aCalculated for a person having 73 kg weight and 174 cm height.

Table 3.1: Principle moments of inertia of the human head in the literature.

**Full Ellipsoid Model**

As shown in Figure 3.3, the brain is assumed to have a full ellipsoid shape with three principle axes. According to the published data in [97], the average nominal length, width, and height of the human brain are 167 mm, 140 mm, and 93 mm, respectively. The mass of the ellipsoid is given by,

$$m = \frac{4}{3} \pi \rho abc,$$

(3.13)

where $\rho$ is the density of the human brain with an average value at 1.05 g/cm³ [14]. $a$, $b$, and $c$ are half the lengths of the principle axes, which coincide with the $X$, $Y$, and $Z$ axes, respectively. Using the nominal dimension of the brain [97], i.e., $2a = 167$ mm, $2b = 140$ mm, $2c = 93$ mm, the mass of the ellipsoid is 1.195 kg, which is slightly less than the average mass of the human brain. By scaling the principle axes of the ellipsoid, the new axes have length of 176 mm, 148 mm, and 98 mm. Calculating the mass again gives 1.403 kg, which is in the range of the average mass of the human brain.
The principle moments of inertia of an ellipsoid are given by,

\[
I_{xx} = \frac{m}{5} \left( b^2 + c^2 \right), \\
I_{yy} = \frac{m}{5} \left( a^2 + c^2 \right), \\
I_{zz} = \frac{m}{5} \left( a^2 + b^2 \right).
\]  \hspace{1cm} (3.14)

Substituting the mass and the new geometrical data into Equations 3.14 yields the moments of inertia of the human brain \( I_{xx} = 0.0022 \text{ kg} \cdot \text{m}^2 \), \( I_{yy} = 0.0028 \text{ kg} \cdot \text{m}^2 \), and \( I_{zz} = 0.0037 \text{ kg} \cdot \text{m}^2 \).

**Half Ellipsoid Model**

Alternatively, the human brain is assumed to be one half an ellipsoid with three principle axes having half lengths \( a \), \( b \), and \( c \), respectively, as shown in Figure 3.4. The mass of the half ellipsoid is given by,

\[
m = \frac{2}{3} \pi \rho abc.
\]  \hspace{1cm} (3.15)
Substituting the nominal average dimensions of the human brain into Equation 3.15 gives $m = 0.598$ kg, which is much less than the average mass of the human brain. It also suggests that the brain shape should be closer to a full ellipsoid than a half ellipsoid. Similarly, by scaling the lengths of the three principle axes, the new half ellipsoid has dimensions of $a = 224$ mm, $b = 186$ mm, and $c = 124$ mm. The mass of this new half ellipsoid is $1.420$ kg with the same density of $1.05$ g/cm$^3$. The corresponding principle moments of inertia of the half ellipsoid are $I_{xx} = 0.0035$ kg·m$^2$, $I_{yy} = 0.0047$ kg·m$^2$, and $I_{zz} = 0.0060$ kg·m$^2$.

![Figure 3.4: The human brain with a half-ellipsoidal shape.](image)

Although it is believed that the human brain should be closer to a full-ellipsoidal shape, the moment of inertia of the brain is estimated by taking the average of the moments of inertia of the full ellipsoid and the half ellipsoid. Therefore, the estimated values may be slightly larger than the actual moments of inertia of the human brain of an average size. The calculation gives $0.0028$, $0.0038$, and $0.0049$ kg·m$^2$ for the moments of inertia of the whole human brain about the $X$, $Y$, and $Z$ axes, respectively. For this new planar brain injury model in the sagittal plane, the moment of inertia of the brain about the $Y$ axis is chosen as $0.0038$ kg·m$^2$. Due to the smaller mass and smaller radius of gyration of the
brain compared with the skull, the moment of inertia of the brain should only contribute a minor portion of the moment of inertia of the whole head. As listed in Table 3.1, the average value in the literature is 0.0228 kg·m\(^2\) about the \(Y\) axis for the moment of inertia of the whole head. The moment of inertia of the human brain about the \(Y\) axis is about one fifth of the total moment of inertia of the whole human head.

By simplifying the brain as a spherical body, the radius is taken from the average nominal human brain sizes, where the length, width, and height are 167 mm, 140 mm, and 93 mm, respectively. Taking the average gives \(r = 67\) mm for the spherical brain. With the average density of 1.05 g/cm\(^3\), the mass of the spherical brain is about 1.32 kg, which falls within the reported range.

### 3.5 Validation of Full Sagittal Plane Model

To validate the brain injury model, the skull kinematics measured by the accelerometer array in Hardy et al’s [41] experiments are used as inputs, and the outputs of the brain injury model are the brain kinematics. The relative brain displacement within the skull is then obtained by comparing the skull and brain kinematics. The NDT data from the experiments are used to compare with the simulation results to tune the model parameters. Note that only the experimental data in the sagittal plane are used for the planar model.

The brain mass and brain moment of inertia have been selected in Section 3.4, where the average brain mass was selected as 1.40 kg and the moment of inertia of the brain about the \(Y\) axis 0.0038 kg·m\(^2\) for all the tests. With the chosen values of the brain mass and brain moment of inertia, a single set of spring and damping coefficients for all six tests were selected through trial and error so as to achieve output brain kinematics that match Hardy et al’s [41] experimental data for the corresponding input skull kinematics.
Simulations were executed in Simulink using variable-step Dormand-Prince integration of Equations 3.12. Having the skull kinematics from the experimental data as the inputs, the relative brain displacement at different NDT locations from the simulations were compared with the experimental data. The errors for all of the NDTs throughout the simulation were summed as a performance index to tune the spring and damping constants. The spring and damping constants were finalized by minimizing the total error. The selected values of the model parameters for all six tests are listed in Table 3.2.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Values</th>
<th>Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>$m_b$</td>
<td>1.40</td>
<td>kg</td>
</tr>
<tr>
<td>$m_s$</td>
<td>3.10</td>
<td>kg</td>
</tr>
<tr>
<td>$I_b$</td>
<td>0.0038</td>
<td>kg·m²</td>
</tr>
<tr>
<td>$I_s$</td>
<td>0.0190</td>
<td>kg·m²</td>
</tr>
<tr>
<td>$r$</td>
<td>0.067</td>
<td>m</td>
</tr>
<tr>
<td>$d$</td>
<td>0.040</td>
<td>m</td>
</tr>
<tr>
<td>$k_1$</td>
<td>25</td>
<td>kN/m</td>
</tr>
<tr>
<td>$k_2$</td>
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<td>110</td>
<td>Ns/m</td>
</tr>
<tr>
<td>$c_3$</td>
<td>20</td>
<td>Ns/m</td>
</tr>
<tr>
<td>$c_4$</td>
<td>350</td>
<td>Ns/m</td>
</tr>
</tbody>
</table>

Table 3.2: Parameters of the full brain injury model in the sagittal plane.

A resultant ±5 mm magnitude of relative brain displacement was achieved. The patterns of the relative displacements along the $X$ and $Z$ axes were also very similar to those observed in Hardy et al’s [41] study, as shown in Figures 3.5 to 3.10. In the figures, “$a_i$”
and “p_i” indicate NDTs in the anterior and posterior columns, respectively. The peak displacements at different NDT locations from the simulations were compared with those in the experimental data, and they are reasonably close, as shown in Figures 3.11 to 3.16.

Figure 3.17 shows that the relative angular displacement of the whole brain in the sagittal plane is about 4 or 5 degrees for all six tests, which is consistent with the findings that the relative rotation of the whole brain under low-severity impacts is about ±5 degrees. Using the measured head kinematics as inputs, the overall patterns of the brain rotation from the planar model agree with the results in Chapter 2 from analysis of the measured relative brain motion data in the same six tests. Tests C755-T2 and C755-T3 present similar patterns for the brain rotation since they are both under occipital acceleration impact. Similar patterns are also found in tests C383-T1 and C383-T3, which are both under frontal deceleration impact. The angular motion obtained from the sagittal plane model agrees with the experimental data.

3.6 Simplified Sagittal Plane Model

3.6.1 Model Description

Developed in Section 3.3.2, the full equations of motion for the brain and skull are highly nonlinear due to the trigonometric, square, and square root functions. However, the angular displacement of the brain with respect to the skull is expected to be small under low-severity impacts. The angle that each Kelvin element makes with the horizontal only has minor variation, so it is assumed that each Kelvin element always makes a 45-degree angle with the horizontal. With this assumption, the equations of motion for the brain and skull can be significantly simplified, and linear equations of motion are obtained. The simplified equations of motion can be solved analytically for simple inputs. This model is
Figure 3.5: Comparison of the relative brain motion at different NDT locations from the experimental data and the simulation results for test C755-T2.
Figure 3.6: Comparison of the relative brain motion at different NDT locations from the experimental data and the simulation results for test C755-T3.
Figure 3.7: Comparison of the relative brain motion at different NDT locations from the experimental data and the simulation results for test C755-T5.
Figure 3.8: Comparison of the relative brain motion at different NDT locations from the experimental data and the simulation results for test C383-T1.
Figure 3.9: Comparison of the relative brain motion at different NDT locations from the experimental data and the simulation results for test C383-T3.
Figure 3.10: Comparison of the relative brain motion at different NDT locations from the experimental data and the simulation results for test C383-T4.
Figure 3.11: Peak displacements in the sagittal plane in test C755-T2. “a_i” and “p_i” indicate NDTs in the anterior and posterior columns, respectively.

Figure 3.12: Peak displacements in the sagittal plane in test C755-T3. “a_i” and “p_i” indicate NDTs in the anterior and posterior columns, respectively.
Figure 3.13: Peak displacements in the sagittal plane in test C755-T5. “a_i” and “p_i” indicate NDTs in the anterior and posterior columns, respectively.

Figure 3.14: Peak displacements in the sagittal plane in test C383-T1. “a_i” and “p_i” indicate NDTs in the anterior and posterior columns, respectively.
Figure 3.15: Peak displacements in the sagittal plane in test C383-T3. \( a_i \) and \( p_i \) indicate NDTs in the anterior and posterior columns, respectively.

Figure 3.16: Peak displacements in the sagittal plane in test C383-T4. \( a_i \) and \( p_i \) indicate NDTs in the anterior and posterior columns, respectively.
Figure 3.17: Relative brain angular displacement for all six tests using the full brain injury model in the sagittal plane. On the top, T2 and T3 are occipital acceleration tests, and T5 is a frontal acceleration test. On the bottom, T1 and T3 are frontal deceleration tests, and T4 is an occipital deceleration test.

similar to the full model (the brain injury model with full equations of motion) depicted in Section 3.3.1. Small angular motion of the brain is assumed for this model under low-severity impacts. The simplified brain injury model in the sagittal plane is shown in Figure 3.18, which is similar to the full model.

3.6.2 Equations of Motion

As shown in Figure 3.19, a spring connecting the skull and the brain has an initial angle of 45 degrees with the horizontal. When the brain undergoes a displacement $\Delta x$ along the $X$ axis with respect to the skull, the spring changes from its original length $L_0$ to a
new length $L$. An assumption is made that the displacement $\Delta x$ along the $X$ axis is small compared with the original spring length $L_0$.

Therefore, the actual change of spring length $\Delta L$, which is the difference between $L_0$ and $L$, is given by,

$$\Delta L = \Delta x \cdot \cos \left( \frac{\pi}{4} \right). \quad (3.16)$$

The force in the spring due to relative motion on the $X$ direction is,

$$P_x = k \cdot \Delta L = k \cdot \Delta x \cdot \cos \left( \frac{\pi}{4} \right). \quad (3.17)$$

Since the spring is assumed to always make a 45-degree with the horizontal, the direction of the spring force $P_x$ does not change. Therefore, the $X$ and $Z$ components of the spring force $P_x$ are,

$$P_{xx} = P_x \cdot \cos \left( \frac{\pi}{4} \right) = k \cdot \Delta x \cdot \cos \left( \frac{\pi}{4} \right) \cos \left( \frac{\pi}{4} \right) = \frac{1}{2} k \cdot \Delta x,$$

$$P_{xz} = P_x \cdot \sin \left( \frac{\pi}{4} \right) = k \cdot \Delta x \cdot \cos \left( \frac{\pi}{4} \right) \sin \left( \frac{\pi}{4} \right) = \frac{1}{2} k \cdot \Delta x. \quad (3.18)$$
Figure 3.19: Change of spring length due to the relative brain displacement along the $X$ axis.

Similar results are obtained when the relative displacement of the brain is along the $Z$ axis.

The force in the spring due to relative motion in the $Z$ direction is,

$$P_z = k \cdot \Delta L = k \cdot \Delta z \cdot \cos \left( \frac{\pi}{4} \right).$$

(3.19)

The $X$ and $Z$ components of the spring force $P_z$ are,

$$P_{zx} = P_z \cdot \cos \left( \frac{\pi}{4} \right) = k \cdot \Delta z \cdot \cos \left( \frac{\pi}{4} \right) \cos \left( \frac{\pi}{4} \right) = \frac{1}{2} k \cdot \Delta z,$$

$$P_{zz} = P_z \cdot \sin \left( \frac{\pi}{4} \right) = k \cdot \Delta z \cdot \cos \left( \frac{\pi}{4} \right) \sin \left( \frac{\pi}{4} \right) = \frac{1}{2} k \cdot \Delta z.$$

(3.20)

As shown in Figure 3.20, the assumption of a small angular displacement of the brain relative to the skull yields the change of the spring length $\Delta L$,

$$\Delta L = L - L_0 \approx d \cdot \Delta \theta,$$

(3.21)

where $\Delta \theta$ is the relative angular displacement of the brain about the $Y$ axis. Therefore, the spring force generated by the rotational motion of the brain is approximately,

$$P_\theta = k \cdot \Delta L = k \cdot d \cdot \Delta \theta.$$

(3.22)
The components of force $P_\theta$ along the $X$ and $Z$ axes are,

$$P_{\theta x} = \frac{\sqrt{2}}{2} k \cdot d \cdot \Delta \theta,$$

$$P_{\theta z} = \frac{\sqrt{2}}{2} k \cdot d \cdot \Delta \theta.$$

(3.23)

Figure 3.20: Change of spring length due to the relative brain rotation about the $Y$ axis. Note that “$P$” and “$Q$” indicate the connecting points here.

Applying the same assumptions as for the springs, the damping forces caused by the relative motion have similar formulas. The forces $Q_x$, $Q_z$, and $Q_\theta$ in the damper corresponding to the three types of relative brain motion are given by,

$$Q_x = \frac{\sqrt{2}}{2} c \cdot \Delta \dot{x},$$

$$Q_z = \frac{\sqrt{2}}{2} c \cdot \Delta \dot{z},$$

88
\[ Q_{\theta} = c \cdot d \cdot \Delta \theta. \]  \hspace{1cm} (3.24)

As shown in Figure 3.21, the \( X \) and \( Z \) axes represent the coordinates of the absolute displacement of the brain and the skull. The \( X' \) axis and \( Z' \) axes define the positive directions of the forces generated in the springs and dampers. Collecting the spring and damping forces expressed in Equations 3.17, 3.19, 3.22, and 3.24, the force in each Kelvin element can be obtained in terms of the relative displacements and velocities between the skull and the brain.

\[
F_1 = \frac{\sqrt{2}}{2} (\dot{x}_s - \dot{x}_b) c_1 + \frac{\sqrt{2}}{2} (\dot{z}_s - \dot{z}_b) c_1 + d (\dot{\theta}_s - \dot{\theta}_b) c_1 \\
+ \frac{\sqrt{2}}{2} (x_s - x_b) k_1 + \frac{\sqrt{2}}{2} (z_s - z_b) k_1 + d (\theta_s - \theta_b) k_1, \hspace{1cm} (3.25)
\]

\[
F_2 = \frac{\sqrt{2}}{2} (\dot{x}_s - \dot{x}_b) (-c_2) + \frac{\sqrt{2}}{2} (\dot{z}_s - \dot{z}_b) c_2 + d (\dot{\theta}_s - \dot{\theta}_b) c_2 \\
+ \frac{\sqrt{2}}{2} (x_s - x_b) (-k_2) + \frac{\sqrt{2}}{2} (z_s - z_b) k_2 + d (\theta_s - \theta_b) k_2, \hspace{1cm} (3.26)
\]

\[
F_3 = \frac{\sqrt{2}}{2} (\dot{x}_s - \dot{x}_b) c_3 + \frac{\sqrt{2}}{2} (\dot{z}_s - \dot{z}_b) c_3 + d (\dot{\theta}_s - \dot{\theta}_b) (-c_3) \\
+ \frac{\sqrt{2}}{2} (x_s - x_b) k_3 + \frac{\sqrt{2}}{2} (z_s - z_b) k_3 + d (\theta_s - \theta_b) (-k_3), \hspace{1cm} (3.27)
\]

\[
F_4 = \frac{\sqrt{2}}{2} (\dot{x}_s - \dot{x}_b) (-c_4) + \frac{\sqrt{2}}{2} (\dot{z}_s - \dot{z}_b) c_4 + d (\dot{\theta}_s - \dot{\theta}_b) (-c_4) \\
+ \frac{\sqrt{2}}{2} (x_s - x_b) (-k_4) + \frac{\sqrt{2}}{2} (z_s - z_b) k_4 + d (\theta_s - \theta_b) (-k_4). \hspace{1cm} (3.28)
\]

The equation of translational motion of the skull along the \( X \) axis is given by,

\[
\sum F_x = F_{sx} - (F_1 - F_2 + F_3 - F_4) \cos \left( \frac{\pi}{4} \right) = m_s \ddot{x}_s, \hspace{1cm} (3.29)
\]

where \( F_{sx} \) is the impact force acting on the skull along the \( X \) axis, and \( F_x \) indicates all of the external forces along the \( X \) axis on the skull. It yields,

\[
m_s \ddot{x}_s + \frac{1}{2} (c_1 + c_2 + c_3 + c_4) (\dot{x}_s - \dot{x}_b) + \frac{1}{2} (c_1 - c_2 + c_3 - c_4) (\dot{z}_s - \dot{z}_b) \]

89
Figure 3.21: Free body diagram of the brain for the simplified sagittal plane model.

\[ + \frac{\sqrt{2}}{2} d \left( c_1 - c_2 - c_3 + c_4 \right) \left( \dot{\theta}_s - \dot{\theta}_b \right) + \frac{1}{2} \left( k_1 + k_2 + k_3 + k_4 \right) \left( x_s - x_b \right) \]
\[ + \frac{1}{2} \left( k_1 - k_2 + k_3 - k_4 \right) \left( z_s - z_b \right) + \frac{\sqrt{2}}{2} d \left( k_1 - k_2 - k_3 + k_4 \right) \left( \theta_s - \theta_b \right) = F_{sx} \quad (3.30) \]

where \( x \) is the absolute displacement along the \( X \) axis, \( z \) is the absolute displacement along the \( Z \) axis, and \( \theta \) is the absolute angular displacement about the \( Y \) axis measured positive clockwise. The “dot” notation indicates derivatives with respect to time. The equation of translational motion of the brain along the \( X \) axis is,

\[ \sum F_x = (F_1 - F_2 + F_3 - F_4) \cos \left( \frac{\pi}{4} \right) = m_b \ddot{x}_b, \quad (3.31) \]

which leads to,

\[ m_b \ddot{x}_b - \frac{1}{2} \left( c_1 + c_2 + c_3 + c_4 \right) \left( \ddot{x}_s - \ddot{x}_b \right) - \frac{1}{2} \left( c_1 - c_2 + c_3 - c_4 \right) \left( \ddot{z}_s - \ddot{z}_b \right) \]
\[ - \frac{\sqrt{2}}{2} d \left( c_1 - c_2 - c_3 + c_4 \right) \left( \dot{\theta}_s - \dot{\theta}_b \right) - \frac{1}{2} \left( k_1 + k_2 + k_3 + k_4 \right) \left( x_s - x_b \right) \]
\[ - \frac{1}{2} \left( k_1 - k_2 + k_3 - k_4 \right) \left( z_s - z_b \right) - \frac{\sqrt{2}}{2} d \left( k_1 - k_2 - k_3 + k_4 \right) \left( \theta_s - \theta_b \right) = 0. \quad (3.32) \]
Similarly, the equation of translational motion of the skull along the $Z$ axis is,

$$\sum F_z = F_{sz} - (F_1 + F_2 + F_3 + F_4) \cos \left(\frac{\pi}{4}\right) = m_s \ddot{z}_s, \quad (3.33)$$

where $F_{sz}$ is the external force acting on the skull along the $Z$ axis, and $F_z$ indicates all the external forces along the $Z$ axis on the skull. It yields,

$$m_s \ddot{z}_s + \frac{1}{2} (c_1 - c_2 + c_3 - c_4) (\ddot{x}_s - \ddot{x}_b) + \frac{1}{2} (c_1 + c_2 + c_3 + c_4) (\ddot{z}_s - \ddot{z}_b) + \frac{\sqrt{2}}{2} d (c_1 + c_2 - c_3 - c_4) (\dot{\theta}_s - \dot{\theta}_b) + \frac{1}{2} (k_1 - k_2 + k_3 - k_4) (x_s - x_b) + \frac{1}{2} (k_1 + k_2 + k_3 + k_4) (z_s - z_b) + \frac{\sqrt{2}}{2} d (k_1 + k_2 - k_3 - k_4) (\theta_s - \theta_b) = F_{sz}. \quad (3.34)$$

The equation of translational motion of the brain along the $Z$ axis is,

$$\sum F_z = (F_1 + F_2 + F_3 + F_4) \cos \left(\frac{\pi}{4}\right) = m_b \ddot{z}_b, \quad (3.35)$$

which gives,

$$m_b \ddot{z}_b - \frac{1}{2} (c_1 - c_2 + c_3 - c_4) (\ddot{x}_s - \ddot{x}_b) - \frac{1}{2} (c_1 + c_2 + c_3 + c_4) (\ddot{z}_s - \ddot{z}_b) - \frac{\sqrt{2}}{2} d (c_1 + c_2 - c_3 - c_4) (\dot{\theta}_s - \dot{\theta}_b) - \frac{1}{2} (k_1 - k_2 + k_3 - k_4) (x_s - x_b) - \frac{1}{2} (k_1 + k_2 + k_3 + k_4) (z_s - z_b) - \frac{\sqrt{2}}{2} d (k_1 + k_2 - k_3 - k_4) (\theta_s - \theta_b) = 0. \quad (3.36)$$

The equation of angular motion of the skull about the $Y$ axis is given by,

$$\sum T_y = T_{sy} - d (F_1 + F_2 - F_3 - F_4) = I_s \ddot{\theta}_s, \quad (3.37)$$

where $T_{sy}$ is the external torque acting on the skull about the $Y$ axis, and $T_y$ indicates all of the external torques about the $Y$ axis on the skull. It yields,

$$I_s \ddot{\theta}_s + \frac{\sqrt{2}}{2} d (c_1 - c_2 - c_3 + c_4) (\ddot{x}_s - \ddot{x}_b) + \frac{\sqrt{2}}{2} d (c_1 + c_2 - c_3 - c_4) (\ddot{z}_s - \ddot{z}_b) + d^2 (c_1 + c_2 + c_3 + c_4) (\dot{\theta}_s - \dot{\theta}_b) + \frac{\sqrt{2}}{2} d (k_1 - k_2 - k_3 + k_4) (x_s - x_b) + \frac{\sqrt{2}}{2} d (k_1 + k_2 - k_3 - k_4) (z_s - z_b) + d^2 (k_1 + k_2 + k_3 + k_4) (\theta_s - \theta_b) = T_{sy}. \quad (3.38)$$
The equation of angular motion of the brain about the $Y$ axis is given by,

$$\sum T_y = d (F_1 + F_2 - F_3 - F_4) = I_b \ddot{\theta}_b, \quad (3.39)$$

which leads to,

$$I_b \ddot{\theta}_b - \frac{\sqrt{2}}{2} d (c_1 - c_2 - c_3 + c_4) (\dot{x}_s - \dot{x}_b) - \frac{\sqrt{2}}{2} d (c_1 + c_2 - c_3 - c_4) (\dot{z}_s - \dot{z}_b)$$

$$- d^2 (c_1 + c_2 + c_3 + c_4) (\dot{\theta}_s - \dot{\theta}_b) - \frac{\sqrt{2}}{2} d (k_1 - k_2 - k_3 + k_4) (x_s - x_b)$$

$$- \frac{\sqrt{2}}{2} d (k_1 + k_2 - k_3 - k_4) (z_s - z_b) - d^2 (k_1 + k_2 + k_3 + k_4) (\theta_s - \theta_b) = 0, \quad (3.40)$$

where $I_s$ and $I_b$ are the moments of inertia of the skull and the brain about the $Y$ axis, respectively. For simplification, new variables are introduced,

\[
\begin{align*}
    c_{11} &= \frac{1}{2} (c_1 + c_2 + c_3 + c_4), \\
    c_{12} &= \frac{1}{2} (c_1 - c_2 + c_3 - c_4), \\
    c_{13} &= \frac{\sqrt{2}}{2} d (c_1 - c_2 - c_3 + c_4), \\
    c_{21} &= \frac{1}{2} (c_1 - c_2 + c_3 - c_4), \\
    c_{22} &= \frac{1}{2} (c_1 + c_2 + c_3 + c_4), \\
    c_{23} &= \frac{\sqrt{2}}{2} d (c_1 + c_2 - c_3 - c_4), \\
    c_{31} &= \frac{\sqrt{2}}{2} d (c_1 - c_2 - c_3 + c_4), \\
    c_{32} &= \frac{\sqrt{2}}{2} d (c_1 + c_2 - c_3 - c_4), \\
    c_{33} &= d^2 (c_1 + c_2 + c_3 + c_4), \\
    k_{11} &= \frac{1}{2} (k_1 + k_2 + k_3 + k_4), \\
    k_{12} &= \frac{1}{2} (k_1 - k_2 + k_3 - k_4), \\
    k_{13} &= \frac{\sqrt{2}}{2} d (k_1 - k_2 - k_3 + k_4),
\end{align*}
\]
\[ k_{21} = \frac{1}{2} (k_1 - k_2 + k_3 - k_4), \]
\[ k_{22} = \frac{1}{2} (k_1 + k_2 + k_3 + k_4), \]
\[ k_{23} = \frac{\sqrt{2}}{2} d (k_1 + k_2 - k_3 - k_4), \]
\[ k_{31} = \frac{\sqrt{2}}{2} d (k_1 - k_2 - k_3 + k_4), \]
\[ k_{32} = \frac{\sqrt{2}}{2} d (k_1 + k_2 - k_3 - k_4), \]
\[ k_{33} = d^2 (k_1 + k_2 + k_3 + k_4). \]

With these new variables, the equations of motion for the skull and brain can be written in matrix form,

\[ m \ddot{x}_b + c \dot{x}_b + k x_b = g x_a, \quad (3.41) \]

where,

\[
\begin{align*}
    m &= \begin{pmatrix}
    m_b & 0 & 0 \\
    0 & m_b & 0 \\
    0 & 0 & I_b 
    \end{pmatrix}, \\
    c &= \begin{pmatrix}
    c_{11} & c_{12} & c_{13} \\
    c_{21} & c_{22} & c_{23} \\
    c_{31} & c_{32} & c_{33} 
    \end{pmatrix}, \\
    k &= \begin{pmatrix}
    k_{11} & k_{12} & k_{13} \\
    k_{21} & k_{22} & k_{23} \\
    k_{31} & k_{32} & k_{33} 
    \end{pmatrix}, \\
    g &= \begin{pmatrix}
    k_{11} & c_{11} & k_{12} & c_{12} & k_{13} & c_{13} \\
    k_{21} & c_{21} & k_{22} & c_{22} & k_{23} & c_{23} \\
    k_{31} & c_{31} & k_{32} & c_{32} & k_{33} & c_{33} 
    \end{pmatrix}, \\
    x_b &= \begin{pmatrix}
    x_b \\
    z_b \\
    \theta_b 
    \end{pmatrix}^T, \\
    x_s &= \begin{pmatrix}
    x_s & \dot{x}_s & z_s & \dot{z}_s & \theta_s & \dot{\theta}_s 
    \end{pmatrix}^T. 
\end{align*}
\]

For many impact tests using human subjects or crash test dummies, the accelerations of the head are directly measured using accelerometers mounted on the head. The measured head accelerations can be used as the skull accelerations for the brain injury model assuming that...
the accelerometers are rigidly connected with the skull. Therefore, Equations 3.30, 3.34, and 3.38 are not used for computing skull kinematics in this case. When the accelerations are not directly measured, however, the full set of Equations 3.41 can be used to calculate the resultant head and brain kinematics by measuring external forces acting on the head. Thus, only Equations 3.32, 3.36, and 3.40 are to be solved to obtain the absolute brain displacement. Finally, comparing the measured skull kinematics and the calculated brain kinematics yields relative brain-skull displacement.

### 3.6.3 Comparing Simplified and Full Models

The simplified equations of motion for the brain injury model have been derived in Section 3.6.2. These equations are now linear and much easier to solve compared with the nonlinear equations of motion derived in Section 3.3.2. To investigate how closely the simplified model (the brain injury model with simplified equations of motion) captures the key characteristics of relative brain motion, the same inputs are used for the simplified and full models to directly compare their outputs. A total of six tests from Hardy et al.’s work [41] have been used for the validation of the full model. These six tests are used here again to compare the simplified model with the full model. Although these tests involve low-severity impacts, the HIC15 values still span a large range from as low as 5.2 for test C755-T5 to as high as 163.7 for test C383-T4.

Figures 3.22 and 3.23 compare the relative displacements at the CG of the brain from the simplified model and the full model in tests C755-T5 and C383-T4. These two tests are selected to represent two different impact scenarios. C755-T2 was under an occipital acceleration impact, and C383-T4 was under a frontal deceleration impact. The results are almost identical for test C755-T5, where the curves are mostly overlapping. As impact
Figure 3.22: Comparing the relative brain displacements at the CG from the simplified brain injury model in the sagittal plane with those from the full model in test C755-T5.

Figure 3.23: Comparing the relative brain displacements at the CG from the simplified brain injury model in the sagittal plane with those from the full model in test C383-T4.
becomes more severe, as in test C383-T4, the results are still very close, as shown in Figure 3.23. The relatively small angular displacement of the brain is consistent with the small angle assumption made for the simplified model development. Further analysis indicates that the full and simplified models also have very similar results for the other four tests.

From the comparison of the simplified and full models, the validation of the full model also applies to the simplified model. Using the same set of model parameters listed in Table 3.2, the relative brain displacements for the simplified model only change slightly compared with the results in Figures 3.5 to 3.10. Therefore, the selection of a different set of model parameters for the simplified model is not necessary.

One of the advantages of the simplified model is that the computational load is much less than that for the full model. This improves the efficiency for the development of head impact simulations to study the brain responses. In addition, with simple inputs, such as impulse and sinusoidal functions, the availability of closed-form solutions for this simplified model will greatly facilitate further analysis, such as sensitivity analysis to be discussed in Chapter 6. Therefore, this simplified model will be used throughout the rest of this study.

### 3.7 Simplified Coronal Plane Model

#### 3.7.1 Introduction

A substantial proportion of injuries in automobile crashes result from side impacts. The Insurance Institute for Highway Safety (IIHS) reported that 31% of all passenger vehicle occupant deaths in 2004 in the U.S. occurred in side crashes, following 49% in frontal crashes [47]. Federal Motor Vehicle Safety Standards (FMVSS) 214 “Side Impact Protection” specifies dynamic and static performance requirements to ensure the crashworthiness
of vehicle side structures and to reduce the risk of serious and fatal injury to vehicle occupants involved in side impact crashes.

The analysis found that the majority of serious injuries are head-related in side impacts [35]. However, no head/brain injury criteria have been proposed nor accepted for current safety regulations under side impact. McIntosh et al. [72] studied head injury tolerance under side impacts using both PMHS tests and an FE head model. They found that head injury criteria in side impact can be based on characteristics of head kinematics. Head acceleration under lateral impacts should be less than 200 g, and preferably below 150 g. Gennarelli et al. [34] produced traumatic coma in 45 monkeys by accelerating the head without impact. They concluded that the tolerance of the brain to acceleration in terms of duration of coma was substantially less in the coronal plane than in the sagittal plane.

To study brain injuries under side impacts, this section develops an analytical model that can capture the key characteristics of brain motion in the coronal plane. This model can be combined with the model in the sagittal plane for better brain injury prediction under 3D head kinematics.

### 3.7.2 Model Description

The results in Section 3.6.3 show that the simplified model in the sagittal has very similar responses to the full model under low-severity impacts. Therefore, this section focuses on the development and validation of a simplified model in the coronal plane. Similar to the simplified model in the sagittal plane, an analytical brain injury model in the coronal plane is developed to study brain responses under side impact. When the impacts are from the lateral direction, head kinematics in the coronal plane are dominant.
As shown in Figure 3.24, the brain injury model in the coronal plane is similar to the one in the sagittal plane. The positive X, Y, and Z axes point to the anterior, left lateral, and superior directions, respectively. Positive angular displacement of the head about the X axis corresponds to right lateral tilt of the neck. The model consists of a circular skull and brain having masses, \( m_s \) and \( m_b \), and moments of inertia, \( I_s \) and \( I_b \), where the subscripts “s” and “b” indicate the skull and brain, respectively. The skull and brain are also connected by four Kelvin elements, each having a spring \( k_i \) and damper \( c_i \) in parallel, where \( i = 1 \cdots 4 \). The center of mass of the skull and the brain are assumed to be coincident at the geometric center. In the neutral position, each Kelvin element makes a 45-degree angle with the horizontal and is attached to the brain at a perpendicular offset distance of \( d \) with respect to the center of mass. The parameter \( r \) is the radius of the circular brain.

Figure 3.24: Schematic of the simplified brain injury model in the coronal plane. A circular skull and brain are connected by four Kelvin elements.
The spring and damping coefficients differ from those used in the sagittal plane model to capture the key characteristics of brain motion in the coronal plane. Similar to the sagittal plane model, the values of the spring and damping coefficients within the four Kelvin elements were selected asymmetrically so as to couple the relative translation and rotation between the brain and the skull. With pure linear or pure angular skull motion as inputs, the brain will have coupled rotation and translation within the skull. The three degrees of freedom in the coronal plane are all coupled.

### 3.7.3 Equations of Motion

The equations of motion are developed in a similar manner to those in the sagittal plane. Comparing the two planar models shown in Figures 3.18 and 3.24 suggests that the arrangement of the four Kelvin elements are the same in terms of the connecting points and orientations, except that they are located in different planes. The equations of motion for the coronal plane model can be simply obtained by replacing \( x \) with \( y \). Similar to Equations 3.32, 3.36, and 3.40, the equations of motion of the brain in the coronal plane are given by,

\[
m_b \ddot{y}_b - \frac{1}{2} \left( c_1 + c_2 + c_3 + c_4 \right) (\dot{y}_s - \dot{y}_b) - \frac{1}{2} \left( c_1 - c_2 + c_3 - c_4 \right) (\dot{z}_s - \dot{z}_b) - \frac{\sqrt{2}}{2} d (c_1 - c_2 - c_3 + c_4) (\dot{\theta}_s - \dot{\theta}_b) - \frac{\sqrt{2}}{2} d (k_1 + k_2 + k_3 + k_4) (y_s - y_b) = 0,
\]

\[
m_b \ddot{z}_b - \frac{1}{2} \left( c_1 - c_2 + c_3 - c_4 \right) (\dot{y}_s - \dot{y}_b) - \frac{1}{2} \left( c_1 + c_2 + c_3 + c_4 \right) (\dot{z}_s - \dot{z}_b) - \frac{\sqrt{2}}{2} d (c_1 + c_2 - c_3 + c_4) (\dot{\theta}_s - \dot{\theta}_b) - \frac{\sqrt{2}}{2} d (k_1 - k_2 - k_3 + k_4) (z_s - z_b) = 0,
\]

\[
I_b \ddot{\theta}_b - \frac{\sqrt{2}}{2} d (c_1 - c_2 - c_3 + c_4) (\dot{y}_s - \dot{y}_b) - \frac{\sqrt{2}}{2} d (c_1 + c_2 - c_3 - c_4) (\dot{z}_s - \dot{z}_b) = 0,
\]
\[-d^2 (c_1 + c_2 + c_3 + c_4) \left( \dot{\theta}_s - \dot{\theta}_b \right) - \frac{\sqrt{2}}{2} d (k_1 - k_2 - k_3 + k_4) (y_s - y_b) \]
\[-\frac{\sqrt{2}}{2} d (k_1 + k_2 - k_3 - k_4) (z_s - z_b) - d^2 (k_1 + k_2 + k_3 + k_4) \left( \theta_s - \theta_b \right) = 0, \quad (3.44)\]

where \(\ddot{y}_b\) is the linear acceleration of the brain along the \(Y\) axis, \(\ddot{z}_b\) the linear acceleration of the brain along the \(Z\) axis, and \(\dot{\theta}_b\) the angular acceleration of the brain about the \(X\) axis. Other quantities have similar definitions to those in Section 3.6. With head kinematics as inputs, the outputs of the model are the brain kinematics. The difference of these two yields relative brain motion with respect to the skull. Similarly, the full equations of motion can be obtained by simply swapping the \(x\) and \(y\) terms in Equations 3.12, and they are not repeated here.

### 3.7.4 Model Validation

One key assumption is that the brain motions in the coronal plane and sagittal plane are decoupled. In other words, the brain motion in the coronal plane only results from the head kinematics in the coronal plane. The head kinematics in the sagittal plane do not cause brain motion in the coronal plane.

The experimental data from Hardy et al.’s [41] work are used to validate the coronal plane model because of the scarcity of experimental data under side impacts. In Hardy et al.’s [41] tests, both 3D head kinematics and relative brain motion were measured under frontal and occipital impacts. Although the relative brain motion in the sagittal plane is particularly interesting because of the nature of the impacts [41] and the head kinematics and relative brain motion in the coronal plane are smaller, they are still significant. As discussed in the experimental data analysis, the peak relative brain motion is about 2-3 mm in the coronal plane, while it is about 4-5 mm in the sagittal plane. In general, the relative
brain rotations are within ±3 degrees and ±5 degrees in the coronal and sagittal planes, respectively.

The NDTs were implanted in the brain in anterior and posterior columns, which form a sagittal plane but lie in two different coronal planes. In the coronal plane, the combination of the two columns does not move as a rigid body, but each individual column still closely follows a rigid body displacement. Therefore, the validation method for the sagittal plane model is not suitable for the coronal plane model. The peak displacement of all NDTs are compared with the experimental data, as well as the angular motion of the brain. In this way, the detailed patterns of brain motion may not closely match the measured motion patterns at each individual NDT location, but the key characteristics of brain motion can be captured. Finally, a single set of spring and damping constants are chosen to yield similar brain motions to the experimental data for both the anterior and posterior columns.

Applying the head kinematics measured in the experiments to the brain injury model, the relative brain displacement can be obtained. Note that only the head kinematics in the coronal plane are used for the validation of the coronal plane model. The model parameters are selected in a similar manner to that for the sagittal plane model. The brain mass is 1.40 kg with an averaged size. The brain mass moment of inertia has been calculated in Section 3.4.2. The calculation gives 0.0028 kg·m² as the mass moment of inertia of the whole brain about the X axis. As discussed, the value is most likely overestimated, so a slightly smaller brain moment of inertia of 0.0025 kg·m² is chosen for the coronal plane model. It is also very close to the computed value of 0.0023 kg·m² with Kleiven’s FE head model [57], which will be discussed in detail in Sections 6.6 and 6.7. The average mass moment of inertia of the whole head is 0.0212 kg·m² from the literature, as listed in Table 3.1.
Simulations were executed in Simulink using variable-step Dormand-Prince integration to solve Equations 3.42, 3.43, and 3.44. With the skull kinematics from the experimental data as the inputs, the peak brain displacement at different NDT locations from the simulations were compared with the experimental data. The error for all of the NDTs throughout the simulation was summed as a performance index to tune the spring and damping constants. The model parameters are summarized in Table 3.3.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Values</th>
<th>Units</th>
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<tr>
<td>$m_b$</td>
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<td>kg</td>
</tr>
<tr>
<td>$m_s$</td>
<td>3.10</td>
<td>kg</td>
</tr>
<tr>
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<td>kg⋅m²</td>
</tr>
<tr>
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<td>0.0180</td>
<td>kg⋅m²</td>
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<td>0.067</td>
<td>m</td>
</tr>
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<td>$k_1$</td>
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<td>kN/m</td>
</tr>
<tr>
<td>$k_2$</td>
<td>5</td>
<td>kN/m</td>
</tr>
<tr>
<td>$k_3$</td>
<td>10</td>
<td>kN/m</td>
</tr>
<tr>
<td>$k_4$</td>
<td>70</td>
<td>kN/m</td>
</tr>
<tr>
<td>$c_1$</td>
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<td>Ns/m</td>
</tr>
<tr>
<td>$c_2$</td>
<td>50</td>
<td>Ns/m</td>
</tr>
<tr>
<td>$c_3$</td>
<td>20</td>
<td>Ns/m</td>
</tr>
<tr>
<td>$c_4$</td>
<td>150</td>
<td>Ns/m</td>
</tr>
</tbody>
</table>

Table 3.3: Parameters of the simplified brain injury model in the coronal plane.

As shown in Figure 3.25, the peak brain displacements at all of the NDT locations from the analytical model are reasonably close to those measured in the experiments. The upper subfigure compares the peak relative brain displacements in the $Y$ direction from the analytical model and experimental data. In general, the peak measured brain motion decreases from NDT $a_1$ to $a_5$ and from $p_1$ to $p_5$. It suggests that the NDTs closer to the CG have relatively larger motion. Note that NDTs $a_1$ and $p_1$ are located closer to the CG of
the brain. The relative motion decreases at the NDT locations approaching the brain-skull boundary. The overall trend of the resultant brain displacement from the analytical model closely matches that of the measured brain motion at different NDT locations. The lower subfigure compares the relative brain displacements in the $Z$ direction for different NDT locations. The variation of the brain motions in the $Z$ direction is generally smaller than that in the $Y$ direction. Compared with the experimental data, the relative brain displacements from the simplified coronal plane model are reasonably close to the measured brain motion in terms of magnitudes.

Similar results are obtained for the other five tests, as shown in Figures 3.26 to 3.30. In general, the peak brain displacements for various NDT locations are reasonably close to the experimental brain motion data, but some of the data points do not match very well. Test C755-T5 presents relatively larger differences between the simulation and experimental results in the $Z$ direction. The difference of the peak brain displacements in the $Y$ direction for the anterior column in test C383-T1 is also considerable. However, the discrepancy of the matching for a few NDTs does not affect the overall validation of the coronal plane model.

Figure 3.31 shows the relative brain rotation for all six tests. The angular displacement for the whole brain is generally within $\pm 3$ degrees for the analytical model. It is consistent with the findings from the experimental data analysis. Smaller relative brain angular motion in the coronal plane may result from the special brain structure and can be explained by the supportive nature of the falx cerebri that divides the brain into hemispheres [58].
Figure 3.25: Peak displacement in the coronal plane for different NDTs in test C755-T2. “a_i” and “p_i” indicate NDTs in the anterior and posterior columns, respectively.

Figure 3.26: Peak displacement in the coronal plane for different NDTs in test C755-T3. “a_i” and “p_i” indicate NDTs in the anterior and posterior columns, respectively.
Figure 3.27: Peak displacement in the coronal plane for different NDTs in test C755-T5. “a_i” and “p_i” indicate NDTs in the anterior and posterior columns, respectively.

Figure 3.28: Peak displacement in the coronal plane for different NDTs in test C383-T1. “a_i” and “p_i” indicate NDTs in the anterior and posterior columns, respectively.
Figure 3.29: Peak displacement in the coronal plane for different NDTs in test C383-T3. 
“a_i” and “p_i” indicate NDTs in the anterior and posterior columns, respectively.

Figure 3.30: Peak displacement in the coronal plane for different NDTs in test C383-T4. 
“a_i” and “p_i” indicate NDTs in the anterior and posterior columns, respectively.
Figure 3.31: Relative brain angular displacement for all six tests using the simplified brain injury model in the coronal plane. The rotation of the whole brain in the coronal plane is generally within ±3 degrees.

3.8 Discussion

Based on the understanding of brain motion patterns under impact, two planar lumped-parameter brain injury models were developed to capture the key characteristics of relative brain motion. The model development was based on the fundamental assumption that the linear and angular movements of the brain with respect to the skull are coupled. The full equations of motion were derived and solved numerically for the model validation using experimental brain motion data. Two simplified planar models were also developed. With
simple inputs, the simplified models can be solved analytically and used for further study of brain injury predictions.

The experimental brain motion data were collected under frontal and occipital impacts. The 3D data of relative brain motion and head accelerations were measured during the tests. Due to the nature of the impacts, the brain and head motions in the sagittal plane are dominant, while the out-of-sagittal-plane motions have smaller magnitudes. The validation of the sagittal plane model was conducted without considering the out-of-sagittal-plane head accelerations since they were less significant.

Ideally, the validation of the coronal plane model should use a similar method, relying on experimental data under side impact. Due to the lack of experimental data in the coronal plane, however, the same set of experimental data under frontal and occipital impacts was used. By assuming that the brain motions in the sagittal and coronal planes are decoupled, taking head accelerations in the coronal plane as inputs yielded relative brain motion in the same plane. With more experimental brain motion data available in the future, the validation of the coronal plane model could be improved and further verified.

These two planar brain injury models both have averaged brain and head size. The mass moment of inertia of the brain has not yet been studied extensively in the past. No values for the brain moment of inertia were found in the literature. The values of the brain moment of inertia were obtained by estimating the human brain with regular shapes, and they may not be very accurate. However, the estimation is very close to the value calculated using an FE model that will be discussed in detail in Sections 6.6 and 6.7. It is believed that these estimations should fall within a reasonable range. With experimental measurements available, the estimation of this important quantity can be verified.
DAI, cerebral contusion, and ASDH are the three types of closed brain injuries that frequently occur under impact. These injury mechanisms may be explained by relative brain motion within the skull. The functional output of the planar brain injury models proposed in this chapter is the relative displacement of the brain, so these models provide information directly related to ASDH. Increased linear and angular head accelerations generate larger relative brain motion within the skull and therefore, higher risk of different brain injuries. The analytical brain injury models need to be further extended with various injury measures for the prediction of different brain injuries.

3.9 Conclusions

Two planar, lumped-parameter human brain injury models having two masses, four springs and four dampers have been developed. The coupling between translation and rotation of the brain relative to the skull is embedded in the structure of the model to capture this important characteristic of brain motion. For simple inputs, analytical solutions are available using the models with simplified equations of motion. Thus, the model can be directly employed to investigate brain injury mechanisms related to relative brain motion under impacts. The parameter selection for the model was based on experimental data of relative brain motion within the skull. The results show that the planar brain injury models are capable of capturing the key characteristics of relative brain motion with reasonable accuracy.
CHAPTER 4

STRAIN MEASUREMENT AND ITS APPLICATION

4.1 Introduction

Occurring over a more widespread area than focal brain injury, DAI is one of the most devastating types of TBI and is the most significant cause of morbidity in patients with TBI [120]. Strain deformation induced by shear force from sudden acceleration or deceleration of the head during impact has been recognized as a major cause of DAI, resulting in tearing axons, which lose the capability of transferring messages between neurons. However, further studies are needed to fully understand the mechanisms of DAI. Various experimental tests and mathematical models have been employed in the past decades to understand the mechanisms of DAI for better injury prediction and prevention.

Based on the planar brain injury models, which were validated against the most advanced experimental brain motion data under low-severity impacts, a simple measure of strain for DAI prediction is presented in this chapter. The objective is to validate the proposed strain measure under low-severity impacts. It is implemented by matching strain measurement with severity of impacts using selected material properties of the brain tissue. The capabilities of brain injury prediction using the strain measure are evaluated in more
severe, realistic crash tests. The simple model is also compared with the HIC criterion and the SIMon FE head model.

4.2 Background

Strain in the brain tissue has been recognized by many researchers as one of the most important factors causing traumatic brain injury from automobile crashes. Experiments have been conducted for years to study strain damage to the brain under impacts. Based on experimental data from primates, Margulies and Thibault [68] concluded that strains ranging from 0.05 to 0.10 correspond to moderate-to-severe DAI. Bain and Meaney [5] conducted experiments by stretching multiple right optic nerves from an adult guinea pig to produce axonal injuries. They found that functional impairment occurs at a threshold of 0.18 Lagrangian strain. Morrison III et al. [76] used a hippocampal slice of a Wistar rat brain to study the tissue-level tolerance criterion. They suggested that a tissue Lagrangian strain of 0.20 may be a transition between non-injurious and injurious loads dependent on strain rates, which probably lie between 10 and 50 s$^{-1}$. Maxwell et al. [69] concluded that a 0.20-0.30 stretching of a neuron’s membrane leads to a chemical poisoning that causes neuronal death after around twelve hours. In Wolf et al’s work [123], however, it was found that neurons were able to survive even under a 0.80 stretching. Bayly et al. [7] measured brain deformation in human volunteers under mild occipital deceleration. The strains were typically 0.02-0.05 during these mild impacts. In general, these studies suggest that the threshold of strain causing brain injury lies between 0.15 and 0.20 depending on different strain rates.

Recent mathematical studies on strain damage to brain tissue are dominated by FE modeling work. Zhang et al. [127] compared brain responses under frontal and lateral
impacts using a 3D FE head model. They found that the strain experienced in the brain tissue due to lateral impact is much higher. Takhounts and Eppinger [111] developed the SIMon FE head model to predict various brain injuries due to impact based on a 3D FE model originally developed by Bandak and Eppinger [6] and later improved by DiMasi et al. [22]. In Takhounts and Eppinger’s work [111], Lagrangian strain of 0.15 is used as the threshold value of DAI in the FE model. Although offering detailed modeling of anatomical structures, these FE models have not been fully validated due to the lack of complete knowledge of the complex material properties and interface conditions of the human head. Lumped-parameter models have also been used to study head/brain injuries under impacts [104, 11, 107, 66, 3, 124]. However, none of those lumped-parameter models has a validated measure for brain injury prediction under impacts.

Due to the limitation of the rigid body model, strain cannot be measured directly using the brain injury model developed herein. However, an alternative method is introduced to make the measure of strain possible. Like other biological materials, human brain tissue is inhomogeneous, anisotropic, and nonlinearly viscoelastic [111]. However, for reducing calculation time and the number of experiments, brain tissue is generally assumed to be homogeneous, isotropic, and linearly viscoelastic in mathematical modeling work [31, 75, 111]. For simplicity, many earlier FE human head models adopted linear elastic material constitutive laws to model brain tissue [118, 44, 51, 95, 130, 18], while more recent FE models used linear viscoelastic material to model brain tissue [23, 22, 1, 127]. The selection of material properties for brain tissue continues to be a big challenge for mathematical modeling work because of the lack of complete knowledge of biological tissues. For FE models having either linear elastic or linear viscoelastic brain tissue, the values of selected material properties span a very large range. This work uses both linear elastic and linear
viscoelastic representations of brain material for the planar brain injury models to develop a simple measure of strain.

4.3 Model Development

4.3.1 Strain Measure for Sagittal Plane Model

Based on the simplified brain injury model in the sagittal plane, a measure of strain for DAI prediction is proposed in this section. The brain is assumed spherical, although its motion is limited within the sagittal plane. The brain is divided into two hemispheres with a horizontal cross section passing through the CG of the whole brain. Figure 4.1 shows the free body diagram of the upper hemispherical brain. The strain profile on the base of the hemisphere is used as the strain measure in the brain tissue.

![Free body diagram for the upper hemispherical brain.](image)

Figure 4.1: Free body diagram for the upper hemispherical brain.

Defined earlier in this work, as shown in Figure 4.1, \( F_1 \) and \( F_4 \) are the forces generated in Kelvin elements 1 and 4. \( V \) is the shear force acting on the base of the hemisphere, and \( N \) is the normal force. The shear and normal forces are assumed to be evenly distributed.
over the cross section so that single forces concentrated at the center of the base can be considered for simplicity of analysis. Note that the rotation of the brain is partially caused by unevenly distributed forces on the base. Recall that the time history of the accelerations of the brain and the forces $F_1$ and $F_4$ have already been obtained; therefore, the shear force $V$ and the normal force $N$ can be derived using Newton’s second law,

\[
\sum F_x = m_h \ddot{x}_b, \\
\sum F_z = m_h \ddot{z}_b, 
\]

(4.1)

where $m_h = 0.7 \text{ kg}$ is the mass of the hemispherical brain. Expanding,

\[
V + (F_1 - F_4) \cos \left(\frac{\pi}{4}\right) = m_h \ddot{x}_b, \\
N + (F_1 + F_4) \cos \left(\frac{\pi}{4}\right) = m_h \ddot{z}_b. 
\]

(4.2)

Substituting $\ddot{x}_b$ and $\ddot{z}_b$ into Equations 4.2 gives the forces on the base of the upper hemispherical brain,

\[
V = \frac{1}{2} (-F_1 - F_2 + F_3 + F_4) \cos \left(\frac{\pi}{4}\right), \\
N = \frac{1}{2} (-F_1 + F_2 + F_3 - F_4) \cos \left(\frac{\pi}{4}\right). 
\]

(4.3)

The brain can also be divided into two hemispheres, back and front, using a vertical cross section passing through the CG, as shown in the Figure 4.2. The shear and normal forces on the base of the back hemisphere are obtained in a similar manner,

\[
V + (F_1 + F_2) \cos \left(\frac{\pi}{4}\right) = m_h \ddot{x}_b, \\
N + (F_1 - F_2) \cos \left(\frac{\pi}{4}\right) = m_h \ddot{z}_b. 
\]

(4.4)

Similarly, substituting $\ddot{x}_b$ and $\ddot{z}_b$ into Equations 4.4 gives the forces on the base of the back hemispherical brain,

\[
V = \frac{1}{2} (-F_1 - F_2 + F_3 + F_4) \cos \left(\frac{\pi}{4}\right), 
\]
\[ N = \frac{1}{2} (-F_1 + F_2 + F_3 - F_4) \cos \left( \frac{\pi}{4} \right). \]  

(4.5)

Figure 4.2: Free body diagram for the back hemispherical brain.

Comparing Equations 4.3 and 4.5, the shear and normal forces on the base of the upper hemisphere are equal to the shear and normal forces on the base of the back hemisphere. The same forces result regardless of whether the cross section is horizontal or vertical. Similarly, the forces on the lower hemisphere are the same as those on the front hemisphere. For this sagittal plane model, the upper hemispherical brain is used to develop the strain measure. Shear stress and normal stress on the base of the upper hemisphere are given by,

\[ \sigma_z = \frac{N}{A}, \]

\[ \tau_{xz} = \frac{V}{A}, \]  

(4.6)
where \( A \) is the area of the base of the hemisphere. The average length, width, and height of the human brain are 167 mm, 140 mm, and 93 mm, respectively [97]; therefore, the average radius of the base is 67 mm.

This section proposes a simple scheme of strain measure for the rigid body model. With known time history of the forces in Kelvin elements 1 and 4, the normal force \( N \) and shear force \( V \) on the horizontal base of the hemispherical brain are calculated using Equations 4.3. The normal and shear stresses are obtained using Equations 4.6. In order to calculate the strains, the material properties of the human brain need to be selected so that the calculated strains are consistent with the severity of impacts.

### 4.3.2 Strain Measure for Coronal Plane Model

Similarly, a measure of strain for the simplified model in the coronal plane is also proposed. The brain is assumed to be spherical, although its motion is limited within the coronal plane. The brain can be divided into two hemispheres using either a horizontal or vertical cross section. As discussed in Section 4.3.1, the forces on the base of the hemispherical brain are the same regardless of the cross section. For this coronal plane model, the brain is divided into left and right hemispheres, with a vertical cross section passing through the CG of the whole brain, as shown in Figure 4.3. It allows an easy construction of 3D strain configuration, which will be discussed in detail in Section 5.3. The right hemispherical brain is used to develop the strain measure. The strain profile on the base of the hemisphere is used as the strain measure in the brain tissue.

With known time history of the forces in Kelvin elements 1 and 2, the normal force \( N \) and shear force \( V \) on the vertical base of the hemispherical brain are calculated using the head kinematics as inputs. The equations for the normal and shear forces are identical to
Equations 4.5. In a similar manner, the stress on the base of the hemisphere is calculated with the known area of the base. The strains are obtained with selected material properties of the brain tissue.

4.4 Material Properties of Brain Tissue

The material properties of human brain tissue are needed to calculate the strains, while other parameters, such as the spring and damping constants, have already been chosen in Chapter 3. By applying experimentally measured head kinematics to the strain model, the normal strain and shear strain in the brain tissue are calculated using the proposed scheme of strain measure. The brain material properties are selected such that the calculated strains can best match the severity of impacts.
4.4.1 Linear Elastic Brain Material

Linear elastic brain tissue was used in earlier modeling work to study brain responses during impacts. However, the value of the Young’s modulus of brain tissue varies in a large range in the literature. Morrison III et al. [76] assumed brain tissue to be linearly elastic with a Young’s modulus of 10 kPa in their FE model to predict the strain field in a stretched culture of rat brain tissue. Galford and McElhaney [31] obtained an elastic modulus of 66.7 kPa for human brain tissue in their vibration tests. This value was widely adopted as the linear elastic modulus in many early FE models with linear elastic material constitutive laws [118, 44, 51, 95]. Ueno et al. [114] used 80.0 kPa as the Young’s modulus of the brain in their FE model. In Chu et al.’s [17] and Huang et al.’s [45, 46] FE work, the Young’s modulus is 250 kPa, while it is as large as 675 kPa in Willinger et al.’s [122] work and 1000 kPa in Claessens et al.’s [18] work. Poisson’s ratios $\nu$ in the literature were all close to 0.50 since brain tissue is nearly incompressible [127, 58]. The Young’s modulus and Poisson’s ratio of brain tissue in the literature are summarized in Table 4.1.

<table>
<thead>
<tr>
<th>Authors</th>
<th>Young’s Modulus $E$ (kPa)</th>
<th>Poisson’s Ratio $\nu$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morrison III et al. [76]</td>
<td>10</td>
<td>0.4999</td>
</tr>
<tr>
<td>Ward &amp; Chan [118]</td>
<td>66.7</td>
<td>0.49-0.4999</td>
</tr>
<tr>
<td>Hosey &amp; Liu [44]</td>
<td>66.7</td>
<td>0.48</td>
</tr>
<tr>
<td>Khalil &amp; Viano [51]</td>
<td>66.7</td>
<td>0.45-0.499</td>
</tr>
<tr>
<td>Ruan et al. [95]</td>
<td>66.7</td>
<td>0.48</td>
</tr>
<tr>
<td>Ueno et al. [114]</td>
<td>80.0</td>
<td>0.49</td>
</tr>
<tr>
<td>Chu et al. [17]</td>
<td>250</td>
<td>0.49</td>
</tr>
<tr>
<td>Huang et al. [45, 46]</td>
<td>250</td>
<td>0.49</td>
</tr>
<tr>
<td>Willinger et al. [122]</td>
<td>675</td>
<td>0.48</td>
</tr>
<tr>
<td>Claessens et al. [18]</td>
<td>1000</td>
<td>0.48</td>
</tr>
</tbody>
</table>

Table 4.1: Material properties of linear elastic brain tissue in the literature.
4.4.2 Linear Viscoelastic Brain Material

Since brain tissue has both elastic and viscous properties, recent modeling work has used viscoelastic models to study brain injuries under impacts. Kelvin material, also called Voigt material, is the simplest constitutive representation for a linear viscoelastic solid [28]. Many analytical models used this two-element Kelvin material to study brain injuries under impacts [12, 27, 67]. More recent FE models used a standard linear solid (SLS) material to study brain responses under impacts [96, 127, 111]. Consisting of two springs and one damper, the three-parameter SLS model is discussed in detail in Appendix A. The shear relaxation function of the SLS model is,

\[ G(t) = G_\infty + (G_0 - G_\infty) e^{-\beta t}, \]  

(4.7)

where \( G \) indicates explicitly the shear modulus of the brain tissue in this study. \( G_0 \) is the short-term shear modulus, \( G_\infty \) is the long-term shear modulus, \( \beta \) is the decay constant, and \( t \) is the duration. The quantities of \( G_0, G_\infty, \) and \( \beta \) are algebraic combinations of the spring and damping coefficients of the SLS model. As listed in Table 4.2, various values of shear modulus for linear viscoelastic brain material have been used for FE models in the past.

In this study, both linear elastic and linear viscoelastic brain materials are used to calculate the strains in the brain tissue using the proposed scheme. The linear elastic model requires the selection of Young’s modulus and Poisson’s ratio, while three parameters, \( G_0, G_\infty, \) and \( \beta \), are needed for the linear viscoelastic SLS model. The material properties of the brain tissue are selected so that the calculated strains can best represent the severity of impacts using the proposed scheme of strain measure.
Table 4.2: Summary of linear viscoelastic brain material in the literature.

<table>
<thead>
<tr>
<th>Authors</th>
<th>Short-Term $G_0$ (kPa)</th>
<th>Long-Term $G_\infty$ (kPa)</th>
<th>Decay Constant $\beta$ (s$^{-1}$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zhang et al. [126]</td>
<td>10-12.5</td>
<td>2.0-2.5</td>
<td>80</td>
</tr>
<tr>
<td>Takhounts et al. [111]</td>
<td>10.3</td>
<td>5</td>
<td>100</td>
</tr>
<tr>
<td>Al-Bsharat et al. [1]</td>
<td>33-43</td>
<td>6-8</td>
<td>500</td>
</tr>
<tr>
<td>Zhang et al. [127]</td>
<td>34-41</td>
<td>6.4-7.8</td>
<td>700</td>
</tr>
<tr>
<td>Bandak &amp; Eppinger [6]</td>
<td>34</td>
<td>17</td>
<td>100</td>
</tr>
<tr>
<td>Gilchrist et al. [36]</td>
<td>41</td>
<td>7.6</td>
<td>700</td>
</tr>
<tr>
<td>Cheng et al. [15]</td>
<td>49</td>
<td>16.2</td>
<td>145</td>
</tr>
<tr>
<td>Willinger &amp; Baumgartner [121]</td>
<td>49</td>
<td>16.2</td>
<td>145</td>
</tr>
<tr>
<td>Kuijpers et al. [62]</td>
<td>338</td>
<td>169</td>
<td>50-10000</td>
</tr>
<tr>
<td>Ruan et al. [96]</td>
<td>528</td>
<td>168</td>
<td>35</td>
</tr>
</tbody>
</table>

4.5 Application to Low-Severity Tests

4.5.1 Parameter Selection

Firstly, the strain measure is applied to Hardy et al.’s [41] experiments under low-severity impact to choose the material properties of the brain tissue. By applying the head kinematics measured during the tests, the normal and shear strains in the brain tissue are calculated. The shear strains in the brain tissue are selected as the indicator of strain measurement using the proposed scheme since DAI most likely results from shear force and shear deformation [126, 58]. In order to have the strain calculation best represent the severity of impact quantified by measured head kinematics during the tests, the Young’s modulus $E$ of the brain tissue is chosen as 150 kPa, and the Poisson ratio $\nu$ is 0.49. They both fall within the range of reported data, as listed in Table 4.1. The shear modulus $G$ is therefore 50.3 kPa for the linear elastic brain tissue.
As listed in Table 4.2, the short-term shear modulus $G_0$ in the existing literature varies from 10 kPa to 528 kPa, while the long-term modulus $G_\infty$ ranges from 2 kPa to 169 kPa. The values of the decay constant $\beta$ also spread across a very large range, from 35 s$^{-1}$ to 10,000 s$^{-1}$. In this study, the parameters of the linear viscoelastic model are chosen as $G_0 = 50$ kPa, $G_\infty = 25$ kPa, and $\beta = 100$ s$^{-1}$. The selected values all fall within the range of reported data and are close to those used in Bandak and Eppinger’s [6] and Cheng et al’s [15] work. In this way, the short-term shear modulus ($G_0 = 50$ kPa) and the overall elastic material characterizations ($G = 50.3$ kPa) of the brain tissue are nearly identical. The selected material properties for brain tissue are summarized in Table 4.3.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Values</th>
<th>Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>Young’s modulus $E$</td>
<td>150</td>
<td>kPa</td>
</tr>
<tr>
<td>Poisson’s ratio $\nu$</td>
<td>0.49</td>
<td></td>
</tr>
<tr>
<td>Shear modulus $G$</td>
<td>50.3</td>
<td>kPa</td>
</tr>
<tr>
<td>Short-term shear modulus $G_0$</td>
<td>50</td>
<td>kPa</td>
</tr>
<tr>
<td>Long-term shear modulus $G_\infty$</td>
<td>25</td>
<td>kPa</td>
</tr>
<tr>
<td>Decay constant $\beta$</td>
<td>100</td>
<td>s$^{-1}$</td>
</tr>
</tbody>
</table>

Table 4.3: Selected material properties of linear elastic and linear viscoelastic brain tissues.

### 4.5.2 Application of Sagittal Plane Model

The measured head accelerations in Hardy et al’s [41] low-severity impact tests are applied to the sagittal plane model as inputs for the strain calculation. Figure 4.4 plots the shear strains in all six tests with both linear elastic and linear viscoelastic brain materials. The results using these two different brain materials are very close, but the linear viscoelastic model gives slightly higher shear strains.
Figure 4.4: Shear strains under low-severity impacts with both linear elastic and linear viscoelastic brain materials using the sagittal plane model.

Table 4.4 lists the configurations of all six tests and the maximum shear strains calculated using the sagittal plane model. The relative magnitudes of maximum shear strains are consistent with the HIC15 values and the maximum resultant linear and angular accelerations. Test C755-T5 has the lowest shear strains of 0.035 and 0.047 with the linear elastic and linear viscoelastic models, respectively. It corresponds to the lowest HIC15 of 5.2, the smallest maximum linear acceleration of 12.1 g, and the smallest angular acceleration of 803.9 rad/s² among all six tests. For test C383-T4, the highest maximum shear strains of 0.136 and 0.153 correspond to the highest HIC15 and maximum linear and angular accelerations. Tests C755-T2 and C755-T3 are similar in impact level, which is slightly more
severe than test C755-T5. The maximum shear strains for these two tests are very close, and they are higher than those in test C755-T5. Likewise, the maximum shear strains in tests C383-T1 and C383-T3 also have similar magnitudes.

<table>
<thead>
<tr>
<th>Test No.</th>
<th>HIC15</th>
<th>Maximum Resultant Acceleration</th>
<th>Maximum Shear Strain</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Linear (g)</td>
<td>Angular (rad/s²)</td>
</tr>
<tr>
<td>C755-T2</td>
<td>16.9</td>
<td>21.8</td>
<td>1753.2</td>
</tr>
<tr>
<td>C755-T3</td>
<td>21.3</td>
<td>24.4</td>
<td>1948.7</td>
</tr>
<tr>
<td>C755-T5</td>
<td>5.2</td>
<td>12.1</td>
<td>803.9</td>
</tr>
<tr>
<td>C383-T1</td>
<td>46.6</td>
<td>62.2</td>
<td>2745.7</td>
</tr>
<tr>
<td>C383-T3</td>
<td>67.6</td>
<td>62.4</td>
<td>3033.3</td>
</tr>
<tr>
<td>C383-T4</td>
<td>163.7</td>
<td>107.7</td>
<td>22393.9</td>
</tr>
</tbody>
</table>

Table 4.4: Test configuration and results of strain measurement in the sagittal plane.

In general, the maximum shear strains in Hardy et al’s [41] six tests fall within the range of 0.04-0.15, which is reasonable compared with strain of 0.02-0.05 observed by Bayly et al. [7] using human volunteers under mild occipital deceleration impacts and with the published injury threshold of strain of 0.15-0.20. The maximum shear strain in each individual test closely matches the severity of impact, which is characterized by the HIC15 and maximum resultant linear and angular accelerations.

### 4.5.3 Application of Coronal Plane Model

Similarly, the shear strains are calculated using the same six tests in the coronal plane model. With both linear elastic and viscoelastic brain tissue, Figure 4.5 plots the shear strains in all six tests using the head kinematics in the coronal plane as inputs. It shows the time history of the shear strains during impact. Comparing Figures 4.4 and 4.5, the shear strains in the coronal plane are lower than the shear strains in the sagittal plane under
the same impacts. The results are reasonable because the sagittal plane head kinematics dominate in all six tests, and higher shear strains are expected with larger head kinematics. In Figure 4.5, the results using the two different brain materials are similar, but the linear viscoelastic model gives slightly higher shear strains. The use of linear viscoelastic brain material shows no significant improvement for brain injury prediction in these tests.

Table 4.5 lists the configurations of all six tests and the maximum shear strains in the coronal plane calculated using the coronal plane model. The 3D head kinematics are included for the calculation of the HIC15 values and the maximum resultant linear and angular accelerations, but the coronal plane model only accepts the head kinematics in the coronal plane as inputs. Therefore, larger head kinematics, referring explicitly to the HIC15.
and maximum resultant linear and angular accelerations in Table 4.5, do not necessarily suggest larger head kinematics in the coronal plane. Instead, Table 4.6 summaries the peak accelerations and peak velocities in the coronal plane, and test C383-T3 has the largest head kinematics considering both the accelerations and velocities.

<table>
<thead>
<tr>
<th>Test No.</th>
<th>HIC15</th>
<th>Maximum Resultant Acceleration</th>
<th>Maximum Shear Strain</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Linear (g)</td>
<td>Angular (rad/s^2)</td>
</tr>
<tr>
<td>C755-T2</td>
<td>16.9</td>
<td>21.8</td>
<td>1753.2</td>
</tr>
<tr>
<td>C755-T3</td>
<td>21.3</td>
<td>24.4</td>
<td>1948.7</td>
</tr>
<tr>
<td>C755-T5</td>
<td>5.2</td>
<td>12.1</td>
<td>803.9</td>
</tr>
<tr>
<td>C383-T1</td>
<td>46.6</td>
<td>62.2</td>
<td>2745.7</td>
</tr>
<tr>
<td>C383-T3</td>
<td>67.6</td>
<td>62.4</td>
<td>3033.3</td>
</tr>
<tr>
<td>C383-T4</td>
<td>163.7</td>
<td>107.7</td>
<td>22393.9</td>
</tr>
</tbody>
</table>

Table 4.5: Test configuration and results of strain measurement in the coronal plane.

<table>
<thead>
<tr>
<th>Test No.</th>
<th>Peak Acceleration</th>
<th>Peak Velocity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Y (m/s^2)</td>
<td>Z (m/s^2)</td>
</tr>
<tr>
<td>C755-T2</td>
<td>64.4</td>
<td>140.4</td>
</tr>
<tr>
<td>C755-T3</td>
<td>72.8</td>
<td>145.2</td>
</tr>
<tr>
<td>C755-T5</td>
<td>29.5</td>
<td>34.6</td>
</tr>
<tr>
<td>C383-T1</td>
<td>62.5</td>
<td>406.5</td>
</tr>
<tr>
<td>C383-T3</td>
<td>153.4</td>
<td>314.2</td>
</tr>
<tr>
<td>C383-T4</td>
<td>193.7</td>
<td>329.8</td>
</tr>
</tbody>
</table>

Table 4.6: Measured head kinematics in the coronal plane for all six tests.

Test C383-T3 has the highest maximum shear strains of 0.048 and 0.055 with the linear elastic and linear viscoelastic models, respectively, as listed in Table 4.5. This is consistent with the largest head kinematics. For test C755-T5, the lowest shear strains of 0.012 and
0.016 corresponds to the smallest head kinematics. The shear strains are all far below the injury threshold value of strain, 0.15-0.20. Tests C755-T2 and C755-T3 have similar head kinematics that are slightly higher than that in test C755-T5. Therefore, the maximum shear strains for these two tests are very close.

The results from the sagittal and coronal plane models suggest that this simple measure of strain is capable of representing the severity of impact, which is characterized by the measured head kinematics during impact. Therefore, the maximum shear strain can be used as the injury indicator for DAI prediction.

4.6 Application to More Severe Tests

4.6.1 Background

The strain measure predicts closely the severity of impact in Hardy et al’s [41] tests under low-severity impacts. To further evaluate its capabilities of DAI prediction, the proposed strain measure in the planar brain injury models is applied to more severe, real crash tests in the New Car Assessment Program (NCAP) conducted by NHTSA. The sagittal plane model is used for frontal crash tests since the impacts are primarily in the sagittal plane, while side impact tests are used to evaluate the coronal plane model since the dummy head responses in the coronal plane dominate.

The crash test data are obtained electronically from the NHTSA Vehicle Crash Test Database [79], which includes records of measured engineering data during various crash tests. For head injury study, a nine accelerometer package (NAP) is instrumented inside the dummy headform to measure nine linear accelerations of the dummy head during impacts. The linear accelerations are used to calculate the HIC value to predict skull fractures. The
measured linear accelerations can also be transformed to calculate three orthogonal angular accelerations that fully describe the kinematics of the dummy head together with the three measured linear accelerations at the CG. The transformation is described in detail in Appendix B. The head kinematics in each test are applied to mathematical models to study different head and brain injuries.

4.6.2 Application to Frontal Crash Tests

Configuration of Frontal Crash Tests

Having dominant head kinematics primarily in the sagittal plane, frontal crash tests are selected to study brain injuries using the sagittal plane model. Table 4.7 shows the configurations of a total of 43 frontal crash tests in NCAP. All of these frontal crash tests have an impact speed of about 56.3 kph (35 mph). The first four digits in the first column of Table 4.7 are the test number assigned by NHTSA. The two digits following the dashed line indicate the test dummy positions, where “01” represents the driver’s seating position and “02” represents the right front passenger’s seating position. Recent crash tests are chosen such that their HIC15 values spread across a large range, representing impacts of various severities. Note that one crash test here explicitly means one set of data collected with one test dummy. More than one set of data from one frontal crash test are possibly used, but are referred to with different numbers. For instance, tests 4500-01 and 4500-02 have different test data from two dummies, but they were conducted during a single crash.

The head kinematics collected with Hybrid III dummies in NHTSA’s NCAP crash tests are listed in Tables 4.8 and 4.9. The peak values of the accelerations and velocities in all three directions are listed. Nine linear accelerations are measured directly using a 3-2-2-2 NAP installed within dummy head, and linear velocities are obtained by integrating the linear accelerations. The angular accelerations are obtained by transforming the nine
<table>
<thead>
<tr>
<th>NHTSA Test No.</th>
<th>HIC15</th>
<th>Vehicle Description</th>
<th>Impact Speed (kph)</th>
<th>Test Configuration</th>
</tr>
</thead>
<tbody>
<tr>
<td>3915-01</td>
<td>379.06</td>
<td>2002 Toyota Tundra</td>
<td>56.16</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
<td>3915-02</td>
<td>534.23</td>
<td>2002 Toyota Tundra</td>
<td>56.16</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
<td>3952-01</td>
<td>540.79</td>
<td>2002 Buick Rendezvous</td>
<td>56.60</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
<td>3952-02</td>
<td>820.82</td>
<td>2002 Buick Rendezvous</td>
<td>56.60</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
<td>4080-01</td>
<td>336.29</td>
<td>2002 Ford Focus 2 Door</td>
<td>56.00</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
<td>4080-02</td>
<td>214.75</td>
<td>2002 Ford Focus 2 Door</td>
<td>56.00</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
<td>4081-02</td>
<td>287.26</td>
<td>2002 Jeep Liberty</td>
<td>56.30</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
<td>4198-01</td>
<td>220.37</td>
<td>2002 Saturn VUE 4 Door</td>
<td>56.30</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
<td>4198-02</td>
<td>524.98</td>
<td>2002 Saturn VUE 4 Door</td>
<td>56.30</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
<td>4205-01</td>
<td>337.31</td>
<td>2002 Ford Thunderbird</td>
<td>56.20</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
<td>4205-02</td>
<td>187.55</td>
<td>2002 Ford Thunderbird</td>
<td>56.20</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
<td>4237-01</td>
<td>414.85</td>
<td>2002 Nissan Frontier</td>
<td>56.16</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
<td>4237-02</td>
<td>329.51</td>
<td>2002 Nissan Frontier</td>
<td>56.16</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
<td>4240-01</td>
<td>414.35</td>
<td>2002 Dodge Ram1500</td>
<td>56.49</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
<td>4240-02</td>
<td>343.88</td>
<td>2002 Dodge Ram1500</td>
<td>56.49</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
<td>4242-01</td>
<td>178.37</td>
<td>2002 Honda Odyssey</td>
<td>56.49</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
<td>4242-02</td>
<td>366.52</td>
<td>2002 Honda Odyssey</td>
<td>56.49</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
<td>4252-01</td>
<td>654.04</td>
<td>2002 Dodge Dakota</td>
<td>56.16</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
<td>4252-02</td>
<td>256.21</td>
<td>2002 Dodge Dakota</td>
<td>56.16</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
<td>4273-01</td>
<td>193.27</td>
<td>2002 Mini Cooper</td>
<td>56.16</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
<td>4273-02</td>
<td>384.51</td>
<td>2002 Mini Cooper</td>
<td>56.16</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
<td>4303-01</td>
<td>156.44</td>
<td>2003 Honda Pilot</td>
<td>55.91</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
<td>4303-02</td>
<td>341.65</td>
<td>2003 Honda Pilot</td>
<td>55.91</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
<td>4500-01</td>
<td>631.51</td>
<td>2003 Isuzu Rodeo MPV</td>
<td>56.65</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
<td>4500-02</td>
<td>365.45</td>
<td>2003 Isuzu Rodeo MPV</td>
<td>56.65</td>
<td>Vehicle into barrier</td>
</tr>
<tr>
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<td>544.04</td>
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Table 4.7: Configuration of NCAP frontal barrier crash tests.
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Table 4.8: Peak head angular velocities and accelerations in NCAP frontal crash tests.
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<th>Peak Linear Velocity</th>
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<td>148.91</td>
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Table 4.9: Peak head linear velocities and accelerations in NCAP frontal crash tests.
linear accelerations. Similarly, the angular velocities are obtained by integrating the angular accelerations. In an FE model, Kleiven [56] concluded that the peak change in angular velocity had the best correlation with the principal strain in the brain tissue under a purely rotational impulse. This suggests that peak angular velocity is an important metric for brain injury prediction. The frontal crash tests are expected to have dominant head kinematics in the sagittal plane due to the nature of the impacts. Comparing the experimental data in Tables 4.8 and 4.9, the angular accelerations and velocities about the $Y$ axis are the largest for most of the tests. The linear accelerations and velocities along the $X$ and $Z$ axes usually have relatively larger magnitudes than those along the $Y$ axis. The dominant head kinematics in the sagittal plane ensure that the planar model can capture the key characteristics of brain motion during impact. For those tests that have relatively large out-of-sagittal-plane head kinematics, the results using the planar brain injury model may not accurately represent the severity of the impacts.

**Results of Strain Measurement**

The head kinematics collected with anthropomorphic test dummies (ATDs) are used as inputs for the sagittal plane model. Including both peak acceleration and velocity, the head kinematics are compared comprehensively to evaluate the severity of impact for different tests. The frontal crash tests are also reconstructed with the SIMon FE head model, which has detailed anatomical structures and was validated against experimental data collected in tests on PMHS and animals. SIMon predicts different brain injuries using three types of injury metrics: cumulative strain damage measure (CSDM), dilatational damage measure (DDM), and relative motion damage measure (RMDM). These three metrics correlate to DAI, cerebral contusion, and ASDH, respectively. Takhounts et al. [111] selected 0.15 strain level as a critical value causing brain damage in SIMon. Twenty-five percent of the
whole brain volume experiencing strain over 0.15 was used as a preset “survivability” for CSDM. The strain measurement in the planar model is compared with the CSDM and HIC values to evaluate their individual advantages and disadvantages for brain injury prediction.

Sorted by the maximum shear strain, Figure 4.6 compares the maximum shear strain from the sagittal plane model with the HIC15 values using linear elastic brain tissue. Except for test 3952-02, all of the HIC15 values are lower than the injury threshold of 700. For the simple model, relatively high shear strains are expected for tests having high HIC15 values because linear kinematics are considered to be injurious to the brain in this study. However, the maximum shear strain is only 0.107 for test 4899-01 having a HIC15 of 544 because this test has relatively small angular head kinematics. Contrarily, for test 5092-02 having a HIC15 of only 144, the maximum shear strain is as high as 0.140 due to the relatively large angular head kinematics. Similar to test 5092-02, test 5245-01 also has a relatively high shear strain but a very low HIC15 value. Moreover, test 3915-01 has the highest maximum shear strain among all the tests, but the HIC15 value of 379 is not very high. In general, the maximum shear strain from the simple model is always significant even for tests having low HIC15 values because of the combined linear and angular head kinematics, which are both treated as injurious inputs.

Also sorted by the maximum shear strain, Figure 4.7 compares the CSDM (0.15) values from SIMon with the maximum shear strain from the sagittal plane model using linear elastic brain tissue. Having a HIC15 of 287, test 4081-02 is the only one that exceeds the preset CSDM threshold value of 25% in SIMon. However, this test does not have a very high maximum shear strain (only 0.114) because the out-of-sagittal-plane kinematics are significant and are not used as inputs for the planar model. Similar results are obtained for test 4500-01. This suggests that the planar model is limited to capture in-plane-only
characteristics of brain response. Since test 4080-01 has relatively small angular head kinematics, SIMon gives a small CSDM value of 2.3%, while the planar model produces a relatively high shear strain of 0.182 due to the large linear head kinematics. For all of the tests having small angular head kinematics, SIMon predicts very low CDSM values, while the sagittal plane model generally does not follow the same trend because the linear head kinematics are also included as injurious inputs.

Using linear viscoelastic brain tissue, Figure 4.8 compares the HIC15 values with the maximum shear strain, and Figure 4.9 compares the CSDM values with the maximum shear strain from the sagittal plane model. Both figures are sorted by the maximum shear strains. Since the overall shear properties are identical, the maximum shear strains are close using the linear elastic and linear viscoelastic brain materials. The overall trends in Figures 4.6 and 4.8 are similar. Similar trends are also obtained in Figures 4.7 and 4.9. No significant improvement was found using the linear viscoelastic brain tissue in spite of its capability of including the effect of strain rates on the brain response.

4.6.3 Application to Side Impact Tests

Configuration of Side Impact Tests

NHTSA conducts different side impact tests to provide safety information for various purposes. Tests are selected in three major categories: vehicle-into-vehicle, barrier-into-vehicle, vehicle-into-pole. In the NCAP side impact tests, a moving deformable barrier impacts a stationary vehicle from the left side at a speed of about 60 kph. In vehicle-into-pole tests, the left side of a vehicle is crashed into a rigid pole at a lower impact speed of about 30 kph to assess the crashworthiness of vehicle structures. More realistic vehicle-into-vehicle crash tests are also conducted. The responses of the dummies are measured and evaluated for all of the side impact tests. The crash test data are also obtained electronically.
Figure 4.6: Comparison of HIC15 and the maximum shear strain from the sagittal plane model with linear elastic brain tissue.

Figure 4.7: Comparison of CSDM (0.15) from SIMon and the maximum shear strain from the sagittal plane model with linear elastic brain tissue.
Figure 4.8: Comparison of HIC15 and the maximum shear strain from the sagittal plane model with linear viscoelastic brain tissue.

Figure 4.9: Comparison of CSDM (0.15) from SIMon and the maximum shear strain from the sagittal model with linear viscoelastic brain tissue.
from the NHTSA Vehicle Crash Test Database [79]. Although nine linear accelerations of the dummy headforms are measured, head injury is not currently included in the safety rating system under side impact. No head or brain injury criteria are available, but the HIC values are reported as a reference.

Table 4.10 shows the configurations of a total of 42 crash tests. The first four digits in the first column of Table 4.10 are the test number assigned by NHTSA. The two digits following the dashed line indicate the test dummy location, where “01” represents driver, and “04” represents rear left passenger. Recent crash tests are chosen such that their HIC15 values spread across a large range, representing impacts of various severities. The impact speeds of these tests are about 30-60 kph. Note that one crash test here explicitly means one set of data collected with one dummy. More than one set of data are possibly used in one side impact test, but are referred to with different numbers. For instance, tests 3799-01 and 3799-04 have crash data from two different dummies, but they were conducted during a single crash and assigned different numbers.

The headform kinematics collected with ATDs, mostly ES-2 dummies, in NHTSA’s crash tests are listed in Tables 4.11 and 4.12. The peak values of the accelerations and velocities in all three directions are listed. Nine linear accelerations are measured directly using a 3-2-2-2 NAP installed within the dummy heads, and linear velocities are obtained by integrating the linear accelerations. Three angular accelerations are obtained by transforming the nine linear accelerations. Similarly, angular velocities are also obtained by integrating the angular accelerations. The side impact tests are expected to have dominant head kinematics in the coronal plane due to the nature of the impacts. Comparing the experimental data in Tables 4.11 and 4.12, the angular accelerations and velocities about the X axis are the largest for most of the tests. The linear accelerations and velocities along the
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<th>Impact Speed (kph)</th>
<th>Test Configuration</th>
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<td>62.00</td>
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<td>61.60</td>
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Table 4.10: Configuration of NHTSA’s side impact tests.
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Table 4.11: Peak head angular velocities and accelerations in side impact tests.
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Table 4.12: Peak head linear velocities and accelerations in side impact tests.

139
and $Z$ axes usually have relatively larger magnitudes than those along the $X$ axis. The dominant head kinematics in the coronal plane ensure that the planar model can capture the key characteristics of brain motion during impact.

**Results of Strain Measurement**

Similar to the sagittal plane model, the head kinematics collected with the ATDs are the inputs for the coronal plane model. The 42 crash tests are also reconstructed using the SIMon FE head model, which predicts DAI using the injury metric of CSDM. The strain measure in the simple model is compared with the CSDM and HIC15 values to evaluate their individual capabilities for brain injury prediction.

Sorted by the maximum shear strain, Figure 4.10 compares the HIC15 values with the maximum shear strain from the coronal plane model using linear elastic brain tissue. Except for test 3819-01, all of the HIC15 values are lower than the injury threshold of 700. The overall trend of the maximum shear strain is reasonably close to the trend of HIC15 values. Tests having higher HIC15 values generally yield higher shear strains using the coronal plane model. However, for some of the tests having relatively low HIC15 values, the coronal plane model gives relatively high shear strains since the angular head kinematics are also included. Test 3800-04 has a high maximum shear strain of 0.303 but a relatively low HIC15 of 370 because of the large angular head kinematics. Similar results are also obtained in tests 4094-04, 3799-01, and 4456-01. In general, the maximum shear strain from the coronal plane model is always significant even for tests having very low HIC15 values because of the combined linear and angular head kinematics, which are both treated as injurious inputs.

Also sorted by the maximum shear strain, Figure 4.11 compares the CSDM (0.15) values from SIMon with the maximum shear strain from the coronal plane model using
linear elastic brain tissue. There are six tests exceeding the preset CSDM threshold value of 25% in SIMon, but their HIC15 values are not very high and mostly below the injury threshold of 700. Among all of the 42 tests, test 3819-01, having the highest HIC15 of 712, yields the highest maximum shear strain of 0.306 and the third highest CSDM of 51%.

The trend of the shear strains from the planar model generally follows the trend of CSDM values. Tests having higher CSDM values tend to accumulate to the right portion of the figure, where the maximum shear strains are higher. However, test 4313-01, having a very low CSDM value of only 2.5%, yields a relatively high shear strain of 0.248. This is due to the small angular head kinematics in test 4313-01. Similar results are also obtained in tests 4471-01, 4480-01, and 3802-01. For all of the tests having small angular head kinematics, SIMon predicts very low CDSM values, while the planar model generally does not follow the same trend because the linear head kinematics are also included as injurious inputs.

Using linear viscoelastic brain tissue, Figure 4.12 and Figure 4.13 compare the HIC15 and CSDM values with the maximum shear strains, respectively. Both figures are sorted by the maximum shear strains. Since the overall shear properties are identical, the maximum shear strains are close using the linear elastic and viscoelastic brain materials. The overall trends in Figures 4.10 and 4.12 are similar. Similar trends are also obtained in Figures 4.11 and 4.13. No significant improvement was found using the linear viscoelastic brain tissue.

Using sagittal and coronal plane models, the trends of maximum shear strains generally match those of the HIC15 and CSDM values, especially when the in-plane head kinematics are dominant. HIC15 predicts head injury based only on linear accelerations of the head during impact, while the brain injury prediction in SIMon is dominated by the angular kinematics of the head. The maximum shear strain from the planar model varies more.
Figure 4.10: Comparison of HIC15 and the maximum shear strain from the coronal plane model with linear elastic brain tissue.

Figure 4.11: Comparison of CSDM (0.15) from SIMon and the maximum shear strain from the coronal plane model with linear elastic brain tissue.
Figure 4.12: Comparison of HIC15 and the maximum shear strain from the coronal model with linear viscoelastic brain tissue.

Figure 4.13: Comparison of CSDM (0.15) from SIMon and the maximum shear strain from the coronal model with linear viscoelastic brain tissue.
closely with the combination of linear and angular head kinematics. It is consistent with the structural coupling between the rotation and translation of the brain.

4.7 Discussion

In this chapter, a simple measure of strain was proposed using the planar brain injury models for DAI prediction. The brain material properties were selected such that the calculated strains closely matched the severity of impacts in Hardy et al’s [41] tests. The planar models with the strain measure were further applied to more severe frontal and side impact tests in NHTSA’s database. The results show that this simple scheme for strain measurement provides reasonable accuracy for brain injury prediction. It also shows that the coupling between brain rotation and translation has significant effects on brain response. Although recent studies [7, 54] provided strong evidence of the coupling, no direct experiments under pure linear or pure angular accelerations have been conducted to study their distinctive contributions to brain motion. In addition, the patterns of relative brain motion inside the skull are not yet fully understood. Further studies will help better understand the coupling between the translation and rotation of the brain.

Although the HIC criterion has been widely used and accepted as a government standard for head injury protection, it is evaluated only based on linear accelerations of the head, and possible injuries caused by angular accelerations cannot be predicted. In contrast, the SIMon prediction is dominated by angular accelerations of the head. With very high linear accelerations as inputs, SIMon only yields very low probability of brain injury in terms of all three proposed injury metrics. The present work is expected to fill the gap between existing simple models and more complex FE models for better brain injury prediction under more severe impacts. The adoption of both types of accelerations as inputs
helps unveil important features of brain injury mechanisms. Compared with several hours in FE models, only several seconds are needed to finish the computation on the simple model for a similar simulation. This reduction in execution time without sacrificing the capability of injury prediction is one of the advantages that would facilitate widespread use of this model.

Compared with the results in frontal crash tests using the sagittal plane model, the maximum shear strains are generally higher for the coronal plane model under side impacts of similar severity. The majority of the frontal and side impact tests in this study have HIC15 values lower than the injury threshold value of 700. Using linear elastic brain tissue, 11 out of 42 tests have maximum shear strains higher than 0.20 under side impacts, while only 3 out of 43 tests do so under frontal impacts. For the same tests, similar results are also obtained using the SIMon FE model. There are 6 tests with CSDM (0.15) values higher than the preset critical 25% under side impacts, while only one test does under frontal impacts. Using the same SIMon model, Takhounts et al’s [111] concluded that side impact was potentially more injurious to the human brain than frontal impact due to more severe angular kinematics. The results from the planar models are consistent with the findings using the SIMon FE model.

The proposed measure of strain on the planar brain injury models for DAI prediction can only capture the in-plane characteristics of brain motion. However, all existing experiments generate 3D head kinematics during impacts. For most of the experiments under frontal and rear impacts, the head kinematics in the sagittal plane are dominant, while the head kinematics in the coronal plane are dominant under side impacts. The planar models are adequate when the out-of-plane head kinematics are insignificant. For those tests that have relatively large out-of-plane head kinematics, the results using planar models may not
accurately represent the severity of impacts. A 3D model is necessary to accept 3D head accelerations as inputs for better injury prediction.

4.8 Conclusions

A simple measure of strain based on the planar brain injury models was presented for DAI prediction. The maximum shear strain was selected as the indicator of brain injury. This simple model for strain measurement was applied to low-severity impact tests in Hardy et al’s [41] work and more severe frontal and side impact tests in NHTSA’s database. The maximum shear strains were calculated for 43 frontal crash tests and 42 side impact tests. These tests were also reconstructed in the SIMon FE head model for purposes of comparison. The results show that the simple measure of strain has the capabilities of predicting brain injuries under impacts. The maximum shear strain from the planar models can be used as a critical element for brain injury prediction. In general, HIC15 predicts head injury based only on linear accelerations of the head during impact, while the brain injury prediction in SIMon is dominated by angular accelerations of the head. The maximum shear strain in the planar models varies more closely with the combination of linear and angular head kinematics. It is consistent with the structural coupling between the relative rotation and translation of the brain.
CHAPTER 5

THREE-DIMENSIONAL MODEL AND ITS APPLICATION

5.1 Introduction

Current regulations contain safety standards for motor vehicle crashes under frontal and side impacts, which account for 35% and 16% of all collisions, respectively, according to averaged statistical data from NHTSA [98]. However, oblique impacts from the front or rear directions account for 39% of all motor vehicle accidents. In addition, real world crashes always produce 3D occupant head kinematics regardless of whether during frontal, rear, or side impacts. When head kinematics in neither the sagittal nor coronal planes are dominant, the two planar models developed earlier in this work are not enough to capture the key characteristics of brain responses under impact. Therefore, it is necessary to develop a 3D model to accept 3D head kinematics as inputs for brain injury study.

This chapter combines the planar models into a 3D version so that 3D head kinematics can be accepted to have a more comprehensive study. Unfortunately, the development of a model in the horizontal plane cannot be currently implemented due to the lack of experimental data. Therefore, the 3D model developed in this chapter is only based on the models in the sagittal and coronal planes. The inputs are the full 3D head kinematics except the rotation about the Z axis. Once available, the horizontal plane model can be
easily integrated into the current 3D model. The simple 3D model is applied to reconstruct ten car-pedestrian accidents to study different brain injuries.

5.2 Background

Among motor vehicle crash deaths, pedestrian deaths comprise the second largest category after vehicle occupants. According to statistical data reported by The Insurance Institute for Highway Safety (IIHS) [48], a total of 4,881 pedestrian deaths occurred in 2005, which accounts for 11 percent of fatalities. Although pedestrian deaths have declined from 17 percent of all motor vehicle crash deaths in 1975 to 11 percent in 2005, the financial and emotional burdens from injuries associated with pedestrian crashes are still substantial. In many other countries, pedestrian deaths even account for a higher percentage of total motor vehicle crash deaths [99]. These data present a critical need to further study car-pedestrian interaction, especially head injuries during head impact with motor vehicles.

For better pedestrian protection, research was conducted based on pedestrian crashes documented in the Pedestrian Crash Data Study (PCDS) initiated by NHTSA. Detailed information from a total of 550 pedestrian accidents was collected in six major cities in the US between 1994 and 1998 [16]. This database provides all detailed vehicle and crash parameters required for case reconstruction. These accidents were mostly frontal car-pedestrian crashes. Among all types of injuries in car-pedestrian crashes, head injury is the most life-threatening and is one of the leading causes of pedestrian deaths. Therefore, more effort is needed to study the mechanisms of head injury in car-pedestrian crashes for better pedestrian protection.

Current regulations need to be improved for better head protection in car-pedestrian crashes. The HIC criterion was initially proposed for the prediction of skull fracture, but it
can be used as a critical element indicating the severity of impact. Although the correlation of HIC values with brain injuries has been studied by some researchers [93, 128], the exclusion of angular head accelerations significantly affects its overall performance. Having three proposed metrics for brain injury prediction, the SIMon FE head model developed by NHTSA [111] is an important tool for brain injury study in car-pedestrian crashes. However, it underestimates the role of linear accelerations. In contrast, the simple 3D model developed in this chapter takes both linear and angular head accelerations as injurious inputs. While each model has its own advantages and drawbacks, this chapter compares the results of car-pedestrian case reconstructions using different models.

### 5.3 Model Development

A simple measure of strain has been proposed and validated for both planar models for brain injury prediction. The combination of these two planar models requires a combined scheme of strain measurement. For a typical element, the 3D strain configuration can be expressed by a symmetrical strain tensor \( \epsilon \),

\[
\epsilon = \begin{bmatrix}
\epsilon_x & \epsilon_{xy} & \epsilon_{xz} \\
\epsilon_{xy} & \epsilon_y & \epsilon_{yz} \\
\epsilon_{xz} & \epsilon_{yz} & \epsilon_z
\end{bmatrix}.
\]  

The strain configuration is graphically shown in Figure 5.1. Note that only the strains on three surfaces are shown. The symmetry of the 3D strain tensor in Equation 5.1 defines the strains on the other three surfaces of the element in Figure 5.1.

To define a 3D strain configuration, the spherical brain is divided into hemispheres by a horizontal cross section for the sagittal plane model. The strain profile on the base of the upper hemispherical brain is used for the strain calculation. With a linear elastic brain material, the normal and shear strains on the base correspond to \( \epsilon_z \) and \( 2\epsilon_{zz} \), respectively, comparing with the element having a full 3D strain configuration. Likewise, the brain is
Figure 5.1: An element with 3D strain configuration.

divided into left and right hemispheres for the coronal plane model. The normal and shear strains on the base of the right hemispherical brain correspond to $\epsilon_y$ and $2\epsilon_{yz}$, respectively. When a model in the horizontal plane is available, the brain can be divided into back and front hemispheres. In a similar manner, the normal and shear strains on the base of the back hemisphere can be defined as the remaining two components and correspond to $\epsilon_x$ and $2\epsilon_{xy}$ in the 3D strain configuration. The three normal strains are orthogonal to each other, as are the three shear strains. Therefore, a full 3D strain configuration is constructed. For the present work, however, the two strain components, $\epsilon_x$ and $\epsilon_{xy}$, from the horizontal plane model are both assumed to be zero. The strain tensor becomes,

$$
\epsilon = \begin{bmatrix}
0 & 0 & \epsilon_{xz} \\
0 & \epsilon_y & \epsilon_{yz} \\
\epsilon_{xz} & \epsilon_{yz} & \epsilon_z
\end{bmatrix}.
$$

(5.2)

Figure 5.2 shows the graphical presentation of the strain tensor in Equation 5.2. Note that the strains on all of the six surfaces are shown. This configuration corresponds to the strain
status at the center of the spherical brain, which is the intersection of the bases of the three hemispherical brain models.

Figure 5.2: The combined strain configuration using two models in the sagittal and coronal planes.

5.4 Strain Transformation

Based on the schemes for the planar models, a measure of strain is proposed for strain calculation in the 3D configuration with 3D head kinematics. The strain profile calculated in the 3D strain configuration can be transformed to find three principal strains in three directions, and all shear strains disappear on the principal planes. Using the principal strains, the maximum shear strain on the element can be easily obtained. The principal strains are the three roots of the cubic equation,

\[ \epsilon_p^3 - J_1 \epsilon_p^2 + J_2 \epsilon_p - J_3 = 0, \]  

(5.3)
where $J_1$, $J_2$, and $J_3$ are three invariants of the strain tensor, usually referred to as strain invariants, which are given by,

$$
J_1 = \epsilon_x + \epsilon_y + \epsilon_z,
$$

$$
J_2 = \epsilon_x \epsilon_y + \epsilon_x \epsilon_z + \epsilon_y \epsilon_z - \left( \epsilon_{xy}^2 + \epsilon_{yz}^2 + \epsilon_{xz}^2 \right),
$$

$$
J_3 = \begin{vmatrix}
\epsilon_x & \epsilon_{xy} & \epsilon_{xz} \\
\epsilon_{xy} & \epsilon_y & \epsilon_{yz} \\
\epsilon_{xz} & \epsilon_{yz} & \epsilon_z
\end{vmatrix}.
$$

(5.4)

Solving Equations 5.3 and 5.4 gives three principal strains $\epsilon_1$, $\epsilon_2$, and $\epsilon_3$, where $\epsilon_1$ has the maximum value and $\epsilon_3$ has the minimum value. The maximum shear strain on the element is defined by,

$$
\epsilon_{max} = \frac{1}{2}(\epsilon_1 - \epsilon_3).
$$

(5.5)

5.5 Application to Low-Severity Impacts

The combination of the two planar models into a 3D model is applied to Hardy et al’s [41] low-severity impact tests to evaluate its capability of predicting brain injuries. The evaluation is performed by comparing the strain results with the HIC values and head accelerations. The principal strains at the center of the spherical brain are calculated with linear elastic brain material, whose Young’s modulus $E$ is 150 kPa and Poisson ratio $\nu$ is 0.49. Note that linear viscoelastic brain material is not used here since it does not outperform linear elastic material for the simple model, as discussed in Chapter 4. The maximum shear strain is used as the indicator of brain injury.

The results for all six low-severity impact tests are summarized in Table 5.1, which also includes the HIC values and maximum resultant linear and angular head accelerations. The calculated maximum shear strains from the planar and 3D models are different due to the different injury metrics. However, the results from the 3D model have the same trend as
those obtained in the sagittal plane model for all six tests. This is reasonable since all six tests are under either frontal or occipital impacts and the head kinematics in the sagittal plane dominate throughout the impacts. Therefore, the sagittal plane model is adequate to capture the key characteristics of brain responses under these impacts.

<table>
<thead>
<tr>
<th>Test No.</th>
<th>HIC15</th>
<th>Max Resultant Acceleration</th>
<th>Max Shear Strain</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Linear (g)</td>
<td>Angular (rad/s²)</td>
</tr>
<tr>
<td>C755-T2</td>
<td>16.9</td>
<td>21.8</td>
<td>1753.2</td>
</tr>
<tr>
<td>C755-T3</td>
<td>21.3</td>
<td>24.4</td>
<td>1948.7</td>
</tr>
<tr>
<td>C755-T5</td>
<td>5.2</td>
<td>12.1</td>
<td>803.9</td>
</tr>
<tr>
<td>C383-T1</td>
<td>46.6</td>
<td>62.2</td>
<td>2745.7</td>
</tr>
<tr>
<td>C383-T3</td>
<td>67.6</td>
<td>62.4</td>
<td>3033.3</td>
</tr>
<tr>
<td>C383-T4</td>
<td>163.7</td>
<td>107.7</td>
<td>22393.9</td>
</tr>
</tbody>
</table>

Table 5.1: Test configuration and results of strain measurement for all six tests in Hardy et al’s [41] study with linear elastic brain material.

### 5.6 Application to NHTSA’s Crash Tests

As discussed in Chapter 4, the brain injury models in the sagittal and coronal planes are capable of capturing the key characteristics of brain responses since most of NHTSA’s frontal and side impact tests have dominant planar head kinematics. The proposed 3D strain measure is applied to NHTSA’s crash tests to compare the performances of the planar and 3D models. The detailed results are presented in Appendix D. The results indicate that the 3D model captures the contributions of the out-of-plane head kinematics to the strain measurement. However, the overall trends using the planar and 3D models are only slightly different. The 3D model does not significantly outperform the two planar models for these
tests. This also suggests that the planar models are adequate for brain injury study under impacts having dominant planar head kinematics.

5.7 Car-Pedestrian Case Study

The configuration of head contact during the car-pedestrian crashes does not follow a specific pattern, and it depends on the motion and reaction of the pedestrians at the time of impact. Therefore, a planar brain injury model in either the sagittal plane or the coronal plane is not adequate to capture the important characteristics of head response. The proposed 3D strain measure is applied to accidents documented in the PCDS database. Case reconstructions are implemented using both the simple 3D brain injury model and the SIMon FE head model. Head/brain injuries recorded in the PCDS are compared with simulation results from the mathematical models to study their capabilities of head injury prediction under severe impacts.

5.7.1 Case Selection

A total of ten cases were chosen from the PCDS database based on the requirements of having: (1) a frontal pedestrian head impact with the front profile of a vehicle, and (2) sufficient pedestrian and vehicle information to complete a reconstruction. The brief description of the selected ten cases and the values of the maximum Abbreviated Injury Scale (AIS) of head injury reported in the PCDS database are summarized in Table 5.2. Maintained by the Association for the Advancement of Automotive Medicine, AIS is a scoring system to evaluate the severity of injury, where injuries are ranked on a scale of 1 to 6, with 1 being minor, 2 moderate, 3 serious, 4 severe, 5 critical, and 6 unsurvivable [20].
<table>
<thead>
<tr>
<th>Case No.</th>
<th>Vehicle Model/Year</th>
<th>Age (year)</th>
<th>Gender</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>MAIS (Head)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1994 Plymouth Sundance</td>
<td>6</td>
<td>M</td>
<td>102</td>
<td>20</td>
<td>5</td>
</tr>
<tr>
<td>2</td>
<td>1996 Dodge Ram</td>
<td>47</td>
<td>M</td>
<td>165</td>
<td>57</td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td>1989 Toyota Celica</td>
<td>8</td>
<td>F</td>
<td>138</td>
<td>62</td>
<td>2</td>
</tr>
<tr>
<td>4</td>
<td>1992 Plymouth Voyager</td>
<td>55</td>
<td>M</td>
<td>183</td>
<td>83</td>
<td>1</td>
</tr>
<tr>
<td>5</td>
<td>1994 Ford F150</td>
<td>42</td>
<td>M</td>
<td>168</td>
<td>83</td>
<td>5</td>
</tr>
<tr>
<td>6</td>
<td>1996 Ford Taurus</td>
<td>48</td>
<td>M</td>
<td>178</td>
<td>82</td>
<td>2</td>
</tr>
<tr>
<td>7</td>
<td>1993 Honda Civic Coupe</td>
<td>13</td>
<td>M</td>
<td>152</td>
<td>43</td>
<td>5</td>
</tr>
<tr>
<td>8</td>
<td>1996 Chevrolet Cavalier</td>
<td>25</td>
<td>F</td>
<td>170</td>
<td>60</td>
<td>5</td>
</tr>
<tr>
<td>9</td>
<td>1990 Honda Civic Hatchback</td>
<td>33</td>
<td>F</td>
<td>150</td>
<td>57</td>
<td>3</td>
</tr>
<tr>
<td>10</td>
<td>1990 Honda Civic Sedan</td>
<td>47</td>
<td>M</td>
<td>196</td>
<td>98</td>
<td>3</td>
</tr>
</tbody>
</table>

Table 5.2: Selected cases from PCDS database for reconstructions.

To accurately reconstruct these accidents, particular care was given to vehicle profile, pedestrian body size, impact velocity, and other relevant parameters, such as material properties of the vehicle hood and bumper. The detailed description of each accident can be found in Ott’s work [87]. Although the car-pedestrian crash simulations were reconstructed as accurately as possible, they were based on information collected by police, medical personnel and witnesses at the scene or after the accidents. Many uncertainties in the documented information may affect the accuracy of the crash simulations.

### 5.7.2 Crash Simulation

Based on the recorded case information from the PCDS database, each individual case was simulated in the Mathematical Dynamic Model (MADYMO). MADYMO is a program to simulate dynamic and kinematic responses of a physical model, especially emphasizing occupant safety analysis in vehicle crashes. For each case, a simulation was executed with known head-vehicle contact properties estimated beforehand. The head acceleration data
calculated from the simulation is input into the HIC criterion, the SIMon FE model, and the simple 3D brain injury model to predict different types of head/brain injuries.

To get an accurate vehicle profile, digital mapping of surrogate vehicles of the same make and model year as those involved in the accidents was completed using a digital position-recording device. Details are given in Ott’s work [87]. A 3D frontal profile of the vehicles was integrated in MADYMO for the crash simulation. Among the ten vehicles, three (Sundance, Voyager and Taurus) were approximated using a combination of simplified cylinders and planes to represent the vehicle profile due to the unavailability of these vehicles for this study.

The pedestrian models used in the crash simulations were Hybrid III ellipsoid standing dummy models provided in MADYMO. Three different dummies, 50th percentile male, 5th percentile female, and 6-year-old child, were used to closely represent their individual actual sizes. According to recorded body parameters, each model was also scaled in MADYMO to match the weight and height of each victim. For purposes of validation, the head impact velocities in MADYMO were also compared with published experimental data using human cadaver subjects in Ishikawa et al’s work [49].

The crash simulation in MADYMO was based on the reported information, including vehicle travel speed, pedestrian walking status, leg impact position, and head impact location and orientation. The accuracy of the simulations highly depends on the accuracy of these parameters at the time of impact. For each case, nine linear accelerations of the head were calculated in MADYMO. The accelerations are used as inputs to the HIC criterion, the SIMon FE model, and the simple 3D model in this study.
5.7.3 Parameter Modification

The two planar brain injury models have employed linear springs and dampers to study brain responses under impacts. The selections are appropriate under impacts of relatively low severity, such as in Hardy et al’s [41] tests and the NHTSA’s frontal and side impact tests. In Hardy et al’s [41] work, the measured relative brain motions are mostly within 5 mm and ±5 degrees. Under slightly more severe impacts in NHTSA’s frontal and side impact tests, they are mostly within 10 mm and ±10 degrees, using the linear springs and dampers. For the car-pedestrian case study, however, some cases generate extremely high head accelerations resulting from severe head impacts with the vehicles. The resultant relative brain displacements become very large. Therefore, linear springs and dampers are not enough to accurately capture the characteristics of brain motion under high-speed impacts. To limit the relative brain displacement with respect to the skull, the spring and damping coefficients are chosen to be nonlinear for the car-pedestrian study. Each spring/damping coefficient is modeled as a power function of the relative displacement at the point where the Kelvin element connects the brain, which indicates the change in length of the Kelvin element.

Figures 5.3 and 5.4 plot the new coefficients for the nonlinear spring $k_1$ and damper $c_1$ in the sagittal plane. The originally selected spring and damping constants are also plotted to compare with the new coefficients. The spring coefficient $k_1$ is a power function of the relative brain displacement at the connecting point $Q_1$ between Kelvin element 1 and the brain,

$$k_1 = 2.5 \Delta Q_1^{3.1} + 25000,$$

(5.6)

where $\Delta Q_1$ is the relative brain displacement at $Q_1$, and it indicates the change in the spring length. The spring coefficient increases with increased relative displacement and
Figure 5.3: The coefficients for linear and nonlinear spring $k_1$. $Q_1$ is the connecting point between Kelvin element 1 and the brain.

Figure 5.4: The coefficients for linear and nonlinear damper $c_1$. $Q_1$ is the connecting point between Kelvin element 1 and the brain.
therefore, the change in the spring length. As the relative displacement becomes larger, the
spring coefficient increases rapidly to limit the overall magnitude of brain movement. As
shown in Figure 5.3, the modified spring coefficient is very close to the originally selected
constant when the relative brain displacement is less than 5 mm. The change becomes more
dramatic as the relative brain displacement exceeds 10 mm. Therefore, the validation of the
planar models still applies since the relative brain displacements are mostly within 5 mm
under low-severity impacts. In the slightly more severe frontal and side impact tests, the
relative brain displacements are mostly within 10 mm, so the linear springs and dampers
also remain unchanged because the variations are not significant.

Similarly, the damping coefficient $c_1$ is also a power function of the relative brain dis-
placement at the connecting point $Q_1$,

$$c_1 = 0.1 \Delta Q_{11}^{3.1} + 300. \tag{5.7}$$

When the relative brain displacement at $Q_1$ is less 5 or 6 mm, the damping coefficient is
nearly a constant, and the difference between the nonlinear and linear dampers is negligible.

In the sagittal plane, the other three spring coefficients have similar profiles to $k_1$, and
their magnitudes vary proportionally to the initially selected spring constants. Likewise,
the other three damping coefficients also have similar profiles to $c_1$ with different magni-
tudes proportional to the originally selected damping constants. For the nonlinear springs
and dampers in the coronal plane, they all have similar profiles to those in the sagittal
plane. For the eight Kelvin elements in the 3D model, each spring/damping coefficient
is a power function of the relative brain displacement at the connecting point $Q_i$ between
Kelvin element $i$ and the brain,

$$y_i = a \Delta Q_{i}^{3.1} + b, \quad i = 1 \cdots 4, \tag{5.8}$$
where $y_i$ is the spring/damping coefficient and $\Delta Q_i$ is the relative brain displacement at $Q_i$.

The constants $a$ and $b$ for each spring/damper are listed in Table 5.3.

<table>
<thead>
<tr>
<th>Spring/damper</th>
<th>Sagittal Plane</th>
<th>Coronal Plane</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$a$</td>
<td>$b$</td>
</tr>
<tr>
<td>$k_1$</td>
<td>2.5</td>
<td>25000</td>
</tr>
<tr>
<td>$k_2$</td>
<td>1.8</td>
<td>18000</td>
</tr>
<tr>
<td>$k_3$</td>
<td>1.5</td>
<td>15000</td>
</tr>
<tr>
<td>$k_4$</td>
<td>3.5</td>
<td>35000</td>
</tr>
<tr>
<td>$c_1$</td>
<td>0.1</td>
<td>300</td>
</tr>
<tr>
<td>$c_2$</td>
<td>0.037</td>
<td>110</td>
</tr>
<tr>
<td>$c_3$</td>
<td>0.0067</td>
<td>20</td>
</tr>
<tr>
<td>$c_4$</td>
<td>0.117</td>
<td>350</td>
</tr>
</tbody>
</table>

Table 5.3: Profiles of nonlinear springs and dampers for the 3D model.

### 5.7.4 Reconstruction Results

#### HIC vs. Skull Fracture

Table 5.4 summarizes the HIC values and the probability of skull fracture (AIS 3) for each case. The injury risk functions for skull fractures of various severities are given in Appendix C. Since the HIC criterion of skull fracture has been studied by many researchers for decades, this study does not attempt to propose a different measure to predict skull fracture using the simple 3D model. Although it is still unclear whether HIC is directly related to a specific type of brain injury or not, HIC is an important factor indicating the severity of impact. For the ten car-pedestrian cases, the HIC values generally match the injury levels of reported skull fractures. However, case 3 with the Celica has a very high HIC value of 2338, but no skull fractures were reported, while case 9 with the Civic Hatchback has a relatively low HIC value of only 215, but an AIS 2 skull fracture was reported.
Table 5.4: Comparing reported skull fractures with HIC values.

<table>
<thead>
<tr>
<th>Case No./Model</th>
<th>Skull Fractures in Accident Report</th>
<th>HIC</th>
<th>Skull Fracture Risk (AIS 3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Sundance</td>
<td>Fracture of right temporal bone (AIS 2)</td>
<td>3133</td>
<td>100%</td>
</tr>
<tr>
<td>2 Dodge Ram</td>
<td>No skull fracture</td>
<td>275</td>
<td>4%</td>
</tr>
<tr>
<td>3 Celica</td>
<td>No skull fracture</td>
<td>2338</td>
<td>99%</td>
</tr>
<tr>
<td>4 Voyager</td>
<td>No skull fracture</td>
<td>740</td>
<td>29%</td>
</tr>
<tr>
<td>5 F150</td>
<td>Orbital plate fracture (AIS 3)</td>
<td>1904</td>
<td>97%</td>
</tr>
<tr>
<td>6 Taurus</td>
<td>No skull fracture</td>
<td>437</td>
<td>10%</td>
</tr>
<tr>
<td>7 Civic Coupe</td>
<td>Fractures of temporal, parietal and occipital bones (AIS 3)</td>
<td>6868</td>
<td>100%</td>
</tr>
<tr>
<td>8 Cavalier</td>
<td>Left &amp; right occipital condyle fractures (AIS 3)</td>
<td>14468</td>
<td>100%</td>
</tr>
<tr>
<td>9 Civic Hatchback</td>
<td>Temporoparietal bone fracture (AIS 2)</td>
<td>215</td>
<td>3%</td>
</tr>
<tr>
<td>10 Civic Sedan</td>
<td>No skull fracture</td>
<td>774</td>
<td>32%</td>
</tr>
</tbody>
</table>

**Brain Rotation vs. ASDH**

Due to the incompressibility of brain tissue and limited space between the brain and skull, significant relative brain translation at the brain/skull interface is not allowed. However, the existence of CSF in the subarachnoid space eases the sliding of the brain with respect to the skull. Relatively large sliding between the brain and skull may result in the breaking of blood vessels, causing ASDH. For the simple 3D model, the relative brain rotation with respect to the skull is used as the injury indicator to predict ASDH, but there is no published data relating brain rotation to injury. For the case reconstructions, the relative brain rotation is compared with the observed ASDH injuries in the car-pedestrian accidents.
to evaluate its capability of injury prediction. The risk functions for different brain injuries in the SIMon FE model are given in Appendix C.

Table 5.5 lists the results of ASDH prediction from both the simple 3D model and the SIMon FE model. The relative brain rotation from the simple model generally matched the severity of ASDH injuries from the accident reports. Case 1 with the Sundance reported an AIS 5 hemorrhaging, while the simple 3D model has a brain rotation of 14.4 degrees in the coronal plane and 5.2 degrees in the sagittal plane. Also having reported hematomas, cases 7 and 8 both have relatively large brain rotation. Without reported bleeding, case 6 with the Taurus had a brain rotation of 7.6 degrees in the coronal plane and 1.4 degrees in the sagittal plane using the simple model. In general, relative brain rotation greater than 13 degrees seems to indicate a severe ASDH injury, such as in cases 1, 7 and 8. An exception exists in case 2 with the Dodge Ram, in which small brain rotation is obtained in the simple model, but an AIS 5 subdural hematoma was reported. For the SIMon FE model, the RMDM measure for ASDH prediction is generally overestimated. In SIMon, a total of 9 cases were predicted with high risks of ASDH (all higher than 88%). Among these cases, however, no injuries were reported for 5 cases.

In addition, the simple 3D model also predicted the locations of ASDH injuries. In cases 1 and 7, ASDH injuries were reported in the left and right side of the brain, respectively. Using the simple 3D model, the large relative brain rotations in the coronal plane correspond to the reported local ASDH injuries for these two cases.

**Maximum Shear Strain vs. DAI**

DAI is predicted using the maximum shear strain from the 3D brain injury model. This maximum shear strain was validated against the experimental data under low-speed impacts by matching the severity of impacts. The simple 3D model only gives a single value of
<table>
<thead>
<tr>
<th>Case No./Model</th>
<th>ASDH Injury from Accident Report</th>
<th>Simple 3D Model</th>
<th>SIMon Head Model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Sagittal (deg)</td>
<td>Coronal (deg)</td>
</tr>
<tr>
<td>1 Sundance</td>
<td>Left intraventricular hemorrhaging (AIS 5)</td>
<td>5.2</td>
<td>14.4</td>
</tr>
<tr>
<td>2 Dodge Ram</td>
<td>Right frontoparietal subdural hematoma (AIS 5)</td>
<td>4.6</td>
<td>5.5</td>
</tr>
<tr>
<td>3 Celica</td>
<td>No reported internal bleeding</td>
<td>2.5</td>
<td>7.2</td>
</tr>
<tr>
<td>4 Voyager</td>
<td>No reported internal bleeding</td>
<td>4.5</td>
<td>10.9</td>
</tr>
<tr>
<td>5 F150</td>
<td>No reported internal bleeding</td>
<td>7.3</td>
<td>11.0</td>
</tr>
<tr>
<td>6 Taurus</td>
<td>No reported internal bleeding</td>
<td>1.4</td>
<td>7.6</td>
</tr>
<tr>
<td>7 Civic Coupe</td>
<td>Right subdural hematoma (AIS 4)</td>
<td>6.4</td>
<td>16.1</td>
</tr>
<tr>
<td>8 Cavalier</td>
<td>Large subarachnoid &amp; epidural hematoma to midbrain (AIS 5)</td>
<td>5.8</td>
<td>13.2</td>
</tr>
<tr>
<td>9 Civic Hatchback</td>
<td>No reported internal bleeding</td>
<td>4.3</td>
<td>11.7</td>
</tr>
<tr>
<td>10 Civic Sedan</td>
<td>No reported bleeding</td>
<td>3.8</td>
<td>10.9</td>
</tr>
</tbody>
</table>

Table 5.5: ASDH prediction using relative brain rotation from the simple 3D model and RMDM results from the SIMon FE model.

strain at the center of the spherical brain, while SIMon predicts DAI by calculating the cumulative strain in the whole brain. This section evaluates the proposed simple strain measure from the simple 3D model for DAI prediction.

Table 5.6 summarizes the results for DAI prediction from the simple 3D model and the SIMon FE model. Cases 1, 5, 7, and 8 reported positive DAI injuries resulting in victims’ deaths. Three severe DAI injuries in cases 5, 7, and 8 are captured by the simple 3D model,
with high resultant maximum shear strain in all cases. Case 1 with the Sundance resulted in a victim’s death, but the strain of 0.37 is not significantly high compared with the other three deadly crashes. Therefore, the strain in case 1 did not closely match the reported DAI. However, the maximum shear strain proposed for DAI prediction using the simple 3D model generally matched the reported injury level of DAI. In SIMon, the CSDM values also mostly matched the reported DAI injuries, but it only predicted a 5% probability of DAI for case 5 with the F150, which actually had a reported AIS 5 level of DAI.

<table>
<thead>
<tr>
<th>Case No./Model</th>
<th>DAI Injury from Accident Report</th>
<th>Simple 3D Model</th>
<th>SIMon Head Model</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sundance</td>
<td>LOC &gt; 24 hrs, diffuse edema (AIS 5), positive DAI</td>
<td>0.37</td>
<td>0.527</td>
</tr>
<tr>
<td>Dodge Ram</td>
<td>No DAI reported</td>
<td>0.10</td>
<td>0.043</td>
</tr>
<tr>
<td>Celica</td>
<td>No DAI reported</td>
<td>0.22</td>
<td>0.193</td>
</tr>
<tr>
<td>Voyager</td>
<td>No DAI reported</td>
<td>0.29</td>
<td>0.075</td>
</tr>
<tr>
<td>F150</td>
<td>Extensive shear injury (AIS 5), positive DAI</td>
<td>0.56</td>
<td>0.021</td>
</tr>
<tr>
<td>Taurus</td>
<td>No DAI reported, LOC (AIS 2)</td>
<td>0.05</td>
<td>0.148</td>
</tr>
<tr>
<td>Civic Coupe</td>
<td>24 hr LOC (AIS 5), positive DAI</td>
<td>0.82</td>
<td>0.859</td>
</tr>
<tr>
<td>Cavalier</td>
<td>Coma, unresponsive, flaccid &gt; 24 hrs, positive DAI</td>
<td>0.97</td>
<td>0.919</td>
</tr>
<tr>
<td>Civic Hatchback</td>
<td>No DAI reported</td>
<td>0.08</td>
<td>0.015</td>
</tr>
<tr>
<td>Civic Sedan</td>
<td>No DAI reported, LOC (AIS 2)</td>
<td>0.38</td>
<td>0.132</td>
</tr>
</tbody>
</table>

Table 5.6: DAI prediction using maximum shear strain from the simple 3D model and CSDM (0.15) results from the SIMon FE model.
DDM vs. Cerebral Contusion

No injury measure for cerebral contusion has been proposed for the simple 3D model. Generally related to the pressure distribution at different locations of the brain, prediction of cerebral contusion is currently infeasible with this simple model. The cases were simulated in SIMon, which uses DDM values to predict cerebral contusions under impacts. The DDM results and the risk of cerebral contusions are summarized in Table 5.7. Although DDM values from SIMon generally match the reported cerebral contusions, there are still some discrepancies between the DDM values and the reported injuries. In case 8 with the Cavalier, there are no reported cerebral contusions, but SIMon predicted a 100% probability of cerebral contusion.

<table>
<thead>
<tr>
<th>Case No./Model</th>
<th>cerebral contusions in Accident Report</th>
<th>SIMon Head Model</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Sundance</td>
<td>No reported contusions</td>
<td>0.030</td>
</tr>
<tr>
<td>2 Dodge Ram</td>
<td>Small frontal contusion (AIS 3)</td>
<td>0.050</td>
</tr>
<tr>
<td>3 Celica</td>
<td>No reported contusions</td>
<td>0.003</td>
</tr>
<tr>
<td>4 Voyager</td>
<td>No reported contusions</td>
<td>0.028</td>
</tr>
<tr>
<td>5 F150</td>
<td>Large parietal contusion (AIS 4)</td>
<td>0.006</td>
</tr>
<tr>
<td>6 Taurus</td>
<td>No reported contusions</td>
<td>0.012</td>
</tr>
<tr>
<td>7 Civic Coupe</td>
<td>Bilateral temporal contusion (AIS 3)</td>
<td>0.134</td>
</tr>
<tr>
<td>8 Cavalier</td>
<td>No reported contusions</td>
<td>0.319</td>
</tr>
<tr>
<td>9 Civic Hatchback</td>
<td>Small right temporal contusion (AIS 3)</td>
<td>0.008</td>
</tr>
<tr>
<td>10 Civic Sedan</td>
<td>Bilateral temporal contusion (AIS 3)</td>
<td>0.045</td>
</tr>
</tbody>
</table>

Table 5.7: cerebral contusion prediction using DDM values in SIMon FE model.
5.8 Discussion

A simple scheme was used to combine the two planar brain injury models into a 3D model. 3D head accelerations, therefore, can be accepted as inputs for better injury prediction. Using this simple 3D model, the results of injury predictions were compared with the experimental data in Hardy et al’s work [41], and they closely matched the severity of impacts. With nonlinear springs and dampers, the model was further applied to much more severe car-pedestrian accidents. The results were compared with medical records and the results from the SIMon FE model. Injury metrics were proposed to predict ASDH and DAI, but not cerebral contusion. Further extension of the simple models developed in this work is needed for cerebral contusion study.

The proposed injury metric for ASDH prediction is the relative brain rotation with respect to the skull. It is believed that the space allowing brain translation at the brain/skull boundary is very limited due to the anatomical structure of the human head [131]. However, shear motion between the brain and skull is much easier because of the existence of CSF. Instead of using relative brain rotation, relative displacement at the brain/skull boundary might be used for ASDH prediction, but more experimental data is needed through direct observation of motion at the brain/skull boundary.

DAI is predicted using the maximum shear strain in the spherical brain of the 3D model. The strain calculation is only at the geometric center, which is the intersection of the three cross sections dividing the brain into hemispheres. In the SIMon FE model, however, CSDM is a cumulative measure of the strains throughout the whole brain. While FE models can provide detailed strain contributions in the brain tissue, the simple 3D model has less computational cost. More importantly, the strain calculations using the simple 3D model are capable of predicting the reported DAI injuries in the car-pedestrian accidents.
For decades, skull fractures resulting from linear accelerations have been investigated in depth using the HIC criterion. Research has also been conducted to directly relate brain injuries to the linear accelerations and HIC values [128]. For brain injuries resulting from head rotation, however, only angular accelerations in the sagittal plane and coronal plane have been highlighted in recent experimental studies. The lack of experimental data under head rotation in the horizontal plane prevents the development and validation of a similar model in this plane. Therefore, this study focuses on the brain responses only in the sagittal and coronal planes. Once more experimental data are available, a horizontal plane model can be developed and integrated into the current 3D model to accept full 3D head accelerations.

The crash simulations executed in MADYMO used different dummies, 50th percentile male, 5th percentile female, and 6-year-old child, to represent each pedestrian. In addition, each MADYMO model was scaled to closely match the actual size of each pedestrian. However, the simple 3D model and the SIMon FE model both used an average head/brain size of an adult for all the cases. This could potentially introduce errors in the case reconstructions, particularly for case 1 involving a 6-year-old child. The head/brain size should be varied in mathematical modeling work to include their effect on injury predictions.

5.9 Conclusions

A total of ten car-pedestrian accidents were selected for crash reconstructions with adequate information of striking vehicles, victims, and reported injuries after impact. Both the simple 3D model and the SIMon FE model were used to study different brain injuries. Using the simple 3D model, injury metrics were proposed for ASDH and DAI prediction, while SIMon also has an injury metric to predict cerebral contusion. The crash simulations
were executed in MADYMO to reproduce the selected car-pedestrian crashes. Nine linear head accelerations were calculated during the simulations and used as inputs for both the simple 3D model and the SIMon FE model. The results from the simple model and SIMon were compared with head injuries documented in accident reports. The results show that the proposed injury metrics for ASDH and DAI prediction using the simple 3D model have the capability of predicting real-world brain injuries in car-pedestrian crashes. The results also suggest that further improvement of the MADYMO simulations is needed to enhance the fidelity of car-pedestrian crash reconstructions.
6.1 Introduction

Research on human brain injury resulting from head impact has employed a variety of experiments and mathematical modeling work. One element of both the experimental and modeling work that has not been adequately investigated to date is the sensitivity of the resulting injury predictions to small parameter changes in the experiment or the model. To determine which parameters are critical to the injury predictions, it is time consuming and financially expensive to execute a large number of physical tests or to run a large number of high-fidelity FE models. Therefore, some fundamental understanding of the problem is needed beforehand to make the sensitivity analysis more efficient.

Based on the simplified brain injury model in the sagittal plane developed earlier in this work, this chapter performs sensitivity analysis to determine the parameters to which injury prediction is most sensitive and to provide guidelines for the development of experimental tests and computational models. Leveraging the closed-form solutions of the simplified model, the sensitivity analysis is performed analytically with simple inputs. A high-fidelity FE head model is used to verify the findings by running a series of simulations with changing parameters.
6.2 Background

Sensitivity analysis is a measure of the effect of parameter variations on the dynamics of a system, such as the time response, the state, the transfer function, or any other quantity characterizing the system dynamics [29]. Its objective is to investigate how output changes result from parameter variation of inputs. The parameter variations include the errors from measurements or approximations made for mathematical modeling. Changes in the parameters are also part of the parameter variations. When mathematical models are developed, assumptions are usually made to simplify the modeling work, such as the assumption of the spherical brain in the present study. Parameter deviations between real systems and mathematical models could be very large, and the solution could be very sensitive to the parameters. Small variation of the system parameters will be studied by assuming that the variation of the system parameters is time-independent.

Let \( \zeta = \zeta(\alpha) \) be a system function that characterizes the behavior of the dynamic system. For example, \( \zeta \) could be the state of the system. \( \alpha = [\alpha_1 \ \alpha_2 \ \cdots \ \alpha_m]^T \) is a system parameter vector, where \( m \) is the number of model parameters. Let the nominal parameter vector be denoted by \( \alpha_0 = [\alpha_{10} \ \alpha_{20} \ \cdots \ \alpha_{m0}]^T \) and the nominal system function by \( \zeta_0 \equiv \zeta(\alpha_0) \). The absolute sensitivity function with respect to the \( j \)th parameter is defined as,

\[
S_j \equiv \left. \frac{\partial \zeta(\alpha)}{\partial \alpha_j} \right|_{\alpha_0} = S_j(\alpha_0), \quad j = 1, 2, \cdots, m,
\]

(6.1)

where the subscript \( \alpha_0 \) indicates that the partial derivative is evaluated at the nominal parameter values. In other words, the sensitivity analysis is evaluated at the nominal values.
Sensitivity functions are usually dealt with in either the time domain or the frequency domain. In the time domain, the commonly used sensitivity functions include output sensitivity function, trajectory sensitivity function, overshoot sensitivity function, and eigenvalue sensitivity function, depending on specific problems. In the frequency domain, the commonly used sensitivity functions include Bode’s sensitivity function, maximum-modulus sensitivity function, and root sensitivity function [29].

For the brain injury model in this work, the trajectory sensitivity analysis in state space is discussed in detail. The trajectory is the time history of the state of a system, and it specifically refers to the displacements and velocities of the skull and brain during impact in this chapter. It is useful to know how the system trajectory varies with some small changes in the model parameters. For the sagittal plane model, the trajectory sensitivity can be directly related to the outputs, providing information about the sensitivity of relative brain displacements to the model parameters. Further reason to choose the trajectory sensitivity analysis is the simplicity of the system equations in state space.

A continuous system can generally be described in state space by a vector differential equation of the form,

\[ \dot{x} = f(x, t, u; \alpha), \quad x(t_0) = x^0, \]  
\[ y = g(x, t, u; \alpha), \]  

where the first equation is called the state equation of the system and the second is the output vector equation. \( x \) denotes an \( n \times 1 \) state vector, \( y \) a \( q \times 1 \) output vector, \( f \) an \( n \times 1 \) vector function, \( u \) an input vector, \( \alpha \) an \( m \times 1 \) parameter vector, and \( x^0 \) an \( n \times 1 \) initial condition vector.
The trajectory sensitivity vector with respect to the \( j \)th parameter is defined as,

\[
S_j(t, \alpha_0) \equiv \left. \frac{\partial x(t, \alpha)}{\partial \alpha_j} \right|_{\alpha_0}, \quad j = 1, 2, \ldots, m, \quad (6.4)
\]

where the trajectory sensitivity vector \( S_j \) has the same dimension as the state vector \( x \). The entirety of all \( n \times m \) trajectory sensitivity functions forms the trajectory sensitivity matrix,

\[
S = \begin{bmatrix} S_1 & S_2 & \cdots & S_m \end{bmatrix} \equiv \begin{bmatrix} \frac{\partial x_1}{\partial \alpha_1} & \cdots & \frac{\partial x_1}{\partial \alpha_m} \\ \vdots & \ddots & \vdots \\ \frac{\partial x_n}{\partial \alpha_1} & \cdots & \frac{\partial x_n}{\partial \alpha_m} \end{bmatrix}. \quad (6.5)
\]

Taking the partial derivative of the state Equation 6.2 with respect to \( \alpha_j \) gives,

\[
\frac{\partial \dot{x}}{\partial \alpha_j} = \frac{\partial f}{\partial x} \frac{\partial x}{\partial \alpha_j} + \frac{\partial f}{\partial \alpha_j}, \quad \frac{\partial x^0}{\partial \alpha_j} = 0, \quad j = 1, 2, \ldots, m. \quad (6.6)
\]

The partial derivative of the initial state with respect to the system parameter \( \alpha_j \) goes to zero since the initial condition vector \( x^0 \) does not depend on \( \alpha \). There are \( m \) equations of this form since \( \alpha \) is \( m \)-dimensional. Interchanging the sequence of the derivatives with respect to \( \alpha_j \) and time \( t \) yields,

\[
\frac{d}{dt} \frac{\partial x}{\partial \alpha_j} = \frac{\partial f}{\partial x} \frac{\partial x}{\partial \alpha_j} + \frac{\partial f}{\partial \alpha_j}. \quad (6.7)
\]

Evaluating Equation 6.7 at nominal values \( \alpha_0 \) gives,

\[
\dot{S}_j = \left. \frac{\partial f}{\partial x} \right|_{\alpha_0} S_j + \left. \frac{\partial f}{\partial \alpha_j} \right|_{\alpha_0}, \quad \dot{S}_j(0) = 0, \quad j = 1, 2, \ldots, m, \quad (6.8)
\]

where \( S_j \equiv \left. \frac{\partial x}{\partial \alpha_j} \right|_{\alpha_0} \) is the trajectory sensitivity vector defined in Equation 6.4. Equation 6.8 is called the trajectory sensitivity equation. A similar procedure on the output vector Equation 6.3 gives,

\[
\sigma_j = \left. \frac{\partial y}{\partial \alpha_j} \right|_{\alpha_0} = \left. \frac{\partial g}{\partial x} \right|_{\alpha_0} S_j + \left. \frac{\partial g}{\partial \alpha_j} \right|_{\alpha_0}, \quad j = 1, 2, \ldots, m, \quad (6.9)
\]
where $\sigma_j \equiv \frac{\partial y}{\partial \alpha_j}\big|_{\alpha_0}$ is the output sensitivity vector, which relates to the trajectory sensitivity vector $S_j$. For a system having a $q \times 1$ output vector $y$ depending on an $m \times 1$ parameter vector $\alpha$, the output sensitivity matrix with respect to parameter $\alpha$ is defined as,

\[
\sigma = \frac{\partial y}{\partial \alpha}\big|_{\alpha_0} = \begin{bmatrix}
\frac{\partial y_1}{\partial \alpha_1} & \cdots & \frac{\partial y_1}{\partial \alpha_m} \\
\vdots & \ddots & \vdots \\
\frac{\partial y_q}{\partial \alpha_1} & \cdots & \frac{\partial y_q}{\partial \alpha_m}
\end{bmatrix},
\]

(6.10)

Combining the trajectory sensitivity matrix $S$ and the output sensitivity matrix $\sigma$ gives the general form of the state sensitivity equations of the system,

\[
\dot{S} = \left. \frac{\partial f}{\partial x} \right|_{\alpha_0} S + \left. \frac{\partial f}{\partial \alpha} \right|_{\alpha_0} x_0(t), \quad S^0 = 0,
\]

\[
\sigma = \left. \frac{\partial g}{\partial x} \right|_{\alpha_0} S + \left. \frac{\partial g}{\partial \alpha} \right|_{\alpha_0}.
\]

(6.11)

For a linear time-invariant system, the state and output Equations 6.2 and 6.3 take the form,

\[
\dot{x} = Ax + Bu, \quad x(t_0) = x^0,
\]

\[
y = Cx + Du,
\]

(6.12)

(6.13)

where $A = A(\alpha)$, $B = B(\alpha)$, $C = C(\alpha)$, $D = D(\alpha)$, $x = x(t, \alpha)$, and $y = y(t, \alpha)$. The matrices $A$, $B$, $C$, and $D$ are the system matrix, input matrix, output matrix, and direct transmission matrix, respectively. Note that the external input vector $u$ of the system is not a function of $\alpha$. Taking the partial derivative of Equation 6.12 with respect to $\alpha_j$ and switching the sequence of derivatives with respect to time $t$ and $\alpha_j$ gives the trajectory sensitivity equation,

\[
\dot{S}_j = A_0 S_j + \left. \frac{\partial A}{\partial \alpha_j} \right|_{\alpha_0} x_0(t) + \left. \frac{\partial B}{\partial \alpha_j} \right|_{\alpha_0} u(t), \quad S_j(0) = 0, \quad j = 1, 2, \cdots, m,
\]

(6.14)

where $A_0 = A(\alpha_0)$. $x_0(t) = x(t, \alpha_0)$ and is the solution of the nominal vector state equation,

\[
\dot{x}_0 = f(x_0, t, u; \alpha_0), \quad x(t_0) = x^0.
\]

(6.15)
A similar procedure applied to Equation 6.13 gives the output sensitivity equation,

\[
\sigma_j = C_0 S_j + \frac{\partial C}{\partial \alpha_j} \bigg|_{\alpha_0} x_0(t) + \frac{\partial D}{\partial \alpha_j} \bigg|_{\alpha_0} u(t), \quad j = 1, 2, \ldots, m, \tag{6.16}
\]

where \(C_0 = C(\alpha_0)\). Writing the trajectory sensitivity equation and output sensitivity equation in a general form gives the state sensitivity equations,

\[
\dot{S} = A_0 S + \frac{\partial A}{\partial \alpha} \bigg|_{\alpha_0} x_0(t) + \frac{\partial B}{\partial \alpha} \bigg|_{\alpha_0} u(t), \quad S(t_0) = 0, \tag{6.17}
\]

\[
\sigma = C_0 S + \frac{\partial C}{\partial \alpha} \bigg|_{\alpha_0} x_0(t) + \frac{\partial D}{\partial \alpha} \bigg|_{\alpha_0} u(t), \tag{6.18}
\]

where \(S\) and \(\sigma\) are the trajectory and output sensitivity matrices, respectively. Equations 6.17 and 6.18 will be directly used to perform sensitivity analysis in this work.

### 6.3 Equations of Motion in State Space

To apply the trajectory sensitivity analysis to the simplified brain injury model in the sagittal plane, the equations of motion in Equations 3.41 developed in Section 3.6.2 are written in state space form. During crash tests, the time history of measured head kinematics is used as an input vector \(u = [u_1 \ u_2 \ \cdots \ u_6]^T\), where \(u_1 = x_s, u_2 = \dot{x}_s, u_3 = z_s, u_4 = \dot{z}_s, u_5 = \theta_s,\) and \(u_6 = \dot{\theta}_s\). The velocity and displacement of the head are obtained by integrating the measured head accelerations. The state vector is \(x = [x_1 \ x_2 \ \cdots \ x_6]^T\), where the state variables are \(x_1 = x_b, x_2 = \dot{x}_b, x_3 = z_b, x_4 = \dot{z}_b, x_5 = \theta_b,\) and \(x_6 = \dot{\theta}_b\). Writing the state space equations of motion in matrix form,

\[
\dot{x} = Ax + Bu, \quad x(t_0) = x^0, \quad y = Cx + Du, \tag{6.19}
\]
where the system matrix $A$, input matrix $B$, output matrix $C$, and direct transmission matrix $D$ are given by,

$$A = \begin{bmatrix}
0 & 1 & 0 & 0 & 0 & 0 \\
\frac{k_{11}}{m_b} & \frac{-c_{11}}{m_b} & \frac{-k_{12}}{m_b} & \frac{-c_{12}}{m_b} & \frac{k_{13}}{m_b} & \frac{-c_{13}}{m_b} \\
0 & 0 & 0 & 1 & 0 & 0 \\
\frac{k_{21}}{m_b} & \frac{-c_{21}}{m_b} & \frac{-k_{22}}{m_b} & \frac{-c_{22}}{m_b} & \frac{k_{23}}{m_b} & \frac{-c_{23}}{m_b} \\
0 & 0 & 0 & 0 & 0 & 1 \\
\frac{k_{31}}{I_b} & \frac{-c_{31}}{I_b} & \frac{-k_{32}}{I_b} & \frac{-c_{32}}{I_b} & \frac{k_{33}}{I_b} & \frac{-c_{33}}{I_b}
\end{bmatrix},$$

$$B = \begin{bmatrix}
0 & 0 & 0 & 0 & 0 & 0 \\
\frac{k_{11}}{m_b} & \frac{k_{11}}{m_b} & \frac{k_{12}}{m_b} & \frac{k_{12}}{m_b} & \frac{k_{13}}{m_b} & \frac{c_{13}}{m_b} \\
0 & 0 & 0 & 0 & 0 & 0 \\
\frac{k_{21}}{m_b} & \frac{k_{21}}{m_b} & \frac{k_{22}}{m_b} & \frac{k_{22}}{m_b} & \frac{k_{23}}{m_b} & \frac{c_{23}}{m_b} \\
0 & 0 & 0 & 0 & 0 & 0 \\
\frac{k_{31}}{I_b} & \frac{k_{31}}{I_b} & \frac{k_{32}}{I_b} & \frac{k_{32}}{I_b} & \frac{k_{33}}{I_b} & \frac{c_{33}}{I_b}
\end{bmatrix},$$

$$C = \begin{bmatrix}
1 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 1 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 1 & 0
\end{bmatrix},$$

$$D = \begin{bmatrix}
-1 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & -1 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & -1 & 0
\end{bmatrix}.$$  

The initial condition of the system is $x^0$. The outputs of the system are the relative brain translations with respect to the skull along the $X$ and $Z$ axes and the relative brain rotation about the $Y$ axis. Comparing the input vector $u$ and state vector $x$ gives the relative brain displacements,

$$y_1 = x_1 - u_1,$$

$$y_2 = x_3 - u_3,$$

$$y_3 = x_5 - u_5.$$  

Similarly, the relative velocities of the brain with respect to the skull can also be obtained by comparing the input vector $u$ and state vector $x$. 

175
6.4 Solution of State Sensitivity Equations

6.4.1 Solution of Nominal State Equations

In order to solve for the state sensitivity function $S_{ij}$, the solution of the nominal state equations $x_0$ in Equation 6.15 is obtained first. For the sagittal plane brain injury model, the system parameters are $m_b$, $I_b$, $d$, $k_i$, and $c_i$, where $i = 1 \cdots 4$; therefore, the parameter vector is $\alpha = [\alpha_1 \alpha_2 \cdots \alpha_{11}]^T$, where $\alpha_1 = m_b$, $\alpha_2 = I_b$, $\alpha_3 = d$, $\alpha_4 = k_1$, $\alpha_5 = k_2$, $\alpha_6 = k_3$, $\alpha_7 = k_4$, $\alpha_8 = c_1$, $\alpha_9 = c_2$, $\alpha_{10} = c_3$, and $\alpha_{11} = c_4$. The nominal parameter vector is $\alpha_0 = [\alpha_{10} \alpha_{20} \cdots \alpha_{110}]^T$, where $\alpha_{10} = 1.40$, $\alpha_{20} = 0.0038$, $\alpha_{30} = 0.040$, $\alpha_{40} = 25$, $\alpha_{50} = 18$, $\alpha_{60} = 15$, $\alpha_{70} = 35$, $\alpha_{80} = 300$, $\alpha_{90} = 110$, $\alpha_{100} = 20$, and $\alpha_{110} = 350$. These are the model parameters for the sagittal plane model, as listed in Table 3.2.

Substituting the model parameters into the state Equation 6.19 gives the nominal state equation,

$$\dot{x}_0 = A_0 x_0 + B_0 u_0, \quad x_0 (t_0) = x^0, \quad (6.21)$$

where,

$$A_0 = \begin{bmatrix}
0 & 1 & 0 & 0 & 0 & 0 \\
-33214.3 & -278.6 & 4642.9 & 50.0 & -545.5 & -10.5 \\
0 & 0 & 0 & 0 & 0 & 0 \\
4642.9 & 50.0 & -33214.3 & -278.6 & 141.4 & -0.8 \\
0 & 0 & 0 & 0 & 0 & 0 \\
-200967.2 & -3870.5 & 52102.6 & -297.7 & -39157.9 & -328.4
\end{bmatrix},$$

$$B_0 = \begin{bmatrix}
0 & 0 & 0 & 0 & 0 & 0 \\
33214.3 & 278.6 & -4642.9 & -50.0 & 545.5 & 10.5 \\
0 & 0 & 0 & 0 & 0 & 0 \\
-4642.9 & -50.0 & 33214.3 & 278.6 & -141.4 & 0.8 \\
0 & 0 & 0 & 0 & 0 & 0 \\
200967.2 & 3870.5 & -52102.6 & 297.7 & 39157.9 & 328.4
\end{bmatrix}. $$

It is well known from state space theory that the linear vector equation,

$$\dot{x} = A (t) x + B (t) u (t), \quad x (t_0) = x^0, \quad (6.22)$$

176
has the general solution,

\[ x(t) = \Phi(t, t_0) x(t_0) + \int_{t_0}^{t} \Phi(t, \tau) B(\tau) u(\tau) d\tau, \]  

(6.23)

where \( \Phi(t, t_0) \) is the state transition matrix satisfying the following equations,

\[ \frac{d}{dt} \Phi(t, t_0) = A(t) \Phi(t, t_0), \]

\[ \Phi(t_0, t_0) = I, \]  

(6.24)

where \( I \) is the identity matrix. For this linear time-invariant system, the state transition matrix is,

\[ \Phi(t, t_0) = \Phi(t-t_0) = e^{A(t-t_0)}. \]  

(6.25)

Therefore, the solution of the nominal state Equation 6.21 takes the form,

\[ x_0(t) = e^{A_0(t-t_0)} x(t_0) + \int_{t_0}^{t} e^{A_0(t-\tau)} B_0(\tau) u(\tau) d\tau. \]  

(6.26)

The initial condition of the system is \( x^0 = 0 \), which means that the system is at rest before the impact occurs at the starting time \( t_0 = 0 \). The solution becomes,

\[ x_0(t) = \int_{0}^{t} e^{A_0(t-\tau)} B_0(\tau) u(\tau) d\tau, \]  

(6.27)

which is a convolution integral. The solution of \( x_0(t) \) is obtained by evaluating this integral.

In order to make the closed-form solution possible for Equation 6.27 and for the trajectory and output sensitivity matrices in Equations 6.17 and 6.18, standard functions are applied to the brain injury model as inputs. Since the head accelerations during automobile accidents are characterized by short duration and large magnitude, the translational acceleration along the \( X \) axis and the rotational acceleration about the \( Y \) axis are idealized as impulse functions \( \delta(t) \). The translational acceleration along the \( Z \) axis is assumed to be
zero to simplify the calculation. Further analysis with nonzero accelerations along the $Z$ axis gives similar results as presented here.

Using impulse linear and angular accelerations for the head, the input vector is $u = [t \ u(t) \ 0 \ 0 \ t \ u(t)]^T$, where $u(t)$ is a step function, the integral of $\delta(t)$ over time $t$. Applying vector $u$ as inputs to the simplified sagittal plane model, the relative brain displacement is about 3 mm in the $X$ direction and 2.5 degrees about the $Y$ axis, while it is small in the $Z$ direction. The results are consistent with the experimental results of brain motion under low-severity impacts. This also suggests that the selection of impulse functions as inputs is appropriate for the sensitivity analysis.

In Bandak and Eppinger’s [6] and Zhang et al.’s [126] work, sinusoidal functions were used as head accelerations for FE models to study brain responses under impact. Therefore, this work also applies sinusoidal functions as inputs to the sensitivity analysis, in a similar manner to the use of impulse functions. With either impulse or sinusoidal functions as inputs, the nominal solution for the state variables has the following general form,

$$x_{0i}(t) = a_i + b_i t + \sum_{j=1}^{3} [c_{1ji} \cos (c_{2ji} t) + c_{3ji} \sin (c_{4ji} t)] e^{d_j t}, \quad i = 1 \cdots 6,$$

(6.28)

where the coefficients $a$, $b$, $c$, and $d$ are all constants. The nominal solution vector is $x_0(t) = [x_{01}(t) \ x_{02}(t) \ x_{03}(t) \ x_{04}(t) \ x_{05}(t) \ x_{06}(t)]^T$.

### 6.4.2 Solution of Trajectory Sensitivity Equations

Recall that the trajectory sensitivity equation is,

$$\dot{S} = A_0 S + \left. \frac{\partial A}{\partial \alpha} \right|_{\alpha_0} x_0(t) + \left. \frac{\partial B}{\partial \alpha} \right|_{\alpha_0} u(t), \quad S(t_0) = 0.$$

(6.29)
In order to evaluate the second and the third terms on the right hand side of Equation 6.29, the following operation is conducted,

$$\frac{\partial (Ax)}{\partial \alpha} = \frac{\partial A}{\partial \alpha} x + \frac{\partial x}{\partial \alpha} A = \frac{\partial A(\alpha)}{\partial \alpha} x(t, \alpha) + \frac{\partial x(t, \alpha)}{\partial \alpha} A(\alpha).$$  \hspace{1cm} (6.30)

It is rewritten as,

$$\frac{\partial A(\alpha)}{\partial \alpha} x(t, \alpha) + \frac{\partial x(t, \alpha)}{\partial \alpha} A(\alpha) = \left[ \frac{\partial [A(\alpha)x(t, \beta)]}{\partial \alpha} + \frac{\partial [x(t, \alpha)A(\beta)]}{\partial \alpha} \right]_{\beta=\alpha}. \hspace{1cm} (6.31)$$

Expanding the term \(A(\alpha)x(t, \beta)\) gives a column vector,

$$L_v = \begin{bmatrix} A_{11}(\alpha)x_1(t, \beta) + A_{12}(\alpha)x_2(t, \beta) + \cdots + A_{16}(\alpha)x_6(t, \beta) \\ A_{21}(\alpha)x_1(t, \beta) + A_{22}(\alpha)x_2(t, \beta) + \cdots + A_{26}(\alpha)x_6(t, \beta) \\ \vdots \\ A_{61}(\alpha)x_1(t, \beta) + A_{62}(\alpha)x_2(t, \beta) + \cdots + A_{66}(\alpha)x_6(t, \beta) \end{bmatrix}. \hspace{1cm} (6.32)$$

The partial derivative of the column vector \(L_v\) with respect to vector \(\alpha\) is defined as the Jacobian matrix. Therefore, the second term in the state sensitivity Equation 6.29 is evaluated by,

$$\left. \frac{\partial A}{\partial \alpha} \right|_0 x_0(t) = \left. \frac{\partial (Ax_0(t))}{\partial \alpha} \right|_0. \hspace{1cm} (6.33)$$

Likewise, the third term in Equation 6.29 is evaluated by,

$$\left. \frac{\partial B}{\partial \alpha} \right|_0 u(t) = \left. \frac{\partial (Bu(t))}{\partial \alpha} \right|_0. \hspace{1cm} (6.34)$$

With the initial condition \(S(t_0) = 0\), the general solution of the trajectory sensitivity equation is,

$$S(t, \alpha_0) = \int_{t_0}^t e^{A_0(t-\tau)} \left[ \frac{\partial A}{\partial \alpha} x_0(\tau, \alpha_0) + \frac{\partial B}{\partial \alpha} u(\tau) \right]_{\alpha_0} d\tau. \hspace{1cm} (6.35)$$

For \(t_0 = 0\), the solution can be written as,

$$S(t, \alpha_0) = \int_0^t e^{A_0(t-\tau)} \left[ \frac{\partial [A x_0(\tau, \alpha_0)]}{\partial \alpha} + \frac{\partial [B u(\tau)]}{\partial \alpha} \right]_{\alpha_0} d\tau. \hspace{1cm} (6.36)$$
For the simplified sagittal plane model, the $6 \times 11$ trajectory sensitivity matrix $S$ is obtained by evaluating Equation 6.36. The complexity of the trajectory sensitivity function in the time domain with respect to the $j$th parameter $S_{ij} \equiv \frac{\partial x_i(\alpha)}{\partial \alpha_j} \bigg|_{\alpha_0}$ does not allow presentation of the closed-form expressions here, but the solutions are the combinations of exponential and trigonometric functions that are similar to the terms in the nominal solution vector $x_0(t)$ in Equation 6.28.

### 6.4.3 Solution of Output Sensitivity Equations

The output sensitivity equation is,

$$
\sigma = C_0 S + \frac{\partial C}{\partial \alpha} \bigg|_{\alpha_0} x_0(t) + \frac{\partial D}{\partial \alpha} \bigg|_{\alpha_0} u(t). \quad (6.37)
$$

Since the output matrix $C$ and the direct transmission matrix $D$ are not dependent on the parameter vector $\alpha$, the partial derivatives all go to zero. The output sensitivity equation becomes,

$$
\sigma(t, \alpha_0) = C_0 S, \quad (6.38)
$$

where $C_0 = C(\alpha_0) = C$. Therefore, the output sensitivity matrix $\sigma$ is $3 \times 11$, and it has the form,

$$
\sigma = \begin{bmatrix}
\frac{\partial \Delta x}{\partial x_0} & \frac{\partial \Delta x}{\partial x_1} & \frac{\partial \Delta x}{\partial x_2} & \cdots & \frac{\partial \Delta x}{\partial x_k} & \frac{\partial \Delta x}{\partial c_0} & \cdots & \frac{\partial \Delta x}{\partial c_4} \\
\frac{\partial \Delta z}{\partial x_0} & \frac{\partial \Delta z}{\partial x_1} & \frac{\partial \Delta z}{\partial x_2} & \cdots & \frac{\partial \Delta z}{\partial x_k} & \frac{\partial \Delta z}{\partial c_0} & \cdots & \frac{\partial \Delta z}{\partial c_4} \\
\frac{\partial \Delta \theta}{\partial x_0} & \frac{\partial \Delta \theta}{\partial x_1} & \frac{\partial \Delta \theta}{\partial x_2} & \cdots & \frac{\partial \Delta \theta}{\partial x_k} & \frac{\partial \Delta \theta}{\partial c_0} & \cdots & \frac{\partial \Delta \theta}{\partial c_4}
\end{bmatrix}_{\alpha_0}, \quad (6.39)
$$

where $\Delta$ indicates the relative displacement of the brain with respect to the skull.

### 6.5 Results of Sensitivity Analysis

Based on the simplified sagittal plane model that was validated against experimental data, the sensitivity analysis is evaluated at the nominal values, which are the model parameters previously selected in Section 3.5. The results using both impulse and sinusoid functions as inputs are presented in this section.
6.5.1 Results using Impulse Functions

With impulse functions for the linear head acceleration along the $X$ axis and the angular head acceleration about the $Y$ axis, the input vector is $u = [t \ u(t) \ 0 \ 0 \ t \ u(t)]^T$. Sensitivity analysis is performed using Equations 6.36 and 6.38. The results indicate that injury prediction with this analytical model is most sensitive to brain moment of inertia, followed by brain mass.

Figure 6.1 shows the sensitivity of the relative displacement on the $X$ axis to different model parameters with impulse inputs. The maximum absolute value for the sensitivity of the brain moment of inertia is about 0.5, while the maximum sensitivity for the brain mass is only about 0.01. This suggests that the relative brain displacement is much more sensitive to the brain moment of inertia than to the brain mass. It is also consistent with the structural coupling between brain rotation and translation. As shown in Figure 6.1, the sensitivities to the spring constants $k_i$ and damping constants $c_i$, $i = 1 \cdots 4$, all have relatively small magnitudes, although their magnitudes vary within a large range that is on the order of $10^{-7}$ to $10^{-5}$. This suggests that the relative brain displacements are insensitive to the selected spring and damping coefficients.

Figures 6.2 and 6.3 show the sensitivities of the relative brain displacement along the $Z$ axis and the relative brain rotation about the $Y$ axis to the model parameters with impulse inputs. The results also show that the relative displacements with the simple planar model are most sensitive to brain moment of inertia, followed by brain mass. The same trends are obtained although the magnitudes are different from those in Figure 6.1. The insensitivity to the spring and damping constants in all the cases indicates the robustness of the model in terms of parameter selection.
Sensitivity of $\Delta x$ to model parameters

Figure 6.1: Sensitivity of relative translation along the $X$ axis to the model parameters with impulse inputs. The most sensitive parameter is the brain mass moment of inertia followed by the brain mass. The model is insensitive to the selected spring and damping constants.
Figure 6.2: Sensitivity of relative translation along the $Z$ axis to the model parameters with impulse inputs. The most sensitive parameter is the brain mass moment of inertia followed by the brain mass. The model is insensitive to the selected spring and damping constants.
Figure 6.3: Sensitivity of relative rotation about the $Y$ axis to the model parameters with impulse inputs. The most sensitive parameter is the brain mass moment of inertia followed by the brain mass. The model is insensitive to the selected spring and damping constants.
6.5.2 Results using Sinusoidal Functions

Using sinusoidal functions as inputs, closed-form solutions are still available for the simplified brain injury model. To simplify the calculation, only a linear head acceleration along the X axis is used as input to perform the sensitivity analysis. Similar to the sinusoidal functions used in earlier FE work [6, 126], a sine function is used as the linear head acceleration along the X axis in this study. The acceleration is selected as $1000\sin(80\pi t)$ (m/s$^2$), which has relatively high magnitude. With this input, the relative brain displacements at the CG are about 10 mm along the X axis, 3 mm along the Z axis, and 10 degrees about the Y axis. Although the relative brain displacements are larger than those obtained with impulse inputs, they are still within a reasonable range.

Figure 6.4 plots the sensitivity of the relative displacement along the X axis to the model parameters with the sinusoidal input. The relative magnitudes of sensitivity indicate that the brain motion is most sensitive to brain moment of inertia, followed by brain mass. Figures 6.5 and 6.6 show the sensitivities of the relative brain displacement along the Z axis and the relative brain rotation about the Y axis to the model parameters. The same conclusions can be drawn, although the magnitudes differ from those in Figure 6.4.

It is shown that the results of sensitivity analysis are similar regardless of whether the inputs are impulse or sinusoid functions. Prange et al. [92] studied the influence of brain size and mechanical properties of the brain on pediatric brain injury using an FE model. They concluded that both brain size and material properties affect brain deformation, but brain size has greater influence. The results of the sensitivity analysis in this study agree with their findings. Prange et al’s [92] work also suggest that smaller brain size tends to introduce smaller strain in brain tissue. This is also supported by the results using the simple brain injury model with changing parameters.
Figure 6.4: Sensitivity of relative translation along the $X$ axis to the model parameters with sinusoid input. The most sensitive parameter is the brain mass moment of inertia followed by the brain mass. The model is insensitive to the selected spring and damping constants.
Figure 6.5: Sensitivity of relative translation along the $Z$ axis to the model parameters with sinusoid input. The most sensitive parameter is the brain mass moment of inertia followed by the brain mass. The model is insensitive to the selected spring and damping constants.
Figure 6.6: Sensitivity of relative rotation about the $Y$ axis to the model parameters with sinusoid functions. The most sensitive parameter is the brain mass moment of inertia followed by the brain mass. The model is insensitive to the selected spring and damping constants.
6.6 Finite Element Model

To verify the findings of the sensitivity analysis, an FE head model developed by Kleiven [57] is used to compare with the simple model. As shown in Figure 6.7, a detailed and parameterized FE model of the adult human head was created, including the scalp, skull, brain, meninges, CSF, and eleven pairs of parasagittal bridging veins. A simplified neck, including the extension of the brain stem to the spinal cord, the dura and pia mater, and the vertebrae, was also modeled.

![Finite element mesh of the human head](image)

Figure 6.7: Finite element mesh of the human head (from Kleiven’s work [57]).

To cope with the large elastic deformations, a Mooney-Rivlin hyperelastic constitutive law was used for the CNS tissues. This homogeneous, isotropic, non-linear and viscoelastic constitutive model was based on the rate-dependent Mooney-Rivlin constants and time decay constants derived by Mendis et al. [73]. The material properties were described in
more detail in [57] and are listed in Table 6.1. Based on the anatomy and physiology, the dura-skull interface was modeled with a tied node contact definition in LS-DYNA, which was used for the analysis and post-processing. Due to the presence of CSF, sliding contact definitions were used between the meningeal membranes and the brain, allowing sliding in the tangential direction and transferring tension or compression in the radial direction. Corresponding to approximately 120 ml of subdural and subarachnoidal CSF, the average thickness of the CSF layer was roughly 2 mm. For all of the sliding interfaces, the coefficient of friction was 0.2, as proposed by Miller et al. [74].

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Young’s Modulus (MPa)</th>
<th>Density (kg/dm³)</th>
<th>Poisson’s Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Outer table/face</td>
<td>15000</td>
<td>2.00</td>
<td>0.22</td>
</tr>
<tr>
<td>Inner table</td>
<td>15000</td>
<td>2.00</td>
<td>0.22</td>
</tr>
<tr>
<td>Diploe</td>
<td>1000</td>
<td>1.30</td>
<td>0.24</td>
</tr>
<tr>
<td>Neck bone</td>
<td>1000</td>
<td>1.30</td>
<td>0.24</td>
</tr>
<tr>
<td>Brain</td>
<td>Hyperelastic/viscoelastic</td>
<td>1.04</td>
<td>~0.5</td>
</tr>
<tr>
<td>CSF</td>
<td>$K = 2.1$ GPa $^a$</td>
<td>1.00</td>
<td>0.5</td>
</tr>
<tr>
<td>Sinuses</td>
<td>$K = 2.1$ GPa</td>
<td>1.00</td>
<td>0.5</td>
</tr>
<tr>
<td>Dura mater</td>
<td>31.5</td>
<td>1.13</td>
<td>0.45</td>
</tr>
<tr>
<td>Falx/tentorium</td>
<td>31.5</td>
<td>1.13</td>
<td>0.45</td>
</tr>
<tr>
<td>Pia mater</td>
<td>11.5</td>
<td>1.13</td>
<td>0.45</td>
</tr>
<tr>
<td>Scalp</td>
<td>16.7</td>
<td>1.13</td>
<td>0.42</td>
</tr>
<tr>
<td>Bridging veins</td>
<td>$EA = 1.9$ N $^b$</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

$^aK$, bulk modulus
$^bEA$, force/unit strain

Table 6.1: Material properties used in the FE head model in Kleiven’s work [57].

This model has been validated against experimental pressure data and relative brain-skull motion data in previous studies [59, 60]. A more comprehensive correlation between
the FE model output and the relative motion between the human brain and skull in anatomical X, Y, Z components has been demonstrated for three impact directions: frontal, occipital, and lateral [58]. Using this well-established FE model, several recent studies investigated the influence of direction and duration of impact on the brain injury prediction [55, 56] and evaluated proposed injury criteria such as HIC and HIP [57].

The parameterized nature of the model allows the scaling of particular dimensions to study their effects on the output. The spatial coordinates of points on the boundaries of different tissues can be scaled individually. The overall size of the skull, the brain, and the neck can be adjusted, as well as the mesh density to ensure sufficient mesh resolution. In this study, the brain of the FE model was scaled independently to create models having different brain size. By increasing or decreasing the thickness of the subarachnoid space filled with CSF depending on the change in brain size, the overall inertia properties of the head remain nearly constant. Simulations with various brain masses and moments of inertia were run while other parameters and inputs remained the same to study the effect of the parameter changes on the outputs.

6.7 Results of Comparison

Five FE models with different brain masses and moments of inertia were created for tests C755-T2 and C383-T1 in Hardy et al.’s work [41], while the other model parameters remained the same. Tests C755-T2 and C383-T1 were adopted since they consisted of different impact scenarios. C755-T2 was an occipital acceleration impact, and C383-T1 a frontal deceleration impact. The same head kinematics are used as inputs for each test with different brain sizes. The brain masses and moments of inertia of the FE models in different simulation cases are listed in Table 6.2. The brain was scaled down by 2.5% for case sc98,
5% for case sc95, 7.5% for case sc93, and 10% for case sc90, compared with the full brain size for case sc100. Table 6.2 summarizes the parameters used for different cases in both the analytical and FE models. Note that only those parameters in the sagittal plane are included for the simple planar model. The estimated brain mass moment of inertia (0.0038 kg·m²) in the sagittal plane for the planar model is close to that (0.0030 kg·m²) calculated in the FE model.

<table>
<thead>
<tr>
<th>Simulated Cases</th>
<th>Brain Mass ( m_b ) (kg)</th>
<th>Brain Moment of Inertia ( I_{xx}) (kg·m²)</th>
<th>( I_{yy}) (kg·m²)</th>
<th>( I_{zz}) (kg·m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>sc100</td>
<td>1.39</td>
<td>0.0023</td>
<td>0.0030</td>
<td>0.0033</td>
</tr>
<tr>
<td>sc98</td>
<td>1.29</td>
<td>0.0021</td>
<td>0.0026</td>
<td>0.0029</td>
</tr>
<tr>
<td>sc95</td>
<td>1.20</td>
<td>0.0018</td>
<td>0.0023</td>
<td>0.0026</td>
</tr>
<tr>
<td>sc93</td>
<td>1.12</td>
<td>0.0016</td>
<td>0.0021</td>
<td>0.0023</td>
</tr>
<tr>
<td>sc90</td>
<td>1.05</td>
<td>0.0014</td>
<td>0.0019</td>
<td>0.0021</td>
</tr>
</tbody>
</table>

Table 6.2: Simulated cases with decreased brain size for the FE and planar models.

The planar model was also run for the different cases, and the results were plotted and compared with those obtained from the FE models. Figure 6.8 indicates that the maximum relative brain displacement decreases with decreased brain mass and moment of inertia for both tests. This can be explained by the lag of brain motion with respect to the skull [41]. A brain of smaller mass and moment of inertia tends to track the motion of the skull more easily and quickly. For test C755-T2 using the FE model, the maximum relative displacement of the brain decreases from 4.39 mm in case sc100 to 3.52 mm in case sc90. Similar results were obtained using the planar model which gives a maximum relative displacement of 4.58 mm in sc100 and 3.70 mm in sc90. The FE model and the planar model have very close brain relative displacements for test C755-T2 with various brain masses and moments.
of inertia. For test C383-T1 using the FE and planar models, the results also have similar trends except the difference of the relative displacement is slightly larger. Reasonably close agreements are observed in these simulations.

Figure 6.8: Decreased brain motion with decreased brain mass and moment of inertia. The results from the planar and FE models are very close for tests C755-T2 and C383-T1, which were under an occipital acceleration impact and a frontal deceleration impact, respectively.

For each test, the decreasing rate of maximum relative displacement with decreased brain mass and moment of inertia for the FE model is very close to that obtained from the planar model. For test C755-T2 and test C383-T1, the slopes of the curves in Figure 6.8 are also very close. For these two low-severity impact tests, the planar and FE models present similar results of sensitivity to model parameters. With the closed-form solution available, the planar brain injury model predicts this result analytically, while the more complex FE model is capable of evaluating the variability in the injury predictions due to the variability in those critical parameters. The planar model can provide guidelines for similar analysis based on FE models. From the sensitivity analysis of the simple planar
model, the relative brain displacement is more sensitive to brain moment of inertia than to brain mass. Although these simulation results could not show the individual effects of brain mass and brain moment of inertia, it is believed that the majority of the effect is from the change of brain moment of inertia, which is indicated by the planar brain injury model.

6.8 Discussion

Based on the simplified sagittal plane model developed earlier in this work, sensitivity analysis was performed to identify critical parameters to which the injury prediction are most sensitive. Followed by the brain mass, the brain moment of inertia was found to be the parameter to which the relative brain displacements is most sensitive. A high-fidelity FE head model was used to verify the findings. With various brain masses and moments of inertia, the results from the FE and planar models were consistent.

The results indicate that the relative brain displacement decreases with decreased brain mass and moment of inertia for both the planar and FE models. This can be explained by the lag of brain motion with respect to the skull during impact. A quicker response for the brain with smaller size results in smaller relative brain displacement within the skull. The decreased relative motion is supported by Holbourn’s scaling law [85] but in contrast to some previously published results using FE models[60]. The conflict among the FE models suggests that more effort is needed to define boundary conditions and material properties for the FE models for better injury study.

Recent FE studies predict brain injuries mostly based on brain responses at the tissue level, like strain, stress, and strain rate [111, 128]. The results obtained using Kleiven’s [57] FE model for the cases in Table 6.2 suggest that the strain in the brain tissue also decreases with decreased brain size. This is consistent with the decreased relative brain displacement
and corresponds to lower chance of brain injury. The simple measure of strain proposed earlier using the planar model gives similar results. Prange et al’s study [92] also suggests that smaller strain in the brain tissue results from smaller brain size.

Although the simple brain injury model provides important insight via the sensitivity analysis, it only contains limited model parameters due to the structural simplicity. The sensitivity of injury prediction to other parameters, such as brain material properties and brain-skull contact definition, is not currently available. For the FE model, the change in brain size always resulted in the change in both the brain mass and brain moment of inertia. To study their individual effects on brain responses, changes in brain mass and brain moment of inertia independently should be made once such FE models are available.

6.9 Conclusions

This chapter performs sensitivity analysis based on the brain injury model in the sagittal plane to determine the parameters to which injury prediction is most sensitive. The sensitivity analysis indicates that the relative brain displacement is most sensitive to the brain moment of inertia followed by the brain mass in low-severity impacts. It is consistent with the structural coupling between the brain translation and rotation. To verify the findings of the sensitivity analysis using the planar model, a number of simulations with various brain sizes were executed on a high-fidelity FE head model, whose parameterized nature allows the scaling of the brain to particular dimensions. It was found that the relative brain displacement decreases with decreased brain mass and moment of inertia, and they are very close in the FE and planar models. This suggests that brain mass moment of inertia, primarily, and brain mass, secondarily, should be varied in focused experimental
and computational modeling work to ensure that conclusions are not drawn from individual data points at which injury predictions are highly sensitive to small parameter changes.
CHAPTER 7

SUMMARY AND FUTURE WORK

7.1 Summary

To bridge the gap between simplified models, such as the HIC criterion, that predict head injuries based only on linear or angular head accelerations and more complex FE models, such as the SIMon FE head model, that require complete knowledge of material properties, new lumped-parameter models were proposed in this work. Based on knowledge from the literature, the model development operated from a fundamental assumption that the linear and angular movements of the brain with respect to the skull are coupled. The model validation was based on experimental brain motion data under low-severity impacts. With proposed injury metrics, the simple models were applied to predict brain injuries under more severe crash tests. A simple 3D model was developed by combining the planar models and was applied to real-world car-pedestrian accidents for crash reconstruction.

The brain motion patterns within the skull were studied using the experimental brain motion data from a total of six tests on two specimens in Hardy et al’s [41] work. The NDTs were implanted in two columns, anterior and posterior, located in the temporoparietal region and the occipitoparietal region. The relative brain motion was measured by tracking the NDTs with a high-speed x-ray system. An analytical method was used to separate the
measured brain motion into rigid body displacement and local deformation. It was found that the whole brain has nearly pure rigid body displacement at low impact speed. As the impact becomes more severe, the increased brain motion primarily is due to deformation while the rigid body displacement is limited in magnitude for both translation and rotation. Under low-severity impacts in the sagittal plane, the rigid body brain translation has a magnitude of 4 to 5 mm, and the whole brain rotation is on the order of ±5 degrees.

Similarly, local brain motion patterns were also analyzed using the two NDT columns separately. It was found that each column individually follows closely a rigid body displacement in both the sagittal and coronal planes in all six tests. As the impact becomes more severe, however, relative motion becomes larger between the two columns, introducing strain deformation, which is dominant in the superior region of the brain. By separating the measured brain motion into rigid body displacement and brain deformation, the brain motion patterns are better understood through this study.

Based on the results of the experimental data analysis, two simple brain injury models were developed in the sagittal and coronal planes. Each planar model consists of a circular skull and brain, both having masses and moments of inertia. The skull and brain are connected by four Kelvin elements, each having a spring and a damper in parallel. By assuming that the brain has the simple geometry of one half an ellipsoid or a full ellipsoid, the brain moment of inertia was estimated because it is unavailable in the literature. The spring and damping coefficients were selected to best match the experimental data of relative brain motion within the skull. The results show that the planar brain injury models are capable of capturing the key characteristics of relative brain motion with reasonable accuracy. The two planar models can be applied to study brain responses when planar head kinematics are dominant, such as in NHTSA’s frontal and side impact tests.
A simple measure of strain for DAI prediction was proposed based on the planar brain injury models. The material properties of the brain tissue were selected to match the strain calculations with the severity of the impacts. The proposed strain measure was evaluated in low-severity impact tests and more severe frontal and side impact tests. The maximum shear strains were calculated for 43 frontal crash tests and 42 side impact tests. The results were compared with the HIC values and the results from the SIMon FE model. The results suggest that the simple measure of strain has the capability of predicting brain injuries under impacts. The maximum shear strain from the planar models can be used as a critical element for brain injury prediction.

A simple 3D brain injury model was developed by combining the two planar models so that 3D head kinematics could be used as inputs to have a more comprehensive study. Injury metrics were proposed for predicting various brain injuries under more general impact scenarios, such as car-pedestrian crashes. A total of ten car-pedestrian accidents were selected for crash reconstructions using both the simple 3D model and the SIMon FE model. The head accelerations were calculated from MADYMO simulations of these accidents. The results from the simple 3D model and SIMon were compared with documented head injuries in the accident reports. Results show that the simple 3D model is capable of predicting the ASDH and DAI injuries in car-pedestrian crashes.

Sensitivity analysis was performed to determine the parameters to which injury prediction is most sensitive. Using the closed-form solutions of the simplified sagittal plane model, the sensitivity analysis was performed analytically with simple inputs, such as impulse and sinusoid functions. The results indicate that the relative brain displacement is most sensitive to the brain moment of inertia followed by the brain mass in low-severity impacts. A high-fidelity FE head model developed by Kleiven [57] was used to verify the
findings by running a series of simulations with changing parameters. It was found that the relative brain displacement decreases with decreased brain mass and brain moment of inertia, and the results are very close in the FE and planar models.

### 7.2 Recommendations for Future Work

This work addresses the analysis and modeling of the biomechanics of brain injury under impact. The brain motion patterns have been better understood via experimental data analysis, and simple brain injury models have been developed for injury studies under impacts of various severities. This section recommends a number of extensions in both the experimental tests and mathematical modeling work.

#### 7.2.1 Recommendations for Experimental Work

The experimental data used in this study was collected under either frontal or occipital impact. Therefore, the measured head kinematics and brain motion in the sagittal plane were dominant. To have a more comprehensive understanding of the brain motion patterns, more experimental data under impacts from other directions, such as the lateral direction, is needed. Although there is one lateral test used in Kleiven and Hardy’s work [58], more data from similar tests is necessary to show the consistency of the results.

In Hardy et al’s six tests [41] used in the present work, the NDTs were located in two columns, representing the temporoparietal region and occipitoparietal region. Although the NDTs occupy a considerably large area of the brain, the brain motions in the areas without NDTs are still unknown, particularly in the frontal area and near the brain-skull interface. A better understanding of the brain motion patterns at the boundary will help define the brain-skull interface for mathematical modeling work.
In all six tests, the spacing of two adjacent NDTs ranges from 7 to 12 mm, while the distance between the two columns is about 50 to 70 mm. Therefore, this set of experimental data could not be used to calculate strain deformation. To provide information of strain distribution in the brain, the NDTs should have much smaller spacing between them. A large number of clusters of three NDTs can be implanted in a large area of brain tissue so that local strain deformation at the location of each cluster can be directly calculated. The strain distribution in the brain can be obtained by collecting strain information at all locations with NDTs implanted.

Although the impacts were all from either frontal or occipital directions in all six tests, they generated both linear and angular accelerations of the head. It is very difficult to separate the individual contributions of linear and angular accelerations to brain motion. Well-controlled experiments can be conducted, allowing the head to only rotate or translate. In this case, the relative brain motion with respect to the skull can be directly observed under pure linear or angular head accelerations. Furthermore, the coupling between brain rotation and translation can also be studied.

The six tests used in the present work were all under low-severity impacts with impact speeds between 2.5 and 3.5 m/s. Although each cadaver head could be repeatedly used for multiple tests under these low-speed impacts, more experimental data under high-speed impacts will help better understand the brain motion patterns. Using the same method, regional brain motions can be separated into rigid body displacement and brain deformation so that relative motion between different areas can be obtained to locate large deformations in the brain under severe impacts.
7.2.2 Recommendations for Modeling Work

Two planar models were developed in the sagittal and coronal planes. The results indicate that the planar models were capable of capturing the key characteristics of brain motion when in-plane head kinematics were dominant. Due to the lack of experimental data, the development of a similar model in the horizontal plane cannot be implemented. Once experimental data is available, a horizontal plane model can be developed and validated in a similar manner to the existing models. This new horizontal plane model can be easily integrated into the current 3D model to accept full 3D head kinematics as inputs. The scheme of strain measure has been readily constructed for the full 3D model.

In the planar models, the four Kelvin elements were all arranged to make a 45-degree angle with the horizontal at the neutral position to have the strongest coupling between the brain translation and rotation. The orientations of the Kelvin elements can be altered to study the effects on the responses of the simple models under impacts. They can be treated as additional model parameters, which also need to be selected to best match the measured brain motion.

One of the limitations of the current models is that the relative motion at the brain boundary is always the largest due to the rigid body displacement of the brain. Research has shown that the relative motion at the brain-skull interface is very limited because of the anatomical structure [41, 131]. The models in the present work can be modified such that the brain will have relatively small motion on the boundary. This extension may require the employment of a deformable brain instead of the rigid brain. Multiple layers may also be used to construct the brain tissue.

In the present work, the relative brain rotation was proposed as the indicator of the ASDH injury. For the 3D model, the relative brain rotations were calculated separately in
the sagittal and coronal planes. A simple scheme can be proposed to combine the two calculations into one for ASDH prediction. In addition, different injury metrics may be used to compare with the currently proposed metric, such as the maximum resultant displacement on the brain surface, including both brain translation and rotation.

The maximum shear strain proposed for DAI prediction in the current work was calculated only at the center of the spherical brain. In more recent FE studies, the strain contributions in the whole brain were calculated. In particular, CSDM in the SIMon FE model is a cumulative measure of strain in the brain for DAI prediction. The present work can be extended to calculate strain contributions for a better study of both diffuse and focal brain injuries.

The simple models in the current work had two injury metrics proposed for predictions of ASDH and DAI injuries, but no injury metric has been proposed to study cerebral contusion, which is one of the three major types of brain injuries under impact. The current models can be extended with a simple measure of pressure for the study of cerebral contusion since cerebral contusions are related to the changes in intracranial pressure. An impact model can be developed to study the pressure distribution when the brain strikes the skull.

The direct inputs of the current models are the head accelerations measured using accelerometer packages, which are assumed to be rigidly fixed on the skull. The current models can be extended with the addition of a neck so that not only head kinematics but also external loads can be directly accepted as inputs. The head accelerations, even the skull deformations, can be calculated with known impact parameters such as impact angle and velocity. The brain kinematics and therefore, relative brain motion, can be obtained in a similar manner to the present work.
7.3 Conclusions

This dissertation found that the brain has nearly pure rigid body displacement under low-speed impact. As the impact speed increases, the increased brain motion primarily is due to brain deformation, while the rigid body displacement is limited in magnitude for both translation and rotation. Under low-severity impacts, the rigid body brain translation has a magnitude of 4 to 5 mm, and the whole brain rotation is on the order of ±5 degrees. Simple brain injury models were developed and validated against the experimental brain motion data. The simple models were applied to more severe frontal and side impact tests and real-world car-pedestrian accidents for brain injury study. The results show that the proposed injury metrics are capable of predicting various brain injuries under impact. Sensitivity analysis was performed, and the results indicate that brain injury prediction is most sensitive to the brain moment of inertia, followed by the brain mass.
APPENDIX A

LINEAR VISCOELASTIC BRAIN TISSUE

Human brain tissue is highly incompressible, with a bulk modulus of approximately 2 GPa [106, 71], which is similar to the bulk modulus of water. Holbourn [42] pointed out that the brain would deform in shear most easily because the shear modulus is low compared with the bulk modulus. This section summarizes two commonly used mathematical models for linear viscoelastic brain tissue: the Kelvin model and the standard linear solid (SLS) model.

A.1 Kelvin Model

A.1.1 Constitutive Law

As shown in Figure A.1, a Kelvin material, also called Voigt material, is the simplest constitutive representation for a linear viscoelastic solid [28]. Many analytical models used this two-element Kelvin material to study brain injuries under various loading conditions [12, 27, 67].

Figure A.1 shows that the Kelvin model has an elastic element, or spring, and a viscous element, or damper, in parallel. This model has equal strain in the two elements, while the total stress is distributed in the spring and damper. This leads to,

\[ \epsilon_E = \epsilon_\eta = \epsilon, \]  

(A.1)
where $\epsilon_E$ is the strain in the spring and $\epsilon_\eta$ is the strain in the damper. The total stress in the Kelvin model is the sum of those in the spring and damper,

$$\sigma = \sigma_E + \sigma_\eta,$$

(A.2)

where $\sigma_E = E\epsilon_E$ and $\sigma_\eta = \eta\dot{\epsilon}_\eta$. Substituting $\sigma_E$ and $\sigma_\eta$ into Equation A.2 and applying Equation A.1 gives the stress-strain equation for the Kelvin model,

$$\sigma = E\epsilon + \eta\dot{\epsilon}.$$  

(A.3)

A.1.2 Experimental Measurements

Viscoelastic material properties are often experimentally measured under periodic oscillations. The stress and strain are both harmonic function of time under periodic loading with frequency $\omega$ (rad/s). Using complex representation, the stress and strain can be written as $\epsilon = \dot{\epsilon}e^{i\omega t}$ and $\sigma = \dot{\sigma}e^{i\omega t}$. Taking the derivative with respect to $t$ gives $\dot{\epsilon} = i\omega\dot{\epsilon}e^{i\omega t} = i\omega\epsilon$ and $\dot{\sigma} = i\omega\dot{\sigma}e^{i\omega t} = i\omega\sigma$. Applying these results to Equation A.3 yields,

$$\sigma = E\epsilon + i\omega\eta\epsilon = E(i\omega)\epsilon,$$

(A.4)

where the complex modulus of elasticity $E(i\omega) = E + i\omega\eta$. It is often written as $E(i\omega) = E' + iE''$, where $E'$ is the storage modulus and $E''$ is the loss modulus. The material
properties can be measured under periodic loadings with various frequencies. Experimental
data of brain properties are summarized in Table A.1.

<table>
<thead>
<tr>
<th>Authors</th>
<th>Specimen</th>
<th>Properties</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Koeneman [61]</td>
<td>Rabbit, rat and pig</td>
<td>Modul. = 1.2-2.2 psi (elastic) Visco. = 35-44 Poise</td>
<td>Creep and cyclic compression, 80-350 Hz</td>
</tr>
<tr>
<td>Ommaya [83]</td>
<td>Rhesus monkey</td>
<td>Visco. = 407 Poise</td>
<td>Falling sphere viscometry</td>
</tr>
<tr>
<td>Fallenstein et al. [25]</td>
<td>Human</td>
<td>Comp. shear modul. $G_1 = 0.08-0.16$ psi $G_2 = 0.04-0.095$ psi</td>
<td>Dynamic simple shear and indentation, 10 Hz</td>
</tr>
<tr>
<td>Galford and McElhaney [31]</td>
<td>Human</td>
<td>Comp. Young’s modul. $E_1 = 9.68$ psi $E_2 = 3.80$ psi</td>
<td>Free vibration, 34 Hz</td>
</tr>
<tr>
<td></td>
<td>Monkey</td>
<td>$E_1 = 13.2$ psi $E_2 = 7.8$ psi</td>
<td>Free vibration</td>
</tr>
<tr>
<td>Wang and Wineman [117]</td>
<td>Human</td>
<td>Comp. Young’s modul. $E_1 = 8.5$ psi $E_2 = 4.9$ psi</td>
<td>Data analysis from [25]</td>
</tr>
<tr>
<td>Shuck et al. [102]</td>
<td>Human</td>
<td>Comp. shear modul. $G_1 = 0.12-0.20$ psi $G_2 = 0.05-0.12$ psi</td>
<td>Dynamic torsional shear, 2-350 Hz</td>
</tr>
</tbody>
</table>

Table A.1: Experimental measurements of brain material properties.

In particular, Shuck et al. [103] and Shuck and Advani [102] measured values of the storage and loss components of the dynamic shear modulus of in vitro human brain tissue up to 350 Hz. Bycroft [12] fitted the experimental data using a two-element Kelvin model and found the constitutive properties of human brain tissue with shear modulus $G = 13.8$ kPa and dynamic viscosity $\mu = 34.5$ Pa·sec (1 Pa·sec = 10 Poise). The same shear modulus and viscosity were also used by Firoozbakhsh and DeSilva [27] and Margulies and Thibault [67] to study brain injury using analytical models subject to torsional loadings.
A.2 Standard Linear Solid Model

A.2.1 Constitutive Law

Some recent FE head models used a SLS material to study brain responses under impacts [96, 127, 111]. Figure A.2 shows a three-parameter SLS model often employed to model biological tissues. Unlike the Kelvin model, SLS has the capability of capturing impact response. In other words, $\sigma(0) \neq \infty$ in SLS under sudden input of strain. Also unlike the Maxwell model having fluid behavior, SLS has non-zero relaxation at equilibrium, i.e., $\sigma(\infty) \neq 0$.

![Figure A.2: Schematic of a standard linear solid.](image)

Since the Kelvin model $(E_1, \eta)$ and the spring $(E_2)$ are in series, the stress in both parts are equal at equilibrium. This gives,

$$\sigma = \sigma_{E_2} = \sigma_{Kelvin}, \quad (A.5)$$

where $\sigma_{E_2}$ is the stress in the spring on the left and $\sigma_{Kelvin}$ is the stress in the Kelvin model.

The total strain in the SLS is the sum of that in both units, i.e.,

$$\epsilon = \epsilon_{E_2} + \epsilon_{Kelvin}. \quad (A.6)$$
This yields,
\[
\dot{\epsilon} = \dot{\epsilon}_E + \dot{\epsilon}_{\text{Kelvin}},
\]  
(A.7)

where in the spring on the left, \( \dot{\epsilon}_E = \frac{\dot{\sigma}}{E_2} \) since \( \epsilon_E = \frac{\sigma}{E_2} \). From Equation A.3, the stress-strain relationship in the Kelvin unit is given by,
\[
\sigma_{\text{Kelvin}} = E_1 \epsilon_{\text{Kelvin}} + \eta \dot{\epsilon}_{\text{Kelvin}}.
\]  
(A.8)

Substituting into Equation A.7 gives,
\[
\dot{\epsilon} = \frac{\dot{\sigma}}{E_2} - \frac{E_1}{\eta} \epsilon_{\text{Kelvin}} + \frac{\sigma}{\eta}.
\]  
(A.9)

From Equation A.6, \( \epsilon_{\text{Kelvin}} = \epsilon - \epsilon_E \). Applying this to Equation A.9 yields,
\[
\dot{\epsilon} = \frac{\dot{\sigma}}{E_2} - \frac{E_1}{\eta} \epsilon + \frac{E_1}{\eta} \epsilon_E + \frac{\sigma}{\eta}.
\]  
(A.10)

Since \( \sigma = E_2 \epsilon_E \), the stress-strain equation of the SLS model is,
\[
\dot{\epsilon} = \frac{\dot{\sigma}}{E_2} + \left(1 + \frac{E_1}{E_2}\right) \frac{\sigma}{\eta} - \frac{E_1}{\eta} \epsilon.
\]  
(A.11)

Rearranging Equation A.11 gives,
\[
\sigma + \frac{\eta}{E_1 + E_2} \dot{\sigma} = \frac{E_1 E_2}{E_1 + E_2} \epsilon + \frac{\eta E_2}{E_1 + E_2} \dot{\epsilon}.
\]  
(A.12)

Equation A.12 has the form,
\[
\sigma + p_1 \dot{\sigma} = q_0 \epsilon + q_1 \dot{\epsilon},
\]  
(A.13)

where \( p_1 = \frac{\eta}{E_1 + E_2} \), \( q_0 = \frac{E_1 E_2}{E_1 + E_2} \), and \( q_1 = \frac{\eta E_2}{E_1 + E_2} \).

**A.2.2 Stress Relaxation**

To study the property of stress relaxation, a constant strain \( \epsilon_0 \) is applied to the SLS model at \( t = 0 \). With the strain is applied instantaneously, the initial stress \( \sigma(0) = E_2 \epsilon_0 \) in
the SLS model since only the spring unit responds immediately. When \( t > 0 \), the strain is constant and \( \frac{d\epsilon}{dt} = 0 \). Equation A.12 becomes,

\[
\dot{\sigma} + \frac{E_1 + E_2}{\eta} \sigma = \frac{E_1 E_2}{\eta} \epsilon_0.
\] (A.14)

Solving this first order differential equation with the initial condition gives,

\[
\sigma(t) = \epsilon_0 \left( \frac{E_2^2}{E_1 + E_2} e^{-\frac{E_1 + E_2}{\eta} t} + \frac{E_1 E_2}{E_1 + E_2} \right) = \epsilon_0 G(t),
\] (A.15)

where the relaxation function \( G(t) \) of a SLS model is often expressed as,

\[
G(t) = G_\infty + (G_0 - G_\infty) e^{-\beta t}.
\] (A.16)

Here, \( G \) indicates explicitly the shear modulus of the brain tissue in this study. \( G_0 \) is the short-term shear modulus, \( G_\infty \) is the long-term shear modulus, \( \beta \) is the decay constant, and \( t \) is the duration, where \( G_0 = E_2, G_\infty = \frac{E_1 E_2}{E_1 + E_2}, \) and \( \beta = \frac{E_1 + E_2}{\eta} \). Knowing the short-term shear modulus \( G_0 \), long-term shear modulus \( G_\infty \), and decay constant \( \beta \), the coefficients of the SLS model are given by \( E_1 = \frac{G_0 G_\infty}{G_0 - G_\infty}, E_2 = G_0, \) and \( \eta = \frac{G_\infty^2}{\beta (G_0 - G_\infty)} \). It is also found that \( G_0 = \frac{q_1}{p_1}, G_\infty = q_0, \) and \( \beta = \frac{1}{p_1} \). Therefore, any set of values \((G_0, G_\infty, \beta), (E_1, E_2, \eta),\) or \((p_1, q_0, q_1)\) fully describes the stress relaxation property of an SLS model.
APPENDIX B

NINE ACCELEROMETER PACKAGE

The nine accelerometer package installed in the dummy head provides nine linear acceleration time histories, which can be applied to calculate three orthogonal angular accelerations in an appropriate body-fixed frame. Padgaonkar et al. [88] arranged the 3-2-2-2 accelerometer array by installing three at the CG of the head, two on the top, two in the front, and two in the left side. The distances, called arm lengths, between the mounting positions to the CG are used to calculate the angular accelerations.

Figure B.1 shows the inertial reference frame $XYZ$ and the body-fixed coordinate system $xyz$. The origin of the body-fixed system is $O$. Nine accelerations are measured in the frame. The arm lengths are $r_1$, $r_2$, and $r_3$.

The acceleration of any point $p$ (not shown in the figure) on a rigid body is given by,

$$\vec{A}_p = \vec{R} + \vec{a} + 2 \times \vec{\omega} \times \vec{v} + \vec{\omega} \times (\vec{\omega} \times \vec{r}_p) + \vec{\ddot{\omega}} \times \vec{r}_p,$$  \hspace{1cm} (B.1)

where $\vec{R}$ is the relative acceleration of the body-fixed frame with respect to the inertial reference frame, $\vec{a}$ is the acceleration of point $p$ in the body-fixed frame, $\vec{\omega}$ is the angular velocity of the body, $\vec{\ddot{\omega}}$ is the angular acceleration of the body, $\vec{v}$ is the velocity of point $p$ in the body-fixed frame, and $\vec{r}_p$ is the position vector of point $p$ from the origin of the body-fixed frame. For a rigid body, $\vec{a}$ and $\vec{v}$ are assumed to be zero. The acceleration of
Figure B.1: Configuration of nine accelerometer package.

point \( p \) then becomes,

\[
\vec{A}_p = \vec{R} + \vec{\omega} \times (\vec{\omega} \times \vec{r}_p) + \vec{\omega} \times \vec{r}_p.
\]  

(B.2)

Therefore, the accelerations at points 1, 2, and 3 on the arms are given by,

\[
\vec{A}_1 = \vec{R} + \vec{\omega} \times (\vec{\omega} \times \vec{r}_1) + \vec{\omega} \times \vec{r}_1,
\]  

(B.3)

\[
\vec{A}_2 = \vec{R} + \vec{\omega} \times (\vec{\omega} \times \vec{r}_2) + \vec{\omega} \times \vec{r}_2,
\]  

(B.4)

\[
\vec{A}_3 = \vec{R} + \vec{\omega} \times (\vec{\omega} \times \vec{r}_3) + \vec{\omega} \times \vec{r}_3,
\]  

(B.5)

where,

\[
\vec{R} = x_0i + y_0j + z_0k,
\]

\[
\vec{\omega} = \omega_xi + \omega_yj + \omega_zk,
\]
\[ \vec{\omega} = \dot{\omega}_x \hat{i} + \dot{\omega}_y \hat{j} + \dot{\omega}_z \hat{k}, \]
\[ \vec{r}_1 = r_1 \hat{j}, \]
\[ \vec{r}_2 = r_2 \hat{i}, \]
\[ \vec{r}_3 = r_3 \hat{k}. \]

Expanding the equation for \( \vec{A}_1 \) and comparing the components yields,
\[ \dot{\omega}_x = (\ddot{z}_1 - \ddot{z}_0) / r_1 - \omega_y \omega_z, \]
\[ \dot{\omega}_z = - (\ddot{x}_1 - \ddot{x}_0) / r_1 + \omega_x \omega_y. \] (B.6)

Similarly, the equations for \( \vec{A}_2 \) and \( \vec{A}_3 \) give,
\[ \dot{\omega}_y = -(\ddot{z}_2 - \ddot{z}_0) / r_2 + \omega_x \omega_z, \]
\[ \dot{\omega}_z = (\ddot{y}_2 - \ddot{y}_0) / r_2 - \omega_x \omega_y, \] (B.7)

and,
\[ \dot{\omega}_x = -(\ddot{y}_3 - \ddot{y}_0) / r_3 + \omega_y \omega_z, \]
\[ \dot{\omega}_y = (\ddot{x}_3 - \ddot{x}_0) / r_3 - \omega_x \omega_z. \] (B.8)

Combining the results in Equations B.6, B.7, and B.8 and canceling the angular velocity terms gives,
\[ \dot{\omega}_x = (\ddot{z}_1 - \ddot{z}_0) / (2r_1) - (\ddot{y}_3 - \ddot{y}_0) / (2r_3), \]
\[ \dot{\omega}_y = (\ddot{x}_3 - \ddot{x}_0) / (2r_3) - (\ddot{z}_2 - \ddot{z}_0) / (2r_2), \]
\[ \dot{\omega}_z = (\ddot{y}_2 - \ddot{y}_0) / (2r_2) - (\ddot{x}_1 - \ddot{x}_0) / (2r_1). \] (B.9)

Given nine linear accelerations, the three angular accelerations can be calculated using the formulas in Equation B.9. Theoretically, six linear accelerations would be sufficient.
to derive three angular accelerations. However, Padgaonkar et al. [88] indicated that the accumulation of error will significantly reduce the accuracy of the measurement. By using three additional linear accelerations, the angular acceleration at each time step does not depend on the previous values, so it gives more accurate results.
APPENDIX C

INJURY RISK FUNCTIONS

C.1 Risk Functions of Skull Fracture

Prasad and Mertz [93] originally developed the risk curve of skull fracture to relate the HIC values with the probability of AIS 4+ skull fracture using the data from cadaver experiments. Based on this curve, a set of curves, called expanded Prasad/Mertz curves, were developed to predict skull fracture at various injury scales [77]. The formulas for the injury probability of different degrees are given as a function of HIC.

\[
P_{AIS1} = \left(1 + e^{1.54 + \frac{200}{HIC} - 0.00650 \times HIC}\right)^{-1},
\]
\[
P_{AIS2} = \left(1 + e^{2.49 + \frac{200}{HIC} - 0.00483 \times HIC}\right)^{-1},
\]
\[
P_{AIS3} = \left(1 + e^{3.39 + \frac{200}{HIC} - 0.00372 \times HIC}\right)^{-1},
\]
\[
P_{AIS4} = \left(1 + e^{4.90 + \frac{200}{HIC} - 0.00351 \times HIC}\right)^{-1},
\]
\[
P_{AIS5} = \left(1 + e^{7.82 + \frac{200}{HIC} - 0.00429 \times HIC}\right)^{-1},
\]
\[
P_{AIS6} = \left(1 + e^{12.24 + \frac{200}{HIC} - 0.00565 \times HIC}\right)^{-1},
\] (C.1)

where \( P_{AIS} \) denotes probability of injury at various degrees.
Corresponding to the brain injury measures of RMDM, DDM, and CSDM in SIMon FE head model, the injury risk curves of ASDH, cerebral contusion, and DAI were plotted in Takhounts et al’s work [111]. The formulas for the injury probability of ASDH, cerebral contusion, and DAI as a function of RMDM, DDM, and CSDM are given by,

\[
\begin{align*}
    P_{ASDH} &= \left(1 + e^{4.908 - 4.904 \times RMDM}\right)^{-1}, \\
    P_{contusion} &= \left(1 + e^{2.149 - 30.011 \times DDM}\right)^{-1}, \\
    P_{DAI} &= \left(1 + e^{3.108 - 5.652 \times CSDM}\right)^{-1},
\end{align*}
\]

(C.2)

where the probability of DAI explicitly indicates injury at a strain level of 0.15.
Two brain injury models in the sagittal and coronal planes were developed in Chapter 4 and applied to NHTSA’s frontal and side impact tests to study brain responses under impact. The results were compared with the HIC criterion and the CSDM values from the SIMon FE model. Since only planar head kinematics were used in the planar models, this section applies the 3D brain injury model to NHTSA’s crash tests and compares the overall performances of the planar and 3D models. The 43 frontal and 42 side impact tests in Chapter 4 are used for the comparison. The test configurations are listed in Tables 4.7 and 4.10. All 85 crash tests are reconstructed using the 3D model to calculate the maximum shear strain. The results are compared with those obtained using the sagittal and coronal plane models alone.

Sorted by the maximum shear strain, Figures D.1-D.4 compare the maximum shear strain using the 3D model with the HIC values and the CSDM measure from SIMon. Note that linear elastic brain material is used in this section. Comparing Figures D.1 and D.2 with Figures 4.6 and 4.7 indicates that the 3D model captures the contributions of the out-of-plane head kinematics to the strain measurements, but the overall trends only change
Figure D.1: Comparison of HIC15 and maximum shear strain from the 3D model in frontal crash tests.

Figure D.2: Comparison of CSDM (0.15) from SIMon and maximum shear strain from the 3D model in frontal crash tests.
Figure D.3: Comparison of HIC15 and maximum shear strain from the 3D model in side impact tests.

Figure D.4: Comparison of CSDM (0.15) from SIMon and maximum shear strain from the 3D model in side impact tests.
slightly. In most of the side impact tests, the impact direction is not perfectly perpendicular to the vehicle, and there is a small oblique angle between the vehicle and impactor. Therefore, larger out-of-plane head kinematics result from side impact than from frontal impact, and a larger difference is obtained when comparing the trends under side impact using the planar and 3D models. By comparing Figures D.3 and D.4 with Figures 4.10 and 4.11, it is clear that the out-of-plane head kinematics make larger contributions to the strain measurement under side impact than under frontal impact in the 3D model. However, the overall trends using the planar and 3D models are similar. The improvement in the results using the 3D model is not significant because most of the frontal and side impact tests have dominant planar head kinematics. This also suggests that the planar models are adequate for these tests.
BIBLIOGRAPHY


