An Investigation into Pressure-Based Abdominal Injury Criteria Using Isolated Liver and Full-Body Post-Mortem Human Subject Impact Tests

Thesis

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By

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Abstract

Research has shown that abdominal injuries account for approximately 3-5% of all injuries that occur during motor vehicle collisions. However these injuries, especially to the solid organs of the abdomen like the liver, represent a much higher percentage of life threatening injuries. Research has suggested that in blunt liver trauma the mechanism of injury is linked to the rapid increase in internal pressure. Previous work also has shown a correlation between vascular pressure and liver injury in human surrogates and in pressurized *ex vivo* human and porcine livers when subjected to blunt impacts. The objective of this work is to further investigate the relationship between pressure and liver injury using full-body post-mortem human surrogates (PMHS) subjected to lateral and oblique impacts with boundary conditions more representative of motor vehicle collisions.

Specifically, the goals of this research were to (1) correct and re-analyze previous experimental work done by Sparks et al (2007) and Gustafson (2009); (2) continue work by Gustafson and complete an additional four rigid impacts to PMHS (for a total of n=10); (3) determine if a correlation exists between several pressure-related variables and liver injury from PMHS data and *ex vivo* work performed by Sparks et al; (4) compare the results to previously proposed biomechanical predictors of abdominal injury.

For the combined PHMS study, each PMHS was instrumented with pressure sensors in the abdominal vessels, these included: the abdominal aorta, the hepatic veins,
and the inferior vena cava. The subjects’ abdominal vessels were pressurized to physiological pressures using saline. Using a pneumatic ram, all subjects were impacted at approximately 7.0 m/s at the estimated level of the liver. Autopsies were conducted on each subject following the impacts to determine the severity of injury to the PMHS and data from the pressure sensors were used to develop injury risk functions correlating pressure to the documented abdominal injuries.

The liver injuries observed in the combined PMHS study (n=10) were similar to those documented in the Crash Injury Research Engineering Network (CIREN) trauma database. Injuries included four livers with serious burst injuries and three livers with superficial lacerations to the capsule. Using binary logistic regression to develop injury risk functions, it was determined the peak rate of pressure change (\( \dot{P}_{\text{max}} \)) was a statistically significant predictor of AIS ≥ 3 liver injury in both the PMHS and ex vivo testing. This suggests that \( \dot{P}_{\text{max}} \) is a good predictor for liver injury regardless of the impact boundary conditions. These results suggest that the peak rate of pressure change could be used in anthropomorphic test devices (ATDs) to predict abdominal injury.
Dedication

Dedicated to my family, especially my parents who are there to support me.
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Chapter 1 : Introduction

1.1 Motivation

Abdominal injuries due to motor vehicle crashes (MVCs) are important to study because they occur commonly and tend to be more life threatening than injuries to other regions of the body. Several studies have investigated the occurrences of abdominal injuries due to (MVCs (Ricci 1980; Bondy 1980; Rouhana and Foster 1985a; Elhagediab and Rouhana 1998; Augenstein et al 2000). From these studies, it was found that abdominal injuries made up only 3-5% of all injuries sustained from crashes. However, even though they comprise only a small percentage of total injuries, abdominal injuries represent a higher proportion of serious injuries. Elhagediab and Rouhana (1998) analyzed the National Automotive Sampling System (NASS) database from 1988 to 1994 and studied the frequency of injuries using the Abbreviated Injury Scale (AIS). The AIS ratings range from 1 for minor injuries to 6 for injuries that likely result in death, Table 1.1. It was found that abdominal injuries comprised 8% of all AIS ≥ 3 injuries but represented 16.5% of AIS ≥ 4 injuries and 20.5% of AIS ≥ 5 injuries. Lee and Yang (2002) reported that abdominal injuries constituted 5.2% of all injuries but 15.6% of AIS ≥ 3 injuries in the NASS database for the years 1993 through 1997. A NASS study from 1998-2004 by Klinich et al (2008) reported abdominal injuries constituted 8% of AIS ≥ 3, 15% of AIS ≥ 4, and
13% of AIS ≥ 5. These studies revealed that when considering higher severity injuries, an increasing percentage of abdominal injuries is a common trend.

<table>
<thead>
<tr>
<th>AIS Code</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>Minor</td>
</tr>
<tr>
<td>2</td>
<td>Moderate</td>
</tr>
<tr>
<td>3</td>
<td>Serious</td>
</tr>
<tr>
<td>4</td>
<td>Severe</td>
</tr>
<tr>
<td>5</td>
<td>Critical</td>
</tr>
<tr>
<td>6</td>
<td>Maximum</td>
</tr>
</tbody>
</table>

The liver has been reported to be a common site of abdominal injury, likely a result of its size and anatomical location. The liver is the largest organ in the body and occupies much of the right upper quadrant of the abdomen. In the study by Elhagediab, it was reported that the liver was the most frequently injured abdominal organ representing 38% of all abdominal injuries. Liver injuries were followed by injuries to the spleen (23%) and digestive system (17%). According to a second study of abdominal injuries based on the NASS database, the frequency of liver injuries was 15.7% of all abdominal injuries and 34% of AIS ≥ 3 abdominal injuries (Lee and Yang, 2002). The direction of impact in MVCs is an important factor in determining injury mechanisms, especially when focusing on injuries to the liver. Yoganandan et al (2000) studied abdominal injuries from MVCs and found that AIS ≥ 3 liver injuries occur more frequently in right side impacts than in left side impacts. This is expected due to the location of the liver in the upper right quadrant of the abdomen.
In summary, abdominal injuries are important to study because they occur in MVCs and represent a high proportion of serious injuries. These serious injuries to the abdomen are commonly to the solid organs, particularly the liver. Right side impacts are more likely than left side impacts to cause liver injury and therefore the current PMHS study will consist only of right-side impacts.

1.2 Anatomy of the Liver

The liver is the largest organ of the body weighing about 1300 grams in an adult. It is located immediately beneath the diaphragm muscle in the upper right quadrant of the abdomen and deep to ribs 7 through 11 as shown in Figure 1.1. The liver has numerous functions including the synthesis, storage, and release of vitamins and glycogen. It also synthesizes blood proteins, removes toxic substances, and produces bile. Bile produced by the liver is stored in the gallbladder, which is located directly inferior and posterior to the liver until it is eventually secreted into the duodenum to help break down food and absorb fats. The inferior vena cava (IVC) passes just posterior to the liver returning filtered venous blood from the liver and lower extremities to the heart.
The liver has four lobes and is supported by five ligaments: the falciform, coronary, left triangular, right triangular and round ligaments. The falciform ligament attaches the liver to the anterior abdominal wall and to the diaphragm. On the superior surface of the liver, the triangular and coronary ligaments connect the liver to the diaphragm. Figure 1.2 and Figure 1.3 show anterior and posterior views of the liver.
Figure 1.2: Anterior view of liver (Netter, 2003)

Figure 1.3: Posterior aspect of liver (Netter, 2003)
The lobes of the liver are classified by two schemes: anatomical and functional. The anatomical scheme divides the liver into the right lobe, left lobe, caudate lobe and quadrate lobe. Anteriorly, the significantly larger right lobe is separated from the left lobe by the falciform ligament. Inferiorly, the caudate lobe is positioned near the IVC, superior to the quadrate lobe which is adjacent to the gallbladder on the visceral surface of the liver. The caudate and quadrate lobes are divided from each other by the porta hepatis. The portal vein, hepatic artery, bile duct, hepatic nerve plexus, and lymph vessels pass through the porta hepatis. Collectively, the portal vein, hepatic artery, and bile duct are called the portal triad.

The liver can also be divided functionally into two lobes, right and left. The functional lobes are divided by the sagittal plane through the gallbladder fossa and the fossa for the IVC. The functional division of the liver is based on the fact that the right and left functional lobes each have their own blood supply and drainage. The hepatic artery and portal vein each divide into right and left branches to supply the lobes, shortly after passing through the porta hepatis.

The liver receives dual blood supply from the portal vein and the hepatic artery. Approximately 75% of the liver’s blood is supplied by the portal vein carrying venous blood from the spleen, gastrointestinal tract, and associated organs. The hepatic artery brings oxygenated blood from the aorta into the liver, via the celiac trunk. The blood exits the liver superiorly through the right, middle or left hepatic vein. The hepatic veins empty into the IVC where the blood is then returned to the heart.
The liver, like many of the abdominal organs, has a high degree of mobility because it is not rigidly fixed by its attachments. The serous fluid reduces the friction between the organ and the abdominal walls. The position of the liver depends on the orientation of the subject and for this reason, it is important to consider subject orientation when performing experimental studies relating to abdominal injury.

1.3 Previous Experimental Work

A lot of research has been done attempting to correlate different biomechanical predictors to abdominal injury. Finding a good biomechanical predictor can provide information which can be used to improve anthropomorphic test devices (ATDs) and ultimately reduce injuries to occupants in motor vehicle collisions (MVCs). Presented here is an overview of experimental work done on abdominal impacts and injury prediction providing context for the current study.

**Applied Force/Applied Pressure**

Studies have shown that peak force can correlate to abdominal injury in both frontal and lateral impacts. Trollope et al (1973) conducted impacts to 85 primate and 15 porcine subjects using various sized impactors to simulate common automotive injuries. The study found that peak applied force and duration had a high level of correlation to injury. It also found that for intact animals, over 1.6 kN (350 lbf) was needed to produce AIS ≥ 3 liver injury, while only 0.67 kN (150 lbf) was needed to produce the same injury to exposed livers. Work by Walfisch et al (1980) dropped unembalmed PMHS (n=11) from heights of one and two meters, corresponding to velocities of 4.5 m/s and 6.3 m/s respectively, to examine lateral
impact response and injury. The work revealed that a force of 4.5 kN was found to correspond to a 50% risk of AIS ≥ 3 liver injury. The study also found that an applied pressure of 260 kPa was associated with a 50% risk of AIS ≥ 3 liver injury. Rouhana et al (1986) performed 214 lateral abdominal impacts on anesthetized rabbits at 5 to 15 m/s using a pneumatic impactor. The impactor face used both rigid and crushable external impacting surfaces. The study found that peak force was well correlated with probability of AIS ≥ 3 renal injury, but not with hepatic injury. Other studies also found applied force to have a good correlation to injury (Talantikite et al 1993, Miller 1989).

Much research has been done which examined pressure applied during impact as a way to account for different impactor shape and size. McElhaney et al (1972) found that approximately 131 kPa (19 psi) of applied pressure was associated with abdominal injury (AIS = 4/5) for contact with “an armrest-like striker” for experiments using a monkey and baboon. Melvin et al (1973) tested organs that were surgically mobilized in anesthetized Rhesus Monkeys. The organs were loaded at controlled velocities and stroke and the resulting load-deflection data were normalized to develop average stress-strain curves for each test. He found that while static tests were confined to crushing of the parenchyma, dynamic tests caused burst injury. Melvin et al (1973) found that an applied pressure of 310 kPa corresponded to an AIS ≥ 4 injury to the liver. Other studies also found a correlation between applied pressure and injury (Lau and Viano, 1981a; Miller, 1989).
**Abdominal Compression**

Williams and Sargent (1963) conducted a series of 45 lower abdominal impact experiments using anesthetized canine subjects and concluded that the mechanism of injury to the intestines in blunt impact is compression of the intestines against spinal column. Miller (1989) performed experiments with anesthetized porcine subjects lying supine in a V-shaped support. The porcine subjects were loading with a belt at velocities varying from 1.6 to 6.6 m/s and compressions varying from 6% to 67%. The study found that maximum compression ($C_{\text{max}}$) correlates to abdominal injury for AIS $\geq 3$. Viano et al (1989) reported the results of lateral impacts ($n=44$) to unembalmed PMHS ($n=14$) using a pneumatic impactor at velocities of 4.5, 6.7, and 9.4 m/s. It was found that a compression of 0.437 and a $V_{C_{\text{max}}}$ of 1.98 m/s corresponded to 25% risk of AIS $\geq 4$ abdominal injury. Talantikite et al (1993) used a 23.4 kg mass driven at 5 to 7 m/s to impact six PMHS. The best correlation for abdominal injury (AIS) was deflection of the half abdomen and found that no AIS $\geq 4$ injuries occurred below 60 mm of deflection.

**Rate Effects and Viscous Injury**

Rate of impact loading has long been recognized as a factor in injury outcome (Kroell et al, 1981). Many of the solid abdominal organs (liver, kidney, spleen) are fluid filled systems that exhibit different mechanical characteristics under different rates of loading (Rouhana, 2002). Studies have shown that small changes in impact velocity can have a large effect on the injury level (McElhaney et al 1972, Melvin et al 1973). A study by Lau and Viano (1981b) held compression constant at 16% and
varied impact velocities between 5 and 20 m/s to anesthetized rabbits and saw a significant increase in hepatic injury with increasing impact velocity. Mertz et al (1997) developed risk curves for AIS ≥ 3 and AIS ≥ 4 as a function of rate of abdominal compression. Work by Rouhana et al (1985b) found that the product of maximum impact velocity ($V_{max}$) and maximum abdominal compression ($C_{max}$), known as abdominal injury criterion (AIC), was well correlated with the severity of abdominal injury from analysis of impacts to 117 anesthetized rabbits. Viano and Lau (1988) extended on this work relating the viscous criterion, $VC(t)_{max}$ the product of the velocity of deformation (V) and compression (C) to injury. Other studies have found the viscous criterion to correlate well to injury (Miller, 1989; Talantikite, 1993; Rouhana, 1986; Viano et al, 1989).

**Abdominal Response**

Cavanaugh et al (1986) performed frontal impacts to the lower abdomen of unembalmed PMHS (n=12) using a 32 or 64 kg pneumatic impactor. The subjects were impacted at various velocities ranging from 4.9-13.0 m/s. Force deflection curves were developed for the abdomen at both speeds. Due to relatively few injuries, correlations between injury and biomechanical variables were not recorded. Mertz (1984) normalized the force-deflection data from Walfisch (1980) and also reported the abdominal response.

**1.4 Previous Experimental Work at OSU**

Work performed by Sparks et al (2007) used 14 *ex vivo* human livers to study pressure as a predictor of injury. Impact-induced pressure changes were produced
using rigid-plate drop tower impacts. For each impact the venous and arterial systems of the liver were perfused using saline. In order to perfuse the venous and arterial systems to physiological pressures two separate standing reservoirs were placed at heights corresponding to physiological pressures. Pressure was measured by transducers inserted into the hepatic veins and in the parenchyma (caudate lobe). The experimental liver injuries were consistent with those documented in the CIREN database. Using binary logistic regression, it was determined that from the impacts the peak midline vascular pressure threshold for 50% risk of serious liver injury was 64 kPa while the peak tissue pressure threshold was 48 kPa. Tissue pressure was a stronger correlate to AIS ≥ 3 liver injury than midline vascular pressure.

Work done by Gustafson (2009) looked to further investigate the relationship between pressure and liver injury using six full body post-mortem human subjects (PMHS) subjected to blunt impacts. The research looked to provide a better understanding of the link between pressure and injury by testing the human liver in situ with boundary conditions more representative of real world trauma. The abdominal vasculature systems were re-pressurized and transducers measured pressure change due to the impact in various locations including the abdominal aorta, hepatic veins, and inferior vena cava (IVC). All subjects were impacted on the right side at a nominal velocity of 7 m/s, with four impacts being lateral impacts and two impacts applied at 30 degrees anterior of lateral or oblique. Binary logistic regression was used to predict injury risk and it was determined that various pressure related variables had statistically significant relationships to injury including peak change in pressure ($P_{max}$), peak rate of pressure change ($\dot{P}_{max}$), as well as $P_{max} \cdot \dot{P}_{max}$ and $[P(t)]^*$.
\(\dot{P}(t)_{\text{max}}\). The tests series successfully produced injuries similar to those documented in the CIREN trauma database. When considering the venous pressure sensors, Gustafson found that the strongest predictor of serious abdominal injury (AIS \(\geq 3\)) was \(\dot{P}_{\text{max}}\) \(p<0.001\), gamma=1.00). A value of \(\dot{P}_{\text{max}}\) of 32.1 kPa/ms corresponded to a 50% risk of AIS \(\geq 3\) injuries.

While reviewing the experimental research mentioned above, it was discovered that the data acquisition system settings used for the pressure transducers were incorrect. Because of the incorrect settings, correction factors must be applied to the pressure signals and new conclusions need to be made.

### 1.5 Goals of Current Study

In order to determine appropriate correction factors for the previous research, experiments with similar setups will be conducted using the pressure sensors with both the previous and correct settings. Sensors will be paired (previous/correct) to measure pressures at identical locations and then their traces will be analyzed to determine what correction factor should be applied.

In addition to correcting the previous research, this study will look to continue the work begun by Gustafson (2009) by conducting an additional four oblique PMHS impact tests to provide a better understanding of the link between pressure and injury to the human liver in situ. This study will provide technique improvements to issues encountered throughout the full-body PMHS testing. The improvements included:
• The use of the C-arm fluoroscopy system to determine liver position of PMHS while upright and ensure that pressurization catheters were located properly in the vasculature

• Flush the arterial and venous systems with embalming pump to remove any obstructions

• Pressurizing the PMHS from both the inferior and superior directions using the Foley catheters to ensure the liver is fluid-filled at impact

• Ensuring that the correct configuration for the Millar® pressure transducers were used in the data acquisition equipment
Chapter 2 : Correction of Previous Research at OSU

2.1 Background of Pressure Sensor Issues

From the beginning of the abdomen project, there were questions concerning the accuracy and reliability of the Millar® pressure transducers. The underlying concern was that the sensors were so highly temperature dependent that getting accurate and repeatable data with the sensors would be very difficult. To gain a better understanding of the Millars and their possible temperature dependence, a series of Millar benchtop tests were conducted by various members affiliated with the Injury Biomechanics Research Laboratory (IBRL), Transportation Research Center (TRC), and the Vehicle Research & Test Center (VRTC). In the first round of benchtop testing, the Millars were lowered several times into a standing column of water to various depths, changing the temperature of the water between tests, Figure 2.1 through Figure 2.3. The following conclusions were drawn:

- The Millars appear to be highly temperature dependent
  - Sensitivity seems to decrease with increase in temperature
  - Initial offset is close to zero at room temperature but inversely related with temperature
• The initial offset can be zeroed at the data acquisition prior to PMHS impacts, but dynamic tests need to be conducted to observe temperature effects on the Millar signal

• The time response of the Millars appear to be very slow

![Figure 2.1: Standing column of water test setup for benchtop tests](image1.png)

![Figure 2.2: Millar pressure transducers with thermocouple for benchtop tests](image2.png)

![Figure 2.3: Initial results focusing on temperature dependence of Millar sensors](image3.png)
Because the Millars had been used in multiple projects (*ex vivo* and full-body PHMS), another round of bench top testing was conducted. These tests were done to investigate the temperature dependent nature of the sensors as well as validate the response of the Millars dynamically. A similar test to that used in round one was conducted, Figure 2.4, along with a new dynamic test. For the dynamic test, a heart pump supplied a pressure impulse through a t-fitting where the Millar and Tektronix pressure transducers were placed in order to compare their signals, Figure 2.5. During these tests, there were drastically different results, Figure 2.6, and thus the following new conclusions were drawn:

- The strong temperature dependence recorded in earlier testing was not observed
- The slow time response documented in earlier testing was not observed
- The Millar readings were similar to the Tektronix® pressure sensor when tested dynamically
Figure 2.4: Second round of Millar benchtop testing (taken from Gustafson, 2009)

Figure 2.5: Dynamic testing using heart pump
In an attempt to understand the difference in results between the two benchtop trials a third round of testing was conducted. While sorting through the methods of the previous rounds, it was noticed that the only main difference in the testing was that the Millar transducer configuration files for the data acquisition system (Yokogawa) were different. In round one, the Millar transducers full scales (FS) were set to 154 psi and in round two the full scales were set to 38 psi. To test whether this was the cause another water column test was conducted in which a combination of full scale settings and excitation voltages were used, Figure 2.7. The plot illustrates how the sensors reacted when set at different configurations. From the test, these final conclusions about the Millar pressure transducers were drawn:

- Results similar to both rounds of benchtop testing were achieved, depending on the full scales settings in the Yokogawa system
- For 5V excitation a FS < 154psi and for 10V excitation a FS < 77 psi results in the pressure sensors being treated as full-bridge sensors, which is the correct configuration
- The excitation voltage does not affect the sensor outputs when used as full-bridge
- The initial offset from dipping the sensors is only present on the sensors configured in half-bridge, with those sensors with 10V excitation having about two times greater offset than those with 5V excitation
- The time response for sensors in full-bridge is much better than those in half-bridge
- For future use, the Millar sensors should only be used with full-bridge configurations with an excitation voltage of 5V
- All data collected using the half-bridge configuration needs to be validated with dynamic testing comparing half-bridge and full-bridge dynamic responses
Figure 2.7: Millar sensor output at different full scales (FS) with 5V excitation

From this test it was determined that something within the Yokogawa was causing the sensors to be treated differently and thus react differently when FS = 154 psi. It was determined that for 5V excitation a FS < 154 psi and for 10V excitation a FS < 77 psi results in the sensors being treated as full-bridge sensors (correct configuration) and any FS greater than these values result in the sensors being treated as half-bridge sensors (incorrect configuration). Below Figure 2.8 and Figure 2.9 show the wiring diagrams for the Millar sensor when treated as full-bridge and half-bridge respectfully. Notice that in Figure 2.8 the output is receiving input from both its + and – inputs and that in Figure 2.9 the sensor output is only receiving input from its + input channel. It is also important to note that when treated as half-bridge, the sensor temperature compensation component is not applied.
2.2 Methods for Determining Correction Factor

Next, all tests previously conducted at The Ohio State University Injury Biomechanics Research Laboratory using the Millar pressure transducers were investigated to determine the data acquisition settings. Table 2.1 shows all the tests in which the data acquisition system had used the incorrect half-bridge configuration settings and therefore required a correction factor for the Millar pressure traces.

<table>
<thead>
<tr>
<th>Project</th>
<th>Test Numbers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sparks et al (2007)</td>
<td>All tests (90°F)</td>
</tr>
<tr>
<td>Gustafson (2009)</td>
<td>FBL01-L, FBL02-L, FBL03-L, FBL04-L (60°F)</td>
</tr>
</tbody>
</table>

In an attempt to salvage the data previously acquired incorrectly using the Millar pressure transducers, a series of impact tests were conducted using redundant or paired Millar pressure transducers located in three different lobes of porcine livers. A porcine liver was impacted five times using a drop tower, increasing the drop height, and thus the impact energies, in 6” increments. The liver was pressurized
using saline reservoirs and allowed to fill prior to each impact. In order to compare the half-bridge and full-bridge configurations, sensors with the two different configurations were placed within the liver at approximately the same location. To do this, the Millars were paired and attached to catheters using shrink-wrap and then placed into separate lobes of the liver through the inferior vena cava (IVC). The instrumentation of the liver is shown in Figure 2.10 and Figure 2.11.

![Figure 2.10: Millars paired on catheter](image1)

![Figure 2.11: Millar pairs inserted into lobes of porcine liver](image2)

After tying off the exit IVC, pressurization tubes were placed into the hepatic artery and portal vein. The tubes were secured using suture string and the liver was moved to the drop tower area where saline reservoirs were connected to pressurize and fill the liver prior to each impact, Figure 2.12 and Figure 2.13.
After the liver was allowed to fill for several minutes, the height of the liver was measured and stoppers were set so that the liver would be compressed to 30% of its pressurized height. Event tape was placed over the liver to trigger the data acquisition system at impact. With the event tape in place, the drop height was set and the drop tower was released by triggering a magnet release, Figure 2.14 through Figure 2.16.
Because the initial benchtop experiments had shown that the Millar pressure transducers are temperature dependent when treated as half-bridge, three different saline temperatures were used. Based on temperatures used in previous testing, the three temperatures chosen were approximately 60°F, 75°F, and 90°F. The first temperature of 60°F was chosen because the core temperature of a post-mortem human subject (PMHS) when tested in a full-body impact is typically between 60-65°F. A saline temperature of 75°F was chosen in order to obtain an additional data
point allowing a better understanding of the sensors’ temperature dependence during dynamic impacts. Finally, the temperature of 90°F was chosen to simulate test conditions by Sparks et al (2007). For the testing, the temperature of the *ex vivo* livers and saline was near 90-98°F at the time of impact. An additional test was conducted using saline near 60°F with an excitation voltage of 10V because one previous PMHS tests had used this configuration. Data from the impacts revealed that the different configurations caused the sensors to record differently under the same loading conditions. From the tests it was also determined that temperature may have a slight effect on the dynamic response of the half-bridge sensors. For each temperature, a correction factor (CF) was determined between the full-bridge and half-bridge configurations.

### 2.3 Results of Correction Factor Impacts

Using MATLAB, the signals from the full-bridge and half-bridge sensors were compared. First the raw data was filtered using the channel frequency class filter, CFC60. Because the maximum pressures are of most importance, a correction factor (CF) was calculated by correlating the peaks of the paired sensors. Individual correction factors for each lobe and each impact were calculated by using equation 2.1, where FB and HB are full-bridge and half-bridge signals respectively. To determine a common correction factor for all of the impacts at one temperature, equation 2.2 was used.
\[ CF_i = \frac{\sum FB(x_{\text{max} - 1}, x_{\text{max}}, x_{\text{max} + 1}) \cdot HB(x_{\text{max} - 1}, x_{\text{max}}, x_{\text{max} + 1})}{\sum [HB(x_{\text{max} - 1}, x_{\text{max}}, x_{\text{max} + 1})]^2} \]  

(2.1)

\[ CF_{\text{total}} = \frac{\sum_{i=1}^{k} FB(x_{\text{max} - 1}, x_{\text{max}}, x_{\text{max} + 1}) \cdot HB(x_{\text{max} - 1}, x_{\text{max}}, x_{\text{max} + 1})}{\sum_{i=1}^{k} [HB(x_{\text{max} - 1}, x_{\text{max}}, x_{\text{max} + 1})]^2} \]  

(2.2)

Figure 2.17 through Figure 2.26 are pressure plots at the various impact heights. The different colored lines represent sensors in different lobes of the porcine liver. The solid lines correspond to the full-bridge sensors and the dashed lines correspond to the half-bridge sensor signals. On the left are plots of the pressure traces for a given impact comparing the full-bridge and half-bridge sensors and on the right are the plots comparing the two with the CF applied to the half-bridge signal.
Full vs. Half Bridge

Full vs. Half Bridge w/ CF applied

Figure 2.17: Impact001 (Drop Ht: 6”)

Figure 2.18: Impact001 (Drop Ht: 6”) adjusted

Figure 2.19: Impact002 (Drop Ht: 12”)

Figure 2.20: Impact002 (Drop Ht: 12”) adjusted

Figure 2.21: Impact003 (Drop Ht: 18”)

Figure 2.22: Impact003 (Drop Ht: 18”) adjusted
Because the previous research had also investigated the correlation between rate of pressure change and the risk of liver injury, the derivatives of the pressure signals shown above were also investigated. It was decided these derivatives were similar enough to allow the data previously collected in the half-bridge configurations to be used when determining risk curves. An example plot of the derivatives of the pressure signals is shown in Figure 2.27.
2.4 Conclusions and Correction Factor Determination

Using the data from the trials at the three different temperatures it was determined that the correction factor should vary depending on the temperature of the saline used for that particular test. The correction factors to be applied for the previous OSU research are shown in Table 2.2.

<table>
<thead>
<tr>
<th>Project</th>
<th>Test Numbers and Conditions</th>
<th>Correction Factor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sparks et al (2007)</td>
<td>All tests (5V, 90°F)</td>
<td>1.76</td>
</tr>
<tr>
<td>Gustafson (2009)</td>
<td>FBL01-L (10V, 60°F)</td>
<td>1.80</td>
</tr>
<tr>
<td>Gustafson (2009)</td>
<td>FBL02-L, FBL03-L, FBL04-L (5V, 60°F)</td>
<td>1.71</td>
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</tbody>
</table>
Chapter 3: Methods of Full Body PMHS Testing

3.1 PMHS Selection and Preparation

Four PMHS were obtained through the willed body donation program at The Ohio State University to complete the overall PMHS test series (n=10). Both males and females were accepted for testing and all subjects met the following criteria:

- The subject had a body mass index (BMI) between 18.5 and 30 kg/m\(^2\). A BMI in this range classifies the PMHS as normal or overweight, but not obese. BMI is calculated by dividing the subject’s weight by height squared:

\[
BMI = \frac{weight \text{ in kg}}{(height \text{ in meters})^2}
\]

- The subject was not osteoporotic. All subjects were scanned using a Dual Energy X-ray Absorptiometry (DXA) bone scan. All subjects with T-scores greater than -2.5 were eligible to be accepted. A T-score is the number of standard deviations a person is below the average bone density of a healthy adult of 30 years old of the same gender. All T-scores correspond to one of the following categories:
  - -1.0 or higher: Normal
  - -2.5 to -1.0: Osteopenic
  - -2.5 or lower: Osteoporotic
- The subject did not have scars indicating major abdominal surgery affecting the liver
- The subject did not weigh more than 95 kg. A subject weighing more than 95 kg would have been difficult to transport and position.
If the PMHS was deemed acceptable, all tests were performed within five days of death. The subjects were kept stored in a cooler set at 4 degrees Celsius to prevent additional tissue degradation. A summary of the tests and subject information is given in Table 3.1. Additional anthropometry measurements are provided in Appendix A.
### Table 3.1: Summary of subject information

<table>
<thead>
<tr>
<th></th>
<th>FBL01-L</th>
<th>FBL02-L</th>
<th>FBL03-L</th>
<th>FBL04-L</th>
<th>FBL05-O</th>
<th>FBL06-O</th>
<th>FBL07-O</th>
<th>FBL08-O</th>
<th>FBL09-O</th>
<th>FBL10-L</th>
<th>Average</th>
<th>Standard Deviation</th>
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<td>Lateral</td>
<td>Lateral</td>
<td>Lateral</td>
<td>Oblique</td>
<td>Oblique</td>
<td>Oblique</td>
<td>Oblique</td>
<td>Oblique</td>
<td>Lateral</td>
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<td>-</td>
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<td>Female</td>
<td>Male</td>
<td>Male</td>
<td>Female</td>
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<td>-</td>
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<td>Age</td>
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<td>79</td>
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<td>77</td>
<td>78</td>
<td>65</td>
<td>76</td>
<td>± 11.3</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>67</td>
<td>59</td>
<td>73</td>
<td>64</td>
<td>54</td>
<td>82</td>
<td>57</td>
<td>69</td>
<td>58</td>
<td>54</td>
<td>64</td>
<td>± 9.2</td>
</tr>
<tr>
<td>Stature (cm)</td>
<td>176</td>
<td>154</td>
<td>188</td>
<td>179</td>
<td>164</td>
<td>179</td>
<td>171</td>
<td>157</td>
<td>171</td>
<td>166</td>
<td>171</td>
<td>± 10.5</td>
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<tr>
<td>Chest Breadth (cm)</td>
<td>26.9</td>
<td>30.8</td>
<td>27.0</td>
<td>27.3</td>
<td>30.0</td>
<td>30.8</td>
<td>26.3</td>
<td>30.5</td>
<td>26.2</td>
<td>28.5</td>
<td>28.4</td>
<td>± 1.92</td>
</tr>
<tr>
<td>Waist Breadth (cm)</td>
<td>25.8</td>
<td>33.0</td>
<td>30.0</td>
<td>29.5</td>
<td>30.0</td>
<td>35.0</td>
<td>27.0</td>
<td>40.5</td>
<td>30.7</td>
<td>31.9</td>
<td>31.3</td>
<td>± 4.18</td>
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<tr>
<td>Seated Height (cm)</td>
<td>93.5</td>
<td>87.0</td>
<td>96.0</td>
<td>97.0</td>
<td>91.5</td>
<td>99.0</td>
<td>94.0</td>
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<td>90.0</td>
<td>92.0</td>
<td>92.0</td>
<td>± 5.5</td>
</tr>
<tr>
<td>Impact Velocity (m/s)</td>
<td>7.2</td>
<td>7.1</td>
<td>7.0</td>
<td>7.1</td>
<td>7.1</td>
<td>7.0</td>
<td>7.1</td>
<td>7.2</td>
<td>7.1</td>
<td>7.1</td>
<td>7.1</td>
<td>± 0.06</td>
</tr>
<tr>
<td>BMD (g/cm²)</td>
<td>1.293</td>
<td>1.008</td>
<td>1.139</td>
<td>1.095</td>
<td>1.039</td>
<td>1.308</td>
<td>1.073</td>
<td>1.000</td>
<td>0.959</td>
<td>1.063</td>
<td>1.098</td>
<td>± 0.12</td>
</tr>
<tr>
<td>T-score*</td>
<td>0.9</td>
<td>-1.5</td>
<td>-1.0</td>
<td>-1.6</td>
<td>-1.1</td>
<td>1.1</td>
<td>-1.8</td>
<td>-1.6</td>
<td>-2.1</td>
<td>-0.8</td>
<td>-1.0</td>
<td>± 1.1</td>
</tr>
</tbody>
</table>
3.2 Internal Instrumentation

The goal of internal instrumentation was to measure pressure changes during impact to the abdomen re-pressurized to physiological pressures. To properly pressurize the abdomen it was essential to assure that no clots were obstructing the vasculature. To ensure that the vasculature was clear of obstructions, both the arterial and venous systems were flushed using warm saline and an embalming pump. The saline was flushed from superior to inferior by dissecting out the right and left internal jugular vein and right and left common carotid artery, as well as the femoral vessels on both sides. This was an improvement made to work done by Gustafson (2009). This procedure helped remove clots and any coagulated blood from the vessels making pressurization easier and allowing for placement of the sensors.

After clearing the vasculature, Millar pressure transducers were attached to angiographic catheters and then routed through a compression fitting connected to a Y-fitting, which inserted into Foley catheters, Figure 3.1 and Figure 3.2. The assembly was then routed into the vasculature.

![Figure 3.1: Millar and Foley assembly (inlet)](image)
To pressurize the venous system and liver, the Foley assemblies were inserted through the right internal jugular vein (superior) and right femoral vein (inferior). Superiorly the Foley balloon was filled between the heart and liver in the IVC, and the Millar pressure transducers were routed into a hepatic veins of the liver. In some instances there was not enough room between the heart and the liver to route the pressure transducers into the hepatic veins. In these cases the pressure transducers were placed in the IVC. Inferiorly the Foley balloon was placed in the IVC just below the liver such that the pressure transducers were located around the level of T10/T11. For both venous assemblies there were two pressure transducers in order to collect multiple pressure measurements, Figure 3.3 and Figure 3.5.

To pressurize the arterial system of the abdomen the Foley assemblies were inserted through the left common carotid artery (superior) and left femoral artery (inferior). Superiorly the Foley was directed passed the arch of the aorta into the descending aorta. The Foley balloon was filled so that the pressure transducers were located around the level of T10. Inferiorly the Foley balloon was placed in the descending aorta so that the pressure transducers were located at the level of L3. This was to ensure that the celiac trunk was included in the pressurization area. For both
arterial assemblies there was only one pressure transducer, Figure 3.4 and Figure 3.6. Following internal instrumentation, a computed tomography (CT) scan was taken of the subject from head to mid femur. The CT scan was used to document the locations of the internal instrumentation.

![Figure 3.3: Hepatic Millar assembly](image3.3)

![Figure 3.4: Inferior aorta Millar assembly](image3.4)

Two additional pressure sensors were inserted directly into the liver tissue just prior to impact, shown in Figure 3.7. This was done by making an incision between the 7th and 8th ribs on the right posterior side of the PMHS and then gluing the sensors into place. Because these sensors were placed prior to impact there is no CT data to determine their location.
3.3 External Instrumentation

In order to track skeletal motion of the PMHS, motion blocks were attached to skeletal structures of the PMHS. These motion blocks, called 3aω motion blocks, consisted of three linear accelerometers and three angular rate sensors that were used to measure acceleration and angular rotation for each impact, Figure 3.8 and Figure 3.9. Three U-brackets were screwed into the bodies of vertebrae T1, T8, and T12, and a flat mount was attached to the sternum, Figure 3.10.
Additionally a chestband was placed around the subject’s abdomen so that it was aligned with the center of impact plate in order to measure deflection along the impact line, Figure 3.11 and Figure 3.12. The chestband was comprised of 40 strain gages mounted to a metal strip covered with flexible rubber. The strain measurements from the chestband were processed using a MATLAB (The Math Works, Massachusetts) coded script called CrashStar v2.5 (NHTSA). The script uses the strain inputs to output planar coordinates for each gauge. These coordinates are then plotted over time and are used to measure thoraco-abdominal deflection at the level of impact.
Figure 3.11: Schematic of external instrumentation (taken from Gustafson, 2009)

Figure 3.12: Chestband placed on subject
3.4 Test Preparation

Prior to final positioning, the PMHS was seated and the C-arm was used to ensure proper blockage of the veins and arteries. This was done by injecting a mixture of contrast and saline through the Foley catheter in the right jugular vein and left common carotid artery and using the C-arm to provide real time images of the solution being blocked by the Foley balloons. At this time, C-arm images were taken to locate the liver with respect to the xiphoid process in order to center the impactor at the level of the liver. An example of this procedure is shown in Figure 3.13 through Figure 3.15. This procedure was one of the improvements made to the methods previously reported by Gustafson and therefore was only performed on the last four PMHS tests. It should be noted that the liver loading prior to implementing this procedure varied only slightly from subject to subject due to the small difference gravity produced on the liver when the subject was seated upright.

![Figure 3.13: Liver in supine position](image1)

![Figure 3.14: Liver in upright position](image2)
The pre-test positioning for oblique and lateral impacts is shown in Figure 3.16 and Figure 3.17 respectively. Also, overhead schematics are shown in Figure 3.18 and Figure 3.19 to show the orientation of the PMHS for the different impact orientations. For the combined PMHS study a total of five lateral and five oblique impacts were performed. Prior to impact, the subject’s lungs were inflated to approximately 6 kPa through an intubation tube inserted directly into the trachea. The arms were crossed and secured on a support system at shoulder level to ensure that the arms would not interfere with the impact.
Once all the instrumentation was in place, a FARO Arm point digitizing system [FARO Technologies, Inc., Florida] was used to digitize various anatomical landmarks and locate the initial position of the 3a0 blocks. These points were used to create local
coordinate systems for the motion blocks and a local body coordinate system that were later transformed into the global lab coordinate system.

Prior to pressurization, air was bled from the tubing. Y-fittings were used at the reservoirs so that the subject could be pressurized from both superior and inferior Foleys on both the arterial and venous sides. This was an improvement made to the methods reported by Gustafson and therefore was also only done on the last four PMHS tests. The main benefit of pressurizing from both directions was that it allowed for the vasculature of the abdomen and liver to fill more quickly helping ensure that at impact the liver and abdomen were re-pressurized. This is represented in the schematic in Figure 3.20. Also shown in the schematic are the saline reservoirs used to pressurize the abdomen. These reservoirs were set at heights corresponding to physiological pressures.

![Figure 3.20: Schematic of setup (edited from Gustafson, 2009)](image-url)
The impactor face on the pneumatic ram was a 15 x 30 cm aluminum plate weighing 1.61 kg. The total ram mass was 23.07 kg. Event tape was placed on the subject and impactor face which triggered the data acquisition system and was used to define time zero. Just prior to impacting the PMHS, a magnetic bracket released the subject’s head restraint allowing the subject to move freely after the impact. A secondary impact protection unit (SIPU) was secured to the lift table to catch the PMHS following the impact.

Two high-speed cameras were used to record the impact. One was located perpendicular to the impact direction and the other was located perpendicular to the subject. A handheld camera was placed on top of the high-speed that was perpendicular to the impact line to provide a real-time recording of the impact. Following the impact, a CT scan of the subject was taken and an autopsy was performed to document injuries.

### 3.5 Data Processing

All data was acquired at 20,000 Hz with a pre-filter of 3000 Hz applied to eliminate high frequency noise. Offsets in the signal were removed by subtracting any bias prior to impact on all channels excluding the 40-band chestband. All data signals were filtered according to standard SAE-J211 using channel filter classes (CFC) ranging from CFC 60-CFC 1000. Because there is no standard filter class for pressure data, a fast Fourier Transform (FFT) analysis of the signals was performed and a CFC60 filter was decided upon. The FFT analysis can be found in Appendix B.
3.6 Calculation of Biomechanical Variables

Biomechanical variables were considered as possible predictors of liver injury. Additional biomechanical variables from the literature search that had been found to be good predictors of abdominal injury were considered as well. A list of all the biomechanical variables considered as predictors and their methods of calculation are show in Table 3.2.

Table 3.2: Biomechanical variables considered for risk predictors and their method of calculation

<table>
<thead>
<tr>
<th>Variable</th>
<th>Units</th>
<th>Method of Calculation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tissue $P_{max}$</td>
<td>kPa</td>
<td>Maximum tissue pressure</td>
</tr>
<tr>
<td>Tissue $\dot{P}_{max}$</td>
<td>kPa/ms</td>
<td>Differentiate tissue pressure signal (maximum)</td>
</tr>
<tr>
<td>Vascular $P_{max}$</td>
<td>kPa</td>
<td>Maximum vasculature pressure (first peak after maximum load)</td>
</tr>
<tr>
<td>Vascular $\dot{P}_{max}$</td>
<td>kPa/ms</td>
<td>Differentiate vasculature pressure signal (maximum)</td>
</tr>
<tr>
<td>$P_{max} \cdot \dot{P}_{max}$</td>
<td>kPa$^2$/ms</td>
<td>Multiply maximum vasculature pressure by maximum rate of vasculature pressure change</td>
</tr>
<tr>
<td>AIC $(V_{max} \cdot C_{max})$</td>
<td>m/s</td>
<td>Take the maximum pre-impact velocity of the ram and multiply it by the maximum compression (from chestband analysis)</td>
</tr>
<tr>
<td>Compression</td>
<td>mm/mm</td>
<td>Take the maximum deflection measured by the chestband and normalize by the initial distance between the gage at the impact location and opposite the impact location along the line of the impact direction</td>
</tr>
<tr>
<td>Scaled Force</td>
<td>N</td>
<td>Taken as maximum normalized force from load cell</td>
</tr>
<tr>
<td>$[V(t) \cdot C(t)]_{max}$</td>
<td>m/s</td>
<td>Using the chestband deflection time history, differentiate to obtain the velocity of deflection and multiply the result by the compression time history (maximum)</td>
</tr>
<tr>
<td>$[\dot{F} \cdot C]_{max}$</td>
<td>N/ms</td>
<td>Differentiate the force and multiply it's time history by the compression time history (maximum)</td>
</tr>
</tbody>
</table>
3.7 Statistical Analysis

Because time of injury could not be determined from the pressure signals, the data needed to be treated as doubly censored. Doubly censored data has both right censored (no injury) and left censored (injured w/out knowing time of injury). One of the most commonly used statistical methods used for doubly censored biomechanical data is the Consistent Threshold method or CT method (Nusholtz, 1999; Di Domenico, 2003, 2005). The CT method is a non-parametric maximum likelihood estimator that can be used to estimate the shape of the unknown biomechanical distribution. Once the CT estimate is obtained it may suggest properties of the underlying distribution. It should be noted that the CT estimate can only operate on the available data and cannot address biases, experimental, or model specification errors.

After using the CT method to determine the shape of the underlying distribution binary logistic regression was performed and compared to the CT method. Binary logistic regression was chosen because it has been used in previous research to develop parametric injury risk curves (Sparks et al, 2007; Gustafson, 2009). The binary logistic regression risk curves were used to evaluate different biomechanical variables for significance and fit. Variables were considered significant if $p \leq 0.05$ and the Goodman-Kruskal’s Gamma ($\gamma$) (Kent et al, 2008) was used as the primary measure of fit for the binary logistic regression model. Additionally, the log likelihood was used as a secondary measure to evaluate which predictor variables.
3.8 Transformations

All acceleration and angular rate sensor data from the 3aω motion blocks were transformed to the laboratory (global) coordinate system. To do this a laboratory coordinate system was created using a FARO arm. With the lab coordinate system created, the 3aω motion blocks were located. Three points at the vertices of the motion block’s face were taken in order to create local coordinate systems for the motion block and related using Euler angle calculations. The transformed accelerations were used in calculating the deflection of the abdomen region during impact.

3.9 Normalization

Due to the difference in subject anthropometry, normalization of the force, deflection, and time data was needed in order to allow for a more meaningful comparison between tests. There are three normalization methods that have been discussed in the literature that help scale parameters, typically to a 50th percentile male. The first method by Eppinger et al (1984) uses simple ratios of the mass of a 50th percentile male (76 kg) to the total body mass of the PMHS to develop scales for time, length, and force. One major limitation of this method is that it does not take into account the build of the subject and assumes that the density and the elastic modulus across all subjects are equal. However, subjects of the same mass may have very different proportions (e.g. tall and slim or short and obese). For the Eppinger method scaling factors were calculated using the following equation:

$$\lambda_m = \frac{M_{50th}}{M_{sub}}$$  \hspace{1cm} (3.1)
Where \( n \) = the total number of trials

\[
M_{\text{sub}} = \text{total mass of the subject}
\]

\[
M_{50\text{th}} = 76\text{kg}
\]

and “50th” refers to the 50th percentile male and “sub” refers to the subject or PMHS being scaled. Using this ratio the following PMHS measurements were scaled according to the following equations:

\[
L_{50\text{th}} = \left( \frac{V_{50\text{th}}}{V_{\text{sub}}} \right) \lambda^{1/3} L_{\text{sub}}
\]

\( (3.2) \)

\[
F_{50\text{th}} = \left( \frac{V_{50\text{th}}}{V_{\text{sub}}} \right) \lambda^{2/3} F_{\text{sub}}
\]

\( (3.3) \)

\[
T_{50\text{th}} = \lambda^{1/3} T_{\text{sub}}
\]

\( (3.4) \)

The second method considered, by Mertz and Viano, (Viano, 1989) proposed a normalization scheme, Impulse-Momentum Based Scaling, which uses a stiffness ratio and a mass ratio that is based on a calculated effective mass. In the Mertz and Viano method, instead of using the total body mass ratio, an effective mass of the loading region is calculated for each subject. The effective mass is calculated using an impulse momentum analysis of the impact event. In all cases, the times over which the integrals are calculated are taken from the time the ram impacts the PMHS (time zero) until maximum deflection of the thoraco-abdominal region. The acceleration of the subject was taken from the 3a\( \omega \) motion block located on T8 (slightly superior to the impact height). The effective mass was then calculated using equation 3.5 for each PMHS:

\[
m_{\text{eff}} = \frac{\int_0^T F dt}{\int_0^T a_{\text{sub}} dt}
\]

\( (3.5) \)
Because effective mass for the impact portion of the subject is not known, an effective mass is calculated using the effective masses calculated in the data set. The effective mass of the 50\textsuperscript{th} percentile and the mass ratio calculations are shown in equations 3.6 and 3.7:

$$m_{\text{eff-50th}} = \frac{\sum_{i=0}^{n} m_{\text{eff-sub}}}{n} M_{50th}$$  \hspace{1cm} (3.6)

$$\lambda_m = \frac{m_{\text{eff-50th}}}{m_{\text{eff-sub}}}$$  \hspace{1cm} (3.7)

Additionally, for this method a stiffness ratio was needed and calculated with the assumption that stiffness was proportional to a characteristic length for the impact region. For this analysis the chest breadth of the subject was used and compared to the 50\textsuperscript{th} percentile chest breadth (taken as 32.65cm for this test). The stiffness ratio was calculated using the following equation:

$$\lambda_k = \frac{\text{Chest Breadth}_{50th}}{\text{Chest Breadth}_{\text{sub}}}$$  \hspace{1cm} (3.8)

Using these ratios the following PMHS measurements were scaled according to equations 3.9 (deflection), 3.10 (force) and 3.11 (time):

$$L_{50th} = \left( \frac{v_{50th}}{v_{\text{sub}}} \right) \sqrt{\frac{\lambda_m}{\lambda_k}} \sqrt{\frac{m_p+m_{\text{sub}}}{m_p+m_{50th}}} L_{\text{sub}}$$  \hspace{1cm} (3.9)

$$F_{50th} = \left( \frac{v_{50th}}{v_{\text{sub}}} \right) \sqrt{\lambda_m \lambda_k} \sqrt{\frac{m_p+m_{\text{sub}}}{m_p+m_{50th}}} F_{\text{sub}}$$  \hspace{1cm} (3.10)

$$T_{50th} = \frac{\lambda_m}{\sqrt{\lambda_k}} \sqrt{\frac{m_p+m_{\text{sub}}}{m_p+m_{50th}}} T_{\text{sub}}$$  \hspace{1cm} (3.11)

The final normalization method considered was one recently submitted for publication by Moorhouse (Moorhouse, In review). This method employs similar assumptions from Mertz and Viano, except that the stiffness ratio was not taken as a ratio
of characteristic length. Instead of using characteristic lengths to develop a stiffness ratio, an effective stiffness of the loading region is calculated based on the energy absorbed by the loaded region during impact. To calculate that energy the loading history is integrated over the deflection history from time zero until time of maximum deflection. This effective stiffness was calculated using the following equation:

\[ k_{\text{eff-sub}} = \frac{\int F dx}{x_{\text{max}}^2} \]  

(3.12)

Similarly the \( k_{\text{eff-50th}} \),

\[ k_{\text{eff-50th}} = \frac{\sum_{i=0}^{n} k_{\text{eff-sub}}}{\sum_{i=0}^{n} L_{\text{50th}}} L_{\text{50th}} \]  

(3.13)

Where \( L_{\text{sub}} \) = characteristic length of subject (chest breadth)

\( L_{\text{50th}} = 32.65 \text{cm} \)

Using these values the mass ratio and stiffness ratios were calculated using the following equations:

\[ \lambda_m = \frac{m_{\text{eff-50th}}}{m_{\text{eff-sub}}} \]  

(3.14)

\[ \lambda_k = \frac{k_{\text{eff-50th}}}{k_{\text{eff-sub}}} \]  

(3.15)

Using these new constants equations 3.9-3.11 can be used as the scaling equations for the PMHS data.
Chapter 4 : Results

4.1 Injury Analysis

*Ex Vivo Liver Injury*

Because this research looks to investigate relationships between the full-body PMHS testing and the *ex vivo* liver tests conducted by Sparks et al (2007) Table 4.1 summarizes all injuries sustained to the livers during drop tower testing.
Table 4.1: Ex vivo liver injury summary (taken from Sparks, 2007)

<table>
<thead>
<tr>
<th>Subject</th>
<th>Injury Description</th>
<th>AIS Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>**HL01</td>
<td>One deep and one superficial laceration on superior aspect of right lobe (9 and 5 cm); Three superficial lacerations on posterior aspect of right lobe (2, 5, and 6 cm)</td>
<td>3</td>
</tr>
<tr>
<td>**HL02</td>
<td>One superficial laceration on superior aspect of right lobe (4 cm); One superficial laceration on lateral aspect of right lobe (10 cm); Moderate internal damage in right lobe (8 cm)</td>
<td>3</td>
</tr>
<tr>
<td>**HL03</td>
<td>One superficial laceration on superior aspect of right lobe (8 cm); Minor internal damage (2 cm)</td>
<td>2</td>
</tr>
<tr>
<td>**HL04</td>
<td>Two deep lacerations on right lobe (5 and 6 cm); Five superficial lacerations on right lobe (2, 3, 4, 5, and 5 cm); Burst injury of right lobe</td>
<td>4</td>
</tr>
<tr>
<td>**HL05</td>
<td>Fiver superficial lacerations on right lobe (3, 5, 5, 6, and 7 cm); Moderate internal damage (5 cm)</td>
<td>3</td>
</tr>
<tr>
<td>**HL07</td>
<td>One superficial laceration on lateral aspect of right lobe (4 cm); One superficial laceration on posterior aspect of right lobe (7 cm)</td>
<td>2</td>
</tr>
<tr>
<td>**HL08</td>
<td>Superficial capsule laceration on posterior aspect of right lobe (7 cm); Minor internal damage in right lobe (2 cm)</td>
<td>2</td>
</tr>
<tr>
<td>**HL09</td>
<td>Superficial right lobe laceration (4 cm)</td>
<td>2</td>
</tr>
<tr>
<td>**HL10</td>
<td>Three lacerations on anterior aspect of right lobe (5, 7, 9 cm); Moderate internal damage in right lobe (5 cm)</td>
<td>3</td>
</tr>
<tr>
<td>**HL11</td>
<td>Two right lobe lacerations (8 and 4 cm); Moderate internal damage (3 cm)</td>
<td>3</td>
</tr>
<tr>
<td>**HL12</td>
<td>Laceration on lateral aspect of right lobe continuing to posterior aspect (7 cm)</td>
<td>2</td>
</tr>
<tr>
<td>**HL13</td>
<td>Seven lacerations on anterior aspect of right lobe (2, 2, 3, 3, 5, 5, 5 cm); One laceration on posterior aspect of right lobe (2 cm); Severe internal damage (10 cm)</td>
<td>4</td>
</tr>
<tr>
<td>**HL14</td>
<td>Three lacerations on anterior aspect of right lobe (6, 6, 8 cm); The 8 cm laceration extended to the posterior aspect of right lobe; Moderate internal damage in right lobe (5 cm)</td>
<td>3</td>
</tr>
<tr>
<td>**HL15</td>
<td>No surface lacerations; Minor internal damage in right lobe (3 cm)</td>
<td>2</td>
</tr>
</tbody>
</table>

**Tests conducted by Sparks et al, 2007

PMHS Injury

Following each test, an autopsy was performed and focused on identifying skeletal and abdominal injury, specifically injury to the liver. Table 4.2 summarizes the liver injuries sustained during the PMHS impacts. Liver injury of AIS ≥ 3 was obtained for four out of ten tests, with three of those occurring during oblique impacts. Full autopsy results for each impact can be found in Appendix C.
4.2 Pressure

Because the liver is primary supplied with blood from the venous system, analysis focused on results obtained from pressure transducers located in the venous vasculature. On a few tests the space between the heart and the liver was too small to route the pressure transducers into the hepatic veins. In these cases the Foley balloon was inflated just past the heart and the pressure sensors were placed in the IVC and included in the analysis. In other cases, pressure sensor locations were incorrectly identified due to the limitations of two-dimensional fluoroscopy. However, because there were multiple sensors located in the vasculature, any sensor not properly located was excluded from the analysis. Below Figure 4.1 through Figure 4.4 show 3-D volume renderings of CT images for several tests. In Figure 4.4 the pressure transducer for FBL06-O was routed.
deep into the hepatic vessel, resulting in a higher pressure reading, and was excluded from the analysis. The reasoning behind this is discussed in the section 5.2. More 3-D volume renderings can be found in Appendix D.

Figure 4.1: FBL08-O sensor placement (as planned with sensors in hepatic veins and IVC)

Figure 4.2: FBL10-L sensor placement (unable to place sensors in hepatic veins; placed in IVC)

Figure 4.3: FBL04-L sensor placement (sensors placed in renal vein instead of hepatic vein) *excluded from analysis

Figure 4.4: FBL06-O sensor placement (sensor placed extremely deep in hepatic vessel) *excluded from analysis
With the assumption that injury is more likely to occur where pressure is the highest, for each test the sensor that reported the highest peak pressure was taken as $P_{\text{max}}$. The same was done for determining $\dot{P}_{\text{max}}$. It should be noted that $P_{\text{max}}$ is not the absolute pressure but rather the change in pressure caused by the blunt impact.

PMHS tests FBL05 through FBL10 included pressure sensors placed in the liver tissue. Readings from the tissue sensors were collected with varying degrees of success due to the uncertain nature in which they were placed (no CT image for location information). Figure 4.5 through Figure 4.7 show pressure traces from one test (FBL08-O) that were typical for each PMHS test. Figure 4.8 show traces of $\dot{P}$ for the same test. This is a direct derivative of the pressure traces seen in Figure 4.5 with units of kPa/ms. Pressure traces for all tests can be seen in Appendix E.

![Figure 4.5: FBL08-O venous traces](image)
Figure 4.6: FBL08-O arterial traces

Figure 4.7: FBL08-O liver tissue traces
4.3 Ex-Vivo Biomechanical Variables and Injury Risk

The CT method discussed in section 3.7 was used to develop a non-parametric risk curve for each variable while treating the data as doubly censored. Binary logistic regression (alpha=0.05) was then performed on the data and the curve’s shape was compared to that of the CT method. Most of the binary logistic regression curves compared well to the CT method. This suggests that using the binary logistic regression curve as the risk function is appropriate. All variables were analyzed for their relationship to AIS ≥ 3 liver injury by fitting data to the risk function of the form:

\[
Probability\ of\ AIS \geq 3\ liver\ injury = \frac{e^{a+bx}}{1 + e^{a+bx}}
\]

Where

- \(x\) = pressure-related variable
- \(a, b\) = model parameter coefficients
Biomechanical predictors were ranked using the Goodman-Kruskal Gamma (labeled GKG on risk plots). Table 4.3 shows the results of all the biomechanical variables analyzed for the ex vivo liver testing performed by Sparks (2007). Data from the venous pressure sensors and tissue pressure sensors were used to calculate variables that could be used to develop injury risk curves. For vascular pressure, in addition to maximum pressure change ($P_{\text{max}}$) and maximum rate of pressure change ($\dot{P}_{\text{max}}$) the product of the two ($P_{\text{max}} \times \dot{P}_{\text{max}}$) was also considered as a possible predictor of injury. It was considered because it makes physical sense for the combination of the two to correlate well to liver capsule failure. The statistically significant variables are highlighted in green.

In addition to the pressure-related variables, other biomechanical variables were considered as possible predictors of liver injury. The list of variables and their method of calculation can be found in Table 3.2. Plots of the binary injury risk functions for all statistically significant biomechanical predictor variables (also shown $P_{\text{max}} \times \dot{P}_{\text{max}}$; p-value=.055) along with the non-parametric CT method curve are given in Figure 4.9 through Figure 4.14.

It should be noted that for the ex vivo impacts only twelve impacts included tissue pressure sensors with two of those signals excluded due to abnormal readings. As a result, the risk curves for the tissue pressure variables only take into account nine data points. Two signals were also excluded for the vascular pressure resulting in the risk curves only including twelve data points. All other biomechanical predictors include data from all fourteen impacts.
Table 4.3: Summary of biomechanical variables and their use as risk predictors for *ex vivo* data

<table>
<thead>
<tr>
<th>Predictor Variable</th>
<th>Units</th>
<th>50% Risk Value</th>
<th>Log Likelihood</th>
<th>P-value</th>
<th>Goodman-Kruskal Gamma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tissue ( \dot{P}_{\text{max}} )</td>
<td>kPa/ms</td>
<td>8.69</td>
<td>-2.235</td>
<td>0.005</td>
<td>0.80</td>
</tr>
<tr>
<td>AIC ( (V_{\text{max}} \times C_{\text{max}}) )</td>
<td>m/s</td>
<td>0.82</td>
<td>-6.110</td>
<td>0.009</td>
<td>0.79</td>
</tr>
<tr>
<td>Vascular ( \dot{P}_{\text{max}} )</td>
<td>kPa/ms</td>
<td>10.6</td>
<td>-5.098</td>
<td>0.013</td>
<td>0.77</td>
</tr>
<tr>
<td>Tissue ( P_{\text{max}} )</td>
<td>kPa</td>
<td>56.3</td>
<td>-3.584</td>
<td>0.023</td>
<td>0.70</td>
</tr>
<tr>
<td>( P_{\text{max}} \times \dot{P}_{\text{max}} )</td>
<td>kPa²/ms</td>
<td>685</td>
<td>-6.308</td>
<td>0.055</td>
<td>0.60</td>
</tr>
<tr>
<td>Compression</td>
<td>%</td>
<td>29.9</td>
<td>-7.303</td>
<td>0.034</td>
<td>0.58</td>
</tr>
<tr>
<td>Vascular ( P_{\text{max}} )</td>
<td>kPa</td>
<td>52.6</td>
<td>-7.026</td>
<td>0.134</td>
<td>0.43</td>
</tr>
<tr>
<td>Scaled Force</td>
<td>N</td>
<td>4260</td>
<td>-9.149</td>
<td>0.364</td>
<td>0.08</td>
</tr>
</tbody>
</table>

Figure 4.9: Injury risk vs. tissue \( \dot{P}_{\text{max}} \) for *ex vivo* liver tests
Figure 4.10: Injury risk vs. AIC for *ex vivo* liver tests

Figure 4.11: Injury risk vs. $\dot{P}_{\text{max}}$ for *ex vivo* liver tests
Figure 4.12: Injury risk vs. tissue $P_{\text{max}}$ for ex vivo liver tests

Figure 4.13: Injury risk vs. $P_{\text{max}} \times \dot{P}_{\text{max}}$ for ex vivo tests
4.4 PMHS Biomechanical Variables and Injury Risk

Using the corrected data from Gustafson (2009) and the additional four PMHS impacts conducted in this study the same statistical analysis performed for the ex vivo drop tower tests was conducted. Table 4.4 shows the results of all the biomechanical predictor variables analyzed. Again, the statistically significant variables are highlighted in green. Plots of the statistically significant binary injury risk functions for the all biomechanical variables along with the non-parametric CT method curve are given in Figure 4.15 and Figure 4.16.

Data from the venous pressure sensors were used to calculate variables that could be used to develop injury risk curves. Tissue pressure was not considered in the analysis because of the limited number of data points (n=5) and because of uncertainty related to their accuracy.
Table 4.4: Summary of biomechanical variables and their use as risk predictors for PMHS data (n=10)

<table>
<thead>
<tr>
<th>Predictor Variable</th>
<th>Units</th>
<th>50% Risk Value</th>
<th>Log Likelihood</th>
<th>P-value</th>
<th>Goodman-Kruskal Gamma</th>
</tr>
</thead>
<tbody>
<tr>
<td>( P_{\text{max}} \cdot \dot{P}_{\text{max}} )</td>
<td>kPa²/ms</td>
<td>622</td>
<td>-4.323</td>
<td>0.028</td>
<td>0.75</td>
</tr>
<tr>
<td>Vascular ( \dot{P}_{\text{max}} )</td>
<td>kPa/ms</td>
<td>9.3</td>
<td>-4.191</td>
<td>0.024</td>
<td>0.58</td>
</tr>
<tr>
<td>Vascular ( P_{\text{max}} )</td>
<td>kPa</td>
<td>66.6</td>
<td>-5.252</td>
<td>0.086</td>
<td>0.50</td>
</tr>
<tr>
<td>( [\dot{F} \cdot C]_{\text{max}} )</td>
<td>N/ms</td>
<td>45.7</td>
<td>-5.969</td>
<td>0.217</td>
<td>0.33</td>
</tr>
<tr>
<td>AIC (( V_{\text{max}} \cdot C_{\text{max}} ))</td>
<td>m/s</td>
<td>2.27</td>
<td>-6.295</td>
<td>0.351</td>
<td>0.25</td>
</tr>
<tr>
<td>([V(t) \cdot C(t)]_{\text{max}})</td>
<td>m/s</td>
<td>1.05</td>
<td>-6.046</td>
<td>0.242</td>
<td>0.17</td>
</tr>
<tr>
<td>Compression</td>
<td>%</td>
<td>31.7</td>
<td>-6.261</td>
<td>0.333</td>
<td>0.17</td>
</tr>
</tbody>
</table>

Figure 4.15: Injury risk vs. \( P_{\text{max}} \cdot \dot{P}_{\text{max}} \) for PMHS tests
4.5 Combined PMHS and Ex Vivo Biomechanical Variables vs. Injury Risk

With the ultimate goal of the this study to develop a pressure-related biomechanical predictor for abdominal injury, specifically liver injury, the data sets were combined to see if any of the pressure-related variables could predict injury despite the different boundary conditions. Table 4.5 shows results of all the pressure-related variables analyzed. Again the statistically significant variables are highlighted in green. Plots of the binary injury risk functions for the pressure-related variables that were found significant along with the non-parametric CT method curve are given in Figure 4.17 through Figure 4.20.
Table 4.5: Summary of pressure-related variables and their use as risk predictors for combined PMHS and ex vivo data

<table>
<thead>
<tr>
<th>Predictor Variable</th>
<th>Units</th>
<th>50% Risk Value</th>
<th>Log Likelihood</th>
<th>P-value</th>
<th>Goodman-Kruskal Gamma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vascular $\dot{p}_{\text{max}}$</td>
<td>kPa/ms</td>
<td>10.2</td>
<td>-9.933</td>
<td>0.001</td>
<td>0.72</td>
</tr>
<tr>
<td>$p_{\text{max}} \times \dot{p}_{\text{max}}$</td>
<td>kPa²/ms</td>
<td>710</td>
<td>-11.759</td>
<td>0.008</td>
<td>0.65</td>
</tr>
<tr>
<td>Tissue $p_{\text{max}}$</td>
<td>kPa</td>
<td>66.5</td>
<td>-9.397</td>
<td>0.164</td>
<td>0.54</td>
</tr>
<tr>
<td>Compression</td>
<td>%</td>
<td>30.4</td>
<td>-13.688</td>
<td>0.015</td>
<td>0.50</td>
</tr>
<tr>
<td>Tissue $\dot{p}_{\text{max}}$</td>
<td>kPa/ms</td>
<td>12.1</td>
<td>-9.053</td>
<td>0.105</td>
<td>0.46</td>
</tr>
<tr>
<td>Vascular $p_{\text{max}}$</td>
<td>kPa</td>
<td>63.5</td>
<td>-13.125</td>
<td>0.039</td>
<td>0.45</td>
</tr>
<tr>
<td>AIC ($V_{\text{max}} \times C_{\text{max}}$)</td>
<td>m/s</td>
<td>1.49</td>
<td>-16.093</td>
<td>0.297</td>
<td>0.23</td>
</tr>
</tbody>
</table>

Figure 4.17: Injury risk vs. $\dot{p}_{\text{max}}$ for combined data sets
Figure 4.18: Injury risk vs. $P_{\text{max}}^* P_{\text{max}}$ for combined data sets

Figure 4.19: Injury risk vs. Compression for combined data sets
4.7 Summary and Comparison of Injury Risk Predictors

Table 4.6 through Table 4.8 summarize the biomechanical predictors and their usefulness as liver injury predictors. Also reported in the tables are the 50% risk values for each predictor. All predictors highlighted green were found to be statistically significant. Vascular $\dot{P}_{\text{max}}$ was the only biomechanical predictor that was found to be statistically significant in all cases (ex vivo, PMHS and combined data sets). Notice that the 50% risk value which ranges from 9.3-10.6 for the different data sets. This small range is encouraging in that the biomechanical predictor is comparable even for tests with drastically different boundary conditions. Also note that $P_{\text{max}} \cdot \dot{P}_{\text{max}}$ was found to be statistically significant for the PMHS and combined data sets and had a p-value=0.055 in the ex vivo data set.
Table 4.6: 50% risk values for *ex vivo* biomechanical predictors

<table>
<thead>
<tr>
<th>Predictor Variable</th>
<th>50% Risk Value</th>
<th>P-value</th>
<th>Goodman-Kruskal Gamma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tissue $\dot{P}_{\text{max}}$</td>
<td>8.69 kPa/ms</td>
<td>0.005</td>
<td>0.80</td>
</tr>
<tr>
<td>AIC ($V_{\text{max}} \cdot C_{\text{max}}$)</td>
<td>0.82 m/s</td>
<td>0.009</td>
<td>0.79</td>
</tr>
<tr>
<td>Vascular $\dot{P}_{\text{max}}$</td>
<td>10.6 kPa/ms</td>
<td>0.013</td>
<td>0.77</td>
</tr>
<tr>
<td>Tissue $P_{\text{max}}$</td>
<td>56.3 kPa</td>
<td>0.023</td>
<td>0.70</td>
</tr>
<tr>
<td>$P_{\text{max}} \cdot \dot{P}_{\text{max}}$</td>
<td>685 kPa²/ms</td>
<td>0.055</td>
<td>0.60</td>
</tr>
<tr>
<td>Compression</td>
<td>29.90%</td>
<td>0.034</td>
<td>0.58</td>
</tr>
</tbody>
</table>

Table 4.7: 50% risk values for PMHS biomechanical predictors

<table>
<thead>
<tr>
<th>Predictor Variable</th>
<th>50% Risk Value</th>
<th>P-value</th>
<th>Goodman-Kruskal Gamma</th>
</tr>
</thead>
<tbody>
<tr>
<td>$P_{\text{max}} \cdot \dot{P}_{\text{max}}$</td>
<td>622 kPa²/ms</td>
<td>0.028</td>
<td>0.75</td>
</tr>
<tr>
<td>Vascular $\dot{P}_{\text{max}}$</td>
<td>9.3 kPa/ms</td>
<td>0.024</td>
<td>0.58</td>
</tr>
</tbody>
</table>

Table 4.8: 50% risk values for combined data sets biomechanical predictors

<table>
<thead>
<tr>
<th>Predictor Variable</th>
<th>50% Risk Value</th>
<th>P-value</th>
<th>Goodman-Kruskal Gamma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vascular $\dot{P}_{\text{max}}$</td>
<td>10.2 kPa/ms</td>
<td>0.001</td>
<td>0.72</td>
</tr>
<tr>
<td>$P_{\text{max}} \cdot \dot{P}_{\text{max}}$</td>
<td>710 kPa²/ms</td>
<td>0.008</td>
<td>0.65</td>
</tr>
<tr>
<td>Compression</td>
<td>30.4%</td>
<td>0.015</td>
<td>0.50</td>
</tr>
<tr>
<td>Vascular $P_{\text{max}}$</td>
<td>63.5 kPa</td>
<td>0.039</td>
<td>0.45</td>
</tr>
</tbody>
</table>

### 4.8 Chestband Analysis

Deflection and compression were measured using a chestband comprised of 40 strain gauges. Maximum deflection was calculated by subtracting the initial position of
the gauge at impact location from the gauge located opposite of it along the line of impact. The deflection was normalized by the initial distance between the gauges in order to determine compression. These chestband contours for each impact are plotted at various points in time in Figure 4.21 through Figure 4.30. These contours hold stationary the gauge placed on the spine in order to make comparing deflection over various times easier to visualize. The average compression for the lateral impacts was 28.7% and the oblique impacts had an average compression of 30.5%, with FBL09-O having the maximum compression of 38%. Note that the contour at t=10 ms for FBL02-L is oddly shaped and is discussed in section 5.3.
Figure 4.23: FBL03-L chestband contours

Figure 4.24: FBL04-L chestband contours

Figure 4.25: FBL05-O chestband contours

Figure 4.26: FBL06-O chestband contours

Figure 4.27: FBL07-O chestband contours

Figure 4.28: FBL08-O chestband contours
4.9 Average Pressure vs. Compression

For each impact, all pressure sensors located in the venous vasculature were averaged to obtain an overall pressure response in the abdomen. The average pressures vs. compression relationships for the lateral and oblique tests are shown in Figure 4.31 and Figure 4.32 respectively. The pressure and compression data were plotted from t=0ms (impact) to time=50ms (after peak pressure and compression).
4.10 Normalization Values

Using the normalization techniques discussed in section 3.9, scaling factors were calculated to scale the data to represent a 50\textsuperscript{th} percentile male impacted under the loading conditions of the test. Table 4.9 shows the data used to calculate the characteristic ratios used to develop the scaling values. Table 4.10 shows the scaling factors for the Moorhouse method. Values for the Mertz and Viano and Eppinger methods can be found in Appendix F.

**Table 4.9: Data and characteristic ratios used in normalization**

<table>
<thead>
<tr>
<th>Test</th>
<th>Chest Breadth (cm)</th>
<th>( m_{\text{eff}} ) (kg)</th>
<th>( \lambda_{\text{m-epp}} )</th>
<th>( \lambda_{\text{m-eff}} )</th>
<th>( \lambda_{k}\text{-length} )</th>
<th>( \lambda_{k}\text{-eff} )</th>
</tr>
</thead>
<tbody>
<tr>
<td>FBL01-L</td>
<td>27.0</td>
<td>25.0</td>
<td>1.14</td>
<td>1.17</td>
<td>1.21</td>
<td>0.99</td>
</tr>
<tr>
<td>FBL02-L</td>
<td>31.0</td>
<td>20.6</td>
<td>1.29</td>
<td>1.43</td>
<td>1.05</td>
<td>1.56</td>
</tr>
<tr>
<td>FBL03-L</td>
<td>27.0</td>
<td>21.8</td>
<td>1.05</td>
<td>1.35</td>
<td>1.21</td>
<td>1.38</td>
</tr>
<tr>
<td>FBL04-L</td>
<td>27.3</td>
<td>23.4</td>
<td>1.20</td>
<td>1.25</td>
<td>1.20</td>
<td>1.18</td>
</tr>
<tr>
<td>FBL05-O</td>
<td>30.0</td>
<td>21.1</td>
<td>1.41</td>
<td>1.39</td>
<td>1.09</td>
<td>1.45</td>
</tr>
<tr>
<td>FBL06-O</td>
<td>31.0</td>
<td>36.7</td>
<td>0.93</td>
<td>0.80</td>
<td>1.05</td>
<td>0.98</td>
</tr>
<tr>
<td>FBL07-O</td>
<td>26.3</td>
<td>31.4</td>
<td>1.33</td>
<td>0.94</td>
<td>1.24</td>
<td>0.82</td>
</tr>
<tr>
<td>FBL08-O</td>
<td>30.5</td>
<td>23.4</td>
<td>1.11</td>
<td>1.26</td>
<td>1.07</td>
<td>1.03</td>
</tr>
<tr>
<td>FBL09-O</td>
<td>26.2</td>
<td>25.2</td>
<td>1.32</td>
<td>1.17</td>
<td>1.25</td>
<td>1.55</td>
</tr>
<tr>
<td>FBL10-L</td>
<td>28.5</td>
<td>16.3</td>
<td>1.42</td>
<td>1.80</td>
<td>1.15</td>
<td>1.11</td>
</tr>
</tbody>
</table>
4.11 Force and Displacement Time Histories

The applied force was calculated using the inertially compensated load cell located on the impactor. For two tests, FBL01-L and FBL06-L, the load cell failed and therefore the force was calculated by multiplying the impactor acceleration by its mass. The error between the two methods was calculated for the remaining (n=8) impacts with the maximum load reported from the accelerometer method on average 3.7% higher. The plots of the non-normalized force-time histories are given in Figure 4.33 (lateral) and Figure 4.34 (oblique). The plots of the normalized force-time histories for the Moorhouse method are shown in Figure 4.35 and Figure 4.36. The Moorhouse method was chosen because it best grouped the curves for the test group. Normalized force-time histories, deflection-time histories, and force-deflection curves using the other normalization methods can be found in Appendix F.

<table>
<thead>
<tr>
<th>Test</th>
<th>Displacement</th>
<th>Time</th>
<th>Force</th>
</tr>
</thead>
<tbody>
<tr>
<td>FBL01-L</td>
<td>1.03</td>
<td>1.04</td>
<td>1.03</td>
</tr>
<tr>
<td>FBL02-L</td>
<td>0.87</td>
<td>0.87</td>
<td>1.36</td>
</tr>
<tr>
<td>FBL03-L</td>
<td>0.92</td>
<td>0.91</td>
<td>1.26</td>
</tr>
<tr>
<td>FBL04-L</td>
<td>0.97</td>
<td>0.97</td>
<td>1.15</td>
</tr>
<tr>
<td>FBL05-O</td>
<td>0.89</td>
<td>0.90</td>
<td>1.30</td>
</tr>
<tr>
<td>FBL06-O</td>
<td>0.97</td>
<td>0.96</td>
<td>0.95</td>
</tr>
<tr>
<td>FBL07-O</td>
<td>1.10</td>
<td>1.09</td>
<td>0.89</td>
</tr>
<tr>
<td>FBL08-O</td>
<td>1.03</td>
<td>1.04</td>
<td>1.07</td>
</tr>
<tr>
<td>FBL09-O</td>
<td>0.83</td>
<td>0.83</td>
<td>1.29</td>
</tr>
<tr>
<td>FBL10-L</td>
<td>1.11</td>
<td>1.10</td>
<td>1.23</td>
</tr>
</tbody>
</table>
The deflection was calculated using the chestband data. The time-histories of the non-normalized and normalized data are shown in Figure 4.37 through Figure 4.40, again separating the lateral and oblique tests.
4.12 Force-Deflection Curves

The response of the thoraco-abdominal region may be characterized by force-displacement curves. These curves provide useful information for designing ATDs. Plots were created for both the non-normalized and normalized data (Moorhouse method). These can be found in Figure 4.41 through Figure 4.44. Again, normalized force-time histories, deflection-time histories, and force-deflection curves using the other normalization methods can be found in Appendix F.
Figure 4.41: Non-normalized force-deflection response (lateral)

Figure 4.42: Non-normalized force-deflection response (oblique)

Figure 4.43: Normalized force-deflection response (lateral), Moorhouse normalization method

Figure 4.44: Normalized force-deflection response (oblique), Moorhouse normalization method
Chapter 5: Discussion

5.1 Injuries

The injuries sustained to the ten PMHS from the lateral and oblique abdomen impacts are displayed in Table 5.1. Liver injury of AIS $\geq 3$ was obtained for four out of ten tests, with three of those occurring during oblique impacts. One possible reason for this injury pattern could be due to the liver compressing against the spine. In the lateral impacts the liver is more likely to move in front of the spine instead of compressing against it. In oblique impacts if the liver follows the direction of force it is probable that the liver will be loaded against the spine and not have the opportunity to slide in front of it. This loading against the spine would likely increase the pressure and rate of pressure change in the liver, which was determined from this study to correlate well to liver injury, resulting in more injuries during the oblique impacts than what was seen in the lateral impacts.

Due to the anatomical location of the liver behind the rib cage, loading the rib cage was unavoidable, and, as a result, skeletal injuries resulted from all impacts. All tests resulted in at least two rib fractures. The number of skeletal injuries varied greatly despite impacting all PMHS at the same velocity. One of the contributing factors to this was probably the difference in bone mineral density (BMD).
Another thing to notice in Table 5.1 is that there can be large discrepancies in the skeletal and liver AIS injury severities scores from the same impact. For instance, in test FBL02-L, the subject sustained an AIS = 5 skeletal injury while sustaining no injury to the liver. On the opposite end, in test FBL08-O, the subject sustained an AIS = 3 skeletal injury while sustaining an AIS = 5 liver injury. This suggests that there is a need to have multiple injury criteria, skeletal and soft tissue, for ATD design in order to determine that maximum AIS injury sustained to an occupant.

Table 5.1: Injuries sustained to PMHS lateral and oblique impacts

<table>
<thead>
<tr>
<th>Test Number</th>
<th>Ribs Injury Description</th>
<th>AIS Level</th>
<th>Liver Injury Description</th>
<th>AIS Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>FBL01-L</td>
<td>Fractures of ribs 5 and 6 on the right side</td>
<td>2</td>
<td>Minor laceration on the right inferior aspect of the liver</td>
<td>2</td>
</tr>
<tr>
<td>FBL02-L</td>
<td>Bilateral flail chest</td>
<td>5</td>
<td>none</td>
<td>0</td>
</tr>
<tr>
<td>FBL03-L</td>
<td>Unilateral flail chest</td>
<td>4</td>
<td>Burst injury to the liver on the posterior side of the liver, primarily to the right lobe of the liver</td>
<td>4</td>
</tr>
<tr>
<td>FBL04-L</td>
<td>Unilateral flail chest</td>
<td>4</td>
<td>none</td>
<td>0</td>
</tr>
<tr>
<td>FBL05-O</td>
<td>Unilateral flail chest</td>
<td>4</td>
<td>Lacerations on the anterior and posterior right lobe, areas of capsular damage</td>
<td>2</td>
</tr>
<tr>
<td>FBL06-O</td>
<td>Unilateral flail chest</td>
<td>4</td>
<td>Three transverse lacerations across anterior surface of liver, right lobe tissue disruption</td>
<td>2</td>
</tr>
<tr>
<td>FBL07-O</td>
<td>Unilateral flail chest</td>
<td>4</td>
<td>Burst injury to the liver on the inferior, posterior side primarily to the right lobe</td>
<td>4</td>
</tr>
<tr>
<td>FBL08-O</td>
<td>Fractures of ribs 3-8 on the right side</td>
<td>3</td>
<td>Lacerations on the anterior and posterior right lobe, burst locations on inferior, right and caudate lobes, pulpefication of liver</td>
<td>5</td>
</tr>
<tr>
<td>FBL09-O</td>
<td>Unilateral flail chest</td>
<td>4</td>
<td>Catastrophic parenchymal disruption, burst injury plus pulpefication</td>
<td>5</td>
</tr>
<tr>
<td>FBL10-L</td>
<td>Unilateral flail chest</td>
<td>4</td>
<td>none</td>
<td>0</td>
</tr>
</tbody>
</table>

In addition to the four AIS ≥ 3 injuries sustained, the test series included three tests with laceration (AIS=2) injuries. The lacerations for these three tests did not appear to be the result of rib fractures since there were no displaced rib fractures corresponding
to the laceration locations. These lacerations are likely due to high stresses in the capsule of the liver caused by the face of the impactor. Table 5.2 compares injuries from the CIREN database to the injuries sustained in the combined PMHS test series. It was found that the injuries sustained in the study were realistic injuries that occur from motor vehicle collisions.

Table 5.2: Comparison of liver injury images from the CIREN database and PMHS test series

<table>
<thead>
<tr>
<th>CIREN Image</th>
<th>Description</th>
<th>Test Image</th>
</tr>
</thead>
<tbody>
<tr>
<td><img src="image1" alt="Image" /></td>
<td>Superficial laceration of the capsule</td>
<td><img src="image2" alt="Image" /></td>
</tr>
<tr>
<td><img src="image3" alt="Image" /></td>
<td>Serious parenchymal disruption</td>
<td><img src="image4" alt="Image" /></td>
</tr>
<tr>
<td><img src="image5" alt="Image" /></td>
<td>Multiple superficial parallel lacerations (“bear claw” lacerations)</td>
<td><img src="image6" alt="Image" /></td>
</tr>
<tr>
<td><img src="image7" alt="Image" /></td>
<td>Parenchymal disruption</td>
<td><img src="image8" alt="Image" /></td>
</tr>
</tbody>
</table>
5.2 Pressure and Pressure-related Variables

For this study, it was decided that the focus should be on pressure sensors located in the venous vasculature. These sensors were chosen for a few reasons, the first reason being the location of the sensors. The venous sensors were located either in the hepatic veins or in the IVC at the level of the liver and therefore more accurately measure the pressure change experienced by the liver. The sensors were also located closer to the impact location resulting in higher pressure readings. Secondly, the liver is mainly supplied with blood from the venous system (75%) and therefore it is expected that the pressure in the venous vasculature would more closely agree to pressures inside the liver.

For each impact, multiple pressure sensors were located in the venous vasculature providing a local ‘pressure map’ of the area around and inside the liver. The traces were typically similar but there were variations in the peak values for both pressure and rate of pressure. For this analysis it was assumed that if the liver were to fail it would be most likely caused by the highest pressure. Using this assumption, the sensor with the highest maximum pressure and highest maximum rate of pressure change was used to create the risk curves.

However, it should be noted that it was determined to exclude the sensor deep in the hepatic vein in test FBL06-O from the analysis. Despite not having an AIS ≥ 3 injury the sensor reported a high pressure reading. However, the liver in FBL06-O did report “right lobe tissue disruption”, Table 5.1. It is hypothesized that this sensor reported a higher reading because it was reading pressures at this injury location. Also, on the tests which did have liver injury of AIS ≥ 3 it was typically on the posterior, lateral aspect of the liver observed in Figure 5.1. This suggests that the pressures at the failure location
are higher than the values reported in this report, since most of the pressure sensors in the
venous vasculature were placed in the IVC or slightly into the hepatic vessels and not
deep into the hepatic on the lateral aspect of the liver where serious liver damage
typically occurred, Figure 5.2. This may also explain why the vascular $P_{\text{max}}$ 50% risk
value is higher for the *ex vivo* data set (pressure transducers located deep into hepatic
vessels) than 50% risk value for the PMHS study. In future studies it may be
advantageous to develop a method to route multiple sensors into the hepatic veins as deep
as possible to get pressure readings closer to the failure location.

![Figure 5.1: Serious liver injuries located on posterior, lateral aspect of liver](image1)

![Figure 5.2: Figure showing pressure transducer locations](image2)
This has important implications if one were to implement a fluid filled abdomen in an anthropomorphic test device (ATD) instrumented with pressure sensors and use the proposed pressure-related biomechanical predictors presented here as the actual 50% risk values may be higher than what is reported here.

From the analysis of the biomechanical predictors for the PMHS data (summarized in Table 4.4) it was determined that $P_{\text{max}} \times \dot{P}_{\text{max}}$ was the best predictor of liver injury of AIS $\geq 3$ ($p=0.028$). This result is encouraging because many other predictors for abdomen injuries like the abdominal injury criterion ($V_{\text{max}} \times C_{\text{max}}$) and the viscous criterion ($[V(t) \times C(t)]_{\text{max}}$) suggest that abdominal and soft tissue injury are rate dependent. It is also encouraging that vascular $\dot{P}_{\text{max}}$ was the only biomechanical predictor considered in this study to be found as a statistically significant predictor for liver injury for the ex vivo, PMHS and combined data sets. Notice also that the 50% risk value for $\dot{P}_{\text{max}}$ ranges only from 9.3-10.6. This small range is encouraging in that the biomechanical predictor is comparable even for tests with drastically different boundary conditions. And as stated earlier, this slight difference could be due to the fact that for the ex vivo liver test, the pressure sensors could be routed deeper into the hepatic vessels near the site of injury. A summary of the 50% risk values for the pressure related variables is located in Table 5.3.
Table 5.3: Comparison of 50% risk values for AIS ≥ 3 for all testing

<table>
<thead>
<tr>
<th>Predictor Variable</th>
<th>Data Set</th>
<th>50% Risk Value</th>
<th>P-value</th>
<th>Goodman-Kruskal Gamma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vasculature $\dot{P}_{\text{max}}$</td>
<td>PMHS</td>
<td>9.3 kPa/ms</td>
<td>0.024</td>
<td>0.58</td>
</tr>
<tr>
<td>Ex Vivo</td>
<td>10.6 kPa/ms</td>
<td>0.013</td>
<td>0.77</td>
<td></td>
</tr>
<tr>
<td>Combined</td>
<td>10.2 kPa/ms</td>
<td>0.001</td>
<td>0.72</td>
<td></td>
</tr>
<tr>
<td>$P_{\text{max}}*\dot{P}_{\text{max}}$</td>
<td>PMHS</td>
<td>622 kPa2/ms</td>
<td>0.028</td>
<td>0.75</td>
</tr>
<tr>
<td>Ex Vivo</td>
<td>685 kPa2/ms</td>
<td>0.055</td>
<td>0.60</td>
<td></td>
</tr>
<tr>
<td>Combined</td>
<td>710 kPa2/ms</td>
<td>0.008</td>
<td>0.65</td>
<td></td>
</tr>
</tbody>
</table>

Intuitively $\dot{P}_{\text{max}}$ and $P_{\text{max}}*\dot{P}_{\text{max}}$ as predictors of liver injury makes sense because a combination of both the rate at which you increase pressure and the total pressure should have an effect on liver injury levels, especially burst injuries. For example, imagine the liver as if it were a water balloon. Slowly loading the balloon with increasing force will eventually pop the balloon because its maximum pressure was reached. However, in this scenario the balloon would most likely reach a higher pressure ($P_{\text{max}}$) than if it were loaded with the same amount of force but much more quickly. This idea helps explain why the rate of pressure change ($\dot{P}_{\text{max}}$) also needs to be considered when thinking about injury for soft tissue organs like the liver.

5.3 Chestband Analysis

The chestband was used as a way to record deformation of the impacted region over time. Figure 4.21 through Figure 4.30 show how the impacted region for each test deformed at various time intervals. Again, it should be noted that the gauge on the spine was held fix in order the make comparing deflections throughout time more easily visual. The average compression for the lateral impacts was 28.7% and the oblique impacts had
an average compression of 30.5%. However, the oblique average was skewed with FBL09-O having a maximum compression of 38%. This compression was more than 6% higher than the next closest compression level of 32% seen in FBL05-O. There are a few possible reasons why this subject had a much higher compression than the others. The first reason is that the subject’s BMD was the lowest (T-score=-2.1). The subject was also had the smallest chest breadth and was the fourth lightest. The combination of these factors likely resulted in a higher maximum compression. If the average is calculated for the oblique impacts excluding FBL09-O it comes out to 28.6% which is near that of the lateral impacts.

From the contour plots it can also be seen that maximum compression happened between 22.9 ms and 28.4 ms. For all tests there was minimal additional compression past 20ms. One major limitation of the chestband is that it is not attached to the skin of the subject. Because of this the chestband may not be in contact with the region of interest during the entire duration of the impact and therefore does not truly represent skeletal deflection. For instance in FBL02-L the contour at t=10ms shown seen in Figure 4.22 is drastically different from the other responses. It is unsure what is happening at this time. It is hypothesized that the chestband was not initially wrapped tightly around the subject prior to impact and because of this the initial loading caused the chestband to initially bend extend away from the impact area and then was able to recover and follow the face of the impactor.
5.4 Pressure vs. Compression

With the limited number of data points for both lateral (n=5) and oblique (n=5) tests, only generalized observations should be made from the average pressure vs. compression curves seen in Figure 4.31 and Figure 4.32. One thing that was noticed is that for all cases the maximum pressure was reached prior to maximum compression. From the limited sample size, it looks that in general the lateral and oblique impacts resulted in similar responses.

5.5 Force and Deflection Time Histories

The time histories of the non-normalized and normalized force (Moorhouse method) are shown in Figure 4.33 through Figure 4.36. It can be seen that when normalizing using the Moorhouse method that the variability of the curves decrease for both the lateral impacts and oblique impacts. For both lateral and oblique impacts the peak normalized force is near 4500 N and occurs between 5 ms and 10 ms.

Figure 4.37 through Figure 4.40 show the non-normalized and normalized time histories for deflection. Here again, the normalization method decreased the variability of both the lateral and oblique curves. For both lateral and oblique impacts the maximum normalized deflection is near 80mm between 20 ms and 25 ms. The same observations hold true for the non-normalized and normalized force-deflection curves seen in Figure 4.41 through Figure 4.44.

5.7 Limitations

For the testing conducted by Gustafson (2009), the PMHS impacts (n=6) aligned the bottom edge of the impactor with rib eleven at the mid-axillary line on the right side
of the subject. This location was selected to maximize the loading of the liver of the impactor for all subjects based on the typical anatomical location of the liver. However, due to anatomical differences in the PMHS the actual amount of the liver loaded may have varied from test to test. The last four PMHS impacts used a C-arm fluoroscope to locate the liver while in the seated position. This procedure helped ensure more direct loading of the liver for these impacts.

Another limitation to the combined PMHS testing was that most of the pressure sensors in the venous vasculature were placed in the IVC or slightly into the hepatic vessels and serious injury to the liver typically occurred to the lateral aspect of the liver. For future testing it is suggested that a method to route multiple sensors into the hepatic veins as deep as possible be developed to get pressure readings closer to the failure location of the liver.

One of the main limitations of this study is the limited number of data points. Even though there were pressure-related variables that were deemed statistically significant predictors of liver injury of AIS ≥ 3, the results should be used cautiously until there is a larger data set supporting the findings. Another limitation of this study is that the impact speeds were not varied for the impacts. Because of this, it was expected that the AIC and viscous criterion values were to be very similar among tests and which may have contributed to them being determined as poor predictors of injury in the current study.

It is hoped that future work will be conducted in order to obtain more data points so that the pressure-related variables discussed in this study can be further validated. It is
also suggested that techniques to place the pressure sensors deep in the hepatic veins be
developed to better measure pressure at or near the location of liver injury.
Chapter 6: Conclusions

This test series used full-body PMHS with re-pressurized abdominal vasculature to study abdominal injury, specifically liver injury. A total of five lateral and five oblique impacts were performed, all at a nominal velocity near 7.0 m/s. This study also reanalyzed data from *ex vivo* liver impacts performed by Sparks et al (2007). The following conclusions from the work were made:

1. The PMHS tests successfully produced injuries similar to those documented in the CIREN trauma database, including four AIS ≥ 3 liver injuries.
2. The consistent threshold (CT) method was used to estimate the shape of the injury risk curve.
3. Binary logistic regression risk functions were created to determine the usefulness of biomechanical predictors. It was determined that $P_{\text{max}} \times \dot{P}_{\text{max}}$ (p-value=0.028 and Goodman-Kruskal Gamma=0.75) was the strongest predictor for AIS ≥ 3 liver injury for the PMHS data set.
4. A value of $\dot{P}_{\text{max}}$ of 9.3 kPa/ms corresponded to 50% risk of AIS ≥ 3 liver injury for the PMHS data set (p-value=0.024). $\dot{P}_{\text{max}}$ was also determined to be a good predictor for the *ex vivo* liver test data (p-value=0.013, 50% risk=10.6 kPa/ms) suggesting that $\dot{P}_{\text{max}}$ is a strong predictor for liver injury of AIS ≥ 3 regardless of boundary conditions.
5. For the PMHS trials most of the pressure sensors in the venous vasculature were placed in the IVC or slightly into the hepatic vessels and not at the lateral aspect of the liver where serious liver damage typically occurred. The pressures at the location or injury are likely higher than the values reported here. For future studies it is suggested that a method to route multiple sensors into the hepatic veins as deep as possible be developed to get pressure readings closer to the failure location.

6. No other biomechanical predictor was found to be significant for both sets of data.

The goal of this study was to relate pressure-related variables to abdominal injury, specifically liver injury. From the data $\dot{P}_{\text{max}}$ and $P_{\text{max}} \cdot \dot{P}_{\text{max}}$ looks to be a good predictor of injury and should be studied further. Implementing fluid-filled abdominal inserts into ATDs with pressure sensors could then be implemented using the findings of this study.
References


Appendix A: Complete anthropometry
## Subject Anthropometry

| Impact Direction | FBL01-L | FBL02-L | FBL03-L | FBL04-L | FBL05-O | FBL06-O | FBL07-O | FBL08-O | FBL09-O | FBL10-L | Average | Standard Deviation |
|------------------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------------------|
| Gender           | Male    | Female  | Male    | Male    | Female  | Male    | Male    | Female  | Female  | Female  | -       | -       |
| Age              | 68      | 80      | 88      | 91      | 53      | 79      | 82      | 77      | 78      | 65      | 76.1    | 11.3    |
| Mass (kg)        | 67      | 59      | 73      | 64      | 54      | 82      | 57      | 69      | 58      | 53.5    | 63.65   | 9.2     |
| Stature (cm)     | 176     | 154     | 188     | 179     | 164     | 179     | 171     | 157     | 171     | 166     | 170.5   | 10.5    |
| Chest Breadth (cm) | 26.9   | 30.8    | 27.0    | 27.3    | 30.0    | 30.8    | 26.3    | 30.5    | 26.2    | 28.5    | 28.4    | 1.9     |
| Waist Breadth (cm) | 25.8   | 33.0    | 30.0    | 29.5    | 30.0    | 35.0    | 27.0    | 40.5    | 30.7    | 31.9    | 31.3    | 4.2     |
| Seated Height (cm) | 94     | 87      | 96      | 97      | 92      | 99      | 94      | 80      | 90      | 92      | 92      | 5.5     |
| Chest Circumference (cm) | 90     | 94      | 93      | 90      | 87      | 98      | 86      | 91      | 78      | 81      | 89      | 6.0     |
| Waist Waist (cm) | 84      | 91      | 88      | 90      | 86      | 94      | 80      | 105     | 87      | 85      | 89      | 6.8     |
| Chest Depth (cm) | 20      | 19      | 19      | 19      | 17      | 23      | 19      | 18      | 14      | 17      | 19      | 2.3     |
| Waist Depth (cm) | 18      | 17      | 14      | 21      | 15      | 22      | 16      | 19      | 17      | 17      | 18      | 2.5     |
Appendix B : FFT Signal Analysis
Because there is not a standard was filter class for pressure data defined by J211 a FFT analysis of the signals was performed seen below. Because most of the signal is comprised of frequencies below 100 Hz a CFC60 filter was decided upon.

Signal analysis of an example PMHS impact (FBL08-O)

Signal analysis of an example *ex vivo* impact (HL05)
Appendix C : Injuries
Rib Fractures

<table>
<thead>
<tr>
<th>Test Number</th>
<th>Injury Description</th>
<th>AIS Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>FBL01-L</td>
<td>Fractures of ribs 5 and 6 on the right side</td>
<td>2</td>
</tr>
<tr>
<td>FBL02-L</td>
<td>Fractures on ribs 2-12 on the right side and ribs 2-4 and 7 and 8 on the left side, bi-lateral flail chest</td>
<td>5</td>
</tr>
<tr>
<td>FBL03-L</td>
<td>Fractures of ribs 6-11 on the right side, unilateral flail chest</td>
<td>3</td>
</tr>
<tr>
<td>FBL04-L</td>
<td>Fractures of ribs 3-12 on the right side and ribs 9 and 10 on the left side, unilateral flail chest</td>
<td>3</td>
</tr>
<tr>
<td>FBL05-O</td>
<td>Fractures of ribs 4-9 on the right side, unilateral flail chest</td>
<td>3</td>
</tr>
<tr>
<td>FBL06-O</td>
<td>Fractures of right ribs 3-10, unilateral flail chest</td>
<td>3</td>
</tr>
<tr>
<td>FBL07-O</td>
<td>Fractures of ribs 3-9 on the right side, unilateral flail chest</td>
<td>4</td>
</tr>
<tr>
<td>FBL08-O</td>
<td>Fractures of ribs 3-8 on the right side</td>
<td>3</td>
</tr>
<tr>
<td>FBL09-O</td>
<td>Fracture of ribs 4-10 on the right side and rib 10 on the left side, unilateral flail chest</td>
<td>4</td>
</tr>
<tr>
<td>FBL10-L</td>
<td>Fracture of ribs 3-11 on the right side and ribs 5, 7-9 on the left side, unilateral flail chest</td>
<td>4</td>
</tr>
</tbody>
</table>

Plotted here are the documented locations of the rib fractures relative to the sternal notch. At autopsy the rib fracture distances were recorded measuring down from the sternal notch and over from the sternal midline. Each rib is indicated by color.

*Taken from Gustafson, 2009

*Taken from Gustafson, 2009
*Taken from Gustafson, 2009
Liver Injuries

<table>
<thead>
<tr>
<th>Test Number</th>
<th>Injury Description</th>
<th>AIS Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>FBL01-L</td>
<td>Minor laceration on the right inferior aspect of the liver</td>
<td>2</td>
</tr>
<tr>
<td>FBL02-L</td>
<td>none</td>
<td>0</td>
</tr>
<tr>
<td>FBL03-L</td>
<td>Burst injury to the liver on the posterior side of the liver, primarily to the right lobe of the liver</td>
<td>4</td>
</tr>
<tr>
<td>FBL04-L</td>
<td>none</td>
<td>0</td>
</tr>
<tr>
<td>FBL05-O</td>
<td>Lacerations on the anterior and posterior right lobe, areas of capsular damage</td>
<td>2</td>
</tr>
<tr>
<td>FBL06-O</td>
<td>Three transverse lacerations across anterior surface of liver, right lobe tissue disruption</td>
<td>2</td>
</tr>
<tr>
<td>FBL07-O</td>
<td>Burst injury to the liver on the inferior, posterior side primarily to the right lobe</td>
<td>4</td>
</tr>
<tr>
<td>FBL08-O</td>
<td>Lacerations on the anterior and posterior right lobe, burst locations on inferior, right and caudate lobes, pulpefication of liver</td>
<td>5</td>
</tr>
<tr>
<td>FBL09-O</td>
<td>Catastrophic parenchymal disruption, burst injury plus pulpefication</td>
<td>5</td>
</tr>
<tr>
<td>FBL10-L</td>
<td>none</td>
<td>0</td>
</tr>
</tbody>
</table>
Appendix D : CT 3-D Volume Renderings
No info available for FBL02-L
Appendix E : Pressure Time Histories
FBL01-L Pressure Traces

Arterial Traces

Venous Traces

FBL02-L Pressure Traces

Arterial Traces

Venous Traces

FBL03-L Pressure Traces

Arterial Traces

Venous Traces
Appendix F: Normalized Force and Deflection Data
### Mertz and Viano Normalization Values

<table>
<thead>
<tr>
<th>Test</th>
<th>Displacement</th>
<th>Time</th>
<th>Force</th>
</tr>
</thead>
<tbody>
<tr>
<td>FBL01-L</td>
<td>0.98</td>
<td>0.99</td>
<td>1.20</td>
</tr>
<tr>
<td>FBL02-L</td>
<td>1.12</td>
<td>1.12</td>
<td>1.17</td>
</tr>
<tr>
<td>FBL03-L</td>
<td>1.04</td>
<td>1.03</td>
<td>1.24</td>
</tr>
<tr>
<td>FBL04-L</td>
<td>1.01</td>
<td>1.01</td>
<td>1.21</td>
</tr>
<tr>
<td>FBL05-O</td>
<td>1.08</td>
<td>1.09</td>
<td>1.19</td>
</tr>
<tr>
<td>FBL06-O</td>
<td>0.98</td>
<td>0.98</td>
<td>1.03</td>
</tr>
<tr>
<td>FBL07-O</td>
<td>0.94</td>
<td>0.93</td>
<td>1.15</td>
</tr>
<tr>
<td>FBL08-O</td>
<td>1.06</td>
<td>1.07</td>
<td>1.15</td>
</tr>
<tr>
<td>FBL09-O</td>
<td>0.97</td>
<td>0.97</td>
<td>1.21</td>
</tr>
<tr>
<td>FBL10-L</td>
<td>1.15</td>
<td>1.14</td>
<td>1.31</td>
</tr>
</tbody>
</table>

### Eppinger Normalization Values

<table>
<thead>
<tr>
<th>Test</th>
<th>Displacement</th>
<th>Time</th>
<th>Force</th>
</tr>
</thead>
<tbody>
<tr>
<td>FBL01-L</td>
<td>1.03</td>
<td>1.04</td>
<td>1.08</td>
</tr>
<tr>
<td>FBL02-L</td>
<td>1.09</td>
<td>1.09</td>
<td>1.18</td>
</tr>
<tr>
<td>FBL03-L</td>
<td>1.03</td>
<td>1.02</td>
<td>1.04</td>
</tr>
<tr>
<td>FBL04-L</td>
<td>1.06</td>
<td>1.06</td>
<td>1.13</td>
</tr>
<tr>
<td>FBL05-O</td>
<td>1.11</td>
<td>1.12</td>
<td>1.25</td>
</tr>
<tr>
<td>FBL06-O</td>
<td>0.98</td>
<td>0.98</td>
<td>0.96</td>
</tr>
<tr>
<td>FBL07-O</td>
<td>1.11</td>
<td>1.10</td>
<td>1.22</td>
</tr>
<tr>
<td>FBL08-O</td>
<td>1.03</td>
<td>1.04</td>
<td>1.06</td>
</tr>
<tr>
<td>FBL09-O</td>
<td>1.09</td>
<td>1.10</td>
<td>1.19</td>
</tr>
<tr>
<td>FBL10-L</td>
<td>1.13</td>
<td>1.12</td>
<td>1.27</td>
</tr>
</tbody>
</table>
Normalized force-time histories using Mertz-Viano and Eppinger methods

Normalized force-time history (lateral).  
Mertz-Viano normalization method

Normalized force-time history (oblique).  
Mertz-Viano normalization method

Normalized force-time history (lateral).  
Eppinger normalization method

Normalized force-time history (oblique).  
Eppinger normalization method
Normalized deflection-time histories using Mertz-Viano and Eppinger methods

Normalized deflection-time history (lateral). Mertz-Viano normalization method

Normalized deflection-time history (oblique). Mertz-Viano normalization method

Normalized deflection-time history (lateral). Eppinger normalization method

Normalized deflection-time history (oblique). Eppinger normalization method
Normalized deflection-time histories using Mertz-Viano and Eppinger methods

Normalized force-deflection response (lateral), Mertz-Viano normalization method

Normalized force-deflection response (oblique), Mertz-Viano normalization method

Normalized force-deflection response (lateral), Eppinger normalization method

Normalized force-deflection response (oblique), Eppinger normalization method
Appendix G : Example MATLAB Correction Factor Code
%% Correction Factor Code
%% Impact001
clc
close all
clear all
% Load Data
load Impact001.csv
load Impact002.csv
load Impact003.csv
load Impact004.csv
load Impact005.csv
% Define Variables
time1=Impact001(:,1);
time2=Impact002(:,1);
time3=Impact003(:,1);
time4=Impact004(:,1);
time5=Impact005(:,1);
% Filter
[b,a] = butter(6,100/10000);
Impact001=filter(b,a,Impact001);
Impact002=filter(b,a,Impact002);
Impact003=filter(b,a,Impact003);
Impact004=filter(b,a,Impact004);
Impact005=filter(b,a,Impact005);
% Right Lobe
rfull1=Impact001(:,9);
rhalf1=Impact001(:,10);
rfull2=Impact002(:,9);
rhalf2=Impact002(:,10);
rfull3=Impact003(:,9);
rhalf3=Impact003(:,10);
rfull4=Impact004(:,9);
rhalf4=Impact004(:,10);
rfull5=Impact005(:,9);
rhalf5=Impact005(:,10);
% Left Lobe
lfull1=Impact001(:,11);
lhalf1=Impact001(:,12);
lfull2=Impact002(:,11);
lhalf2=Impact002(:,12);
lfull3=Impact003(:,11);
lhalf3=Impact003(:,12);
lfull4=Impact004(:,11);
lhalf4=Impact004(:,12);
lfull5=Impact005(:,11);
lhalf5=Impact005(:,12);
% Center Lobe
cfull1=Impact001(:,13);
chalf1=Impact001(:,14);
cfull2=Impact002(:,13);
chalf2=Impact002(:,14);
cfull3=Impact003(:,13);
chalf3=Impact003(:,14);
cfull4=Impact004(:,13);
chalf4=Impact004(:,14);
cfull5=Impact005(:,13);
chalf5=Impact005(:,14);

%%
% Assign variable names to each column from 'Data'
TR1=time1;
XR1=rfull1;
YR1=rhalf1;
TR2=time2;
XR2=rfull2;
YR2=rhalf2;
TR3=time3;
XR3=rfull3;
YR3=rhalf3;
%Assign variable names to each column from 'Data'
TL1=time1;
XL1=lfull1;
YL1=lhalf1;
TL2=time2;
XL2=lfull2;
YL2=lhalf2;
TL3=time3;
XL3=lfull3;
YL3=lhalf3;
TL4=time4;
XL4=lfull4;
YL4=lhalf4;
TL5=time5;
XL5=lfull5;
YL5=lhalf5;

%Assign variable names to each column from 'Data'
TC1=time1;
XC1=cfull1;
YC1=chalf1;
TC2=time2;
XC2=cfull2;
YC2=chalf2;
TC3=time3;
XC3=cfull3;
YC3=chalf3;
TC4=time4;
XC4=cfull4;
YC4=chalf4;
TC5=time5;
XC5=cfull5;
YC5=chalf5;

%Find point of peak measured pressure
[PeakR1,IR1] = max(XR1);
[PeakL1,IL1] = max(XL1);
[PeakC1,IC1] = max(XC1);
[PeakR2,IR2] = max(XR2);
[PeakL2,IL2] = max(XL2);
[PeakC2,IC2] = max(XC2);
[PeakR3,IR3] = max(XR3);
[PeakL3,IL3] = max(XL3);
[PeakC3,IC3] = max(XC3);
[PeakR4,IR4] = max(XR4);
[PeakL4,IL4] = max(XL4);
[PeakC4,IC4] = max(XC4);
[PeakR5,IR5] = max(XR5);
[PeakL5,IL5] = max(XL5);
[PeakC5,IC5] = max(XC5);

%Fit interval for C
CminR1=IR1-1; CmaxR1=IR1+1;
CminL1=IL1-1; CmaxL1=IL1+1;
CminC1=IC1-1; CmaxC1=IC1+1;
CminR2=IR2-1; CmaxR2=IR2+1;
CminL2=IL2-1; CmaxL2=IL2+1;
CminC2=IC2-1; CmaxC2=IC2+1;
CminR3=IR3-1; CmaxR3=IR3+1;
CminL3=IL3-1; CmaxL3=IL3+1;
CminC3=IC3-1; CmaxC3=IC3+1;
CminR4=IR4-1; CmaxR4=IR4+1;
CminL4=IL4-1; CmaxL4=IL4+1;
CminC4=IC4-1; CmaxC4=IC4+1;
CminR5=IR5-1; CmaxR5=IR5+1;
CminL5=IL5-1; CmaxL5=IL5+1;
% Calculate C using cumulative variance formulation
Cmin = IC5 - 1; Cmax = IC5 + 1;

% Calculate C using cumulative variance formulation

CR1num = sum(XR1(CminR1:CmaxR1).*YR1(CminR1:CmaxR1));
CR1den = sum(YR1(CminR1:CmaxR1).^2);

CL1num = sum(XL1(CminL1:CmaxL1).*YL1(CminL1:CmaxL1));
CL1den = sum(YL1(CminL1:CmaxL1).^2);

CC1num = sum(XC1(CminC1:CmaxC1).*YC1(CminC1:CmaxC1));
CC1den = sum(YC1(CminC1:CmaxC1).^2);

CR2num = sum(XR2(CminR2:CmaxR2).*YR2(CminR2:CmaxR2));
CR2den = sum(YR2(CminR2:CmaxR2).^2);

CL2num = sum(XL2(CminL2:CmaxL2).*YL2(CminL2:CmaxL2));
CL2den = sum(YL2(CminL2:CmaxL2).^2);

CC2num = sum(XC2(CminC2:CmaxC2).*YC2(CminC2:CmaxC2));
CC2den = sum(YC2(CminC2:CmaxC2).^2);

CR3num = sum(XR3(CminR3:CmaxR3).*YR3(CminR3:CmaxR3));
CR3den = sum(YR3(CminR3:CmaxR3).^2);

CL3num = sum(XL3(CminL3:CmaxL3).*YL3(CminL3:CmaxL3));
CL3den = sum(YL3(CminL3:CmaxL3).^2);

CC3num = sum(XC3(CminC3:CmaxC3).*YC3(CminC3:CmaxC3));
CC3den = sum(YC3(CminC3:CmaxC3).^2);

CR4num = sum(XR4(CminR4:CmaxR4).*YR4(CminR4:CmaxR4));
CR4den = sum(YR4(CminR4:CmaxR4).^2);

CL4num = sum(XL4(CminL4:CmaxL4).*YL4(CminL4:CmaxL4));
CL4den = sum(YL4(CminL4:CmaxL4).^2);

CC4num = sum(XC4(CminC4:CmaxC4).*YC4(CminC4:CmaxC4));
CC4den = sum(YC4(CminC4:CmaxC4).^2);

CR5num = sum(XR5(CminR5:CmaxR5).*YR5(CminR5:CmaxR5));
CR5den = sum(YR5(CminR5:CmaxR5).^2);

CL5num = sum(XL5(CminL5:CmaxL5).*YL5(CminL5:CmaxL5));
CL5den = sum(YL5(CminL5:CmaxL5).^2);

CC5num = sum(XC5(CminC5:CmaxC5).*YC5(CminC5:CmaxC5));
CC5den = sum(YC5(CminC5:CmaxC5).^2);

% Total C-factor
Ctot = (CR1num + CL1num + CC1num + CR2num + CL2num + CC2num + CR3num + CL3num + CC3num + CR4num + CL4num + CC4num + CR5num + CL5num + CC5num)/(CR1den + CL1den + CC1den + CR2den + CL2den + CC2den + CR3den + CL3den + CC3den + CR4den + CL4den + CC4den + CR5den + CL5den + CC5den);

% Plots
figure
plot(TR1,XR1,TR1,YR1*Ctot); axis([0 0.02 -2.5 2.5]); ylabel('Pressure (psi)'); xlabel('Time(s)'); grid on; legend ('Full','Half(Adjusted)');
figure
plot(TL1,XL1,TL1,YL1*Ctot); axis([0 0.02 -4 12]); ylabel('Pressure (psi)'); xlabel('Time(s)'); grid on; legend ('Full','Half(Adjusted)');
figure
plot(TC1,XC1,TC1,YC1*Ctot); axis([0 0.02 -1 10]); ylabel('Pressure (psi)'); xlabel('Time(s)'); grid on; legend ('Full','Half(Adjusted)');
figure
plot(TR2,XR2,TR2,YR2*Ctot); axis([0 0.02 -3 6]); ylabel('Pressure (psi)'); xlabel('Time(s)'); grid on; legend ('Full','Half(Adjusted)');
figure
plot(TL2,XL2,TL2,YL2*Ctot); axis([0 0.02 -10 20]); ylabel('Pressure (psi)'); xlabel('Time(s)'); grid on; legend ('Full','Half(Adjusted)');
figure
plot(TC2,XC2,TC2,YC2*Ctot); axis([0 0.02 -2 14]); ylabel('Pressure (psi)'); xlabel('Time(s)'); grid on; legend ('Full','Half(Adjusted)');
figure
plot(TR3,XR3,TR3,YR3*Ctot); axis([0 0.02 -4 10]); ylabel('Pressure (psi)'); xlabel('Time(s)'); grid on; legend ('Full','Half(Adjusted)');
figure
plot(TL3,XL3,TL3,YL3*Ctot); axis([0 0.02 -5 25]); ylabel('Pressure (psi)'); xlabel('Time(s)'); grid on; legend ('Full','Half(Adjusted)');
figure
plot(TC3,XC3,TC3,YC3*Ctot); axis([0 0.02 -4 16]); ylabel('Pressure (psi)'); xlabel('Time(s)'); grid on; legend ('Full','Half(Adjusted)');

% Impact 001
CR1 = CR1num/CR1den

% Impact 002
CR2 = sum(XR1(CminR1:CmaxR1).*YR1(CminR1:CmaxR1))./sum(YR1(CminR1:CmaxR1).^2)

% Impact 003
CR3 = sum(XR2(CminR2:CmaxR2).*YR2(CminR2:CmaxR2))./sum(YR2(CminR2:CmaxR2).^2)

% Impact 004
CR4 = sum(XR3(CminR3:CmaxR3).*YR3(CminR3:CmaxR3))./sum(YR3(CminR3:CmaxR3).^2)

% Impact 005
CR5 = sum(XR4(CminR4:CmaxR4).*YR4(CminR4:CmaxR4))./sum(YR4(CminR4:CmaxR4).^2)

% Impact 006
CR6 = sum(XR5(CminR5:CmaxR5).*YR5(CminR5:CmaxR5))./sum(YR5(CminR5:CmaxR5).^2)

% Impact 007
CR7 = sum(XR6(CminR6:CmaxR6).*YR6(CminR6:CmaxR6))./sum(YR6(CminR6:CmaxR6).^2)

% Impact 008
CR8 = sum(XR7(CminR7:CmaxR7).*YR7(CminR7:CmaxR7))./sum(YR7(CminR7:CmaxR7).^2)

% Impact 009
CR9 = sum(XR8(CminR8:CmaxR8).*YR8(CminR8:CmaxR8))./sum(YR8(CminR8:CmaxR8).^2)

% Impact 010
CR10 = sum(XR9(CminR9:CmaxR9).*YR9(CminR9:CmaxR9))./sum(YR9(CminR9:CmaxR9).^2)
figure
plot(TR4,XR4,TR4,YR4*Ctot); axis([0 0.02 0 -2 18]); ylabel('Pressure (psi)');
xlabel('Time(s)'); grid on; legend ('Full','Half(Adjusted)');
figure
plot(TL4,XL4,TL4,YL4*Ctot); axis([0 0.02 0 -5 30]); ylabel('Pressure (psi)');
xlabel('Time(s)'); grid on; legend ('Full','Half(Adjusted)');
figure
plot(TC4,XC4,TC4,YC4*Ctot); axis([0 0.02 0 -5 30]); ylabel('Pressure (psi)');
xlabel('Time(s)'); grid on; legend ('Full','Half(Adjusted)');
figure
plot(TR5,XR5,TR5,YR5*Ctot); axis([0 0.02 0 -5 20]); ylabel('Pressure (psi)');
xlabel('Time(s)'); grid on; legend ('Full','Half(Adjusted)');
figure
plot(TL5,XL5,TL5,YL5*Ctot); axis([0 0.02 0 -10 20]); ylabel('Pressure (psi)');
xlabel('Time(s)'); grid on; legend ('Full','Half(Adjusted)');
figure
plot(TC5,XC5,TC5,YC5*Ctot); axis([0 0.02 0 -10 20]); ylabel('Pressure (psi)');
xlabel('Time(s)'); grid on; legend ('Full','Half(Adjusted)');
Appendix H : Example Diadem Code for Data Processing
Call CHNLINSCALE("[1]/Time", "[1]/Time (msec)", 1000, 0)
chnDim("[1]/Time (msec)")="msec"

Removing the Bias (look at raw data to determine the duration)

Dim i, k, mean
Dim StartTime, StopTime, StartPoint, StopPoint, action

StartTime = -0.1 'Put in Start Time
StopTime = -0.07 'Put in Stop Time
StartPoint = PNo("[1]/Time", StartTime)
StopPoint = PNo("[1]/Time", StopTime)
Call ChnGet(0, 1, "Select Channels for Offset Removal")
For i=1 To ChnSelCount(ChnNoStr1)
    k=ChnSelGet(ChnNoStr1,i)
    STATSEL(1) = "No"
    STATSEL(2) = "No"
    STATSEL(3) = "No"
    STATSEL(4) = "No"
    STATSEL(5) = "Yes"
    STATSEL(6) = "No"
    STATSEL(7) = "No"
    STATSEL(8) = "No"
    STATSEL(9) = "No"
    STATSEL(10) = "No"
    STATSEL(11) = "No"
    STATSEL(12) = "No"
    STATSEL(13) = "No"
    STATSEL(14) = "No"
    STATSEL(15) = "No"
    STATSEL(16) = "No"
    STATSEL(17) = "No"
    STATSEL(18) = "No"
    STATSEL(19) = "No"
    STATSEL(20) = "No"
    STATSEL(21) = "No"
    STATSEL(22) = "No"
    STATCLIPCOPY = 0
    STATCLIPVALUE = 0
    STATFORMAT ="
    STATRESCHN = 0
    Call ChnPropSet(k, "description")
    Call STATBLOCKCALC("Channel", StartPoint & "-" & StopPoint, k)
    Mean=statarithmean
    unit = ChnDim(k)
    action = ChnPropGet(k, "description")
    call FormulaCalc("Ch(" & k & "):=Ch(" & k & ")-" & mean)

    Call ChnPropSet(k, "description", action & "zeroed from " & StartTime & " - " & StopTime & ", ")
    ChnDim(k) = unit
Next

'---------------------------------------------------------------
' Ram Accelerometer
'---------------------------------------------------------------

Call CHNCFILTCALC([1]/Time","[1]/RAMXG","[1]/RAMXGp","CF_60",0,"EndPoints",10)
ChnDim([1]/RAMXGp) = "g"

' Velocity from Ram accelerometer
Call FormulaCalc("ch([1]/RAMXGpM):= ch([1]/RAMXGp)*9.80665")' g -> M/sec^2
Call CHNINTEGRATE("[1]/Time","[1]/RAMXGpM","[1]/RAMXGp_Vel")
ChnDim([1]/RAMXGp_Vel) = "m/Sec"
Call CHNDELETE("[1]/RAMXGpM")

' Displacement from Ram accelerometer (double integrated)
Call CHNINTEGRATE("[1]/Time","[1]/RAMXGp_Vel","[1]/RAMXGp_Dis")
Call FormulaCalc("ch([1]/RAMXGp_Dis'):= ch([1]/RAMXGp_Dis)*1000")
ChnDim([1]/RAMXGp_Dis) = "mm"

'---------------------------------------------------------------
' Finding Force From Accel and Ram Mass of 23.068
'---------------------------------------------------------------

Call FormulaCalc("ch([1]/Force_Accel'):= ch([1]/RAMXGp')*9.80665*23.068")
ChnDim([1]/Force_Accel) = "N"

'---------------------------------------------------------------
' Finding Compensation Load of Ram
'---------------------------------------------------------------

' Inertial force of the impactor including half of loadcell and 4-screws
Call FormulaCalc("ch([1]/InertialFy'):= ch([1]/RAMXGp')*9.80665*1.763")
ChnDim([1]/InertialFy) = "N"

' Ram 6-axis Load Cell

Call CHNCFILTCALC([1]/Time","[1]/RAMFX","[1]/RAMFXp","CF_180",0,"EndPoints",10)
ChnDim([1]/RAMFXp) = "N"
Call CHNCFILTCALC([1]/Time","[1]/RAMFY","[1]/RAMFYp","CF_180",0,"EndPoints",10)
ChnDim([1]/RAMFYp) = "N"
Call CHNCFILTCALC([1]/Time","[1]/RAMFZ","[1]/RAMFZp","CF_180",0,"EndPoints",10)
ChnDim([1]/RAMFZp) = "N"
Call CHNCFILTCALC([1]/Time",","[1]/RAMMX","[1]/RAMMXp","CF_180",0,"EndPoints",10)
ChnDim([1]/RAMMXp) = "N"
Call CHNCFILTCALC([1]/Time","[1]/RAMMY","[1]/RAMMYp","CF_180",0,"EndPoints",10)
ChnDim([1]/RAMMYp) = "N"
Call CHNCFILTCALC([1]/Time",","[1]/RAMMZ","[1]/RAMMZp","CF_180",0,"EndPoints",10)
ChnDim([1]/RAMMZp) = "N"

' Impact force including inertial compensation and displacement from linear pot
Call FormulaCalc("ch(['1]/CompForce'):= (ch('['1]/RAMFYp')-ch('['1]/InertialFy'))/1")
ChnDim("['1]/CompForce") = "N"

' Ram Displacement from linear pot

Call CHNCFCFILT CALC("['1]/Time","['1]/RAMXD","['1]/RAMXDp","CFC_180",0, "EndPoints",10)
ChnDim("['1]/RAMXDp") = "Cm"

' Process Millars (Filter and convert to kPa)

Call CHNCFCFILT CALC("['1]/Time","['1]/AortaSup","['1]/AortaSupp_psi","CFC_180",0,"EndPoints",10)
Call CHNCFCFILT CALC("['1]/Time","['1]/AortaInf","['1]/AortaInf_psi","CFC_180",0,"EndPoints",10)
Call CHNCFCFILT CALC("['1]/Time","['1]/HepaticLat","['1]/HepaticLatp_psi","CFC_180",0,"EndPoints",10)
Call CHNCFCFILT CALC("['1]/Time","['1]/HepaticMed","['1]/HepaticMedp_psi","CFC_180",0,"EndPoints",10)
Call CHNCFCFILT CALC("['1]/Time","['1]/IVCSup","['1]/IVCSupp_psi","CFC_180",0,"EndPoints",10)
Call CHNCFCFILT CALC("['1]/Time","['1]/IVCInf","['1]/IVCInfp_psi","CFC_180",0,"EndPoints",10)
Call CHNCFCFILT CALC("['1]/Time","['1]/LiverTissue1","['1]/LiverTissue1p_psi","CFC_180",0,"EndPoints",10)
Call CHNCFCFILT CALC("['1]/Time","['1]/LiverTissue2","['1]/LiverTissue2p_psi","CFC_180",0,"EndPoints",10)
chnDim("['1]/AortaSupp_psi")="PSI"
chnDim("['1]/AortaInf_psi")="PSI"
chnDim("['1]/HepaticLat_psi")="PSI"
chnDim("['1]/HepaticMed_psi")="PSI"
chnDim("['1]/IVCSupp_psi")="PSI"
chnDim("['1]/IVCInfp_psi")="PSI"
chnDim("['1]/LiverTissue1p_psi")="PSI"
chnDim("['1]/LiverTissue2p_psi")="PSI"

'-- VBS script file
'-- Created on 01/28/2010 12:54:05
'-- Author: ---
'-- Comment: ---Convert to kPa

Call CHNLINSCALE("['1]/AortaSupp_psi","['1]/AortaSup","6.89475729",0)
Call CHNLINSCALE("['1]/AortaInf_psi","['1]/AortaInf","6.89475729",0)
Call CHNLINSCALE("['1]/HepaticLat_psi","['1]/HepaticLat","6.89475729",0)
Call CHNLINSCALE("[1]/HepaticMedp_psi","[1]/HepaticMedp",6.89475729,0)
Call CHNLINSCALE("[1]/IVCSupp_psi","[1]/IVCSupp",6.89475729,0)
Call CHNLINSCALE("[1]/IVCInfp_psi","[1]/IVCInfp",6.89475729,0)
Call CHNLINSCALE("[1]/LiverTissue1p_psi","[1]/LiverTissue1p",6.89475729,0)
Call CHNLINSCALE("[1]/LiverTissue2p_psi","[1]/LiverTissue2p",6.89475729,0)
chnDim(["1/AortaSupp"])="kPa"
chnDim(["1/HepaticLatp"])="kPa"
chnDim(["1/HepaticMedp"])="kPa"
chnDim(["1/IVCSupp"])="kPa"
chnDim(["1/IVCInfp"])="kPa"
chnDim(["1/LiverTissue1p"])="kPa"
chnDim(["1/LiverTissue2p"])="kPa"

Call ChnDifferentiate(["1/Time (msec)","[1]/AortaSupp","[1]/AortaSuppMean","[1]/AortaSupp_Rate"])
Call ChnDifferentiate(["1/Time (msec)","[1]/AortaInfp","[1]/AortaInfMean","[1]/AortaInfp_Rate"])
Call ChnDifferentiate(["1/Time (msec)","[1]/HepaticLatp","[1]/HepaticLatMean","[1]/HepaticLatp_Rate"])
Call ChnDifferentiate(["1/Time (msec)","[1]/HepaticMedp","[1]/HepaticMedMean","[1]/HepaticMedp_Rate"])
Call ChnDifferentiate(["1/Time (msec)","[1]/IVCSupp","[1]/IVCSupMean","[1]/IVCSupp_Rate"])
Call ChnDifferentiate(["1/Time (msec)","[1]/IVCInfp","[1]/IVCInfMean","[1]/IVCInfp_Rate"])
Call ChnDifferentiate(["1/Time (msec)","[1]/LiverTissue1p","[1]/LiverTissue1Mean","[1]/LiverTissue1p_Rate"])
Call ChnDifferentiate(["1/Time (msec)","[1]/LiverTissue2p","[1]/LiverTissue2Mean","[1]/LiverTissue2p_Rate"])
chnDim(["1/AortaSupp_Rate"])="kPa/ms"
chnDim(["1/AortaInfp_Rate"])="kPa/ms"
chnDim(["1/HepaticLatp_Rate"])="kPa/ms"
chnDim(["1/HepaticMedp_Rate"])="kPa/ms"
chnDim(["1/IVCSupp_Rate"])="kPa/ms"
chnDim(["1/IVCInfp_Rate"])="kPa/ms"
chnDim(["1/LiverTissue1p_Rate"])="kPa/ms"
chnDim(["1/LiverTissue2p_Rate"])="kPa/ms"

' Delete raw data channels

Call CHNDELETE("[1]/AortaSuppMean")
Call CHNDELETE("[1]/AortaInfMean")
Call CHNDELETE("[1]/HepaticLatMean")
Call CHNDELETE("[1]/HepaticMedMean")
Call CHNDELETE("[1]/IVCSuppMean")
Call CHNDELETE("[1]/IVCInfpMean")
Call CHNDELETE("[1]/LiverTissue1Mean")
Call CHNDELETE("[1]/LiverTissue2Mean")
Call CHNDELETE("[1]/RAMXD")
Call CHNDELETE("[1]/RAMPSI")
Call CHNDELETE("[1]/RAMXG")
Call CHNDELETE("[1]/RAMFX")
Call CHNDELETE("[1]/RAMFY")
Call CHNDELETE("[1]/RAMF2")
Call CHNDELETE("[1]/RAMMX")
Call CHNDELETE("[1]/RAMMY")
Call CHNDELETE("[1]/RAMMZ")
Call CHNDELETE("[1]/AortaSup")
Call CHNDELETE("[1]/AortaInf")
Call CHNDELETE("[1]/IVCSup")
Call CHNDELETE("[1]/IVCInf")
Call CHNDELETE("[1]/HepaticLat")
Call CHNDELETE("[1]/HepaticMed")
Call CHNDELETE("[1]/LiverTissue1")
Call CHNDELETE("[1]/LiverTissue2")
Call CHNDELETE("[1]/AortaSupp_psi")
Call CHNDELETE("[1]/AortaInfp_psi")
Call CHNDELETE("[1]/HepaticLatp_psi")
Call CHNDELETE("[1]/HepaticMedp_psi")
Call CHNDELETE("[1]/IVCSupp_psi")
Call CHNDELETE("[1]/IVCInfp_psi")
Call CHNDELETE("[1]/LiverTissue1p_psi")
Call CHNDELETE("[1]/LiverTissue2p_psi")
Call CHNDELETE("[1]/STRNXG")
Call CHNDELETE("[1]/STRNYG")
Call CHNDELETE("[1]/STRNZG")
Call CHNDELETE("[1]/STRNXdeg")
Call CHNDELETE("[1]/STRNYdeg")
Call CHNDELETE("[1]/STRNZdeg")
Call CHNDELETE("[1]/T1XG")
Call CHNDELETE("[1]/T1YG")
Call CHNDELETE("[1]/T1ZG")
Call CHNDELETE("[1]/T1Xdeg")
Call CHNDELETE("[1]/T1Ydeg")
Call CHNDELETE("[1]/T1Zdeg")
Call CHNDELETE("[1]/CHST1")
Call CHNDELETE("[1]/CHST2")
Call CHNDELETE("[1]/CHST3")
Call CHNDELETE("[1]/CHST4")
Call CHNDELETE("[1]/CHST5")
Call CHNDELETE("[1]/CHST6")
Call CHNDELETE("[1]/CHST7")
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Call CHNDELETE("[1]/CHST9")
Call CHNDELETE("[1]/CHST10")
Call CHNDELETE("[1]/CHST11")
Call CHNDELETE("[1]/CHST12")
Call CHNDELETE("[1]/CHST13")
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Call CHNDELETE("[1]/CHST15")
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Call CHNDELETE("[1]/CHST19")
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Call CHNDELETE("[1]/CHST25")
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Call CHNDELETE("[1]/CHST39")
Call CHNDELETE("[1]/CHST40")
Call CHNDELETE("[1]/T8XG")
Call CHNDELETE("[1]/T8YG")
Call CHNDELETE("[1]/T8ZG")
Call CHNDELETE("[1]/T8Xdeg")
Call CHNDELETE("[1]/T8Ydeg")
Call CHNDELETE("[1]/T8Zdeg")
Call CHNDELETE("[1]/T12XG")
Call CHNDELETE("[1]/T12YG")
Call CHNDELETE("[1]/T12ZG")
Call CHNDELETE("[1]/T12Xdeg")
Call CHNDELETE("[1]/T12Ydeg")
Call CHNDELETE("[1]/T12Zdeg")
Call CHNDELETE("[1]/Channel44")
Call CHNDELETE("[1]/Channel45")
Call CHNDELETE("[1]/Channel46")
Call CHNDELETE("[1]/Channel47")
Call CHNDELETE("[1]/Channel48")
Call CHNDELETE("[1]/Channel49")
Call CHNDELETE("[1]/Channel50")
Call CHNDELETE("[1]/Channel51")
Call CHNDELETE("[1]/Channel52")
Call CHNDELETE("[1]/Channel53")
Call CHNDELETE("[1]/Channel54")
Call CHNDELETE("[1]/Channel55")
Call CHNDELETE("[1]/Channel56")