Correlation of Liver Injury and Vascular Pressure: A Study of Lateral Impacts to Post-Mortem Human Subjects

H.M. Gustafson[1], J.A. Stammen[2], R.G. Herriott[3], and J.H. Bolte IV[1]
[1] The Ohio State University; [2] NHTSA-VRTC; [3] Transportation Research Center

ABSTRACT

Abdominal injuries are a relatively common result of motor vehicle crashes and the injuries represent a high proportion of serious injuries. Previous work 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 impact. The purpose of this work was to further investigate the relationship between pressure and liver injury using post-mortem human subjects (PMHS). Specifically, the goals were to (1) conduct lateral impacts on PMHS with re-pressurized abdominal vascular systems and measure the vascular pressure and (2) determine if a correlation exists between the measured vascular pressure and liver injury. In an ongoing study, four PMHS have been instrumented with pressure sensors in the abdominal vessels, including the abdominal aorta, the hepatic veins, and the inferior vena cava. For each test, the subject's abdomen was pressurized to physiological pressures using saline. The seated subject was held upright by a head restraint which was released immediately before contact by the pneumatic ram, ensuring the subject was not suspended at the time of impact. The lateral impact was applied to the right side of the subject at the level of the liver. Following each test, autopsy was performed on the subject. One test resulted in a serious (AIS 3+) injury to the liver. The test in which a liver injury was obtained resulted in the highest peak hepatic vein pressure and the highest peak rate of pressure increase. The compression (C), viscous criteria ($[V(t)*C(t)]_{max}$), and abdominal injury criteria ($V_{max}C_{max}$) were also highest for this test. These results can be applied to improve the abdominal injury assessment in both anthropomorphic test devices and in computer models of the human body used in vehicle safety research.

This paper has not been peer- reviewed and should not be referenced in open literature.

INTRODUCTION

Abdominal injuries account for only 3 to 5% of the total number of injuries due to motor vehicle crashes (MVC) (Ricci, 1980; Rouhana, 1985). However, abdominal injuries, especially to the solid organs of the abdomen, represent a higher proportion of serious injuries (Elhagediab, 1998). For example, Lee and Yang (2002) reported that abdominal injuries constituted 5.2% of all injuries but 15.6% of Abbreviated Injury Scale (AIS) 3+ injuries in the National Automotive Sampling System (NASS) database for the years 1993 through 1997. Furthermore, the liver has been reported to be a common site of abdominal injury, likely a result of its size and anatomical location. According to a study of abdominal injuries based on the NASS database, the frequency of liver injuries was15.7% of all abdominal injuries and 34% of AIS 3+ abdominal injuries (Lee, 2002). The liver is the largest solid organ with relatively little mobility in the abdomen due to ligamentous attachments to the diaphragm. Three types of injury mechanisms are common for the liver: (a) laceration of the liver due to rib edges that have fractured, (b) compression of the liver between the rib cage and the spine or posterior abdominal wall, and (c) deceleration injuries which result in the liver tissue shearing, particularly at attachment points (Feliciano, 2008).

Previously, it has been shown that pressure measured in the abdomen relates to injury in experimental animal surrogate research. Lau and Viano (1981) investigated abdominal injury using white New Zealand rabbits. A pressure sensor was placed in the esophagus at mid-thoracic level and the impact was applied at velocities from 5 to 20 m/s. Pressure showed a strong (r^2=0.76) correlation to abdominal injury. Prasad and Daniel (1984) also found a relationship between injury and blood pressure measured in the descending aorta when performing high speed accelerations on piglets. It was found that blood pressure peaks greater than 53.3 kPa are associated with AIS 3+ injuries in pigs. Miller (1989) placed sensors in the right carotid artery and the jugular vein of swine to measure the arterial and venous pressure. Occurrence of AIS 3+ injuries generally increased with higher measured pressures. Additionally, Sparks et al. (2007) studied *ex vivo* human livers and performed rigid plate, drop tower impacts on fourteen specimens. During the testing, the venous and arterial systems were perfused with saline. The change in internal vascular pressure of the fluid was measured as well as the tissue pressure in the liver parenchyma. Analysis of the *ex vivo* data shows that a vascular pressure of 46.0 kPa corresponds to a 50% risk of AIS 3+ injury.

Animal models and *ex vivo* testing have shown that internal pressure is a predictor of abdominal injury. To expand on this, the purpose of this research is to determine if pressure is a good predictor of abdominal injury in full body post-mortem human subjects (PMHS). This research will test the human liver *in situ* with boundary conditions more representative of real world trauma. In addition to pressure, other biomechanical variables will be measured and analyzed for their relationship to injury.

This paper has not been peer- reviewed and should not be referenced in open literature.

METHODS

For the testing, PMHS (n=4) were obtained through the willed body donation program at Ohio State. Both males and females were accepted for testing and all subjects met the following criteria:

- Classified as neither emaciated or obese according to the body mass index
- Not osteoporotic according to the Dual Energy X-ray Absorptiometry (DXA) bone scan
- No scars indicating major abdominal surgery
- Mass of less than 95 kg for ease of positioning

All tests were performed within four days of death and subjects were not frozen prior to testing.

For the internal instrumentation, pressure sensors (Millar Instruments model SPR-524, Houston, TX) were inserted through Foley catheters of size 16 to 20 French with 30 cubic centimeter balloons. The purpose of the Foley catheters was to occlude vessels in order to pressurize only the abdomen. The goal of the instrumentation was to locate the pressure sensors in the vasculature of the abdomen. The number of sensors placed for the testing ranged from four to eight and the positions were identified in real-time using a fluoroscope. The target locations for the internal instrumentation were the hepatic vein, the inferior vena cava, the superior border of the liver in the descending aorta, and the inferior border of the liver in the descending aorta. The locations are shown in Figure 1.

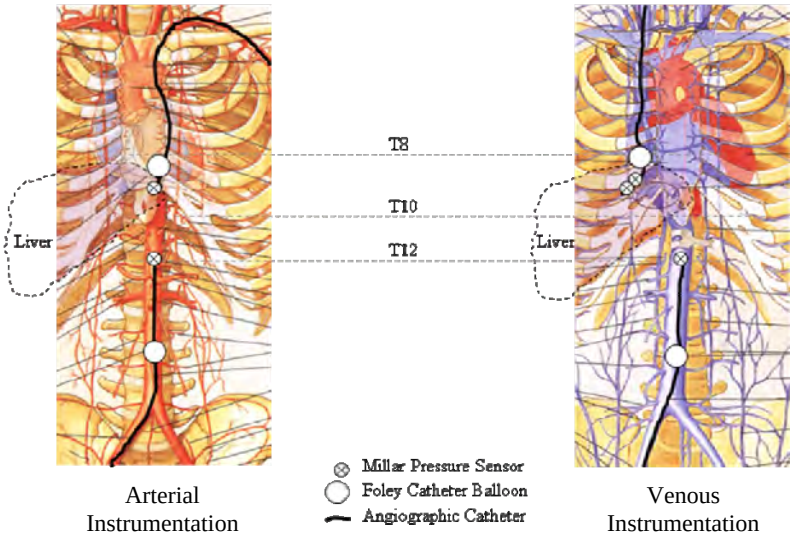


Figure 1: Internal Instrumentation

The subject was positioned in front of a pneumatic ram. The inferior edge of the rib cage on the right side of the subject was aligned with the lower edge of an aluminum impact plate. The aluminum plate, intended to simulate a blunt lateral loading, was 15 cm high and 30 cm wide. To measure the motion of the thorax, three accelerometers and three angular rate sensors were placed on blocks and attached to the sternum and vertebral arches of T1, T8, and T12. A chestband was placed at the midline of impact in order to measure compression of the subject's lower thorax due to impact. Figure 2 shows the external instrumentation relative to the skeletal structure of the thorax.

This paper has not been peer- reviewed and should not be referenced in open literature.

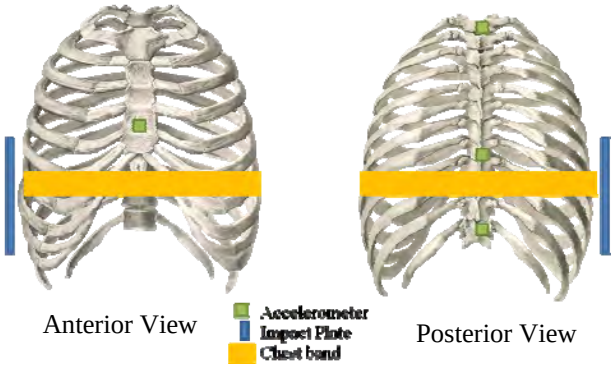


Figure 2: External Instrumentation.

The subject's abdomen was pressurized for approximately three minutes by connecting the Foley catheters of the superior insertion points to saline reservoirs, as shown in Figure 3. The heights of the reservoirs corresponded to the physiological pressures of the arterial and venous vasculature. The seated subject was held upright by a head restraint which was released immediately before contact with the pneumatic ram. The nominal energy of impact was 587 J. Test-specific data is given in Table 1.

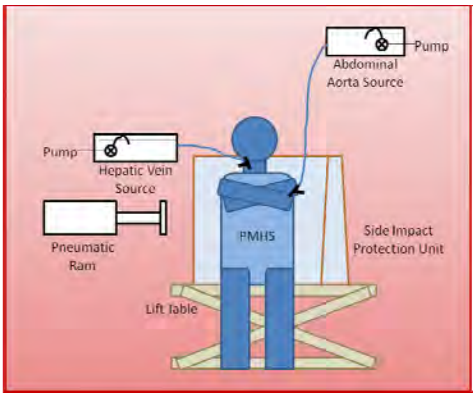


Figure 3: Test Setup.

Table 1: Subject and Test Data

	FBL01	FBL02	FBL03	FBL04
Gender	Male	Female	Male	Male
Age	68	80	88	91
Mass (kg)	66.7	59.0	72.6	63.5
T-Score*	0.9	-1.5	-1.0	-1.6
Stature (cm)	176	154	188	179
Seated Height (cm)	94	87	96	97
Impact Velocity (m/s)	7.20	7.09	7.03	7.06
Notes	Sensors intended for Hepatic Vein were in Right Atrium of the Heart			Sensors intended for Hepatic Vein were in Right Renal Vein

* T-Score measured by DXA. -1.0 or higher: Normal bone mass density, -2.5 to -1.0: Osteopenic, -2.5 or lower: Osteoporotic

This paper has not been peer- reviewed and should not be referenced in open literature.

RESULTS

Injuries

Following each test, the subject was autopsied to identify injuries, and injuries were rated on the AIS scoring system. Table 2 summarizes the injury findings with the shaded injuries representing serious (AIS 3+) injuries.

Table 2: AIS Injuries Ratings and Descriptions of injury

	FBL01		FBL02		FBL03		FBL04	
Injury Location	AIS Code	Description	AIS Code	Description	AIS Code	Description	AIS Code	Description
Ribs	450220.2	Fractures of ribs 5 and 6 on the right side	450266.5	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	450230.3	Fractures on ribs 6-11 on the right side, unilateral flail chest	450262.3	Fractures of ribs 3-12 on the right side and ribs 9 and 10 on the left side, unilateral flail chest
Liver	541822.2	Minor laceration on the right inferior aspect of the liver			541826.4	Burst injury to the liver on the posterior side of the liver, primarily to the right lobe of the liver		
Lungs			441406.3	Lung contusion on the right side with pneumothorax				
Transverse Process Fractures			650620.2	Fractures of the lumbar transverse processes 1-4 on the right side and 2-4 on the left side			650620.2	Fractures of the lumbar transverse processes 1-5 on the right side

Data Analysis

The frequency of the data acquisition was 20,000 Hz. The raw data signals were processed as follows to obtain the biomechanical variables:

- **Peak Change in Pressure (P_{max}):** Filter raw pressure signal at 1650 Hz, Zero at time=-0.09 seconds prior to initial ram movement, report maximum change in pressure
- **Peak Rate of Change of Pressure ($\dot{P}_{max}$):** Filter raw pressure signal at 1650 Hz, Zero at time=-0.09 seconds, differentiate using 5-point central difference formula shown below, report maximum rate of change of the pressure

$$f'(x)= \frac{-f(x+2h)+8*f(x+h)-8*f(x-h)+f(x-2h)}{12h}$$

- **Peak of Rate of Change in Pressure times Pressure ($[P(t)*\dot{P}(t)]_{max}$):** Filter raw pressure signal at 1650 Hz, Zero at time=-0.09 seconds, differentiate using 5-point central difference formula, multiply pressure times the rate of change of the pressure, report maximum
- **Peak Rate of Change in Pressure times Peak Pressure ($P_{max}*\dot{P}_{max}$):** Filter raw pressure signal at 1650 Hz, Zero at time=-0.09 seconds, find maximum of pressure, differentiate pressure using

This paper has not been peer- reviewed and should not be referenced in open literature.

5-point central difference formula, find maximum rate of change, multiply the maximums and report value for each set of pressure data

- **Displacement (d):** Process chestband data using numerical computation software (Matlab, Natwick, MA), indentify the gages initially at the mid-axillary lines, calculate compression between gages as a function of time, report maximum displacement from original position at time zero
- **Compression (C):** Normalize the displacement by the chest breadth of the subject
- **Abdominal Injury Criterion (V_{max}*C_{max})**: Multiply the velocity of the ram at the time of the event (t=0) by the compression, report the maximum
- **Viscous Criterion ($[V*C]_{max}$):** Obtain the time history of compression from the chestband, differentiate the compression using a 5-point central difference equation to obtain velocity of compression, multiply compression by velocity, report the peak value
- **Kinetic Analog to the Viscous Criterion ($[\dot{P} * C]_{max}$):** This was variable was suggested by Kent et al. (2008). Filter the ram load cell force in the direction of impact at 1650 Hz, zero at time=-0.09, filter the ram acceleration at 1650 Hz, zero at time=-0.09, multiply acceleration by the mass of the impactor plate and half the mass of the load cell in order to get the inertial force, add the load cell force to the inertial force, differentiate the force using the 5-point central difference formula, multiply the rate of change of force by the compression, report the peak value

Results from the testing are summarized in Table 3.

Table 3: Results from Full Body Liver Testing

Variable	Units	FBL01	FBL02	FBL03	FBL04
Peak Change in Pressure, Superior Descending Aorta	kPa	11.5/ 10.2	20.4	9.7	22.5
Peak Change in Pressure, Inferior Descending Aorta	kPa	19.4/ 18.6	27.4	16.7	23.3
Peak Change in Pressure, Hepatic/Renal Vein	kPa	NM	21.2/ 20.9	41.9/ 37.2	29.9*
Peak Change in Pressure, Inferior Vena Cava	kPa	36.6/ 33.7	19.4	37.5	34.2
$\dot{P}_{max}$, Hepatic/Renal Vein	kPa/ms	NM	9.6	75.0	23.1*
$[P(t) * P(t)]_{max}$, Hepatic/ Renal Vein	kPa^2/ms	NM	200.1	3142.7	690.0*
P_{max}*$\dot{P}_{max}$, Hepatic/Renal Vein	kPa^2/ms	NM	175.4	1466.6	267.3*
d, Displacement	mm	77.71	75.6	95.0	87.5
C, Compression	mm/mm	0.26	0.24	0.31	0.29
$V_{max}C_{max}$, Abdominal Injury Criterion	m/s	1.84	1.70	2.19	2.03
$[V*C]_{max,}$ Viscous Criterion	m/s	1.03	1.02	2.47	1.10
$[\dot{P} * C]_{max,}$ Kinetic Analog to the Viscous Criterion	N/s	833.1	713.7	1095.0	950.4

Shaded indicates AIS 3+ liver injury
NM- Value Not Measured
*Sensor located in Renal Vein, not in Hepatic Vein for test FBL04

This paper has not been peer- reviewed and should not be referenced in open literature.

Discussion

In general, the measured pressures in the venous vessels were higher than in the arterial vessels. The pressures also tended to rise at a higher rate on the venous side. One explanation for this is the proximity of the venous vasculature to the loading since the inferior vena cava is located closer than the descending aorta to the right side of the body. Additionally, venous vessels walls are thinner than the artery walls and the venous vasculature, especially the hepatic veins, may be subject to deformation by the surrounding liver tissue. In some cases, two pressure transducers were placed near the same location to obtain a redundant measurement. For these pairs of sensors, the variations were relatively small with errors of 1.4 to 12.6% of the pressure. Additionally, the arterial and venous measurements had consistent shapes for a given test. This provides confidence that the pressure changes measured were not spurious but rather the result of real fluid effects.

A serious liver injury was obtained in test FBL03. In this test, the peak change in pressure in the hepatic vein was 41.9 kPa, the highest of all the tests. The rate of increase of pressure was the highest of the tests (75.0 kPa/ms) and the highest level of compression from the four tests was found in this test (31%). This high compression contributed to test FBL03 also having the highest abdominal injury criterion ($V_{max}C_{max}$), the highest viscous criterion ($[V*C]_{max}$), and the highest kinetic analog of the viscous criterion ($[\rho * C]_{max}$). These results are promising in determining a strong, consistent predictor of liver injury.

Subject FBL02 experienced the most extensive skeletal injuries including flail chest of both the right and left sides and fractures of numerous lumbar transverse processes. However, the subject did not experience any liver injury. The subject had a significant amount of subcutaneous tissue over the rib cage. It is hypothesized that the energy from the ram was dissipated by the fracturing ribs and the subcutaneous fat. This resulted in a lower ram force in the direction of impact and less compression of the lower thorax which may have resulted in less liver deformation.

Previous *ex vivo* human liver testing by Sparks et al. (2007) indicated that a measured change in vascular pressure of 46.0 kPa corresponded to a 50% risk of injury. The current *in situ* testing has not obtained pressures that high. One possible reason for the discrepancy is the boundary conditions are different from the *ex vivo* testing. Future work includes modification of the test setup to more directly load the liver. It is hypothesized that this will increase the pressure in the liver. This may lead to an increase in the occurrence of liver injuries which will add left-censored samples to the overall dataset and thus allow a better understanding of the injury risk distribution.

Limitations

One clear limitation of the current study is the number of subjects, especially the number of subjects with the instrumentation in the hepatic veins. Since only one test (FBL03) resulted in liver injury, it is difficult to say if our results are significant until more tests are performed. While it was assumed that the pressurization of the abdomen created a closed system, there may have been leakage if a secure seal was not created between the vessel and the Foley balloon.

This paper has not been peer- reviewed and should not be referenced in open literature.

Additionally, clotted blood or plaque may have affected the measurements recorded from the pressure transducers.

CONCLUSIONS

- Four subjects were laterally impacted using a pneumatic ram and the pressure in the abdomen was measured
- One impact (FBL03) resulted in liver injury and also produced the highest compression, pressure change in the hepatic veins, rate of change of pressure, abdominal injury criterion, viscous criterion, and kinetic analog to the viscous criterion.
- The peak change of pressure in the hepatic vein in FBL03 was slightly lower than the value determined in *ex vivo* testing for a 50% risk of AIS 3+ injury
- The current study shows promise in establishing a relationship between pressure and other pressure related variables such as $\dot{P}_{max}$ or $[P(t) * P(t)]_{max}$.

ACKNOWLEDGEMENTS

This research was supported by the U.S. Department of National Highway Traffic Safety Administration (Contract No. DTNH22-08-D-00082). The authors would like to thank Bruce Donnelly and Dan Rhule for their assistance in data analysis. The authors also gratefully acknowledge Amanda Agnew, Yun-Seok Kang, Brian Suntay, Kyle Icke, Matt Long, and Austin Meek for their assistance in testing.

This paper has not been peer- reviewed and should not be referenced in open literature.

REFERENCES

ELHAGEDIAB, A.M., ROUHANA S.W. (1998). Patterns of abdominal injury in frontal automotive crashes. ESV Conference Proceedings, 16, 327-337.

FELICIANO, D. V., MATTOX, K. L., MOORE, E. E. (2008). Trauma, McGraw-Hill Medical, New York. 111.

KENT, R., STACEY, S., KINDIG, M., WOODS, W., EVANS, J., ROUHANA, S.W., HIGUCHI, K., TANJI H., ST. LAWRENCE, S., ARBOGAST, K.B. (2008). Biomechanical Response of the Pediatric Abdomen, Part 2: Injuries and Their Correlation with Engineering Parameters. 52nd Stapp Car Crash Conference proceedings, 135-166.

LAU, V. K., VIANO, D. C. (1981). Influence of impact velocity on the severity of nonpenetrating hepatic injury. The Journal of trauma, 21(2), 115-123.

LEE, J.B., YANG K.H. (2002). Abdominal Injury Patterns in Motor Vehicle Accidents: A Survey of the NASS Database from 1993 to 1997. Traffic Injury Prevention, 3(3), 241-246.

MILLER, M. A. (1989). The biomechanical response of the lower abdomen to belt restraint loading. Journal of Trauma, 29(11), 1571-1584.

PRASAD, P., DANIEL, R. P. (1984). A biomechanical analysis of head, neck, and torso injuries to child surrogates due to sudden torso acceleration. 28th Stapp Car Crash Conference Proceedings, 25-40.

RICCI, L. L. (1980). NCSS statistics: passenger cars. UM-HSRI report. 68-75.

ROUHANA, S. W., FOSTER, M. E. (1985). Lateral impact - an analysis of the statistics in the NCSS. 29th Stapp Car Crash Conference proceedings, 79-98.

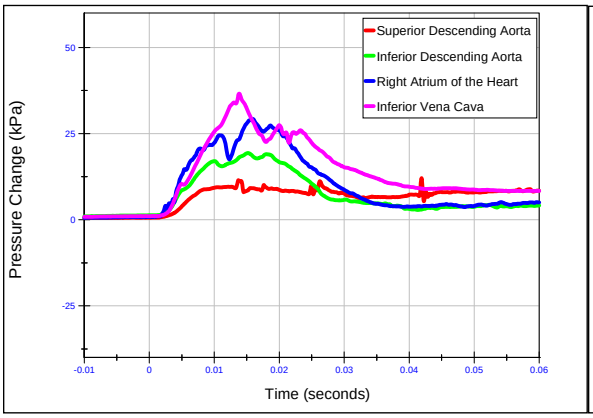
SPARKS, J. L., BOLTE, J. H., DUPAIX, R. B., JONES, K. H., STEINBERG, S. M., HERRIOTT, R. G., STAMMEN, J. A., DONNELLY, B. R. (2007). Using Pressure to Predict Liver Injury Risk from Blunt Impact. 51st Stapp Car Crash Proceedings, 401-432.

This paper has not been peer- reviewed and should not be referenced in open literature.

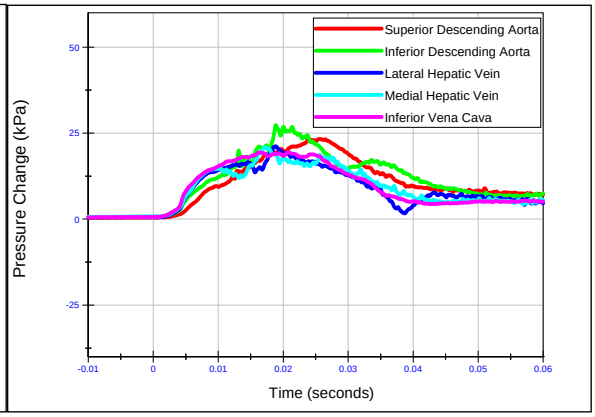
APPENDIX

Pressure Results

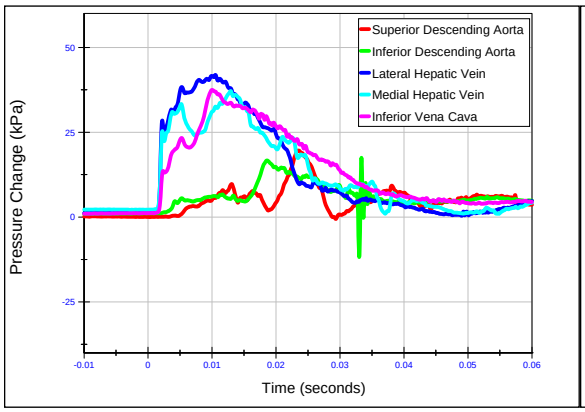
The change in pressure for each test is shown here. For the majority of the tests, the sensors in the same vessels follow a similar trend. The venous side tends to have higher pressures. This could be due to the fact that the IVC is closer than the descending aorta to the impact side.



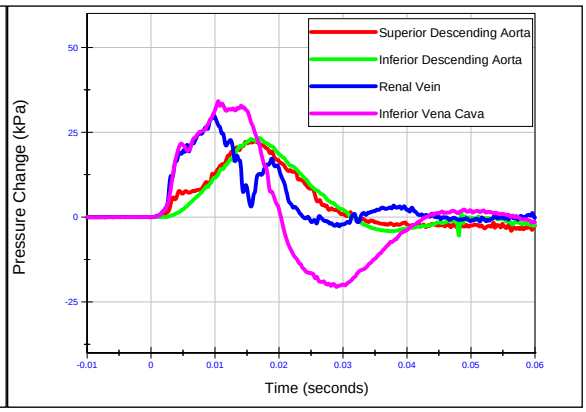
FBL01



FBL02



FBL03

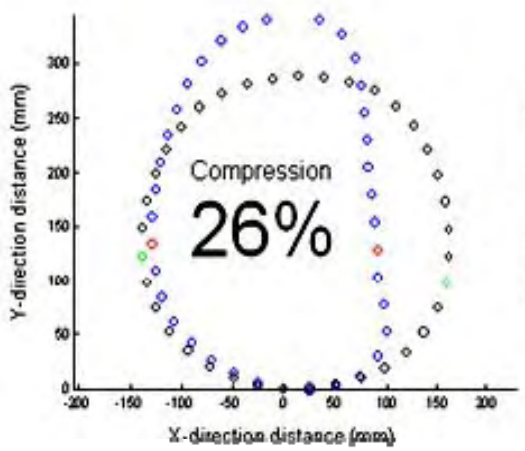


FBL04

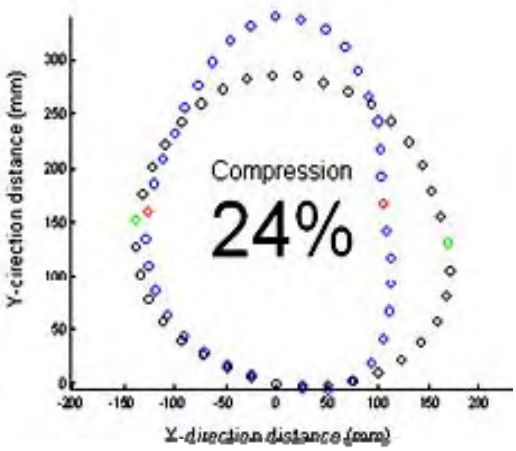
This paper has not been peer- reviewed and should not be referenced in open literature.

Compression Results

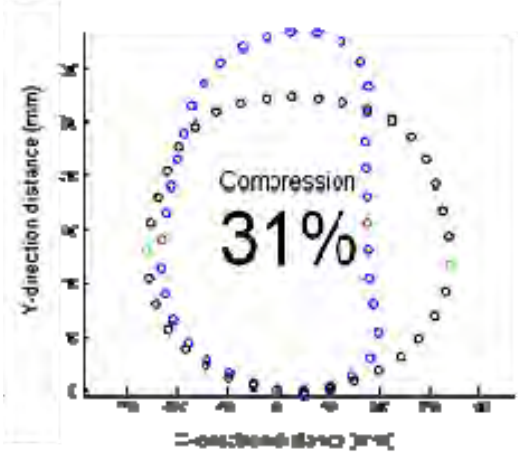
The plots here show the initial position of the chestband (shown in black) and the final position of the chestband (shown in blue). The gages at the mid-axillary lines are shown in green in their initial position and in red in their final position.



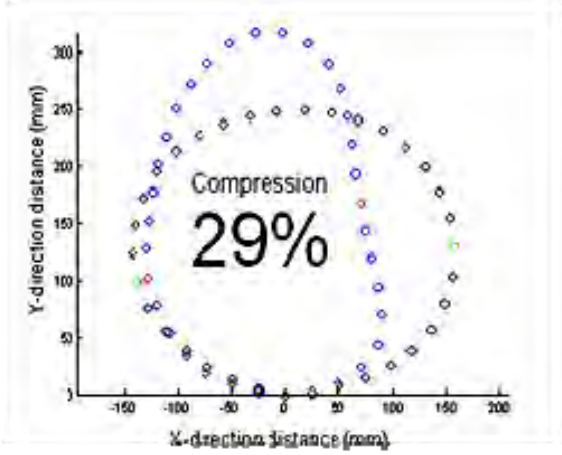
FBL01



FBL02



FBL03



FBL04

This paper has not been peer- reviewed and should not be referenced in open literature.

AUTHOR LIST

1. **Hannah M. Gustafson**
   279 Hamilton Hall
   1645 Neil Ave.
   Columbus, Ohio 43210
   (614) 292-4448 (office)
   (614) 292-7659 (fax)
   Gustafson.30@osu.edu
   The Ohio State University
   Masters Student
   Dr. John Bolte IV
   June, 2009

2. **Jason A. Stammen**
   VRTC/DOT
   PO Box B37
   East Liberty, Ohio 43319
   (937) 666-3251
   Jason.Stammen@dot.gov

3. **Rod G. Herriott**
   TRC
   PO Box B37
   East Liberty, Ohio 43319
   (937) 666-4511 ext. 281
   Rod.Herriott@dot.gov

4. **John H. Bolte IV**
   The Ohio State University
   279 Hamilton Hall
   1645 Neil Ave.
   Columbus, Ohio 43210
   (614) 292-4448
   bolte.6@osu.edu

This paper has not been peer- reviewed and should not be referenced in open literature.