

The Effect of Obesity on the Restraint of Automobile Occupants

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ABSTRACT – As obesity rates increase, the protection of obese occupants will become increasingly important in vehicle and restraint design. As a first step in this effort, this study seeks to compare the kinematics, dynamics, and injuries of obese post mortem human surrogates (PMHS) to (approximately) 50th percentile adult male PMHS in frontal impact sled tests with a force-limiting, pre-tensioning restraint system. Forty-eight km/h, frontal impact sled tests were performed with a sled buck representing the rear seat occupant compartment of a 2004 mid-sized sedan. The restraint system consisted of a 3-point belt with a pretensioner and a progressive force-limiter at the retractor. The test subjects were either obese PMHS or approximately 50th percentile adult male PMHS. Instrumentation included accelerometer packages on the spine. Deformation of the subjects' chests were measured using chestbands placed nominally at the superior-inferior locations of the 4th and 8th ribs. Tension in the restraint system was measured at the upper shoulder belt, lower shoulder belt, and the lap belt. Motion of the head, shoulder, pelvis, and knee were recorded using high-speed video. Two obese PMHS (average mass 137 kg, average stature 186 cm) and three approximately mid-sized male PMHS (average mass 68 kg, average stature 176 cm) were tested. The obese PMHS exhibited significantly greater forward motion of the head and the pelvis compared to the mid-sized PMHS. The obese PMHS also exhibited backwards torso rotation at the time of maximum forward excursion, whereas the mid-sized PMHS did not. The obese PMHS exhibited average maximum chest compressions of approximately 44% (\pm 9% standard deviation) of their initial chest depths, and exhibited 26 g (\pm 2 g) average 3 ms clip maximum chest resultant acceleration. In comparison, the mid-sized PMHS exhibited averages of 29% (\pm 9%) maximum chest compression and 35 g (\pm 4 g) maximum 3 ms clip chest acceleration. The obese PMHS exhibited 7 and 2 rib fractures, with maximum chest AIS scores of 3 and 2. The mid-sized PMHS exhibited 12, 2, and 17 rib fractures, with maximum chest AIS scores of 4, 1, and 4, respectively. This study is the first (to the authors' knowledge) to compare the kinematic, dynamic, and injury behaviors of obese and mid-sized PMHS in frontal impact sled tests with a force-limiting, pretensioning restraint system. The unfavorable kinematics observed with the obese PMHS highlights the difficulty of designing restraint systems to adequately restrain obese occupants, even with currently available advanced restraint technologies.

INTRODUCTION

Obesity is one of the greatest epidemics of our generation. From 1980 to 2000, the prevalence of obesity in Americans increased from 14.4% to 30.5% (Flegal et al. 2002). In 2005-2006, approximately 72 million Americans, over one third of the adult U.S. population, were obese (Ogden et al. 2007). This trend may be exacerbated in the coming years by a trend towards increasing obesity in younger people. Between the time periods of 1988-1994 and 2003-2004, the largest increases in the prevalence of abdominal obesity in U.S. adults occurred in the age range of 20-29 years (Li et al. 2007). Among 20-29 year olds, the prevalence of abdominal obesity increased by 100% in men, and 82% in women.

Obesity is most commonly defined by a person's Body Mass Index (BMI). BMI is calculated by dividing a person's body mass (in kilograms) by the square of their stature (in meters). The American Obesity Association designates a person as overweight if they have a BMI between 25 and 30 kg/m². Obesity is defined as a BMI greater than 30 kg/m².

Obesity is often associated with increased comorbidity and complications resulting from blunt impact trauma (Boulanger et al. 1992). In studies of patients admitted to level I trauma centers, Chohan et al. (1991) and Neville et al. (2004) both observed increased mortality in obese patients (compared to non-obese patients) despite similar injury severities. Chohan et al. (1991) observed an increase in

complications (mainly pulmonary) and length of stay in the obese group. Neville et al. (2004) and Boulanger et al. (1992) also observed that the majority of obese patients admitted to level I trauma centers with blunt trauma retained those injuries in motor vehicle collisions.

Obesity may affect fatality risk in automobile collisions because of the observed increase in post-injury complications and morbidity. In studies of NASS-CDS data, Viano et al. (2008a) and Mock et al. (2002) both observed increased fatality risk in obese occupants (defined by BMI > 30 kg/m²) compared to non-obese occupants. Injury trends with obesity, however, are less consistent. In a matched-pair analysis with NASS-CDS data (1993-2004), Viano et al. (2008a) observed that obese automobile occupants exhibited an approximately 57% greater risk of AIS 3+ injury in collisions than non-obese occupants. Mock et al. (2002) observed a non-significant trend in increased Injury Severity Score (ISS) with increased BMI when the analysis controlled for potentially confounding factors such as age, gender, and restraint use.

Obesity may also affect the distribution of body regions injured in automobile collisions. In their studies of blunt trauma patients admitted to level I trauma centers, Boulanger et al. (1992) and Neville et al. (2004) both observed that obese patients were more likely than non-obese patients to exhibit lower extremity injuries. Boulanger et al. (1992) also observed that obese patients were more likely to exhibit rib fractures than non-obese patients. In a study of NASS-CDS data, Mock et al. (2002) observed that increased BMI tended to increase the prevalence of AIS 3+ chest injuries. Jakobsson et al. (2005) observed an increase in AIS 2+ chest and lower extremity injuries in obese occupants in Volvo's automobile collision database compiled in Sweden, but did not observe those trends in data from NASS-CDS (1998-2002).

Despite the prevalence of obesity in our society and its affects on post-trauma outcomes, very little attention has been paid to obesity in automobile safety research. Current U.S. Federal Motor Vehicle Safety regulations and New Car Assessment Program testing rely on crash testing with a dummy with a mass of approximately 76.2 kg and a stature of approximately 173 cm. This corresponds to a BMI of approximately 25.5 kg/m² (Mackay et al. 1994). Furthermore, it is not feasible to scale (geometrically) the behavior of a 50th percentile male dummy to infer the mechanical response of an obese occupant in a collision. Body mass is distributed throughout the

body differently in the obese than in the non-obese (Friess et al. 2004). The physical relationship between an automobile occupant and the restraints and interior of a vehicle also change with obesity. For example, in a study of driver position and clearance with vehicle interior structures, Bove et al. (2006) found that the minimum distance between the steering wheel and the driver decreased with increasing BMI. This type of change in the relationship between the occupant and the vehicle interior would affect contacts, forces, and event timings in a collision in manners not accounted for by the geometric scaling of dummy responses.

Due to differences in weight distribution, tissue depth, etc., the effects of obesity on automobile occupant collision biomechanics are poorly understood. Although many studies have studied vehicle occupant biomechanics using post mortem human surrogates (PMHS, e.g. Kallieris et al 1982, Forman et al. 2006a), none have used PMHS to specifically study the collision biomechanics of obese automobile occupants. Obesity holds the potential to change the interaction with the vehicle restraints due to an increased depth of subcutaneous tissues between the restraints and osseous structures of the shoulder and pelvis. Obesity also holds the potential to affect occupant kinematics due to restraint interactions and due to the occupant mass and weight distribution. This study seeks to compare the kinematics, restraint interactions and injuries of obese and non-obese adult PMHS subjected to frontal impact sled tests.

METHODS

Test Configuration

Five PMHS were subjected to frontal sled tests with a nominal change in velocity (ΔV) of 48 km/h. The sled acceleration pulse (Figure 1) was approximately trapezoidal, with a nominal 20 g plateau and 80 ms duration.

As a first step in the study of the restraint of obese occupants, it was desired to test these subjects in an environment that isolated the subject interactions with the belt system and the seat from other factors (e.g., contact with an airbag or knee bolster). Thus it was desired to use a sled buck that included a seat and seatbelt anchor points, but did not include a knee bolster or airbag. This was accomplished using a sled buck representing the rear bench seat of a 2004 mid-sized sedan, with the front seat removed. The bench seat included a sheet-metal seat pan (reinforced to prevent deformation) formed over the rear wheel-

well (with no anti-submarining structure). This seat pan was covered by a foam seat cushion.

The restraints consisted of a deck-mounted, retractor pretensioned, progressive force-limiting 3-point belt system. The specific characteristics of this restraint system were described by Forman et al. (2008).

Test Subjects

Two of the test subjects were obese (BMI > 30). Three of the subjects were not obese (Table 1), and were chosen to target the stature and anthropometry of a 50th percentile male adult (approximately 175 cm stature and 75 kg mass). All subjects were screened for HIV and hepatitis A, B, and C. The subjects were also screened for pre-existing pathologies via CT scan. The subjects were unembalmed and were preserved until the time of testing by freezing. The subjects' pulmonary systems were pressurized (with compressed air) via tracheostomy to a nominal *in vivo* level (approximately 10 kPa measured

externally) immediately prior to testing. The subjects' cardiovascular systems were also pressurized (with 6% Hetastarch blood plasma replacement solution) to approximately 10 kPa (measured externally) immediately prior to testing. Injuries were identified post-test via autopsy. All PMHS test and handling procedures were approved by the University of Virginia institutional review board.

To the extent possible, the subjects were positioned according to the rear seat occupant posture study of Reed et al. (2005). While this was largely possible for the mid-sized subjects, the position and posture of the obese subjects was dictated mostly by their superficial tissue and the manner in which they settled into the seats. For those subjects, efforts were instead focused on attaining as upright a sitting position as possible, with the pelvis rotated as upright and pressed against the seat back to the extent possible. The initial seated postures of the subjects are shown in Figure 2.

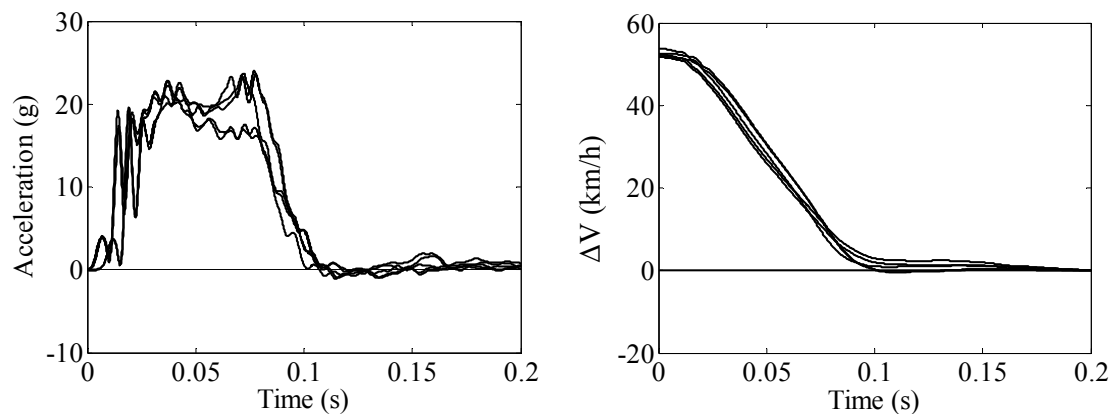


Figure 1: Composite plots of the sled accelerations (left) and changes in velocity (ΔV , right) for all tests.

Table 1: Test Matrix and Subject Characteristics

Test #	Subject #	Age/Gender	Stature (cm)	Mass (kg)	BMI* (kg/m ²)	ΔV (km/h)
1333	404	54/M	189	124	35	48.7
1335	400	53/M	182	151	45	48.2
1386	429	67/M	175	69	23	48.2
1387	444	69/M	171	67	20	49.6
1389	457	72/M	183	72	22	49.4

*BMI: Body Mass Index = Mass / Stature²

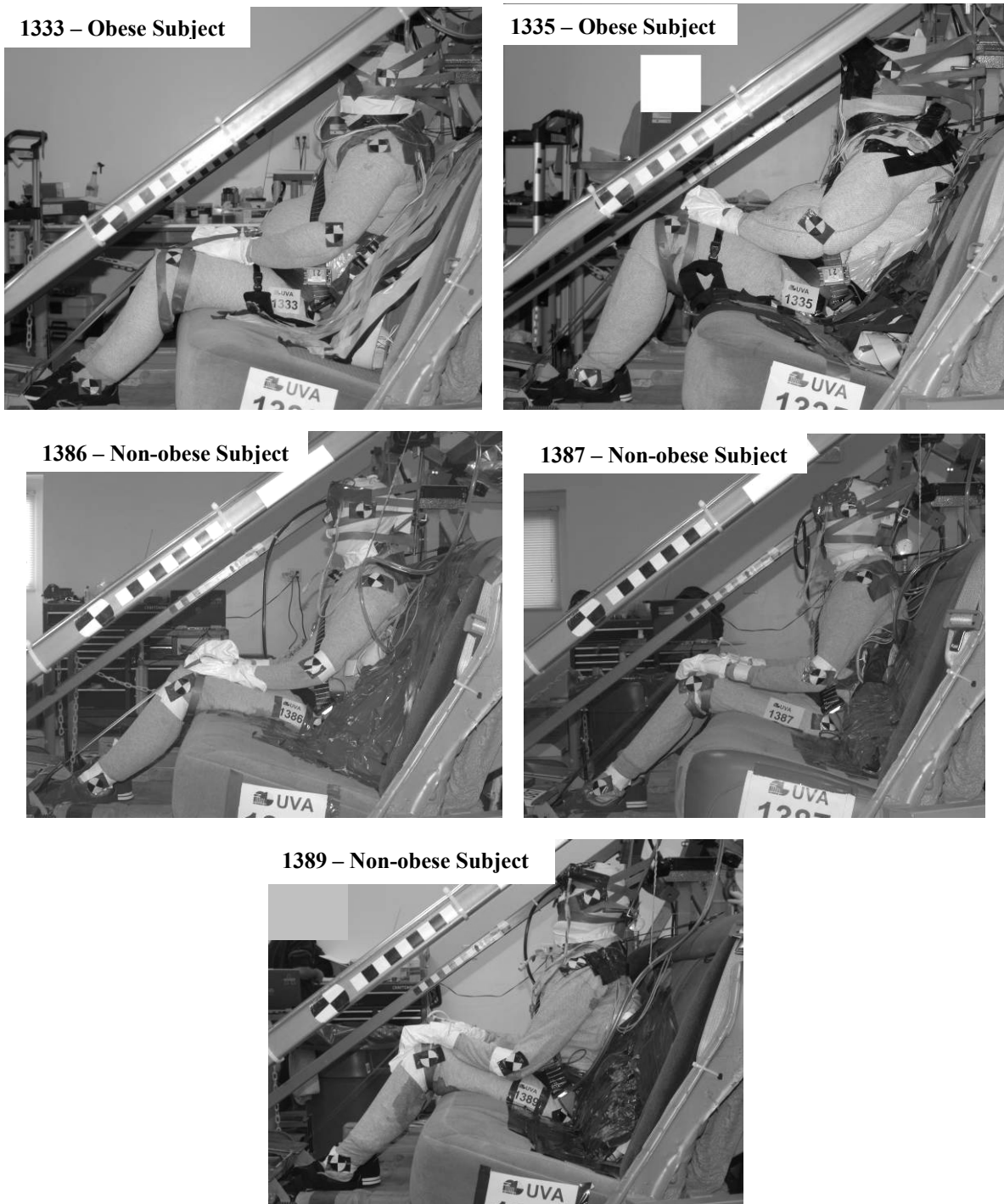


Figure 2: Pictures of each of the subjects positioned in the buck just prior to testing

Subject Instrumentation

Occupant accelerations were recorded with tri-axial accelerometer packages mounted to several locations on the body. Among other sites, the acceleration of the mid-spine was recorded with an accelerometer package mounted nominally to the eighth thoracic

vertebra. All data were collected at 10 kHz, and filtered according to SAE J211 specifications.

The overall kinematics of the occupants were recorded with off-board high-speed video (1000 frames per second). The trajectories of select anatomical targets were then digitized in the buck

reference frame. Points that were tracked included the head, the shoulder (defined as the lateral point of the acromion process), the hip (the greater trochanter), and the knee (the center of rotation). These points were marked externally with photo targets that were fixed to the skin (through the clothing) with surgical staples.

Chestbands were used to measure the deformation of the subjects' chests caused by loading from the seatbelt. Chestbands consist of a series of strain gauges attached to a thin, flexible strip of steel that is potted in a protective layer of rubber. The strain gauges are sensitive to bending of the strip. By wrapping the chestband around an object (e.g., a test subject's torso), the external contour of the circumference of that object may be calculated (for each step in time) by integrating the signals from each of the strain gauges on the band.

The chestbands used here (Model 4592, Robert A. Denton, Inc.) each contained 59 strain gauges (Forman et al. 2006b). One chestband was wrapped around each subject's torso approximately at the superior-inferior location of the lateral-most point of the 4th rib (the "upper" chestband). For most tests, a second chestband was wrapped around the subject's torso at the superior-inferior location of the lateral-most point of the 8th rib (the "lower" chestband). For the largest subject (test 1335) one chestband alone would not fit around the entire circumference of the upper chest. Thus, for that subject, two chestbands were used on the upper chest (one around the anterior surface of the chest, the other around the posterior surface of the chest), and the data from these two bands were combined to calculate a single closed contour for the upper chest.

The chestband data were analyzed according to the method described by Michaelson et al. (2008). The compression of the chest (defined as displacement of the anterior surface of the chest towards the posterior surface of the chest) was calculated by finding the change in length of a chord connecting the posterior aspect of the chestband contour to the anterior aspect of the chestband contour (Figure 3). Chest compression was calculated in the mid-sagittal plane (i.e., the mid-sternum), however the maximum chest compression most often occurred at some location lateral to the mid-sagittal plane. Thus, maximum chest compression was also calculated by allowing the lateral position of the chord connecting the anterior surface to the posterior surface to vary until the maximum value was found (Figure 3). Chest compression values are reported in absolute

displacement units (mm), and are reported as a percentage of the initial depth of the chest.

Restraint Measurements

One of the concerns regarding the restraint of obese subjects with a force-limiting belt is the amount that the belt may pay out of the retractor as the force-limiting element yields. To investigate this, the length of belt payout off of the retractor was measured using a high-speed imager that was mounted to the buck. The belt was marked at 1.5 cm increments. The imager recorded the motion of the belt off of the retractor as these markings passed a fixed reference point.

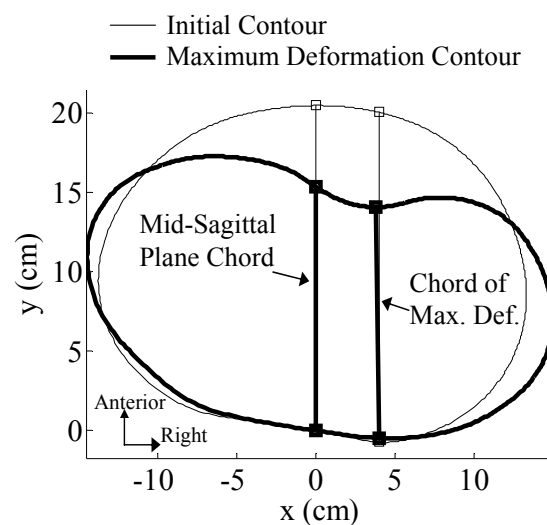


Figure 3: An example of chestband contours prior to testing and at the time of maximum chest compression. Also shown are the chords used to calculate the compression of the chest in the mid-sagittal plane (the mid-sternum) and at the lateral location at which maximum compression occurred.

The restraint system used in these tests included a torque-bar type progressive force-limiter in the retractor. This type of device provides a force-limit to the belt by yielding an element in the retractor spindle when the torque on the spindle exceeds a certain threshold. In theory, the force-limit resulting in the belt should be equal to the torque limit in the retractor divided by the radius of the outer layer of belt wrapped around the spindle (the "moment arm"). The radius of this moment arm is related to the number of times the belt wraps around the spindle, and is thus related to the amount of belt retracted into the retractor. As a result, the amount of belt in the retractor may affect the force limit observed in the upper shoulder belt during the test. To investigate this, the amount of belt wound around the retractor

was measured when the subjects were in their initial, restrained positions. The extra amount of belt wound into the retractor by the pretensioner was then observed via a high speed imager mounted to the buck.

Belt tension gauges were used to record the forces in the seat belts at three locations – the shoulder belt between the shoulder and the retractor (upper shoulder belt), the in-board portion of the shoulder belt between the subject and the buckle latch plate (lower shoulder belt), and the outboard portion of the lap belt between the subject and the lap belt anchor.

RESULTS

Peak recorded sensor data are included in Table 2. The peak data recorded for the obese group and the non-obese group were compared using a two-tailed Student's t-test, and data that were found to be significantly different ($p < 0.05$) are indicated in Table 2.

Plots of the upper shoulder belt time histories are shown in Figure 4. The initial length of belt on the retractor and the gross payout off of the retractor are included in Table 2. High speed video captures at 80 ms and 120 ms post-trigger are shown in Figure 5 and Figure 6. Trajectories of the head, shoulder, hip, and knee in the buck reference frame (up to the time of maximum forward head excursion) are shown in Figure 7.

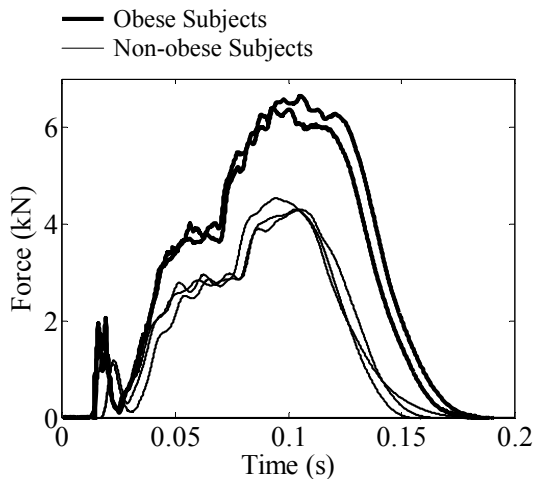


Figure 4: Upper shoulder belt force time-histories.

Sensor Data and Restraint Measurements

The tests on the obese subjects resulted in significantly greater upper shoulder belt force, greater lower shoulder belt force, and greater lap belt force than the tests on the non-obese subjects. The obese

subjects exhibited an average (\pm standard deviation) peak upper shoulder belt force of 6.6 ± 0.2 kN, compared to 4.4 ± 0.1 kN with the non-obese subjects. The obese subjects also had significantly less belt in the retractor prior to the tests. After pretensioning occurred, the obese subjects had an average of 47 ± 9 cm of belt wound around the spindle in the retractor, compared to 81 ± 9 cm with the non-obese subjects. The obese subjects also exhibited an average of approximately 4.8 cm greater gross belt payout (length of payout after pretensioning occurred) than the non-obese subjects.

The obese subjects exhibited significantly lower mid-spine resultant peak accelerations and 3 ms clip peak accelerations than the non-obese subjects. The obese subjects exhibited an average of 26 ± 1.8 g 3 ms clip peak resultant mid-spine acceleration, compared to 35 ± 4.3 g with the non-obese subjects.

The obese subjects exhibited a trend towards increased maximum upper chest compression (120 ± 21 mm, compared to 68 ± 29 mm) that approached the significance threshold ($p = 0.06$). Similarly, the obese subjects exhibited a trend towards increased maximum normalized chest compression ($44 \pm 10\%$ of the initial chest depth, compared to $29 \pm 9\%$). The lower chestband malfunctioned for one of the non-obese tests, thus lower chestband data are only available for one obese subject and two of the non-obese subjects. Although the statistical significance was not investigated, the obese subject appeared to exhibit greater maximum lower chestband compression (58 mm, 24% of the initial chest depth) than the two non-obese subjects (average of 29 ± 11 mm, $12 \pm 4\%$).

Kinematics and Trajectories

The obese subjects exhibited significantly greater forward motion of the pelvis (average of 49.1 ± 6.4 cm) and the head (74 ± 9.3 cm) compared to the non-obese subjects (18 ± 4.3 cm and 58 ± 4.3 cm, respectively). As shown in Figures 5, 6, and 7, the obese subjects also exhibited much less forward rotation of the torso. Because of their limited pelvis motion, the torsos of the non-obese subjects rotated forward as the shoulder belt paid out of the retractor. In contrast, the obese subjects exhibited trajectories that were largely straight forward. As their pelvis moved forward, the torsos of the obese subjects rotated backwards. This forward pelvis motion was great enough to cause the obese subjects to fall off of the front edge of the seat, resulting in the subjects coming to rest on the floor of the buck at the end of the test.

Table 2: Peak Data Results

Test #	Obese		Non-obese		
	1333	1335	1386	1387	1389
Subject	404	400	429	444	457
Length of belt on retractor (after pretensioning), cm*	53.4	40.0	87.5	86.8	74.3
Gross belt payout, cm*	28.0	26.0	23.1	20.3	23.1
Mid-spine acceleration*, g (time, ms)	33.0 (117)	29.1 (99)	40.9 (86)	41.0 (55)	35.0 (57)
Mid-spine 3ms clip acceleration, g*	26.9	24.4	35.4	39.2	30.6
Upper shoulder belt tension, kN* (time, ms)	6.43 (93)	6.68 (106)	4.29 (106)	4.30 (104)	4.53 (95)
Lower shoulder belt tension, kN* (time, ms)	6.29 (94)	7.27 (106)	3.52 (108)	3.25 (58)	4.74 (96)
Outboard lap belt tension, kN* (time, ms)	8.29 (91)	9.45 (95)	4.63 (85)	4.39 (63.5)	4.23 (89)
Upper chest band maximum deflection, mm	135	105	58	52	101
[%]† (time ms)	[50] (110)	[37] (120)	[25] (100)	[23] (100)	[39] (100)
Lower chest band maximum deflection, mm	58	--	21	36	--
[%]† (time ms)	[24] (110)	-- --	[9.6] (50)	[15] (50)	-- --
Upper chest band sternal deflection, mm	120	53	58	49	91
[%]† (time ms)	[44] (109)	[16] (95)	[25] (112)	[21] (111)	[34] (100)
Lower chest band sternal deflection, mm	8	--	3	24	--
[%]† (time, ms)	[2.8] (101)	-- --	[1.4] (52)	[9.4] (49)	-- --
Number of rib fractures	2	7	12	2	17
Chest Max AIS (AIS 2005)	2	3	4	1	4

* Peak results of the obese group are significantly different than the non-obese group ($p < 0.05$, one-tailed student's t-test)

** There was a large spike in the pelvis acceleration late in test 1335 presumably due to the subject falling off of the seat and striking the floor. That spike was removed from this maximum calculation and did not affect the calculation of the 3 ms clip maximum.

† Chest compression as a percent of the initial chest depth.

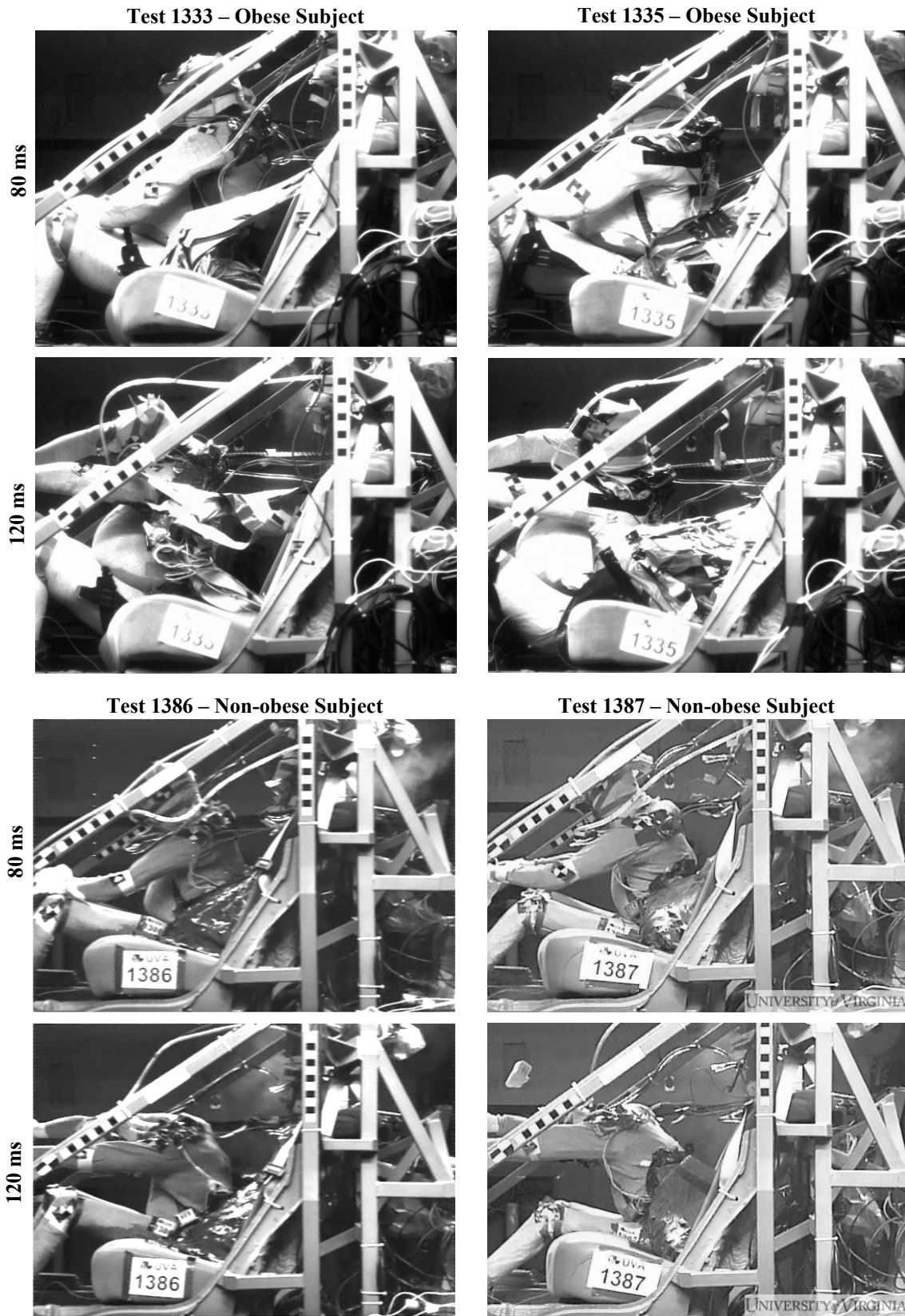


Figure 5: High speed video captures at 80 ms and 120 ms post-trigger comparing the typical kinematic behaviors between the obese subjects and the non-obese subjects.

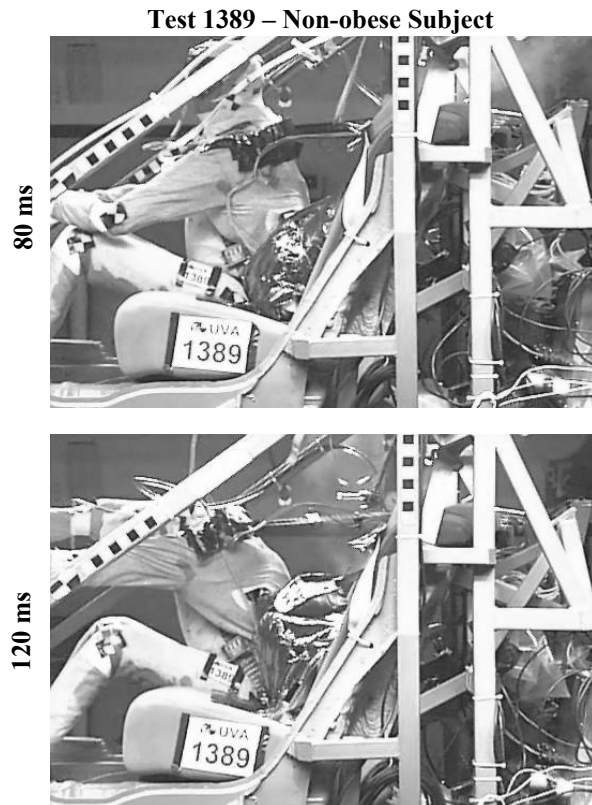


Figure 6: High speed video captures of test 1389 at 80 ms and 120 ms post-trigger.

Injuries

Each of the subjects tested exhibited some rib fractures post-test. The number of rib fractures for each subject (Table 2) varied greatly between subjects, and no trend was observed between the two test groups. The only other injuries that were observed are as follows:

- Test 1386 resulted in complete bilateral fractures through the neural arch of the second cervical vertebra (AIS 3).
- Test 1389 resulted in unilateral fractures of the right transverse processes of the 10th and 12th thoracic vertebrae and the 1st lumbar vertebra (AIS 2 each).

DISCUSSION

Kinematics

Contemporary restraint systems are designed to take advantage of the naturally strong structures of the body to decelerate automobile occupants safely in a collision. The lap belt is designed to load the

relatively strong structure of the anterior superior iliac spines (ASIS) of the pelvis; the shoulder belt is designed to load the relatively stiff structures of the clavicle and the upper ribcage. With the non-obese subjects, the lap belt appeared to primarily load the pelvis (slip of the lap belt into the abdomen was observed only in test 1386). The combination of limited pelvis motion and payout of the shoulder belt off of the retractor resulted in forward rotation of the torso. This forward torso rotation may tend to beneficially concentrate the shoulder belt force on the clavicle and upper ribcage. The trajectories of the non-obese subjects in Figure 5 and 6 illustrate the kinematic sequence that results from designed control of the occupant's motion by the restraint system. The pelvis is restrained by a combination of the removal of the belt slack by the pretensioner and by engagement of the ASIS, but the torso and head are allowed to rotate forward in a controlled manner as the belt force limiter absorbs energy.

In contrast, it appears that the restraint system was not as able to engage the targeted bony structures with the obese subjects. The obese subjects exhibited a large amount of forward pelvis motion, which may be indicative of limited engagement of the pelvis with the lap belt. This large forward pelvis motion also caused the torsos of the obese subjects to rotate backwards, likely resulting in increased belt loading on the lower chest and abdomen. The limited control of the obese subject's motion is illustrated by the relatively straight line trajectories in Figure 7, and was highlighted by the inability of the restraints to confine the subjects to remain seated on the seat cushion. Instead of the controlled restraint of the pelvis and forward torso rotation exhibited by the non-obese subjects, the obese subjects appear to have simply translated straight forward until they were fully decelerated.

The obese subjects also exhibited considerably greater forward motion of their knees than the non-obese subjects. Although this forward knee motion (and consequently, the forward pelvis motion) would have likely been decreased by the presence of a knee bolster or a front seat, it still suggests a few challenges facing the restraint of obese occupants. The increased subject mass, the increased forward knee motion, and the presumed decrease in the restraint of the pelvis by the lap belt all would tend to increase the forces generated in the lower extremity resulting from interaction with a front seat or knee bolster. This is consistent with studies that have observed a relationship between obesity and an increase in lower extremity injury risk in automobile collisions (e.g. Jakobsson et al. 2005).

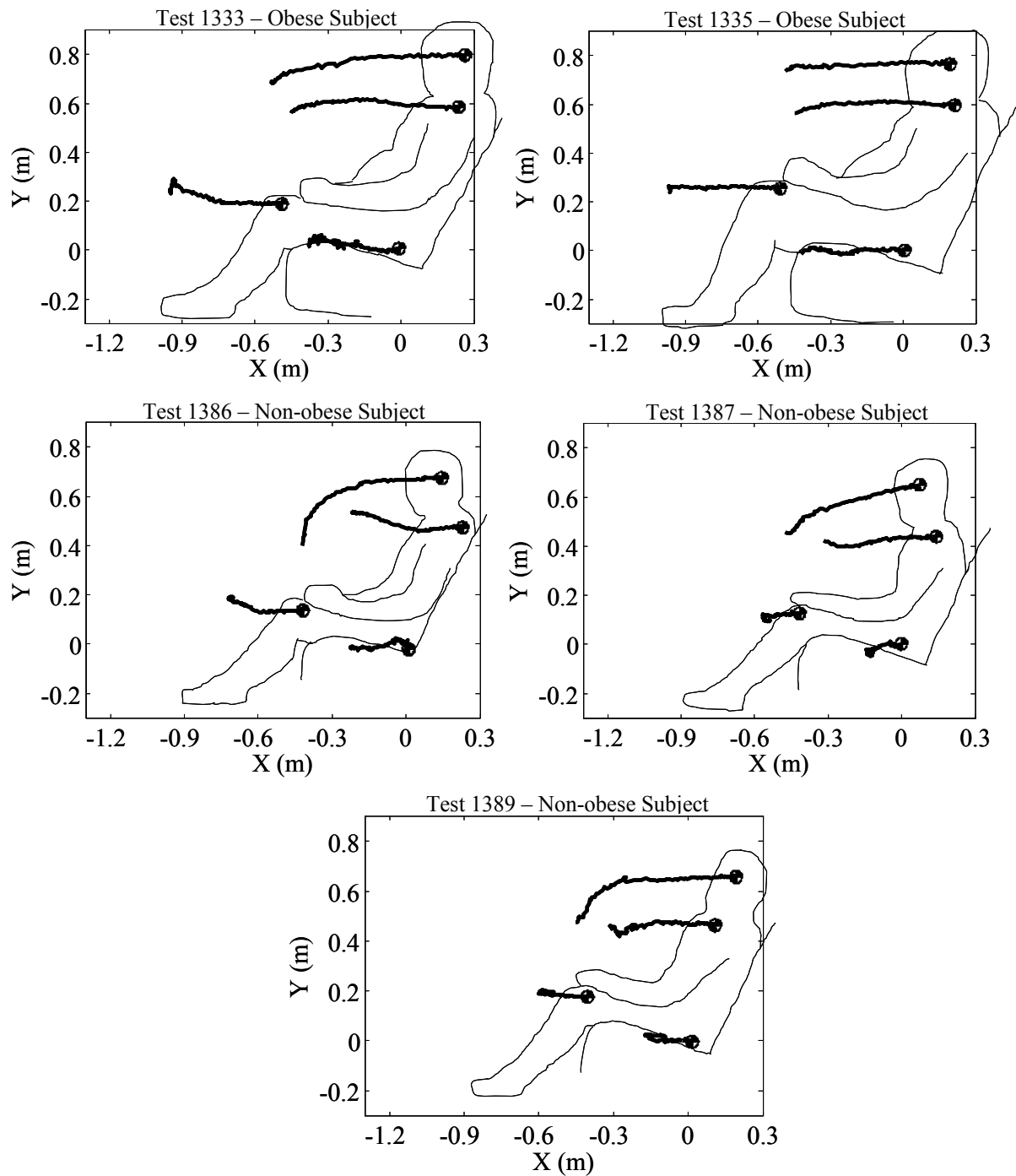


Figure 7: Plots of the trajectories of the subjects in the buck reference frame up to the time of maximum forward head excursion. The outlines illustrate the approximate initial positions of the occupants.

The large forward motions of the obese subjects are not entirely explained by increased belt payout off of the retractor. The obese subjects experienced an average of approximately 5 cm greater belt payout than the non-obese subjects, compared to an increase in forward head motion of approximately 16 cm, an

increase in forward shoulder motion of approximately 25 cm, and an increase in forward pelvis motion of approximately 30 cm. The increase in forward excursion may have been affected by the compliance and depth of the superficial tissue present in the obese subjects. The belt system likely had to

compress a considerable depth of this tissue prior to engaging the osseous structures of the shoulders and pelvis. Furthermore, although it was not possible to directly observe the interaction between the lap belt and pelvis (due to the large amount of superficial tissue), it is possible that the lap belt may have either slipped off of the pelvis (into the abdomen) or missed the pelvis entirely with the obese subjects. If either of these occurred, then the lap belt would have slipped into the abdomen, requiring considerable compression of the abdomen for the lap belt to engage the lumbar spine (possibly contributing to the observed forward motion of the pelvis).

Belt Forces

Although force-limiting restraints of the same model were used for the two test groups, the obese group exhibited greater upper shoulder belt forces than the non-obese group. This may be explained, at least partially, by the differences in the amount of belt wound around the retractor. As described in the introduction, the force limiters in the retractors operated by yielding an element in the retractor spindle at a prescribed torque. This torque is related to the force limit experienced in the upper shoulder belt by the radius of the outer layer of belt wrap around the retractor spindle. This radius is related to the number of wraps of belt around the spindle.

After pretensioning occurred, the obese subjects had an average of 47 cm of belt wrapped around the spindle, compared to 81 cm with the non-obese subjects. The amount of belt wrapped around the spindle at the time of maximum upper shoulder belt force (near the end of the tests) may be approximated by subtracting from these values the gross total amount of belt payout. This results in average of approximately 11 cm of belt around the spindle for the obese subjects, and 52 cm for the non-obese subjects.

The circumference of the spindle (without any belt) is approximately 12.8 cm. The number of wraps of belt around the spindle may be approximated by dividing the length of belt in the retractor by this circumference. For the obese subjects, there were an average of approximately 0.8 wraps of belt around the spindle at the end of the test; for the non-obese subjects there were approximately 4.1 wraps of belt around the spindle. The thickness of a single layer of the belt (when wrapped around the retractor) was approximately 0.18 cm. The radius of the spindle (without any belt) was approximately 2.03 cm. Thus, the average radius of the out layer of belt wrapped around the spindle was approximately $2.03 \text{ cm} + 0.8 \times 0.18 \text{ cm}$, or 2.18 cm, for the obese subjects. The

average outer spindle belt radius for the non-obese subjects was approximately 2.75 cm. This is a difference of approximately 26%.

The force in the upper shoulder belt should be approximately equal to the torque in the spindle divided by the radius of the outer wrap of belt around the spindle. Thus, a 26% increase in the outer spindle belt wrap radius should result in a 21% decrease in the upper shoulder belt force for a given amount of spindle torque. The non-obese subjects exhibited an average peak upper shoulder belt force approximately 33% less than the obese subjects. The predicted 21% decrease in belt force appears to account for a substantial portion of this observed difference (and is relatively close given the assumptions made above).

The relationship between belt force limit and the amount of belt wrapped around the retractor spindle may provide some benefit because, in essence, it results in the belt system acting as a passively adaptive restraint. As in the case presented here, larger vehicle occupants require a greater length of belt to properly wear their restraints. This results in less belt left in the retractor and less belt wrapped around the spindle. Because of their mass, these occupants also require greater belt forces to limit their forward excursions in a frontal collision. The analysis presented above suggests that the torque-limiting type of device used here will tend to result in greater belt forces applied to larger occupants simply due to the relationship between the length of belt in the retractor and the radius of belt wrap around the spindle. The relationship between a person's weight and the length of belt in the retractor is, of course, dependent on the individual's specific body habitus, and further work is needed to study the effects of the belt wrap around the spindle for persons of different body shapes. Using this phenomenon, however, it may be possible to specifically design a force-limiting retractor to be passively adaptive based on the general size of the occupant, and to optimize a retractor design to best protect a range of occupant sizes.

Injuries

Chest Injuries

The chest injuries observed (in terms of the number of rib fractures) varied considerably within both of the test groups. The two obese subjects exhibited 2 and 7 rib fractures; the number of rib fractures ranged from 2 to 17 in the non-obese groups. As a result, there was no significant difference in the number of rib fractures between the two test groups. Furthermore, the comparison of the thoracic injuries between the obese subjects and the non-obese

subjects is confounded by the subjects' ages. Kent et al. (2003) determined that the risk of rib fractures occurring from anterior loading increases with age. The obese subjects in this study were younger (ages 54 and 53) than the non-obese subjects (ages 67, 69, and 72). As a result, it is not appropriate to draw conclusions on the effect of obesity on thoracic injury risk based solely on the thoracic injuries observed here.

Some inconsistencies are present in the limited studies that have investigated the relationship between body habitus and injury risk for properly restrained automobile occupants. In a study of 1995-1999 NASS-CDS data, Moran et al. (2002) observed that for restrained drivers in frontal collisions, there was no significant relationship between body weight and the occurrence of thoracic injury. In a study of belted occupants in frontal impacts, Jakobsson et al. (2005) observed a greater risk of AIS2+ chest injury with obesity (defined by BMI) in data from Volvo's accident database in Sweden, but not in data from NASS-CDS (1998-2002). In a study of the 1993-2004 NASS-CDS data, Viano et al. (2008a and 2008b) did observe a general trend towards increasing risk of AIS3+ injuries in restrained drivers and right front passengers. That study did not, however, describe the distribution of injuries by body region or control for the type/direction of collision.

Nearly all of the available field data suggest, however, that for a given injury severity, there is an increased risk of mortality for the obese due to pulmonary complications, thromboembolic disease, infection, difficulties in treatment, and other factors (Boulanger et al. 1992). In a study of blunt trauma patients admitted to a level I trauma center over a period of seven months, Choban et al. 1991 observed that obese patients exhibited a greater mortality rate than non-obese patients, despite no observed difference in Injury Severity Score (ISS). The obese group exhibited a greater incidence of complications (mainly pulmonary), and the obese survivors tended to require a longer hospital stay than the non-obese survivors. Furthermore, the obese survivors also exhibited a significantly lower ISS than the non-obese survivors, prompting the authors to postulate that "[the data] suggests that in order to survive severely overweight patients can have no more than trivial injuries". In a similar study, Neville et al. (2004) also observed that of blunt trauma patients admitted to a level I trauma center, obese patients exhibited a greater incidence of mortality than non-obese patients, despite no significant differences in age, gender, or ISS. This is also reflected in vehicle collision field data. Using NASS-CDS data (of

various time periods) Viano et al. (2008a and 2008b), Moran et al. (2001), and Mock et al. (2002) all observed a greater risk of fatality for obese automobile occupants.

As a result, even if the effects of age are ignored then it is necessary to discuss the injuries presented here not just in terms the number of rib fractures, but also in terms of the effect of the subject's body habitus on their ability to recover from these injuries. The number of rib fractures in the two test groups studied here were not significantly different, but the obese subjects would be more likely than the non-obese subjects to develop complications from these injuries, resulting in a longer hospital stay and possibly death. Thus, if injury severity is evaluated based on cost of care or risk of death, then the injuries observed in the obese subjects may actually be more severe than those of the non-obese subjects due to the increased risk morbidity and complications associated with obesity.

Rib Fractures and Chest Compression

These results also illustrate the difficulty of predicting injury in obese subjects using injury risk functions developed with non-obese subjects. Kent et al. (2003) developed an age-dependent injury risk function relating chest compression measured at the sternum of PMHS to the risk of six or greater rib fractures. When applied to the subjects and chest compression results observed here, this function predicts a 79% risk of ≥ 6 rib fractures for the obese subject test 1333, and 16% risk for the obese subject test 1335 (Table 3). The actual number of rib fractures that occurred in these subjects, however, is juxtaposed with the predicted injury risk. Test 1333 resulted in 2 rib fractures, and test 1335 resulted in 7 rib fractures.

The inconsistency in test 1335 likely resulted from the maximum chest compression (37% of the initial chest depth) occurring lateral to the sternum. Although this type of phenomenon may occur when measuring the sternal compression of non-obese subjects, it may be exacerbated in obese subjects by differences in belt fit and the greater chest area over which the maximum chest compression may occur.

In contrast the inconsistency in test 1333 likely resulted from compression of the subcutaneous tissue superficial to the ribcage. In a non-obese person, 44% sternal chest compression is normally associated with a severe risk of multiple rib fractures, ribcage collapse, and consequent thoracic organ injury. In an examination of the blunt hub thoracic impact tests of Kroell et al. (1971, 1974), Viano (1978) suggested

that sternal chest compressions exceeding 40% of the initial chest depth would cause the ribcage to fail entirely, resulting in direct loading and injury of the underlying organs. Similarly, in a computed-tomography study of the kinematics of deformation of the thorax under anterior load, Kent et al. (2001) suggested that the physical limit of chest compression was 60% (compared to the 50% maximum chest compression of subject 404), at which point the posterior surface of the sternum would contact the anterior surface of the spine. The subject of test 1333, however, exhibited considerably greater superficial soft tissue than the non-obese subjects (Figure 8). During test 1333, some of the observed chest compression likely resulted of compression of soft tissue superficial to the ribcage. Thus, the 44% sternal chest compression observed in that subject is likely not related to compression of the ribcage and injury risk in the same manner as with non-obese persons.

Table 3: Prediction of Rib Fracture Risk from the Maximum Sternal Chest Compression

Test	Age	Sternal Comp.*	Risk of ≥ 6 Rib Fx.**	# of Rib Fx.
1333	54	44%	79%	2
1335	53	16%	2%	7
1386	67	25%	18%	12
1387	69	21%	10%	2
1389	72	34%	58%	17

* Maximum chest compression measured at the sternum, normalized by the initial chest depth.

** Predicted with the age-dependent injury risk function described by Kent et al. (2003).

Neck Injuries

The C2 fracture in test 1386 (non-obese) may be consistent with a hyper-flexion type of injury mechanism. Forward torque on the dens caused by forward flexion of the head would cause bending stresses in the neural arch/pedicles of C2, consistent with the observed location of this injury. This is also consistent with the rear seat, frontal impact sled tests of Michaelson et al. (2008), which observed flexion-related injuries to the cervical spine in PMHS restrained by a standard 3-point belt system. As explained in that study, however, it is unclear how neck injuries of this type, observed in PMHS studies, relate to neck injury risk in living humans. PMHS are unable to model the muscle tone and muscle activation present in living humans. In addition, post-mortem changes may decrease the stiffness of passive muscle tissue relative to living tissue (Van Ee et al.

1998). Computational studies have suggested that active neck musculature may change the injury tolerance of the neck and the location of neck injuries under tensile loading, flexion, and lateral bending (Van Ee et al. 2000, Oi et al. 2004, Brodin et al. 2005). In addition, the limited field data suggest that inertial neck injuries are rare compared to head and thoracic injuries in belted occupants in frontal collisions, even when an airbag is not present (e.g., in restrained rear seat occupants, Kuppa et al. 2005). Thus, because it is uncertain how the neck injuries observed here relate to injury risk in living humans, conclusions on the effect of obesity on inertial neck injury risk in frontal collisions should not be drawn from the observations of this study alone.

Abdomen

No abdominal injuries were observed in any of the test subjects, despite the lap-belt loading of the abdomen that presumably occurred with the obese subjects. This is consistent with other frontal-impact cadaver studies that have observed submarining with no abdominal injury (e.g., Kallieris et al. 1982). This may have been affected, however, by post-mortem degeneration of the abdominal tissues. Post-mortem discoloration of the skin and a lack of blood flow may make it difficult to observe acute skin contusions that result from abdominal seatbelt loading in living humans. Autolysis of the abdominal organs may also make it difficult to identify visceral damage that may be readily identifiable in living humans due to bleeding or other acute pathologies. Due to the uncertainty resulting from these post-mortem degenerative changes, the possibility of abdominal injury should not be excluded based on the lack of identification in these tests.

Limitations

The study presented here is the first step towards investigating the fundamental effects of obesity on occupant kinematics, restraint interactions, and injuries in automobile collisions. There are, however, some important limitations to this study. First, the addition of other restraining structures typically found in automobiles (e.g. airbags, knee bolsters, or the front seat back for rear seat occupants) may affect the occupant kinematics, restraint interactions, and resulting injuries. As discussed above, the addition of a knee bolster or front seat back would likely decrease the forward pelvis motion of the obese occupants, but may result in injury to the legs. Likewise, the addition of an airbag may help to limit the forward motion of the head and flexion of the neck, possibly helping to protect against neck injuries similar to those observed in tests 1386 and 1389 (e.g., Forman et al. 2006a). An airbag may also change the

kinematics of the torso and the distribution of restraining force over the chest. Thus, future studies should investigate the effects of obesity on occupant kinematics and injuries in more complex vehicle environments involving the multiple restraint components present in contemporary automobiles.

Second, these tests studied one specific restraint environment and one specific collision scenario. The kinematics and injury responses of the test subjects are likely dependent on the specific characteristics of the seat and the restraint system. For example, it is

unknown if the kinematic trends observed here would also occur if a bucket seat with an anti-submarining structure were used. Likewise, it is unknown how obesity would affect occupant kinematics and injury in other collision scenarios (such as side impact or roll-over) and at other collision speeds. Thus, although this study provides a first look at some of the biomechanical issues facing the restraint of obese occupants, additional work is required to investigate the effects of obesity in other restraint, vehicle, and collision environments.

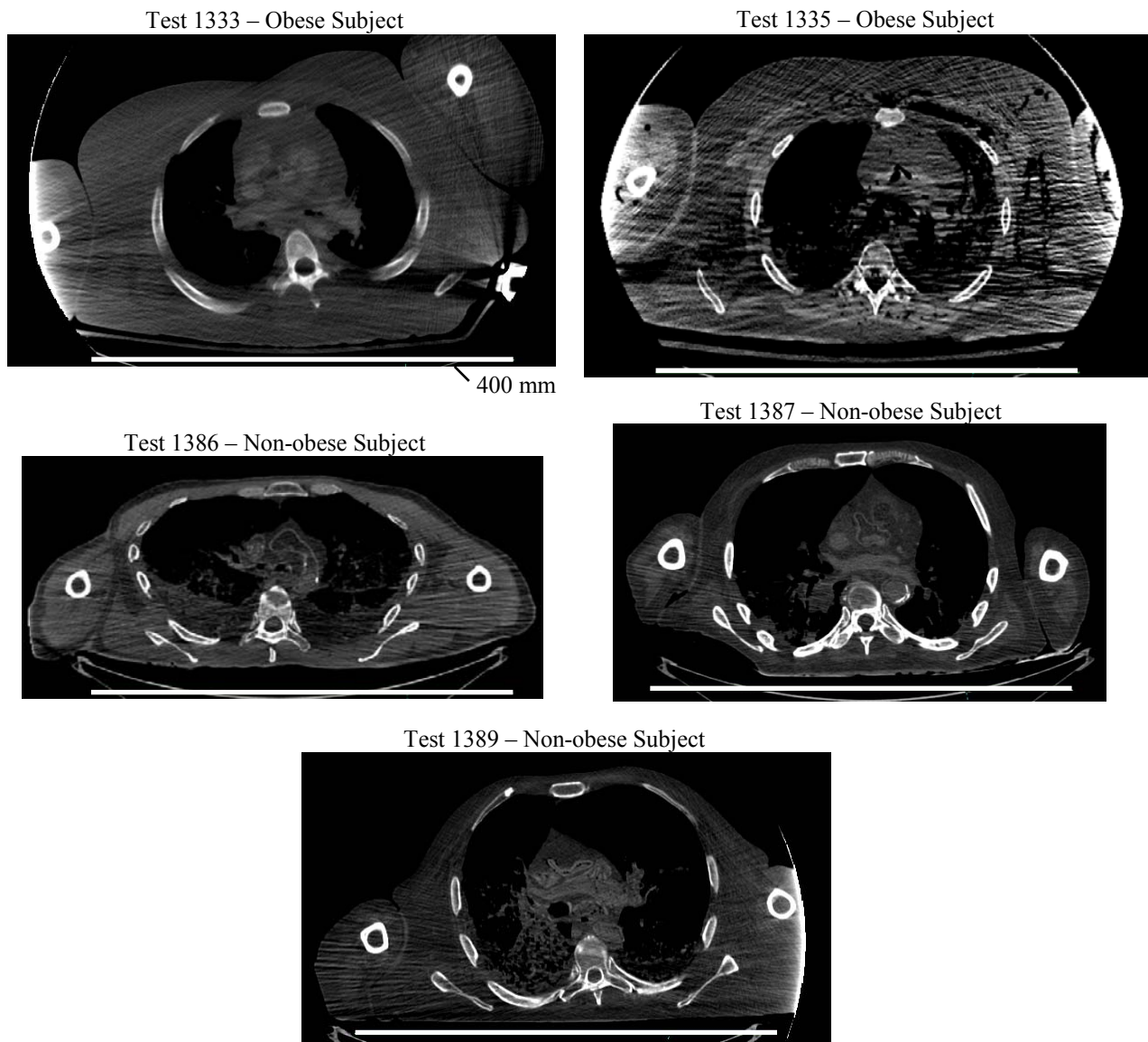


Figure 8: CT scan cross-sections of the five test subjects illustrating the differences in superficial soft tissue depth. These cross sections were taken approximately at the superior-inferior locations of the upper chest bands (i.e., the lateral-most location of the 4th rib). All images are to scale.

CONCLUSIONS

Frontal impact, 48 km/h sled tests were performed to compare the kinematics and injuries of obese PMHS to non-obese PMHS restrained by a progressive force-limiting, pretensioning restraint system in a rear seat automobile environment. The obese subjects exhibited significantly greater peak belt forces; greater forward excursions of head, knee, and pelvis; and significantly lower mid-spine resultant accelerations. The obese subjects exhibited a non-significant trend towards increased peak chest compression, but did not exhibit any observable difference in chest injuries. To the author's knowledge, this is the first study to compare the kinematics, restraint interactions, and injuries of obese subjects to non-obese subjects using frontal impact sled tests with PMHS.

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REFERENCES

- Boulanger BR, Milzman D, Mitchell K, Rodriguez A. Body Habitus as a Predictor of Injury Pattern After Blunt Trauma. Journal of Trauma Vol. 33, No. 4, pp 228-232, August 1992.
- Brolin K, Halldin P, Leijonhufvud I. The Effect of Muscle Activation on Neck Response. Traffic Injury Prevention Vol. 6, pp 67-76, 2005.
- Choban PS, Weireter LJ, Maynes C. Obesity and Increased Mortality in Blunt Trauma. Journal of Trauma Vol. 31, No. 9, pp 1253-1257, September 1991.
- Forman J, Lessley D, Kent R, Bostrom O, Pipkorn B. Whole-Body Kinematic and Dynamic Response of Restrained PMHS in Frontal Sled Tests. Stapp Car Crash Journal Vol. 50, pp 229-336, 2006a.
- Forman J, Lessley D, Shaw CG, Evans J, Kent R, Rouhana SW, Prasad P. Thoracic Response of Belted PMHS, the Hybrid III, and the THOR-NT Mid-Sized Male Surrogates in Low Speed, Frontal Crashes. Stapp Car Crash Journal Vol. 50, pp 191-215, 2006b.
- Forman J, Michaelson J, Kent R, Kuppa S, Bostrom, O. Occupant Restraint in the Rear Set: ATD Responses to Standard and Pre-Tensioning, Force-Limiting Belt Restraints. Annual Proceedings of the Association for the Advancement of Automotive Medicine, Vol. 52, pp 141-154, 2008.
- Friess M, Corner BD. From XS to XL: Statistical Modeling of Human Body Shape Using 3D Scans. Society of Automotive Engineers, Paper No. 2004-01-2183, 2004.
- Jakobsson L, Lindman M. Does BMI (Body Mass Index) Influence the Occupant Injury Risk Pattern in Car Crashes? Proceedings of the International Research Council on the Biomechanics of Impact, pp 427-430, September 2005.
- Kallieris D, Mellander H, Schmidt G, Barz J, Mattern R. Comparison Between Frontal Impact Tests with Cadavers and Dummies in a Simulated True Car Restrained Environment. Proceedings of the 26th Stapp Car Crash Conference, pp 353-367, 1982.
- Kent R. Dynamic Response of the Thorax: Restraint-Specific Injury Prediction and Constitutive Description. Ph.D. Dissertation, University of Virginia, 2001.
- Kent R, Patrie J, Poteau F, Matsuoka F, Mullen C. Development of an Age-Dependent Thoracic Injury Criterion for Frontal Impact Restraint Loading. Proceedings of the Conference on the Enhances Safety of Vehicles, 2003.
- Kroell C, Schneider D, Nahum A. Impact Tolerance and Response of the Human Thorax. Society of Automotive Engineers, Paper No. 710851, 1971.
- Kroell C, Schneider D, Nahum A. Impact Tolerance of the Human Throat II. Society of Automotive Engineers, Paper No. 741187, 1974.
- Kuppa S, Saunders J, Fessahaie O. Rear Seat Occupant Protection in Frontal Crashes. 19th Proceedings, International Technical Conference on the Enhanced Safety of Vehicles, 2005.
- Li C, Ford ES, McGuire LC, Mokdad AH. Increasing Trends in Waist Circumference and Abdominal Obesity Among U.S. Adults. Obesity Vol. 15, No. 1, pp 216-224, January 2007.
- Mackay M. Smart Seat Belts – some Population Considerations Applied to Intelligent Restraint Systems. Society of Automotive Engineers, Paper No. 940531, 1994.
- Michaelson J, Forman J, Kent R, Kuppa S. Rear Seat Occupant Safety: Kinematics and Injury of PMHS Restrained by a Standard 3-Point Belt in Frontal

- Crashes. Stapp Car Crash Journal Vol. 52, pp 295-325, 2008.
- Mock CN, Grossman DC, Kaufman RP, Mack CD, Rivara FP. The Relationship Between Body Weight and Risk of Death and Serious Injury in Motor Vehicle Crashes. Accident Analysis & Prevention Vol. 34, pp 221-228, 2002.
- Moran SG, McGwin G, Reiff DA, Rue LW. The Association Between Body Habitus, Restraint Use, and Fatality in Motor Vehicle Collisions. 45th Annual Proceedings, Association for the Advancement of Automotive Medicine, pp 107-123, 2001.
- Moran SG, McGwin G, Metzger JS, Windham ST, Reiff DA, Rue LW. Injury Rates Among Restrained Drivers in Motor Vehicle Collisions: The Role of Body Habitus. Journal of Trauma Vol. 52, pp 1116-1120, June 2002.
- Neville AL, Brown CV, Weng J, Demetriades D, Velhamos GC. Obesity is an Independent Risk Factor of Mortality in Severely Injured Blunt Trauma Patients. Arch. Surg. Vol. 139, pp 983-987, September 2004.
- Ogden CL, Carroll MD, McDowell MA, Flegal KM. Obesity Among Adults in the United States – No Statistically Significant Change Since 2003-2004. Centers For Disease Control, Data Brief Number 1, pp. 8, November 2007.
- Oi N, Pandey M, Myers B, Nightingale R, Chancey V. Variation of Neck Muscle Strength Along the Human Cervical Spine. Stapp Car Crash Journal 48, pp 397-417, 2004.
- Reed MP, Ebert-Hamilton SM, Schneider LW. Development of ATD Installation Procedures Based on Rear-Seat Occupant Postures. Stapp Car Crash Journal Vol. 49, pp 381-421, 2005.
- Van Ee C, Chasse A, Myers B. The Effect of Postmortem Time and Freezer Storage on the Mechanical Properties of Skeletal Muscle. Society of Automotive Engineers, Paper No. 983155, 1998.
- Van Ee C, Nightingale R, Camacho D, Chancey V, Knaub K, Sun E, Myers B. Tensile Properties of the Human Muscular and Ligamentous Cervical Spine. Stapp Car Crash Journal 44, pp 85-102, 2000.
- Viano DC. Thoracic Injury Potential. Proceedings of the 3rd Conference of the International Research Council on the Biomechanics of Impact, 1978.
- Viano DC, Parenteau CS, Edwards ML. Crash Injury Risks for Obese Occupants Using a Matched-Pair Analysis. Traffic Injury Prevention Vol. 9, No. 1, pp 69-64, 2008a.
- Viano DC, Parenteau CS. Crash Injury Risks for Obese Occupants. Society of Automotive Engineers, Paper No. 2008-01-0528, 2008b.