Is There Really a "Cushion Effect"?: A Biomechanical Investigation of Crash Injury Mechanisms in the Obese

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The objective of this study was to document the motion and potential injury mechanisms of obese occupants in frontal car crashes compared to a control group of nonobese occupants in controlled laboratory impacts. Eight cadavers were divided into obese (n = 3) and a nonobese (n = 5) groups and exposed to a 48 km/h impact. High-speed digital video documented the motion of the belted subjects. Compared to the nonobese cohort, the obese exhibited a characteristically different set of motions. As expected, the obese (heavier) subjects experienced greater maximum forward displacement (excursion) before their motion was arrested by the restraint. In addition, the obese exhibited a different distribution of excursions among body segments. The primary difference between the cohorts was substantially larger hip excursion in the obese (452 ± 83 mm vs. 203 ± 42 mm, $P < 0.01$), which was the proximate cause of a tendency of the obese cadavers' torsos to pitch forward less during impact. Some of the published epidemiology can be elucidated by the results reported here. The increased hip excursion and concomitant decreased torso pitch may reduce the risk of the head striking some component of the vehicle interior. Furthermore, the reclined torso during belt loading may increase the risk of rib and pulmonary trauma because the load is concentrated on the compliant and vulnerable lower thorax and less on the stiff upper ribs and clavicle. The lower extremities also experience increased excursion as a result of this hip excursion, and thus an increased risk of a hard contact and resulting injury.

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INTRODUCTION

An increasing rate of obesity has been documented for both children and adults in many developed nations (e.g., refs. 1–3). The health consequences of this trend have also been well documented, including increased risk of coronary disease, diabetes, certain cancers, hypertension, and dyslipidemia (4).

Less well understood are the consequences of obesity related to trauma. The literature is reasonably consistent in the finding that obesity is an independent risk factor for adverse outcomes, including death, following blunt trauma. Neville *et al.* (5) analyzed 242 patients critically injured from blunt trauma and found that those with a BMI of 30 kg/m^2 had a higher incidence of multiple organ failure and mortality. Zhu *et al.* (6) found an increased risk for death with increasing BMI in male drivers involved in crashes. Mock *et al.* (7) studied 36,206 crash-involved automotive occupants, which represented a national sample of nearly 12 million crashes and found a significant increase in fatality risk with increasing weight or BMI when the confounding factors occupant age, gender, belt use, seated position, and vehicle weight were controlled. Choban *et al.* (8) considered 351 patients with blunt trauma and reported a substantially higher mortality rate and rate of complications, especially pulmonary, for patients with BMI greater than 31 kg/m^2. Interestingly, Tremblay and Bandi (9) found that low BMI, but not high BMI, was associated with poor outcomes in a population of 41,011 patients admitted to an intensive care unit for any reason, including nontrauma, and concluded that overweight and obese patients have improved mortality and discharge functional status. No explanation for this difference between trauma patients and all intensive care unit patients was given.

It seems appropriate to conclude, based on the above studies, that the risk of death is greater for obese people who sustain blunt trauma. It is important to consider, however, that the risk of death is influenced by two factors potentially related to obesity: (i) the risk of death given a particular injury, which includes obesity-related treatment and diagnostic factors that may increase the risk of misdiagnosis and complications following an injury, and (ii) the risk of sustaining the injury in the first place. The literature is less conclusive on the

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second point. Some of the studies mentioned above attempted to quantify a relationship between obesity and injury risk. In ref. 7, the adjusted odds ratios for weight or BMI and an injury outcome did not attain statistical significance. Whitlock *et al.* (10) reported a 10-year prospective study of New Zealand men and women in which participants in the highest and lowest quartiles of BMI were more likely to have sustained an injury while driving, but concluded that further research is needed to assess the hypothesis that BMI is a risk factor for driver injury in car crashes. Similarly, Moran *et al.* (11) did not identify consistent relationships between height, weight, and injury outcome in crash victims.

Some studies have hypothesized that obesity is related to different patterns of injury if not risk and have even hypothesized differences in crash kinematic for obese and nonobese drivers, including the assertion that fat acts as an "intrinsic airbag" with a "cushioning effect," though none of the assertions regarding injury mechanism is based on actual observation (12,13). A group at the University of Michigan (12,14) found lower mean injury severity in the abdomen of obese patients compared to leaner patients and a higher mean severity in the lower extremities. Both studies concluded that the lower mean severity in the abdomen was the result of a protective cushioning effect from abdominal fat, but the use of a convenience sample of trauma patients with no exposure information limits the conclusions that can be drawn regarding risk and the lack of direct observation of the crash mechanics limits the conclusions that can be drawn regarding injury mechanism. Jakobsson and Lindman (15) did not observe a decrease in abdominal injury rate for obese crash victims when exposure was considered in nationally representative databases of Swedish and US crashes and, in fact, did not find any consistent trends associated with BMI and injury outcome. Recently, Viano *et al.* (16) used a matched-pair analysis to assess fatality and serious injury risk for obese drivers in crashes and a novel model of body deformation to account for the increased kinetic energy associated with increased body mass. This study and a companion article (17) reported an increase in both fatality and serious injury associated with obesity. The definition of serious injury in both studies, however, included death, so it is difficult to isolate any obesity-related effect on injury risk from the well established obesity-related risk of death given an injury.

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 US Federal Motor Vehicle Safety regulations and New Car Assessment Program testing rely on crash testing with a dummy with a mass of ~76.2 kg and a stature of ~173 cm. This corresponds to a BMI of ~25.5 kg/m^2. 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 nonobese. The physical relationship between an automobile occupant, the restraints, and the interior of a vehicle also change with obesity, which affects the magnitude and timing of the applied loads during a crash in manners that cannot be inferred from crash test dummy behavior.

The literature is lacking a controlled prospective observational study of the crash mechanics of obese people. Such a study is critical for elucidating the field observations and currently unconfirmed hypotheses regarding injury mechanism and obesity. The purpose of this article is to report differences in restraint interaction and crash biomechanics for obese and nonobese car occupants in a small set of well controlled and documented laboratory impacts. Retrospective studies of even large databases of crash victims have not adequately described the consequences of obesity in terms of occupant motion and injury mechanisms. The goal of the study reported here is to identify key aspects of obesity that may lead to better interpretation of retrospective crash outcome studies.

METHODS AND PROCEDURES

Frontal impacts account for the majority of crashes and fatal crashes (e.g., ref. 18), so that crash mode was chosen for study. Impact severity was based on Federal Motor Vehicle Safety Standard 208 (48 km/h impact speed) with a deceleration pulse based on the crash test performance of a popular mid-size US sedan (19,20). Two different seatbelt configurations were considered. The first was a typical three-point lap-shoulder belt system with sliding latch plate and no advanced features (standard belt). The second was identical except a retractor with force-limiting and pretensioning was used (force-limited belt-pretensioned). The pretensioner retracted several centimeters of belt webbing upon impact and the force-limiter yielded to allow webbing to be pulled off of the spool as the occupant loaded the belt system. These advanced features are present in most new vehicles (21) and are intended to reduce the belt system response time (pretensioner) and mitigate the risk of thoracic injuries from excessive belt impingement (force-limiter). An unbelted configuration was considered but eventually discarded due to the wide range of initial conditions that an unbelted occupant can assume and the complex injury mechanisms acting on a completely unrestrained occupant. An airbag was not used so that the biomechanics of the belt interaction would not be confounded by an additional load path and also to avoid occlusion of the video documentation of the occupant's kinematics.

Obese crash test dummies do not exist, so obese and nonobese human cadavers were used for this study. All cadaver handling and test protocols were approved by an institutional oversight committee at the University of Virginia. Eight cadavers were divided into an obese group ($n = 3$) and a nonobese group ($n = 5$) (**Table 1**). Cadavers were preserved until the time of testing by freezing at 0 °C. Prior to freezing blood was drawn and screened for Hepatitis B, C, and HIV. Freezing occurred ~1 week after death, during which time the subjects were stored in a refrigerator. Thawing occurred at room temperature and occurred over ~48 hours. Age, weight, height, and cause-of-death criteria were used to screen the cadavers. Subjects that were nonambulant for an extended period prior to death or that had a cause of death associated with bony lesions were excluded from the study. Pretest computed tomography scans (0.65 mm slice thickness) (Lightspeed, GE Healthcare, Little Chalfont, UK) were taken and read by a radiologist to exclude subjects with bone pathology, including osteoporosis. Pulmonary and cardiovascular systems were pressurized to nominal *in vivo* levels immediately before testing using pressurized (7 kPa nominal) air introduced through a tracheostomy and a plasma replacement solution (Hetastarch) injected under pressure into the common carotid artery.

Test subjects were positioned in the right rear position of a contemporary mid-size vehicle buck mounted to a deceleration sled (Via Systems model HITS 713, Brighton, MI) with a programmable hydraulic decelerator (Via Systems model 931-4000, Brighton, MI) (**Figure 1**). High-speed digital imagers documented the crash

Table 1 Experimental matrix and subjects test

	Belt	Subject ID	Age/gender	Stature (m)	Weight (kg)	BMI (kg/m²)	Cause of death
Nonobese							
1262	SB	362	51/M	1.75	54.9	17.9	Anoxic brain injury
1264	SB	367	57/M	1.79	59.0	18.4	Brain tumor
1386	FLB-PT	429	67/M	1.75	71.0	23.2	Parkinson's disease
1387	FLB-PT	444	69/M	1.71	59.9	20.5	Melanoma
1389	FLB-PT	457	72/M	1.83	72.6	21.7	Pneumonia
Mean nonobese			63.2	1.77	63.5	20.3	
Obese							
1263	SB	394	57/F	1.65	108.9	40.0	Myocardial infarction
1333	FLB-PT	404	54/M	1.89	124.0	34.7	Schizophrenia[a]
1335	FLB-PT	400	53/M	1.82	151.0	45.6	Sudden death[a]
Mean obese			54.7	1.79	128.0	40.1	

FLB-PT, force-limited belt-pretensioned; SB, standard belt.
[a]No other information available from tissue supplier. No bone pathology, including metastatic tumors, identified in either pretest CT or post-test autopsy of any subject.

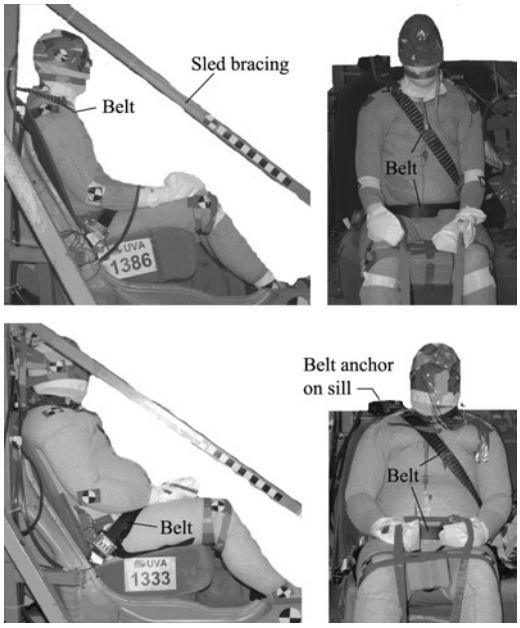

Figure 1 Initial positions of a typical nonobese cadaver (top two photos, test 1386) and an obese cadaver (bottom two photos, test 1333). Note the differences in initial belt fit.

kinematics at a frame rate of 1,000 Hz. Targets mounted to the ankle (lateral malleolus), knee (lateral femoral epicondyle), hip (greater trochanter), and shoulder (humeral head) were tracked throughout the impact to quantify the occupant motion in the sagittal plane. Excursion was defined as the forward displacement relative to the vehicle interior.

RESULTS

Compared to the nonobese cohort, the obese subjects exhibited a characteristically different set of motions regardless of belt type (**Figure 2** and **Figure 3**). As expected, the obese (and heavier) subjects generally experienced greater maximum excursion before their motion was arrested by the restraint. In addition, the obese subjects exhibited a substantially different distribution of excursions among their body segments. The primary difference between the two cohorts was substantially larger hip excursion in the obese cadavers (obese 452 ± 83 mm vs. 203 ± 42 mm, $P < 0.01$, **Figure 3**), which was the proximate cause of a tendency of the obese cadavers' torsos to pitch forward much less during impact (**Table 2**). This difference in torso pitch, which is apparent in the video images (**Figure 2** and **Figure 3**), can be quantified using the ratio of maximum shoulder excursion to maximum hip excursion, where a small number indicates less forward pitch of the torso (obese 1.23 ± 0.37 vs. 2.05 ± 0.80, $P < 0.05$, **Figure 3**). Despite this lack of forward pitch, the heads of the obese cadavers tended to experience slightly greater maximum excursion relative to the vehicle interior (obese 657 ± 151 mm vs. 554 ± 47 mm, $P = 0.19$, **Figure 3**). The knees of the obese cadavers exhibited significantly greater excursion (obese 395 ± 103 mm vs. 220 ± 58 mm, $P < 0.05$, **Figure 3**).

DISCUSSION

To the extent that these experiments reflect crash conditions experienced by obese occupants in the field, some of the published epidemiology can be elucidated by the kinematics of the subjects reported here. Boulanger *et al.* (13) reported a lower incidence of head trauma in obese patients and an increased incidence of rib fractures, pulmonary contusions, pelvic fractures, and extremity fractures. Wang *et al.* (14) also advanced the idea of increased risk of injury to the extremities associated with obesity. These findings are consistent with the injury mechanics observed in the cadavers. First, the increased hip excursion and concomitant decreased torso pitch in the obese cadavers may reduce the risk of the head striking some component of the vehicle interior in frontal or near-frontal impacts. Furthermore, the reclined torso attitude during belt loading may increase the risk of rib fractures and pulmonary trauma because the load on the chest is concentrated on the more compliant and vulnerable lower thorax and less on the stiff

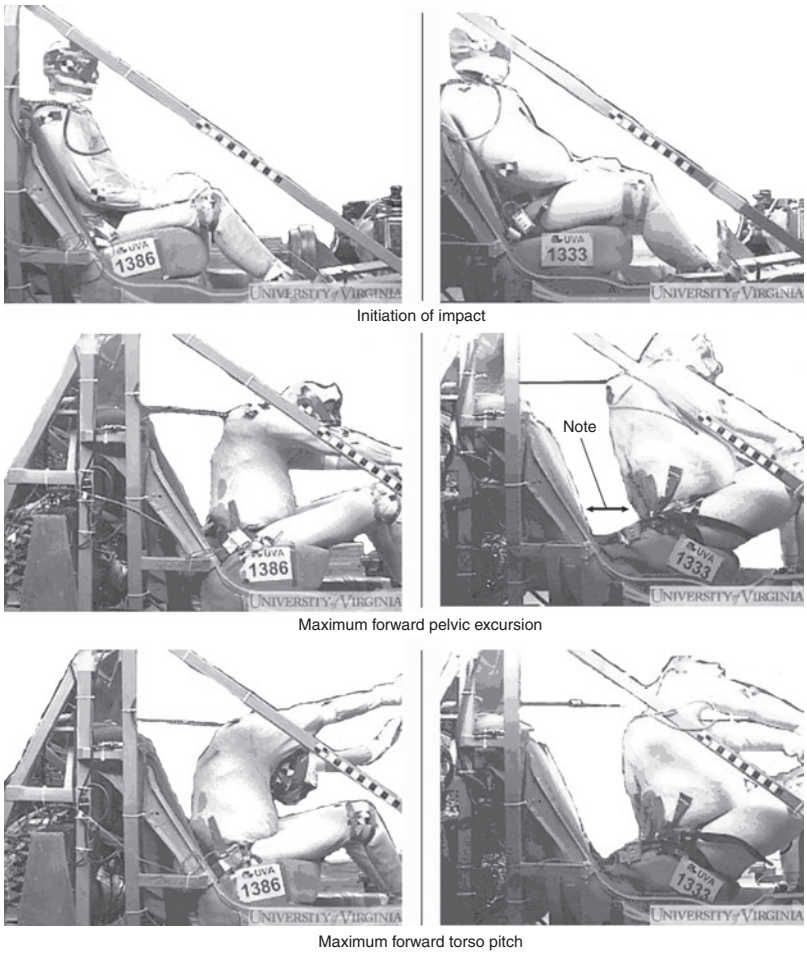

Figure 2 Kinematics of a nonobese (test 1386, left) and an obese (test 1333, right) subject. Note the significantly greater forward pelvic excursion for the obese subject, the resulting decreased forward pitch of the obese torso, and the consequent difference in head trajectory.

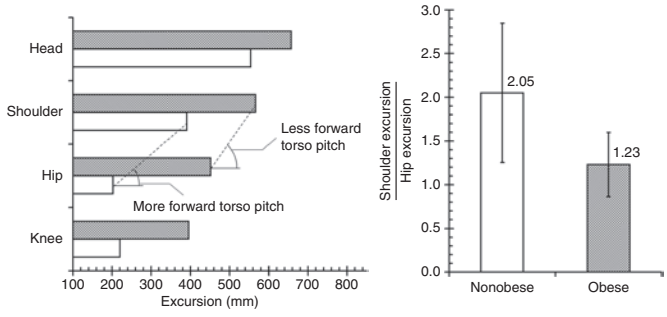

Figure 3 Head, shoulder, hip, and knee excursion of obese (gray bars) compared to nonobese (white bars) cadavers showing greater excursion for all body regions and decreased torso pitch reflected in a lesser ratio of shoulder excursion to hip excursion in the obese subjects.

upper ribs and clavicle. The hip motion of the obese cadavers in this study was similar to that associated with submarining, wherein the lap belt translates over the ilia and penetrates into the softer abdomen, thus allowing a large amount of pelvic excursion relative to the vehicle (22). The mechanism in these experiments was different, however, as the belt did not necessarily have to ride over the ilia in order for substantial hip excursion to occur. The depth of subcutaneous tissue between the lap belt and the anterior superior iliac spines was sufficient to allow the excessive hip motion without abdominal penetration by the lap belt. The consequences of submarining are not limited to abdominal trauma from lap belt loading. Large hip excursion leads to a reclined torso and results in an unfavorable biomechanical interaction with both the lap and shoulder portions of the seatbelt (23). Thus, there exists a mechanism for increased risk of injury to the obese subjects regardless of whether the belt translates over the iliac wings and into the abdomen. The lower extremities also experience increased excursion as a result of this hip excursion, and thus a mechanism for increased risk of a hard contact and resulting injury.

The results of this study indicate that the "fat as airbag" hypothesis advanced by Boulanger *et al.* (13) and Arbabi *et al.* (12) may not be a primary mechanism of reduced head injury risk for obese occupants who use a seatbelt. A more likely explanation is the overall unfavorable kinematics that result from increased hip excursion before the lap belt engages bony structures in an obese occupant, which leads to the hips and chest "leading" the head as the torso remains reclined throughout the impact. For unbelted occupants and other types of trauma, the "cushion effect" of fat should be studied further before it is accepted as an explanation for any retrospective field observations.

Table 2 Correlations between excursions and obesity

Parameter	Pearson correlation	*P*
Maximum head excursion	0.516	0.191
Maximum shoulder excursion	0.570	0.140
Maximum hip excursion	**0.922**	**0.001**
Maximum knee excursion	**0.790**	**0.020**
Head/hip excursion	**–0.763**	**0.027**
Shoulder/hip excursion	–0.556	0.152

Boldface values indicate $P < 0.05$

This study supports the concept of the injury model proposed by Viano *et al.* (16) in that the obese occupants by virtue of their greater mass and hence kinetic energy do require more work from the restraint system before their forward motion is arrested. This is another explanation for increased risk of thoracic and extremity injuries, though it would also suggest an increased risk of head and abdominal trauma. It is possible that the torso-reclined kinematic observed in this study is sufficiently protective of the head to offset the increased risk associated with the greater kinetic energy of an obese occupant. The cadaver study did not, however, identify any mechanism by which abdominal injury would be reduced by obesity. Additional research is necessary to determine whether obesity actually reduces the risk of abdominal injury and to identify the mechanism of any reduction.

Finally, it is important to realize that this study is limited by the use of cadavers as a model of the living human. Factors such as active and passive musculature, tissue lividity, and autolysis limit cadavers as a model of living humans for studying crash mechanics. Furthermore, crashes in the field are characterized by unquantifiable variability in factors such as initial position, seatbelt routing and fit, vehicle geometry, and crash mechanics. The test conditions considered here, while representative of a typical and common laboratory test configuration (48 km/h frontal impact), do not consider the wide array of conditions that lead to injury in the field. Detailed clinical data linked with crash reconstructions and biomechanical investigations of field cases, such as that performed under the US Department of Transportation's Crash Injury Research and Engineering Network program (http://www.nhtsa.dot.gov/portal/site/nhtsa/menuitem.1c5bf5af32c6dfd24ec86e10dba046a0/) may guide future laboratory studies of crash mechanics and injury mechanisms specific to the obese.

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DISCLOSURE

The authors declared no conflict of interest.

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