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New Jersey Medical School: UMDNJ Crash Injury Research & Engineering Network Center

Fatal Versus Potentially Survivable Motor Vehicle Crash (MVC) Aortic Injuries (AI): The Ratio of Deceleration Energy to Change in Velocity on Impact and the Presence of Associated Injuries as Determinants of Outcome

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Abstract

Objective: To examine the difference in force mechanisms between fatal and potentially survivable MVC aortic injuries (AI) compared to non-AI severe thoracic injuries (ST). **Methods:** Of 324 autopsied MVC driver or front seat passenger fatalities (1997-2000), there were 43 fatal AI (36 scene deaths, 7 hospital deaths) and 5 additional AI survivors. **Results:** Of the 48 AI, there was only a 42% survival for those reaching hospital alive. 80% of AI survivors had isthmus lesions and all had no or minimal brain injury (GCS \geq 13), no cardiac injury and only 20% ribs 1-4 fx or shock; of AI non-survivors reaching hospital alive,

67% had GCS \leq 12, 50% cardiac injury, 83% ribs 1-4 fx and 83% shock; AI scene deaths had 78% severe brain injury, 56% cardiac injury, 69% lung injury and 78% ribs 1-4 fx. Quantifying forces in AI scene mortality: the **Instantaneous Velocity on Impact** of the subject vehicle (delta V1) and the Impact Energy Dissipated (IE) on the subject vehicle (V1) in joules demonstrated a linear regression in fatal car MVC AIs: **Energy dissipated (joules) =**

$$-56.65 \times (\text{delta V1})^2 + 15972 \times \text{delta V1} - 454661, r^2 = 0.83.$$

However, for 27 patients with non-AI but severe thoracic (ST) injury (AIS \geq 3), the relationship of IE to delta V1 had a linear regression of Energy dissipated (joules) = $-5.0787 \times (\text{delta V1})^2 + 4282.1 \times \text{delta V1} - 57182.1$, $r^2 = 0.84$, with the slope difference between the regression for AI scene deaths and that of ST and AI survivors being significant

($p < 0.05$). Based on these relationships, a Critical Zone limited by MVC Impact Energy level of 336,000 joules and a delta V1 of 64 kph appears to be the limit of potential survivability in MVCs producing aortic injuries. All AI above these thresholds died. In contrast, ST had greater use of seatbelts (AI 10% vs all ST 60%) and airbags (AI 50% vs all ST 72%), and an 83% survival. **Conclusion:** The data suggest different mechanisms of force delivery and injury patterns in fatal vs potentially survivable AI, and vs ST MVCs. They suggest that an approach to improving vehicle safety measures for AI may involve better safety devices and mechanisms for reducing that fraction of Impact Energy dissipated on V1 for a given delta V1 which is focused on the upper portion of the subject's thoracic cage between the levels of ribs 1-8.

Introduction

A variety of mechanisms have been proposed as determinants of motor vehicle crash induced aortic injuries. These include shearing stresses secondary to differential decelerations of the aorta and the thorax at the time of impact [Zehnder, 1956; Sevitt, 1977; Feczko, Lynch, Pless *et al*,



1992; Shkrum, McClafferty, Green *et al*, 1999] as a function of the direction of the motor vehicle crash, frontal versus lateral [Ben-Menachem, 1993; Katyal, McLellan, Brenneman *et al*, 1997; Careme, 1989] chest wall compression in a cephalic direction with a “shoveling effect” on the heart and proximal aorta with tethering at the aortic isthmus by the ligamentum arteriosum in frontal crashes [Voigt, Wilfert, 1969] and a similar compression of the chest wall with lateral movement of the more mobile heart relative to the fixed proximal descending aorta in lateral MVCs [Viano, 1983; Nahum, Kroell, Schneider, 1971]; an “osseous pinch” effect whereby the proximal descending aorta is caught between the sternum or upper ribs and the vertebral bodies [Crass, Cohen, Motta *et al*, 1990]; and an intravascular “water hammer” effect due to a sudden rise in intravascular arterial pressure when the external compression effect occurs at a critical point in the cardiac ejection cycle, thus bursting the aorta at its weakest point [Lundevall, 1964; Lasky, Nahum, Siegel, 1969; Saylam, Melo, Ahmad *et al*, 1980]. In addition to these theories as to causation, a major component of the literature has focused on the differences in mechanisms between aortic disruption survivors and deaths in the group of patients who reach hospital alive [Turney, Attar, Ayella *et al*, 1976; Fabian, Richardson, Croce *et al*, 1997]. Conversely, a number of important studies of aortic injury have confined themselves solely to autopsy series of scene-fatal medical examiner cases [Shkrum *et al*, 1999; Ben-Menachem, 1993; Dischinger, Cowley, Shankar *et al*, 1988; Williams, Graff, Uku *et al*, 1994].

While all of these theories have some merit, very few studies have combined their clinical or post mortem patient observations with professional crash reconstruction-derived data regarding the mechanism and direction of the MVC, the calculation of the impact deceleration velocity ($\Delta V1$) and the estimated impact energy (IE) dissipated upon the patient's vehicle (V1), and the identification of the passenger compartment structure responsible for the delivery of these physical factors to the patient's thorax and the modulating or contributing factors induced by seat belts and/or airbag deployment. Moreover, it is essential to identify the nature and severity of any other associated injuries and their physiological consequences, which may convert a potentially survivable aortic injury into a fatal one, regardless of the timing and proficiency of the aortic reconstruction surgery. Finally, there is a need to examine the entire demographic distribution of scene fatal versus potentially survivable aortic injuries within a given geographic region, since the qualitative mechanisms and quantitative forces may be quite different in these two groups, or within subgroups, of these patients. The present study was designed to consider all of these factors by obtaining detailed crash reconstruction compared to anatomic/physiologic data on a specific group of aortic injury patients, front seat drivers and

passengers who remained in the vehicle after the MVC, to include all aortic injury patients encountered during a continuous time period, hospital survivors, hospital deaths and medical examiner scene fatalities.

Methods

All cases of fatal aortic injury (AI) occurring between 1997 and 2000 in either drivers or front seat passengers of cars, sport utility vehicles or light pickup trucks in the three county area (Essex, Passaic, & Hudson) which falls under the administrative authority of the Regional Medical Examiners Office (ME) of Newark, New Jersey, and those patients surviving their AI injury long enough to be admitted to the Level I Trauma Center at the New Jersey Medical School University Hospital, also located at Newark, were included in this study. All of the fatal AI cases were autopsied under the authority of the ME and the cases surviving to reach hospital who were treated by surgical therapy had clinical, radiological and operative confirmation of the nature, location and outcome of their AI, as well as diagnosis and appropriate therapy for any associated anatomic or physiologic injuries. Thus, there were three categories of AI: Scene Deaths autopsied by the Medical Examiner (ME), Hospital Deaths (H), and Hospital Survivors (S). The pathologic anatomic findings of all injuries were documented by direct contemporaneous observation (autopsy or surgical/radiological procedures), the physiologic consequences of all injuries in patients surviving long enough to reach hospital alive were directly observed (shock, Glasgow Coma Scale, evidence of organ dysfunction and length of hospital and ICU stay) and documented, as was the final outcome of the patient's AI (survival or death). In all cases, crash scene reconstruction was done by examination of police and EMS reports, as well as by direct questioning by the study EMS Coordinator of the EMS personnel involved in transporting the hospital directed cases. In the Scene Deaths (ME cases), no vehicle could be moved, nor could the corpse be removed until the ME's office photographer had documented the exact location of the vehicles and the position of the bodies.

Following the identification of each crash which met the criteria of the study, with the assistance of the study's EMS Coordinator, the study Crash Investigation Team located the vehicle (V1) of the AI victim and the crashing vehicle(s) (V2, V3, etc.), or the fixed object impacted by V1. The team made detailed measurements of the vehicle deformities and identified the location of the occupant contact sites within the passenger compartment, which had been made by the subject at the time of the crash. The deployment of any frontal airbags was noted, as was the use of any type of seatbelt restraints. None of these vehicles were equipped with side airbags or air curtains. From these measurements, wherever possible the change in velocity on impact ($\Delta V1$) and the deceleration energy dissipated on impact (IE) were estimated using the National Highway

Traffic Safety WINSMASH 1.2.1 program.

In addition, for purposes of comparison with those AI patients reaching hospital alive, a group of patients who sustained severe thoracic trauma of AIS 3 or greater (mean AIS for most severe thoracic injury=3.6) (ST), similar in all respects save AI, were studied in the identical manner with regard to injury identification, hospital course and crash and vehicle reconstruction with delta V1 and IE also being calculated from the WINSMASH program supplied by NHTSA. In all patients reaching hospital alive, an informed consent to record the patient data and to examine the vehicle approved by the UMDNJ: IRB was obtained from the patient or next of kin. In the Scene Deaths (ME), the autopsy findings and crash reconstruction were obtained under the legal authority of the Medical Examiner. However, in all cases (ME, H, & S), all patient and vehicle identifiers, protected by a Certificate of Confidentiality, were removed and each sanitized case was identified only by a randomly selected case identification number. These sanitized data were entered into a computer generated relational data base and the data interrelationships were analyzed for patterns and significance with ANOVA and regression techniques by the use of a standard statistical program (SAS). A value of $p < 0.05$ was used as the minimum basis for significance.

Results

As examples of the CIREN methodology two representative cases are presented. The first is an example of a proximal aortic rupture in a front seat passenger produced by a frontal motor vehicle crash (MVC) impact of a sedan into a telephone pole and the second is an example of a descending aortic laceration sustained by the driver of a sedan also in a frontal MVC with a delivery truck. Both subjects were immediate deaths and consequently were autopsied immediately after the MVC by the Regional Medical Examiner.

In the first case, the patient was a 73 year old male who was the unrestrained front seat passenger in a 1997 Mercury Sable (1676 kg). He weighed 90kg (198lbs) and was 5ft

11 inches (180cm) in height. As shown in the scene diagram (Figure 1), the driver lost control of the vehicle and it impacted a telephone pole at the side of the road on the driver's side with a primary direction of force (PDOF) of 350o, and then rotated in a counter clockwise direction.

The Delta V was 73kph (41mph) and the maximum crush was 81cm at C2 (Figure 2). The passenger airbag deployed on impact, but because the passenger was unrestrained and the primary impact was on the driver's side at 350o PDOF, the patient missed the airbag and impacted his anterior left chest wall and sternum on the left side of the central instrument panel (Figure 3).

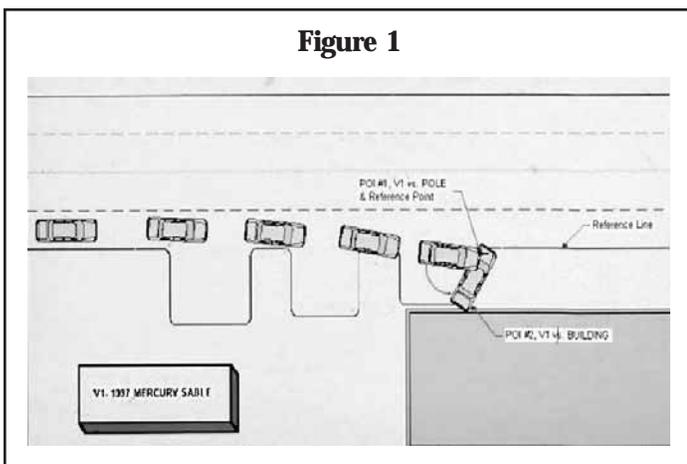
Figure 2



Figure 3



Figure 1



As a result of the impact, the patient sustained a bursting type of rupture of the proximal aorta on the anterior aspect just above the aortic valve (Figure 4). On inspection, after opening the aorta and the left ventricular chamber (Figure 5), it can be seen that the transverse rupture lies just above the aortic valve ring and the coronary artery cusps close to the right coronary artery orifice in the aorta. This type of lesion is unfortunately always fatal, since the high-pressure

stream of blood ejected from the left ventricle immediately enters the pericardium with acute pericardial tamponade and if there is also a rent in the pericardium, rapidly fills the thoracic cavity with the entire cardiac output with consequent acute hypovolemic shock and subsequent cardiac arrest.



Figure 4

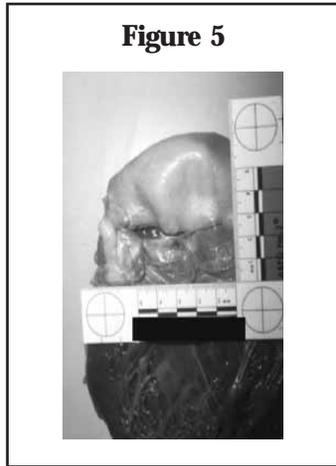


Figure 5

The second case example, which demonstrates a shearing type of rupture of the descending aorta, was a 51 year old female, 62 kg (135 lbs), 5ft 4 inch (163cm) unrestrained driver of a 1985 Ford Thunderbird (1505 kg) who apparently lost control of her vehicle at a curve in a four lane

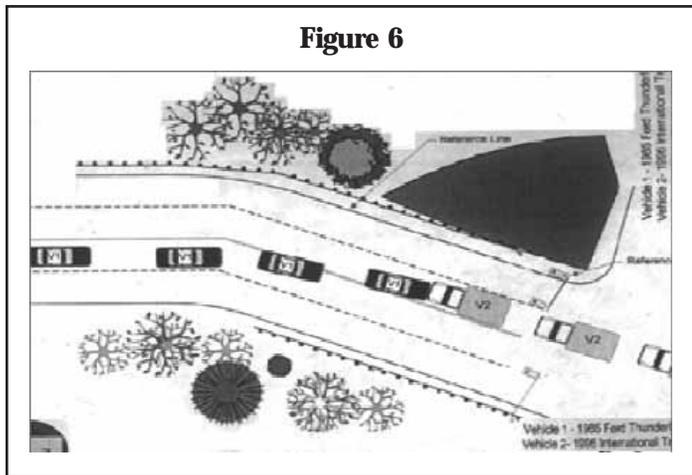


Figure 6

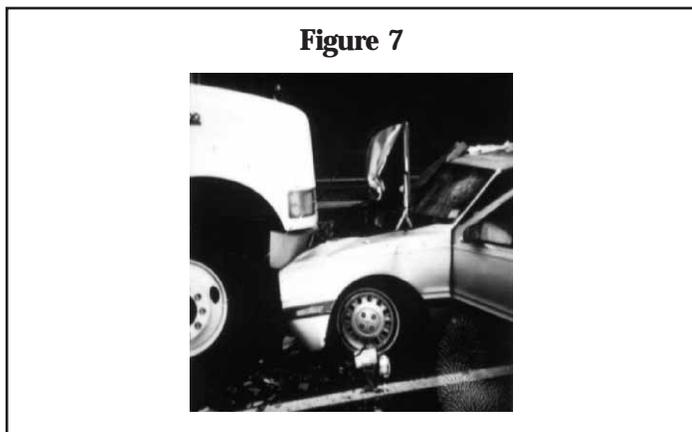


Figure 7

undivided roadway and crossed the mid-line to sustain a frontal impact with a 1996 International CBE/490 delivery truck (14923 kg) (Figure 6 and Figure 7).

The impact occurred at a Delta V of 60 kph (33 mph) with a maximum crush of 76cm at C6. There was a major degree of bumper override of the delivery truck with the sedan, so that the engine was driven back toward the passenger compartment with intrusion of the steering wheel into the driver's space (Figure 8).

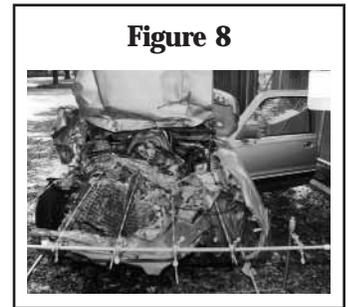


Figure 8

As there was no airbag to deploy and the patient was unrestrained, she impacted with the steering wheel hub with her left anterior chest wall (Figure 9). The force of this unbuffered impact produced fractures of the first through the fifth ribs on the left, just to the left of the costal-sternal junction (Figure 10).

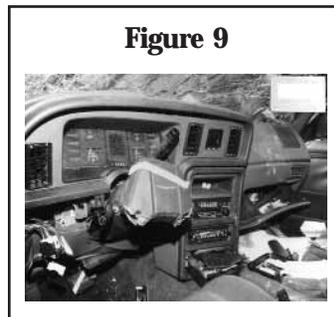


Figure 9

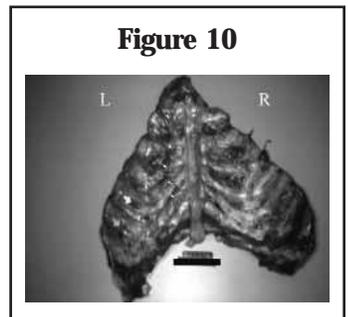


Figure 10

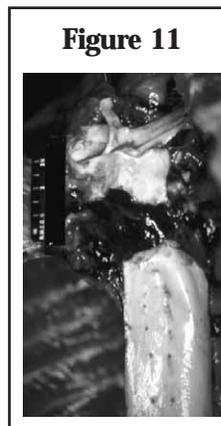


Figure 11

This impact produced a shearing type of complete transverse laceration of the aorta in the freely mobile isthmus region below the take-off of the left subclavian artery and just proximal to the aortic orifices of the third intercostals arteries which fix the descending aorta to the vertebral column. The leading edge of the disruption appeared to be at the point of fixation of the aorta to the left pulmonary artery by the ligamentum arteriosum.

This rupture was not contained by the posterior mediastinal tissues, and consequently the entire cardiac output was ejected into the left thoracic cavity, with immediate death of the patient at the scene of the MVC (Figure 11).

Demographics of Aortic Injury

Of 324 autopsied MVC drivers and front-seat passenger fatalities (1997-2000), there were 43 fatal aortic injuries, or 13% of these fatalities (Table 1).

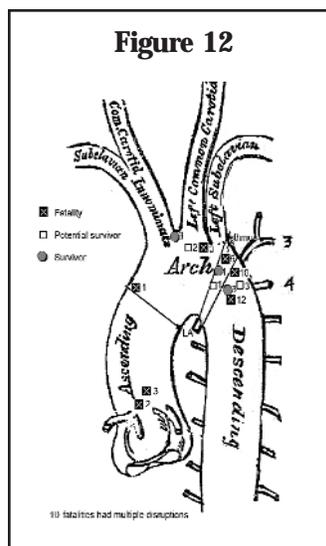
Total aorta injury cases:	48
Dead at scene:	36 (75%)
Potentially survivable:	12 (25%)
Hospital deaths:	7 (15%)
Actual survivors:	5 (10%)

Of the fatal AI, 36 were scene deaths (ME) and 7 were hospital deaths (H). In addition there were 5 AI who were hospital discharge survivors (S). Thus, AI represented 13.3% of all fatally injured front seat MVC occupants. Moreover, it is discouraging to note that 75% of all AI patients were dead at the scene of the crash; only 25% were sufficiently vital to be able to reach the hospital alive in spite of brief urban transport times, and of these potential survivors less than half, 42%, (but representing only 10% of the total MVC induced aortic injuries) actually left the hospital alive.

With regard to the mechanism of the MVC induced AI, of the 48 cases, two cases were in so massive an MVC that the exact direction of the crash could not be assigned with certainty. Of the remaining 46, 74% were caused by Frontal (F) MVCs and 26% by Lateral (L) MVCs. Seatbelt restraints were used by only 12% of the Frontal crash AI and 36% of the Lateral crash AI patients. Airbag deployment occurred in 50% of the Frontal crashes, but surprisingly, in 36% of the Lateral crashes the airbag actually deployed, either due to the sudden arrest of the subject vehicle's forward motion, or due to the massive deformity of the subject vehicle consequent to the force of the Lateral impact. As a result, 74% of the Frontal-MVC and 82% of the Lateral MVC were ME cases. Neither seatbelt use nor airbag deployment appeared to influence the final outcome in patients with AI. However, when compared to a group of 27 crash study patients admitted to the hospital with similar severe thoracic injuries (ST), but without AI, there was a significant difference ($p < 0.05$) with respect to a greater use of seatbelts (AI 10% vs ST 60%) and a higher incidence of airbag deployment (AI 50% vs ST 72%) in the non AI thoracic injury patients. Possibly as a result of the use and/or deployment of these protective devices, there was a significantly ($p < 0.05$) higher survival rate in the ST patients (83%) compared to the potentially survivable AI patients, where only 42% of the AI patients who reached the hospital while still viable actually left the hospital alive.

Location of Aortic Injuries

The sites of the primary aortic injuries is shown in Figure 12, with the majority of the patients who reached the hos-



pital alive and the actual hospital survivors having their main lesion in the isthmus region or the descending thoracic aorta.

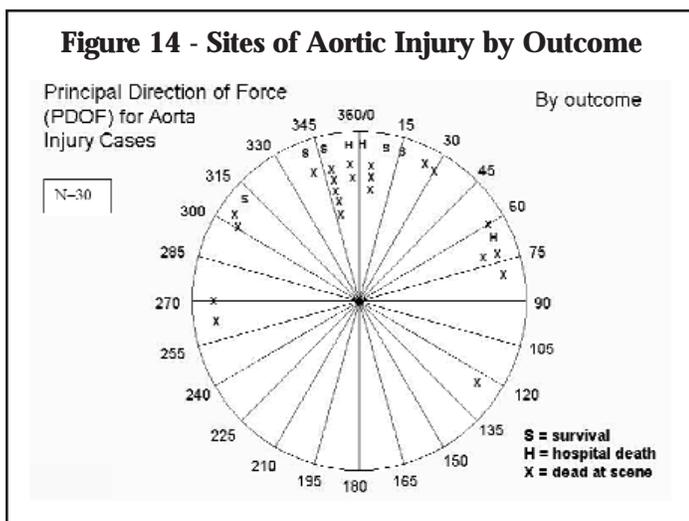
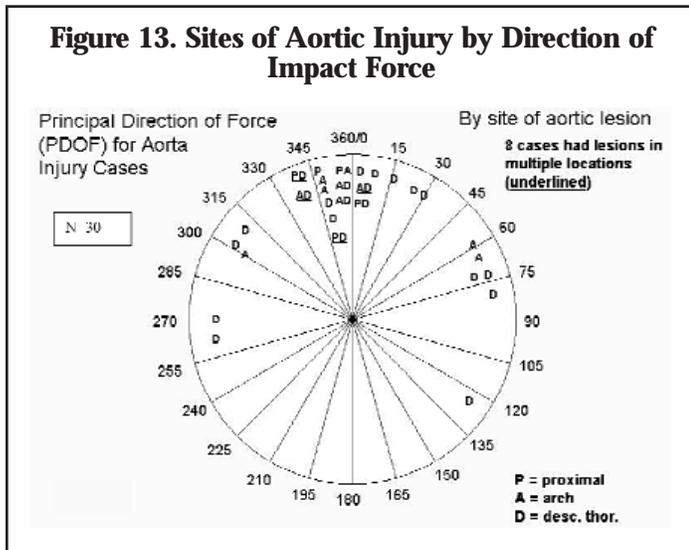
However, there were frequently multiple areas of aortic injury, some of which did not result in a complete disruption of the aorta, but were limited to the intima and the media of the aortic wall.

(24% had multiple lesions)	Survival	Hospital deaths	Dead at scene
N	5	7	36
Proximal aorta injury	1 (2%)*	0	6 (13%)
Aortic arch injury	1 (2%)	3 (6%)	12 (25%)
Descending aorta injury	4 (8%)	4 (8%)	29 (60%)

The site of the principal aortic injury appeared to play a major role in the type of outcome. As shown in Table 2, while 13% of the AI (all were ME cases) had a major proximal aortic injury, only one* of the hospital survivor AI cases in this series sustained such a lesion and it was only a small non-ruptured bulge with the primary pseudoaneurysm occurring at the aortic isthmus. Of the total AI, 33% had an injury to the aortic arch, only one of whom survived (2%), but of those with an aortic arch injury 25% were scene death ME cases. However, 76% of the AI had an aortic injury at the aortic isthmus, or just below the ligamentum arteriosum in the proximal descending aorta, but of these 37 cases, only 4 actually left the hospital alive. There were 29 ME cases, representing 60% of the total AI patients, who had their lesion in a similar location. In regard to the sites of AI, it is important to emphasize that in 24% of all the cases there were multiple sites including various combinations of proximal, arch and isthmus lesions.

In addition to the general direction of the MVC (Frontal vs Lateral), the Principal Direction of Force (PDOF) appeared to play some important role in the mechanism and location of the aortic injury. In 30 of the cases, a precise PDOF could be related to the site (or sites) of the aortic injury.

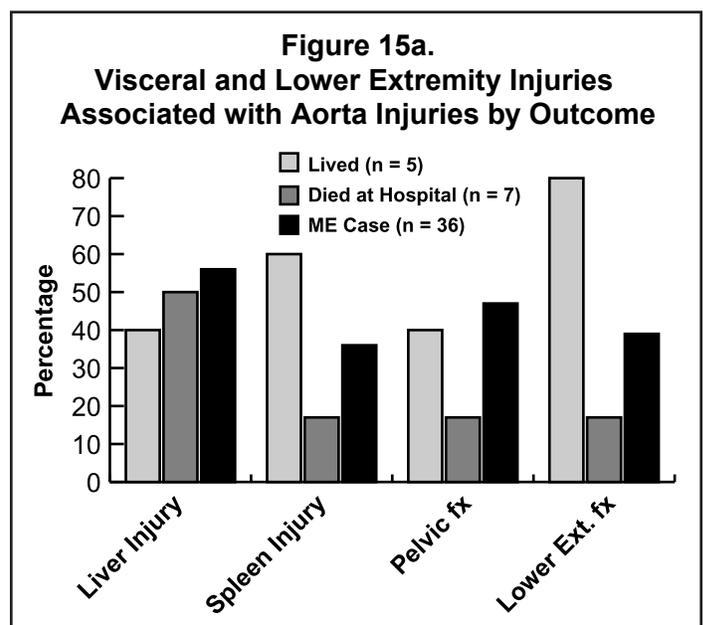
These data are shown in Figure 13 where it can be seen that all of the proximal aortic (P) and most of the aortic arch injuries (A) occurred in frontal MVCs in which the PDOF was between 0 and 30 degrees to the right, or between 360 and 330 degrees to the left of the vehicle's front center point (0/360 degrees). Moreover, all of those cases in which there were multiple sites (PA, PD, AD)



resulted from impacts within this range of PDOFs. However, while 3 of the 4 Frontal MVC hospital survivors (S) had their major lesion in the isthmus or proximal descending aorta (Figure 14), one also had a small contained lesion of the proximal aorta. The only Frontal MVC AI survivor who did not have an isthmus or proximal descending aortic lesion had a small pseudoaneurysm contained within the adventitia of the aortic arch between the innominate artery and the left carotid artery. The majority of the patients whose AI resulted from a Lateral MVC had their lesion in the isthmus or descending aorta. There was no proximal AI in this group.

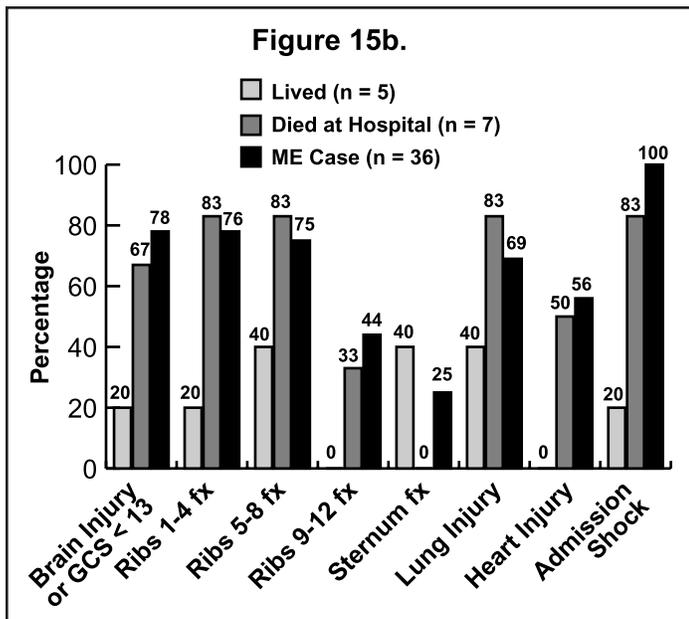
In weighing the major factors which contributed to the final outcome of those patients with AI who did not instantly exsanguinate from their injury, there are two critical relationships which must be considered. The first, although not necessarily the most important variable, is age. While the incidence of aortic injury was greatest among the younger age groups, with a peak in the 26 to 35 year range (13 cases), there were cases seen steadily even in the 96 to 100 year age range and surprisingly, there were survivors even in the aged population. Not unexpectedly, the ratio of male to female AI patients was slightly more than 3:1, but in this small population the only potential survivors in the over 65-year group were male.

The second and undoubtedly most important factor affecting the final outcome in the AI patient is the incidence and severity of the associated injuries and their consequent physiologic complications; namely circulatory shock and severe brain injury as measured by their admission Glasgow Coma Scale (GCS) score. These data are shown in Figures 15A and 15B. Figure 15A demonstrates the incidence of major visceral (liver and spleen) and non-thoracic fracture injuries (pelvic and lower extremity fractures) in each of the three groups: hospital survivors (S), hospital deaths (H), and scene fatalities (ME). It can be seen that while the incidence of hepatic injury rose progressively from S (40%) through H (50%) to ME (59%), it was the hospital survivor group who had the highest incidence of splenic injuries (60%) and lower extremity injuries (80%) compared to the other two groups. Indeed, even in the ME cases, injuries to the spleen were only found in 35% and those to the lower extremities were only found in 38% of the cases. Pelvic fractures in the S and ME groups were of similar incidence (40% and 47% respectively) and both were higher than the incidence seen in the H group.



However, perhaps the most revealing differences in the pattern of injuries which separated the hospital survivor AI patients (S) from the ME and H patients is seen in Figure 15B. Here it can be noticed that the pattern of incidence of the most critical associated injuries was vastly different in the S patients compared to both the hospital death (H) and the scene death (ME) groups. The incidence of associated severe intrathoracic injuries to lung (40%) in S was substantially less than that seen in H (83%) or ME (68%) and there was no evidence of cardiac injury in S, compared to a 50% H and 56% ME incidence, respectively. As a result of this vastly reduced incidence of associated injuries to other vital organs or major skeletal systems, there was a marked decrease in the consequent state of physiologic shock in S patients (20%) compared to 83% in the hospital deaths (H), and of course all of the ME cases by definition had passed beyond shock into final irreversible physiologic collapse.

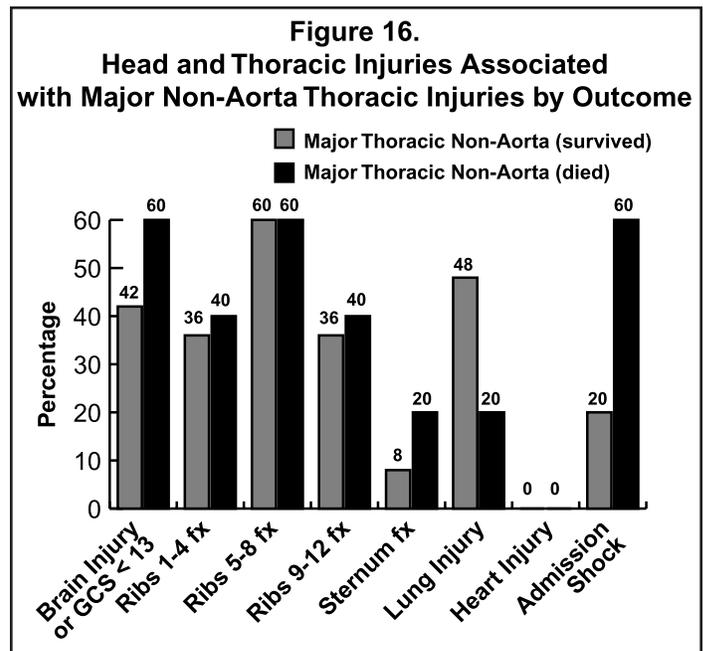
A perhaps equally important difference in the incidence of physiologic injury was seen with regard to the brain (Figure 15B). Again, only 20% of the hospital survivors (S) had a brain injury severe enough to produce a Glasgow Coma Scale (GCS) score of less than 13, while 67% of the H patients reached this level. In the ME cases, autopsy findings showed that there was a similar incidence (76%) of pathologic evidence of significant brain injury (epidural, subdural, or intraparenchymal hemorrhage, as well as brain laceration, or contusion) which would have been expected to have produced at least a similar level of brain deterioration as that represented by a GCS of <13.



Of considerable interest with regard to the site and outcome of the AI was the incidence of rib fractures at different levels of the chest wall. As also shown in figure 15B, while only 20% of the AI survivors (S) had rib fractures involving ribs 1-4, 83% of the H patients and 76% of the

ME cases had fractures at this level. Indeed the one patient in the S group with rib fractures at this level was the one with a small contained rupture of the aortic arch between the innominate and right carotid artery. Also, S patients generally had a reduced incidence of rib 5-8 fractures (40%) compared to those found in the H (83%) and ME (74%) groups and no S patient among the AI survivors had a fracture involving ribs 9-12. In contrast, the AI survivors sustained a greater incidence of sternal fractures than the other two groups, but had no cardiac injuries.

Indeed as shown in Figure 16, which documents the pattern of injuries seen in the 27 patients with severe thoracic trauma not involving an aortic injury (ST), the general pattern of thoracic organ, skeletal and rib injuries is similar to the AI survivor (S) group, especially with respect to the incidence of rib 1-4 fractures and lung injuries, and the total absence of cardiac injuries. These data suggest that the main force of the MVC impact injury in the AI hospital-survivor group is delivered at a lower level on the body generally sparing the head and upper thorax, but causing a greater incidence of splenic and lower extremity injuries. The S patients also had an incidence of pelvic fractures which was greater than that found in the H patients, although it approached that seen in the ME cases.



However, both the H and ME patients generally had much more severe multitrauma than the S, as evidenced by the far greater incidence of admission shock found in H (83% vs 20% in S) and of course the 100% loss of all circulatory and cardiorespiratory function in the scene deaths (ME). Also, while the pattern of injuries in surviving AI patients might suggest a greater likelihood that these were sustained in a Lateral MVC, as seen in Figure 14, four of the five AI hospital survivors (S) had impacts delivered in the Frontal

direction with PDOFs between 330 degrees to the left and 30 degrees to the right of the 0 degree front-center point of the vehicle.

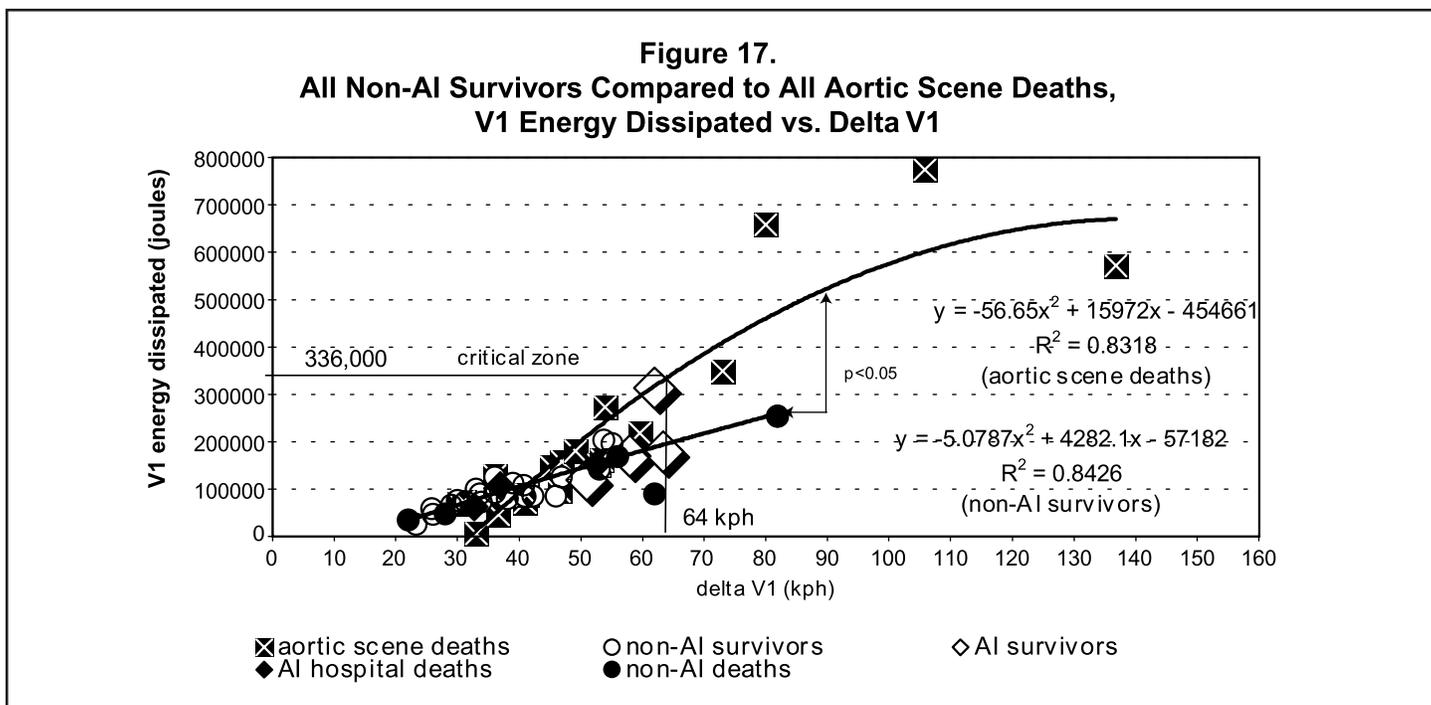
The major difference between Frontal and Lateral MVCs responsible for the production of the resulting AI is the component of the passenger compartment which becomes the specific thoracic contact/intrusion site producing the AI. The incidence of these sites as the cause of the aortic disruption(s) is shown in Table 3. In 5 of the ME MVCs (4 Frontal and 1 Lateral) the impact was so catastrophic that no specific site could be identified and in 3 Frontal MVCs the identification was equivocal. However, in those Frontal MVCs where a contact/intrusion site could be identified with security, it was determined that in 53% of the cases the steering wheel assembly was responsible, in 15% of cases the AI was produced by anterior chest contact with an expanding airbag cover due to the patient's too close proximity to the SRS, and in 12% of cases there was thoracic contact with the instrument panel. In contrast, in the

Lateral MVCs in which an AI occurred, all of the identified contacts productive of an AI came from crash-induced intrusions of the side-door panel and/or the B-pillar.

These data concerning the differing causes, sites and severities of AI, and the incidences of multiple AI lesions, as well as the different patterns of associated injuries found in the hospital survivors, compared to the in-hospital and scene deaths, suggests that a different magnitude of forces may have played a part in the creation of this lesion in these groups. To attempt to examine this question the calculated instantaneous deceleration on impact (delta V1) of the subject vehicle (V1) was evaluated with respect to the estimated deceleration energy (IE) imparted to V1 on impact, as computed from the WINSMASH program based on the direction of the MVC, the relative masses of the struck and striking vehicles and the magnitude of the V1 vehicle deformity and its stiffness characteristics. The data from all the ME scene deaths (including AI secondary to car crashes with other cars, sport utility vehicles, trucks, or fixed objects) was used to develop a linear regression which was then compared with a similar regression computed from the data of surviving front seat drivers or passengers with severe thoracic trauma (ST-survivors) who did not have an AI. These regressions, together with the data points from the AI hospital survivors and the in-hospital AI and ST deaths are shown in Figure 17.

Here it can be seen that each of the two regressions (ST-survivors and ME-deaths) has a coefficient of determination (R2), which explains more than 80% of the variability in the data. More important, these two regressions have significantly different slopes (p<0.05), with the deceleration impact energy (IE) rising to a greater extent per unit

	Catastrophic	Steering wheel	Air bag	Instrument panel	Side door/B pillar	unknown
Frontal (34)	4 (12%)	18 (53%)	5 (15%)	4 (12%)		3 (9%)
Lateral (11) (6 right, 5 left)	1 (9%)				10 (91%)	



increase in delta V1 in the ME aortic disruption cases than in the surviving severe thoracic trauma patients (ST-survivors) who did not sustain an AI. When the data points for the potential and actual survivors of AI (H&S) are plotted on this graph, it can be seen that, with one exception, they tend to lie on, or close to, the mean regression slope for the ST survivors. This relationship suggests that, in addition to the direction of the crash, the age of the patient, and the anatomic level of the primary impact point on the patient's body, there is a critical threshold of impact energy dissipation (IE) for a given level of delta V1 which cannot be exceeded without making the incidence of AI more likely. Also to be noted is that there appears to be a Critical Zone, delimited by a level of delta V1 of 64KPH and an IE of 360,000 joules, above which the likelihood of a fatal AI secondary to a thoracic impact at MVC becomes extremely high, regardless of the direction of the crash. All AI in this series who lay above these thresholds died at the scene of the MVC. Nevertheless, even within this Critical Zone, when compared to all the ST cases (survivors and deaths), the potentially survivable AI patients, i.e., (S&H) those who reached hospital alive, had more severe brain injury (S&H 45% vs ST 17%), more fractures of ribs 1-4 (S&H 55% vs ST 27%), more injuries of heart (S&H 20% vs ST 0%) and lung (S&H 64% vs ST 43%), a higher incidence of shock (S&H 55% vs ST 27%) and a lower survival (S&H 42% vs ST 83%). However, this pattern of better outcomes even within the delta V1/IE Critical Zone also may have been facilitated by the much higher use of seatbelts (S&H 10% vs ST 60%) and the greater incidence of airbag deployment (S&H 50% vs ST 72%) in the thoracic trauma patients who did not sustain an AI, compared to the potentially survivable AI patients.

Discussion

As noted by Parmley et al (1986), the occurrence of a disruption of the thoracic aorta as the consequence of blunt trauma to the thorax was first described by Andreas Vesalius in 1557. However, it was considered a rare catastrophe until the modern era of mechanized transportation. Indeed, even as a result of domestic motor vehicle collisions, it was demonstrated to be a disease of small incidence in studies of fatal MVCs done during the Great Depression until the beginning of World War II, when the autopsy series of 7000 motor vehicle crashes done by Strassman (1947) showed an incidence of AI of only 0.73%. However, as America's love affair with cars and speed became more passionate in the quarter century of affluence beginning after 1950 which solidified the American Century, studies of fatal MVCs by Greendyke (1966) and by Sutorius, Schreiber and Helmsworth (1973) showed that the incidence of AI in fatal crashes had risen to between 10 to 15%. Similar autopsy studies carried out between 1984 - 1988 by Feczko et al (1992) and between 1980 - 1985 by Williams et al (1994) in mid-sized cities surrounded by

largely rural areas showed a similar incidence, 12% and 17% respectively. In contrast, Dischinger et al's (1988) observations of autopsied victims in a highly urbanized state traversed by many high speed highways demonstrated an incidence of 27% AI in MVC drivers and 19% in passengers for an overall occurrence rate of 26% of MVC fatalities. These horrifying statistics, it must be emphasized, represented an era where seatbelt use was minimal and no airbag technology had been introduced into the increasingly overpowered US automotive fleet.

Nevertheless, not dissimilar findings were seen in the 1991-1995 Canadian study carried out in metropolitan Toronto by Katyal et al (1997), where seatbelt use was legally mandatory and used by 70% of their cases. In spite of this high use of restraints, AI was found in 21% of the autopsied fatalities. However, the absence of airbag deployment was indicated as a factor by these authors. Nevertheless, they introduced an important observation into the demographics of this injury; namely, they also included the 16 additional AI cases that survived to reach hospital alive, so that the true incidence of aortic disruption in their area during this period could be ascertained. Thus, there were 97 cases of AI, with a scene mortality rate of 83%. Although, the authors did no detailed crash reconstruction, they did identify the direction of the crash and the site of the primary point of impact on the subject vehicle. In this study, 49.5% of the AI resulted from a lateral impact, 50.5% were induced by a frontal MVC and in 94% of all the AI cases at least one of the aortic injuries lay in the peri-isthmic region.

It is of interest to compare these data obtained in time periods when an airbag SRS was not included as a feature of the automotive safety package, with the AI and ST patient data from the present study. In contrast to the Canadian study, done at a time when none of their AI cases appears to have had airbag protection, the present group of AI patients had airbag deployment in 50% of the Frontal MVC cases. 74% of all these cases were frontal in character. While it is not clear whether airbag deployment played a statistically significant role in preventing AI, it is of interest to note that in the Toronto study [Katyal et al, 1997] the overall incidence of AI averaged 24 cases per year. However, in the present study the average incidence was only 16 cases per year and the scene death rate was reduced to 75%, compared to 83% in the Canadian series. As noted earlier, the incidence of fatal AI in this study was 13%, compared to 21% in the Toronto study. Thus, both the occurrence of AI and the incidence of ME cases appear to be lower after the introduction of airbag technology into at least part of the car fleet than was found in the most immediately prior series [Katyal et al, 1997] analyzed before this safety device was introduced into the car fleet to any great extent.

Moreover, when the present group of AI cases is compared to a contemporaneous group of patients who sustained similar severe thoracic injuries (ST) without AI, it can be seen that the incidence of both airbag deployment and seatbelt usage were substantially greater in the ST group. Also, the data presented in Figure 3B demonstrate that those AI patients who were hospital survivors had a significantly lower incidence of moderate to severe brain injury (as defined by autopsy findings, or by clinical evidence of a GCS <13) than either the hospital deaths (H), or the ME cases. Thus, since previous studies [Loo, Siegel, Dischinger *et al*, 1996; Siegel, Loo, Dischinger *et al*, 2001] have demonstrated that airbag deployment in Frontal MVCs provides a statistically significant reduction in the functional severity of MVC induced traumatic brain injury, it seems probable that in those cases where the AI is contained within the periaortic tissues long enough to allow corrective surgical therapy, the airbag protective effect with respect to the brain may be a critical factor in increasing the potential for ultimate survival. In this regard, it is also of interest to note that four of the five hospital survivors sustained their AI in a frontal (PDOF 330 to 0 to 30 degrees) MVC, and all were airbag protected.

The importance of other associated visceral injuries seen in this series in determining the ultimate outcome in AI patients who survive the MVC long enough to reach the hospital alive is consistent with the data found in the 1993-1996 multi-institutional prospective study of blunt aortic injury patients who survived long enough to be admitted to a trauma center [Fabian *et al*, 1997]. While that 50 center study by its definition excluded ME cases, and was not stratified with respect to AI patients with or without other major visceral injuries, it can be seen that there was a similar high incidence of brain, liver, and lung injuries, as well as a 46% incidence of multiple rib fractures, and that the non AI causes of death were most heavily weighted to brain injury, Multiple Organ Failure, and Acute Respiratory Distress Syndrome, all of which would appear related to these identified organ injuries.

While a great deal of speculation has been made about the mechanisms involved in aortic transection injuries, it has been difficult to reproduce this injury in human cadaveric models [Eppinger, 1978; Bass, Darvish, Bush *et al*, 2001]. However, most investigators agree that a differential rate of deceleration between the fixed and mobile portions of the aorta produces a point of stress which is maximal at the isthmus. At this portion of the aorta, the arch of the aorta tethered by the major aortic arch vessels proximally is briefly free, until fixed by the third and subsequent distal intercostal arteries which arise directly from the descending aorta, and by the ligamentum arteriosum which connects the isthmus region to the left pulmonary artery. Indeed, in many of the cases of descending AI autopsied in this study, the tear appeared to begin at the ligamentum and to extend

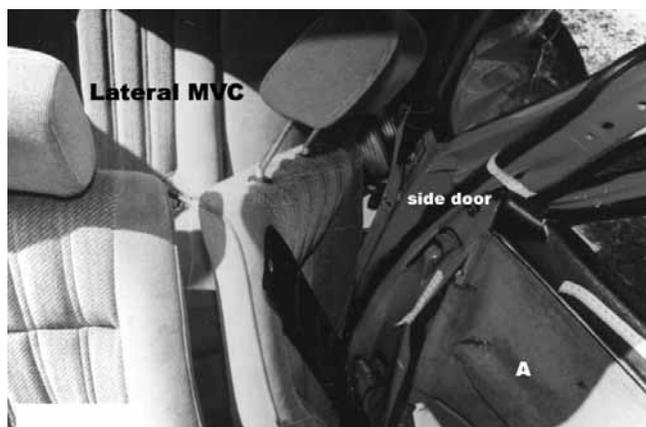
posteriorly in a circumferential manner leaving only a small bridge of intact aorta connecting the proximal and distal segments just above the intra-aortic orifices of the third intercostal arteries.

Obviously, somewhat different mechanisms have been proposed for AI secondary to frontal as opposed to lateral MVCs. Wilfert and Voigt (1971) have proposed a “shoveling” effect whereby the patient’s contact with the lower portion of the steering wheel rim forces the intrathoracic cardiac and proximal aortic structures upward with a fulcrum at the isthmus. This may account for some of the distal AI injuries seen here which resulted from frontal crashes, but as seen in Figure 14 all of the proximal AI occurred in Frontal MVCs, which suggests a powerful direct anterior-posterior compression producing a bursting force. This mechanism is made more probable by the observation that in 15% of the Frontal MVCs, the AI was due to an anterior chest contact with a fully powered exploding airbag cover and in 12% of Frontal MVC cases the AI followed the chest contact of an unrestrained passenger with the instrument panel. A number of experimental studies in animal models [Moffat, Roberts, Berkas, 1966; Viano, Haut, Golocovsky *et al*, 1978] and using human cadaveric aortas [Bass *et al*, 2001; Viano, King, Melvin *et al*, 1989; Nahum, Kroell, Schneider, 1973] have explored the concept that the deceleration induced chest compression may induce extremely high intraortic pressures which may be responsible for the AI. However, in Bass’s very precise studies of longitudinal and circumferential stresses induced by in vitro pressure impulse testing of human aortas, he found that pressure increases rising to more than 85kPa at rates exceeding 1000kPa/sec were more likely to produce longitudinal ruptures at the isthmus, rather than the clinically found transverse AI disruptions. Also, their studies noted that both the circumferential as well as the longitudinal stress-to-stretch ratios were most rapidly achieved in the stiffer aortas of patients older than 65 years, whereas, as widely reported in the literature, and in the present study, the vast majority of AI cases (both survivors and fatalities) occur in the young.

A great deal of comment has been made about the absence of chest wall fractures [Shkrum *et al*, 1999; Hossack, 1980] in many AI patients. However, as shown in Figure 15B, there appears to be a high incidence of upper rib fractures (ribs 1-4 and 5-8) in the ME and H cases, but these were less common in the hospital survivors and, since most surgical series reported deal only with patients reaching hospital alive, it is likely that the high incidence of thoracic wall bony injury has been overlooked. However, these data do not support the largely discredited “osseous pinch” theory [Crass *et al*, 1990].

Rather, they suggest that the mechanism of AI due to differential deceleration between the relatively tethered and more mobile parts of the aorta may occur in response to a

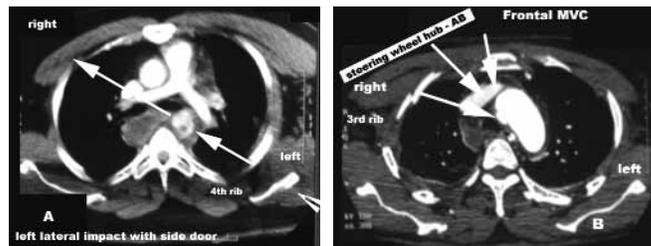
Figure 18. Site of Impact Resulting in Aortic Injury. A: Lateral MVC Left Chest Impact with Side Door and B Pillar; B: Frontal MVC Right Chest Impact with Air Bag Cover and Steering Wheel Hub



highly focused trajectory of impact force at the time of the MVC, initiated at the precise point of the patient's bodily contact with the structures of the passenger compartment. This would appear to be a more likely mechanism in Lateral MVCs where the patient's body is in very close proximity to the intruded side door (Figure 18A) and B-pillar structures, which directly transmit the crash impact energy to the thoracic structures. However, it can also occur in Frontal MVCs when the anterior chest has a very discrete point of impact with the steering wheel hub (Figure 18B), airbag cover or instrument panel.

Evidence for this sort of mechanism can be seen in the Computer Tomographic pictures (Figures 19A and 19B) of the Lateral and Frontal MVC patients whose points of vehicle compartment contact were shown in Figures 18A and 18B, respectively. In these CT cross-sections, the level of the rib fractures which mark the site of the passenger compartment structure's impact on the body are seen to lie at a thoracic level that is in the direct line of a trajectory which passes through the point of the aortic disruption. Obviously, such a highly focused impact energy transmission need not always result in rib or sternal fractures, but

Figure 19. Computer Tomographic Images of Aortic Injury of Patients Injured in the A: Lateral and B: Frontal Impacts Shown in Figure 18



the concept that a force vector may be transmitted with a different degree of efficiency at different levels of the thorax needs further investigation. This idea is further supported by the evidence, also shown in Figure 15, that the AI hospital survivors, with or without rib 9-12 fractures, tend to have more of their associated injuries in the lower thoracic cage protected structures (i.e.: spleen) and in the pelvis and lower extremities, than do the ME and H cases.

Finally, having now introduced the issue of the Impact Energy (IE) imparted to the subject vehicle (V1) at the time of the MVC as a function of the deceleration impact velocity ($\Delta V1$) modulated by the stiffness characteristics of the motor vehicle which result in the magnitude of the vehicle and passenger compartment deformation, it is of interest to consider the data presented in Figure 17. While there are many sources of inaccuracy in the estimations of $\Delta V1$ and the impact energy (IE) dissipation derived from WINSMASH, nevertheless, in the absence of direct measurements of the primary variables involved (which may come from future studies using Automatic Crash Notification sensor technology), perhaps these derived data offer some clues as to the nature and quality of the force mechanisms responsible for AI. As can be seen, when the impact energy (IE) dissipated in the MVC is considered as a function of the $\Delta V1$, there appears to be a highly significant relationship found in the AI scene death ME cases which forms an upper limit for potential patient survival.

In contrast, patients with severe thoracic MVC trauma (ST), but who did not manifest AI, had a significantly lower IE to $\Delta V1$ slope, regardless of their final outcome. However, those AI patients who survived long enough to reach hospital alive (the potential AI survivors) fell between these two regression derived slopes and in general were closer to the ST slope than to the ME one. Moreover, the deaths in the H cases were generally secondary to an interaction of the AI induced shock state with one or more of the associated organ injuries, especially that of the brain, as has been noted previously in other types of trauma cases [Siegel, Rivkind, Dalal et al, 1990]. This may be particularly relevant in two of the H cases where there was a substantial delay in definitive treatment due to a misriage of the

patient to a non-trauma center hospital before the AI was suspected. In these cases, the prolonged hypoperfusion due to their semi-contained AI, by producing an increasing oxygen debt [Siegel et al, 1990; Rixen, Raum, Bouillon et al, 2001], may have contributed to their ultimately fatal outcome.

Also, as shown in Figure 17, there appears to be a Critical Zone within which factors of associated injuries, effectiveness of field resuscitation, time to definitive therapy, etc. may play a role in influencing the final outcome. However, outside and above this IE/delta V1 threshold it would appear that there is little chance of field survival. This implies that an engineering, rather than a medical solution to this particular disease of trauma must be found, by the introduction of safety devices which will reduce the focused force thresholds below the critical level for AI and which will also prevent, or ameliorate the severity of the associated organ injuries, especially those of the brain and the heart. These engineering modifications may include sensor driven frontal and side airbags which are modulated with respect to occupant body weight and position, improved side door and B-pillar construction which directs the MVC impact forces away from the occupants, and universal Automatic Crash Notification technology which will allow a more effective early response that may reduce the shock driven oxygen debt injury [Siegel et al, 1990; Rixen et al, 2001] to critical associated organs so as to provide a greater margin of safety to potentially survivable AI patients who can be brought to hospital alive.

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