AN ANALYSIS OF TRAUMATIC RUPTURE OF THE AORTA IN SIDE IMPACT SLED TESTS

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ABSTRACT

Traumatic rupture of the aorta (TRA) is a leading cause of death in high velocity blunt trauma, particularly motor vehicle accidents. However, little is understood about the mechanisms of TRA and thus, there is little understanding of how to increase occupant protection to prevent TRA in the motor vehicle environment. The objective of this study is to increase the understanding of the relationship between impact response and TRA through analyses of data from cadaver tests that successfully generated TRA in lateral impacts.

Seventeen Heidelberg-style side impact sled tests were conducted using unembalmed human cadavers. Three sled speeds were used: 6.7, 9.0, and 10.5 m/s. Three barrier configurations were used: rigid flat wall, rigid wall with a 152-mm offset toward the pelvis, and a flat wall with padding of varying stiffness. Multiple load and acceleration measurements were made on the barrier and cadaver. Potential injury parameters were evaluated and their relative predictive abilities were examined.

Five of the seventeen tests resulted in AIS 4 or 5 TRA. Most were partial circumferential tears in the periisthmic region. All tears resulted from tests involving the rigid barrier or stiff padding. Tests involving softer padding did not result in TRA. Based on logistic regression analysis Average Spine Acceleration (T12 Y-direction), peak lateral acceleration of the eighth rib on the unstruck side and VCmax had correlation to the presence of TRA. Predictive ability was improved with the inclusion T12-z or upper sternum-x acceleration. The inclusion of age provided modest improvement.

INTRODUCTION

Because the aorta is the major blood vessel emerging from the heart and supplies blood to most of the body, tearing can result in extensive blood loss, hemorrhagic shock and death. The majority of victims of TRA die at the scene of the accident. Rupture of the intrathoracic aorta in blunt trauma accounts for 10-25% of all deaths from motor vehicle accidents (Ochsner et al, 1989; Greendyke, 1966; Hurley, 1986; Pamley, 1958). In the 1980s it was estimated that 7500-8000 cases of blunt aortic injury occurred annually in the United States and Canada (Jackson, 1984; Mattox, 1989). Eighty to eighty-five percent of the victims die at the scene of the accident (Pamley, 1958; Smith and Chang, 1986. Thus, 1000-1500 cases of blunt aortic injury arrive at the hospital annually. Of these, one-third will die within the first six hours and if untreated 90% will die by three months. In a prospective study using 50 trauma centers in North America, Fabian et al (1997) reported 274 (199 men 75 women, mean age 38.7 years) blunt aortic injury cases studied over 2.5 years. Motor vehicle crashes were responsible for 222 of the 274 cases with 72% of these being head-on and 24% from side impacts. The other causes included motorcycle crashes, auto-pedestrian impacts and falls. Forty-six percent had multiple rib fractures, 38% pulmonary contusion, 31% pelvic injury, and 51% closed head injury. Overall mortality was 31%, with 63% of deaths attributable to the aortic rupture itself. The authors concluded that rupture after hospital admission remained a major problem.

That aortic rupture is also associated with lateral directions of impact has been documented in both the laboratory (Cavanaugh et al 1990, 1993) and the field (Newman and Rastogi, 1984; Ben-Menachem, 1993; Katyal et al, 1997; Horton et al, 2000; Augenstein et al, 2000). Newman and Rastogi (1984) reported six frontal, four side and two rollover cases of TRA. They noted that the angle of impact in frontal collisions was not head on in any case, and ranged from 30 degrees to the nearside to sixty degrees to the offside. Ben-Menachem (1993) reported findings in 13 patients who experienced lateral impact and suffered aortic laceration. In most cases the injury appeared to involve partial shearing of the distal aortic arch. As in the study of Ben-Menachem (1993) a shearing mechanism was hypothesized to occur between the fixed descending aorta and the more mobile aortic arch. In a Canadian study Katyal et al (1997) reported that 49.5% of aortic injury cases were the result of lateral impacts. Horton et al (2000) investigated motor vehicle crashes to determine crash scene information that would help clinicians identify patients at risk for aortic trauma. Velocity change (delta V) greater than 20 miles per hour and near-side impact had the strongest correlation with thoracic aortic injury. In another study Augenstein et al (2000) noted that brain and aortic injuries were most frequently associated with side impact fatalities.

Based on retrospective and prospective clinical studies and field accident investigations, several thoracic loading mechanisms appear to be associated with TRA. However, the reproduction of TRA in human cadaveric studies is rare. Thus, the proposed mechanisms of rupture have not been verified. Furthering the understanding of TRA mechanisms can help trauma teams determine if an accident victim may have this injury. In addition, better insight into injury mechanisms can aid the automotive safety engineer in design approaches that may reduce the incidence of this injury, particularly in side impact. The purposes of this study were to review the impact responses in 17 side impact sled tests with unembalmed cadavers and to determine if any pattern of responses was related to the probability of aortic trauma.

METHODOLOGY

Seventeen side impact sled tests were conducted with a horizontally accelerated sled and a Heidelberg type seat fixture using unembalmed human cadavers. The sled was accelerated up to velocities of 6.7 m/s to 10.5 m/s. At the end of the acceleration stroke the sled was disengaged from the propulsion mechanism and allowed to strike a hydraulic snubber. During this impact phase the cadaver slid laterally across a low-friction bench seat and impacted a wall instrumented with load cells (Figure 1). The test series included combinations of different padding stiffness and velocity. Cavanaugh et al (1990, 1993) describe the methods for cadaver preparation, sled preparation, instrumentation and autopsy. Instrumentation location and photo target positions are listed in Table 1.

The seventeen tests are summarized as follows:

1. 9 m/s with the pelvic load plate offset six inches towards the body from the rest of the load plate (N= 2)
2. 10.5 m/s with the pelvic load plate offset six inches toward the body from the rest of the load plate (N= 1)
3. 9 m/s unpadded flat wall (N=2)
4. 6.7 m/s unpadded flat wall (N =3)
5. 9 m/s with 6 inch thick soft padding (N =2)
6. 9 m/s with 4 inch thick soft padding (N =3)
7. 9 m/s with 3 inch thick stiff padding (N =2)
8. 9 m/s with 4 inch thick stiff padding (N =2)

Initial Data Processing

Analog data was initially filtered at 1000 Hz (SAE channel class) and digitized at 8000-10000 Hz. The peak values of these data are shown in Table 3. Data quality checks were performed and any unusable data is labeled "NA" in the tables that follow.

Filtering

Before filtering, the baseline was zeroed for the first 300 points before the cadaver made contact with the barrier. The acceleration data and sidewall force data were digitally filtered with a 300 Hz Butterworth Filter (BWF). This filter corresponds to an SAE CFC 180 Hz filter per the specifications of SAE J211 (2003). The peak responses of filtered data are shown in Table 4.

Normalizing

Data were normalized using the equal stress equal velocity scaling procedure outlined by the Eppinger et al. (1984). Values of lamda, the basic scaling factor used in this method are listed in Table 2. The peak responses of normalized data are shown in Table 5.

Lamda= (standard mass/subject mass) $^{1/3}$
Normalized force =(force x lamda ^{2})
Normalized acceleration = (acceleration /lamda)
Normalized deflection= (deflection x lamda)
Normalized time = (time x lamda)

RESULTS

Tables 3a and 3b list the peak values of the 1000 Hz filtered and non-normalized data. Tables 4a and 4b list the peak values of the 300 Hz BW filtered and non-normalized data. Tables 5a and 5b show the peak values of the 300 Hz BW filtered and normalized data. These latter data were used in the logistic regression analyses that follow. In addition, ASA (T12y Average Spine Acceleration), VCmax and Cmax were evaluated as injury

criteria. Peak values of these responses are listed in the Table 6. These were obtained from Cavanaugh et al (1993).

Table 7 summarizes the aortic injuries based on the autopsy reports. Aortic injury occurred in five cases. In all cases the tears was just distal to the ligamentum arteriosum and proximal to the descending thoracic aorta. In all cases the aortic laceration had a transverse orientation. In four cases the tear was through all layers of the aorta (intima, media and adventitia) resulting in AIS 5. In one case the tear was intimal, resulting in AIS 4.

Logistic Regression Analysis

A logistic regression analysis was performed using SPSS software to determine if a relationship existed between TRA and independent variables in this study. Aortic injury was considered the dependent variable and was assigned a value of 0 or 1. Biomechanical responses including rib, spine and sternum accelerations, chest compression, viscous criterion and barrier forces as well as age were analyzed as independent variables. The equation for the linear logistic regression model is as follows:

$P= 1/(1+\exp(-\text{alpha}- \text{beta}_i * x_n))$

Where:
P is the probability of aortic injury.
Alpha is the intercept and beta_i are the coefficients for each independent variable.
x_n are the independent response variables.

Table 8 summarizes the chi-square values and levels of significance (p) from these analyses. The higher the chi-square distribution and lower the p-value the better the goodness of fit of the model. Right rib 8 acceleration, ASA at T12 and VCmax had the best goodness-of- fit outcomes and had p values less than 0.05. The probability curves for these are shown in Figures 2a-2c.

Multivariate analyses:

Age was combined with the most promising responses for predicting injury; ASA, VCmax and right rib 8 acceleration. In addition, better performing responses were combined with T12-z acceleration to determine if combining a vertical component of torso acceleration would increase the ability to predict aortic injury. Finally, promising predictors were paired in multivariate analyses. The results are summarized in Table 9. Those combinations with p less than 0.05 are shown in bold. Probability curves for three multivariate functions are shown in Figures 3a-3c. ASA, Cmax and VCmax combined with T12z acceleration were the best combinations, with the combination of VCmax and T12z having the best predictive ability. Another combination of orthogonal responses (ASA and upper sternum X acceleration) was also promising.

Padding and aortic injury:

Aortic injury occurred in unpadded pelvic offset tests (SIC 02, SIC03), unpadded 6.7 m/s tests (SIC07, SIC08) and a 9 m/s test with 4-inch thick stiff padding (SIC12). VCmax averaged 2.8 m/s in these tests. There were no aortic injuries in the five soft padded tests (55-70 kPa crush strength) and VCmax averaged 0.98 m/s in these tests.

DISCUSSION

All five aortic tears in our study occurred in the region just distal to the isthmus and proximal to the descending aorta. Ninety percent of thoracic aortic injuries are reported to occur in the region of the aortic isthmus, just distal to the origin of the left subclavian artery (Creasy et al, 1997). The isthmus is that portion of the proximal descending aorta between the origin of the left subclavian artery and the ligamentum arteriosum. The aortic isthmus averages only about 1.5 cm in length in adults, and the region of the proximal descending aorta is often referred to loosely as the region of the aortic isthmus (Creasy et al, 1997). Clinically, thoracic aortic injury involves the ascending aorta in only 5% of cases. This area of the aorta is associated with grave complications such as valve rapture and coronary artery laceration that are threats to life. Hilgenberg et al (1992) reported managing 51 patients with thoracic aortic injuries between 1977 and 1990. Forty-nine were in the upper descending aorta. Kodali et al (1991) reported on 116 patients diagnosed with traumatic aortic rupture between 1969-89. The site of injury in 105 was in the region of the aortic isthmus, distal to the origin of the left subclavian artery.

Aortic rupture has three major hypothesized mechanisms: (1) traction or shear forces generated between relatively mobile portions of the vessel and points of fixation, (2) direct compression over the vertebral column, (3) and excessive sudden increases in intraluminal pressure (Creasy et al, 1997). It has been noted that vertical shearing forces may account for an increased incidence of aortic raptures in victims of plane crashes and falls (Pickard, 1977; Smith and Chang, 1986). Viano (1983) also noted that inertial loading of the blood-filled heart can cause the heart to displace in the chest cavity and stretch points of attachment of the aortic arch such as the superior arteries or the ligamentum arteriosum. This may occur if the heart is displaced vertically, laterally or obliquely. Horton et al (2000) noted that inertia of the heart may produce traction on the thoracic aorta along its long axis, putting tensile strain in the region of the isthmus. In a review of 25 cases of aortic rupture admitted to the University of Virginia 23 were caused by MVAs and 2 from falls. The aortic tears were just distal to the left subclavian artery in 19 patients [Tribble and Crosby (1988)]. The authors contended that most injuries are produced when the heart and mediastinum are pushed posteriorly and superiorly into the left hemithorax. The aortic arch would then be forced superiorly and twisted, causing shear and stretch at the isthmus. This mechanism was not proven, however. Gotzen et al (1980) reported that the predominant direction of impact in 26 cases of aortic rupture was from the right ventrocaudal direction. In the current study T12-z itself was not a significant predictor of aortic injury. ASA and VCmax had some level of significance. However, when T12-z acceleration was combined with ASA, VCmax or Cmax the predictive ability was much improved. This suggests the importance of a vertical component of acceleration in producing aortic injury. The modest improvement seen with

the inclusion of sternum acceleration in the x-direction suggests that posterior-to-anterior stretching of the aortic arch can contribute to TRA.

Five of seventeen surrogates had aortic rupture with the tears having a transverse orientation distal to the ligamentum arteriosum. Mohan and Melvin (1982) demonstrated in uniaxial dynamic tensile tests that when pulling along the longitudinal axis of the aorta, ultimate stress was only 0.65 of that obtained when pulling along a transverse axis. Thus, transverse tears occurred at lower stresses than longitudinal tears. Mohan and Melvin (1983) demonstrated in biaxial inflation tests that high internal vascular pressures may balloon out the vessel wall, and a transverse rupture of the aortic wall could be expected with large hydrodynamic pressures.

In this study including the subject's age in a multivariate analysis improved the predictive ability of response parameters. The aorta loses strength with age. Yamada (1970) reported the ultimate tensile strength for the 50-79 year old age groups was about 62% of the 0-19 year old age group.

In the current study the aortic tears were close to points restraint; between the ligamentum arteriosum and attachments of the aorta to the posterior chest wall. In his review Viano (1983) points out that the most widely held view of aortic rupture is that the descending aorta remains relatively fixed at the posterior chest wall by the intercostal arteries while the heart and aortic arch displace and induce tension at sites of attachment. This appears to be a point of either confusion or controversy. Some investigators report (Tripp, 1995; Moar, 1985) the relative motion is between the more fixed attachment of the aortic arch and the more mobile descending thoracic aorta. Observation by Cavanaugh et al (1993) in side impact studies supports the review by Viano because indeed the descending thoracic aorta was fixed to the chest wall in post-impact necropsy studies and the transverse tears in the aorta occurred above this attachment to the posterior chest wall.

Shah et al (2001) and Richens et al (2004) have developed finite element models of the aorta and surrounding thoracic structures. Continued development of these models can lead to further understanding of aortic injury mechanisms.

CONCLUSIONS

1. Aortic injury occurred in 5 of 12 unpadded or stiff padded impacts. In the five impacts with soft padding no aortic injury occurred.
2. ASA, unstruck rib-8 acceleration and VCmax showed correlation with aortic injury.
3. The inclusion of subject age modestly improved the predictive ability of these response variables.
4. Combining upper sternum-x acceleration with ASA and combining T12-z (vertical acceleration) with VCmax, Cmax and ASA resulted in the best predictors of aortic injury.

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TABLES

Table 1. Instrumentation

Instrumentation	**Location and axes**
Accelerometers	Head- 3,2,2,2 Shoulder - X, Y, Z Up sternum- X, Y Lower sternum- X, Y T1- X, Y, Z T12- X, Y, Z S1- X, Y, Z Rib4 Y (right and left) Rib 8 Y (right and left)
Barrier load cells	Shoulder 1, 2 Thorax 1, 2 Abdomen 1, 2 Pelvis 1, 2 Knee
Photo Targets	Upper sternum, lower sternum T1, T5, T12 Sacrum, right iliac crest Rib 4 (Right) Rib 8 (Right) Medial and lateral ends of the clavicle Acromion and spine of the scapula Acromion of the right scapula

Table 2. Cadaver and scaling data

RUN NO.	AGE	GENDER	MASS (kg)	LAMDA Standard = 75 kg
SIC01	60	M	705	1.021
SIC02	64	F	49.5	1.149
SIC03	37	M	70.0	1.023
SIC04	69	M	57.6	1.092
SIC05	67	M	44.0	1.195
SIC06	60	M	61.2	1.070
SIC07	66	M	74.8	1.001
SIC08	64	F	73.9	1.005
SIC09	61	M	54.9	1.110
SIC10	60	M	62.1	1.065
SIC11	54	F	55.3	1.107
SIC12	68	F	54.4	1.113
SIC13	62	M	66.7	1.040
SIC14	72	M	55.3	1.107
SIC15	43	F	68.9	1.029
SIC16	58	F	56.7	1.098
SIC17	65	M	93.0	0.931

Table 3a. Peak responses of 1000 Hz filtered and non-normalized data

TEST TYPE	AIS TO AORTA	ACCELERATIONS g' S					
SIC RUN NO AND TYPE		T1 X	T1 Y	T1 Z	T12X	T12Y	T12Z
UNPADDEDPELVIC OFFSET 9 m/s							
1	0	96.3	255.9	102.2	NA	NA	NA
2	4	70.2	84.8	103.7	97.2	147.3	61.6
UNPADDED PELVIC OFFSET 10.5 m/s							
3	5	131.5	228.1	93.5	98.0	192.0	135.9
UNPADDED 9 m/s							
4	0	124.9	104.8	85.3	109.8	84.3	42.6
6	0	110.1	149.7	94.4	62.2	117.0	39.2
UNPADDED 6.7 m/s							
5	0	124.4	103.3	96.1	44.8	123.4	37.7
7	0	44.9	78.2	39.5	39.2	50.1	24.8
8	5	102.7	69.5	44.6	52.1	54.4	N/A
6 INCH SOFT PAD 9 m/s							
10	0	14.2	57.5	23.3	70.5	137.3	47.2
17	0	NA	51.2	46.7	41.1	77.3	65.5
4 INCH SOFT PAD 9 m/s							
11	0	222.0	89.2	NA	38.8	67.9	40.0
13	0	49.3	70.9	NA	N/A	N/A	N/A
15	0	38.1	N/A	72.5	41.2	58.1	163.8
3 INCH STIFF PAD 9 m/s							
9	5	N/A	138.8	N/A	68.6	90.9	100.0
16	0	40.3	58.5	69.6	92.9	N/A	48.4
4 INCH STIFF PAD 9 m/s							
12	5	83.2	101.4	NA	48.5	82.7	65.3
14	0	194.4	85.7	N/A	81.5	143.9	69.0

Table 3b. Peak responses of 1000 Hz filtered and non-normalized data

TEST TYPE	AIS TO AORTA	ACCELERATIONS G						BEAM FORCES KN		
SIC RUN NO AND TYPE		R4 LEFT	R8 LEFT	R4 RIGHT	R8 RIGHT	UPPER STERNUM-X	LOWER STERNUM-X	THORAX	SHOULD + THOR	SHOULD
UNPADDED PELVIC OFFSET 9 m/s										
1	0	N/A	N/A	N/A	227.6	147.1	112.3	4.9	7.3	3.4
2	4	287.0	179.2	53.5	N/A	163.1	94.4	2.7	5.7	3.7
UNPADDED PELVIC OFFSET 10.5 m/s										
3	5	478.0	98.6	325.0	NA	173.8	229.8	4.6	11.4	8.4
UNPADDED 9 m/s										
4	0	291.0	415.7	318.0	NA	76.4	142.2	3.9	8.3	5.3
6	0	447.0	97.9	107.0	124.5	161.6	114.1	3.4	7.9	5.3
UNPADDED 6.7 m/s										
5	0	440.0	263.8	216.0	375.6	76.2	57.9	1.2	4.1	3.1
7	0	157.0	99.0	205.0	NA	20.8	NA	2.9	6.6	3.9
8	5	N/A	397.0	168.0	254.7	NA	96.1	3.2	5.9	3.0
6 INCH SOFT PAD 9 m/s										
10	0	284.0	173.9	44.8	77.8	56.0	91.6	3.3	5.9	3.2
17	0	239.0	220.1	40.8	58.9	27.0	66.3	3.4	8.2	5.5
4 INCH SOFT PAD 9 m/s										
11	0	114.0	234.2	59.3	63.9	65.1	60.1	2.8	5.0	2.9
13	0	168.0	589.1	348.0	106.8	48.6	56.3	3.0	8.5	5.7
15	0	110.0	76.0	68.1	54.1	22.4	28.9	3.2	8.0	5.5
3 INCH STIFF PAD 9 m/s										
9	5	341.0	153.1	190.0	335.5	382.0	N/A	3.4	6.6	3.7
16	0	549.0	465.0	178.0	104.4	138.0	106.9	3.8	6.8	3.3
4 INCH STIFF PAD 9 m/s										
12	5	767.0	450.0	125.0	86.0	82.3	68.9	3.9	7.5	3.7
14	0	370.0	531.2	103.0	65.6	96.0	72.1	7.3	10.9	4.2

Table 4a. Peak responses of 300 Hz BW filtered and non-normalized data

TEST TYPE	AIS TO AORTA	ACCELERATIONS g' S					
SIC RUN NO AND TYPE		T1 X	T1 Y	T1 Z	T12X	T12Y	T12Z
UNPADDEDPELVIC OFFSET 9 m/s							
1	0	61.7	214.4	92.8	NA	NA	NA
2	4	34.7	75.8	65.7	52.8	133.7	52.2
UNPADDED PELVIC OFFSET 10.5 m/s							
3	5	63.7	197.9	86.2	84.5	171.9	116.8
UNPADDED 9 m/s							
4	0	116.7	53.1	79.5	99.7	79.6	41.4
6	0	41.0	139.2	95.4	59.6	107.7	38.9
UNPADDED 6.7 m/s							
5	0	40.7	75.5	61.1	40.0	109.7	22.8
7	0	41.1	72.9	38.1	33.4	46.2	23.1
8	5	41.3	63.6	33.9	51.5	52.4	N/A
6 INCH SOFT PAD 9 m/s							
10	0	13.2	51.7	20.5	65.7	127.7	38.9
17	0	N/A	49.5	38.8	35.2	74.5	20.8
4 INCH SOFT PAD 9 m/s							
11	0	226.0	76.0	NA	37.7	65.7	38.9
13	0	27.7	58.9	NA	N/A	N/A	N/A
15	0	34.7	N/A	65.2	38.8	55.8	126.0
3 INCH STIFF PAD 9 m/s							
9	5	N/A	138.8	N/A	60.8	86.4	78.2
16	0	28.5	53.0	55.0	85.5	N/A	40.7
4 INCH STIFF PAD 9 m/s							
12	5	46.1	97.1	NA	63.5	102.9	47.0
14	0	69.4	75.7	N/A	76.8	118.0	52.0

Table 4b. Peak responses of 300 Hz BW filtered and non-normalized data

TEST TYPE	AIS TO AORTA	ACCELERATIONS G						BEAM FORCES KN		
SIC RUN NO AND TYPE		R4 LEFT	R8 LEFT	R4 RIGHT	R8 RIGHT	UPPER STERNUM-X	LOWER STERNUM-X	THORAX	SHOULD + THOR	SHOULD
UNPADDED PELVIC OFFSET 9 m/s										
1	0	N/A	N/A	N/A	103.8	101.0	77.5	4.4	6.7	3.0
2	4	241.0	130.3	41.8	N/A	102.3	85.9	2.6	5.4	3.5
UNPADDED PELVIC OFFSET 10.5 m/s										
3	5	211.2	348.9	202.4	N/A	119.5	109.3	3.9	10.6	8.0
UNPADDED 9 m/s										
4	0	268.3	315.6	278.5	N/A	74.4	75.0	3.2	7.7	4.8
6	0	210.6	324.0	89.8	124.4	95.0	81.6	2.6	6.7	4.5
UNPADDED 6.7 m/s										
5	0	142.9	205.0	115.1	177.0	83.9	84.8	1.1	3.6	2.5
7	0	64.0	151.8	61.4	N/A	81.5	N/A	2.6	6.1	3.5
8	5	N/A	77.1	46.1	239.2	N/A	75.4	2.9	5.6	2.8
6 INCH SOFT PAD 9 m/s										
10	0	126.1	150.4	40.9	75.1	84.8	74.9	2.5	5.0	2.7
17	0	181.0	157.7	31.2	46.4	100.0	68.6	3.0	7.9	5.4
4 INCH SOFT PAD 9 m/s										
11	0	98.2	163.7	14.7	61.8	81.8	83.9	2.6	4.9	2.6
13	0	147.7	187.5	56.3	45.0	113.6	87.3	2.9	7.9	5.5
15	0	86.5	63.2	61.0	48.7	81.3	72.3	2.6	7.7	5.5
3 INCH STIFF PAD 9 m/s										
9	5	156.5	101.2	100.2	212.0	79.0	63.9	3.3	6.1	3.3
16	0	99.0	415.1	144.5	102.9	84.9	85.3	3.7	6.6	3.0
4 INCH STIFF PAD 9 m/s										
12	5	153.3	98.3	85.1	79.6	90.9	88.8	3.5	7.1	3.6
14	0	81.5	246.7	89.3	60.5	85.6	75.4	7.0	10.5	4.0

Table 5a. Peak responses of 300 Hz BW filtered and normalized data

TEST TYPE	AIS TO AORTA	ACCELERATIONS g' S					
SIC RUN NO AND TYPE		T1 X	T1 Y	T1 Z	T12X	T12Y	T12Z
UNPADDEDPELVIC OFFSET 9 m/s							
1	0	60.4	210.0	90.9	N/A	N/A	N/A
2	4	30.2	66.0	57.2	46.0	116.4	45.4
UNPADDED PELVIC OFFSET 10.5 m/s							
3	5	62.3	193.5	84.3	82.6	168.0	114.2
UNPADDED 9 m/s							
4	0	106.9	48.6	72.8	91.3	72.9	37.9
6	0	38.3	130.1	89.2	55.7	100.7	36.4
UNPADDED 6.7 m/s							
5	0	34.1	63.2	51.1	33.5	91.8	19.1
7	0	41.1	72.8	38.1	33.4	46.2	23.1
8	5	41.1	63.3	33.7	51.2	52.1	N/A
6 INCH SOFT PAD 9 m/s							
10	0	12.4	48.5	19.2	61.7	119.9	36.5
17	0	N/A	53.2	41.7	37.8	80.0	22.3
4 INCH SOFT PAD 9 m/s							
11	0	204.2	68.7	N/A	34.1	59.3	35.1
13	0	26.6	56.6	N/A	N/A	N/A	N/A
15	0	33.7	N/A	63.4	37.7	54.2	122.4
3 INCH STIFF PAD 9 m/s							
9	5	N/A	125.0	N/A	54.8	77.8	70.5
16	0	26.0	48.3	50.1	77.9	N/A	37.1
4 INCH STIFF PAD 9 m/s							
12	5	41.4	87.2	N/A	57.1	92.5	42.2

14	0	62.7	68.4	N/A	69.4	106.6	47.0

Table 5b. Peak responses of 300 HZ BW filtered and normalized data

TEST TYPE	AIS TO AORTA	ACCELERATIONS g						BEAM FORCES KN		
SIC RUN NO AND TYPE		R4 LEFT	R8 LEFT	R4 RIGHT	R8 RIGHT	UPPER STERNUM-X	LOWER STERNUM-X	THORAX	SHOULD + THOR	SHOULD
UNPADDED PELVIC OFFSET 9 m/s										
1	0	N/A	N/A	N/A	101.7	98.9	75.9	4.6	7.0	3.2
2	4	209.7	113.4	36.4	N/A	89.0	74.8	3.4	7.1	4.6
UNPADDED PELVIC OFFSET 10.5 m/s										
3	5	206.5	341.1	197.8	N/A	116.8	106.8	4.1	11.1	8.3
UNPADDED 9 m/s										
4	0	245.7	289.0	255.0	N/A	68.1	68.7	3.9	9.2	5.7
6	0	196.8	302.8	83.9	116.3	88.8	76.3	3.0	7.7	5.3
UNPADDED 6.7 m/s										
5	0	119.6	171.5	96.3	148.1	70.2	71.0	1.6	5.2	3.7
7	0	63.9	151.6	61.3		81.4	N/A	2.6	6.1	3.5
8	5	N/A	76.7	45.9	238.0	N/A	75.0	2.9	5.6	2.9
6 INCH SOFT PAD 9 m/s										
10	0	118.4	141.2	38.4	70.5	79.6	70.3	2.9	5.8	3.1
17	0	194.4	169.4	33.5	49.8	107.4	73.7	2.6	6.8	4.6
4 INCH SOFT PAD 9 m/s										
11	0	88.7	147.9	13.3	55.8	73.9	75.8	3.1	6.0	3.3
13	0	142.0	180.3	54.1	43.3	109.2	83.9	3.1	8.5	5.9
15	0	84.1	61.4	59.3	47.3	79.0	70.3	2.7	8.2	5.8
3 INCH STIFF PAD 9 m/s										
9	5	141.0	91.2	90.3	191.0	71.2	N/A	4.0	7.6	4.1
16	0	90.2	378.1	131.6	93.7	77.3	77.7	4.4	8.0	3.6

4 INCH STIFF PAD 9 m/s										
12	5	137.7	88.3	76.5	71.5	81.7	79.8	4.3	8.9	4.5
14	0	73.6	222.9	80.7	54.7	77.3	68.1	8.6	12.8	4.9

Table 6. Peak values (from Cavanaugh et al, 1993)

TEST NO.	**AIS Aorta**	**ASA-T12y**	**Cmax**	**VCmax**
		G	**%**	**m\s**
SIC 01	0	NA	83	4.8
SIC 02	4	65.5	59	3.3
SIC 03	5	57.0	48	4.5
SIC 04	0	52.7	49	1.7
SIC 05	0	23.6	41	1.0
SIC 06	0	43.6	45	1.1
SIC 07	0	28.8	41	1.3
SIC 08	5	29.5	61	2.6
SIC 09	5	41.1	56	1.6
SIC 10	0	21.1	36	1.0
SIC 11	0	23.3	35	0.7
SIC 12	5	46.3	56	2.0
SIC 13	0	NA	35	0.9
SIC 14	0	36.8	42	1.1
SIC 15	0	27.6	52	1.3
SIC 16	0	N/A	55	2.1
SIC 17	0	19.5	43	1.0

Table 7-autopsy report summary

Test No.	AIS Aorta	Aortic Injury	Site Of Injury And Comments
UNPADDED PELVIC OFFSET 9 m/s			
SIC01	0		
SIC02	4	1 cm intimal tear of descending aorta (AIS 4)	Just below the ligamentum arteriosum
UNPADDED PELVIC OFFSET 10.5 m/s			
SIC03	5	1.5 cm transverse complete tear of thoracic aorta	Just below the level of ligamentum arteriosum
UNPADDED 9 m/s			
SIC04	0		
SIC06	0		
UNPADDED 6.7 m/s			
SIC05	0		
SIC07	0		
SIC08	5	1 cm tear of thoracic aorta	In an area of marked atheroslcerosis just distal to left subclavaian artery and ligamentum arteriosum
6 INCH SOFT PAD 9 m/s			
SIC10	0		
SIC17	0		
4 INCH SOFT PAD 9 m/s			
SIC11	0		
SIC13	0		
SIC15	0		
3 INCH STIFF PAD 9 m/s			
SIC09	5	Transverse 1 cm tear of thoracic aorta (AIS5)	Just distal to left subclavian artery and

SIC16	0		ligamentum arteriosum
4 INCH STIFF PAD 9 m/s SIC12 SIC14	 5 0	1.5 cm transverse partial traumatic aortic rupture (AIS5)	Just distal to left subclavian artery and ligamentum arteriosum

Table 8: Logistic Regression Analysis
Single Variables

Response variable	Chi square	Significance
Peak spine Accelerations		
T1-X	0.386	0.5346
T1-Y	1.058	0.3037
T1-Z	0.005	0.9453
T12-X	0.261	0.6094
T12-Y	1.289	0.2562
T12-Z	1.960	0.1615
Peak rib accelerations		
R4-LEFT	1.952	0.1623
R8-LEFT	1.506	0.2197
R4-RIGHT	0.042	0.8370
R8-RIGHT	5.166	0.0230
Peak sternum accelerations		
U.STERN-X	0.403	0.5255
L.STERN-X	3.750	0.0528
Peak barrier forces		
SHOULDER	0.467	0.4944
THOROAX	0.035	0.8507
SHOULD+THORAX	0.188	0.6649
Average spine acceleration		
ASA 10 at T12	5.216	0.0224
Deflection-based film data		
Cmax	2.329	0.1270
VCmax	3.959	0.0466

Table 9: Multivariate Analysis

Combination	k1	k2	k3	Chi-square	P value
k1*Age+k2*T12Z+k3	0.0918	0.0513	-9.1772	2.657	0.1031
k1*Age+k2*R8r+k3	**0.3454**	**0.0416**	**-28.0385**	**7.057**	**0.0079**
k1*Age+k2*UpsX+k3	-0.0351	0.0171	-0.4842	0.690	0.4063
k1*Age+k2*LosX+k3	**0.1566**	**0.2446**	**-29.6114**	**5.008**	**0.0252**
k1*Age+k2*ASA+k3	**-0.0324**	**0.1088**	**-2.8124**	**5.403**	**0.0201**
k1*Age+k2*Cmax+k3	-0.0322	0.0733	-2.6504	1.662	0.1973
k1*Age+k2*VCmax+k3	**0.0191**	**0.9727**	**-4.0128**	**4.021**	**0.0449**
k1*T12Z+k2*R8r+k3	0.0189	0.0242	-4.7658	2.249	0.1337
k1*T12Z+k2*UpsX+k3	0.0226	0.0393	-5.3859	2.576	0.1085
k1*T12Z+k2*LosX+k3	**0.0297**	**0.5884**	**-47.0305**	**6.631**	**0.0100**
k1*T12Z+k2*ASA+k3	**0.0426**	**0.2123**	**-12.0304**	**8.985**	**0.0027**
k1*T12Z+k2*Cmax+k3	**0.0236**	**0.3666**	**-20.9704**	**8.438**	**0.0037**
k1*T12Z+k2*VCmax+k3	**0.0294**	**4.6622**	**-10.4518**	**9.760**	**0.0018**
k1*R8r+k2*Upsx+k3	0.0200	-0.0431	-0.1473	2.360	0.1245
k1*R8r+k2*LosX+k3	**0.0443**	**0.5547**	**-48.9654**	**5.271**	**0.0217**
k1*R8r+k2*ASA+k3	**0.0250**	**0.1696**	**-9.8907**	**6.363**	**0.0116**
k1*R8r+k2*Cmax+k3	**0.0274**	**0.0355**	**-6.2468**	**5.506**	**0.0190**
k1*R8r+k2*VCmax+k3	**0.0286**	**0.1948**	**-4.8498**	**5.244**	**0.0220**
k1*UpsX+k2*LosX+k3	**-0.0534**	**0.2770**	**-18.1059**	**4.822**	**0.0281**
k1*UpsX+k2*ASA+k3	**0.0964**	**0.1889**	**-16.1679**	**8.405**	**0.0037**
k1*UpsX+k2*Cmax+k3	0.0228	0.0558	-5.8607	1.726	0.1889
k1*UpsX+k2*VCmax+k3	-0.0269	1.0226	-0.9134	3.497	0.0615
k1*LosX+k2*Cmax+k3	**0.1894**	**0.0699**	**-19.2160**	**5.652**	**0.0174**
k1*LosX+k2*VCmax+k3	**0.1664**	**0.7958**	**-15.4318**	**5.761**	**0.0164**

R8r: Right rib 8 acceleration
UpsX: Upper sternum X acceleration
LosX: Lower sternum X acceleration

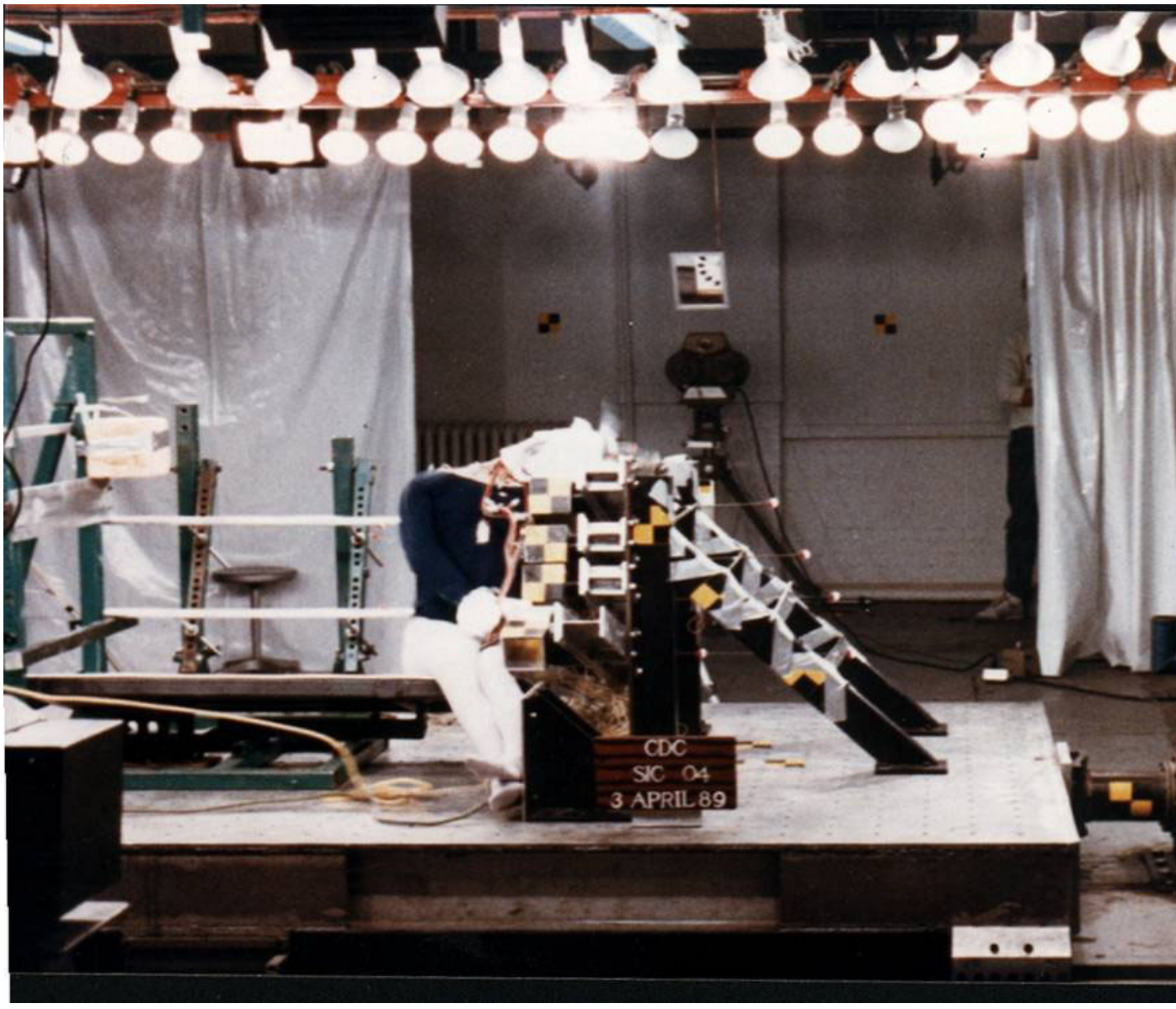

Figure 1. Photo of 9 m/s impact of cadaver into unpadded flat wall (CDC SIC04).

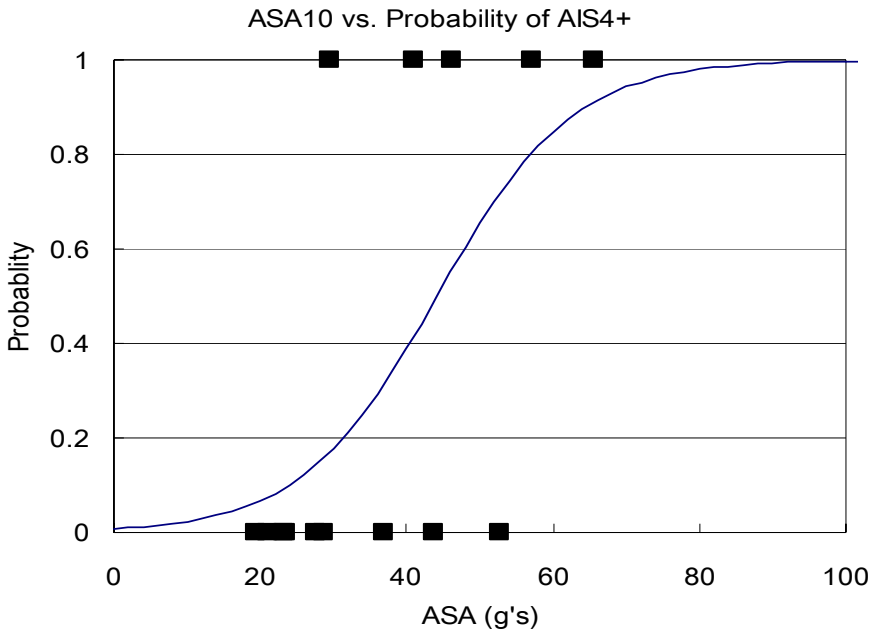

Figure 2a. Logist plot of probability of AIS 4 or higher to the aorta vs. ASA10 (Average Spine Acceleration, Chi square = 5.216, P = 0.0224)

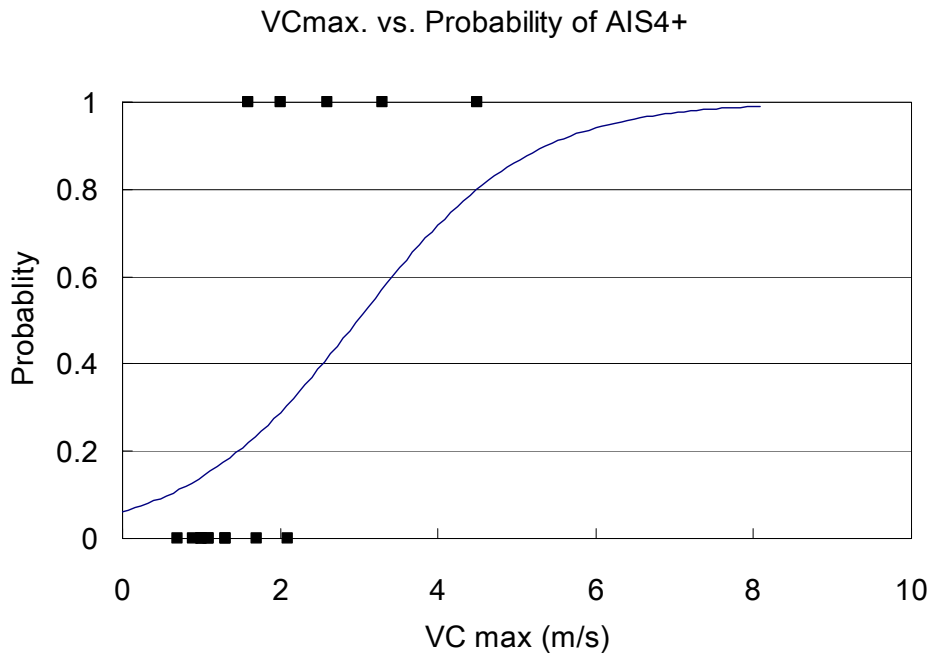

Figure 2b. Logist plot of probability of AIS 4 or higher to the aorta vs. VCmax (Chi square = 3.959, P = 0.0466)

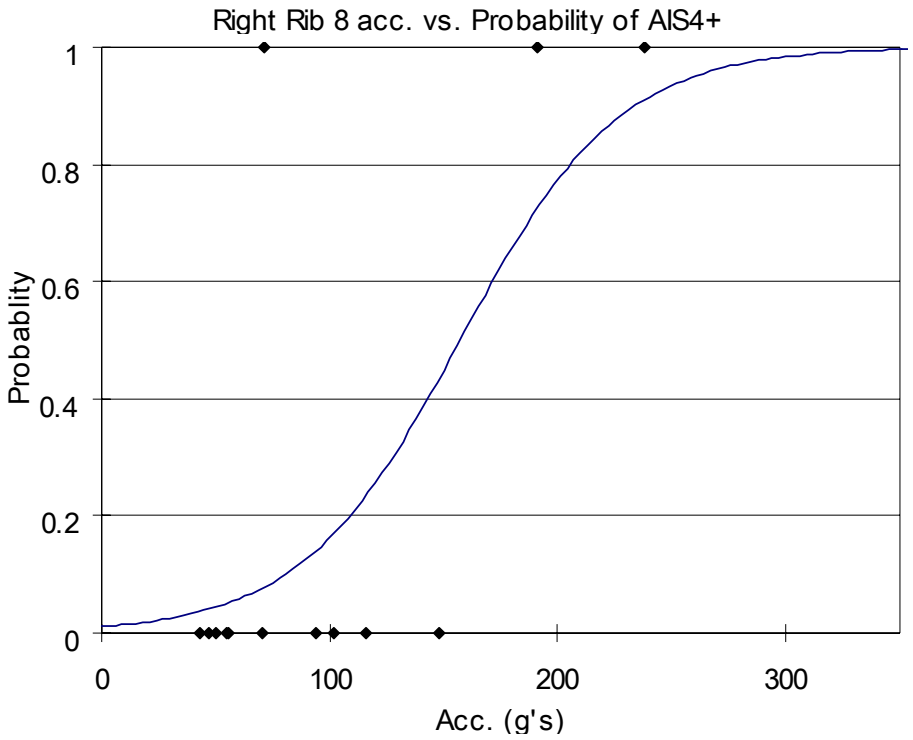

Figure 2c. Logist plot of probability of AIS 4 or higher to the aorta vs. Right rib 8 acceleration (Chi square = 5.166, P = 0.0230)

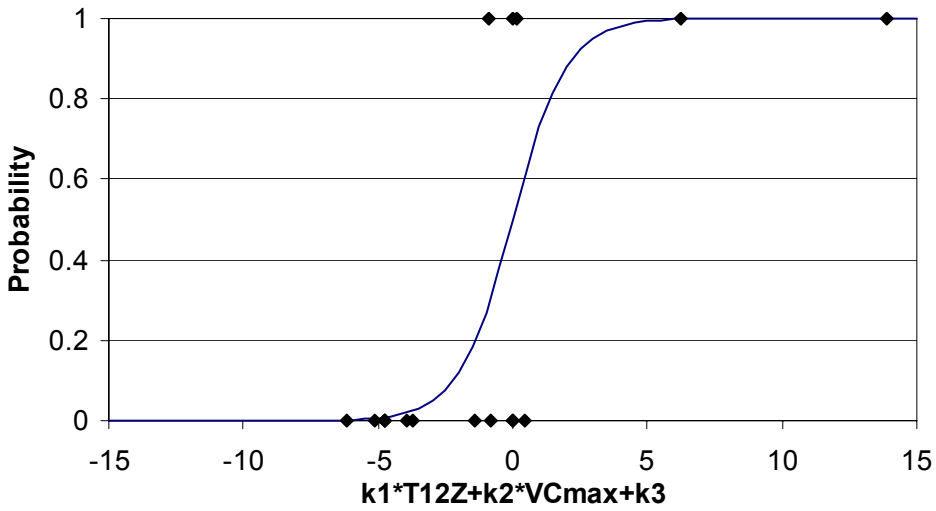

Figure. 3a. Logist plot of probability of AIS 4 or higher to the aorta vs. combination of T12Z acceleration and VCmax (Chi square = 9.760, P = 0.0018)

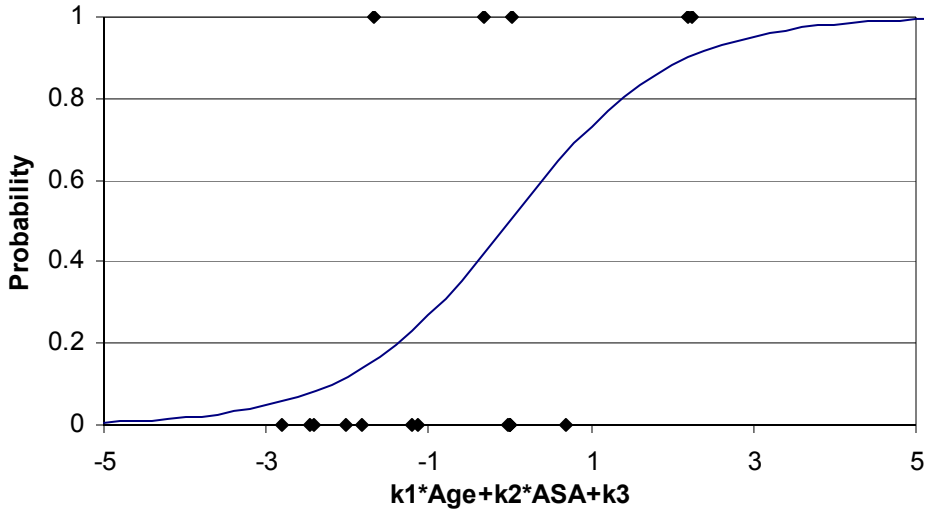

Figure 3b. Logist plot of probability of AIS 4 or higher to the aorta vs. combination of Age and ASA (Chi square = 5.403, P = 0.0201)

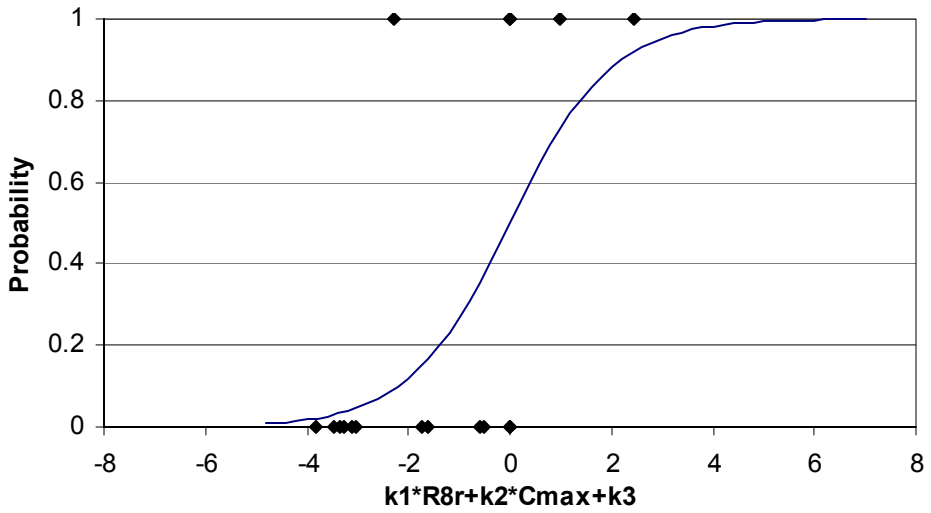

Figure 3c. Logist plot of probability of AIS 4 or higher to the aorta vs. combination of right rib 8 acceleration and Cmax (Chi square = 5.506, P = 0.0190)