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Mechanisms of Aortic Injury in Fatalities Occurring in Motor Vehicle Collisions*

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ABSTRACT: Case reviews based on autopsy studies have shown that motor vehicle collisions cause between 50 and 90% of traumatic aortic ruptures. Very few studies have analyzed the nature and severity of the collision forces associated with this injury. Our passenger car study (1984–1991) examined 36 collisions in which 39 fatally injured victims sustained aortic trauma. In this injury group, a disproportionate number of heavy truck and roadside fixed-object impacts occurred. Vehicle crash forces were generally severe and were either perpendicular or oblique to the vehicle surface. Intrusion into the occupant compartment was a significant factor in most of these fatal injuries. Occupant contact with vehicle interior surfaces was identified in most cases, and occupant restraints were often ineffective, especially in side collisions. The more elderly victims were seen in the least severe collisions.

The most frequent site of aortic rupture was at the isthmus. A majority of victims had rib/sternal fractures indicating significant chest compression. Of the various traumatic aortic injury mechanisms proposed in motor vehicle impacts, the favored theories in the literature combine features of rapid deceleration and chest compression. This study supports that predominant impression, concluding that rapid chest deceleration/compression induces torsional and shearing forces that result in transverse laceration and rupture of the aorta, most commonly in the inherently vulnerable isthmus region.

KEYWORDS: forensic science, wounds and injuries, wounds, nonpenetrating, accidents, traffic, aorta, aortic rupture

Chest injury is common in automobile accident victims and is a factor in about 50% of motor vehicle occupant deaths (1–4). The incidence of traumatic aortic rupture in deceased motor vehicle occupants, as documented in autopsy studies since the 1960s, ranges from 12 to 26% (2,4–8). Understanding the mechanisms resulting in this injury requires not only integration of clinical and postmortem observations of injured individuals but also reconstruction of the actual motor vehicle collisions in which they were involved, including the predicted occupant kinematics (1,9,10). Identification of common features could assist in the early clinical recognition and treatment of cases at risk for aortic injury as well as its prevention (10). The medical literature on aortic trauma has been hindered by a corresponding lack of detailed crash descriptions (3,8). Research designed to study injury risk in car occupants has been hindered by unsatisfactory simulations of the human body (9,11). Clinical research of any type of injury is usually based on case selection influenced by the investigator's medical practice or specialty area and is usually focused on timely diagnosis and treatment to achieve reduction in morbidity and mortality (1). Medical studies tend to group trauma arising during motor vehicle crashes with other types of injury scenarios (1). Reviews based on autopsy cases have shown that automobile collisions cause about half to 95% of aortic ruptures (5,7,12–16). Long-term survival from aortic rupture is low even in areas with access to advanced trauma care and ranges from 10 to 16% (12,15). Postmortem observations, therefore, can provide insight into the factors responsible for this injury (6).

Methods

All cases of aortic trauma occurring in motor vehicle collision fatalities investigated during the Passenger Car Study (PCS), 1984 to 1991, by the University of Western Ontario (U.W.O) Multi-Disciplinary Accident Research Team were reviewed (17). The U.W.O. Team is part of a Canadian national network of university-based teams, funded by Transport Canada, that investigate representative fatal and nonfatal motor vehicle collisions in a defined geographic area.

Certain types of collision were selected: frontal and side impacts involving either a single car or light truck with either another similar vehicle, heavy truck (>5000 kg or 11 000 lb) or fixed object. Side collisions were considered as either near-side, that is, the fatally injured occupant was on the same side as the collision or far-side, that is, the occupant fatality was seated opposite to the impact. Information about the vehicle(s) and occupants was derived from PCS files. Specific data included age and sex of the deceased, occupant location, restraint system use, role of ejection and intrusion in injury causation, force direction and Vehicle Deformation Extent (VDE). VDE is a component of the Collision Deformation Classification (CDC) used to classify vehicle deformation due to impact (18). The maximum degree of deformation is numbered (1 to 9) according to extent zones across either the length (frontal collision) or width (side collision) of the vehicle (Figs. 1 and 4).

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The Equivalent Barrier Speed (EBS) was calculated for the frontal and side impacts when the necessary data were available. The EBS is often a good predictor of the velocity change, or delta-V, that a vehicle undergoes during a collision. Both delta-V and EBS can be used to measure collision severity, compare collision types and predict the potential for injury (19,20).

Campbell determined a linear relationship between vehicle crush and impact speed in full-frontal barrier crash tests using 1971–1974 General Motors cars (21). Following the determination of this relationship, a number of commercial computer programs were devised which calculate the delta-V and EBS using crush measurements, vehicle stiffness and inertial properties (22–24).

EBS is determined by equating a vehicle's crush energy to the change in kinetic energy which would occur in a collision with a fixed barrier (25,26). If the kinetic energy after the barrier impact is negligible, such as in a central impact, the EBS is simply the impact speed into the fixed barrier. In these cases

$$\text{EBS} = \sqrt{(2 \times \text{crush energy/vehicle mass})}$$

The crush energy is computed using measurements of vehicle deformation (crush), principal direction of impacting force and pre-determined vehicle stiffness values. Crush is measured at six equidistant intervals perpendicular to the original undeformed plane according to a certain protocol (27). The direction of force is determined from vehicle and collision scene inspection. Frontal and side stiffness values have been determined from staged crash tests of similar vehicles.

The autopsy reports of occupant fatalities with aortic trauma were reviewed using the centralized file of the Office of the Chief Coroner for Ontario (Toronto, Canada). Information about the severity and location of the aortic trauma and other associated injuries was correlated with the PCS data.

Results

From 1984 to 1991, 69 frontal impacts and 123 side impacts in 188 collisions causing 237 fatalities (frontal impact fatalities = 87; side impact fatalities = 150) were investigated by the U.W.O. Team. Of the 135 victims autopsied, there was documentation of aortic trauma in 39 individuals (27 males and 12 females; 29% of the autopsied fatalities) in 36 crashes (at least 19% of the total fatal collisions). The deceased ranged in age from 15 to 87 years ($21 < 40$ years; average age 44 years).

Vehicle Frontal Collisions (Fig. 1)

Seventeen deaths (13 drivers) occurred in 15 impacts. The age range was 15 to 78 years (average 44 years). The force direction was ''head-on'' (12 o'clock) in ten crashes. The Vehicle Deformation Extent (VDE) was at or beyond the windshield (i.e., ≥ 5) in at least 2/3 ($n = 10$) of the collisions. Intrusion into the occupant space was a factor in injury causation in almost all of the cases (exception—see Case 2) and chest contact with either the steering

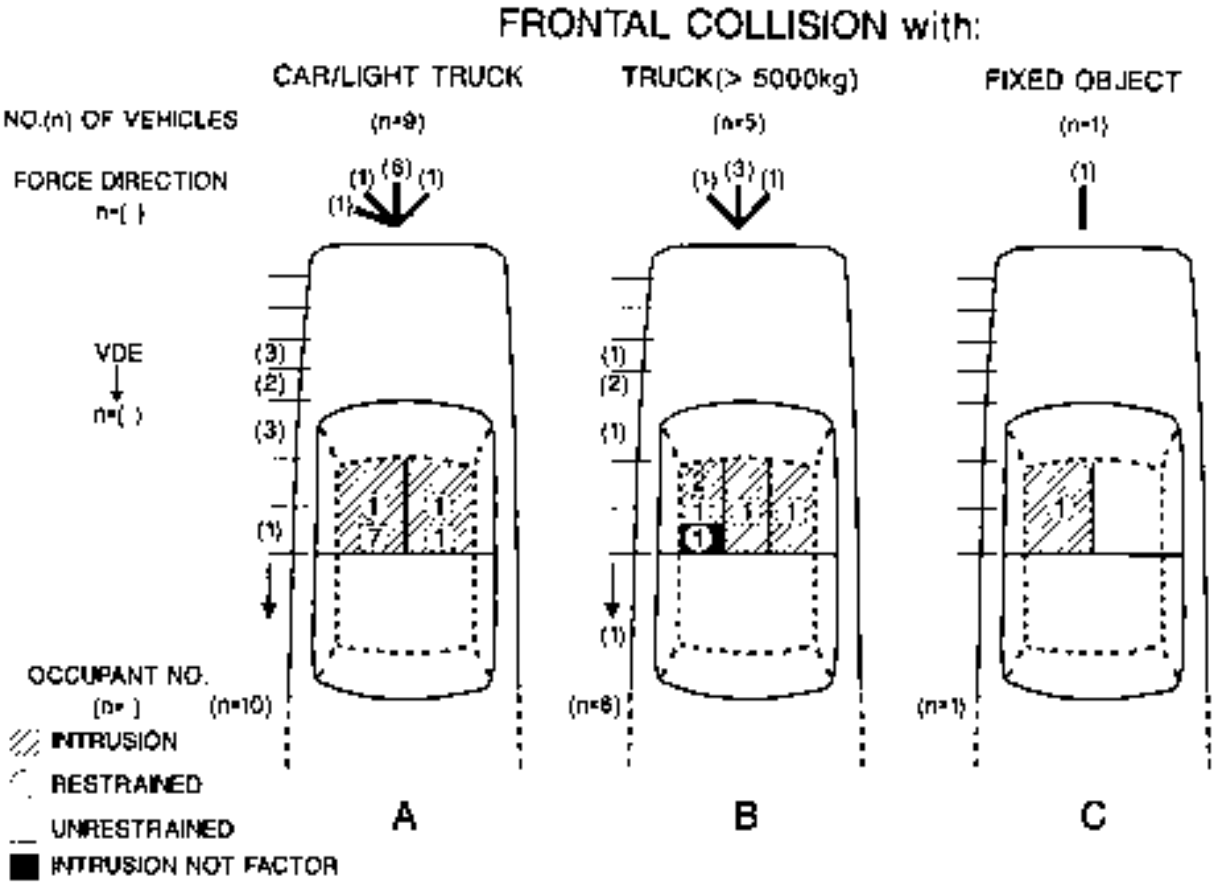

FIG. 1—*Motor vehicle (car/light truck)—frontal collision with: (A) car/light truck, (B) heavy truck, (C) fixed object. VDE = vehicle deformation extent.*

wheel by drivers or the instrument panel by passengers was noted (Fig. 2). The majority ($n = 11$) were unrestrained. Ten of the thirteen driver fatalities were associated with steering wheel rim deformation described as ''major'' or ''massive.'' In two cases, deformation was ''moderate.'' No rim deformation was noted in one impact; however, upward rotation of the steering wheel column was evident during the investigation of this impacted vehicle. The 57-year-old unrestrained driver sustained sternal and bilateral rib fractures and cardiac and aortic arch ruptures. Upward column rotation was also documented in at least three other vehicles, and steering assembly damage due to extrication by emergency personnel could not be excluded. Another one of these victims had ruptured the aortic arch and sustained sternal and rib fractures. Two others suffered chest wall fractures, hepatic, splenic and jejunal lacerations and had torn either the isthmus or descending aorta (Fig. 2).

Collisions with Car/Light Trucks (Fig. 1A)

Ten deaths (8 drivers) occurred in nine impacts. A driver and a passenger, both restrained, sustained isthmic ruptures after their vehicle collided with another car.

The EBS in seven impacts ranged from 54 km/h (34 mph) to 127 km/h (79 mph) (average 81 km/h; 50 mph) (Fig. 3). The lowest EBS (54 km/h and 57 km/h) were associated with the deaths of two 57-year-old males. The higher EBS observed were seen in victims ranging from 21 to 30 years of age.

Case 1—A 57-year-old male, a restrained right front passenger, was in a ''head-on'' collision (EBS = 54 km/h; 34 mph). Inspection of the vehicle revealed moderate intrusion (VDE = 4) and his chest had contacted the instrument panel. There was evidence that the seat was fully forward at the time of collision and seat back loading from cargo in the hatch area had occurred. A tear at the aortic isthmus and left rib fractures were found at autopsy.

Collisions with Heavy Trucks (Fig. 1B)

Six deaths (4 drivers, 3 restrained) occurred in five impacts. Two individuals in the same vehicle sustained aortic trauma (unrestrained driver—ascending aorta; unrestrained passenger—ascending aorta).

EBS values in three impacts ranged from 48 km/h (30 mph) to 86 km/h (54 mph) (average 67 km/h; 42 mph) (Fig. 3).

Case 2—A 78-year-old restrained female driver (155 cm or 5 ft 2 in; 73 kg or 160 lb), was involved in a ''head-on'' collision (EBS = 48 km/h or 30 mph; VDE = 4). There was no significant passenger compartment intrusion. The bottom rim of the steering wheel was ''moderately'' bent. The ascending aorta was lacerated.

FIG. 2—*Eighteen-year-old restrained male driver suffered complete aortic transection at isthmus following collision with heavy truck. Severe passenger compartment intrusion. Chest contact (''abrasion'') with steering wheel that shows major deformation and rotation upward. Fracture of sternum. Ribs intact. Hepatic, splenic and jejunal lacerations.*

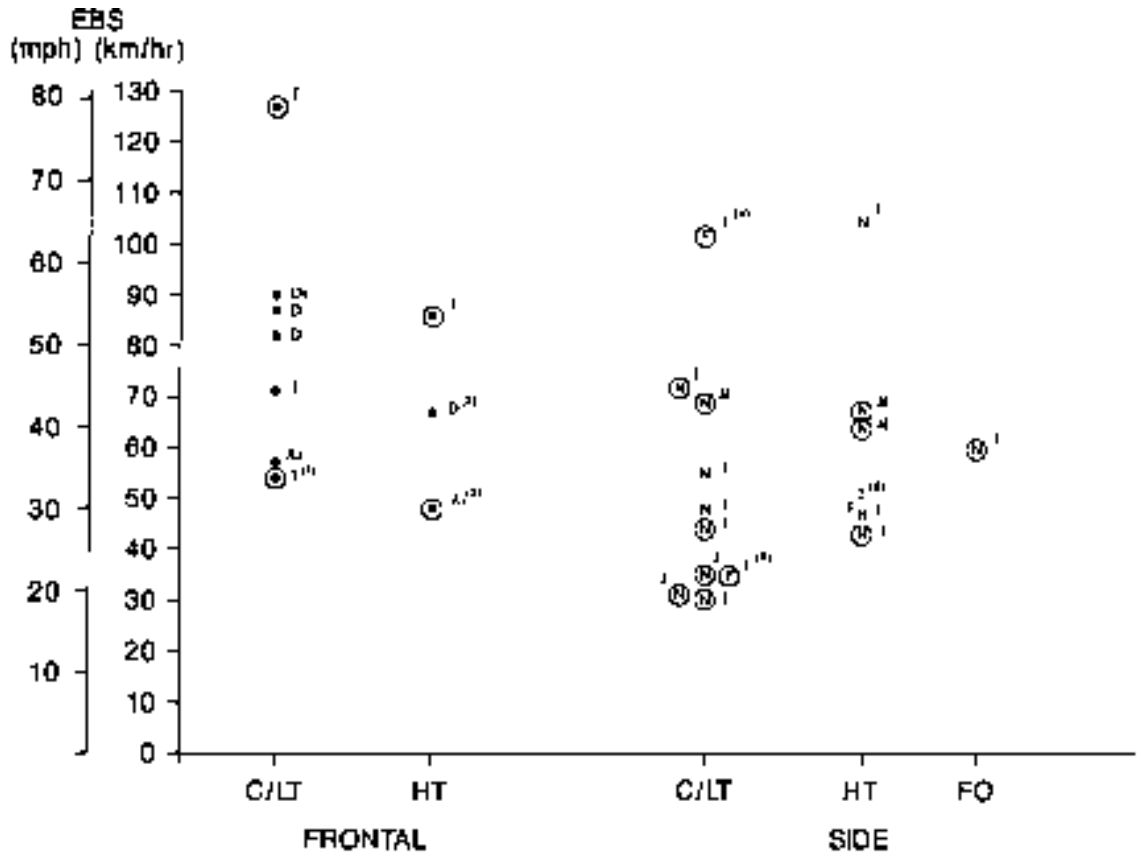

FIG. 3—*Equivalent barrier speed (EBS) available in some frontal and side collisions. Car/light truck (C/LT). Heavy truck (HT). Fixed object (FO). Restrained (open circle). Near-side impact (N). Far-side impact (F). Numbered superscripts refer to case descriptions (see Results). Lettered superscripts refer to site of aortic injury: I = isthmus; D = descending aorta, not specified; i = intimal tear; Ar = arch of aorta; As = ascending aorta; M = multiple tears.*

Other injuries included pericardial and right atrial lacerations, sternal and multiple bilateral rib fractures and atlanto-occipital dislocation. Her age-related osteoporosis and seatbelt loading (an irregular bruise was observed on the upper chest) were considered by investigators to have contributed to the thoracic trauma.

Case 3—A 15-year-old female, an unrestrained middle front passenger, was in a ''head-on'' collision (EBS = 67 km/h or 42 mph; VDE = 5). Her chest contacted the instrument panel. She suffered a ruptured descending thoracic aorta. No other injuries were found at autopsy.

Collisions with Fixed Objects (Fig. 1C)

A 44-year-old driver was killed after his car, which was being pursued at high speed, hit a tree stump (VDE unknown). The torso part of his seatbelt was under his left arm. Multiple intimal tears were seen in his thoracic aorta. Biventricular cardiac lacerations and bilateral rib and sternal fractures were also seen.

Vehicle Side Collisions (Fig. 4)

There were 22 fatalities (15 drivers) in 21 collisions (3 far-side). The age range was 19 to 87 years (average 43 years). Over half of the fatally injured occupants (13/22) were restrained. The force direction was either perpendicular (''T-bone'' or 3/9 o'clock) in nine crashes (2 of 3 far-side) or oblique (2/10 o'clock) in the rest. The VDE was up to the middle of the passenger compartment (i.e., ≤5) in 14 cases and intrusion resulted in chest contact by most victims with the side interior surface (Fig. 5) Exceptions included a 22-year-old rear middle seat passenger ejected after the rear section of his car was torn away by the impact and Case 5 (see below).

Collisions with Car/Light Trucks (Fig. 4A)

There were 12 deaths (6 drivers) in 11 impacts (2 far-side). Eight (5 drivers) were restrained.

EBS was available in nine impacts (2 fatal aortic injuries in one crash—see Case 5) and varied from 30 km/h (19 mph) to 102 km/h (64 mph) (average 54 km/h; 34 mph) (Fig. 3). The lower EBS values tended to be associated with older individuals (30 km/h—87-year-old woman; 31 km/h—77-year-old man; 35 km/h—76-year-old and 47-year-old men, see Case 5). The age range of the victims associated with the remaining EBS determinations was 25 to 54 years.

Case 4—A 54-year-old restrained female driver died following a ''T-bone'' far-side collision (EBS = 102 km/h; 64 mph). There was massive intrusion into the vehicle (VDE = 7). Autopsy findings included aortic isthmic rupture, bilateral rib fractures, hepatic and mesenteric lacerations, atlanto-occipital dislocation and right pelvic fracture.

Case 5—A 76-year-old man, a restrained front seat passenger,

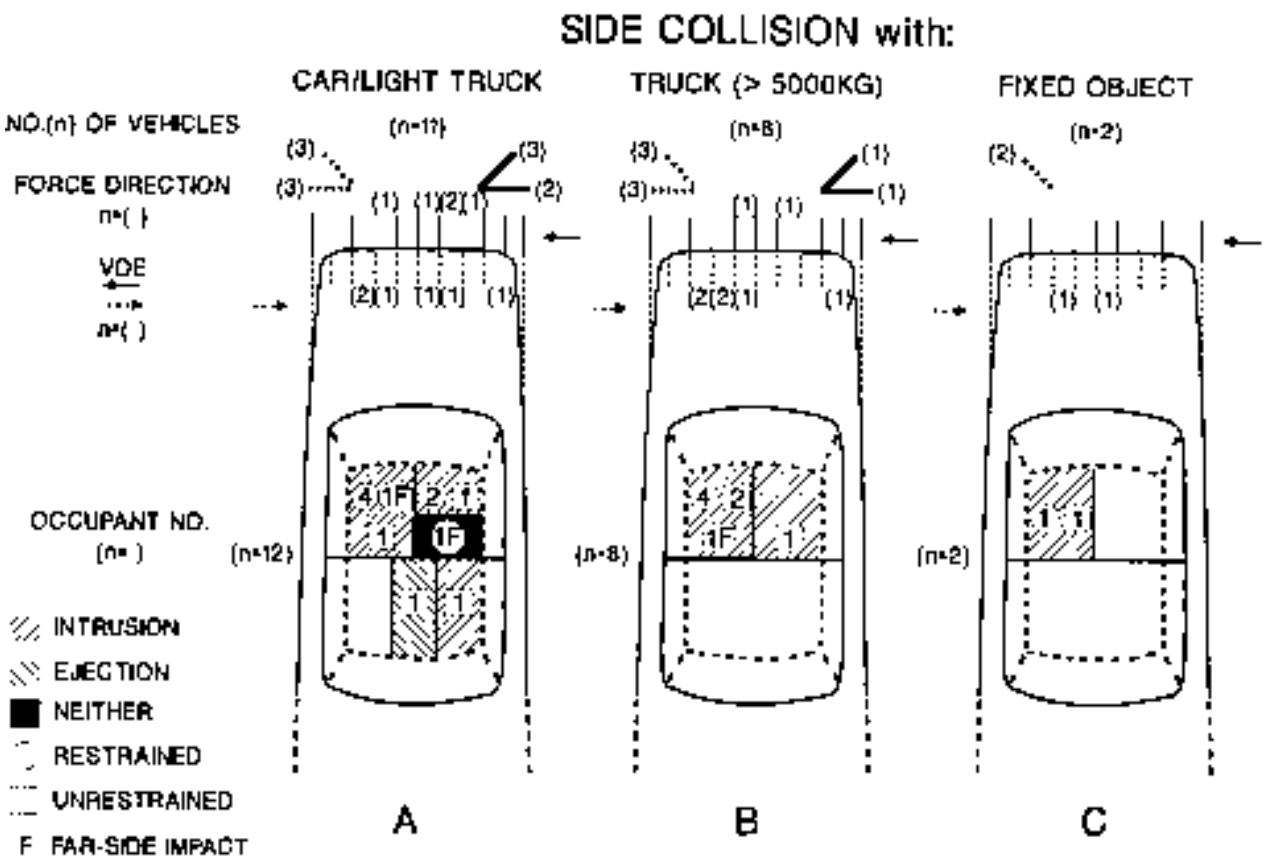

FIG. 4—*Motor vehicle (car/light truck)—side collision with (A) car/light truck, (B) heavy truck, (C) fixed object. VDE = vehicle deformation extent.*

sustained an aortic isthmic tear following an oblique far-side impact (EBS = 35 km/h; 22 mph). Intrusion was not considered a factor in injury causation (VDE = 3). The likelihood of this individual slipping out of his shoulder restraint and then contacting either the steering wheel or middle seat passenger was raised. The deceased had multiple (n = 18) left rib fractures. The 47-year-old belted driver was also killed. He had also transected his aorta at the level of the isthmus.

Collisions with Heavy Trucks (Fig. 4B)

Eight people (7 drivers) were killed in the same number of collisions (1 far-side). Four drivers were restrained.

The EBS values in six cases ranged from 43 km/h (27 mph) to 105 km/h (66 mph) (average 63 km/h; 39 mph) (Fig. 3).

Case 6—A 19-year-old unrestrained female driver suffered an aortic isthmic tear, bilateral rib fractures and hepatic and splenic lacerations after a ''T-bone'' far-side crash (EBS = 50 km/h; 31 mph). There was severe intrusion into the occupant compartment (VDE = 6).

Collisions with Fixed Objects (Fig. 4C)

Two drivers, one restrained, died. One had driven into an earth embankment (EBS = 60 km/h; 38 mph), the other into a tree.

Aortic Trauma and Associated Injuries (Table 1)

Thirty-eight of 39 individuals were dead at the scene. A 30-year-old man died of multiple traumatic injuries within 24 h of hospital admission. He had multiple intimal tears in the descending aorta but no rupture.

The cause of death, based on the autopsy reports, was either multiple trauma (9 deaths in frontal collisions; 14 in side collisions), aortic rupture (4 frontal fatality cases; 7 in side impacts) or multiple cardiovascular trauma (4 frontal impact deaths; 1 in a side collision).

Aortic and Other Major Vessel Trauma

Twenty-seven individuals sustained either rupture of the aortic isthmus (23 victims) or descending aorta. These included two restrained drivers and an unbelted passenger in heavy truck frontal collisions, five (4 drivers, 2 unrestrained, and 1 unrestrained passenger) in heavy truck side collisions and two drivers (1 unrestrained) in fixed object side impacts. Of the five cases of ascending aortic rupture, three (2 drivers, 1 restrained, and 1 unrestrained passenger) occurred in heavy truck frontal impacts and one (an unbelted driver) in a heavy truck side crash. All of the victims with multiple rupture sites were in side collisions. Two involved impacts with heavy trucks (a 75-year-old male, ascending aorta and isthmus; a 61-year-old male, isthmus and descending aorta) and the other followed a collision with another car (a 35-year-old man, isthmus and upper abdominal aorta). Multiple superficial intimal lacerations were observed in two frontal collisions, one with a fixed object and the other with another car involving an unrestrained 30-year-old driver (EBS = 90 km/h).

Tears of the inferior vena cava (superior vena cava in one case

FIG. 5—*Thirty-five-year-old restrained male driver: isthmic rupture. Oblique near-side impact with car (EBS = 69 km/h; 43 mph). Massive intrusion at driver's door. Other major injuries cardiac laceration, bilateral rib fractures with left diaphragmatic tear, splenic and mesenteric lacerations, fracture of* T_5- T_6*, tear of the upper abdominal aorta.*

also) were found in three frontal crashes. The pulmonary artery was torn in three side impacts.

Rib and Sternal Fractures

One of the occupants, an 18-year-old-restrained driver, had a sternal fracture but intact ribs (Fig. 2). Three of the four frontal collision cases with ascending aortic rupture had sternal fractures.

Of the individuals sustaining bilateral rib fractures in frontal collisions, at least six drivers had either broken the first or second ribs or both. The specified number of rib fractures in these six individuals ranged from 7 to 17 (average 12). In the rest, ''multiple'' rib fractures were seen. The side collision fatalities (including 2 far-side) with bilateral rib fractures included at least 13 occupants (9 drivers) with fractured first or second ribs or both. In these cases, and another unbelted passenger without these injuries, the number of fractures was specified as varying from 6 to 25 (average 15). Multiple fractures were present in one other victim.

Only two frontal collision deaths had unilateral rib fractures (driver, right 4 to 7; passenger, 12 fractures, left side including first and second ribs). Of the side collision cases with unilateral fractures (including 1 far-side collision), the specified number of fractures in four drivers was 1 to 7 (three with 5, 6 or 7). Two restrained passengers had either 18 (Case 5) or multiple fractures. Generally fractures were located on the side impacted except for a belted driver in a near-side impact with a fixed object.

No chest wall fractures were observed in two individuals (Case 3; also a 32-year-old-male restrained driver in a near-side collision with a heavy truck).

Diaphragmatic Rupture

Of the 12 cases, left-sided rupture of the diaphragm was specified in nine (3 drivers in frontal crashes; 5 drivers and 1 passenger in near-side impacts). Bilateral diaphragm tears were found in one driver in a near-side impact.

Cardiac Trauma

Myocardial laceration was the most common type of cardiac trauma. Evidence of cardiac contusion was seldom present. Of the three deaths with valvular tears arising from frontal collisions with other passenger cars, the tricuspid valve was specified in one case. A restrained driver in a heavy truck side collision had a mitral valve rupture. An unrestrained passenger in a side crash with another car tore mitral and tricuspid leaflets. No aortic valve ruptures were seen.

Abdominal Trauma

Hepatic and splenic lacerations occurred frequently in frontal and side impacts. Injury of the gastrointestinal tract (1 case each of esophageal, gastric and small bowel laceration; small bowel contusions in 3 deaths) was noted only in frontal crashes.

Other Fractures

Pelvic fractures were more common in fatalities in side collisions (15/22) as compared to frontal impact cases (2/17). Fractures at the following thoracic vertebral levels were observed in the

TABLE 1—*Aortic trauma and associated injuries.*

	Frontal Collisions $D(D^R):P(P^R)$* 17 = 8(5):3(1)	Side Collisions $D(D^R):P(P^R)$* 22 = 5(10):4(3)
AORTA:		
Rupture		
isthmus	5 1(3):(1)	18 4(7):4(3)
descending nos.†	4 3:1	. . .
ascending	4 1(1):2	1 1
arch	2 2	. . .
multiple	. . .	3 (3)
Intimal laceration‡		
descending nos.†	2 1(1)	. . .
Other major intra-thoracic vessels	3 2:1	3 (2):1
CHEST WALL FRACTURES:		
sternum	9 5(3):1	4 (2):2
Ribs		
bilateral	13 7(4):2	15 2(8):4(1)
unilateral	2 1:(1)	6 3(1):(2)
clavicle	4 2(1):1	4 3:(1)
Diaphragmatic tear	4 1(2):1	8 3(4):1
Hemomediastinum	6 2(3):1	10 2(4):4
Hemothorax(ces)	12 5(5):1(1)	19 5(8):3(3)
Pericardial tear	3 1(2)	6 (4):2
Cardiac		
contusion (myocardial)	1 1	1 :1
laceration (myocardial)	10 5(3):2	6 (5):1
laceration (valvular)	3 2:1	2 (1):1
Pulmonary contusion/laceration	7 4(1):2	15 4(8):3
Hepatic laceration	10 6(3):1	15 3(7):4(1)
Splenic laceration	6 2(2):1(1)	14 3(8):2(1)
Gastrointestinal injury§	6 2(2):2	. . .
Mesenteric injury	2 1(1)	3 (2):1
Pelvic fractures	2 1(1)	15 3(7):3(2)
Renal laceration	2 (1):1	4 1(1):2
FRACTURES:		
face	9 4(3):2	1 1
skull	7 4(1):2	8 3(4):(1)
Atlanto-occipital dislocation	2 1(1)	6 (4):1(1)
Spinal fractures		
cervical	1 :1‖	. . .
thoracic	6 3(1):2	4 (3):1
lumbar	. . .	. . .
Brain injury§	7 4(1):(2)	10 2(5):3
Subarachnoid/subdural hemorrhage only	3 1(2)	5 2(2):(1)
Extremity fractures		
upper	10 4(4):1(1)	4 1(3)
lower	15 8(4):2(1)	7 3(3):1

* D = unrestrained driver; P = unrestrained passenger; (D^R) = restrained driver; (P^R) = restrained passenger.
† Not otherwise specified.
‡ Superficial tear without rupture.
§ Injury = contusion/laceration.
‖ C_1-C_2 by radiographic examination.

frontal collisions: T_2/T_3 (2 cases, ascending or descending aorta); T_4/T_5 (1 case, isthmus); T_5/T_6 (1 case, descending aorta); T_6 (1 case, ascending aorta) and ''upper thoracic'' (1 case, ascending aorta). All four side collision cases had isthmic tears: T_1 and T_5/T_6 (3 cases). No lumbar spinal fractures were observed.

Extremity fractures were relatively less frequent in side collisions. In three cases, fractures were found opposite the side of impact. None of the far-side collisions had documented extremity fractures.

Discussion

A spectrum of aortic injuries can result from blunt trauma. Hemorrhage can be seen in the intima or periaortic soft tissue (16). Lacerations beginning in the intima can extend into or through the media and adventitia leading to rupture (13,16,28,29). Aneurysms formed are dependent on the tensile strength of the media and adventitia (16,28–30). In the present series, 37 cases of rupture and two deaths associated with intimal lacerations were studied. Three of these cases involved multiple ruptures. In other studies of blunt aortic trauma, the frequency of multiple ruptures varies from 1% to about 25% (7,13,14,16,31,32).

The isthmus is the area of the descending thoracic aorta in the region of the ostium of the left subclavian artery and this ''classic site'' was the most frequently injured (at least 5/17 frontal collisions and a tear noted in the descending aorta in 6 other fatalities; 21/22 side impacts including all cases of multiple rupture). Other reviews of aortic trauma occurring in motor vehicle occupant fatalities have shown a similar site predilection despite its relatively protected location (5–8,12–14,16,28,29,31–37). Some series have observed up to almost 90% of traumatic aortic ruptures in the isthmus area (2). Another frequently injured location is the ascending aorta, above the aortic valve cusps, observed in four frontal collisions in the present series (5,7,12–14,16,28,31,32). Other injured sites observed have included the descending thoracic aorta below the isthmus and at the diaphragmatic opening, the aortic arch and the abdominal aorta above and below its bifurcation (5,7,12–14,16,31,38,39).

Various proposed mechanisms of injury incorporating elements of chest crushing-compression, deceleration, increased intravascular pressure and/or hemodynamic forces have been advanced to explain the development of aortic tears (7,16,28,33,38–43).

Predisposition to Injury

The aortic isthmus incorporates the ligamentum arteriosum, the scarred remnant of the fetal ductus arteriosus, which attaches the aorta to the pulmonary artery. *The isthmus is considered to be inherently weak* (29,40,41,44,45). The isthmus is less resistant to tension strain, particularly from dynamic longitudinal stretching as could occur during a motor vehicle collision (41,46,47). This is supported by the observation, during either experimental testing or postmortem examinations, of aortic intimal tears which are typically transverse, consistent with longitudinal shearing forces (5,7,11,13,14,16,28,29,31,38,46–48).

The role of disease weakening the aorta and predisposing to traumatic rupture has been debated. Although atherosclerosis was sometimes documented in the autopsy reports reviewed in this study, it was not mentioned as a factor, an observation supported by others (5,13,14,35). Other types of vascular pathology that can weaken the media (e.g., cystic medionecrosis, syphilis) have also been considered to be noncontributory to rupture (35). In contrast, other clinical and experimental studies have linked the presence of disease, particularly severe atherosclerosis and cystic medionecrosis with aortic laceration in some cases (16,38,40,49,50). Underlying pathology should, therefore, be excluded by gross and microscopic examination (16,40).

The tensile strength or elasticity of the aorta decreases with age (44,47). The increased susceptibility of the aorta to traumatic laceration because of aging was observed in a study of 105 vehicular fatalities (6). Strassman, however, noted a wide age distribution

in his series and concluded, as others, that traumatic aortic laceration occurs independent of age (13,14,32). More than half (42/70) of his subjects were less than 50 years old and free of aortic pathology. The remaining older subjects had varying degrees of atherosclerosis. In the present series, the age range was 15 to 87 years (average 44 years), comparable to other studies (8,16,31,32). Although 60% (23/39) of victims were less than 50 years old, older victims were seen in frontal and side crashes with the lowest determined EBS values. Only one adolescent, aged 15 years (Case 3), was found highlighting the relative infrequency of this injury in younger age groups, i.e., $<$ 16 years (36,42,51). Of 551 children and adolescents dying accidentally, 12 (2.1%) ranging in age from 3 to 15 years had aortic rupture (36). Five of these victims were motor vehicle occupants. In Strassman's series, four were less than 20 years of age including two under 10 years (13). A number of factors could account for the infrequency of aortic rupture in the young. Significant atherosclerosis is absent in this age group. Greater chest wall elasticity could cushion the internal viscera from trauma (42,51). A child is less likely to sustain localized chest trauma, either as a pedestrian or motor vehicle passenger (i.e., striking a steering wheel) (36,42). Nevertheless, the severity and pattern of associated injuries in children are comparable to adults which suggests similar injury mechanisms (36).

Deceleration—The Absence of Chest Wall Fractures

When one part of the body is decelerated at a different rate from another, the connections or points of fixation between them are stressed or sheared proportional to the differences in deceleration rates (48). Parts of the aorta are variably mobile and points of fixation exist which are potentially stressed by blunt trauma (5,13,16,28). The isthmus connects the descending aorta, bound to the spinal column by fascia, pleura and intercostal arteries, and the relatively moveable proximal aorta (29,38,40,42,43,49). Alternatively, the ascending and distal descending parts of the aorta have been considered to be mobile and the proximal descending aorta or the aortic arch tethered by the major aortic arch vessels (7,16,35–37,39,41,52). The ascending aorta between the heart and arch, the descending aorta at the diaphragmatic hiatus and the abdominal aorta proximal to its bifurcation are other fixation sites (5,16,38,41).

Aorta injuries have been associated with deceleration which has been either vertical (cranial or caudal direction, e.g., falls) or horizontal (anterior or posterior, e.g., frontal or rear-end motor vehicle collisions) (16,28,29,33,40,43,45). Sudden forward displacement of the lung hilum can tense the parietal pleura, compressing the aorta against the spine resulting in tearing of this vessel (49). A motor vehicle occupant thrown forward and upward sustaining head impact will experience cranially directed deceleration which can tense the aortic arch and induce tears of the descending aorta (isthmus) (28). Case 1, a frontal collision, also involved seatback loading of a front seat passenger. Backward jerking of the head (''whiplash effect'' or ''traction effect'') in a rear-end collision can stretch cervical vessels and exert traction on the aortic arch (38,40,41). Notably, the two cases of aortic arch tears observed in the present study were associated with upward rotation of the steering column. Lateral decelerative forces and compression arising during side collisions are also recognized in the causation of aortic injury (7,29,38,45,48,53).

Severe thoracic compression or impact as indicated by chest wall trauma or fractures may not be necessary for internal injury to occur (5,13,28,29,31,32,44,45,48,54). In some cases of aortic rupture, no other significant injuries will be found as observed in Case 3 (7,35). In Hossack's study of 198 cases of aortic rupture in road crash victims, about 1/4 had no documented chest wall damage and 2% had no other injuries (32). This suggests that horizontal deceleration with resulting shearing forces alone could cause aortic laceration (5,28,29,31,45,54). Whether identifiable evidence of concomitant chest wall trauma will occur is dependent on the magnitude and direction of the external force striking the thorax, the area over which the force is distributed and the elastic resistance of the rib cage (40).

''Viscous tolerance'' could explain the lack of chest wall fractures in some individuals with aortic rupture (11,55). As the speed of deformation (velocity change per time) increases, parts of the body are not as tolerant of compression or deformation compared with other sites because of the variable viscous nature of the tissues and organs (11,55,56). Minimal thoracic wall trauma but severe aortic injury has been seen resulting from rapid organ/tissue deformation in the experimental setting of high frontal or lateral impact velocity (55,56).

The absence of thoracic injury could relate to the age of the victim. Younger individuals have more flexible rib cages that are less prone to fracture (e.g., Case 3—15-year-old; 32-year-old in a side collision in the present series) (1,3,13,37,57–61).

Chest impact centered above or at the mid-sternum of rabbits or dogs, respectively, can cause cardiovascular injuries but few rib fractures (present series—an 18-year-old restrained driver, Fig. 2) (50,62). A ''shoveling'' mechanism has been proposed by which cranially and posteriorly directed deceleration (i.e., vertical and horizontal forces) or displacement of mediastinal structures from abdominal and lower chest wall impact can stretch fixation points of the thoracic aorta, i.e., the isthmus, but not necessarily result in chest wall fractures (9,28,31,57). Voigt and Wilfert observed that, following frontal collisions, unrestrained drivers and passengers in vehicles with a deep leg recess and high instrument panels allowing knee contact with the firewall sustained isthmic tears from impacting the lower edge of the steering wheel or slipping under the instrument panel respectively (9,57). Aortic tears would be expected to be proximal to the ligamentum arteriosum under these circumstances although the large majority of the Voigt and Wilfert series were distal (28,31,57). Associated tears of the inferior vena cava can occur and, on occasion, the ascending aorta ruptures (57). ''Shoveling'' has also been observed in frontal oblique collisions involving passengers sustaining isthmus tears (9). In the present series, injury of the gastrointestinal tract was observed in six frontal collisions but in no side impacts. Only drivers, not passengers, sustained this type of injury in Greendyke's series (5). Five patients in Verdant's series of aortic trauma had evidence of abdominal trauma only (29). In the present series, 10 of 12 diaphragm tears were seen in drivers. Diaphragm rupture, an indicator of a sudden increase in intra-abdominal pressure, was observed most frequently on the left side. In another series, rupture of the diaphragm, more commonly on the left side, was seen in 10 of 21 traumatic tears (14). Herniation of intra-abdominal organs into the left chest cavity could shift the lung hilum superiorly exerting traction on the aortic arch leading to isthmic rupture (41).

The ability of a steering wheel to absorb the energy of chest impact will determine the severity of internal thoracic injury seen in a driver (63). The extent of steering wheel disruption has been linked with severe thoracic injuries (1). In the present series, significant steering wheel rim deformation was seen in most of the driver fatalities resulting from frontal crashes.

Chest Wall Compression

Vertebral fractures can tear the descending thoracic and abdominal aorta (5,7,13,16,28,29,31–33,40). In Hossack's series, forcible hyperextension fractures from the level of T_2 to T_9 were seen in 8% (16/198) of road traffic deaths with aortic rupture (32). The T_2-T_3 vertebral level was most commonly associated with aortic laceration (32). In the present study, upper and mid-thoracic fractures likely reflected forces acting at this level rather than causing direct injury to the aorta.

At maximum compression, the sternum can approximate the spinal column as supported by experimental studies and can lead to cardiac displacement (30,64–67). Cardiac displacement posteriorly, downward and laterally into the left chest cavity has been demonstrated by radiographic imaging in dogs sustaining sternal impact while held stationary on a rigid frame (65–67). This resulted in the aorta being lengthened and stretched in a craniocaudal direction in these subjects leading to transverse aortic tears (65,67). Cardiac displacement, therefore, can stress points of fixation (e.g., isthmus, ascending aorta) leading to tearing (28,29,31,38,40,57). Jagged anterior chest wall fractures can directly lacerate the underlying ascending aorta (32,35,68). Ascending aorta tears have a frequent association with cardiac injuries (3,16).

Aortic trauma has been linked with fractures of the clavicle-first rib (6,69). In theory, if sufficient energy can cause these bones to fracture, then associated intrathoracic injuries are possible (69). But reviews of the literature show no clear-cut relationship between rib/sternal fractures and the nature of the underlying cardiovascular injury. Their presence has little predictive value in indicating whether aortic rupture has occurred (39,69). The probability of aortic injury is the same whether upper or lower rib fractures are present (69). In the present series, although first and second rib fractures occurred frequently, they were not observed in all cases. ''Pinching'' of the aorta between the spine and superior anterior bony thorax (manubrium, clavicle, first rib) by abrupt chest compression has been proposed as a cause of isthmic lacerations (70).

Chest wall fractures have been considered a function of thoracic deflection (59). Viano described the concept of the ''thoracic stability limit'' (64). Below this limit the chest wall is structurally stable and protective of internal organs. Relatively few rib fractures will be caused by chest compression. Above this threshold, the thorax is unstable and prone to collapse by a cascade or ''avalanche'' effect as manifested by extensive fracturing of the remaining intact ribs. This is potentiated in the older individual. Based on human cadaver studies in frontal impacts, the limit is exceeded once more than six or seven rib fractures occur. In the present study, most individuals in frontal and side impacts had a minimum of five to seven rib fractures which would support this experimental concept. With chest wall collapse, the potential for intrathoracic organ and vessel injury is then substantially increased; however, the injury severity varies and is not necessarily correlated linearly with the degree of chest compression, which may be about 1/3 or more of the chest dimension (range of 3 to 4 in. or 7.5 to 10 cm or more) and can occur in either frontal or side impacts (1,44,60,64).

Chest Wall Compression and Deceleration

Although deceleration alone has been thought to account for some cases of aortic rupture, this study and others have documented a high frequency of rib/sternal fractures consistent with chest compression (5,7,12–14,16,31,52). *Of the various blunt trauma injury mechanisms proposed in frontal and side motor vehicle impacts, the favored theories combine features of rapid deceleration and chest compression* (3,7,8,16,28,31,40,43). Sudden decelerative displacement of the heart during chest compression, e.g., due to steering wheel impact, results in aortic fixation points, particularly the isthmus, being subject to torsion, shearing and bending stresses (30,43,65). Torsional forces are exerted on the aortic root, i.e., ascending aorta; shearing and bending occurs at the isthmus (30,41,43,71). Overbending of the aortic arch, due to the mass inertia of the heart, over transverse hilar structures could lead to stretching at the isthmus (33,40).

Increased Intravascular Pressure and Hemodynamic Forces

A transient excessive elevation of blood pressure is another explanation as to how the aorta ruptures, particularly in young victims without chest wall fractures (13,33). Whether a pulse of sufficiently high intravascular pressure can be generated by chest impact or deceleration is a mechanism debated in the literature (7,28,38,40,41,45). High pressures observed in aortas or aortic segments experimentally inflated with air, water or blood have been associated with rupture (35,39,41,46,57). However, in animal models, there is disagreement whether enough pressure rise can occur following chest impact and, if it does, whether it is the sole mechanism of rupture (50,62,65). Chronic hypertension has been considered as a predisposing cause of aortic rupture (37). Dogs, hypertensive by pharmacologic means and subjected to sternal impact, were not more likely to sustain vascular tears (62,65). Vessel tears have even been observed in dogs rendered hypotensive (66). Some studies have used human cadavers usually representing older age groups. These subjects are not physiologic (e.g., lack of pulsatile blood flow) and undergo changes in soft tissue consistency (11,60,63). In one study, cadavers, subject to mid-sternal impact velocities ranging from 17.3 to 30.9 mph (27.7 to 49.4 km/h) and chest deflections up to 3 to 4 in. (7.5 to 10 cm) resulting in intra-aortic pressures greater than 1000 mm Hg, suffered no aortic rupture (60).

The pressure rise could be localized by sudden compression or trapping of part of the aorta displacing noncompressible blood and expanding the adjacent undilated aorta and tearing sites of weakness (16,37,38,40,41,46,47). Torsional stress exerted at the base of the heart results in a pressure wave of blood that is transmitted to the isthmus where rupture can happen because of shear stresses (30). Tears of the ascending aorta are sometimes associated with aortic valve rupture (31,68). Aortic valve rupture could result from increased ascending aortic pressure acting on the closed valve in diastole resulting from temporary occlusion of the proximal ascending aorta due to compression against the sternum (68). No aortic valve tears were seen in the present series. Studies on monkeys have shown trapping and occlusion of the descending aorta between the posterior ventricular wall of the heart and spinal column following anterior chest impact (72).

A ''waterhammer'' or ''hydraulic ram'' effect has been described (41,44). Following a frontal collision, sudden forward inertial deceleration of blood in the aortic arch impacts the anterior wall of the ascending aorta and exerts traction on the isthmus leading to rupture (41,43,71).

Cases of multiple aortic ruptures have been attributed to a sudden rise in aortic pressure leading to more than one site of tearing (52).

Vehicular Factors

Effectiveness of Restraint Systems—In frontal collisions, unrestrained drivers can contact the steering wheel and passengers can

strike diverse components, including the instrument panel (1,3,9,31,73). In near-side collisions, restrained and unrestrained occupants contact the intruding door and other side structures, an impacting vehicle or a fixed object (1,7,73–75). In far-side collisions, occupant contact can occur with vehicle front structures (e.g., instrument panel, windshield) and other occupants, which can contribute to chest wall injury (74). In far-side impacts a restrained occupant's upper body can escape from the shoulder belt (76,77). Case 5 involved a 76-year-old passenger who slipped out of his seatbelt following a far-side impact. Notably, his driver also sustained an aortic rupture of the isthmus. Greendyke also observed aortic tears in the deceased occupants seated in the same vehicle (5). Contact with intruding side structures and doors is frequently associated with pelvic fractures (74,75). In this study, pelvic fractures were observed in 15/22 side collisions.

Previous reviews of aortic trauma occurring in frontal collisions have noted low seatbelt usage, implying that increased utilization would reduce the incidence of this injury, particularly in frontal collisions (7,36). Although about 1/3 ($n = 6/17$) of the frontal impact deaths in the present study were restrained, intrusion was a major factor in these cases resulting in occupant contact and consequent injury (73). Although injury severity can be related to intrusion, intrusion can be associated with varying degrees of injury (78). In a prior U.W.O. study of restrained passenger car occupant fatalities, there was a disproportionate number of crashes with either a heavy truck or fixed object leading to occupant compartment intrusion in both frontal and side impacts (73). In the present study, 7/17 frontal and 10/22 side collisions were with a heavy truck or fixed object. Green et al. also stated that side collisions predominated among the restrained occupant fatalities (in present study, 13/22 side collision fatalities restrained; 6/17 frontal impact deaths restrained), reflecting not only the more effective protection offered by seatbelts in frontal collisions but also the greater likelihood of contacting intruding surfaces in near-side impacts (7,53,73,75,78). Severe intrusion was also evident in two of three far-side collisions (Cases 4 and 6), a factor occasionally mentioned in other studies which include far-side impacts (7,74).

Despite the prominence of ejection in Greendyke's series and others (13/25 drivers and passengers), only one such case, in a side collision, was observed in the present series (5,29). Other data have suggested no significant association between ejection and traumatic aortic rupture (6).

Seatbelt loading can cause chest injury and rarely aortic rupture in frontal and side collisions (58,74). In Hill's series, 1025 of 3276 restrained front seat occupants suffered injuries of varying severity caused solely by their seatbelts (58). Twelve fatalities were attributed to seatbelt loading. Three cases in this series suffered aortic rupture. One of the case descriptions was similar to Case 2 of the present study. A 75-year-old-woman of ''average height'' was in a frontal collision (delta-V estimated to be 18 km/h or 11 mph) and sustained sternal/right rib fractures and lacerations of the right ventricle, liver and aorta (58). In the previous U.W.O. study, four elderly motor vehicle occupants had fatal chest injuries attributed to seatbelt loading (73).

Improperly worn restraints, e.g., a lap belt slack or improperly positioned on the abdomen, and seatbelt misuse can contribute to lumbar vertebral fracture and abdominal aorta rupture (79). In the present study, a 70-year-old woman whose restraint showed ''too much slack'' sustained a torn descending aorta following a collision with a heavy truck. A 44-year-old man, a driver in a frontal collision with a tree, developed multiple aortic intimal tears. The torso part of his belt was under his left arm.

Impact Speed—Aortic trauma has been linked with motor vehicle crashes involving excessive speed (5,29,31,36,52,80). Although lowering the highway speed limit from 70 mph to 55 mph has been associated with a reduction of driver and passenger fatalities, morbidity and some types of severe chest injury, this initiative did not have any significant effect on the frequency of cardiac and aortic injury in one reported study (81). Motor vehicle fatalities including cases of aortic rupture occur not only on highways but also in the lower-speed-limit zones of city roads (4,6).

Impact speeds are either not available or estimated in police reports following scene and vehicle investigation and witness statements (6,82). Hudock and Imajo described four cases of aortic rupture in motor vehicle crashes occurring at estimated speeds of 40 to 45 mph (64 km/h to 72 km/h) (82). Charles et al. described a 56-year-old male driver who sustained a partly lacerated ascending aorta during a head-on car crash with a tree at an estimated speed of 35 mph (56 km/h) (68). Impact speed of a single vehicle, however, is not a good indicator of collision severity.

In general, the delta-V a vehicle experiences during a collision will depend on the closing velocity at impact, the inertial properties (e.g., mass ratio) of the colliding vehicles, and the location and direction of the impact forces. The closing velocity depends on the impact velocities of both vehicles. For example, a rear-end collision involving the front of a bullet vehicle traveling 120 km/h which strikes the rear of a target vehicle going 110 km/h in the same direction results in a closing velocity of just 10 km/h. Although the impact speed of the bullet vehicle is high, the rear-end collision would actually be very minor. The ratio of the colliding vehicles' masses plays an important role in collision severity and the delta-V experienced. In a collision between a large vehicle and a small vehicle, the smaller vehicle will experience the greater delta-V. For example, when the colliding vehicle mass ratio is 2 to 1, the small vehicle would experience twice the delta-V of the large vehicle. In the present series, an over-representation of heavy truck and fixed-object collisions was observed. The location and direction of the resultant impact force can also be significant. For a given closing velocity and mass ratio, the maximum delta-V will occur in a central collision when the line of action of the impact force passes near or through the vehicles' mass centers. A golfer will relate to the greater delta-V and distance achieved when a golf ball is stroked squarely. Similarly in a motor vehicle collision, a central impact between two colliding vehicles at a given closing velocity results in the largest delta-V.

Delta-V calculations by Newman and Rastogi in five cases of descending aortic laceration (frontal and side impacts not specified) determined an average of 35.4 $\pm$14.5 mph (56.6 $\pm$23 km/h) (8). Two cases of ascending aorta tears had delta-V values of 33.1 mph (53 km/h) and 50 mph (80 km/h) (8). In the present study, the range of EBS for isthmic and descending aorta tears occurring in frontal collisions was 54 to 127 km/h (34 to 79 mph) (average, 84 km/h; 53 mph); in side collisions, 31 to 105 km/h (19 to 66 mph) (average, 52 km/h; 33 mph).

Superficial intimal tears are less frequently seen at autopsy (14,16,31). In our series, their presence did not indicate a less severe collision. They were observed in two collisions, one at EBS = 90 km/h and the other also of high severity.

Impact Direction—Aortic rupture occurs in frontal and side collisions (6,7,29,53). It is seen not only in head-on (force direction 12 o'clock) and right-angled crashes (force direction, 3 o'clock or 9 o'clock) but also in collisions involving obliquely directed forces causing shearing stresses (7–9,28). In the present series, 5 of 15

frontal collisions and 12 of 21 side impacts involved obliquely directed forces. In side collisions, although injuries are expected to occur on the side impacted, the higher frequency of bilateral (15/22) compared to unilateral (6/22) rib fractures in our study could indicate the asymmetric nature of the forces arising during this type of collision (53). Lateral impacts are associated with secondary rotatory and forward velocities (54). This can also happen in far-side impacts and aortic trauma can result (7,53,54). Bilateral rib fractures in two far-side impacts, in the present study, suggest a degree of forward chest compression.

Factors Influencing Survival

In the present series, all fatally injured occupants were dead at the scene except for one individual, who lived about 24 h. Although a significant number of individuals suffering traumatic aortic rupture can survive more than 1/2 h following a vehicle collision, a number of factors will decrease survival (4,6,7,12,13,15,16,32,33). An isthmic tear is more likely to be associated with initial survival since it can be temporarily sealed by adventitia and pleura (12,13,31,33,52). Ascending aorta lacerations are not invested by pleura and more likely to be associated with cardiac injuries (39,71). Victims dead at the scene have other more severe and life-threatening injuries than those individuals hospitalized (12,80). Cases of traumatic aortic rupture frequently have other injuries which can cause death (4,5,14–16,31). Injuries at these other sites can result in aortic trauma being overlooked in the clinical setting (3,5,12,13,16,29,35,39,52,71). Based on the cause of death assigned in autopsy reports, almost three quarters of the deceased had other life-threatening injuries. For example, in the present series, a significant number of occupants had life-threatening craniocerebral trauma (7/17 frontal; 10/22 side) (7,8,12,29). Not only can there be a lack of external or bony chest trauma to alert medical personnel of the possibility of aortic injury, but also signs and symptoms can be delayed (15,16,39,40,42,44,52,71). The rarity of aorta trauma in children can mean that this injury may be missed in this age group (36).

Conclusion

Motor vehicle collisions are the major cause of traumatic aortic injury with its life-threatening consequences. The present study has combined autopsy information with the associated vehicle collision and occupant kinematic events to identify the likely injury mechanisms. Multiple factors are usually considered in assessing the risk of aortic rupture in a motor vehicle occupant involved in a crash.

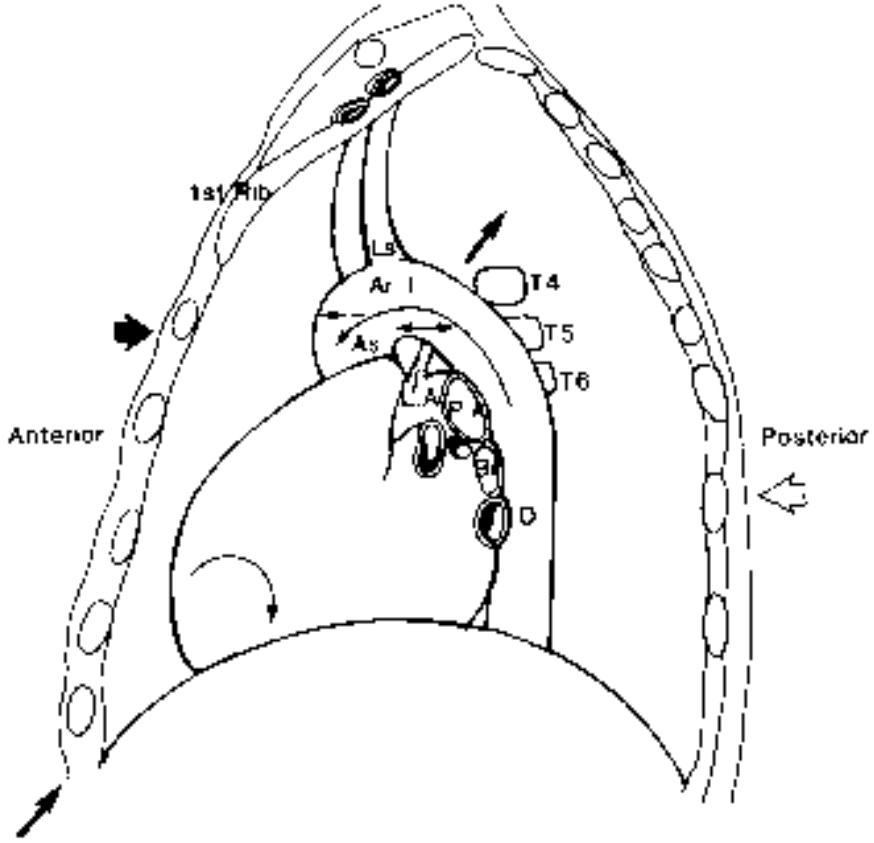

FIG. 6—*Some proposed mechanisms of traumatic aortic rupture in motor vehicle collisions. Left aspect of mediastinum. Largest solid arrow—sternal fracture from anterior chest compression directly injuring ascending aorta (As). Largest open arrow—posterior compression causes vertebral fracture tearing the descending aorta (D). Solid arrows (angled at 45°)—''shoveling'' mechanism leading to isthmic (I) tears by displacement and deceleration of mediastinal structures cranially and posteriorly. Cranially directed forces can also lead to stretching of the major aortic arch vessels (left subclavian artery = Ls) and consequent traction on the arch (Ar). Chest compression/deceleration: curved arrow on heart—torsion resulting in twisting of aortic root, i.e., ascending aorta; curved arrow on aortic arch— ''bending'' or stretching due to cardiac displacement thereby stressing isthmus; double-headed arrow—shear stresses focused on aortic isthmus. Horizontal interrupted arrow—''waterhammer'' effect following frontal collision with force exerted on ascending aorta resulting in stretching of isthmus. LA = ligamentum arteriosum. PA = pulmonary artery.* T_4, T_5, T_6 = *thoracic vertebrae. Br = left bronchus.*

Age of the victim, the presence of underlying vascular disease, anatomic and physiological features related to the heart, aorta and circulation, thoracic wall and other associated injuries, the type and severity of the impact, the direction of decelerative forces experienced by the victim, occupant compartment integrity, occupant contacts and the restraint system can play roles in the causation of this injury. Review of the literature details numerous mechanisms attempting to explain the development of aortic rupture due to nonpenetrating blunt chest trauma (Fig. 6). Our study supports the predominant impression in the literature that sudden chest compression/deceleration is the major injury mechanism inducing torsional and shearing forces that result in transverse laceration and rupture of the aorta, most commonly in the inherently vulnerable isthmus region. Our vehicle crash analysis reveals severe collision forces with intrusion into the occupant compartment in most cases. From this study and others, it is evident that efforts to reduce the incidence of life-threatening injury to restrained passenger car occupants must include diminishing the severe intrusion forces that impinge directly on vulnerable human tissues.

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