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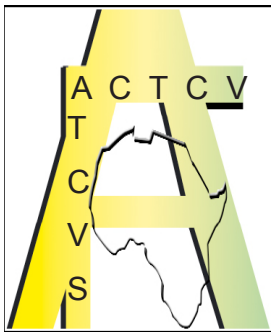

CHIRURGIE CARDIAQUE/CARDIAC SURGERY

BLUNT THORACIC AORTIC INJURY (BTAI) : ADVANCES IN THE ERA OF INNOVATION. A REVIEW (PART 1)

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ABSTRACT

Evolving technology and its applications have improved the diagnostic and therapeutic approaches in managing blunt thoracic aortic injuries (BTAI) over the last two decades. Case reports, prospective and retrospective reviews, technical advances and statistical meta-analyses have all contributed towards a better understanding of this potentially lethal injury. Yet consensus has not been reached re. timing of intervention or treatment options. Level one and two evidence based data is not yet available re. BTAI.

This review summarizes the incidence, natural history, initial evaluation, diagnostic capabilities, and the therapeutic options, including delayed management strategies, operative aspects of open surgery/ spinal cord protection, and emerging endovascular techniques/approaches.

Introduction

The World Health Organization (WHO), in 2002, reported more than 1.2 million deaths and 50 million injuries globally from road-traffic injuries (RTI)[1]. The resultant global cost was estimated >$500 billion. By 2020, the WHO estimates that 60 to 80% of the future deaths, injuries, and cost from RTI will occur in low and middle income countries (LMIC)[1]. Further, the second leading cause of global mortality in the 15-44 age group is RTI. The incidence is not balanced as evidenced by mortality rates of 15/100,000 population in the USA, 51/100,000 in other high income countries, and 89/100,000 in LMIC[2].

Overall thoracic trauma mortality from all causes (blunt, penetrating, blast) is estimated >10%[2]. With an annual incidence of 7,000-8,000 new cases in North America, blunt thoracic aortic injury (BTAI) is highly lethal[3,4]. Autopsy studies reveal that BTAI is the second most common fatal blunt injury accounting for 21 % of all deaths from road traffic accidents[3,4,5], with head injuries the major cause of death[6]). Untreated or unrecognized, the estimated mortality at the scene averages 85%, and, of the survivors, 30 % will die within the first six hours after admission, and 55 % within twenty four hours, with an average in-hospital mortality of

1 % per hour for the initial 48 hours[3,4,5]. In the USA, an average of 2.5-14.3 cases of BTAI are seen per year at the major urban or regional trauma centers, with in hospital mortality of 10-20%[7,8,9].

BTAI has been well studied over the past 50 years. Yet a unified consensus re.evaluation, diagnosis or treatment has not been fully achieved. Emerging diagnostic modalities, especially CT scanning, and therapeutic approaches that include the importance of blood pressure control, delayed operative management, spinal cord protection with open repair, and endovascular stenting, continue to generate debate and evolution[4,6-15].

The American Association for the Surgery of Trauma reported a dramatic change in diagnosis and treatment of BTAI in a comparison of prospective multicenter center studies from 1997 and 2007 (table 1)[7,8]. During the intervening 10 years spiral or helical CT scanning has become the primary diagnostic screening modality, with an average of 2 days delay in treatment, endovascular stent-grafts emerging as the primary therapeutic modality, and overall decrease in hospital mortality, despite the lack of Level 1 or 2 evidence based data From J. Bone & Joint surg. 2003;85A:2).

Table 1 : Diagnostic and treatment

Diagnosis to repair	16.5 h	54.6 h
Diagnosis		
Aortogram	87%	8.3%
CT scan	34.8%	93.3%
TEE	11.9%	1%
Treatment		
Method of repair		
Open repair	100%	35.2%
Clamp/sew	35.2%	16.2%
Bypass	64.7%	83.8%
Endovascular	0	64.8%
Paraplegia		
Open repair	8.7%	1.6%
Clamp/sew	16.4%	2.9%
Bypass	4.5%	3.5%
Endovascular	0	0.8%
Complications		
Pneummonia	32.9%	32.6%
Renal Failure	8.7%	8.8%
Repair site complication	0.5%	13.5%
Open repair	0.5%	1.5%
Endovascular	0	20%
Mortality	22%	13%

Definition/Historical Aspects

Injury or trauma is considered as damage to the body caused by an exchange with environmental energy that is beyond the body's resilience. Acute blunt thoracic aortic injury is defined further as external or internal forces causing traumatic dehiscence of all, or part of the thoracic aortic wall[16]. The time period of acute injury is within 14 days, beyond which they are classified as sub acute or chronic[16]. This is the time period within which a periaortic hematoma liquefies and expands, and is most susceptible to rupture. Pate et al[17] classified injuries :

I. Acute- less than 8 days after injury, with: A. Continuing free hemorrhage, or B. Periaortic hematoma contained within the mediastinum.

II. Chronic- pseudo aneurysm present > than 1week after injury. Fattori et al[18] further classified BTAI as acute < 14 days, subacute 14 days to 3 months, and chronic >3 months.

Vesalius, in 1557, first reported BTAI in a victim who fell off a horse[4,5,12]. Strassman in 1947 reported an autopsy series of 72 cases from 1936-1942[19]. Parmley's classic paper in 1948 of 296 autopsy cases has become the basis of our continued understanding of the incidence and natural history of BTAI[5]. Greendyke in 1966 noted 41 deaths from BTAI in an autopsy report of 1174 MVA victims[21]. Additionally he noted an associated overall incidence of 16% BTAI in MVA victims.

The first successful repair of BTAI is credited to Dfhanelidze in 1923, followed by Gerbode in 1957, and Klassen in 1958[16]. Debakey performed the first graft repair for traumatic chronic aneurysm in 1958[10,13]. Symbas[10], in a literature review through 1973 found 204 cases of BTAI treated surgically. VonOppel et al.[15] subsequently reported more than 1742 patients from 1972-1992 with BTAI reaching the hospital alive. Through 1997 more than 3,000 scientific papers have appeared re. BTAI[4]. The majority of papers are case reports, technical advances, retrospective series, and recently meta-analysis, and retrospective non-randomized comparative studies. Mattox and Wall[13] in 2000 gave a complete historical review of BTAI. They divided the recent history into decades: From

1950-1960- Decade of Description

1960-1970- Decade of Diagnostic Approaches and Epidemiological analysis

1970-1980- Decade of Repair technologies

1980-1990-Decade of Imaging controversies

1990-2000- Decade of Analysis and Conservatism

The present decade is the era of elective planned delay, with priority given to controlling blood pressure,

treating associated injuries, and open surgery vs. endovascular stent graft (EVSG) vs. non-operative treatment.

Incidence

The exact global incidence of BTAI is unknown, but estimated at 7,000 to 8,000 per year in North America[3,4]. Fitzharris et al[20] reported an incidence of 1.5 and 1.9% for USA and UK victims, respectively, following MVA. This was based on autopsy and documented clinical survivors. Assuming a 5% mortality from BTAI, then over 60,000 deaths per year globally from motor vehicle accidents or RTI's are attributable to BTAI[4]. The majority of studies regarding incidence is based on autopsy studies[4,5,21-23]. Confusion arises when causation of mortality is from BTAI alone or multi-trauma (polytrauma). It is difficult to document from the reported autopsy studies whether true rupture with exsanguinations was the primary cause of death.

Malhotra et al.[11] reported 15,000 patients evaluated with screening helical CT scans. BTAI was suspected in 1.3%, and confirmed in 0.6%. Only 2.3% of the injuries were the ascending aorta, with 97.7% the remaining aorta. In a previous publication from the same center, Pate et al.[17] reported 24,681 patients with multisystem trauma, with 12,587 from MVA. 51 patients had BTAI, yielding an incidence of approximately 4/1,000 cases of trauma. VonOppell et al.[15] point out that the mean number of patients with BTAI admitted to the major medical centers was 2.6/year (range 0.2-10.7 patients). This limits the experience to a small number of major regional trauma centers in the USA[16].

It may be postulated that the global incidence of BTAI will decrease from MVA secondary to increased seat belt use, air bags, and improved vehicle design (puncture-resistant gas tanks, shock absorbing steering columns)[20,24]. The Haddon study and matrix has shown a reduction of fatalities following MVA in the USA from 30 deaths/100,000 population in the 1930's to 15/100,000 presently[25].

Etiology

The major cause of BTAI is motor vehicle accidents (MVA) or road traffic injuries (RTI). Other causes include pedestrian vs. vehicle, falls >3meters, crush injuries, airplane crashes, and work/recreational-related events[5,26]. Burkhart et al[27] noted the etiology in 242 autopsy cases : 68% MVA; 17% pedestrian injury from MVA; and 17% motorcycle crash. Feczko et al[28] in an autopsy study of 142 cases reported: 72% MVA; 12% pedestrian; 8% motorcycle; 5% falls >3 meters; and 5% miscelleanous. Airplane accidents deserve mention, since there is a higher incidence of BTAI in this group, and higher mortality (>96%)[4,5,26].

Pathology/Pathogenesis

The thoracic aorta is divided into ascending, arch, and descending segments. The arch gives rise to the innominate (brachiocephalic), left carotid, and left subclavian branches. A common branching of the innominate and left carotid occurs in 13%; left carotid from the innominate in 9%; origin of left vertebral from arch <3%; bilateral innominate <1%; and aberrant right subclavian <1%[30]. The average diameter of the descending aorta in the majority of BTAI victims is 18-24 mm, given that the majority of patients are young male adults[6]. The thoracic aorta consists of the tunica intima, media, and adventia. The tunica media has about 50 layers or lamella, compared to 28 for the abdominal aorta. The lamella is composed of distensible elastic fibers, along with muscle cells, collagen,and ground substance. The adventitial layer contains the strong collagen fibers and provides the major tensile strength of the aorta[31].

The major pathological feature is a transverse or horizontal tear, occasionally spiral or tangential, and rarely a dissection[6,12,28]. The tear varies from a small partial intimal tear to full or complete circumferential rupture and separation or distraction of proximal and distal ends. (figure1)[11,12].

Partial tears occur more commonly posteriorly, involving both the intima and media[6]. They can also form intramural hematomas and focal dissection with/without retrograde extension. Fattori et al[18] point out that fibroplastic proliferation begins soon after injury, and by 2-3 weeks endothelial cells appear. Increased rate of endothelial growth rate occurs in the first 7-15 days. The associated periaortic hematoma begins to liquefy at 14 days, with resultant pseudaneurysm formation[16]. The adventitia provides the major component of tensile strength of the aorta. Premortem antegrade or retrograde propagation or dissection of small tears can be difficult to diagnose[6]. Dissection has been reported in blunt trauma patients with underlying aortic disease or states e.g. Marfan's disease, Ehlers-Danlos syndrome, pregnancy, or aortitis[32]. The location of injury is based on both autopsy and clinical studies (table 2)[5,27-29].

The lower incidence of the ascending aortic injuries in the clinical data is reflective of the higher severity and initial on scene mortality. The mechanisms for BTAI are considered to be attributable to :

1- bending and deceleration-acceleration shearing forces applied at a fixed point of the aorta (most commonly aortic isthmus) (figure 2,3),

2- "osseous pinch" compression of the aorta between the spine and the thoracic cage,

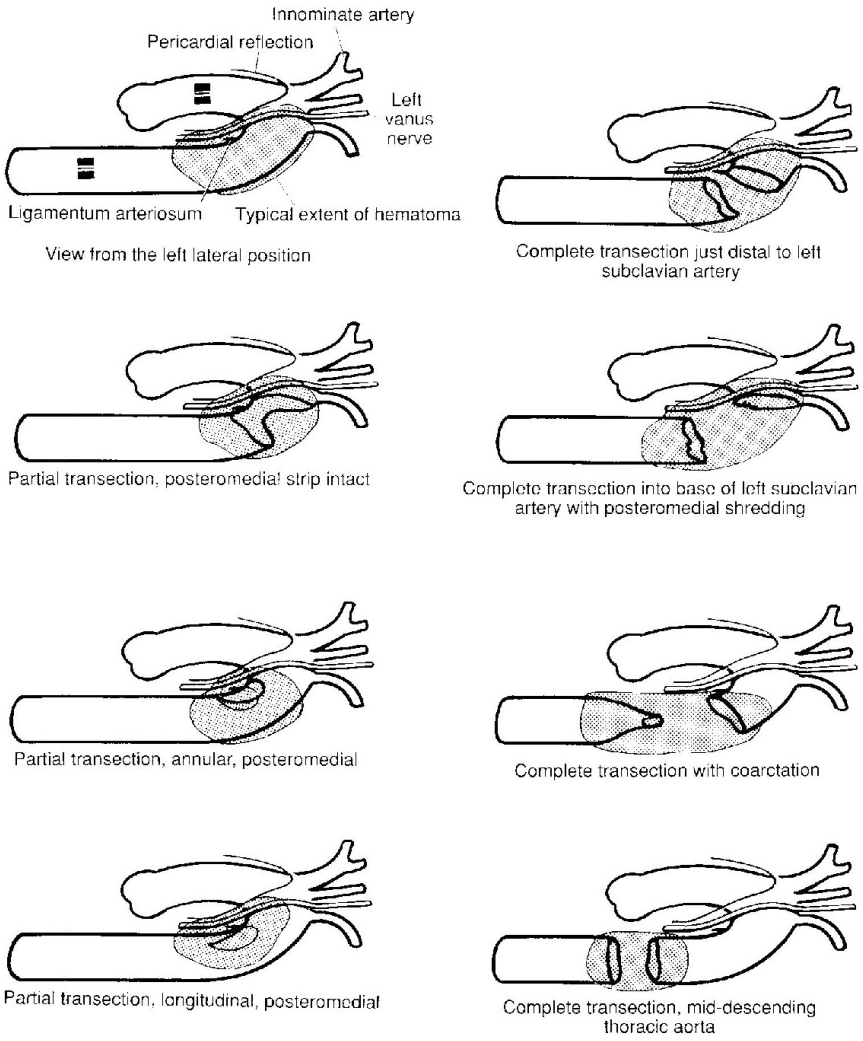


Fig. 1 : Spectrum of BTAI involving descending thoracic aorta

*From Turney SZ, Rodriguez A. Injuries to the Great Vessels. In: Turney SZ, Rodriguez A, Cowley RA. Management of Cardiothoracic Trauma. Williams & Wilkins. Baltimore. 1990: page 243.

3- profound intra-luminal hypertension and endovascular pressure waves (hydrostatic force) during a severe traumatic event,
4- torsion and "water-hammer" effect at the ascending aorta, and
5- traction forces in association with hyperextension of the spine accompaning a vertebral fracture[33-41] (figures 2,3).
Cammack et al[36] estimated that a head-on collision of 60 mph generates an intra-aortic pressure of 1250mmHg. However, Zehnder et al[40] determined the internal force required to cause a tear is an intraluminal pressure >2,000 mm Hg. Historically, Abbot suggested that the aortic isthmus is congenitally weak[12]. Yet this was in the presence of bicuspid aortic valve or coarctation. No true weakness of the isthmus has been demonstrated, with the adventitia, as noted, providing the major tensile strength of the aorta[6].

Table 2 : Anatomical location of BTAI

Autopsy ⟶ Clinical

Location	P[5]	B[27]	F[28]	W[29]
Isthmus	124	58	54	65
Ascending	64	8	8	14
Arch	22	7	2	..
Distal	35	8	11	12
Multiple	17	16	18	13
Diaphragm/ Abdomen	13	7	7	9

P : Parmely[5] - B : Burkahart[27] - F : Feczko[28] - W : Williams[29]

The pathogenesis of BTAI is closely correlated with the type of accident, the applied forces, the application of passive and/or active vehicular restraints, age and associated co-morbidities. A thorough evaluation is required to determine the impact of the traumatic event on the patient's physiology. The direction of the applied forces on the vehicle in a crush or the pedestrian in a vehicular-pedestrian accident, the extrinsic/disruptive type and the inertial (dP/dT, dBP/dT) type of forces, the impulse angle, the intrusion, the extent of passenger compartment deformation (DEF) and the delta-V(a measure of crash severity expressed in km/hour or miles/hour) are all impact-related factors that determine the likelihood of BTAI.

In recent studies, individuals at risk for BTAI can be reliably excluded in collisions when delta-V > 20mph and near-impact and intrusion factor > 15 inches are not present (negative predictive value of 100 %)[33,35,42]. Furthermore, the risk for BTAI in car occupants was the highest in broadside or lateral collisions, with a direct correlation of higher AIS thorax, ISS and clinical course (ICU length of stay and ventilation time). In addition higher delta-V and DEF occurred in older (> 55 years old) and pedestrian victims[20,35,42,43].

Richens et al[44] has nicely summarized four theories of pathogenesis. Sudden stretching at the fixed aortic isthmus is the most common theory. Sudden rise of intra-aortic blood pressure to >2,000 mmHg is the second theory. The "water hammer" effect, occuring with high pressure waves being reflected back on the vessel wall is the third theory. Finally, the "osseous pinch" theory implies entrapment of the aorta between the anterior bony chest structures and the vertebral bodies. Further, it is theorized by Richens that a combination of all four postulates are present[44].

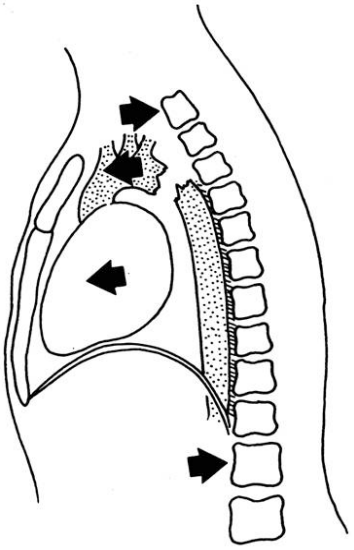


Figure 2* : Mechanism for BTAI (*From McSwain NE. Kinematics of Chest Trauma. In: Webb WR, Besson A. ed. Thoracic Surgery: Surgical Management of Chest Injuries Vol 7. Mosby/Year Book 1991; p.8.

The heart and arch of the aorta are relatively free to move forward, whereas the descending aorta is secured tightly to the vertebral column. The junction of the arch and descending aorta is the point of maximum stress and location of stress disruption of the aorta.

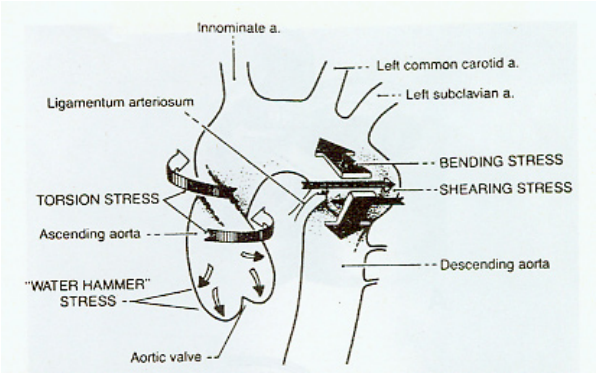


Figure 3 : Pathophysiologic features of blunt thoracic aortic injury (From Symbas PN. Cardiothoracic trauma. Philadelphia: Saunders; 1989. P. 193.)

This is also supported by Crass et al[37] who maintain that individual mechanisms alone are inadequate to cause injury, given the high aortic tensile strength.

Those that survive BTAI to receive medical attention have some degree of contained aortic rupture in the form of intimal tear, partial thickness injury (pseudoaneurysm, dissection), full thickness contained injury (rupture with periaortic hematoma contained by the adventitia and mediastinal pleura), or non-contained complete transection. (figure 4,5).

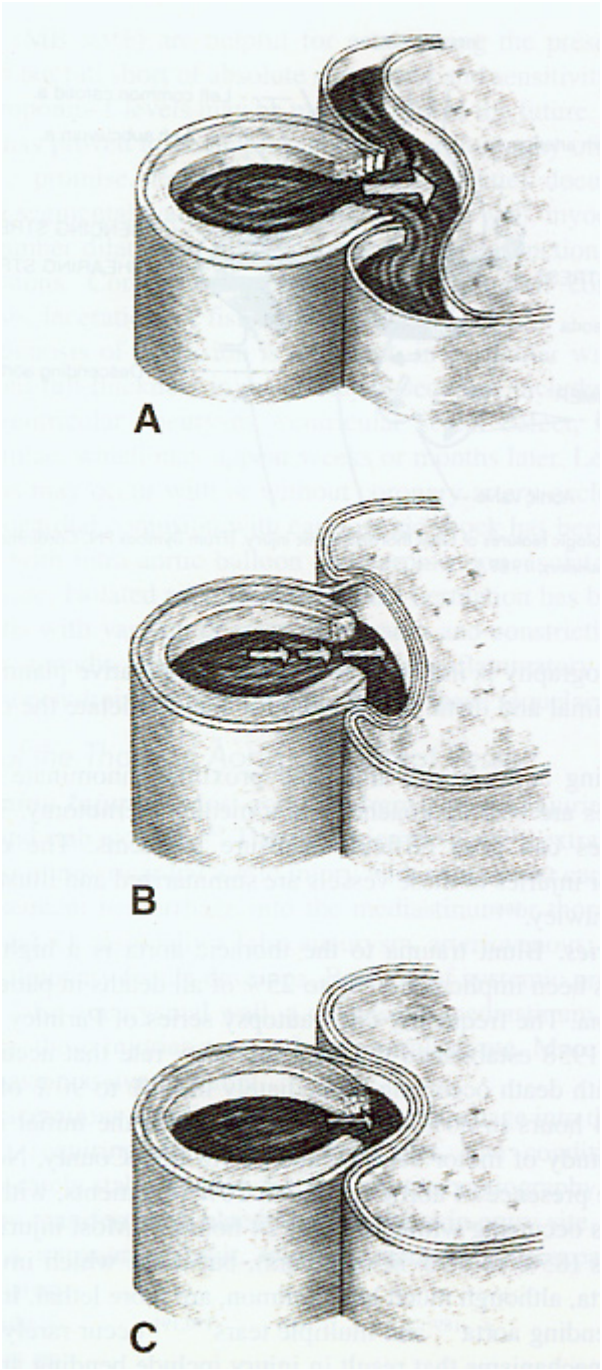


Figure 4 : Degree of aortic injury from (A) rupture to pleural cavity, (B) contained by adventitia, to (C) chronic aneurysm (*From Naclerio EA. Chest injuries, physiological principles and emergency management. New York: Grune and Stratton, 1971 pages 282-288.)

The majority of free ruptures through the mediastinal pleura into the pleural cavity or the pericardium are lethal, secondary to exsanguinations. Uncommonly, the initial injury leads to a post-traumatic chronic dissection or development of either a true or false aneurysm in 2-3% of victims[5,20].

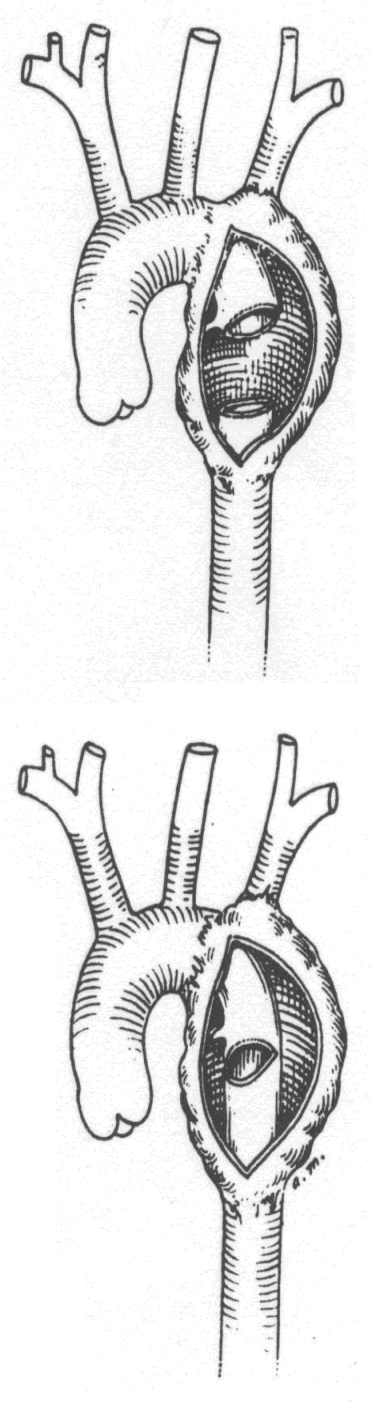


Figure 5 : Circumferential tear with distracted ends and pseudoaneurysm, compared to partial tear and pseudoaneurysm. (From Langlois j, DeBrux JL, Binet JP, Khoury W. Traumatic Aortic Rupture. In: Grillo, HC, Eschapasse H, ed. Major Challenges- International Trends in General Thoracic Surgery Vol.2. WB Saunders. Philadelphia. 1987. P. 275.

Natural History (Table 3)

Though the majority (85-90 %) of BTAIs occur at the isthmus, only 58 % are initially or immediately lethal. Non-isthmus injuries, though uncommon (ascending aorta and transverse arch 12 %, mid- and distal descending aorta 10 %), are highly lethal[5,11,45,47]. Multiple injured aortic sites have been reported in as many as 18 %, with a mortality greater than 70 %[29,48]. Finally, aortic branched vessel injury, isolated, or, in association with thoracic aortic involvement has been reported from 15 to 38 %[48,49]. A composite of survival in days, months, and years is illustrated in figures (6,7,8,9)[50]. These statistics were gathered primarily from Parmley's classic autopsy study at the Armed Forces Institute of Pathology (AFIP) in 1958[5]. More recent clinical data from Hartford et al[51] in 1986 noted 70 of 84 patients died at the scene or were dead on arrival, Seven more died in the hospital prior to diagnosis, and 7 ultimately survived following operative repair. The long term actuarial survival of unrecognized, undiagnosed, or untreated BTAI is unknown. In 50% of chronic traumatic aneurysms the time interval to recognition was 10 years, and in 12% the interval was >20years[52].

The injury location was 92% descending aorta, 2% arch, and 6% ascending aorta. Finkelmeier et al[53] noted 5, 10, and 20 year survival of 71%,66%, and 62% respectively with >90% of the injuries at the isthmus. BTAI as the primary cause of death can be difficult. Confirmation of rupture with exsanguinations is the primary cause of death can only be gleaned, or confirmed, by the presence of massive hemothorax. Pezzella[26] in a study of autopsies from 3 airplane crashes categorized massive hemothorax from aortic rupture as the primary cause of death.

Pate[54] has challenged the natural history figures. He rightly points out that the classic Parmley study was an autopsy study with no cause of death given. The victims studied were from the 1862 to 1957 timeframe, from a variety of causes, including military. Pate comments further on the study of Williams et al.[29]. In that series of 90 patients with BTAI, no data are given implicating massive hemothorax as the primary cause of death. Clearly, in the era of rapid evacuation from the accident scene, early diagnosis, and anti-pulse therapy, the natural history has changed.

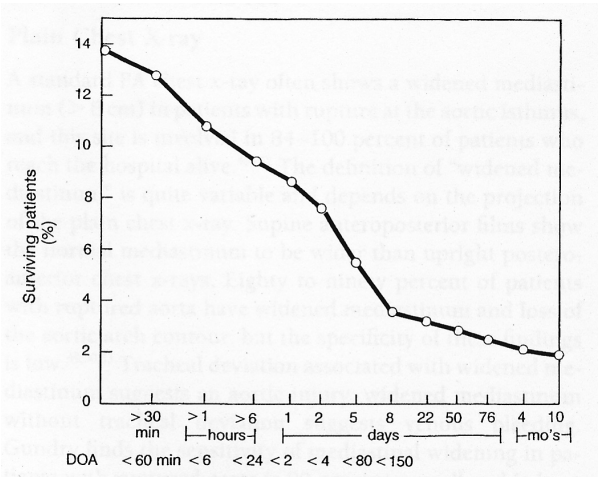
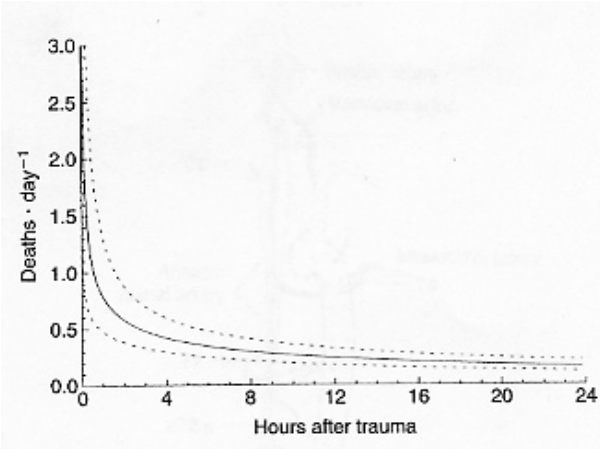


Fig. 6 : Duration of survival after injury in patients who died with traumatic aortic rupture. (*From Parmley L, Mattingly T, Manion W: Nonpenetrating traumatic injury of the aorta. Circulation 1958; 17:1086-1111 (5)

Table 3 : Death intervals **Natural History**

81% death at scene or arrival	42% 96 hours
12.5% 6 hours	72% 8 days
25% 24 hours	83% 3 weeks
29% 48 hours	90% 10 weeks
	2%/ year thereafter



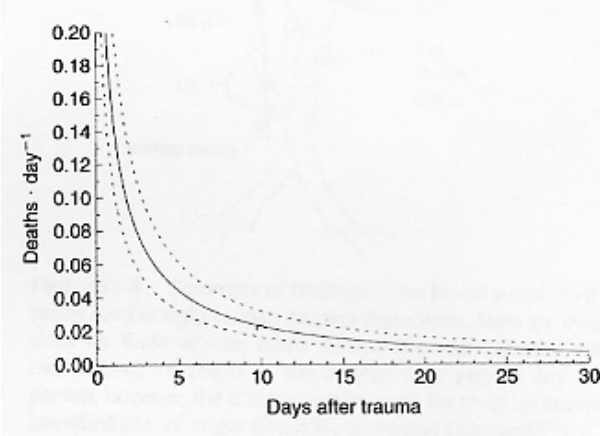


Fig. 8 : Hazard function (deaths • day^{-1}), or instantaneous risk of death across time, after acute traumatic aortic transection. Time zero is time of trauma. Dashed lines enclose 70% confidence limits. There is an early phase of rapidly falling risk and a constant late phase. The two graphs differ only in the scales of the axes; in **A** the horizontal axis is *hours* after time zero, and in **B** it is *days*, and the vertical axis is expanded. The relationships are such that the following are conditional probabilities of survival without treatment :

Time after Trauma	Probability (%) of Survival for :	
	24h	7 days
0 h	74	50
12 h	85	61
24 h	89	66
2 d	92	73
3 d	94	77
4 d	95	80
5 d	96	82

(From Kouchoukos NT,Blackstone EH, Doty DB, Hanley FL, Karp RB. Kirklin/Barratt-Boyes Cardiac Surgery, 3rd ed. Churchill Livingstone. Philadelphia. 2003; p.1802.)

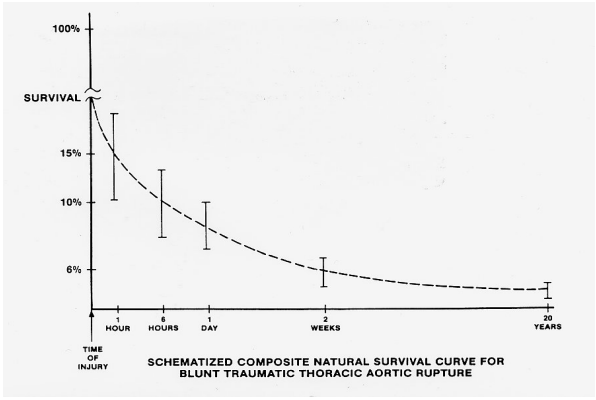


Fig. 9 : Schematized composite natural survival curve for blunt traumatic thoracic arotic rupture (From Turney SZ, Rodriguez A. Injuries to the Great Vessels. In: Turney SZ, Rodriguez A, Cowley RA. Management of Cardiothoracic Trauma. Williams & Wilkins. Baltimore. 1990; p. 231.)

Clinical Aspects

The need for prompt diagnosis and a high level of suspicion is critical. The average age range of victims is 36-40 years, with male dominant[6]. Less than 50% of the patients with BTAI have clinical evidence of chest trauma or physical findings associated with aortic pathology. Parmely reported 36% of victims had no clinical evidence of BTAI[5]. Supraclavicular bruit or hematoma, midscapular murmur, pseudocoarctation syndrome, persistent hypotension, or cardiac tamponade are commonly absent[6,55,56]. Other clinical manifestations include dyspnea, back pain, presternal contusion, palpable chest wall instability or pain, and differential hypertension in the upper extremities relative to the lower extremities (pseudocoarctation)[6]. Isolated systemic hypertension requires aggressive evaluation and treatment, given the risk of increased intra-aortic propagating forces.

Often the mechanism and suspicion of injury (e.g. driver hitting steering wheel) represents the strongest indicator guiding the diagnostic work-up and management, especially in the asymptomatic patient with a negative screening chest x-ray. Details of the MVA, especially the rate of speed (>50 km/hour), driver vs. passenger, location of the patient, site of impact (direct vs. lateral or broadside) restrained/unrestrained with seat belt/harness, pedestrian injury, motorcycle, and ejection from vehicle are important details. Falls > 3 meters, airplane crashes, train accident, and details of crush injury are other suspected injuries with risk for BATI.

In the study of Simon et al[57], the mean time from transient hypotension to free rupture, in the setting of decelerating trauma for patents with "triad of impending

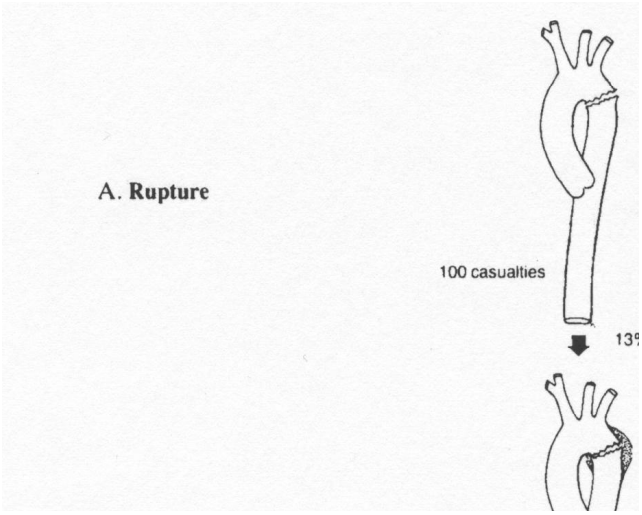


Fig. 7 : Parmley Autopsy Data[5]

rupture" (eg the constellation of widened mediastinum with hemothorax and transient hemodynamic instability), was 25.6 min. The authors concluded that "this association has a strong diagnostic value and warrants immediate surgical intervention without confirmational diagnostic studies"[57]. The presence of a positive history for mechanism of injury, large left hemothorax, pseudocoarctation syndrome, or supraclavicular hematoma also warrants prompt confirmatory diagnosis, and even urgent surgery without confirmatory diagnosis.

Basically, patients with BTAI can be categorized clinically into 3 groups[13]: 70-80% die at the scene within 0-2 hours, secondary to exsanguinations from rupture or from associated injuries; 2-5% arrive at the hospital in an unstable condition, and worsen over 1-6 hours, usually from associated injuries; and 15-25% are stable over 4-18 hours, with or without associated injuries. The unstable are usually intubated, given the high incidence of associated respiratory problems. The first group die from early exsanguination, the second group from exsanguination or associated injuries, and the third group from associated injuries or postoperative complications.

Associated Injuries

Associated injuries include pelvic, long bone fractures and posterior hip dislocation (55 – 62 %), solid abdominal organs (38 – 51 %), closed head trauma (35 – 70 %), lung contusion (up to 40 %) and other intra-thoracic structures, including heart (3 – 10 %). Liver and spleen injuries are by far the most frequently coexisting solid abdominal organ injuries in blunt trauma (table 4)[7,58-64]. Fatal BTAI is associated with closed head injuries (85 %), rib fractures (79 %), other intra-thoracic organ injuries (78 %), and intra-abdominal injuries (48%)[15,65,66]. Despite rib fractures having a statistically significant higher incidence in patients with BTAI (46%) than those without, the specificity (57.4 %) and the positive predictive value (14.8 %) are poor[67]. Interestingly, the incidence of associated cardiac injury has decreased to < 9%)[7].

Table 4 : Associated injuries in hospitalized patients with traumatic aortic disruption

Associated (surgical) injuries				
	Schmidt[58]	**Hilgen-berg[59]**	**Duhay-longsod[60]**	**Kirsh[61]**
Central nervous system	25	39	34	50
Thorax	-	-	-	-
Diaphragm	13	2	12	9.3
Lung	38	41	43	58
Heart	10	10	18	19
Rib/clavicle fractures	40	39	55	65
Abdominal				
Spleen	20	10	17	
Liver	10	12	15	
Kidney	9	12	11	
Bowel	10	-	15	
Other abdominal	11	-	9	
Skeletal				
Extremity	81	71	59	
Spine	5	10	20	
Pelvis	24	25	26	
Maxillofacial	5	10	20	

Table 4 : continued

Associated (surgical) injuries				
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Thorax				
Diaphragm	13	2	12	9.3
Lung	38	41	43	58
Heart	10	10	18	19
Rib/clavicle fractures	40	39	55	65
Abdominal				
Spleen	20	10	17	
Liver	10	12	15	
Kidney	9	12	11	
Bowel	10	-	15	
Other abdominal	11	-	9	
Skeletal				
Extremity	81	71	59	
Spine	5	10	20	
Pelvis	24	25	26	
Maxillofacial	5	10	20	

Severity Index/ Risk factors

Age, Glascow coma scale (GCS) at the scene and on arrival in the Emergency department, Injury severity score (ISS), Abbreviated injury severity score-Thorax (AISthorax), APACHE II and mechanism of injury with applied forces all have statistically significant impact on the lethality of the BTAI[47]. Age (>55 years old) is associated with higher associated injuries and comorbidity, yet has not been a constant risk factor[68]. The majority of patients are males 36-40 average

years of age[6]. Simon et al[57] reported 51 patients with BTAI. Subgroups of free rupture and contained rupture were compared. A widened mediastinum, hemothorax, and transient hypotension were increased risk factors for free rupture. The mechanism of injury is important. More than 75% of BTAI involve MVA related events. Ejection, frontal, and lateral crashes carry high risk, whereas seat belts and air bags decrease risk[6].
The ISS has been the most common risk score used for BTAI. The ISS is an anatomical score that uses values from 1-75. An ISS >16 is associated with a mortality of 10%[69]. Globally, mortality with ISS >9 has been reported 35% in high income countries, 55% in middle income countries, and 63% in low income areas like Subsaharan Africa[2]. Sturm et al[62] reported a mean ISS of 59.3+/-13.8 in 51 patients who died at the scene of injury. Camp et al.[68,70] noted a 71.9% survival in a stable cohort of 233 BATI patients with an ISS of 40+/-16. Other associated risks in their study included age >55 years, and coronary artery disease. Other studies report similar scores[71,72,73]. Lebl et al.[72] showed similar results in 3 treatment groups of surgery, delayed treatment, or stenting. Survival was 80% with ISS similar in all 3 groups (34.9, 29.9, and 35.1 respectively). The mean ISS was 42.1 in the AAST-1 trial, with a proportionate increase in mortality[7]. It is clear that more victims with increased associated injuries are reaching the hospital, with earlier diagnosis of BTAI, and higher interval mortality, unless treated.

Diagnostic Evaluation

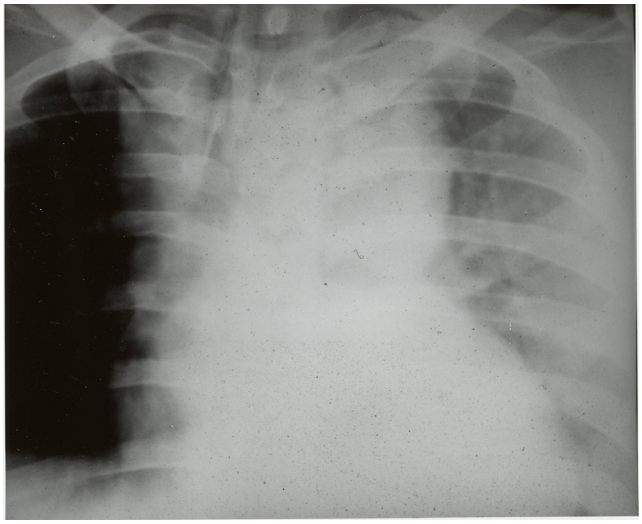
The diagnostic time interval or sequence is a critical phase in the care and evaluation of BTAI. This includes initial evaluation, monitoring, and eventual surveillance. The time interval from diagnosis to treatment has increased from 16.5 hours to 54.6 hours in the 2 AAST studies[7,8]. This is a reflection of the increase in delayed management strategies, principally blood pressure control, treatment of life threatening associated injuries, and repair of BTAI with open surgery or EVSG.

Laboratory

Aside from routine hematological, chemistry panel testing, and blood gas analysis, cardiac enzymes are measured, given the 5-10% associated cardiac injuries[74]. Creatinine kinase with myocardial specific fractions (CPKMB) and myocardial specific enzyme troponin I (cTnI) are usually measured[23]. Combined with ECG and ECHO, a negative predictive value of 100% for myocardial injury can be achieved[74,75].

Chest X-ray (CXR)

The supine anteroposterior (AP) chest roentgenogram is part of the standard evaluation of chest trauma in the emergency room, even though the sensitivity (90 %) for diagnosing a widened mediastinum lacks specificity. Its predictive ability may be improved with the addition of an upright AP chest x-ray view or a supine reverse Trendelenburg view[6] (figure 10,11).



Radiographic findings (table 4,5) suggestive of aortic injury include a widened mediastinum greater than 8 cm; mediastinum : chest width ratio greater than 0.25, irregular aortic knob; and opacified aortopulmonary window reach sensitivity above 80 %, with nasogastric tube deviation, depression of the left main stem bronchus, wide left paraspinal line and thoracic spinal fracture being more specific indicators of BTAI[13,76-79]. Dyer et al[80] recommended that with a low impact mechanism and normal chest-x ray, no further imaging is warranted. However, the suspicion of injury should guide the evaluation, despite an asymptomatic presentation and normal chest x-ray, given the chest x-ray may be interpreted as negative in 4-15% of patients[6,48,76].

It is important to note that the cause of a widened mediastinum, aside from BTAI, may be caused by small tears of arteries or veins surrounding the aortic arch, or superior mediastinal veins[52]. The natural history of these causes is gradual resolution with return to normal, yet no long term serial CXR follow-up has been performed looking for chronic changes or return to normal.

Table 5 : Radiographic findings suspicious for BTAI

	Sensitivity (%)	Specificity (%)
Mediastinum > 8 cm		
M:C width ratio> 0.25	53	59
Opacified aortopulmonary (AP) window Irregular or	72	47
loss of aortic knob	63	53
Blurred aortic contour		
Nasogastric tube elevation or deviation to right		
Trachea shifted to right or anterior displacement Pulmonary contusion		
Wide left paraspinal line or loss of paraspinal stripe		
Depressed left main bronchus (> 140 degrees)		
Left apical cap (pleural hematoma)		
First rib fracture		
Thoracic spinal fracture		
Clavicle or scapula fracture		
Large left hemothorax		
Calcium "layering" in aortic arch		

*From: Woodring JH, Dillon ML. Radiographic manifestations of mediastinal hemorrhage from blunt chest trauma. Ann Thorac Surg 1984;37:171-178.

Mirvis SE, Bidwell JK, Buddemeyer EU, et al. Value of chest radiography in excluding aortic rupture. Radiology 1987;163:487.

Mattox KL. Approaches to trauma involving the major vessels of the thorax. Surg Clin N Am 1989;69:77-91.

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CT Scanning

Computed tomography (CT), with or without contrast (volumetric helical or spiral), is the currently recommended initial screening modality for BTAI[6,49,81] (figure 12,13,14). It is readily available, fast, low cost, and easily interpreted. The CT scan should also include the head and abdomen, as part of the total torso evaluation. Though helical CT lacks positive predictive value (PPV) (55%), primarily due to aortic wall motion, prominent bronchial or mediastinal vessels, prominent atheromatosis or volume averaging, it has 100 % sensitivity and negative predictive value (NPV), with specificity up to 99%. As an effective screening tool, and being less invasive, less expensive, faster to obtain, and more readily available compared to angiography, the need for conventional aortography (CA) has drastically decreased by more than 56%[82]. Gavant et al[83] reported in a prospective analysis that helical CT has 100% sensitivity compared to conventional angiogram (94.4%) and 81.7% specificity versus 96.3% respectively. Mirvis et al[67] concluded that, with specificity reaching 99.7 %, helical CT can reliably exclude thoracic aortic injury.

Findings suggestive of BTAI are a periaortic hematoma (PPV 97 % for traumatic aortic rupture), disruption of normal low attenuation periaortic plane, irregular aortic contour, poorly defined fat plane, aortic caliber change, and intraluminal irregularity[84]. With the introduction of high resolution technology (1.25-3.00 mm thin views),with positive predictive value (PPV) up to 98 % and NPV and specificity 100 % respectively, minimization of volume averaging and wall motion has been accomplished by multi-slice multidetector three-dimension CT angiography (3D MDCTA or 3D CTA)[49,81,84].

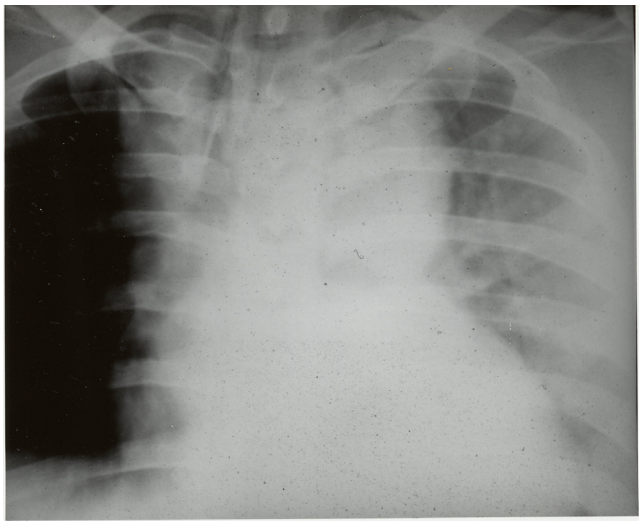
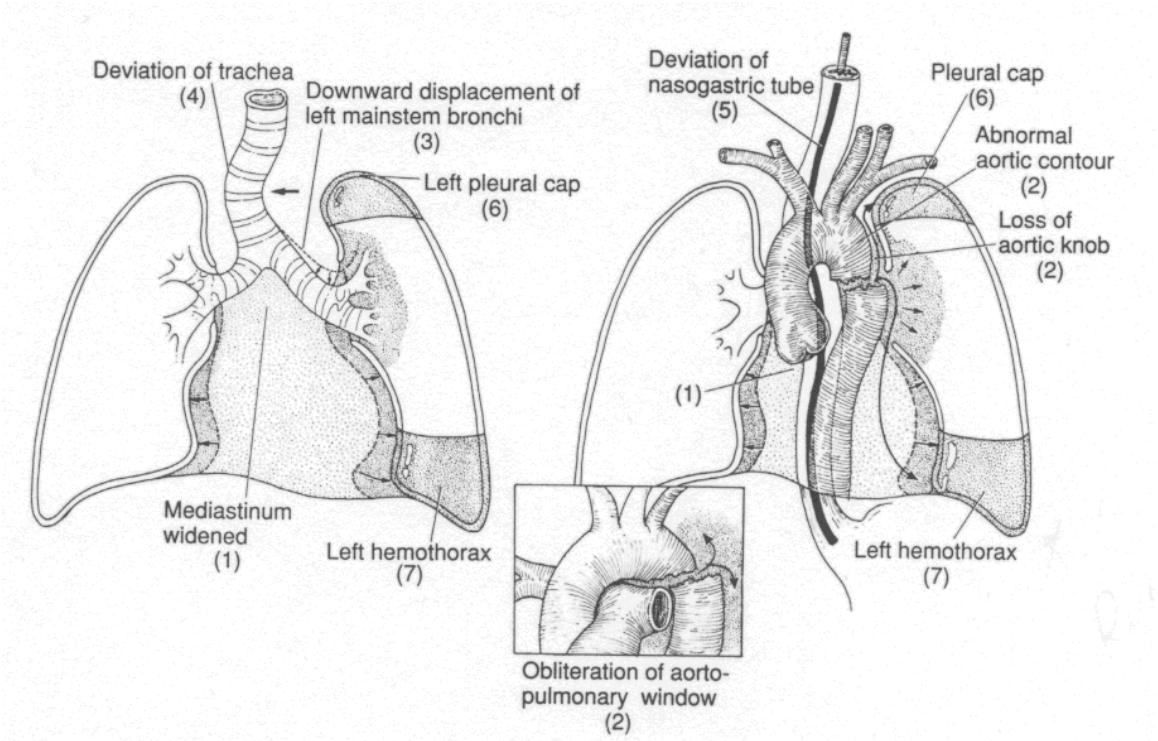


Fig. 10 : AP Chest Roentgenogram – Supine view showing widened mediastinum

CTA provides a reliable, faster than helical CT, non-invasive, diagnostic and preoperative planning tool (especially with sagital,coronal and axial reconstructions) with superiority in visualizing intimal tears, and identifying proximal branch injuries, thus making the role of conventional preoperative angiogram further limited[56,85]. Recent studies now confirm the role of primary routine screening CT scanning for major chest trauma[86-89]. The availability of three dimensional reconstruction establishes the diagnosis, and gives the surgeon detailed information re. operative approaches, be it open surgery or endovascular stenting. However, stenting may require angiography as the final determinant of stent placement and suitability.



Roentgenographic signs of mediastinal hematoma: *1,2*, widened mediastinum, loss of superior contour of aortic knob, abnormal aortic contour, obliteration of aortopulmonary window; *3*, downward displacement of the left mainstem bronchus; *4*, deviation of the trachea to the right; *5*, deviation of the nasogastric tube to the right; *6*, left-sided pleural cap; *7*, left-sided hemothorax.

Fig. 11 : *From Maggisano R, Cina C. Traumatic rupture of the thoracic aorta. In: McMurtry RY, McLellan BA, eds. Management of Blunt Trauma. Baltimore. Williams & Wilkins, 1990; p.213.

Given the increased use of screening CT scans, smaller intimal tears (<1cm) can be seen, as well as ductus diverticulum remnants[46]. These findings may be more difficult to differentiate. The absence of intimal irregularity and mediastinal hematoma with ductal remnant may be helpful[6]. In these situations, additional 3D CT reconstruction, or angiography may be necessary[81].

The Agee criteria for suspected BTAIs remain relevant[9]. These criteria for reliable negative CT scans include: good contrast enhancement of the aorta; no interfering artifacts; complete study; experienced interpretation; and absence of "positive criteria". Criteria for positive CT scans include: mediastinal hematoma contiguous with the aorta (figure 13); false aneurysm; irregular aortic contour; divided aortic lumen (figure 14); and intimal flap.

Angiography

Despite the low yield (< 10 %) for conventional angiogram (CA) as a diagnostic study, catheter angiography remains the "gold standard" for diagnosis of BTAI and of progressively broadened value, in light of emerging catheter-based endovascular treatment solutions[81]. The yield will probably increase as screening CT scans decrease the need for angiography. The common findings on CA include aortic tear (65%) (figure 15), pseudoaneurysm (29%), contrast extravasation (14%), intimal tear (14%) (figure 16), and dissection (8%)[47].

It has 95-100 % specificity, PPV 98% (false positive results due to ductus diverticulum/remnant or thrombosis of the lacerated intimal site occur in 2% of the studies), and more than 95% NPV, provides accurate appreciation of the arch and branched aortic vessels (often injuries at this level represent intimal tears – 69% and psudoanurysms – 31%), and has unique value for outlining the operative plan, especially in the light of equivocal CT findings[25,26,51].

As an invasive study complications are primarily due to access-related morbidity (1-5% femoral artery pseudoaneurysm, 5-9% hematoma, or intimal injury)[91]. At present, digital subtraction

angiography (DSA) is the preferred technique[81]. It subtracts background information, thus providing more accurate aortic information, as well as requiring less contrast agents. Newer agents are nonionic and low osmolar, with resultant decreased incidence of contrast nephropathy[81].

Pate et al.[92] suggested that the helical CT findings that trigger an aortogram include:

1- intimal tear,
2- pseudoaneurysm,
3- large periaortic hematoma not explained by other injuries,
4- an unestablished proximal extension of the aortic injury, and
5- indeterminate aortic findings[56,71,85].

Angiography has been replaced by 3D CTA for most EVSG procedures. Yet angiography still plays a role in associated injuries, especially pelvic fractures where embolization may be needed for retroperitoneal hematomas, as well as situations where CTA is unavailable or inconclusive.

Intravenous Ultrasonography (IVUUS)

As noted, highly suspicious CT findings, even with negative angiography, should undergo further evaluation by other diagnostic modalities, usually in the form of intravenous ultrasonography (IVUS) (figure17), magnetic resonance angiography (MRA) or transesophageal echocardiogram (TEE)[57,71,93].

IVUS has PPV and specificity of 100% especially for minimal aortic injuries (MAI) with intimal flap less than 10 mm and no evidence of periaortic hematoma. Even though IVUS is highly operator-dependent and invasive, it represents a promising, highly accurate tool in the diagnostic armamentarium, complimentary to CTA or conventional angiography for diagnosis and surveillance of MAI during non-operative management, ruling-out ductus diverticulum in false positive angiograms, guiding the accurate placement of endovascular stent grafts (EVSG) in acute aortic traumatic dissections, and confirming obliteration of the false lumen by the device[57,71,94].

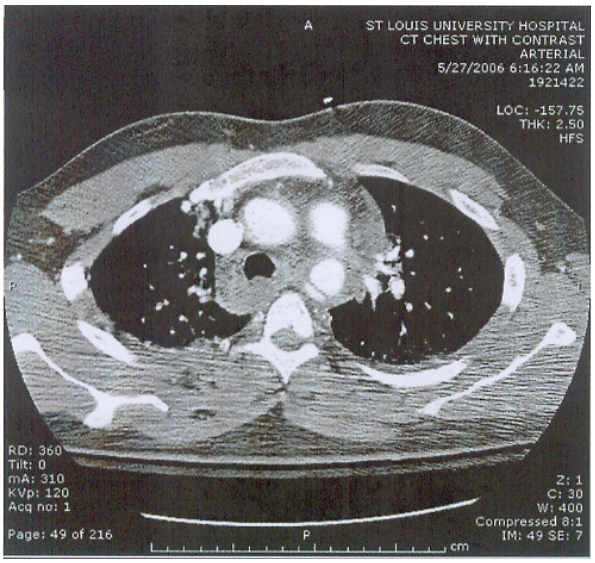


Figure 12 : CT scan of acute descending thoracic transaction with periaortic hematoma (arrow

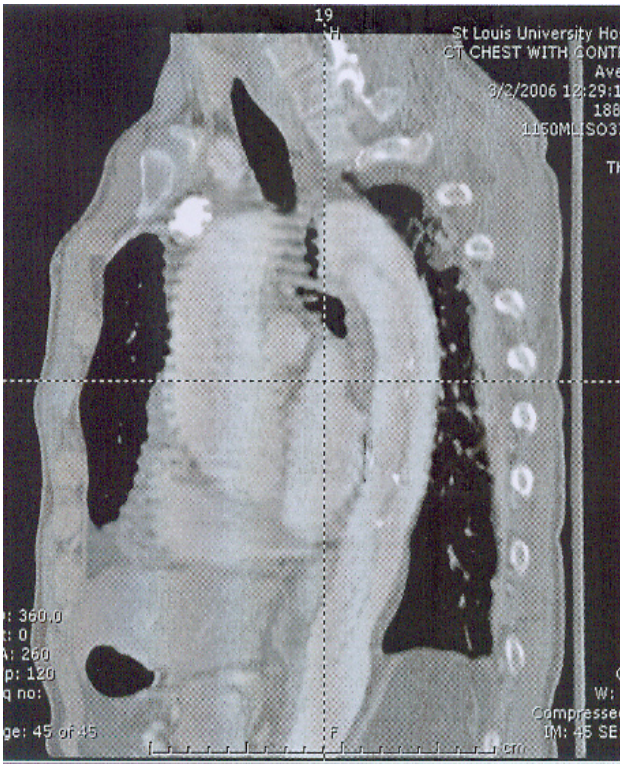


Fig. 13 : Sagittal CT scan showing retrograde dissection of arch and ascending aorta following acute descending thoracic traumatic injury (arrow).

Fig. 14 : Postraumatic descending thoracic aortic aneurysm with opacification of false channel (arrow).

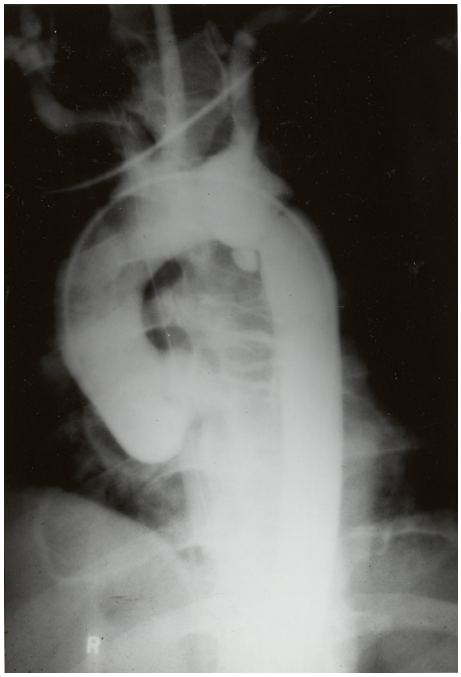


Fig. 15 : Conventional Aortogram (CA) with injury at aortic isthmus distal to left subclavian artery (arrow).

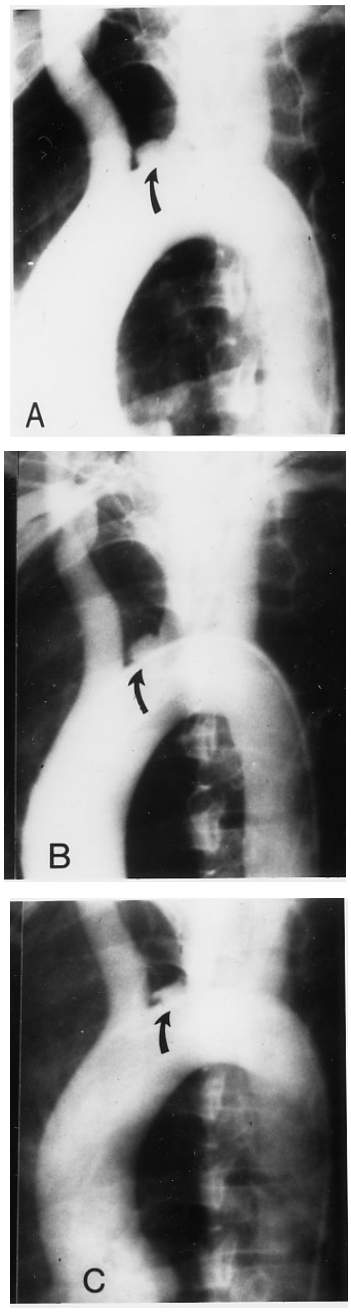


Fig. 16 : Delayed medical management with Serial aortograms showing a small intimal tear in aortic arch between innominate artey and left carotid artery (arrows): A- initial; B- 6 months ; C -2 years. (*From Pezzella AT, Todd EP, Dillon ML, Utley JR, Griffen WO. Early Diagnosis and Individualized Treament of Blunt Thoracic Aortic Trauma. Am Surgeon 1978;44:699-703.)

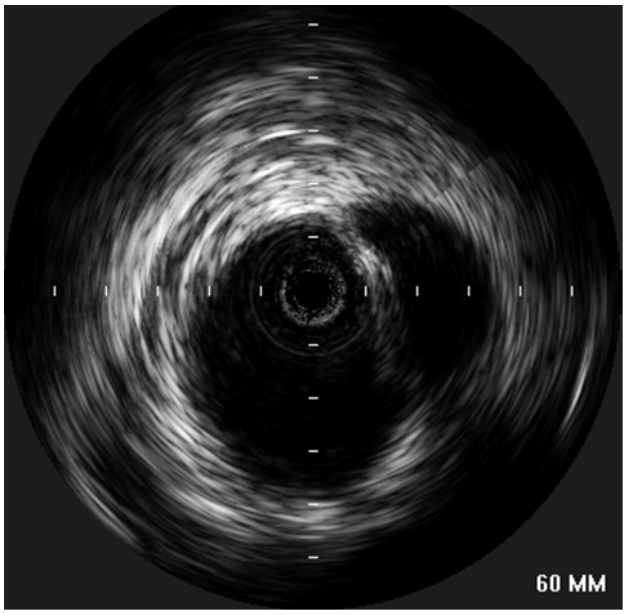


Fig. 17* : IVUS showing dissection flap (arrow).
(From Khoynezhad A, Donayre CE, Kopchok G, Eugene J, White RA. Use of Intravascular Ultrasound in Endovascular Stenting of Traumatic Rupture of the Descending Thoracic Aorta http://www.ctsnet.org/sections/clinicalresources/clinicalcases/article-12.html Accessed 10/26/08.)

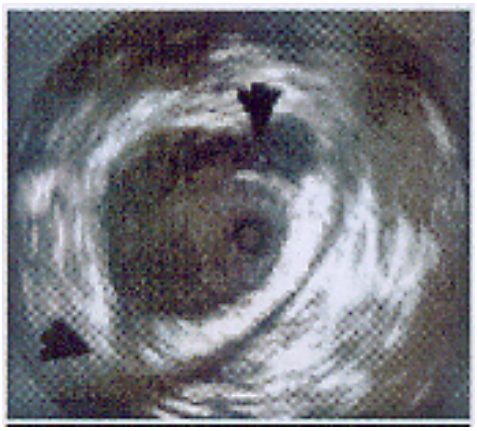


Fig. 18 : Transesophageal echocardiogram of traumatic thoracic aortic dissection. Upper arrow highlights intimal flap.

MRI/MRA

The role of MRA is currently limited to surveillance in patients treated non-operatively to assess and rule out an expanding pseudoaneurysm, and in highly suspicious CT findings with negative conventiona angiography[18,57,71,95]. It has the advantage of requiring no contrast agents, yet it takes longer to perform, and is not readily available. Fattori et al[18] used MRI. They stressed the ability of MRI to detect the hematic content of the injured area because of its high signal intensity. It was also demonstrated that the growth rates of circumferential lesions were greater than partial lesions or tears (1.8 mm vs. 5.9 mm).

TEE

Transesophageal echocardiography (TEE) has emerged as a valuable diagnostic modality over the last decade[6,96,97,98]. (figure 18) It carries no requirements for intravenous contrast and no exposure to radiation, facilitates concurrent assessment of cardiac function (particularly useful in unstable blunt thoracic trauma when definitive diagnosis is in question), is portable, has sensitivity 62 – 100 % and specificity from 92 – 100 %[96,97,98]. TEE can document a mural flap, aortic wall thickness, and low flow patterns[5]. It can be invaluable for hemodynamically unstable patients who arrive in the ED with radiographic and clinical findings highly suspicious for BTAI, but non-suitable for diagnostic work-up with CTscan or angiogram (especially when operative intervention is highly anticipated). As a surveillance modality, TEE can be a valuable tool for assessment and surveillance of MAI, especially in nonoperative or delayed operative approach. Due to institutional differences, TEE may not be readily available, and is operator-dependent. Technical limitations of TEE include: uncooperative patient, a difficult airway, suspected maxillofacial trauma, and suspected or confirmed cervical spinal cord injuries that preclude neck manipulation. Inherent limitations include the assessment of the distal ascending aorta (about 3 cm above the aortic valve), transverse arch and brachiocephalic vessels by the interposition of the air-filled trachea[16]. In addition,TEE is inconsistent and suboptimal in the light of extensive atheromatous aortic disease and pneumomediastinum[49,80,99,100].
TEE is especially useful for intraoperative monitoring. It can help in guiding femoral venous cannulation of the right atrium, as well as assessing cardiac function in operations for associated injuries, especially intra-abdominal injuries. Wall motion changes, volume status, and intracardiac valve assessment can all be readily evaluated and monitored preoperatively and intraoperatively with TEE[23].

(To be continued)