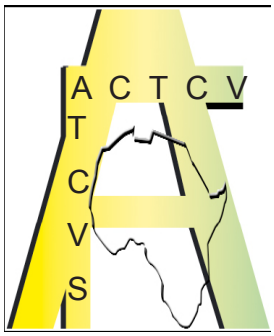
Ann. Afr. Chir. Thor. Cardiovasc. 2008;4(1):7-13



CHIRURGIE CARDIAQUE/CARDIAC SURGERY

BLUNT THORACIC AORTIC INJURY (BTAI) : ADVANCES IN THE ERA OF INNOVATION. A REVIEW (PART 2)

A. THOMAS PEZZELLA, MD[1], ANASTASIOS C. POLIMENAKOS, MD[2]

Treatment

The treatment strategy of the BTAI patient must consider a number of factors: time interval from injury to diagnosis, age, clinical status (emergent, unstable, stable), associated injuries, and underlying comorbidity.

A major therapeutic challenge is treatment of the asymptomatic minimal aortic injury (MAI) (defined as an intimal flap < 1cm and no periaortic hematoma or pseudoaneurysm), and especially those with delayed referral or dignosis[6,11]. Given the sophistication of the aforementioned diagnostic modalities, these small lesions are being increasingly identified[11]. The natural history of MAI is unknown. Malhotra et al.[11] reported a 10% (9 patients) incidence of MAI in a series of 87 patients. Of 6 discharged patients 2 had resolution of the MAI, and 3 developed small pseudoaneurysms. Delayed medical management and surveillance offer a safe and prudent approach[18]. However, EVSG has gained increased support in this scenario.

The larger series of more extensive lesions are the present concern. Mattox et al.[4,13] stress the three objectives of surgical treatment :

1- to prevent exsanguinations from a partial tear with a contained perivascular hematoma;

2- to control blood loss from vessel wall rupture;

3- and to restore vessel continuity.

Symbas[46] proposed an early algorithm (figure 19). Downing et al[49] has proposed an updated algorithm for diagnostic evaluation and treatment (figure 20). A contemporary algorithm features CT scan and EVSG playing prominent roles (figure 21). This has been hightlighted in the recent AAST report (table 1)[8], as well as the updated ATLS changes with level 3 and 4 evidence (table 2)[101].

The ATLS protocol is mandated in evaluation of suspected BTAI[102]. Globally, in low and middle income countries (LMIC), the Essential Trauma Care Project, and its publication Guidelines for Essential Trauma Care, has addressed the need and value of early coordinated, standardized care of the trauma patient[2]. Both stress the ABC's, and primary/secondary survey.

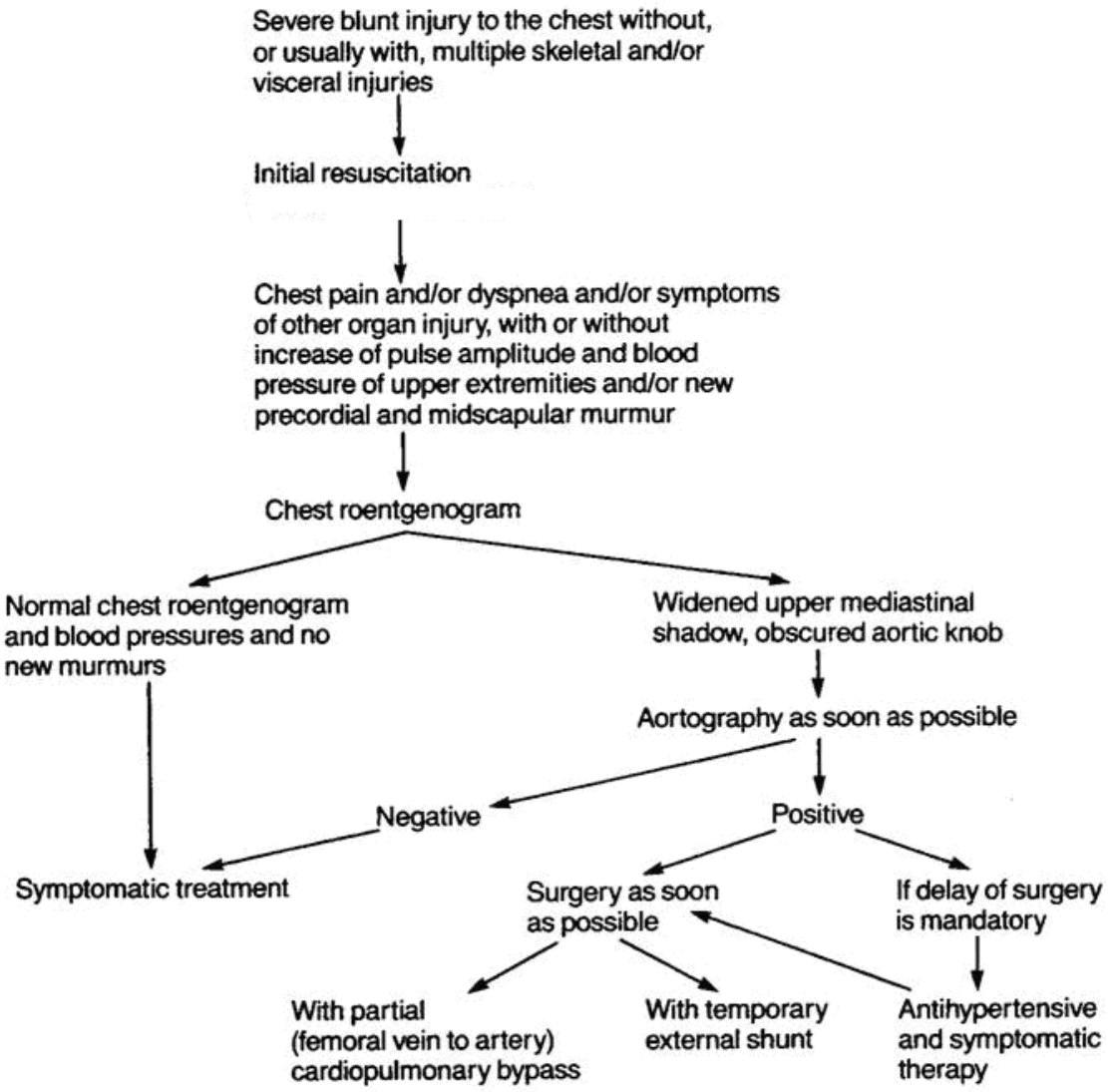
Tension pneumothorax, cardiac tamponade, and massive hemothorax warrant an early response in the primary survey. This requires ready availability of chest tube thoracostomy, subxyphid pericardial window, or emergency/urgent thoracotomy. The patient's clinical status ultimately dictates the subsequent evolution of action.

There are eight thoracic lethal injuries following blunt, penetrating or blast injury: progressive pneumothorax, increasing hemothorax, pulmonary contusion with ARDS, tracheobronchial injury with large air leak, blunt cardiac injury, BATI, diaphragmatic rupture, and mediastinal traverse injury[102]. Diagnostic studies can be precluded in the light of extreme scenarios. With few exceptions a definitive sequence of intervention is mandated[103].

A major decision to be made in the triage period for suspected or diagnosed BTAI is whether the accepting facility can accommodate and treat the injury, or requires transfer to a higher echelon trauma facility (Level I or II). Undertriage places the victim at higher risk, whereas overtriage increases transfer to higher level centers, with resultant greater cost and utilization of resources. The availability of advanced diagnostic studies, cardiopulmonary bypass, and endovascular stent-graft capability are important considerations.

Regarding blunt thoracic trauma, emergency or resuscitative thoracotomy for cardiac arrest, when vital signs are present at the scene, and neurologically intact, has a documented survival rate ranging from 0.6 – 4.5 %[23,64,65]. The role in blunt trauma has not been effective, as compared to penetrating trauma[101,104,105]. The ATLS revised guidelines are noted[101]: " Patients

sustaining blunt injuries who arrive pulseless but with myocardial electrical activity (PEA) are not candidates for resuscitative thoracotomy (RT)". It must be stressed that closed compressions for traumatic cardiac arrest is generally ineffective[23]. The goal of emergency thoracotomy is to gain more effective internal cardiac massage, control bleeding, relieve cardiac tamponade, control air embolism, and cross clamp the descending aorta. All of these goals do not help in the presence of massive hemothorax from aortic rupture, especially if performed outside the operating room.



Algorithm for diagnosis and management of rupture of the aorta.

Fig. 19 : Early historical algorithm with CXR, aortogram, and early open surgery options. (From Symbas PN. Cardiothoracic Trauma. WB Saunders, Philadelphia. 1989. P.190.)

The concept of damage control in thoracic trauma is limited and not well understood. Simpler techniques, including crycothyroidotomy, subxyphoid window, and chest tube thoracostomy are well know and practiced[105]. Yet, unlike packing for abdominal wounds, this is not an option for thoracic wounds. Chest closure may be difficult, though use of delayed chest closure (eg Bogota bag) has been utilized. Thoracoscopy has not been widely utilized in diagnosis or treatment strategies for BTAI.

temporized by the concept of permissive hypotension, in the absence of significant head injury. A minimum systolic pressure of 90 mmHg is the goal. This is achieved with crystalloid, colloid products, or blood. Alam et al.[106] has reviewed new developments in fluid resuscitation. Delaying or restricting fluids did not increase mortality. They noted 6 randomized clinical studies reviewed by the Cochrane Database. These studies failed to show increased mortality in restricted large volume fluid administration. The major volume

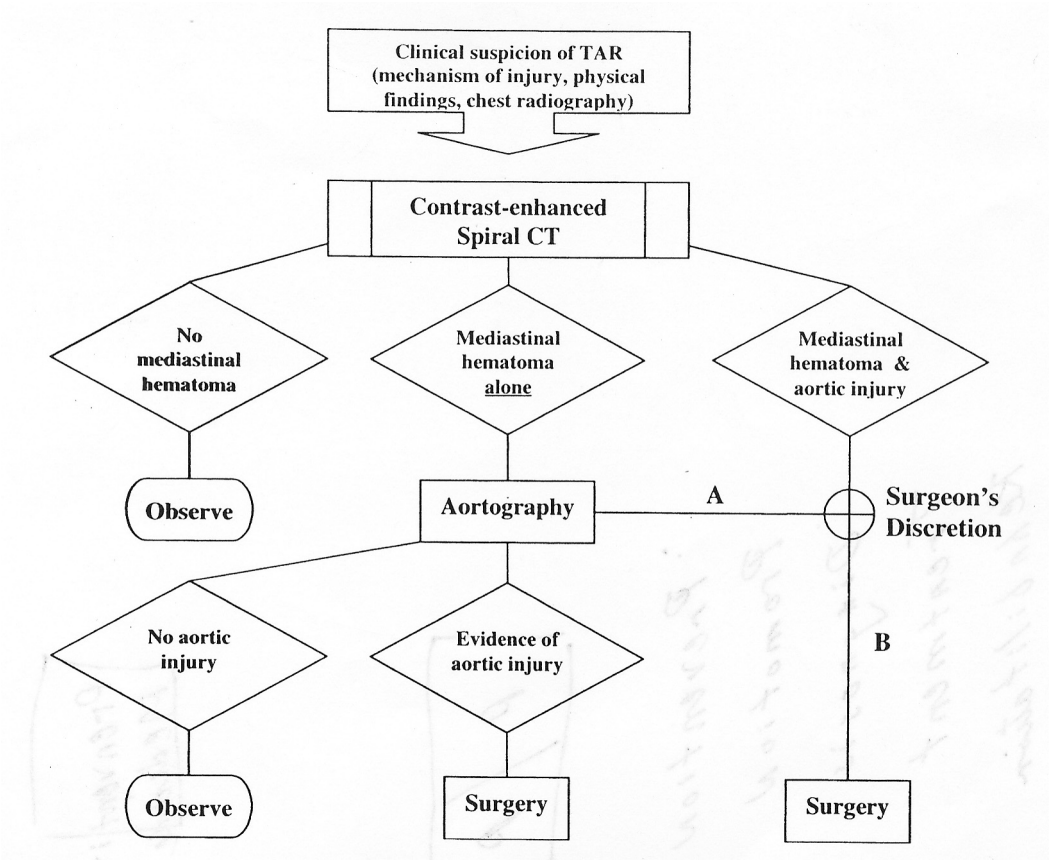


Fig. 20 : Evolving algorithm with CT scan and delayed medical management options.
Diagnostic algorithm for traumatic aortic rupture. Patients with no mediastinal hematoma are observed. Patients with mediastinal hematoma but no definitive sign of aortic injury (a "nonnegative" spiral computed tomograph [CT]) undergo aortography. Patients with mediastinal hematoma and signs of aortic injury proceed either down path A to aortography or path B directly to operation at the surgeon's discretion. (From Downing et al[49])

Addressing massive hemothorax with tube thoracostomy, obtaining two large bore intravenous access and invasive monitoring represent the first line of action. Emergency or urgent thoracotomy is indicated in the initial blood loss of 1,500cc., or 200cc/hour for 2-4 hours[102]. This is better accomplished in the operating room.

Blood products and crystalloid infusion are paramount, but excessive use should be avoided. Massive volume replacement in the multiple injured patient has been

sources include : 5% hypertonic saline (HTS), isotonic crystalloids, artificial colloids, fresh frozen plasma, fresh whole blood, and artificial blood.

The principle of anti-impulse therapy, permissive hypertension, or "intended reduction of aortic wall stress (dP/dT)" by keeping mean arterial pressure between 60-80 mmHg and heart rate <100, principally with β-blocker and vasodilator drugs, should be instituted on all BTAI patients, and continued throughout the clinical course, especially perioperatively on open

surgery or EVSG repairs, in both the adult and pediatric populations[6,48,99,100]. The effects of therapy are illustrated in figure 22[31]. Blood pressure control has become well accepted in the early management of suspected or documented BATI[8]. Yet, caution is warranted with sudden or progressive hypotension, since it may be difficult to distinguish drug effect from volume vasodilation, or bleeding.

and anesthesia induction can increase systemic pressure.

For endovascular techniques, the principle of arterial pressure reduction is not unanimous, and maintaining a mean BP > 70, or systolic pressure of 100-120 is the goal during deployment of the device[110,111,112]. With associated head injuries higher systemic pressures should also be maintained[112,113].

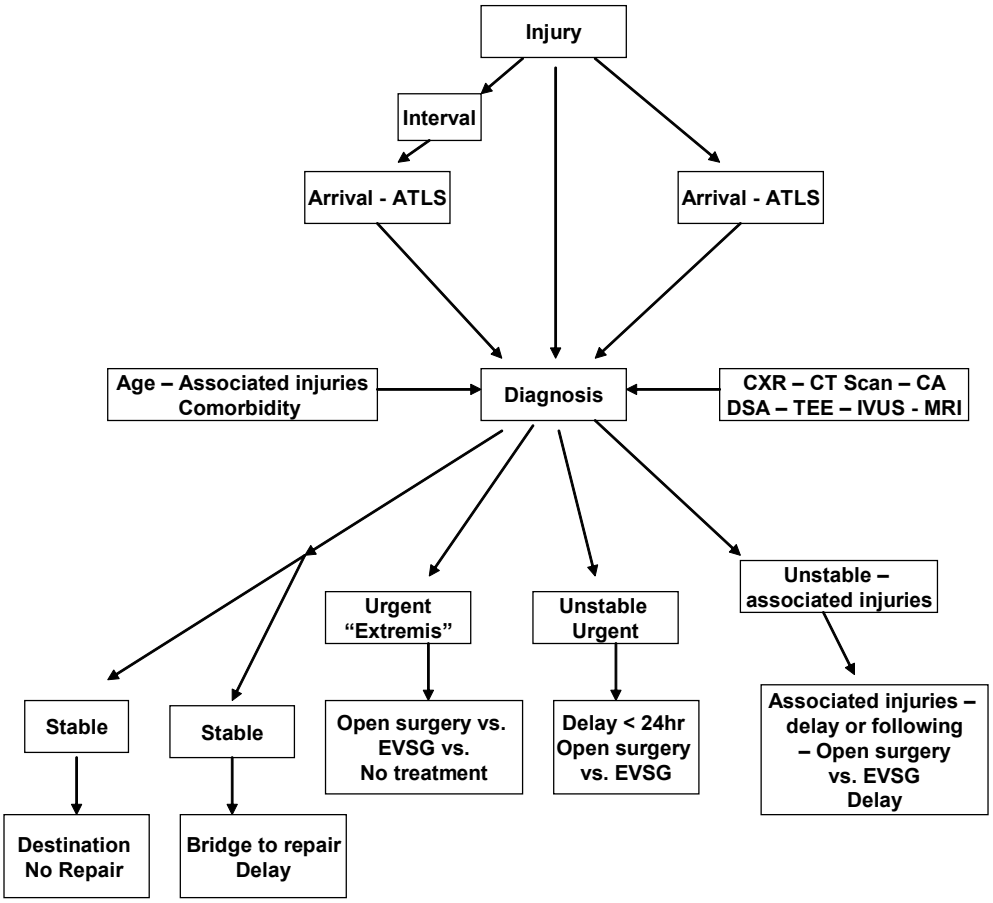


Fig. 21 : Contemporary Algorithm with more diagnostic and EVSG options

The major therapeutic intravenous agents include: esmolol, propranolol, labetalol, sodium nitroprusside, enalaprilat, nicardipine, fenldopam, and nitroglycerin (table 7). Caution re. afterlaod reduction is warranted, since these agents, especially nitroprusside have been implicated in causing spinal cord ischemia[107,108]. Pate et al[109] also cautioned that anti-hypertensive treatment, as well as volume restriction, should be initiated in patients with initial adequate blood pressure and urinary output. Further, treatment should be maintained throughout the diagnostic process since increased stress during transportation, manipulation,

Prioritization of treatment must be considered in patients with BTAI, as multiple associated injuries are frequently present. Camp et al.[70] in 1997 evaluated 395 cases over 11 years from 14 regional trauma centers. 102 cases were categorized as extremis. 99 died shortly after admission, and 3 died after reaching the operating room. BTAI was the primary cause of death in 56 (55%). This means 45% died from associated injuries, thus highlighting the fact that sicker patients are reaching the hospital.

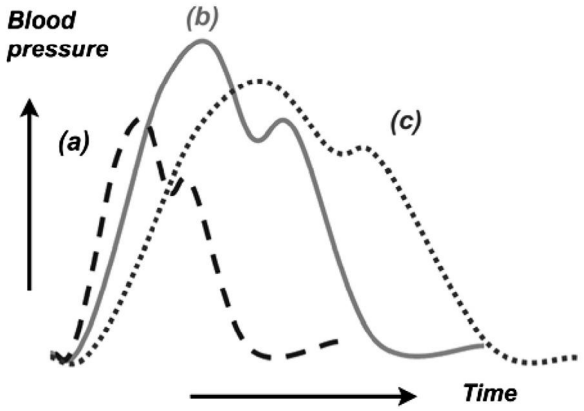
able 7*# : Parental antithypertensive drugs

Drug	Dose	Onset/ Duration
Esmolol (Brevibloc)	Bolus: 500mcg/kg IV, repeat after 5 minutes. Infusion:50-100mcg/kg mn,upto300mcg/kg/min. Titrate to systolic pressure of 100mmHg and heart rate <100 beats/minute	Onset:1-5min Duration:15-30-min
Labetalol (Normodyne)	Bolus : 20mg IV x1,then 20-80mgq10 in to maximum dose 300mg. Infusion : 0.5-2mg/min.	Onset: 5-10 min. Duration: 3-6 hr.
Nitroglycerin	Initial: 5mcg/min. IV Maintenance: titrate q 3-5 min to 100mcg/min	Onset: 2-5 min. Duration: 5-15 min.up
Sodium nitro-prusside (SNP)	Initial: 0.2-0.5 mcg/kg/min continuous IV infusion. Maintenance: titrate to goal BP (systolic 90-100); Up to 8-10 mcg/kg/min continuous infusion	Onset : Seconds Duration : 2-3 min
Enalaprilat (Vasotec)	Initial : 1.25 mg IV over 2-5 min. q 6 hours, then increase up to 5 mgs. q 6 hours.	Onset : 15 min. Duration: 11 hrs.
Nicardipine (Cardene)	Initial: 5mg. /hour continuous infusion, up to 15mgs./hour.	Onset: 10 min. Duration: 2-4 hrs.
Fenoldopam (Corlopam)	Initial: 0.01 mcg./ kg./ min IV, titrated up to 0.3 mcg./kg./min.	Onset: 2-3 min. Duration: 30 min.

*Cooper DH. Hypertensive Emergencies. In: Kollef MH, Bedient TJ, Isakow W, Witt CA.The Washington Manual of Critical Care. Woltors Kluwer/ Lippincott Williams & Wilkins 2008; page 149-150.

Brenner M. Critical Care Medicine, 2007 edition. Current Clinical Strategies Publishing. Laguna Hills, CA. 2007. P. 55.

Major long bone and pelvic fractures are common, with 49% requiring early operative intervention. Pelvic fractures, especially with associated retroperitoneal hematomas, have a high mortality[114]. This injury is particularly significant since operative positioning is hazardous. With respect to concomitant intra-abdominal solid organs injury, the FAST (focused abdominal sonography for trauma) evaluation is extremely valuable in ruling out abdominal injury, as well as pericardial effusions/tamponade. Treatment can be delivered in one of three ways: exploratory laparotomy with delayed repair of the aorta; exploratory laparotomy with subsequent repair of the aorta during the same operation; or aortic repair prior to addressing concomitant solid abdominal organ injuries. Only for hemodynamically stable patients with known BTAI and associated solid organ intraabdominal injury, without ongoing hemorrhage, should repair of the aortic injury take precedence. A positive FAST for pericardial effusion warrants immediate evaluation. This requires a subxyphoid window. If positive, then conversion to a median sternotomy is warranted. Medical anti-shock trousers (MAST) should not be used in patients with suspected BTAI, since increased afterload, and systemic pressure may aggravate the injured aorta[4].



Pharmacologic anti-impulse therapy. Diagram of aortic pressure curves under various conditions. The continuous line (B) represents the baseline state. Administration of a vasodilator agent such as nitroprusside is represented by the dashed curve (A). There is significant decrease in pressure levels and acceleration in heart rate, but this is accompanied by a steepest slope of the ascending portion of the curve (increased dp/dt_{max}). Beta-blockade administration is represented by the dotted line (C). Although the degree of pressure lowering is usually smaller, the drug's negative inotropic and chronotropic effects result in decreased impulse and dp/dt_{max}

Fig. 22 : Pharmacology antipulse therapy diagram of aortic pressure curves

Tatou et al.[115] noted an incidence of 21.3% of BTAI having an abdominal procedure prior to aortic repair. Santaniello et al.[116] concluded that nonoperative management of grade I and II liver and spleen lacerations who undergo systemic anticoagulation for aortic repair (utilizing partial left heart bypass) is a safe approach and associated with no statistically significant impact on transfusion rates, length of stay (LOS), or mortality. Fabian[117] has nicely outlined laparotomy damage control for acute and chronic trauma. He emphasizes the concept of the compartment syndrome where ischemia and soft tissue injury leads to edema and increasing tissue pressures. This requires open abdomen techniques and more required time perioperatively for stabilization.

Cardiac contusion has decreased from 62% to < 9%% of patients with BTAI, yet the role of TEE has been invaluable in determining the extent of cardiac dysfunction, or presence of anatomical injury[7,48,74]. This subgroup of patients suffers higher rates of perioperative arrhythmias, cardiac arrest, ARDS and mortality[118]. The AAST has classified blunt cardiac

injury (BCI) into scales I-VI. Scale I is minor ECG changes (non-specific ST or T wave changes, PAC, PVC, or persistant tachycardia). The extreme scale VI is lethal blunt avulsion of the heart[23].

25% have sustained severe closed head injuries and require intracranial pressure monitoring[7,15.] Closed head injuries accompanying BTAI represent a challenging task, with often increased mortality, and surgical management of BTAI should be addressed after a neurosurgical procedure. Chestnut[113] has nicely reviewed traumatic brain injuries. He points out that cerebral perfusion pressure (CPP) equals mean systemic arterial pressure (MAP) minus intracranial pressure (ICP). ICP is directly proportional to central venous pressure (CVP). Cerebral pressure autoregulation maintains cerebral blood flow (CBF) over a CPP range of 50-150 mm Hg. This highlights the need to maintain higher MAP and lower total body volume. Neurological non-invasive monitoring is helpful in tracking trends and sudden changes (e.g. Cerebral oxygen saturation—Invos Cerebral Oximeter, Somanetics Corp., Troy, Michigan, USA). The goal is to not to precipitate increases in intra-cranial pressure (ICP), particulary with increased volume loading.

If indicated, heparinless or decreased heparin (ACT<150 with heparin coated bypass systems with centrifugal pumps, or endovascular stent-graft techniques) are recommended[113,119]. A shortened delay in the setting of severe CNS injuries may not be sufficient enough for prognostication. Delaying the aortic repair and applying the principle of "intended reduction of aortic wall stress (dP/dT)", with maintaining mean arterial pressure between 60-80 mmHg, may complicate the management of patients with head injuries and impaired cerebrovascular regulatory mechanisms[113].

Delayed (bridge) or Non operative (destination) Treatment

Though urgent intervention of well documented BTAI is the recommended therapeutic approach for unstable patients without associated injuries, there is a subgroup of selected patients who may benefit from delayed aortic repair (defined here as planned delay in intervention any time 14-18 hours from admission to operation or non-operative treatment)[7,8,109]. This also includes patients with delayed admission or transfer >24 hours after injury. As noted, patients with associated injuries, especially severe head injury, cardiac injury, unstable intra-abdominal hemorrhage or extensive pelvic fracture, ongoing sepsis and major burns, severe multi-organ trauma (high ISS) with poor physiologic reserve, massive lung contusion with ARDS and respiratory compromise, coagulopathy and no signs of impending aortic rupture or rapid growth of aortic pseudoaneurysm, are all relative indications for expectant or delayed approaches[100,109,120-122]. In recents years patients with increased ISS, as well as patients with minimal aortic injury (MAI) are being seen. This is a reflection of faster and more effective treatment at the scene, more rapid transfer, improved initial treatment (ATLS), and improved/ rapid diagnostic screening.

The history and evolution of delayed management was empiric and anecdotal. Rice[123] in 1951 recognized hypertension as the cause of death in a pregnant woman with a distant chest injury. The medical treatment (Wheat regimen of antihypertensive therapy) was shown to be effect in type B thoracic dissections in 1965[124]. Aronstam[125] in 1970 treated 2 acute traumatic aortic injuries initially medically, followed by elective repair. Fox et al.[126] in 1979 noted the occurrence of acute hypertension in BTAI. Two theories emerged. The first was the pseudocoarctation syndrome of upper body hypertension. The second and most plausible was the positive feedback spinal reflex mechanism related to thoracic aortic stretch. The presence of sympathetic afferent nerve fibers in the aortic isthmus area responding to stretch with reflex hypertension was postulated as the cause.

Akins et al.[120] from Massachusetts General Hospital popularized the` concept of delayed repair in patients with severe associated injuries as noted above. These injuries were more qualitative than specific[100]. Maggisano et al.[121] developed more quantitative associated injuries. These included :

1- Head injury- GCS <6 or intracranial bleeding on CT scan;

2- Pulmonary injury- PaO2/FiO2 <200, or inability to tolerate one lung ventilation;

3- Cardiac injury requiring pharmacological support;

4- PT and PTT >1.5 normal, despite attempted correction. Holmes et al[122] included an INR >1.5, Platelet count <100, 000, and age > 55 years.

As noted, since that time, individualized approaches have evolved with early repair for both stable and unstable patients, with no prohibitive associated injuries or comorbidities, and delayed repair for unstable patients with severe associated injuries[109,121,127-130]. Through 2000, it is estimated that more than 500 patients have been treated with a delayed management protocol[13]. It is estimated that more than 20% of patients may not be candidates for early repair[122].

The cornerstone of delayed management is the principle of permissive hypotension and "intended reduction of aortic wall stress (dP/dT)" by intravenous β-blockers and the selective use of vasodilatory agents

when necessary (figure 22) (table 7)[31]. Intensive care unit and invasive cardiovascular monitoring (arterial and central venous pressure) for adequate resuscitation and monitoring of adequate tissue perfusion status are fundamental components of delayed repair or non-operative management[109].

Results/ Complications

In a ten-year follow-up of delayed management, Pacini et al.[128] proposed that all trauma patients with BTAI who arrive alive in the ED, without signs of impending aortic rupture or rapid growth of pseudoaneurysm present, should be considered for delayed planned aortic repair after the resolution of all other significant associated injuries. Less than 10 % of their patients required conversion to urgent repair, with decreased mortality to 4.2 % and no recorded spinal cord ischemia, regardless of the type of repair (open with total cardiopulmonary bypass, partial left heart bypass, or endovascular graft stenting).

Wahl et al[85], in their multicenter retrospective review, found no statistically significant mortality benefit to delayed repair, but highlighted a significant increase in length of ICU stay and a two-fold increase in cost-analysis. Both Langanay[129], and Hemmila[130] stress the importance of delayed management in the setting of severe associated injuries, with no resultant danger of interval rupture and death. The recent AAST study revealed increased time interval from diagnosis to treatment (16.5 hours to 54.6 hours). This is related to increased ISS, associated injuries, and planned delayed medical management, including permissive hypotension[7,8].

Pate et al.[109] has championed the delayed approach. In 15 patients surgical repair was performed at 2 days to 6 months. The mortality was 13.3%.11 patients had non operative treatment with 54.5% mortality from associated injuries. In neither group was there spontaneous aortic rupture. There was no pseudoaneurysm enlargement in 4 non operated patients at 18-49 months follow up. Holmes et al.[122] reported 10 nonoperative patients at a median survival of 2.5 years without progression of injury, or need for operation.

A subset of BTAI (10%), involving only an intimal flap (MAI), has been considered an indication for nonoperative treatment with no delayed repair. Kepros et al.[99] found no complications related to the aortic injuries during a mean follow-up of 16.8 months. Malhorta et al.[11] had > 90 % success rate with nonoperative management, no recorded complications, and complete resolution of MAI in 3 weeks for 50 % of patients, and stable pseudoaneurysm formation in 40 %. Hemmila et al.[130] in 2004 compared their series of delayed repair (DR) with early repair (ER), and compared the results with the National Trauma Data Bank (NTDB). DR was established to be 16 hours. Beta blockers were used in the DR group (goal of systolic pressure <120 and heart rate < 100). They noted a decrease in rupture in the treated group to 1.5%, compared to the earlier AAST study of 12% in patients not treated with antihypertension strategy[7]. They also noted increased complications, higher hospital and ICU length of stay (LOS), as well as ventilator time. This was reflective of higher GCS and Head/Neck AIS in DR vs ER groups.

However, the results of initial delayed approach or the long-term natural history of BTAI treated with delayed or nonoperative technique is not known and a cautious surveillance plan should be exercised with serial TEE, MRI, CTA or helical CT during hospitalization and following discharge. Fattori et al.[18] used surveillance MRI. As noted, they found MRI more accurate in assessing the evolution of the periaortic hematoma. Long-term complications include : 1) formation, enlargement or rupture of pseudoaneurysm, 2) thomboembolic events by loose intima or thrombus, 3) progressive dissection of the aortic wall and, 4) fistulization with formation of aorto-bronchial, aorto-esophageal or aorto-pleural fistula. Mortality with this approach ranges from 0 % to 14%[48,71,109,122,128].

(To be continued)