Evolution of lesion-specific management of blunt thoracic aortic injury

Donald G. Harris, MD,[a] Joseph Rabin, MD,[b] Benjamin W. Starnes, MD,[c] Ali Khoynezhad, MD, PhD,[d] R. Gregory Conway, MD,[a] Bradley S. Taylor, MD, MPH,[c,f] Shahab Toursavadkohi, MD,[a,f] and **Robert S. Crawford, MD,[a,f]** *Baltimore, Md; Seattle, Wash; and Los Angeles, Calif*

Developments in diagnosis and treatment have transformed the management of blunt thoracic aortic injuries (BTAIs). For patients in stable condition, treatment practice has shifted from early open repair to nonoperative management for low-grade lesions and routine delayed endovascular repair for more significant injuries. However, effective therapy depends on accurate staging of injury grade and stability to select patients for appropriate management. Recent developments in BTAI risk stratification enable lesion-specific management tailored to the patient and aortic lesion. This review summarizes advances in lesion assessment and treatment and proposes an integrated scheme for the modern management of BTAI. (J Vasc Surg 2016;64:500-5.)

Among blunt trauma patients, thoracic aortic injury is the second leading cause of death after head injury.[1-3] Most victims die in the field,[1,2] and the historical management paradigm for those admitted to the hospital has been framed by the seminal autopsy study of Parmley et al that described invariable progression to aortic rupture.[3] Screening chest radiography for blunt thoracic aortic injury (BTAI) was performed, with subsequent aortography in those suspected of having aortic injury.[1] Because of the perceived high risk of impending rupture, the standard of care for BTAI until recently was early open aortic repair,[4] which was associated with considerable operative morbidity and mortality.[1,4-6]

The management of BTAI has evolved significantly during the past decade, particularly regarding injury diagnosis, medical therapy, and, for patients requiring repair, the selection, timing, and technique for aortic intervention. Computed tomography (CT) angiography enables rapid screening and planning for intervention, whereas anti-impulse therapy with beta-blockade limits injury progression.[1,7] In large part owing to better characterization of specific lesions by CT and the ability to medically stabilize the aorta, nonoperative management of selected BTAIs and delayed repair (after 48 hours from injury) of more significant lesions have become routine and preferred; contrary to historic practice, few aortic injuries undergo early repair.[8-11] These strategies represent a paradigm shift in the understanding of the natural history of BTAI.[12] The purpose of this review is to summarize the evolution of nonoperative and delayed repair strategies for BTAI in adults and recent developments in selection of patients for lesion-specific management.

TRADITIONAL MANAGEMENT OF BTAI

In their extensive autopsy analysis published in 1958, Parmley et al recognized that most blunt aortic trauma led to vessel rupture at the time of injury. For lesions that remained intact, the suggested natural history was progression and subsequent aortic rupture.[3] The latter assumption dictated an aggressive operative approach for patients with BTAI for the next five decades. Accordingly, screening chest radiography was performed for blunt trauma patients presenting after a significant mechanism of injury to evaluate for signs of BTAI, including mediastinal widening, displacement of mediastinal structures, and obscured aortic knob or aortopulmonary window. Contrast aortography was indicated in the presence of such findings to confirm or to exclude BTAI. Unless contraindicated by severe polytrauma or concurrent life-threatening injuries, expeditious surgical repair was standard.[1,4]

From the Division of Vascular Surgery, Department of Surgery,[a] R Adams Cowley Shock Trauma Center,[b] and Division of Cardiac Surgery, Department of Surgery,[c] University of Maryland School of Medicine, Baltimore; the Division of Vascular Surgery, Department of Surgery, University of Washington School of Medicine, Seattle[c]; the Division of Cardiothoracic Surgery, Department of Surgery, Cedars-Sinai Medical Center, Los Angeles[d]; and the Center for Aortic Disease, University of Maryland Medical Center, Baltimore.[f]

Author conflict of interest: B.W.S. is a consultant to Endologix and is a cofounder and shareholder of Aortica Corporation. A.K. is a consultant to Medtronic and principal investigator for Medtronic device studies. B.S.T. is a consultant to Medtronic.

Correspondence: Donald G. Harris, MD, Division of General Surgery, University of Maryland Medical Center, 22 S Greene St, Baltimore, MD 21201-1590 (e-mail: dharris@smail.umaryland.edu).

The editors and reviewers of this article have no relevant financial relationships to disclose per the JVS policy that requires reviewers to decline review of any manuscript for which they may have a conflict of interest.

0741-5214

http://dx.doi.org/10.1016/j.jvs.2015.12.066

Techniques for operative repair of BTAI evolved since Parmley's seminal work.[6] The "clamp and sew" method, although simple and expeditious, was associated with mortality and paraplegia rates of 16% to 31% and 10% to 23%, respectively. The addition of active distal perfusion with cardiopulmonary bypass improved operative outcomes and reduced paraplegia, presumably because of improved spinal cord perfusion pressure during aortic cross-clamping. However, open repair under these circumstances continued to entail substantial in-hospital mortality.[6,13,14] National outcomes for open BTAI repair were evaluated in the first American Association for the Surgery of Trauma (AAST-1, 1994-1996) aortic injury study. Although most repairs were performed using active or passive distal perfusion, 9% of patients developed paraplegia, and inpatient mortality was 31% and was predominantly aortic related.[5] In this context, delayed repair arose as a reasonable alternative to early open repair in patients with severe concurrent injuries or comorbidities associated with prohibitive operative risk.

INITIAL EXPERIENCE WITH DELAYED REPAIR AND NONOPERATIVE MANAGEMENT

Prompt aortic repair on diagnosis was common practice and standard therapy for BTAI through the early 2000s.[4] The average time from admission to thoracotomy was 15 hours in AAST-1, and guidelines from the Eastern Association for the Surgery of Trauma (EAST) recommended early repair unless there were competing management priorities.[4,5] Nonoperative management of patients who had prohibitive comorbidities to undergo thoracotomy or were too unstable from other injuries led to early experience with medical therapy for BTAI. Provided they could tolerate beta-blockade for heart rate and blood pressure control, no patients progressed to aortic rupture awaiting repair, and some remained stable without definitive intervention.[7,15,16]

This observation broadly reflects the natural history of BTAI, namely, most deaths occur at the scene, and patients surviving to admission have been automatically selected for a variable degree of aortic stability.[2,3,17] Indeed, even Parmley recognized "that the natural course of traumatic aortic aneurysms may be prolonged and before surgical treatment is decided upon its risks must be carefully evaluated."[3] Given that most patients with BTAI have significant concurrent trauma, medical therapy either as definitive treatment or as a bridge to delayed repair enabled stabilization and treatment of other life-threatening injuries.[2,5,10,18] In early experiences, no patients receiving blood pressure control progressed to aortic rupture. Aortic-related mortality among patients subsequently undergoing delayed repair was low (0%-14%), and long-term follow-up of those managed nonoperatively reported no late ruptures or lesion progression.[7,15,16]

In more recent surveys, delayed repair became normative and associated with better outcomes. Between AAST-1 and AAST-2, the mean time from admission to aortic repair increased from 15 to 55 hours, and patients having delayed repair in AAST-2 had 65% lower mortality.[5,18] Delayed repair allows stabilization of other critical injuries and may enable repair under optimized circumstances, such as the ability to administer anticoagulation to patients who initially present with major bleeding.

Single-institution series support the benefit and safety of delayed repair and indicate that it may have a specific benefit in patients with BTAI and traumatic brain injury.[13,19,20] Recognizing this, updated EAST guidelines endorsed delayed repair when possible, provided patients are candidates for preoperative anti-impulse therapy.[11] Similarly, nonoperative management of low-grade lesions became common practice. In a recent multicenter study by DuBose et al, one-third of patients with BTAI were managed without aortic repair.[10] In Canada, despite stable incidence, the rate of aortic repair has decreased from 55% in 2002 to 36% in 2010, a trend that coincided with a decrease in BTAI-related mortality.[8] However, safely selecting patients for nonoperative management or delayed repair is challenging and requires accurate assessment of lesion grade and stability.

LESION ASSESSMENT AND RISK STRATIFICATION

Advances in imaging techniques, particularly CT angiography, have helped define BTAI as a continuum demonstrating a spectrum of lesions with varying severity and natural histories. Several characteristics make contrast CT the best screening and diagnostic test for BTAI,[1,11] including 100% sensitivity, specificity that matches or exceeds that of conventional aortography, inclusion in a whole-body protocol to detect other injuries, and ability to plan for thoracic endovascular aortic repair (TEVAR).[1,7,11,21,22] Between AAST-1 and AAST-2, the primary modality for BTAI diagnosis shifted away from aortography (87% in AAST-1) to CT (93% in AAST-2).[5,18] CT is able to detect intimal injuries, which are defined by an intimal defect and no external aortic contour abnormality and represent the most limited type of BTAI.[23] These constitute low-grade or minimal aortic injuries together with intramural lesions.[1,23] Although minimal aortic injuries are diagnosed by modern imaging techniques with increasing frequency, in the era of chest radiographic screening and selective diagnostic aortography, they were clinically occult.[24]

Follow-up imaging of minimal aortic injuries indicates a high rate of resolution and low rate of progression to more severe injury.[23,25-30] For example, in a series of 43 patients with low-grade lesions and medium-term follow-up imaging reported by Osgood et al, 55% of lesions resolved and 40% remained stable. Progression to a higher grade injury occurred in two patients, but late intervention was not required.[27] Similarly, of 91 patients with low-grade BTAI managed nonoperatively in the DuBose study, only one patient required salvage aortic repair and no patients died of aortic rupture.[10] Indeed, current Society for Vascular Surgery guidelines support nonoperative management and radiographic surveillance of intimal lesions.[31]

Table I. Summary of blunt thoracic aortic injury (BTAI) grading scales

	Classification system					
Lesion	*SVS*[31]	*Stanford*[29]	*Presley*[33]	*Vancouver*[28]	*Shock trauma*[35]	*Harborview*[17]
Isolated MH			I			
Intraluminal defect		I				
Intimal injury	I	II	IIa IIb with MH	I: <1 cm II: > 1 cm	I	I
Mural hematoma or defect	II	III			I	
Pseudoaneurysm	III	IV: <50% circumference V: >50% circumference	IIa: <1 cm IIb: <1 cm + MH IIIa: >1 cm IIIb: >1 cm and proximal to LSCA	III	II: <50% circumference III: >50% circumference	II
Rupture/transection	IV		IV	IV	IV	

LSCA, Left subclavian artery; *MH,* mediastinal hematoma; *SVS,* Society for Vascular Surgery.
For each grading system, injury grades, denoted by Roman numerals, are mapped to specific aortic lesions. Blank cells indicate a lesion not included in the scale.

Table II. Risk factors associated with traumatic aortic pseudoaneurysm instability

	Risk factors		
Study	*Admission physiology*	*Pseudoaneurysm size*	*Mediastinal hematoma*
Starnes et al[17]	SBP <90 mm Hg	N/A	>15 mm at the aortic arch
Rabin et al[35]	N/A	≥50% aortic circumference	Hematoma with mass effect, aortic pseudocoarctation, or left hemothorax
Harris et al[39]	Lactate >4 mM	>1.4 × normal aortic diameter	>10 mm at posterior descending aorta

N/A, Not applicable; *SBP,* systolic blood pressure.

More extensive aortic disruption results in formation of a traumatic pseudoaneurysm,[3,32] which is defined by focal, contained aortic dilation resulting in an external contour abnormality.[3,17,32,33] Whereas pseudoaneurysms are associated with aortic-related morbidity and mortality because of greater potential for progression to rupture,[3,10,17] some remain stable and have been successfully managed nonoperatively.[10,16,29,30,34-36] For example, Rabin et al reported a nonoperative management rate of 28% (17/59) for uncomplicated pseudoaneurysms, with no aortic mortality.[35] Similarly, in preliminary data from Osgood et al, 24% (37/153) of patients with pseudoaneurysms were managed medically, although one patient with a larger lesion subsequently ruptured and died.[34] Complete aortic disruption represents the most severe form of BTAI and occurs by primary transection or from rupture of an unstable lesion. Although these patients frequently present in extremis, a contained rupture may stabilize and enable survival to diagnosis and emergency intervention.[28,33,35]

The spectrum of blunt aortic trauma is graded by several CT-based scales that describe the anatomic severity of the injury (Table I).[17,28,29,33,35,37] For example, the system described by Azizzadeh et al and subsequently adopted by the Society for Vascular Surgery has four grades: I, intimal tear; II, intramural hematoma; III, pseudoaneurysm; and IV, rupture.[31,37] Other schemes, such as those by the Vancouver, Presley, Stanford, and Maryland Shock Trauma groups, further stratify pseudoaneurysms according to size.[29,33,35] In contrast, a simplified system was proposed by Starnes et al that classifies injuries as minimal or significant according to the presence of an external aortic contour abnormality.[17]

However, categorizing BTAI by grade does not accurately predict stability or guide therapy.[38] In particular, despite being high-grade injuries, pseudoaneurysms may remain stable. Analyses by the Maryland Shock Trauma and Harborview groups have evaluated BTAI features associated with aortic-related mortality. In a study of clinical and CT characteristics associated with death from aortic injury, Starnes et al identified that hypotension (systolic blood pressure <90 mm Hg) and a large mediastinal hematoma (>15 mm at the aortic arch) were independent risk factors for mortality.[17] Similarly, for patients with large pseudoaneurysms (>50% circumference), Rabin et al found that mediastinal hematoma resulting in mass effect, aortic pseudocoarctation, and extensive left hemothorax were high-risk findings that should prompt urgent aortic repair as soon as feasible on the basis of concurrent management priorities.[35]

Harris et al specifically evaluated factors associated with traumatic aortic pseudoaneurysm instability.[39] Patients with pseudoaneurysms presenting in stable condition who either remained stable without aortic intervention for at least 48 hours or progressed to rupture were

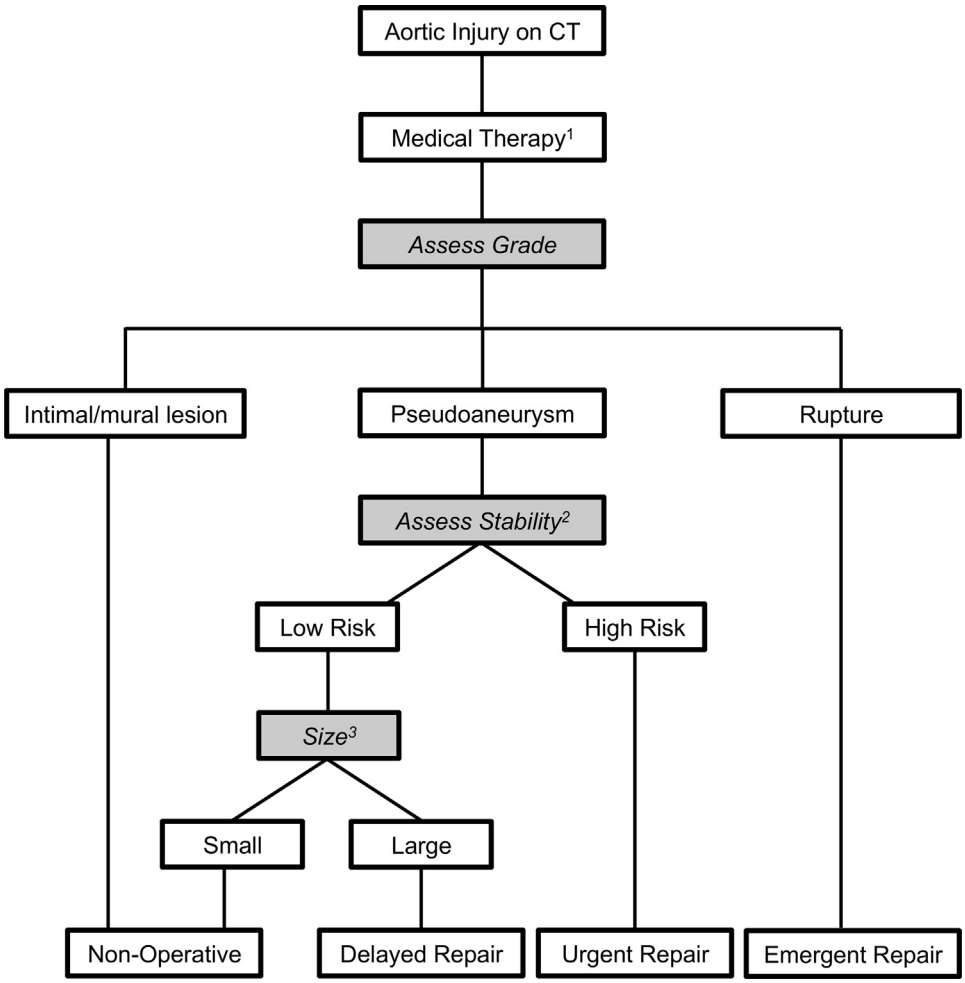

Fig. Suggested management of blunt thoracic aortic injury (BTAI) based on injury staging. [1]Antiplatelet therapy and beta-blockade targeting a systolic blood pressure of 100 to 120 mm Hg and heart rate of 60 to 90 beats/min with additional antihypertensive agents as required for blood pressure control; repeated computed tomography (*CT*) within 48 to 72 hours for patients managed nonoperatively or by delayed repair to assess stability. [2]High-risk factors include two or more of the following: signs of hypotension, large pseudoaneurysm, and extensive mediastinal hematoma (refer to Table II). [3]Small defined as <50% aortic circumference or <1 cm in maximal dimension.

analyzed, enabling comparison of high-grade, stable injuries vs similarly graded but unstable lesions. Analysis of clinical and radiographic variables demonstrated that large pseudoaneurysms (>1.4 × normal aortic diameter), extensive mediastinal hematoma (>10 mm along the descending aorta), and admission serum lactate concentration (>4 mM) were independently associated with subsequent aortic rupture. Patients who progressed to rupture were identified by the presence of two or more of these factors with 100% sensitivity and 84% specificity. The overall accuracy of this risk assessment score was significantly higher than blinded, routine clinical evaluation by trained vascular surgeons.[39]

Integrating these analyses indicates that pseudoaneurysm size, extent of the mediastinal hematoma, and shock are critical risk factors that can be used to assess BTAI stability (Table II). Importantly, these features can be readily ascertained early in the initial trauma evaluation and are clinically relevant. Whereas the primary purpose of stratifying pseudoaneurysm stability is identification of patients with high-risk lesions who may benefit from early intervention, an important secondary outcome is the ability to reliably select patients for delayed repair and nonoperative management, thus extending the benefit of lower procedural morbidity and mortality to patients who may otherwise unnecessarily undergo early repair.

LESION-DIRECTED MANAGEMENT

Management of patients with blunt trauma starts with the primary and secondary trauma surveys and initial resuscitative maneuvers. Contrast CT is indicated in those at risk for BTAI on the basis of the mechanism of injury (Fig).[11] Unless it is contraindicated by other injuries, once diagnosed, medical therapy is instituted to stabilize the patient

until aortic repair or definitive nonoperative therapy. Standard regimens including anti-impulse therapy with beta-blockade to maintain a systolic blood pressure below 100 to 120 mm Hg and heart rate of 60 to 90 beats/min appear to prevent lesion progression and rupture, and anti-platelet therapy may also be considered.[15,17,24,26,27,29,35] Although infrequent, early repair within 48 hours after injury may be warranted in patients who are not candidates for medical therapy, such as those requiring pharmacologic blood pressure support to maintain cerebral perfusion in the setting of intracranial hypertension.[17,20]

The treatment of patients with low- or high-grade BTAI is relatively straightforward. As previously discussed, intimal and intramural lesions can be managed nonoperatively with follow-up imaging. At the other end of the spectrum, aortic rupture is immediately life-threatening. These patients often present in extremis or decompensate in the trauma bay, but tamponade within the mediastinum can enable survival to diagnosis and treatment. Although mortality is high in this group, emergency aortic repair is indicated and may salvage a significant portion of these patients.[3,10,33,35,39]

Pseudoaneurysms represent an intermediate lesion for which optimal management requires individual assessment and further evaluation of aortic stability and lesion size. Large lesions associated with high-risk features are relatively unstable, and urgent aortic repair in these patients may be beneficial and prevent death from rupture.[17,35,39] Absent such features, the need for and timing of repair remain uncertain. Selective nonoperative management of traumatic pseudoaneurysms occurs in 15% to 40% of major series; for small pseudoaneurysms, this strategy appears safe, provided proper medical therapy, radiographic surveillance, and ready access to follow-up care are available.[16,17,25,29,34,35,40] The cutoff for defining this category is uncertain, but a size <50% aortic circumference and <1 cm are consistent parameters associated with safe nonoperative management, although the long-term safety of this approach warrants dedicated study.[7,10,15,17,29,35,40] Large but otherwise uncomplicated pseudoaneurysms may still be at greater long-term risk for enlargement and rupture, and these patients may benefit from delayed repair,[34,35] usually during the index admission.

Regardless of timing, TEVAR is the primary and preferred intervention when aortic repair is required.[8,10,11,18] It is associated with significantly lower mortality, paraplegia, and procedural morbidity rates than open repair,[13,18] and it is the recommended treatment modality for patients undergoing BTAI repair.[11] Whereas heparinization is recommended, TEVAR can be performed without anticoagulation in patients in whom major bleeding is a concern, although with a risk for thrombotic complications.[31,35] Coverage of the left subclavian artery appears well tolerated in patients with BTAI, with low rates of stroke or need for subclavian revascularization.[10,41] After TEVAR, patients require standard clinical and radiographic follow-up, with imaging obtained before hospital discharge and at 1-, 6-, and 12-month intervals followed by annual surveillance thereafter. Open repair still has a role for patients with severe lesions not amenable to endovascular coverage or as salvage therapy after TEVAR[10,11] and is optimally performed with distal bypass.[5,6] More broadly, transfer to an aortic referral center may be considered because repair at a high-volume center is associated with fewer complications.[18]

For patients managed nonoperatively, the duration of medical therapy and optimal surveillance regimen remain uncertain. Neither may be required if the aorta heals. For lesions that persist, routine follow-up imaging before hospital discharge and at 1 month, 6 months, and 12 months and then annually is reasonable. Magnetic resonance aortography may be a useful alternative to CT, especially in the relatively younger trauma population who may have more significant risk for malignant disease if lifelong serial CT studies are required.[31] Finally, given potential concerns about compliance and follow-up diligence among trauma survivors, successful selection for long-term nonoperative management requires a thorough discussion about and plan for long-term care.

Whereas these studies may be used to refine the selection of patients for appropriate management, they have important limitations. All are retrospective, single-institution analyses limited to adult patients, and the underlying data may be prone to selection bias. None of the risk factors have been validated externally or in a prospective fashion, which, along with longer term outcomes and surveillance, should be an avenue of further research. Additional insight may be obtained from novel techniques, such as magnetic resonance angiography with shear stress analysis, to better characterize lesion stability.

CONCLUSIONS

Routine nonoperative management and delayed repair strategies represent a transformative change in the management of BTAI. Initial experience with medical management of patients at prohibitive risk for open aortic repair has evolved to enable primary nonoperative management for minimal aortic injuries, including small stable pseudoaneurysms. Recent studies have helped define high-risk features that may be used to assess the stability of larger lesions for urgent repair. As such, comprehensive BTAI staging by assessment of grade, stability, and size enables lesion-directed management and optimal care for patients with aortic injury.

AUTHOR CONTRIBUTIONS

Conception and design: DH, JR, RSC
Analysis and interpretation: DH, JR, BS, AK, BT, ST, RSC
Data collection: DH, JR, RGC, RSC
Writing the article: DH, RGC, RSC
Critical revision of the article: JR, BS, AK, BT, ST
Final approval of the article: DH, JR, BS, AK, RGC, BT, ST, RSC
Statistical analysis: Not applicable
Obtained funding: Not applicable
Overall responsibility: RSC

REFERENCES

1. Neschis DG, Scalea TM, Flinn WR, Griffith BP. Blunt aortic injury. N Engl J Med 2008;359:1708-16.
2. Dosios TJ, Salemis N, Angouras D, Nonas E. Blunt and penetrating trauma of the thoracic aorta and aortic arch branches: an autopsy study. J Trauma 2000;49:696-703.
3. Parmley LF, Mattingly TW, Manion WC, Jahnke EJ. Nonpenetrating traumatic injury of the aorta. Circulation 1958;17:1086-101.
4. Nagy K, Fabian T, Rodman G, Fulda G, Rodriguez A, Mirvis S. Guidelines for the diagnosis and management of blunt aortic injury: an EAST Practice Management Guidelines Work Group. J Trauma 2000;48:1128-43.
5. Fabian TC, Richardson JD, Croce MA, Smith JS, Rodman G, Kearney PA, et al. Prospective study of blunt aortic injury: multicenter trial of the American Association for the Surgery of Trauma. J Trauma 1997;42:374-83.
6. Cardarelli MG, McLaughlin JS, Downing SW, Brown JM, Attar S, Griffith BP. Management of traumatic aortic rupture: a 30-year experience. Ann Surg 2002;236:465.
7. Fabian TC, Davis KA, Gavant ML, Croce MA, Melton SM, Patton JH, et al. Prospective study of blunt aortic injury: helical CT is diagnostic and antihypertensive therapy reduces rupture. Ann Surg 1998;227:666.
8. de Mestral C, Dueck A, Sharma SS, Haas B, Gomez D, Hsiao M, et al. Evolution of the incidence, management, and mortality of blunt thoracic aortic injury: a population-based analysis. J Am Coll Surg 2013;216:1110-5.
9. Demetriades D, Velmahos GC, Scalea TM, Jurkovich GJ, Karmy-Jones R, Teixeira PG, et al. Blunt traumatic thoracic aortic injuries: early or delayed repair—results of an American Association for the Surgery of Trauma prospective study. J Trauma 2009;66:967-73.
10. DuBose JJ, Leake SS, Brenner M, Pasley J, O'Callaghan T, Luo-Owen X, et al. Contemporary management and outcomes of blunt thoracic aortic injury: a multicenter retrospective study. J Trauma Acute Care Surg 2015;78:360-9.
11. Fox N, Schwartz D, Salazar JH, Haut ER, Dahm P, Black JH, et al. Evaluation and management of blunt traumatic aortic injury: a practice management guideline from the Eastern Association for the Surgery of Trauma. J Trauma Acute Care Surg 2015;78:136-46.
12. Rabin J, Harris DG, Drucker C, Bhardwaj A, June A, Taylor BS, et al. The evolution of management strategies for blunt aortic injury. Curr Surg Rep 2016;4:1-10.
13. Estrera AL, Gochnour DC, Azizzadeh A, Miller CC, Coogan S, Charlton-Ouw K, et al. Progress in the treatment of blunt thoracic aortic injury: 12-year single-institution experience. Ann Thorac Surg 2010;90:64-71.
14. Von Oppell UO, Dunne TT, De Groot MK, Zilla P. Traumatic aortic rupture: twenty-year metaanalysis of mortality and risk of paraplegia. Ann Thorac Surg 1994;58:585-93.
15. Pate JW, Gavant ML, Weiman DS, Fabian TC. Traumatic rupture of the aortic isthmus: program of selective management. World J Surg 1999;23:59-63.
16. Holmes JH, Bloch RD, Hall RA, Carter YM, Karmy-Jones RC. Natural history of traumatic rupture of the thoracic aorta managed non-operatively: a longitudinal analysis. Ann Thorac Surg 2002;73:1149-54.
17. Starnes BW, Lundgren RS, Gunn M, Quade S, Hatsukami TS, Tran NT, et al. A new classification scheme for treating blunt aortic injury. J Vasc Surg 2012;55:47-54.
18. Demetriades D, Velmahos GC, Scalea TM, Jurkovich GJ, Karmy-Jones R, Teixeira PG, et al. Operative repair or endovascular stent graft in blunt traumatic thoracic aortic injuries: results of an American Association for the Surgery of Trauma Multicenter Study. J Trauma 2008;64:561-71.
19. Di Eusanio M, Folesani G, Berretta P, Petridis FD, Pantaleo A, Russo V, et al. Delayed management of blunt traumatic aortic injury: open surgical versus endovascular repair. Ann Thorac Surg 2013;95:1591-7.
20. Rabin J, Harris DG, Crews GA, Ho M, Taylor BS, Sarkar R, et al. Early aortic repair worsens concurrent traumatic brain injury. Ann Thorac Surg 2014;98:46-52.
21. Mirvis SE, Shanmuganathan K, Buell J, Rodriguez A. Use of spiral computed tomography for the assessment of blunt trauma patients with potential aortic injury. J Trauma 1998;45:922-30.
22. Nguyen D, Platon A, Shanmuganathan K, Mirvis SE, Becker CD, Poletti PA. Evaluation of a single-pass continuous whole-body 16-MDCT protocol for patients with polytrauma. AJR Am J Roentgenol 2009;192:3-10.
23. Gunn ML, Lehnert BE, Lungren RS, Narparla CB, Mitsumori L, Gross JA, et al. Minimal aortic injury of the thoracic aorta: imaging appearances and outcome. Emerg Radiol 2014;21:227-33.
24. Malhotra AK, Fabian TC, Croce MA, Weiman DS, Gavant ML, Pate JW. Minimal aortic injury: a lesion associated with advancing diagnostic techniques. J Trauma 2001;51:1042-8.
25. Paul JS, Neideen T, Tutton S, Milia D, Tolat P, Foley D, et al. Minimal aortic injury after blunt trauma: selective nonoperative management is safe. J Trauma 2011;71:1519-23.
26. Kepros J, Angood P, Jaffe CC, Rabinovici R. Aortic intimal injuries from blunt trauma: resolution profile in nonoperative management. J Trauma 2002;52:475.
27. Osgood MJ, Heck JM, Rellinger EJ, Doran SL, Garrard CL, Guzman RJ, et al. Natural history of grade I-II blunt traumatic aortic injury presenting over 10 years at a single institution. J Vasc Surg 2013;59:334-42.
28. Lamarche Y, Berger FH, Nicolaou S, Bilawich AM, Louis L, Inacio JR, et al. Vancouver simplified grading system with computed tomographic angiography for blunt aortic injury. J Thorac Cardiovasc Surg 2012;144:347-54.
29. Caffarelli AD, Mallidi HR, Maggio PM, Spain DA, Miller DC, Mitchell RS. Early outcomes of deliberate nonoperative management for blunt thoracic aortic injury in trauma. J Thorac Cardiovasc Surg 2010;140:598-605.
30. Forman MJ, Mirvis SE, Hollander DS. Blunt thoracic aortic injuries: CT characterisation and treatment outcomes of minor injury. Eur Radiol 2013;23:2988-95.
31. Lee WA, Matsumura JS, Mitchell RS, Farber MA, Greenberg RK, Azizzadeh A, et al. Endovascular repair of traumatic thoracic aortic injury: clinical practice guidelines of the Society for Vascular Surgery. J Vasc Surg 2011;53:187-92.
32. Richens D, Field M, Neale M, Oakley C. The mechanism of injury in blunt traumatic rupture of the aorta. Eur J Cardiothorac Surg 2002;21:288-93.
33. Gavant ML. Helical CT grading of traumatic aortic injuries: impact on clinical guidelines for medical and surgical management. Radiol Clin North Am 1999;37:553-74.
34. Osgood MJ, Heck JM, Rellinger EJ, Garrard CL, Naslund TC. Selective nonoperative and delayed operative approaches for the management of grade III blunt traumatic aortic injury. J Vasc Surg 2015;61:119S-20S.
35. Rabin J, DuBose J, Sliker CW, O'Connor JV, Scalea TM, Griffith BP. Parameters for successful nonoperative management of traumatic aortic injury. J Thorac Cardiovasc Surg 2014;147:143-50.
36. Forcillo J, Philie M, Ojanguren A, Le Guillan S, Verdant A, Demers P. Outcomes of traumatic aortic injury in a primary open surgical approach paradigm. Trauma Mon 2015;20:33-9.
37. Azizzadeh A, Keyhani K, Miller CC, Coogan SM, Safi HJ, Estrera AL. Blunt traumatic aortic injury: initial experience with endovascular repair. J Vasc Surg 2009;49:1403-8.
38. Heneghan RE, Aarabi S, Singh N, Quiroga E, Tran N, Starnes BW. Blunt aortic injury: a call for a new classification system and treatment strategy. J Vasc Surg 2015;62:532-3.
39. Harris DG, Rabin J, Kufera JA, Taylor BS, Sarkar R, O'Connor JV, et al. A new aortic injury score predicts early rupture more accurately than clinical assessment. J Vasc Surg 2015;61:332-8.
40. Tanizaki S, Maeda S, Matano H, Sera M, Nagai H, Nakanishi T, et al. Blunt thoracic aortic injury with small pseudoaneurysm may be managed by nonoperative treatment. J Vasc Surg 2016;63:341-4.
41. Khoynezhad A, Donayre CE, Azizzadeh A, White R. One-year results of thoracic endovascular aortic repair for blunt thoracic aortic injury (RESCUE trial). J Thorac Cardiovasc Surg 2015;149:155-61.

Submitted Jul 31, 2015; accepted Dec 19, 2015.