Traumatic rupture of the descending thoracic aorta

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Summary

The management of acute traumatic rupture of the descending thoracic aorta at Groote Schuur Hospital between January 1984 and December 1989 is reviewed. Aortic rupture was diagnosed angiographically in 18 of 150 patients (12%), who underwent aortography because this injury was suspected. However, 3 of these patients had false-positive angiograms. The diagnosis was initially missed in 31% of patients, and this contributed to morbidity and mortality. Simple aortic cross-clamping ($N = 8$) was used before September 1988 and 3 patients died — 1 intra-operatively from cardiac arrhythmia and 2 postoperatively, where major peri-operative haemorrhage had occurred. In contrast, partial heparin-less bypass ($N = 5$) using a centrifugal vortex pump was used after September 1988, and there were no haemorrhagic or paraplegic complications or mortality in this group. This technique is safe and appears to be superior to simple aortic cross-clamping in managing this condition.

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The incidence of acute traumatic rupture of the thoracic aorta has increased over the past few decades, coincident with an increase in high-speed motor vehicle accidents (MVA).[1] At autopsy, ruptured aortas are found in 13 - 16% of fatally injured MVA victims, and the incidence is higher in those ejected from the vehicle.[2,3] These fatalities constitute the 70 - 85% immediate mortality group of patients with aortic rupture; most die before reaching hospital.[1,3] If the diagnosis is missed at the admitting hospital, the majority of the remaining patients will die within 2 weeks.[1] In a minority chronic aneurysms will form; these tend to become symptomatic or rupture fatally within 5 years of the initial injury.[4] It is therefore imperative that all medical personnel treating victims of severe blunt trauma be cognisant of this condition.

The surgical management of these patients remains controversial, particularly with regard to preventing peri-operative paraplegia. At Groote Schuur Hospital before September 1988 patients were managed by simple aortic cross-clamping without augmentation of the distal circulation. Thereafter, partial heparin-less bypass with a centrifugal vortex pump (Bio-Medicus, Minneapolis, Minnesota, USA) was used.

The case records of all patients diagnosed as having acute traumatic rupture of the descending thoracic aorta from January 1984 to December 1989 were analysed retrospectively. Information regarding the accident was obtained from hospital records, operation reports and direct communication with surviving patients or their relatives.

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Patient characteristics and mechanism of injury

During this 6-year period at Groote Schuur Hospital 150 patients underwent aortography because acute traumatic rupture of the thoracic aorta was suspected; it was diagnosed in 18 patients (12%). However, in 3 of these patients the diagnosis was false; a previously reported ductal diverticulum[5] and non-aneurysmal fusiform dilatations of the aorta were found at thoracotomy in these patients. One patient underwent surgery without pre-operative angiography. Therefore, 16 patients had confirmed rupture; they were predominantly male and their ages ranged from 13 to 69 years — all had been involved in road traffic accidents (Table I).

TABLE I. PATIENT CHARACTERISTICS, MECHANISM OF INJURY AND ASSOCIATED INJURIES IN PATIENTS WITH ACUTE TRAUMATIC RUPTURE OF THE THORACIC AORTA DIAGNOSED AT GROOTE SCHUUR HOSPITAL, JANUARY 1984 - DECEMBER 1989

Age (yrs)	**13 - 69 (mean 29,4)**
Sex	
M	**12**
F	**4**
Mechanism of injury	
Pedestrian vehicle accident	
Motor cycle accident	**3**
Motor vehicle accident	
Ejected	**6**
Not ejected	**1**
Associated injuries ($N = 16$)	
Cerebral trauma ($N = 12$)	
Severe	**5**
Moderate	**7**
Orthopaedic trauma ($N = 13$)	
Lower limb fractures	**9**
Upper limb fractures	**5**
Pelvic fractures	**7**
Spinal fractures	**0**
Abdominal trauma ($N = 7$)	
Ruptured bowel	**2**
Ruptured left diaphragm	**2**
Ruptured bladder	**2**
Renal contusion	**7**

The majority of patients were resuscitated and assessed in the trauma unit of Groote Schuur Hospital; 5 were transferred from outlying hospitals because of chest radiographs suggestive of a ruptured thoracic aorta (Fig. 1). The most frequent radiographic signs noted were: widening of the mediastinum > 8 cm at the aortic knob (100%); obliteration of the aorto-pulmonary window (94%); and left apical pleural cap (87,5%). The other radiographic features associated with this diagnosis are listed in Table II.

TABLE II. INCIDENCE OF RADIOGRAPHIC SIGNS SUGGESTIVE OF TRAUMATIC RUPTURE OF THE DESCENDING THORACIC AORTA

Radiographic sign	No. of patients
Wide mediastinum > 8 cm at aortic knob	16
Obliteration of aortopulmonary window	15
Left apical pleural cap	14
Oesophageal deviation to right demonstrated by nasogastric tube	5*
Depression of left main bronchus > 40° from horizontal	11
Tracheal deviation to right	10
Left pleural effusion	5
Fractures of sternum or upper 3 ribs	2

Sixteen patients had confirmed rupture and the number of patients having these signs on initial chest radiograph is shown. However, none of these signs are specific for the diagnosis.
* This could only be assessed in 6 patients, in whom a nasogastric tube had been inserted pre-operatively, on admission.

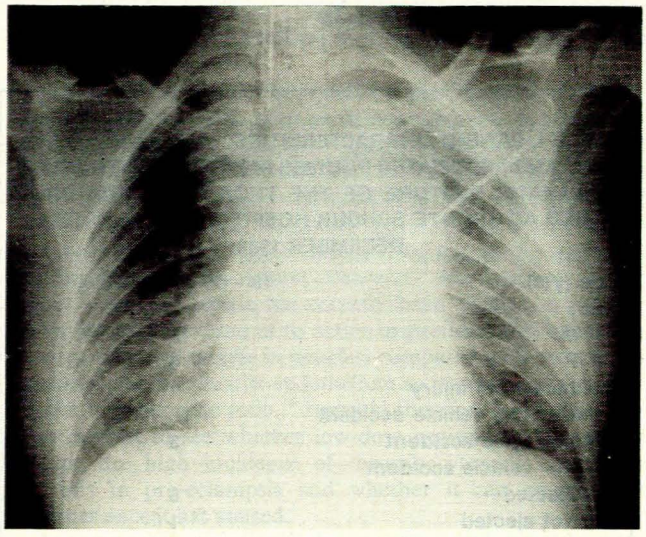

Fig. 1. Chest radiograph of a patient with traumatic rupture of the descending thoracic aorta. The common radiographic signs — a wide mediastinum, left apical pleural cap, loss of aortic knob and obliteration of the aortopulmonary window — are easily seen.

Associated injuries and timing of surgery

All patients had multiple injuries (Table I), and only 4 were haemodynamically stable on admission. Cerebral injuries were present in 75% of patients, and this resulted in 1 pre-operative death within 3 hours of admission. In addition, 1 patient required evacuation of a progressing extradural haematoma before management of the aortic injury.

Orthopaedic injuries were common (81%), and 2 patients had orthopaedic procedures before management of the aortic injury. In 1 patient this antecedent management was because the aortic injury was initially missed. Seven patients (44%) required laparotomy. In 3 patients this was done first, again because the aortic injury had not been diagnosed.

Therefore, in summary, the diagnosis was initially missed (for 15 hours - 5 days) in 5 patients (31%), 4 of whom successfully underwent other surgical procedures before the aortic surgery. However, in the remaining misdiagnosed patient, the diagnostic delay probably contributed to his pre-operative death. This patient bled to death 46 hours after the accident, immediately after aortography. Once diagnosed, no patient died as a result of elective delay in order for transfer to Groote Schuur Hospital ($N = 4$) or as a result of attending to more urgent associated injuries ($N = 2$).

In the majority of patients ($N = 10$) aortography and surgery were undertaken within 12 hours of injury.

Surgical technique

A left posterolateral thoracotomy, entering the pleural cavity above the 5th rib, was performed. Double-lumen endotracheal tubes were used whenever possible. While attempting not to enter the mediastinal haematoma, the descending thoracic aorta and left subclavian artery were carefully dissected and encircled with tapes.

One patient with a circumferential tear died at the onset of surgery while we were attempting to obtain proximal and distal aortic control, and is not included in the analysis of surgical techniques (Table III).

TABLE III. OUTCOME OF 16 PATIENTS WITH TRAUMATIC RUPTURE OF THE DESCENDING THORACIC AORTA AND SURGICAL TECHNIQUE USED

Initial deaths ($N = 3$)		
Severe cerebral trauma		1
Exsanguination after aortography (diagnosis missed for 46 h)		1
Exsanguination at commencement of surgery (inability to obtain proximal aortic control)		1
Simple aortic cross-clamping ($N = 8$)		
Deaths		
Intra-operative cardiac arrest on cross-clamping		1
Postoperative, consequential to major secondary haemorrhages requiring re-operation		2
Pre-operative paraplegia		1
Postoperative		
Paraplegia	No paralysis	
3	4	
Cross-clamp time (min)		
35 - 70 (mean 46,7)	19 - 25 (mean 21,75)	
Interposition grafts		
2	2	
Partial heparin-less bypass ($N = 5$)		
Pre-operative paraplegia		1
Postoperative		
Paraplegia	No paralysis	
1	4	
Cross-clamp time (min)		
26	17 - 37 (mean 24,5)	
Interposition grafts		
1	3	

Simple aortic cross-clamp (8 patients)

In this group, the aortic repair was managed by simple aortic cross-clamping without augmentation of the distal circulation. One patient died intra-operatively as a result of a cardiac arrhythmia during the cross-clamp period, and a further 2 patients died in the early postoperative period. Major anastomotic haemorrhage necessitating re-exploration contributed to these delayed deaths.

The ischaemic cross-clamp time ranged from 19 minutes to 70 minutes (mean 32,4 minutes). Paraplegia was documented

postoperatively in 3 patients, and all had cross-clamp times > 35 minutes (Table III). However, 1 patient had *pre-operative* evidence of partial paresis.

At repair, incomplete aortic tears were found in 5 patients and circumferential tears in 3 patients. If totally transected the aortic ends were retracted for up to 6 cm, therefore these were repaired with interposed Dacron grafts (14 - 22 mm diameter), as were 2 incomplete tears.

Partial heparin-less bypass (5 patients)

In this group of patients, a centrifugal vortex pump with standard non-heparin-bonded tubing was used to pump blood from the left atrium or ascending aorta to the descending thoracic aorta or left femoral artery. At the onset of the procedure a 'non-heparinising' dose of heparin (3 000 - 5 000 U) was administered intravenously in some patients because of concern regarding possible left atrial thrombus formation.

There were no deaths in this group and no patient developed paraplegia after aortic repair, although 1 patient was *pre-operatively* documented to have complete paralysis at the level of T 10 (Table III). The ischaemic cross-clamp time in this group ranged from 17 minutes to 37 minutes (mean 25 minutes), and there were no haemorrhagic complications. Moreover, the majority of this group ($N = 3$) had complete transections, and 1 patient had two sequential circumferential tears separated by an intervening 2 cm portion of aorta. All except 1 patient with an incomplete 90% tear were managed by interposing a prosthetic graft of Dacron (20 mm diameter) or Goretex (18 mm).

Complications independent of surgical techniques

One left phrenic nerve palsy and 3 left recurrent laryngeal nerve palsies were diagnosed postoperatively in patients who could be assessed ($N = 11$). These complications were not related to the use of partial bypass or the insertion of interposition grafts.

No patient developed prosthetic graft sepsis, despite potentially septic associated injuries, such as compound fractures and ruptured bowel.

Discussion

All acute thoracic aortic ruptures during this 6-year period at Groote Schuur Hospital were the result of MVAs and occurred at the aortic isthmus. This region, between the origin of the left subclavian artery and the ligamentum arteriosum, has been postulated to be inherently weaker and prone to tearing because of a transition in the degree of fixity of the aorta.[6] In this study 94% of patients were either ejected from moving vehicles or motor cycles, or were pedestrians struck by a high-speed vehicle. The mechanism of injury thus involved violent uncontrolled upward and forward trajectory paths when flung through the air, followed by sudden deceleration and impact on the head or chest area. This high incidence of aortic rupture in persons ejected from a vehicle has been previously reported by Greendyke.[2] Although not seen in our series, alternative mechanisms such as falls from a height, direct sternal blows, etc. can also produce aortic rupture.[7]

Delayed diagnosis due to the injury initially being missed was noted in 31% of our cases, and contributed to 1 pre-operative death. Thus a high index of suspicion for aortic rupture must be maintained by medical personnel treating victims of severe trauma. The radiographic features suggestive of this injury that we noted are neither specific for this injury, nor prerequisites for requesting angiography.[8] In fact, a normal admission radiograph has been reported in 27% of patients, especially in the elderly.[9] Thus either specific radiographic features or a history of violent uncontrolled acceleration/deceleration events should alert the physician to the possibility of this injury and thus the need for an aortic angiogram. Our 12% incidence of positive angiograms in patients suspected of having this condition is acceptable in order not to miss this lethal injury, as are the negative thoracotomies (16,7%) owing to false-positive angiograms.

The management of traumatic rupture of the aorta deserves the highest priority because of its poor natural history.[1,4] However, progressive cerebral or intra-abdominal bleeding may occasionally have to be controlled first. The risk of exsanguination from secondary delayed rupture of the contained mediastinal haematoma is high,[1] and was the cause of 1 pre-operative death in this series. If surgical repair needs to be electively delayed, e.g. for transportation to another centre, it is absolutely essential that post-resuscitation hypertension, which is common, is appropriately controlled with a combination of sodium nitroprusside and ß-blockade.[10] The ß-blocker is thought essential in order to decrease the systolic ejection force that is postulated to be the cause of progression of dissecting aortic aneurysms and complete rupture in those patients with contained traumatic rupture.[11]

The controversial aspect highlighted in our study is the occurrence of paraplegia and the need to provide protection for the spinal cord. The traumatic injury itself can cause pre-operative paraplegia and this was documented in 2 of our patients. Flexion forces causing the aortic injury are also transmitted to the spinal cord and its arteries, and this can cause neurological damage and vascular thrombosis. In addition, the aortic tear may cause an acute 'coarctation syndrome' with distal ischaemia, as seen in 2 patients, 1 of whom developed pre-operative paresis and 1 acute pulmonary oedema.

Paraplegia complicated the surgical procedure in 20% of surviving patients. It developed only in those managed by simple aortic cross-clamping who had long (> 30 minutes) ischaemic cross-clamp times. Paraplegia did not develop in the partial bypass group, although only 1 patient had a cross-clamp time > 30 minutes. The association of paraplegia and cross-clamp times of > 30 minutes has been previously documented,[12,13] and adjunctive measures such as pre-operative administration of intrathecal papaverine has been suggested in order to improve collateral spinal blood flow.[14] However, intrathecal papaverine is not uniformly effective and therefore augmentation of distal blood flow with shunts or partial cardiopulmonary bypass is still an accepted alternative. Unfortunately, bypass does not entirely prevent paraplegia, since major radicular arteries may be incorporated within the cross-clamped portion of the aorta. Therefore surgery must still be performed expeditiously, collateral vessels preserved, and the distal aortic cross-clamp should be applied as close as possible to the area of transection.

Nevertheless partial bypass also decreases the high myocardial 'after-load' associated with aortic cross-clamping,[15] which might have contributed to the fatal arrhythmia seen in the patient managed by simple cross-clamping.

Systemic heparinisation is not required for partial bypass with the centrifugal vortex pump, in contrast to cardiopulmonary bypass with a roller pump and oxygenator.[16] Moreover, heparinisation is contraindicated if there are associated craniocerebral injuries, documented in 75% of our cases. Thromboembolic, cannulation or bleeding complications were not seen when this technique was used. If partial bypass is used, it is started before any aortic dissection is performed. A note of caution should, however, be mentioned — the vortex pump is

non-occlusive when not in motion, therefore it will allow retrograde blood flow from the distal aorta to the left atrium if lines are unclamped while the centrifugal pump is not rotating. There is therefore a risk of cerebral air embolism if air bubbles (possibly introduced when connecting the tubing) are present in the lines.

In 50% of our operative cases the ruptures were transverse, linear and circumferential with retraction of both ends of the intima and media for up to 3 - 6 cm. In agreement with others,[12,17] total transections were managed by using interposition prosthetic grafts rather than direct suture approximation. Although this might incur a longer ischaemic time, we believe that a tensionless anastomosis is safer. In this series major peri-operative haemorrhagic complications only occurred in patients who had direct suture repair. The completeness of rupture or the presence of multiple tears (noted in 1 patient), and thus the need for graft interposition is also rarely predictable pre-operatively. This further emphasises that a short cross-clamp time of < 30 minutes cannot always be guaranteed before cross-clamping the aorta.

No patient had a septic complication related to the prosthetic graft, despite associated injuries commonly thought to increase the risk of sepsis. Therefore other potentially septic injuries should not delay management of the aortic rupture, as has been suggested by Akins *et al.*[10]

The axiom 'more haste, less speed' is most appropriate in cardiovascular surgery, since the complexity of the surgery often increases after intra-operative mishaps. This might well have contributed to the major peri-operative bleeding complications seen, which were only documented in the simple cross-clamp group of patients. Although not a randomised study, we advocate partial heparin-less bypass as the technique of choice in managing this condition. In addition to the advantages discussed above, it also allows for partial neutralisation and removal of 'time stress' during the operative procedure and, additionally, few surgeons can guarantee a short cross-clamp time before starting the repair.

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REFERENCES

1. Akins CW. Major injuries: aortic rupture. In: Grillo HC, Eschapasse H, eds. *International Trends in General Thoracic Surgery*. Vol. 2: Major Challenges. Philadelphia: WB Saunders, 1987: 268-272.
2. Greendyke RM. Traumatic rupture of the aorta: special reference to automobile accidents. *JAMA* 1966; **195**: 527-530.
3. Smith RS, Chang FC. Traumatic rupture of the aorta: still a lethal injury. *Am J Surg* 1986; **152**: 660-663.
4. Finkelmeier BA, Mentzer RM, Kaiser DL, Tegtmeyer CJ, Nolan SP. Chronic traumatic thoracic aneurysm: influence of operative treatment on natural history: an analysis of reported cases, 1950-1980. *J Thorac Cardiovasc Surg* 1982; **84**: 257-266.
5. Von Oppell UO, Beningfield SJ, Odell JA, Reichart B. Cardiac rupture caused by blunt trauma as well as false angiographic aortic rupture. *S Afr Med J* 1988; **74**: 519-520.
6. Sevitt S. The mechanisms of traumatic rupture of the thoracic aorta. *Br J Surg* 1977; **64**: 166-173.
7. Parmley LF, Mattingly TW, Manion WC, Jahuke EJ. Non-penetrating traumatic injury of the aorta. *Circulation* 1958; **17**: 1086-1101.
8. Woodring JH, Dillon ML. Radiographic manifestations of mediastinal hemorrhage from blunt chest trauma. *Ann Thorac Surg* 1984; **37**: 171-178.
9. Gundry SR, Williams S, Burney RE, Cho KJ, Mackenzie JR. Indications for aortography in blunt thoracic trauma: a reassessment. *J Trauma* 1982; **22**: 664-671.
10. Akins CW, Buckley MJ, Daggett W, McIlduff JB, Austen WG. Acute traumatic disruption of the thoracic aorta: a ten-year experience. *Ann Thorac Surg* 1981; **31**: 305-309.
11. Wheat MW. Acute dissecting aneurysms of the aorta: diagnosis and treatment, 1979. *Am Heart J* 1980; **99**: 373-387.
12. Antunes MJ. Acute traumatic rupture of the aorta: repair by simple aortic cross clamping. *Ann Thorac Surg* 1987; **44**: 257-259.
13. Katz NM, Blackstone EH, Kirklin JW, Karp RB. Incremental risk factors for spinal cord injury following operation for acute traumatic aortic transection. *J Thorac Cardiovasc Surg* 1981; **81**: 669-674.
14. Svensson LG, Stewart RW, Cosgrove DM *et al*. Intrathecal papaverine for the prevention of paraplegia after operation on the thoracic or thoracoabdominal aorta. *J Thorac Cardiovasc Surg* 1988; **96**: 823-829.
15. Pate JW. Traumatic rupture of the aorta: emergency operation. *Ann Thorac Surg* 1985; **39**: 531-537.
16. Hess PJ, Howe HR, Robicsek F *et al*. Traumatic tears of the thoracic aorta: improved results using the Bio-Medicus pump. *Ann Thorac Surg* 1989; **48**: 6-9.
17. Langlios J, De Brux JL, Binet JP, Khoury W. Major injuries: traumatic aortic rupture. In: Grillo HC, Eschapasse H, eds. *International Trends in General Thoracic Surgery*. Vol. 2: Major Challenges. Philadelphia: WB Saunders, 1987: 273-284.