

REVIEW

Diagnosing traumatic rupture of the thoracic aorta in the emergency department

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Thoracic aortic rupture (TAR) is recognised as a cause of death in victims of blunt trauma. Immediate mortality is 85% but in the group who survive to reach hospital there is a reasonable chance of successful surgical repair. TAR can be remarkably occult and the emergency physician is paramount in making the initial diagnosis. If suggestive, but often subtle features are not recognised in the early phase they will go undetected until full rupture and death occurs. This article reviews the mechanism of injury and describes the signs and symptoms of TAR in the acute phase. Features suggestive of TAR on the initial primary survey chest radiograph are described. The use of this film as a screening tool, and of other imaging modalities, is discussed.

usefulness of various imaging modalities available.

It does not discuss the treatment of TAR; this remains a surgical remit, but rather considers the difficulty of diagnosis, a challenge that can perplex even the most experienced emergency physician.

INCIDENCE

As far back as 1557 Vesalius reported a necropsy finding of aortic rupture presumed to be traumatic (cited by Sailer in 1942).¹ For centuries this was assumed to be a rarity, universally immediately fatal and therefore of little interest to the medical world. Then the era of high speed transportation arrived and brought with it a changing pattern of traumatic injury. During the first world war, aortic rupture was often noted in the victims of airplane crashes. Shortly afterwards in 1947 Strassman reported 72 cases of aortic injury among 7000 necropsies performed in Manhattan between 1936 and 1942 (a rate of about 1%).² As cars became more widely available the lesion became more common. By 1966 Greenadyke reported on the

Rupture of the thoracic aorta is not infrequent in critically injured patients. Most victims die at scene but for those who do reach hospital, if the diagnosis is made, surgical repair is possible.

Clinicians continue to be surprised by how such a catastrophic lesion is accompanied by so few reliable symptoms and signs. As with all traumatic injury, the emergency physician combines an understanding of the mechanism of injury, knowledge of the suggestive signs and symptoms, with accurate interpretation of the primary survey chest radiograph. The challenge with this injury is that although the injury is uncommon, the suggestive mechanism of injury is extremely frequent. Clinical examination is sometimes helpful, but may be normal with the injury remaining occult until a second fatal bleed occurs. Interpretation of the supine chest film is difficult. Clues to the presence of thoracic aortic rupture (TAR) may be subtle and missed on initial review. The opposite situation is more common. Positive plain film findings may be misleading and apparent widening of the mediastinum noted during resuscitation may lead to a change in treatment priority; while appropriate this may occur at the expense of other life threatening injury.

This review investigates the incidence, mechanism of injury, clinical features, and plain film radiological signs of TAR.

The exsanguinating patient needs immediate laparotomy and thoracotomy but for the haemodynamically stable patient imaging is appropriate. Therefore this article also reviews the

“remarkable frequency with which aortic rupture is encountered in patients dying of traumatic injury”.³

In his series, 420 victims of accidental death underwent necropsy—40 had sustained aortic rupture (a rate of about 10%), 35 of which were automobile accidents. He stated that one in six of all victims of fatal automobile accidents sustained aortic rupture. More recent figures from the Central Statistics Office in Ireland record, that among victims of road traffic accident, “injury to blood vessels of thorax” resulted in an average of 12 deaths per year for the years 1995–1998. This represents about 3% of all road traffic deaths.

NATURAL HISTORY

In his landmark paper in 1958, Parmley looked at 296 cases of blunt aortic injury among young soldiers and found that about 15% survive long enough to get to hospital.⁴ The significance of traumatic thoracic aortic rupture to emergency physicians had been established.

Abbreviations: TAR, thoracic aortic rupture; CT, computed tomography; CCT, conventional computed tomography; TOE, transoesophageal echocardiography; SCT, spiral computed tomography; MOI, mechanism of injury

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Of the 15% that survive to get to hospital, 99% will die without surgical intervention. Fifteen per cent survive only the first hour, 30% die within six hours, 49% die within 24 hours, 72% within eight days, and 90% within four months.⁵

A traumatic pseudoaneurysm is formed in survivors. In most cases this undergoes secondary rupture resulting in delayed death but even before surgical intervention there were rare reports of long term survivors in the literature.⁶

SURGICAL REPAIR

Successful early surgical repair was first reported by Passaro in 1959.⁷ In 1976 Kirsh reported a surgical success rate of 70% based on a 10 year experience.⁸ The American Association of the Surgery of Trauma supported this figure and in 1997 quoted an overall mortality rate of 31% based on a multi-centre trial.⁹ Therefore once diagnosed the patient with TAR has a reasonable chance of survival. Surgery remains the only treatment for this injury. A discussion on surgical technique is beyond the scope of this review.

The challenge for all those involved in the resuscitation of the victim of blunt trauma is to establish the diagnosis.

MECHANISM

Mechanism of injury (MOI) remains the most important factor in establishing the diagnosis—falls from >10 feet, motor vehicle crashes at speeds >30 mph, unrestrained drivers, ejected passengers, and pedestrians struck by motor vehicles. A recent article emphasised that severe crush can also cause TAR—the authors reported a patient who sustained rupture of the aortic isthmus having been pinned to a wall by his truck.¹⁰ In his paper in 1978 Mattox found that 27 of 28 patients with definite aortic rupture had been involved in a road traffic accident.¹¹ Most victims of blunt trauma will not have sustained TAR but unreliable clinical signs means a high index of suspicion should be based on MOI. Indeed some authors feel that all victims of significant decelerating trauma should undergo definitive aortography.¹²

Blunt trauma can damage the thoracic aorta by several mechanisms. A displaced thoracic vertebral fracture can cause direct shearing injury to the aorta. Bony intrusion by the first rib and clavicle can cause “osseous pinch” or bony compression of the aortic isthmus.^{13 14}

The aorta is also be damaged by indirect forces. Tremendous pressure can build up within the aorta at the moment of impact (water hammer effect) and rupture is caused by an explosive outburst.^{15 16} The most commonly quoted cause of rupture is the differential forces set up within the chest by deceleration in either the horizontal or vertical plane. The descending aorta remains fixed to the posterior chest wall, while the heart and ascending aorta swing forward and tear free at the isthmus. Parmley and others found the aortic isthmus to be by far the most common site of rupture.^{3 4} Other sites include rupture of the ascending aorta and avulsion of vessels off the arch. Most of those who survive to hospital sustain rupture at the isthmus.

Tears begin in the intima and progress outwards through the media and adventitia. Ayella *et al* reported a series of 36 TAR, 60% of which only involve intima and media, leaving adventitia intact.¹⁷ Surprisingly, complete transection is compatible with survival. In his series Parmley discovered that 40% of those that survived for a time sustained complete transection.⁴

CLINICAL FEATURES

Symptoms of TAR include chest pain (quoted as the most common in a review by Symbas), dyspnoea, back pain, hoarseness, dysphagia, and cough.

Signs include anterior chest wall contusion, unexplained hypotension, upper limb hypertension, or acute coarctation syndrome (decreased lower limb pulses with normal upper limb pulses), differences in pulse amplitude, and a systolic murmur audible over the base of the heart or between the scapulae. Symbas described a frequently occurring triad of

- Increased blood pressure in the upper limbs.
- Decreased blood pressure in the lower limbs.
- Widened mediastinum on rdiography.¹⁸

Many reviewers have concluded that the presence of certain signs and symptoms are useful indicators that urgent investigation is required, but their absence does not exclude the diagnosis.¹⁹ Parmley reported 30% of patients with TAR will have no external signs of injury to the chest and 75% will have fractures of bones other than ribs thus distracting attention away from the chest injury.⁴ In a recent review of 54 cases of TAR, all had other serious injury—17% neurological, 60% abdominal, 35% had pelvic fracture, and 65% had fractures elsewhere.²⁰

Because of this dearth of reliable clinical signs or symptoms and the frequency of distracting injury, emergency clinicians have come to rely on radiographic imaging in the management of these patients.

IMAGING

Plain film

The importance of routine chest radiograph during the initial examination of the trauma patient was first emphasised in the 1960s.²¹ In the 1980s ATLS enshrined a chest radiograph as part of the trauma series, taken during the primary survey assessment of the trauma victim.

In 1953 the first description of the “roentgen” signs of aortic rupture was found in the literature.²² The most important sign was felt to be widening of the mediastinum. A widened mediastinum occurs when a traumatic pseudoaneurysm changes the contour of the mediastinum or more commonly when mediastinal haemorrhage or haematoma occurs. However, Ayella in a review of 149 cases of mediastinal haematoma, found the aortic adventitia to be intact in 60% of cases. Therefore the haematoma could not be from aortic leak; rather it came from small arteries and veins within the mediastinum.¹⁷ Mediastinal widening is not specific for TAR.

However, Gundry *et al* reported the results of a panel of radiologist and surgeons, blinded to outcome, reviewing the chest films of 149 trauma victims who subsequently went on to have aortography to rule out TAR. They found mediastinal widening to be correctly interpreted in 89% of instances in which TAR was present and decided it was the most reliable sign of TAR.²³

Most authors acknowledge that supine radiograph in the resuscitation room results in a magnified mediastinal image. This is a significant problem for the emergency physician as the apparently wide mediastinal image seen on chest radiograph during the primary survey may misrepresent the underlying abnormality. Radiographic techniques vary in the degree of magnification they produce. Work in the 1970s tried to define a widened mediastinum. Marsh found all the patients with TAR in his series had a mediastinum of greater than 8 cm at the level of the aortic knob.²⁴ In 1979 Sturm looked at 18 patients with TAR and found the average mediastinal width to be 8.8 cm.²⁵ These figures have long been quoted as the upper limit of normal for mediastinal width. This work was performed at a time when a history of trauma did not significantly change the way a chest radiograph was taken. The patient was lifted to place the x ray

cassette beneath them or simply sat up for an erect film.^{17 26} This minimised magnification.

With the advent of spinal immobilisation, the distance from the patient's mediastinum to the x ray cassette (object to film distance or OFD) became longer and the distance from x ray machine to cassette (focus to film distance or FFD) shorter. The primary survey chest radiograph is now always supine. All these factors have resulted in greater magnification, making assessment in the resuscitation room more difficult.

Recent work investigated some of these issues.²⁷ Gleeson *et al* showed how modern trauma room techniques magnify the normal mediastinum up to a width of 10–12.5 cm. This negates the usefulness of figures quoted by Marsh and Sturm and reduces the usefulness of the chest radiograph in screening for TAR. Gleeson *et al* went on to show how a minor change in radiographical technique resulted in less magnification and a more useful image. Using these new techniques they produced a range of new upper limits (7.38 cm–9.68 cm) for mediastinal width in the resuscitation room. TAR can occur within these limits (indeed as noted below a proportion of TAR results in no mediastinal widening) but the techniques described by the authors result in a more useful mediastinal image.

In 1981 Seltzer retrospectively compared the plain chest films of 20 patients with confirmed TAR to 20 patients with blunt chest trauma but no TAR.²⁸ He introduced the concept of the M/C ratio, that is the ratio of mediastinal width to chest width at the level of the aortic arch. He concluded that mediastinal widening was a sensitive but non-specific sign of TAR. He defined that an M/C ratio of >0.20 would pick up 100% of cases but would only be 15% specific; a ratio of >0.25 would be 95% sensitive and 75% specific and >0.28 would be 85% sensitive and 100% specific. Sefczek retrospectively reviewed the chest films of 54 trauma patients referred for angiography to rule out TAR. He validated Seltzer's work and felt that the difficulty of evaluating the mediastinum on a supine radiograph was overcome by using the M/C ratio.²⁹

Marnocha *et al* disagreed.³⁰ They looked at 54 patients, 10 of whom had surgically confirmed rupture. The mean M/C ratio for those with rupture was 0.386 and for those without was 0.359 ($p > 0.29$). Marnocha suggested differences in radiographic techniques (he standardised the FFD to 101.6 cm) might have accounted for the disparate results. Although this would have resulted in a different scale of ratios, it does not obviate the fact that Marnocha found the M/C ratio to be unreliable in confirming or excluding aortic rupture.

Several authors have made the point that it is not just the actual width but rather the clinicians overall impression of the mediastinal contour that is important.^{12 24} In speaking to radiologists and clinicians this seems to be the most common approach.

Mediastinal abnormality remains the cornerstone for screening for TAR. Mirvis has shown that aortography is normal in at least 98% of blunt chest trauma victims who do not have mediastinal abnormality.²⁶

In his extensive work in 1990, Woodring reviewed 52 articles covering 656 patients with blunt traumatic rupture of the aorta or brachiocephalic arteries.¹² Some 92.7% of these had an abnormal mediastinum on initial radiograph thus permitting early detection of the vascular injury. Mediastinal abnormalities include

- widened mediastinum
- abnormal aortic outline
- opacification of the aortopulmonary window
- downward displacement of the left mainstem bronchus
- deviation of trachea to the right of midline

- deviation of nasogastric tube to the right of midline
- widened right paratracheal stripe

Some 7.3% of patients had a normal mediastinum on their initial radiograph. This appears to occur when the traumatic pseudoaneurysm is not accompanied by associated mediastinal haemorrhage or haematoma formation, and the pseudoaneurysm is either small or is situated in such a way that does not change the mediastinal contour.

The use of accessory clinical or radiographic signs would have permitted early detection of a further 5.6% of reported cases. Other signs quoted include

- large haemothorax
- first and second rib fractures
- left apical cap
- sternal fracture
- posteriorly displaced clavicular fracture
- multiple rib fractures with a crushed chest
- brachial plexus injury
- diminished or absent pulses or blood pressure in upper extremities
- systolic murmur
- palpable supraclavicular haematoma
- unexplained haematoma

Using any one of these signs as an indication for more invasive imaging, results in a very high negative aortography rate of 80–90%. This is deemed acceptable by most authors.^{23 29 31}

Others have made some attempts to reduce the number of aortograms requested by looking at combinations of specific signs. Mirvis *et al* indicated that when the aortic arch and descending aorta, aortopulmonary window, trachea, and left paraspinal space are normal, there is a 92% probability of no aortic rupture.²⁶ Woodring said that when there is simultaneous absence of deviation of a nasogastric tube and visualisation of a normal right paratracheal stripe there is a 98% probability of no aortic rupture.¹² Marnocha and Maglinte stated that if the aortic arch and its contour are normal and there is no deviation of either nasogastric tube nor trachea then aortic rupture can be excluded.³⁰ But as Woodring pointed out in his meta-analysis any combination of radiological and clinical signs will still miss 1.7% of TAR. Basing your request for definitive imaging on MOI alone is the only way of detecting this remaining group.

Angiography

Aortography has been the gold standard imaging modality for demonstrating TAR. Many authors suggest liberal aortography and aortography based on MOI remains the only way to 100% confirm a normal aorta.¹² Good anatomical detail aids surgical repair.

The angiographic diagnosis of an intimal injury rests on the demonstration of an intimal irregularity or filling defect caused by an intimal flap. The presence of contrast outside the lumen of the aorta is indicative of a transmural laceration, which may be contained (that is, pseudoaneurysm) or a free extravasation (that is, rupture). False negatives can occur but the sensitivity approaches 100%.^{8 26} False negatives reported are of small intimal tears in the descending aorta. Aortography is considered to be 98% specific but incorrect interpretation of anatomical variants, atheromatous plaques, or physiological streaming of contrast can result in unnecessary thoracotomy.

One difficulty with aortography is that it is invasive and requires injection of iodinated contrast material. Because of the volume of dye required some authors report incidences of

full rupture after injection. Concern exists regarding the possibility of the guidewire/catheter penetrating the injured aortic wall. This is not found to be a problem in practice.³² Complications, including puncture site haematoma, anaphylaxis, renal problems, and pseudoaneurysm rupture occur in less than 1%.

The difficulty with aortography lies in the fact that the clinician must assess the risk of transferring a potentially unstable patient to the vascular suite for a prolonged period (60–90 minutes). It may also compromise the investigation and management of other potentially life threatening injuries. Many units in the UK and Ireland who receive trauma patients do not have on site angiography. Therefore the patient would need to be transferred for aortography. This may present difficulties for the multiply injured patient. Of note all units receiving trauma should have on site access to computed tomography (CT).

In an interesting paper, Richardson investigates the subject of potential TAR in the patient with multi-system injury.³¹ He says "There is great risk to the patient if an aortic injury is not detected but considerable time and expense is incurred to exclude the diagnosis if it is not present". As about 85% of aortography is normal, he wondered if the management of other potentially lethal injuries was being compromised. He looked at a series of 408 patients who had suffered multi-system trauma and had a widened mediastinum on initial radiograph. There were seven errors in triage sequence, when an overriding concern for the evaluation of the widened mediastinum led to a delayed diagnosis of intra-abdominal bleeding. He suggested an algorithm for the management of patients with a widened mediastinum, which is based on early diagnostic peritoneal lavage. When diagnostic peritoneal lavage reveals gross blood, laparotomy should precede angiography. Some of these difficulties can be overcome by the consecutive high speed CT of multiple sites.

Transoesophageal echocardiography (TOE)

TOE emerged in 1976 as a modality to complement and possibly replace the use of arch aortography in evaluating the unstable, multiply injured patient with potential aortic disruption. The benefits of TOE include the fact that the study is less invasive, requires no contrast, and can be done at the bedside, only taking 15 minutes to complete. TOE is the study of choice in non-trauma patients with possible aortic abnormalities and initial reports had suggested that it was highly accurate in trauma patients also.^{33–34} In 1995 Saletta *et al* reviewed the use of TOE as "sole" diagnostic modality in 114 consecutive patients with possible aortic trauma.³⁵ They reported a sensitivity of 63% and specificity of 84%. The low sensitivity reflects three missed aortic disruptions all of which had considerably widened mediastinal shadows on chest radiography. In two the correct diagnosis was delayed and all died from massive aortic injury. Disparity exists between results published from different authors. This probably results from the fact that TOE is very user dependent and there is a long learning curve involved in its use, a fact alluded to by Saletta *et al*. Saletta concluded that "while a negative examination may lower the suspicion of injury to the thoracic aorta, it cannot rule it out absolutely". They advise that highly suspicious injuries, unexplained hypotension, or other clinical or radiological findings inconsistent with the TOE findings still warrant further investigation. They recommend arch aortography. Like aortography, immediate TOE is not always available in centres receiving trauma patients. Larger prospective studies on the role of TOE in the evaluation of blunt chest are awaited.

Computed tomography

When conventional CT (CCT) was first introduced into the management of TAR it was used as a screening tool in an

attempt to reduce the rate of negative aortography. Advocates found it efficient to scan the chest along with other systems such as head and abdomen (in which the role of CCT was already established).

Initial reports on the use of contrast enhanced CCT were very positive. Heidberg reported the potential of CCT to identify mediastinal haemorrhage and to depict actual aortic injury.³⁶ Reports of false negatives followed and the role of CCT became very controversial, with some authors advocating its exclusion from the management protocols of these patients.^{12–31–37} Raptopoulos scanned the aortic arch of 326 trauma patients who were already being scanned for abdominal abnormality.³⁸ He found no false negatives but the number of false positives (that is, positive scan and negative aortogram) meant that CCT could only be advocated as a screening tool albeit a better one than plain radiography. The need for aortography was reduced by 56%. Mirvis *et al* re-emphasised the negative predictive value of CCT for excluding TAR (NPV 100%).³⁹ He also clarified the difficulty—CCT is excellent at depicting mediastinal haematoma but less reliable at showing actual aortic injury. As most patients with mediastinal haematoma will not have TAR, aortography will still be required before surgery is contemplated. This need to perform aortography regardless of findings at CCT, initially limited the role of CCT in the triage of critically injured patients.

Gavatt's large study in 1995 marked the advent of contrast enhanced helical or spiral CT (SCT) and the superior visualisation of the aorta itself.⁴⁰ A total of 1518 patients with non-trivial blunt trauma underwent chest SCT scan. Altogether 127 (8.3%) with abnormal SCT findings underwent aortography. Imaging abnormalities were correlated with surgical or clinical outcome. They found helical CT to be even more sensitive than aortography in their study (100% compared with 94.4%) and only a little less specific (81.7% compared with 96.3%) in the detection of aortic injury. They concluded that SCT was effective for screening critically injured patients with possible TAR. They also concluded that patients with mediastinal haematoma but no obvious aortic injury on SCT need not undergo aortography. Aortography was indicated only if SCT was indeterminate or if further definition of the extent of the injury was required preoperatively.

Patel emphasised the technical difficulties encountered in performing high quality helical CT scans and concluded that CT abnormalities are not 100% diagnostic, and so aortography is still necessary.¹⁹ Other authors comment on the ease with which good images were achieved despite difficulties with positioning injured upper limbs.⁴¹ Images can be compiled during one breath cycle, though cardiac motion can cause difficulties.⁴² Scaglione commented that aortic abnormality was no more difficult than any other anatomy on CT and various grades of radiologist were involved in his study.⁴²

In 1998 Wicky reviewed 487 chest SCT, detecting aortic injury in 29% and reporting 100% sensitivity and 99.8% specificity.⁴¹ Scaglione in 2001 reported a large review of 1419 chest SCT after blunt trauma.⁴² They clarified "direct signs" of aortic injury as "intimal flap, pseudoaneurysm, contour irregularity, lumen abnormality or contrast extravasation". They detected 23 of these abnormalities and 21 were confirmed at thoracotomy. The two false positives were movement artefact and most significantly, occurred in the absence of mediastinal haematoma. The authors emphasise the invariable association of mediastinal haematoma when aortic injury has occurred. They conclude that when direct signs of TAR are detected, no further investigation is required before thoracotomy. They advocate caution if signs are subtle and no mediastinal haematoma is present. They conclude

that isolated mediastinal haematoma should lead to a search for its source. If no obvious alternative source is detected aortography is warranted. In 2001 Downing *et al* looked specifically at SCT as a "sole diagnostic method" for TAR.²⁰ They divided 54 cases of TAR into those who had SCT only, those who had confirmatory aortography despite diagnostic SCT, and those who proceeded to aortography because of equivocal SCT findings. Of note 52 of the 54 had simultaneous scanning of another body part. "Positive or diagnostic" chest scans were those with direct signs of aortic injury and associated mediastinal haematoma, "negative" scans had neither, and "non-negative or equivocal scans" showed periaortic haematoma without detectable aortic injury. They found cases with positive scans could proceed safely to thoracotomy without further studies, negative scans outruled aortic injury and only the final group required aortography. There was one false positive in both the SCT and the aortography group: thoracotomy revealed mediastinal haematoma and coincidental ductus diverticulum in both. The authors also reported "time from admission to diagnosis". For those who only had SCT this was 1.7+/-1.7 hours, for those who had aortography despite diagnostic SCT it was 5.2+/-3.0 hours and for those who had aortography because of equivocal SCT findings was 7.9+/-4.5 hours, (p<0.01). They concluded that proceeding to the operating theatre on the basis of diagnostic SCT is safe and expeditious.

CONCLUSION

A high index of suspicion remains important in the detection of TAR. The injury may be difficult to diagnose and if missed is almost always fatal. Mechanism of injury remains an important clinical indicator. Trauma resuscitation must include a thorough hunt for specific clinical signs. Optimal radiographic techniques will improve the diagnostic quality of the resuscitation room chest radiograph. Mediastinal widening remains the most reliably sought sign. Other radiological abnormalities do occur and if the clinician is aware of these, plain chest radiography remains a practical screening tool.

When suggestive clinical features or plain film abnormality are detected in the haemodynamically unstable patient, immediate surgical thoracotomy is required. For the stable patient further imaging is appropriate. The reliability of transoesophageal echo has not been established although it may be of use for screening in centres where the expertise is rapidly available. Aortography is also limited in availability and the logistics entailed make it less suitable for the multiply injured patient. Emergency physicians still use it to assist diagnosis in cases where SCT is equivocal and some cardiothoracic surgeons still request it, to further delineate the injury before repair. The need to perform aortography will lead to significant delays.

Contrast enhanced SCT has been established as the imaging modality of choice for stable patients with a high risk mechanism of injury and clinical features or chest radiological abnormalities suggestive of TAR. Although some authors still advocate caution the role of SCT is progressing from screening tool towards sole diagnostic tool.²²⁻⁴³ A negative SCT excludes TAR. Diagnostic SCT—signs of direct aortic injury with associated mediastinal haematoma—is a safe and speedy basis on which to proceed to thoracotomy. Authors differ on the need for angiography when isolated mediastinal haematoma is detected. It seems sensible that this finding should lead to a search for source. Most authors now include "mediastinal haematoma without obvious source", under the heading of "equivocal" SCT findings. Because of the invariable association of TAR with mediastinal haemorrhage, this group also includes subtle aortic contour findings in the absence of mediastinal haematoma. Those

with equivocal SCT should proceed to aortography. Therefore aortography still has a significant role and both imaging modalities have complementary roles.

It is worth emphasising once again, the remarkably occult nature of TAR. A small percentage of TAR will remain undiagnosed unless we perform SCT based on mechanism of injury alone. This may not be practical but the emergency physician must retain a high index of suspicion when deciding to screen for TAR. The threshold for deciding to perform contrast enhanced SCT of the thorax must be low despite the resulting large number of negative scans.

Departmental trauma guidelines should emphasise identification of those at risk and list plain film findings. All departments receiving trauma should have access to SCT as the imaging modality of choice. If SCT detects abnormality, cardiothoracic involvement is essential. Local protocols compiled by emergency physicians and cardiothoracic surgeons need to establish when and where aortography should be performed.

REFERENCES

- 1 Sailler S. Dissecting aneurysm of the aorta. *Arch Pathol* 1942;**23**:704.
- 2 Strassmann G. Traumatic rupture of the aorta. *Am Heart J* 1947;**33**:508.
- 3 Greendyke RM. Traumatic rupture of the aorta. *JAMA* 1966;**195**:527-30.
- 4 Parmley LF, Mattingly TW, Manion WC, *et al*. Nonpenetrating traumatic injury of the aorta. *Circulation* 1953;**17**:1086-101.
- 5 Beel T, Harwood AL. Traumatic rupture of the thoracic aorta. *Ann Emerg Med* 1980;**9**:483-6.
- 6 Steinberg I. Chronic traumatic aneurysm of the thoracic aorta: report of five cases with a plea for conservative treatment. *N Engl J Med* 1957;**257**:91.
- 7 Passaro E, Pace WG. Traumatic rupture of the aorta. *Surgery* 1959;**46**:787-91.
- 8 Kirsh MM, Behrendt DM, Orringer MB, *et al*. The treatment of acute traumatic rupture of the aorta: a 10-year experience. *Ann Surg* 1976;**184**:308-16.
- 9 Fabian TC, Richardson JD, Croce MA, *et al*. Prospective study of blunt aortic injury: multicenter trial of the American Association for the Surgery of Trauma. *J Trauma* 1997;**42**:374-80.
- 10 Reid C, Livesey SA, Egleston C. Aortic rupture as a result of low velocity crush. *J Accid Emerg Med* 1999;**16**:299-300.
- 11 Mattox KL, Pickard L, Allen MK, *et al*. Suspecting thoracic aortic transection. *JACEP* 1978;**7**:12-15.
- 12 Woodring JH. The normal mediastinum in blunt traumatic rupture of the thoracic aorta and brachiocephalic arteries. *J Emerg Med* 1990;**8**:467-76.
- 13 Crass JR, Cohen AM, Motta AO, *et al*. A proposed new mechanism of traumatic aortic rupture. The osseous pinch. *Radiology* 1990;**176**:645-9.
- 14 Cohen AM, Crass JR, Thomas HA, *et al*. CT evidence for the 'osseous pinch' mechanism of traumatic aortic rupture. *Am J Radiol* 1992;**159**:271-4.
- 15 Coermann R, Dotzauer G, Lange W, *et al*. The effects of the design of the steering assembly and the instrument panel on injuries (especially aortic rupture) sustained by car drivers in head-on collision. *J Trauma* 1972;**12**:715-24.
- 16 Lundevall J. The mechanism of traumatic rupture of the aorta. *Acta Pathol Microbiol Scand* 1964;**62**:34-46.
- 17 Ayella RJ, Hankins JR, Turney SZ, *et al*. Ruptured thoracic aorta due to blunt trauma. *J Trauma* 1977;**17**:199-205.
- 18 Symbas PN, Tyras DH, Ware RE, *et al*. Rupture of the aorta: a diagnostic triad. *Ann Thorac Surg* 1973;**15**:405-10.
- 19 Patel NH, Stephens KE, Mirvis SE. Imaging of acute thoracic aortic injury due to blunt trauma; a review. *Radiology* 1998;**209**:335-48.
- 20 Downing SW, Sperling JS, Mirvis SE, *et al*. Experience with spiral computed tomography as the sole diagnostic method for traumatic aortic rupture. *Ann Thorac Surg* 2001;**72**:495-501.
- 21 Sandor F. Incidence and significance of traumatic mediastinal haematoma. *Thorax* 1967;**22**:43-62.
- 22 Wyman AC. Roentgenologic diagnosis of traumatic rupture of the thoracic aorta. *Arch Surg* 1953;**66**:656.
- 23 Gundry SR, Burney RE, Mackenzie JR, *et al*. Assessment of mediastinal widening associated with traumatic rupture of the aorta. *J Trauma* 1983;**22**:293-.
- 24 Marsh DG, Sturm JT. Traumatic aortic rupture: roentgenographic indications for angiography. *Ann Thorac Surg* 1976;**21**:337-40.
- 25 Sturm JT, Marsh DG, Kenton CB. Ruptured thoracic aorta: evolving radiological concepts. *Surgery* 1979;**85**:363-7.
- 26 Mirvis SE, Bidwell JK, Buddemeyer EU, *et al*. Value of chest radiography in excluding traumatic aortic rupture. *Radiology* 1987;**163**:487-93.
- 27 Gleeson CE, Spedding RL, Harding LA *et al*. The mediastinum—Is it wide? *Emerg Med J* 2001;**18**:183-5.
- 28 Seltzer SE, D'Orsi C, Kirshner R, *et al*. Traumatic aortic rupture: plain radiographic findings. *AJR Am J Roentgenol* 1981;**137**:1011-14.
- 29 Sefczek DM, Sefczek RJ, Deeb ZL. Radiographic signs of acute traumatic rupture of the thoracic aorta. *AJR Am J Roentgenol* 1983;**141**:1259-62.

- 30 **Marnocha KE**, Maglente DDT, Woods J, *et al.* Mediastinal-width/chest-width ratio in blunt chest trauma: a reappraisal. *AJR Am J Roentgenol* 1984;**142**:275–7.
- 31 **Richardson JD**, Wilson ME, Miller FB. The widened mediastinum: diagnostic and therapeutic priorities. *Ann Surg* 1990;**211**:731–7.
- 32 **Fisher RG**, Hadlock F, Ben-Menachem Y. Laceration of the thoracic and brachiocephalic arteries by blunt trauma. *Radiol Clin North Am* 1981;**19**:91–110.
- 33 **Erbel R**, Engberding R, Daniel W, *et al.* Echocardiography in diagnosis of aortic dissection. *Lancet* 1989;*i*:457.
- 34 **Kearney PA**, Smith DW, Johnson SB, *et al.* Use of transesophageal echocardiography in the evaluation of traumatic aortic injury. *J Trauma* 1993;**34**:696.
- 35 **Saletta S**, Lederman E, Fein S, *et al.* Transoesophageal echocardiography for the initial evaluation of the widened mediastinum in trauma patients. *J Trauma* 1995;**39**:137–42.
- 36 **Heiberg E**, Wolverson MK, Sundaram M, *et al.* CT in aortic trauma. *AJR Am J Roentgenol* 1983;**140**:1119–24.
- 37 **Goodman PC**, Jeffrey RB, Minagi H, *et al.* Angiographic evaluation of the ductus diverticulum. *Cardiovasc Intervent Radiol* 1982;**5**:1–4.
- 38 **Raptopoulos V**, Sheiman RG, Phillips DA, *et al.* Traumatic aortic tear: screening with chest CT. *Radiology* 1992;**182**:667–73.
- 39 **Mirvis SE**, Shanmuganathan K, Miller BH, *et al.* Traumatic aortic injury: diagnosis with contrast-enhanced thoracic CT—five year experience at a major trauma center. *Radiology* 1996;**200**:413–22.
- 40 **Gavant ML**, Menke PG, Fabian T, *et al.* Blunt traumatic aortic rupture: detection with helical CT of the chest. *Radiology* 1995;**197**:125–33.
- 41 **Wicky S**, Capasso P, Meuli R, *et al.* Spiral aortography: an efficient technique for the diagnosis of traumatic aortic injury. *Eur Radiol* 1998;**8**:828–33.
- 42 **Scaglione M**, Pinto A, Pinto F, *et al.* Role of contrast enhanced helical CT in the evaluation of acute thoracic aortic injuries after blunt chest trauma. *Eur Radiol* 2001;**11**:2444–8.
- 43 **Collier B**, Hughes KM, Mishok K, *et al.* Is helical computed tomography effective for diagnosis of blunt aortic injury? *Am J Emerg Med* 2002;**20**:558–61.