REVIEW

Non-penetrating cardiac and aortic trauma

Adrian P Banning, Ravi Pillai

Department of
Cardiology, John
Radcliffe Hospital,
Headington, Oxford
OX3 9DU, UK
A P Banning
Department of
Cardiac Surgery
R Pillai
Correspondence to:
Dr Banning.
Accepted for publication
15 May 1997

Blunt, non-penetrating trauma to the heart and great vessels occurs most commonly following road traffic accidents. Other causes include direct blows to the chest, falls from great heights, sporting and industrial injuries, and kicks by animals. The potential mechanisms by which blunt chest trauma can result in cardiac and aortic injury have been described as: direct force against the chest; bi-directional forces against the thorax; increases in thoracic pressure resulting from sudden compression of the abdomen or lower limbs; sudden deceleration; and blast and concussive forces.[1] It is presumed that the frequency of cardiac injury reflects the incidence of such trauma and the relative position of the cardiac chambers within the thorax.

Diagnosis

The actual incidence of cardiac and aortic injury following thoracic trauma is poorly defined. Clinical examination is unreliable as the absence of signs of direct trauma to the chest wall does not exclude significant internal injury, and performing a detailed physical examination in a hectic emergency room can be difficult. In cases of multiple trauma, these diagnostic problems are exaggerated by the urgent need to treat clinically obvious injuries.

In most trauma centres, a chest x ray is used routinely to screen survivors of major trauma for either cardiac or aortic injury. Potential radiographic markers of cardiac and aortic injury include mediastinal widening, sternal or multiple high rib fractures, displacement of the trachea or nasogastric tube, haemothorax, diaphragmatic rupture, apical extrapleural capping, and an indistinct aortic knuckle.[2 3] Unfortunately none of these signs is specific and interpretation of a supine portable x ray can be difficult. ECG and cardiac enzyme estimation are also used as screening tests for myocardial injury following chest trauma. However, several studies have shown a poor correlation between ECG abnormality and either raised creatine kinase MB or evidence of regional wall motion abnormality demonstrated by either echocardiography[4 5] or radionuclide ventriculography.[6]

Aortography is the standard investigation when either clinical or radiographic examination suggests aortic injury. Disadvantages of aortography include its invasive nature, the associated delay and cost, and the limited diagnostic data that it provides as aortography cannot exclude traumatic myocardial injury. Despite these disadvantages the extensive clinical experience that has been accrued using aortography make it the gold standard investigation in most centres for assessing possible aortic trauma (fig 1). Aortographic images are familiar to thoracic surgeons and guidelines for recommending surgical or medical management based upon the results of aortography are well defined.

Transthoracic echocardiography has a limited role in assessing patients with traumatic cardiac injury, but it is of no value when aortic injury is suspected. Difficulty in obtaining diagnostic images is the principal limitation.[5 7] In contrast, transoesophageal echocardiography is safe, rapid, and provides high quality images of the heart and aorta in the majority of trauma patients.[4 8] In experienced hands, transoesophageal echocardiography is the
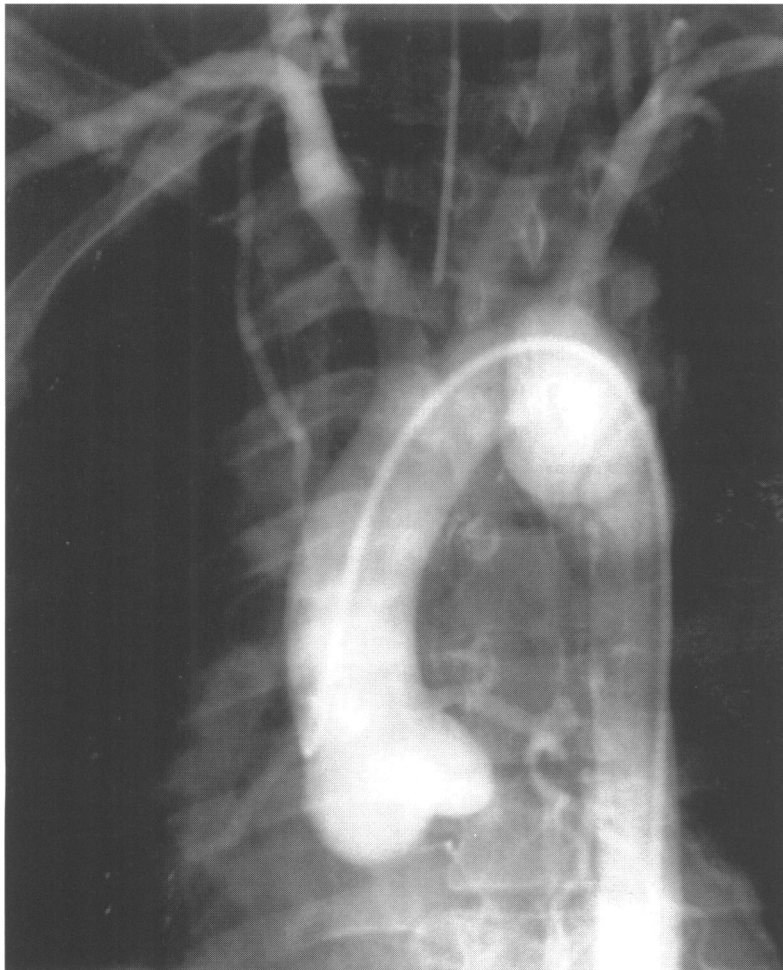

Figure 1 Left anterior oblique projection of an aortogram. There is a localised swelling of upper descending aorta at the level of the isthmus with evidence of extensive intimal disruption.
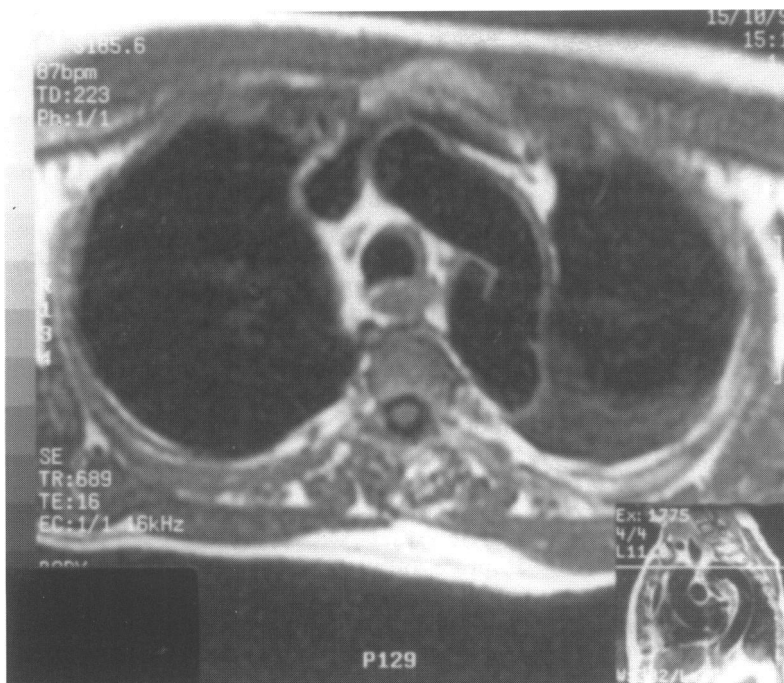
Figure 2   Axial magnetic resonance image demonstrating aneurysm and linear structures in the distal arch proximal descending aorta.

diagnostic technique of choice for assessing traumatic myocardial or valve injury. It is also a sensitive method of detecting aortic injury, however, there is a learning curve for operators,[8 9] and uncertainty persists over the interpretation of some aortic abnormalities demonstrated by transoesophageal echocardiography.[3 9] Minor abnormalities including periaortic haematoma and small mobile intimal flaps may not be prognostically important and are unlikely to be visible by aortography.[8] Thus, before management decisions can be made, careful and expert interpretation of aortic images obtained by transoesophageal echocardiography are necessary.

Magnetic resonance imaging (fig 2) and spiral computed tomography both provide accu-

rate images of the thoracic aorta. Increasing sophistication is improving their ability to provide images of the heart but their general application as a screening tool for cardiac trauma is likely to be restricted because of limited availability and patient access.

Ventricular contusion and myocardial rupture

Ventricular contusion occurs in up to 20% of patients following blunt chest trauma.[5] Right ventricular contusion is most common, presumably because the position of the right ventricle immediately behind the sternum predisposes it to compressive injury. Falls in cardiac output of up to 33% have been demonstrated following experimental acute right ventricular contusion,[10] but clinical observations have demonstrated that long term recovery of systolic function is usually complete and survivors usually have no long term sequelae.[11]

Parmley *et al* described the relative frequency of traumatic cardiac chamber rupture as: right ventricle, left ventricle, right atrium, intraventricular septum, left atrium, and, least commonly, rupture of the intra-atrial septum.[1] This is a reflection of the higher compliance of the atria and their protected posterior position within the thorax. External ventricular rupture is usually fatal. Occasionally patients present in tamponade following contained atrial rupture (fig 3). This occurs most commonly at the junctions of the right atrium with the vena cavae where the heart is relatively fixed within the thorax.

Injury to the pericardium and coronary arteries

Some degree of pericarditis occurs commonly after non-penetrating cardiac injury. This is usually coincidental but occasionally pericardial constriction may develop some years later. Pericardial laceration and rupture occur in fatal cases of severe multiple trauma but rarely in isolation.

Traumatic coronary artery injury resulting in myocardial infarction has been reported. The left anterior descending artery appears to be most commonly involved but accurate diagnosis may be hampered by the limitations of the ECG and the other screening tests in the emergency situation.

Valve injury

Isolated injury to cardiac valves following blunt chest trauma is unusual. Historically, aortic valve disruption was regarded as the most common valve injury[1] but this is probably a reflection of a high incidence of aortic valve injury in fatal cases of severe multiple thoracic injury. Aortic regurgitation may result from avulsion of the valve from the annulus or from laceration of one or more of the aortic cusps. Compressive injury occurring during early ventricular systole is considered to be the usual mechanism of injury.[12] Aortic valve
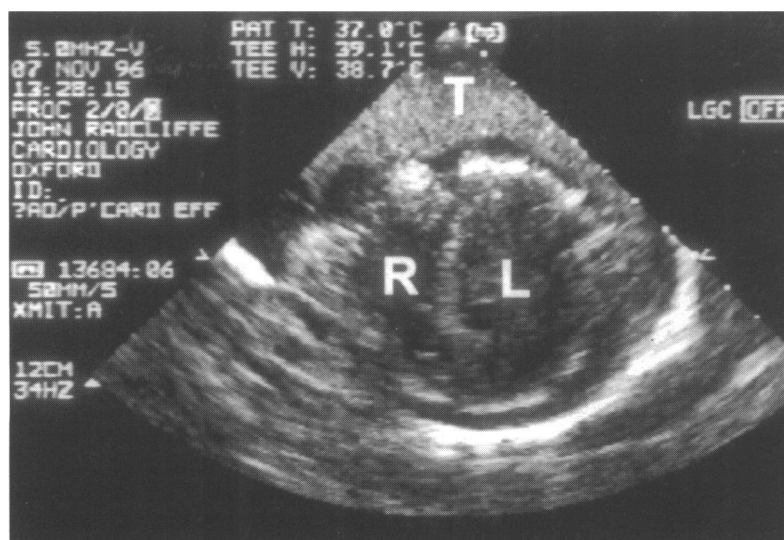

Figure 3   Transoesophageal transgastric transverse cross sectional echocardiographic image demonstrating a large pericardial effusion containing thrombus (T) compressing both right (R) and left (L) ventricles. The patient presented in tamponade following a road traffic accident. Rupture at the junction of the superior vena cava and right atrium was demonstrated during surgery.
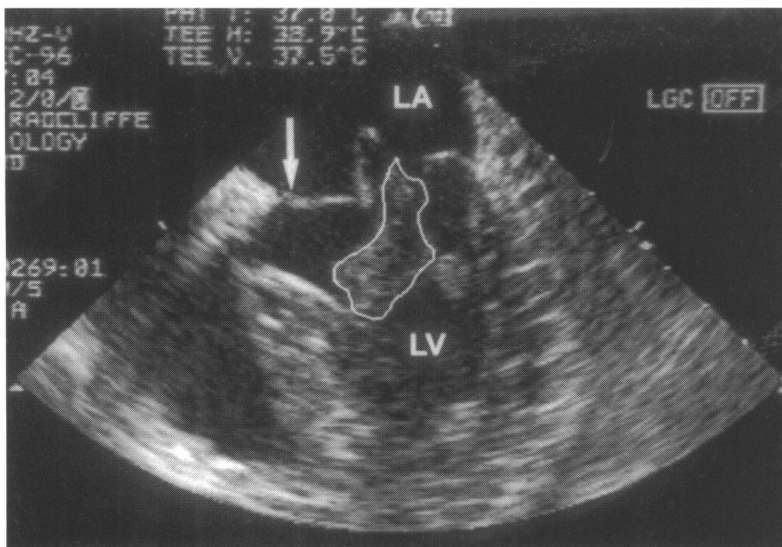
Figure 4   Traumatic avulsion of a mitral valve papillary muscle in a patient who developed severe pulmonary oedema 24 hours after a road traffic accident. Transoesophageal echocardiography demonstrated frank prolapse of both mitral valve leaflets into the left atrium during left ventricular systole (LV). The avulsed papillary muscle (outlined in white) subtending the anterior leaflet of the mitral valve (white arrow) is mobile within the LV cavity.

replacement may be necessary if the extent of the injury makes valve repair impossible.[12]

Traumatic papillary muscle rupture can result in severe mitral regurgitation (fig 4). The mechanism is thought to reflect either rupture of the valve following compression of a full left ventricle (isovolumetric contraction) or papillary muscle contusion followed by inflammation and necrosis with subsequent delayed rupture.[13] Our literature review suggested that the latter mechanism is dominant as clinical presentation with pulmonary oedema tends to occur 2–7 days or more after injury. Valve repair is the treatment of choice but occasionally the extent and location of the injury is prohibitive and valve replacement is necessary.

Traumatic rupture of the tricuspid valve is often well tolerated haemodynamically and its

frequency may be underestimated. A recent review of 13 cases demonstrated a median duration between trauma and surgery of 17 years with a postoperative median survival of 12 years.[14] Compressive injury of the lower thorax/upper abdomen during isovolumic contraction of the right ventricle, resulting in a sudden increase in right sided cardiac pressure, may be the mechanism of injury. Following compressive abdominal injury, severe tricuspid regurgitation may exacerbate abdominal haemorrhage. When traumatic rupture of the tricuspid valve occurs with right to left intracardiac shunting (fig 5) the presentation may be fulminant and emergency surgery may be necessary.[15]

Aortic injury
Rupture of the thoracic aorta occurs commonly following unrestrained high velocity motor vehicle collisions and it is usually fatal. The aortic isthmus at the site of the attachment of the ligamentum arteriosum in the upper descending aorta is the typical site of injury, as at this point the descending aorta is relatively immobile compared with the arch and ascending aorta, and this appears to predispose it to injury. Data from highly sensitive imaging techniques such as magnetic resonance imaging and transoesophageal echocardiography has resulted in a greater understanding of the pathophysiology of traumatic aortic injury. Traumatic involvement of the aortic wall may vary from a discrete subintimal haemorrhage (invisible on aortography) to complete laceration of all three layers of the aorta resulting in fatal intrathoracic haemorrhage. In cases of multiple trauma when the aortic adventitia and surrounding tissues are intact on magnetic resonance imaging, corrective surgery can be safely delayed allowing treatment of other injuries.[16] During this period, treatment with β blockers is recommended and frequent imaging should be performed to determine the optimal timing for surgery. Immediate graft replacement of the affected area is recommended if there is evidence of extravasation or enlargement of the aortic false aneurysm.

Conclusions
Continuing improvements in car safety and emergency care are likely to result in increased numbers of survivors of major trauma reaching hospital. Determining whether these patients have significant myocardial or aortic injury requires a high index of suspicion and a multidisciplinary approach. Improvements in imaging are increasing our understanding of the pathophysiology of blunt thoracic injury, but as yet no single imaging technique is ideal in the emergency setting. Each hospital must consider how to use its expertise to translate advances in thoracic imaging into a reduction in emergency room mortality.
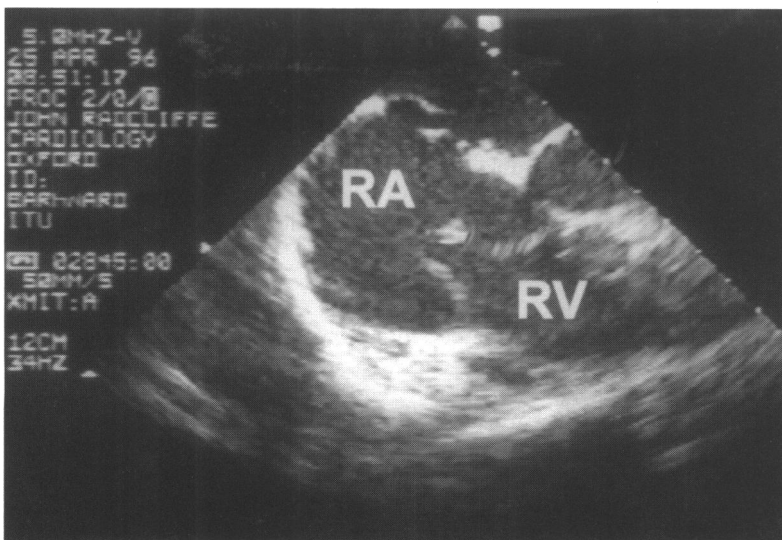

Figure 5   Transverse plane, cross sectional transoesophageal echocardiographic cardiac image during right ventricular (RV) systole. The septum between the left atrium and the right atrium (RA) is disrupted and the tricuspid valve is completely everted with remnants of the subvalvar apparatus prolapsing into the RA.

1 Parmley LF, Manion WC, Mattingly TW. Nonpenetrating traumatic injury of the heart. *Circulation* 1958;18:371–96.

2 Vignon P, Lagrange P, Boncoeur MP, Francois B, Gastinne H, Lang RM. Routine transoesophageal echocardiography for the diagnosis of aortic disruption in trauma patients without enlarged mediastinum. *J Trauma* 1996;40:422–7.

3 Minard G, Schurr MJ, Croce MA, Gavant ML, Kudsk KA, Taylor MJ, *et al*. A prospective analysis of transoesophageal echocardiography in the diagnosis of traumatic disruption of the aorta. *J Trauma* 1996;40: 225–30.

4 Catoire P, Orliaguet G, Liu N, Delaunay L, Guerrini P, Beydon L, *et al*. Systematic transoesophageal echocardiography for detection of mediastinal lesions in patients with multiple injuries. *J Trauma* 1995;38:96–102.

5 Weiss RL, Brier JA, O'Conner W, Ross S, Braithwaite CM. The usefulness of transoesophageal echocardiography in diagnosing cardiac contusions. *Chest* 1996;109:73–7.

6 Hendel RC, Cohn S, Aurigemma G, Whitfield S, Dahlberg S, Pape L, *et al*. Focal myocardial injury following blunt chest trauma: a comparison of indium 111 antimyosin scintigraphy with other invasive methods. *Am Heart J* 1992;123:1208–15.

7 Karalis DG, Victor MF, Davies GA, McAllister MPJ, Covalesky VA, Ross JJ, *et al*. The role of echocardiography in blunt chest trauma: a transthoracic and transoesophageal echocardiographic study. *J Trauma* 1994;36: 53–8.

8 Chirillo F, Totis O, Cavarzerani A, Bruni A, Farnia A, Sarpellon M, *et al*. Usefulness of transthoracic and trans-oesophageal echocardiography in recognition and management of cardiovascular injuries after blunt chest trauma. *Heart* 1996;75:301–6.

9 Saletta S, Lederman E, Fein S, Singh A, Kuehler D, Fortune JB. Transoesophageal echocardiography for the initial evaluation of the widened mediastinum in trauma patients. *J Trauma* 1995;39:137–42.

10 Diebel LN, Tagget MG, Wilson RF. Right ventricular response after myocardial contusion and haemorrhagic shock. *Surgery* 1993;114:788–93.

11 Struraitis M, McCallum D, Sutherland G, Cheung H, Dreidger AA, Sibbald WJ. Lack of significant long term sequelae following traumatic myocardial contusion. *Arch Intern Med* 1986;146:1765–9.

12 Pretre R, Faidutti B. Surgical management of aortic valve injury after nonpenetrating trauma *Ann Thorac Surg* 1993;56:1426–31.

13 McDonald ML, Orszulak TA, Bannon MP, Zeitlow SP. Mitral valve injury after blunt chest trauma. *Ann Thorac Surg* 1996;61:1024–9.

14 van Son JAM, Danielson GK, Schaff HV, Miller FA. Traumatic tricuspid valve insufficiency-experience in thirteen patients. *J Thorac Cardiovasc Surg* 1994;108: 893–8.

15 Banning AP, Durrani A, Pillai R. Rupture of the atrial septum and tricuspid valve following blunt chest trauma. *Ann Thorac Surg* 1997;64:240–2.

16 Fattori R, Celletti F, Bertaccini P, Galli R, Pacini D, Pierangeli A, *et al*. Delayed surgery of traumatic aortic rupture. Role of magnetic resonance imaging. *Circulation* 1996;94:2865–70.