posteriorly. The chest wall does have a certain degree of compliance, and due to its visco-elastic properties, it can withstand considerable deformation [2].

The decelerating force leads to a change in the antero-posterior diameter of up to 50% [3], which is compounded at impact by the deployment of the airbag. The latter prevents the forward expansion of the anterior chest wall. As the heart is relatively unfixed, it retains the potential for high inertia, which is transmitted to the cardiac muscle and structures causing distortion to the cardiac chambers by shear stress. The right ventricle (RV) is in a disadvantageous position, being right behind the sternum, hence receiving the full impact of the airbag on the anterior chest wall. Moreover, its usual low pressure system ill-prepares the RV for this significant change in pressure. This is worsened if there is an associated abdominal compression by the seat belt. The latter forces the blood within the venous system into the right side of the heart, i.e. an increase in the intravascular hydrostatic pressure. The combined end-result of all these forces leads to an acute elevation of right intraventricular pressure, which has been shown by Perlroth et al. [4] to injure the tricuspid valve apparatus. Valve rupture is more likely if these sudden acute changes occur during the isovolumetric phase of systole when the intraventricular pressure is low and the valve is closed, as this represents the timing of the maximal transvalvular gradient.

The rupture of the chordal apparatus is explained by the rush of blood towards the closed valve during the initial phase of deceleration (antegrade wave). The latter is caused by the initial anterior displacement of the heart, leading to blood acceleration towards the valve, and if the valve is closed, producing a significant extension-tensile tug on the chordal apparatus. This is quickly followed by the posterior displacement of the heart, creating a ‘reverse haemodynamic wave’ and a sudden increase in intracardiac pressure. Some of the above theories have been proven in an experimental laboratory using mathematical modelling principles [5].

The timing of intervention in the injured heart valve depends on the patient’s clinical condition. In the case described above, the life-threatening injuries were dealt with at the outset and the cardiac intervention was delayed. Repair of the valve is preferable if possible. Long-term outcome is usually very good. If the patient is haemodynamically unstable due to the severe TR, then optimization of the medical therapy including the use of diuretics as well as the RV assist device to support the cumbered RV or extracorporeal membrane oxygenation could be considered.

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REFERENCES


Figure 1: Intraoperative finding of anterior leaflet chordal rupture.

eComment. Tricuspid valve and blunt chest trauma

Authors: Georgios Dimitrakakis1 and Inetzi A. Dimitrakaki2

1Department of Cardiothoracic Surgery, University Hospital of Wales, Cardiff, UK
2Department of Cardiology, Metropolitan Hospital of Athens, Athens, Greece

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We read with interest the article by Thekkudan and associates regarding the tricuspid valve chordal rupture due to blunt injury and we agree with their comment that the timing of the surgical intervention in the heart valve depends on the patient’s general clinical condition [1].

Cardiac valvular injuries are quite uncommon after blunt thoracic trauma and the tricuspid valve is perhaps the more vulnerable because of the proximity of the right ventricle to the sternum [2].

Bailey et al. have highlighted that even a moderate blunt chest injury can provoke a significant injury of cardiac valves [2]. The major causes of injury are related to motor vehicle accidents followed by falls from one or more stories, falls off a horse or bus and compressed air explosions [2, 3].

Van Son et al. have reported on their experience with the surgical management of thirteen patients with tricuspid insufficiency due to blunt chest trauma (twelve cases were related to motor vehicle accidents and one to an explosion of a tank of compressed air). The median duration between trauma and surgical treatment was 17 years (range, 1 month to 37 years) and the primary cause of tricuspid insufficiency was flail of the anterior leaflet because of chordae tendinae rupture (n = 9), rupture of anterior papillary muscle (n = 3), or tear in the anterior leaflet (n = 1). In one patient, the septal leaflet was missing and in another it was retracted and adherent to the ventricular septum. Eight patients underwent tricuspid valve replacement and four tricuspid valve repair. No early or late deaths occurred at a median follow-up of 12 years [3].

Choi and Kim have reported on the successful management of simultaneous rupture of mitral and tricuspid valve with left ventricular rupture due to blunt chest injury. Their brief review of the relevant literature found that only eight patients had simultaneous rupture of the atrioventricular valves [4].

The diagnostic and therapeutic approach is a real challenge for these complex cases. When cardiac trauma is suspected after blunt chest trauma, transthoracic echocardiography can facilitate its appropriate management. However, the atrioventricular valve rupture can be missed due to cardiac tamponade and resultant ventricular collapse or due to other significant thoracic injuries [4].
controversial cases or in extremely unstable patients transesophageal echocardiography (preoperative and/or intraoperative) can establish the definitive diagnosis [2].

Post-traumatic tricuspid and mitral valve repair has to be considered very carefully because these patients are hemodynamically unstable and there are anatomic valvular difficulties such as myocardial contusion, focal endocardial ischemia, friable endocardium and necrotic papillary muscle. Because the right heart is a low-pressure system and the septomarginal trabecula is thick, the ruptured anterior papillary muscle can be reattached to its primary position with the use of the native chordae or neochordae with pledgeted sutures [1, 4].

In conclusion, the experience of valve repair in blunt chest trauma is limited however, especially for the tricuspid valve this therapeutic modality can be effective.

References