A 28-year-old man was admitted to hospital with progressive exertional dyspnea and peripheral edema of 2 months. One year previously he had experienced multiple fractures of the ribs and lower extremities after a 6-m fall. At that time, chest X-ray and echocardiographic examination had not showed abnormalities of the heart and great vessels. On this admission, his vital signs were normal and stable. Physical examination revealed peripheral edema, widening of the pulse pressure, elevation of the jugular venous pressure and a thrill at the upper left sternal border. On auscultation, there was a systolic ejection murmur maximal at the aortic area, and a diastolic murmur (grade III/VI) was appreciated along the left sternal border. Chest X-ray demonstrated widening of the mediastinum. Echocardiographic examination detected a large echo-free space that compressed the right heart. The right ventricle and right atrium were clearly defined as narrow distorted cavities (Fig 1). Continuous wave Doppler examination demonstrated increased peak diastolic transtricuspid blood flow velocity up to 2.7 m/s with a corresponding pressure gradient of 33 mmHg (Fig 2). Careful examination of the parasternal long-axis view of the left heart detected a communication between the large anterior echo-free space and the adjacent ascending aorta approximately 3 cm above the aortic valve (Fig 3). The color Doppler flow imaging showed shunt flow from the ascending aorta into the false aneurysm through the neck of the false aneurysm (Fig 4), and pulsed wave Doppler examination demonstrated that the shunt flow occurred in systole (Fig 5). Aortic regurgitation was also detected by color Doppler flow imaging.

He underwent surgical repair soon after the echocardiographic examination without cardiac catheterization. Surgical exploration revealed a large pseudoaneurysm (PSA) extending anteriorly to the undersurface of the anterior chest wall. The site of the neck of the false aneurysm was approximately 3 cm above the aortic annulus. The PSA was excised and the communication between it and the ascending aorta was surgically repaired. In addition, tears in the right coronary and noncoronary cusps were found and so
aortic valve replacement was performed. Histological examination showed that the wall of the PSA was composed of fibrous connective tissue with hyaline degeneration (Fig 6).

The postoperative echocardiographic examination performed 3 months later showed normalization of the right atrium and ventricle (Fig 1), and of the diastolic transtricuspid blood flow velocity (Fig 2).

**Discussion**

Rupture of the aorta commonly follows blunt trauma such as automobile accidents or falls from heights, with the most frequent site of rupture being the ligamentum arteriosum. The second most frequent site is the ascending aorta, followed by the thoracic aorta, arch, and abdominal aorta.1

In 1958, Parmley et al reviewed 296 cases involving non-penetrating aortic rupture and reported the first pathological series on this injury: 45% of injuries involved the isthmus, 23% the ascending aorta, 13% the thoracic aorta, 8% the arch, 6% multiple sites, and finally 5% involved the abdominal aorta. The regions at highest risk of damage are the junctions with the relatively immobile arch. Concomitant cardiac injury as shown at necropsy was 73% in those with injuries involving the ascending aorta, 27% in the arch, 23% in isthmus and thoracic, and only 15% in those with abdominal injury. It is clear from the few published...
studies that rupture of the ascending aorta is quickly followed by death and those patients who survive the initial trauma may later develop a PSA of the ascending aorta. Survival is associated with minor lesions, incomplete wall rupture, or favorably located injuries; that is, free hemorrhage and tamponade do not occur. A chronic PSA is defined as one that persists longer than 3 months after the original traumatic event as was the case for the present patient whose accident preceded the diagnosis by 1 year.

Clinical symptoms associated with PSA are caused by the impingement on adjacent structures, and the exact manifestation of these symptoms is related to the size and location of the PSA. For example, Stoney et al reported the development of superior vena cava syndrome by an expanding PSA of the aortic root. In the present patient the massive PSA of the ascending aorta compressed the right heart, causing obstruction of the right ventricular inflow and subsequent peripheral edema.

Pseudoaneurysm of the ascending aorta has been described in association with aortic valve endocarditis as a postoperative complication of cardiac surgery and secondary to blunt and penetrating trauma. There are several reports in the literature regarding descending thoracic aortic PSA with one reporting a patient diagnosed 32 years after the initial blunt trauma. In contrast, chronic PSA of the ascending aorta following blunt trauma are rare. Albuquerque et al reported one case diagnosed more than 5 months before surgical repair was undertaken.

The traditional method of diagnosis of PSA of the ascending aorta prior to surgery remains aortography, but other noninvasive studies, including magnetic resonance imaging, computed tomography and echocardiography are also valuable. Computed tomography with contrast can show the relationship between the PSA and the sternum, which is important for preventing the rupture of the aneurysm at the time of median sternotomy. In recent years, numerous case reports have cited echocardiography as a potentially useful tool because 2-dimensional echocardiography combined with the continuous wave Doppler, pulsed wave Doppler and color Doppler flow imaging can show the size and location of the PSA, adjacent structures and the shunt flow between the PSA and aorta through the neck. In the present case, 2-dimensional echocardiography showed the large echo-free space and compression of the right heart, and the Doppler technique, including color Doppler flow imaging, was then used to display the flow from the ascending aorta into the PSA and the increased diastolic tricuspid flow velocity. In essence, this accomplished the same goal as angiography but with a greatly reduced risk and cost.

Because 23% of patients have nonpenetrating aortic rupture in the ascending aorta and chronic PSA may occur after closed chest trauma, follow-up of such patients is suggested. Symptoms associated with chronic traumatic PSA of the ascending aorta include dyspnea and those caused by the impingement on adjacent structures. A newly developed cardiac murmur on physical examination and the presence of a widened mediastinum on chest X-ray are important diagnostic clues. Color Doppler flow imaging is a useful screening tool in the follow-up period after closed chest trauma.

Because traumatic pseudoaneurysms of the ascending aorta tend to rupture even several years after development, they must be treated surgically. Data from patients with chronic PSA of the ascending aorta suggest that the mortality rate is far less for surgical repair than for delayed rupture (4.6% vs 33%, respectively). Aortic regurgitation may also occur, as in the present case. Simple tears of the aortic valve should be repaired and the native aortic valve should be preserved, especially in young patients. Aortic valve replacement may be suggested in more complex aortic valve lesions, such as multiple tears in several cusps or tears of the cusp free edge. In the present case, tears in both the right coronary and noncoronary cusps were found at surgery, and so aortic valve replacement was performed.

Acknowledgment
This study was supported in part by a grant from the National Science Foundation of China (30270560), Beijing, China.

References