Spurious tricuspid regurgitation

Three conditions mimicking tricuspid regurgitation diagnosed at operation

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Brown, A. H., and Braimbridge, M. V. (1973). *Thorax*, 28, 495–497. Spurious tricuspid regurgitation: Three conditions mimicking tricuspid regurgitation diagnosed at operation. The diagnosis of tricuspid incompetence is difficult. Three patients are described in whom the diagnosis of tricuspid regurgitation was made but disproved by the findings at surgery. The first patient had aortic regurgitation, mitral regurgitation from chordal rupture, and constrictive pericarditis; the right atrium was compressed between the pulsating left atrium and the tight pericardium. The chordal rupture caused the mitral murmur to radiate parasternally. The second patient had severe mitral and aortic regurgitation and an interatrial septal defect with transmission of the left-sided 'v' waves to the right atrium. The third patient had an iatrogenic Gerbode defect from a previously repaired ostium primum atrial septal defect. Intracardiac phonocardiography failed to distinguish the anatomical situation from tricuspid regurgitation. The best assessment of tricuspid valvular disease is still that of the surgeon at operation.

THE DIAGNOSIS OF TRICUSPID REGURGITATION

'A survey of the current status of tricuspid incompetence shows that the diagnosis remains fraught with difficulty' (Domanchich and Koenker, 1971). A raised jugular venous pressure with a dominant 'v' wave may result from congestive cardiac failure alone and hepatic pulsation may be transmitted directly from the heart. A systolic murmur may be absent and atrial fibrillation obscures the distinction between stenosis and regurgitation. A catheter used for right ventricular angiography may itself produce distortion of the tricuspid valve (Cairns *et al.*, 1968). Direct right ventricular puncture and angiography is probably the most accurate method of diagnosing tricuspid regurgitation but the danger of tearing the right ventricle is too great for this to be a routine procedure (Domanchich and Koenker, 1971). Catheter velocitometry may be an alternative technique.

'The preoperative diagnosis of tricuspid valvular regurgitation is unreliable, and definite diagnosis is usually made by the surgeon at the time of operation' (Nolan *et al.*, 1971). We report here three patients in whom this statement was true.

CASE REPORTS

PREOPERATIVE DIAGNOSIS: AORTIC, MITRAL, AND TRICUSPID REGURGITATION A man aged 63 had rheumatic fever at the age of 20 but was well until two

years before admission when he became increasingly dyspnoeic on effort. Examination showed a malar flush, atrial fibrillation and a waterhammer pulse. His venous pressure was raised (6 cm) with a marked 'v' wave and rapid 'y' descent. On auscultation there was a pansystolic murmur audible at the apex and in the praecordium, a third sound, and a short apical mid-diastolic murmur. In the aortic area there was a loud ejection murmur, a normal second sound, and an aortic regurgitant murmur down the left sternal border. There were crepitations audible at the left base of the chest. The liver was enlarged five fingers' breadth, but there was no peripheral oedema. Electrocardiography showed atrial fibrillation, a normal axis, and some digitalis effect. Chest radiography showed an enlarged heart, with enlargement of both atria, the ventricular mass, and the pulmonary artery. Some calcification was seen in the posterior aspect of the heart on penetrated lateral films. Cardiac catheterization showed a raised mean right atrial pressure (16 mmHg) with a high 'v' wave. The mean pulmonary artery pressure was 58/35 mmHg and the wedge pressure was 32 mmHg. The left ventricular end-diastolic pressure was 25 mmHg. Angiography demonstrated a poorly contracting left ventricle, severe mitral regurgitation, moderate aortic regurgitation, and normal coronary arteries. It was thought that the aortic, mitral, and probably tricuspid valves would need replacement.

At operation (November 1969) through a median sternotomy the pericardium was found to be grossly adherent, thickened, and calcified, and was constricting the heart. After its removal the high right atrial pressure fell to normal and subsequent palpation of the tricuspid valve showed it to be entirely normal with no

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regurgitation. At exploration of the mitral valve under cardiopulmonary bypass the lesion was found to be rupture of the chordae tendineae of the anterior cusp with resultant severe mitral regurgitation. This valve and a thickened, shrunken aortic valve were replaced with Starr-Edwards prostheses.

The signs of tricuspid regurgitation in this patient therefore were due to constrictive pericarditis, the mobile atrial septum being forced by the expansile left atrium into the inelastic constricted right atrium producing the 'v' wave.

PREOPERATIVE DIAGNOSIS: MITRAL, AORTIC, AND TRICUSPID REGURGITATION A man aged 25 had had rheumatic fever at the age of 10. He developed bacterial endocarditis four years later but remained asymptomatic after recovery from this until he was 24 years old when he developed dyspnoea on exertion followed by congestive cardiac failure.

On examination he was in atrial fibrillation with a waterhammer pulse, his blood pressure being 130/49 mmHg. The mean jugular venous pressure was normal but there was a visible 'v' wave in the neck. Biventricular hypertrophy was suggested by palpation, and the pulmonary second sound was palpable. A loud pansystolic murmur was audible at the apex, and a loud third sound but no diastolic murmur. A pansystolic murmur was also audible at the left border of the sternum. An aortic mid-systolic and early diastolic murmur and single second sound were heard.

Electrocardiography was normal apart from atrial fibrillation. Chest radiography showed a very large heart with ventricular and bi-atrial enlargement. At cardiac catheterization the pulmonary artery pressure was 50/30 mmHg with saturation of 75%, no superior vena cava sample having been taken. The left atrial pressure had a 'v' wave of 25 mmHg, and the 'v' wave in the right atrium was 5 mmHg; the left ventricular end-diastolic pressure was 10 mmHg. Angio-

cardiography demonstrated severe aortic regurgitation and moderate mitral regurgitation. Mitral, aortic, and possibly tricuspid valve replacement was therefore advised.

At operation (October 1969) palpation of the tricuspid valve disclosed that the source of the 'tricuspid regurgitant murmur' and 'v' wave was a forceful jet of blood coming through a small secundum atrial septal defect during systole. The left atrium was pulsatile as a result of severe mitral regurgitation. The mitral and aortic valves, at exposure with cardiopulmonary bypass, were regurgitant because of thickening and contraction of the cusps. They were replaced with Starr-Edwards prostheses and the interatrial communication was closed.

PREOPERATIVE DIAGNOSIS: REOPENED OSTIUM PRIMUM ATRIAL SEPTAL DEFECT, MITRAL AND TRICUSPID REGURGITATION A man aged 21 had had an ostium primum atrial septal defect, with cleft mitral and tricuspid valves, repaired at the age of 15 by another surgeon. The patient's impaired exercise tolerance was improved by this for five years but was again diminishing with exertion inducing palpitations.

On examination he was in sinus rhythm. The venous pressure was raised with a 'v' wave of 6 cm. There was right ventricular hypertrophy and a pansystolic murmur at the apex, an opening snap and a mid-diastolic murmur. The pansystolic murmur could be heard in the praecordium. In the pulmonary area there was an ejection murmur and fixed splitting of the second sound. The liver was enlarged by 2 cm.

Electrocardiography showed a P mitrale, a long P-R interval, left axis deviation, and right bundle-branch block. There was moderate enlargement of the cavae, right atrium, ventricular mass, and pulmonary artery on the chest radiograph. At cardiac catheterization there was a 'v' wave of 10 mmHg in the right atrium, whose mean pressure was 3 mmHg.

FIGURE. The right atrial and ventricular pressures in patient 3, the intracardiac phonocardiogram below showing a loud pansystolic murmur at the level of the tricuspid valve, not detected elsewhere; R.V.=right ventricle; R.A.=right atrium.

The catheter crossed the interatrial septum and the mean left atrial pressure was 7 mmHg with a 'v' wave of 14 mmHg. The oxygen saturation rose from 62% in the superior vena cava to 85% in the right atrium. Intracardiac phonocardiography demonstrated a mitral pansystolic murmur which was, however, less loud than a pansystolic murmur just above the tricuspid valve (Figure). There was an opening snap and mid-diastolic murmur in the right ventricle also, demonstrated by intracardiac phonocardiography. Gross pathology of the tricuspid valve was thus suggested in addition to the reopened ostium primum atrial septal defect and mitral incompetence.

At operation (November 1969) great enlargement of the right atrium was found. The patch for the atrial septal defect inserted at the previous operation had separated at its upper and lower extremities. The lower separation included the septal attachment of the mitral valve, resulting in a Gerbode defect with communication between the left ventricle and the right atrium, producing a systolic jet. There was no tricuspid regurgitation whatsoever. The mitral regurgitation was minimal although the central part of the sutured split in its cusp had separated. Under cardiopulmonary bypass a new patch was sutured to the atrial septal defect and the Gerbode defect was closed with Teflon-buttressed stitches.

DISCUSSION

The notorious difficulty of diagnosing tricuspid valve disease in the presence of disease of other valves is well demonstrated by these three patients. One had in fact constrictive pericarditis, the second had an atrial septal defect, and the third had an iatrogenic left ventricular/right atrial shunt.

Although spurious tricuspid valve disease caused by constrictive pericarditis has been reported (Pitt *et al.*, 1969), the syndrome in those cases was compression of the tricuspid annulus by cystic complications of pericarditis producing a picture of tricuspid stenosis. In our first patient with constrictive pericarditis and aortic and mitral regurgitation, the right atrial 'v' waves were the result of the interatrial septum bulging into the right atrium because of the massive mitral incompetence and the constrictive pericardium preventing the right atrium from absorbing this pulsation. The

illusion of tricuspid regurgitation was augmented in this patient by the parasternal radiation of the pansystolic murmur because the mitral regurgitation was due to chordal rupture (Brown *et al.*, 1968).

Simulation of right-sided by left-sided abnormalities with an incomplete interatrial septum is well illustrated by patient 2. The atrial communication was missed because a superior vena cava sample had not been taken.

The third patient with an iatrogenic left ventricular right atrial shunt illustrates the fallability of intracardiac phonocardiography in the diagnosis of tricuspid regurgitation. There was no doubt about the position of the pansystolic murmur in the right atrium but murmurs caused by tricuspid reflux and Gerbode defects may be found in the same place. The same fallacy would also be found in the readings from velocitometers (Nolan *et al.*, 1971) in this region.

It seems that definite diagnosis of tricuspid regurgitation must be made by the surgeon at the time of operation.

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