LETTERS TO THE EDITOR

- The British Heart Journal welcomes letters commenting on papers that it has published within the past six months.
- All letters must be typed with double spacing and signed by all authors.
- No letter should be more than 600 words.
- In general, no letter should contain more than six references (also typed with double spacing).

Balloon dilatation of tricuspid stenosis caused by carcinoid heart disease

SIR,—Mullins *et al*'s conclusion that balloon dilatation of the tricuspid valve is safe and effective for tricuspid stenosis caused by carcinoid may not be consistent with clinical and haemodynamic data in their patient (*Br Heart J* 1990;63:249–50). They state that the jugular venous pulse showed a prominent systolic wave. This clearly means that tricuspid regurgitation was a clinically important abnormality. This accords with the haemodynamic data because in the basal state the 'v' wave in the right atrium was 44 mm Hg whereas the 'a' wave was 24 mm Hg. They say that the chest x ray showed pulmonary oedema. This is an unusual and unlikely feature of primary tricuspid valve disease, especially when there is no additional left sided disease. Because there was no mention of myocardial or valve disease affecting the left heart I presume that these structures were normal.

Right ventricular hypertension in carcinoid heart disease affecting the right side of the heart is possible only if there is stenosis of the pulmonary valve. The pulmonary valve was normal in this case, so what was the cause of the right ventricular pressure of 52 mm Hg? In addition, a right ventricular diastolic pressure of 18 mm Hg is rather surprising in the presence of important tricuspid stenosis without any additional cause for diastolic restriction of the right ventricle.

After dilatation of the tricuspid valve the 'a' wave in the right atrium remained at 24 mm Hg whereas the height of the 'v' wave fell from 44 to 36 mm Hg. This needs to be explained. Mullins *et al* claim that the severity of tricuspid regurgitation did not change after dilatation so what was the cause of reduction in the height of the 'v' wave?

Though after dilatation the mean pressure drop across the tricuspid valve fell from 10.5 to 7.6 mm Hg and the valve area increased from 0.9 to 1.4 cm², are Mullins *et al* justified in calling this dilatation "effective"? The reduction in the mean pressure drop could have been solely related to the reduction in the height of the 'v' wave, especially as the height of the 'a' wave was unchanged. Without knowledge of the cardiac output the effectiveness of dilatation cannot be judged solely on the values of the pressure drops.

Though the area of the tricuspid valve increased, its absolute value of 1.4 cm² still indicates a critical tricuspid stenosis.¹

What is more impressive is the fact that despite an 'a' wave of 24 and a 'v' wave of 36 mm Hg in the right atrium after balloon dilatation the "patient is doing well with reduction of dyspnoea and pulmonary and peripheral oedema". Why did the pulmonary oedema resolve after the release of the tricuspid stenosis?—when it seems logical to expect an increase in pulmonary oedema because of increased flow in the pulmonary circuit.

Unless these discrepancies are sorted out I cannot accept that this was predominantly a case of tricuspid stenosis requiring balloon dilatation. Moreover, on the basis of these data alone I believe that it is not correct to call this dilatation effective and recommend it for carcinoid valve disease.

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- 1 Kitchin A, Turner R. Diagnosis and treatment of tricuspid stenosis. *Br Heart J* 1964;26: 354–79.

This letter was shown to the authors who reply as follows:

SIR,—We are grateful for Dr Dalvi's interest in the case we presented. He raises three points for debate.

Firstly, was there significant tricuspid stenosis? Tricuspid stenosis was confirmed by a transvalvar gradient of 10.5 mm Hg and an orifice area of 0.9 cm² estimated by Doppler echocardiography. The criticism that the presence of a cardiac catheter across the valve during the haemodynamic study leads to an overestimation of the gradient can be levelled at all gradient estimations. This criticism, however, is not valid when the orifice area is estimated by Doppler echocardiography at a different time and the method is not subject to the same measurement constraints.

Secondly, why did the patient have a raised right ventricular diastolic pressure and pulmonary oedema? These abnormalities are not a feature of pure tricuspid stenosis. This elderly woman, presenting with carcinoid, also had poor left ventricular function, the cause of which was not investigated further. We postulated that fluid retention caused by tricuspid stenosis and poor ventricular function was responsible for the pulmonary congestion.

Thirdly, was the tricuspid dilatation successful? We showed a reduced mean gradient and an increase from 0.9 cm² to 1.4 cm² in orifice area estimated by Doppler echocardiography. The patient's symptoms of fluid retention and dyspnoea rapidly improved. The increase in tricuspid valve orifice area was maintained on a later echocardiogram.

This patient was terminally ill and in a very poor clinical state. Our aim was to palliate the symptoms associated with her severe tricuspid valve disease. The small improvement in tricuspid valve orifice was sufficient for this purpose.

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Diagnosis of a pseudoaneurysm of the right ventricular outflow tract

SIR,—Sreeram *et al* reported the diagnosis of a pseudoaneurysm of the right ventricular outflow tract (1990;63:129–31). Their colour Doppler picture certainly shows clearly the narrow communication between the right ventricle and the pseudoaneurysm.

Those who do not have a colour Doppler machine may be interested to know that a communication such as this can also be detected by contrast echocardiography, as we reported a few years ago.¹

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- 1 Garty BZ, Berant M, Weinhouse E, Levinski L. False aneurysm of the right ventricle due to endocarditis in a child. *Pediatr Cardiol* 1987;8:275–7.

This letter was shown to the author, who replies as follows:

SIR,—As Berant and Garty point out in their letter, the use of contrast echocardiography may be a valuable adjunct to the investigation of patients in whom a pseudoaneurysm of the right ventricular outflow tract is suspected.

While contrast echocardiography is a useful alternative non-invasive approach to diagnosis where colour flow mapping is not available, we suggest that colour flow mapping is a more appropriate technique because it can be quickly and easily carried out as part of the ultrasound examination itself. It does not require an intravenous line and administration of a contrast agent. Furthermore, the contrast effect produced can be variable in such patients and thus the results of such studies could be misleading. We expect colour flow mapping will prove to be a more sensitive and specific technique.

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CORRECTION

Review. Late potentials as predictors of risk after thrombolytic treatment? Günter Breithardt, Martin Borggrefe, Ulrich Karbenn (September 1990 issue, volume 64: pages 174–6)—We regret that an editorial error was introduced into this article. Lines 9, 10, and 11 of the second paragraph on page 174 should have read: There was an inverse relation between the duration of late potentials and the rate of ventricular tachycardia.