

FEATURED CLINICAL CASE

Pacemaker lead related tricuspid stenosis: a report of two cases

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Abstract

Only four cases of tricuspid stenosis related to endocardial pacemaker leads have been reported. Two further cases associated with perforation of a tricuspid valve leaflet by a pacemaker lead are presented: a 46 year old woman and a 60 year old man. It is possible that tricuspid valve disease related to endocardial pacemaker and non-thoracotomy defibrillator leads is underrecognised. Diagnosis requires clinical suspicion and the use of Doppler echocardiography. Recent evidence of fibrosis affecting the tricuspid valve in hearts from patients who have had non-thoracotomy defibrillator implants suggests that this problem could be more common in the future.

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Tricuspid valve disease related to endocardial pacemaker leads is uncommon. Tricuspid stenosis has only been reported in four cases in the literature, two associated with endocarditis.^{1–4} We report two cases of severe tricuspid stenosis requiring surgical intervention. In each case a tricuspid valve leaflet was perforated by a pacemaker lead.

Case 1

A 46 year old woman had a DDD pacemaker implanted in 1984, age 32, for symptomatic congenital heart block. The pulse generator was replaced in 1990 using the original leads. In June 1998 the second pulse generator battery had depleted and the leads had a manufacturer's notice advising lead replacement. At admission to hospital she had mild exertional dyspnoea and refractory hypertension, but no signs of tricuspid valve disease. An elective attempt was made to extract the leads using the Cook-Byrd technique but it proved technically difficult and was abandoned. A new dual chamber pacing system was implanted.

Three weeks later she presented again with severe dyspnoea. Venous pressure was raised with a dominant "a" wave. She was hypertensive with no pulsus paradoxus. Cardiac auscultation was unremarkable. Chest radiography showed cardiomegaly. Transthoracic echo-

cardiography showed a small antero-apical pericardial effusion with no evidence of tamponade. Because of concern that the attempted lead extraction may have caused a loculated pericardial haematoma she underwent trans-oesophageal echocardiography. This showed a large right atrium with a small underfilled right ventricle. Tricuspid valve Doppler revealed stenosis, peak pressure drop 16 mm Hg in late diastole, E wave pressure drop 8 mm Hg.

At operation there was no evidence of cardiac trauma. The right atrium was tense and the right ventricle underfilled. The original atrial lead had perforated one of the tricuspid valve leaflets and then looped back through the central orifice to lie on the inter-atrial septum. There was massive fibrosis of the valve leaflets and subvalvar apparatus with severe tricuspid stenosis. An attempt was made to perform a valvotomy but the fibrosis was too severe. The valve was excised and replaced with a St Jude prosthesis (Minnesota, USA). Macroscopically the valve was 3–4 mm thick. Histology showed fibrosis, calcification, and dense chronic inflammation. The patient was paced epicardially and has made an uneventful recovery. At nine months' follow up she is well with satisfactory pacemaker and prosthetic valve function. She is now normotensive on co-amifrufruse and warfarin.

Case 2

A 60 year old man had a Starr-Edwards (California, USA) aortic valve replacement in 1985, age 47, because of symptomatic severe aortic stenosis. One year later he presented with syncope and 10 seconds of asystole. A VVI pacemaker was implanted, but he continued to experience presyncope. After carotid sinus hypersensitivity was demonstrated, his pacemaker was upgraded to a DDI unit and he became asymptomatic.

In 1998 he presented again with exertional syncope and chest pain. Venous pressure was raised with a dominant "a" wave. The carotid pulse was low volume. In addition to prosthetic sounds there was a soft diastolic rumble with presystolic accentuation. ECG showed sinus rhythm with left bundle branch block. Transthoracic echocardiography showed pronounced obstruction of the aortic prosthesis (peak pressure drop of 74 mm Hg) along with severe tricuspid stenosis (peak A pressure drop

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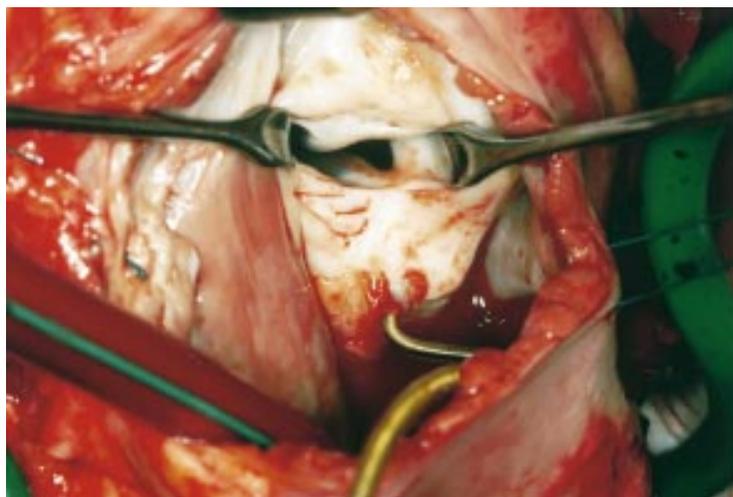


Figure 1 Intraoperative photograph of the tricuspid valve in case 2. The atrial lead has been removed. The stenosed tricuspid valve is held open by two small retractors. The ventricular lead is seen perforating the septal leaflet of the valve.

15 mm Hg, mean 9 mm Hg). The mitral valve was unremarkable. Coronary angiography was normal.

At operation it was shown that the ventricular pacing wire had perforated the septal leaflet of the tricuspid valve. There was notable fibrosis of the valve and subvalvar apparatus with severe tricuspid stenosis (fig 1). There was fibrous pannus on the left ventricular outflow surface of the Starr-Edwards sewing ring. A tricuspid valvotomy was performed, with splitting of several papillary muscles to allow better cusp mobility. The Starr-Edwards prosthesis was replaced with a St Jude valve. The endocardial pacing system was explanted and replaced with an epicardial system. The patient made an uneventful recovery and he was well at seven months' follow up. Echocardiography showed mild tricuspid stenosis (mean pressure drop 3 mm Hg) and no tricuspid regurgitation. Pacemaker function is satisfactory, although he now has complete heart block and is pacemaker dependent.

Discussion

Tricuspid stenosis related to endocardial pacing was first reported in 1980 in an 80 year old man presenting with right heart failure.¹ Chest radiography showed the atrial lead looped into the tricuspid orifice with the tip at the right atrial free wall. Surgery revealed that the atrial lead had perforated the lateral right atrial wall. The perforation was sealed by fibrous adhesions to the pericardium. There was severe fibrosis, calcification, and thickening of the atrial free wall, which followed the leads through to the tricuspid valve causing severe stenosis. The leads were removed, the valve replaced, and an epicardial pacing system implanted. The patient died three days postoperatively from septic shock.

The second reported case was a 60 year old man who presented with mild exertional fatigue and clinical signs of tricuspid stenosis.² Chest radiography showed the ventricular lead

executed a loop at the level of the tricuspid orifice. Echocardiography showed reduced tricuspid valve leaflet mobility, thickened subvalvar structures, and a mean diastolic tricuspid pressure drop of 9 mm Hg. The patient was offered surgical correction but declined.

In the third and fourth cases an attempt was made to treat endocarditis related to a pacing system with antibiotics alone, rather than early system explantation.^{3 4} Large vegetations developed on the leads in the right atrium, causing stenosis by prolapsing into the tricuspid valve orifice. Both cases were treated with surgical explantation of the endocardial pacing system, tricuspid valve repair/valvotomy, and use of an epicardial pacing system.

In our two cases trauma to the tricuspid valve was caused by perforation of a valve leaflet. We suggest that endothelial injury at the level of the tricuspid valve may eventually lead to fibrosis, calcification, and eventually stenosis. The injury may be from perforation of a cardiac structure, or secondary to a loop of lead at the level of the tricuspid valve causing a whiplash injury to the valve as postulated by Old *et al.*² Endocardial injury may occur more consistently with non-thoracotomy defibrillator leads.⁵ In eight hearts examined a mean 640 days after implantation of a non-thoracotomy defibrillator, the interface between the distal shocking coil and the ventricular endomyocardium was characterised by intense endocardial fibrosis and a sheath of fibroelastic tissue around the lead. In five hearts the tricuspid valve was involved.

Although rarely reported, tricuspid stenosis related to endocardial leads may occur more commonly than is clinically appreciated and might increase with long term clinical use of defibrillator leads. Tricuspid stenosis should be considered in any patient with endocardial leads presenting with symptoms or signs of right heart failure. Clinical signs may be subtle therefore there should be a low threshold to investigate with echocardiography. In our cases cross sectional echocardiography and colour Doppler were unhelpful, and pulse and continuous wave Doppler were necessary. There is also a strong case for systematic tricuspid valve Doppler in patients with endocardial leads investigated with echocardiography.

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- 1 Lee ME, Chaux A. Unusual complications of endocardial pacing. *J Thorac Cardiovasc Surg* 1980;**80**:934-40.
- 2 Old WD, Paulsen W, Lewis SA, *et al.* Pacemaker lead-induced tricuspid stenosis: diagnosis by Doppler echocardiography. *Am Heart J* 1989;**117**:1165-7.
- 3 Enia F, Mauro RL, Meschisi F, *et al.* Right-sided infective endocarditis with acquired tricuspid valve stenosis associated with transvenous pacemaker: a case report. *Pacing Clin Electrophysiol* 1991;**14**:1093-7.
- 4 Unger P, Clevenbergh P, Crasset V, *et al.* Pacemaker-related endocarditis inducing tricuspid stenosis. *Am Heart J* 1997;**133**:605-7.
- 5 Epstein AE, Kay GN, Plumb VJ, *et al.* Gross and microscopic pathological changes associated with nonthoracotomy implantable defibrillator leads. *Circulation* 1998;**98**:1517-24.