Balloon dilatation of pacemaker induced stenosis of the superior vena cava

A A Grace, M Sutters, P M Schofield

Abstract
A 53 year old woman with symptomatic pacemaker associated superior vena cava syndrome was treated successfully with balloon angioplasty. She was well six months after the procedure.

The superior vena cava syndrome is a well recognised but unusual complication of permanent pacemaker implantation. The syndrome results from the development of a stenosis in the superior vena cava with symptoms arising secondary to impaired regional venous drainage. Management is controversial and thoracotomy and caval reconstruction have been used to relieve symptoms. We report the successful use of balloon angioplasty as the primary treatment of this condition.

Case report
A 53 year old woman presented with a long history of severe palpitation. Atrioventricular nodal reentrant tachycardia was confirmed at electrophysiological study. The arrhythmia was refractory to medical treatment and in 1985 a PASAR (Telectronics) antitachycardia pacemaker was implanted. A Telecronics atrial lead was advanced to the right atrial appendage without difficulty. This relieved her symptoms considerably. A year after the procedure she presented with pleuritic chest pain and treatment with warfarin was started after a clinical diagnosis of pulmonary embolism. In 1988 she developed swelling of her hands and face and an angiogram showed stenosis of the superior vena cava proximal to its entry into the right atrium. Pulmonary artery pressure was normal and a pulmonary angiogram showed no evidence of pulmonary embolism. No action was taken. By 1990 the swelling of her upper body, which was especially severe in the morning and was made worse by activity, had increased and in addition she was experiencing worsening headaches. There was mild pitting oedema of the arms. The jugular vein was non-pulsatile and visible beyond the angle of the jaw. The electrocardiogram showed minor repolarisation abnormalities and the chest x ray was normal. Computed tomography showed a normal mediastinum. A repeat angiogram of the superior vena cava showed that the stenosis was more severe than before (fig 1). There was a gradient of 9 mm Hg between the superior vena cava above the stenosis and the right atrium. The angiographic appearance was suggestive of a fibrotic stricture with no evidence of associated thrombus. Contrast injections into the cephalic vein in the left antecubital fossa showed another stenosis at the junction of the brachiocephalic and the subclavian veins on that side. Multiple collaterals were also present. Balloon angioplasty of the stenosis of the superior vena cava was undertaken. A 0.035 inch straight guide wire and 7F Gensini catheter were introduced from the right femoral vein and the wire was passed through the stenosis to the right subclavian vein. The catheter was then advanced through the stenosis to allow a 0.035 inch exchange J guide to be introduced. The stenosis was then dilated with an 8 mm balloon catheter initially and then with a 10 mm balloon catheter (Mansfield). A good angiographic result was obtained (fig 2) and the gradient across the stenosis was abolished. The procedure was uneventful apart from two brief episodes of supraventricular tachycardia, during which the pacing unit continued to function normally. On examination the next day the jugular vein was no longer visible above the sternal angle. She remained symptom free six months after the procedure.

Discussion
Though venous abnormalities induced by pacemaker leads are common, stenosis of the superior vena cava and associated symptoms are unusual. The condition is more common

Cardiac Unit, Papworth Hospital, Cambridge
A A Grace
M Sutters
P M Schofield

Correspondence to Dr P M Schofield, Regional Cardiac Unit, Papworth Hospital, Cambridge CB3 8RE.

Figure 1 Dye injection into the right subclavian vein showing stenosis of the the superior vena cava above its junction with the right atrium. The Telectronics atrial lead can be seen in position.
in patients with retained leads and in those with infectious complications after pacemaker implantation and is the result of thrombosis, which may be associated with fibrosis. Ideal management is not established but thrombotic occlusion may be reduced by the administration of anticoagulants. Several patients have been given oral anticoagulants but the risk of resultant cerebral haemorrhage is probably increased by coexistent cerebral venous hypertension. Thrombolytic agents should be used only when coexistent thrombus is visible on angiography and they should be used with caution.

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