Reminder of important clinical lesson

Severe mitral regurgitation due to right ventricular apical pacing

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Correspondence to Dennis T L Wong, drdenniswong@yahoo.com.au**Summary**

A 75-year-old man with history of paroxysmal atrial fibrillation developed acute pulmonary oedema immediately after permanent pacemaker insertion for symptomatic bradycardia and was transferred to our institution. Echocardiography prior to pacemaker insertion showed normal left ventricle (LV) function and mild *mitral regurgitation* (MR). A single-chamber pacemaker had been inserted with the ventricular lead positioned in the right ventricular apex. He was treated with diuretics with symptomatic improvement.

Investigations failed to reveal a cause for cardiac failure. Patient subsequently had multiple readmissions for heart failure and echocardiography revealed severe MR. Patient was referred for mitral valve (MV) surgery.

Intraoperatively, when patient was in sinus rhythm and not paced, transoesophageal echocardiogram showed a significant reduction in the severity of MR. MV surgery was aborted and further echocardiographic characterisation revealed worsening of MR during ventricular pacing. The device was upgraded to a dual-chamber system and programmed to atrial pacing with intrinsic ventricular rhythm. He has had no further admissions over the following year.

BACKGROUND

We believe that the case report demonstrates the importance of attention to mode of pacing in the evaluation of mitral regurgitation (MR) in those with permanent pacemakers.

CASE PRESENTATION

A 75-year-old man with history of paroxysmal atrial fibrillation was transferred to our institution after developing acute pulmonary oedema immediately after permanent pacemaker insertion for symptomatic bradycardia. Echocardiography prior to pacemaker insertion had demonstrated normal left ventricle (LV) function and mild MR. A single-chamber pacemaker had been inserted with the ventricular lead positioned in the right ventricle (RV) apex. We postulated that a single-chamber pacemaker was chosen because patient was in atrial fibrillation at the time of insertion.

He was treated with diuretics with symptomatic improvement. Investigations failed to reveal a cause for cardiac failure. In particular, he had no elevation in cardiac enzymes, echocardiography failed to show any change in function, and coronary angiography was unremarkable. The presumptive diagnosis was volume overload in the periprocedural period. However, over the ensuing months, the patient experienced recurrent hospital admissions due to symptomatic LV failure. Subsequent echocardiography revealed severe MR that was not apparent prior to pacemaker insertion. The patient was referred for MV surgery.

At the time of the planned operative procedure, the patient was in sinus rhythm and had not required pacing. Intraoperative transoesophageal echocardiogram showed a significant reduction in the severity of MR. MV surgery was aborted and further echocardiographic characterisation

was undertaken. This demonstrated that during ventricular pacing there was severe MR that reduced markedly when not paced (figures 1 and 2, table 1). The device was upgraded to a dual-chamber system and programmed such that he was atrial paced with intrinsic ventricular rhythm. At pacemaker follow-up, he was in atrial fibrillation and hence 1:1 ventriculoatrial conduction during V pacing could not be determined. He has had no further admissions over the following year.

INVESTIGATIONS

- ▶ Transthoracic echocardiogram prior to permanent pacemaker insertion – mild MR.
- ▶ Transthoracic echocardiogram after permanent pacemaker insertion – severe MR with VVI pacing, mild MR when in intrinsic ventricular rhythm.
- ▶ Coronary angiogram – normal.
- ▶ Intraoperative transoesophageal echocardiogram – reduction in severity of MR when not paced.

TREATMENT

Upgrade of permanent pacemaker to dual-chamber system and programmed to atrial pacing with intrinsic ventricular rhythm.

OUTCOME AND FOLLOW-UP

No further admissions over the following year with no symptoms of heart failure.

DISCUSSION

This case first illustrates the acute and potentially dramatic effects of intra-LV dyssynchrony upon MV incompetence. Second, it highlights the deleterious effects of RV apical

Figure 1 Mitral regurgitation with VVI pacing.

Figure 2 Mitral regurgitation with AAI pacing.

Table 1. Echocardiographic parameters of right ventricular pacing versus intrinsic ventricular rhythm

Parameter	VVI pacing	AAI pacing/sinus rhythm
Septal to posterior wall motion delay (ms)	110	50
Interventricular mechanical delay	30 ms RV before LV	30 ms LV before RV
Dyssynchrony index (SD of 12 segment model) (ms)	40	30
Peak opposing wall delay (ms)	71	29
Ejection fraction 3D (%)	38	75
PISA EROA (cm ²)	0.57	0.17
Area of MR jet	26/32	4.8/22
A reversal (cm/s)	47	52

There had been about 10 case reports in the past 10 years highlighting worsening MR as a result of right ventricular pacing. Most have attributed left ventricular dyssynchrony secondary to RV pacing as a major cause. In several cases, patients were treated with cardiac resynchronisation therapy with good outcome. EROA, effective regurgitant orifice area; LV, left ventricle; MR, mitral regurgitation; RV, right ventricle.

pacing on both intra-LV synchrony and the severity grading of MR. Third, asynchronous ventricular pacing due to the loss of atrioventricular (AV) synchrony during VVI pacing may also have contributed to the MR.

VVI pacing delays LV activation and inverts the ventricular depolarisation sequence leading to dyssynchronous ventricular contraction and relaxation.¹ This results in significant modification in LV systolic function and LV filling, and increase in end diastolic volume.² This change in LV geometry may enhance pre-existing MR.³ Subsequent alteration in papillary muscle function may increase pre-existing MR, especially in the presence of leaflet motion abnormalities.⁴

During physiological activation, the mitral annulus moves forward in systole and decreases in area by 25%.⁵ Borgenhagen *et al*⁶ showed that this phenomenon was associated with a 59% reduction in MR orifice size. It is likely that the inversion of the ventricular activation sequence is associated with a delayed reduction of both mitral annulus size and MR orifice size and that this enhances MR severity.⁷

This case demonstrates the importance of attention to mode of pacing in the evaluation of MR in those with permanent pacemakers. In this instance, unnecessary MV surgery was avoided by preservation of intra-LV synchrony. It remains unclear whether strategies to promote intra-LV synchrony, such as alternate-site RV pacing or bi-ventricular pacing, can reduce the incidence of MV dysfunction or LV impairment over conventional RV apical pacing.

Competing interests None.

Patient consent Obtained.

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Learning points

- ▶ Right ventricular apical pacing can worsen functional MR.
- ▶ Right ventricular pacing can cause left ventricular dyssynchrony leading to worsening of MR.
- ▶ It is important to pay attention to mode of pacing when evaluating MR in patients with pacemaker.
- ▶ It is important to follow recommended guidelines when choosing mode of pacing for patients.
- ▶ As demonstrated in this case, right ventricular apical pacing can potentially worsen MR. Therefore, in patients with new or worsening heart failure symptoms after right ventricular apical pacing, MR severity should be reassessed.

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