Deep septal pacing to upgrade patients with pacing-induced cardiomyopathy

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Introduction

Chronic right ventricular (RV) apical pacing causes electrical and mechanical dyssynchrony, which in turn can lead to left ventricular (LV) dysfunction and symptomatic heart failure (HF) in a non-negligible proportion of patients.[1] Alternative pacing sites, such as the RV septum or the RV outflow tract, have not consistently shown improved clinical outcomes.[2]

Cardiac resynchronization therapy (CRT) can reduce LV dyssynchrony and reverse pacing-induced cardiomyopathy (PICM).[3] However, patients with prior RV pacing have been excluded from randomized trials of CRT, so the impact of CRT upgrade on hard clinical endpoints is less established than that of de novo CRT implantation and CRT upgrade is recommended with lower level of evidence in clinical practice guidelines.[4] Pacing the His bundle or the left bundle branch (left bundle branch pacing; LBBP) has emerged not only as a very attractive option to replace RV pacing but also as a potential alternative to CRT.[5] Deep septal pacing or LV septal pacing, even in the absence of conduction system capture, may also provide a more synchronous LV activation and better clinical outcomes as compared to RV pacing.[6]

To date, no data are available about deep septal pacing in patients with PICM. We present the 6 months outcome of the first 2 patients with PICM upgraded to deep septal pacing at our institution. In both cases deep septal pacing was achieved using a SelectSecure 3830 pacing lead delivered through a fixed-curve C315-HIS sheath. Both patients gave their written informed consent to the procedure. The final position of the leads in these 2 patients is shown in Figure 1.

Case report

The first patient was a 71-year-old man with multiple cardiovascular risk factors and advanced chronic kidney disease, with normal (>55%) left ventricular ejection fraction (LVEF). A dual-chamber pacemaker was implanted for advanced atrioventricular block. Paced QRS duration was 160 ms (Figure 2). A few weeks later, the patient developed severe LV dysfunction (LVEF of 30%) accompanied by worsening HF (NYHA class III) and worsening renal function, which required starting hemodialysis. Given the important comorbidities, upgrade to LBBP was the first choice. However, clear left bundle branch (LBB) capture could not be achieved (Figures 2 and 3). Despite absence of LBB capture, deep septal pacing obtained a paced QRS of 125 ms, which was considered satisfactory. Pacing threshold was 0.9 V at 0.4 ms. Prior RV lead was easily removed. Six months later, the LVEF at echocardiography improved to 42% and the patient was in NYHA class I.

The second patient was a 70-year-old man affected by myelodysplastic syndrome, who underwent dual-chamber pacemaker implantation for complete atrioventricular block. Paced QRS duration was 170 ms (Figure 2). At the time of implant his echocardiogram was unremarkable. One month later, the myelodysplastic syndrome evolved to an acute myeloid leukemia and a new echocardiography was performed before starting the chemotherapy, showing a mildly dilated left ventricle, significant LV dyssynchrony, LVEF of 37%, and moderate functional mitral regurgitation. These findings contraindicated chemotherapy, so he was scheduled for an upgrade. LBBP was attempted; however, deep septal pacing without clear criteria of LBB capture was associated with a paced QRS of 120 ms (Figures 2

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KEY TEACHING POINTS

- Deep septal pacing can produce a narrow paced QRS, even in the absence of left bundle branch capture.
- Deep septal pacing was able to revert pacemaker-induced cardiomyopathy in 2 patients.
- Deep septal pacing might be a simpler alternative to left bundle branch pacing if larger studies confirm our initial results, and it may be a cost-effective strategy in patients with pacing-induced cardiomyopathy.

and 3), so it was considered an adequate final position for the lead, with a pacing threshold of 0.5 V at 0.4 ms. An echocardiogram performed 6 months later showed a significant improvement in LVEF up to 52%, the absence of LV dyssynchrony, the normalization of LV diameters, and mild mitral regurgitation. Thus, the patient was allowed to start chemotherapy.

Discussion

This report describes 2 patients with PICM who experienced reverse remodeling after upgrade to deep septal pacing, in the absence of LBB capture.

Recently, several studies have evaluated criteria for LBB capture. A paced QRS morphology of right bundle branch block (either complete or incomplete) was reported as a necessary (although not sufficient) condition for LBB capture.[7] In our patients, paced QRS had QS morphology in V_1 without a clear r wave; thus, this morphological criterion should already exclude LBB capture in both cases. However, we performed additional tests to exclude LBB capture. The criterion of "paced V_6 R-wave peak time (RWPT) (measured from QRS onset) $\leq$ native V_6 RWPT (+ 10 ms)" has shown high sensitivity and specificity for LBB capture in a recent report.[8] As shown in Figure 3, none of our patients fulfilled this criterion. Different cut-off values of paced V_6 RWPT measured from the pacing spike (for example, 75 ms[7] and 83 ms[8]) have been reported in patients without baseline LBBB to predict LBB capture and both our patients had a paced V_6 RWPT above these values. Changes in V_6 RWPT as well as in QRS morphology by pacing at different outputs can demonstrate transition from selective LBB capture to nonselective LBB capture or from nonselective LBB capture to LV septal pacing. As shown in Figure 2, pacing at different outputs did not produce any significant change in the morphology of the paced QRS or in the V_6 RWPT.

In both cases deep septal pacing was associated with a significant reduction in QRS duration with respect to RV pacing and with an improvement in the LVEF by >10%. These findings are complementary to the initial evidence from 2 small observational studies about the benefit of LBBP in patients with PICM.[9,10] With respect to LBBP, deep septal pacing

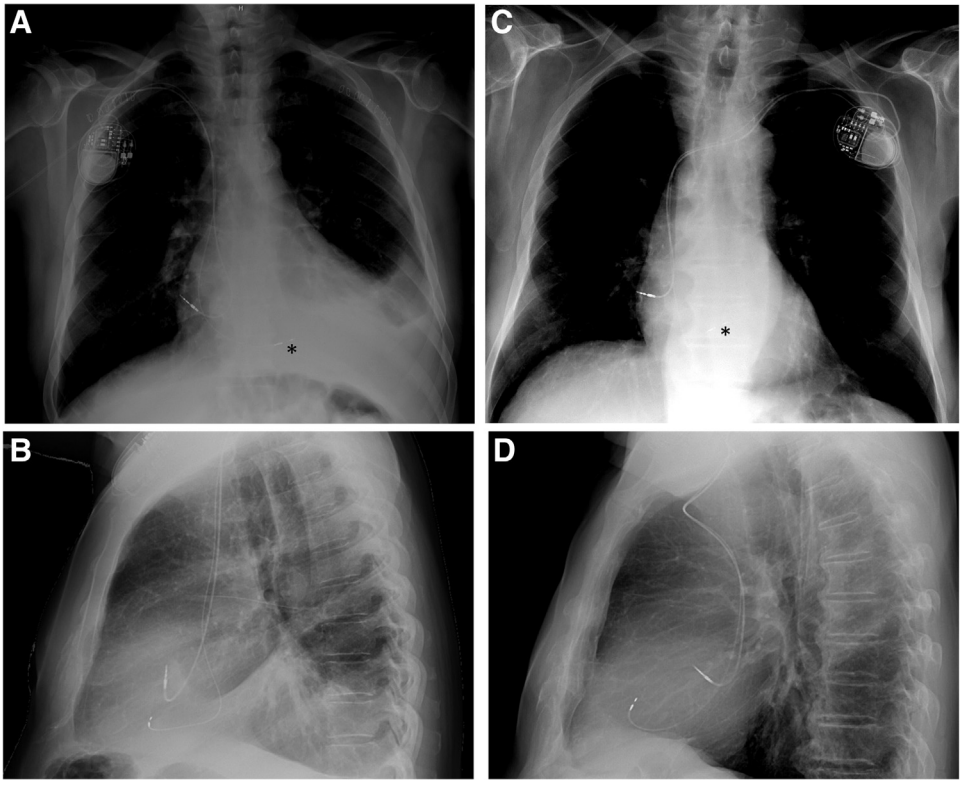

Figure 1 Final leads position in patient 1 (**A:** posteroanterior view; **B:** lateral view) and patient 2 (**C:** posteroanterior view; **D:** lateral view). Asterisk (*) indicates the tip of the septal lead in the posteroanterior view.

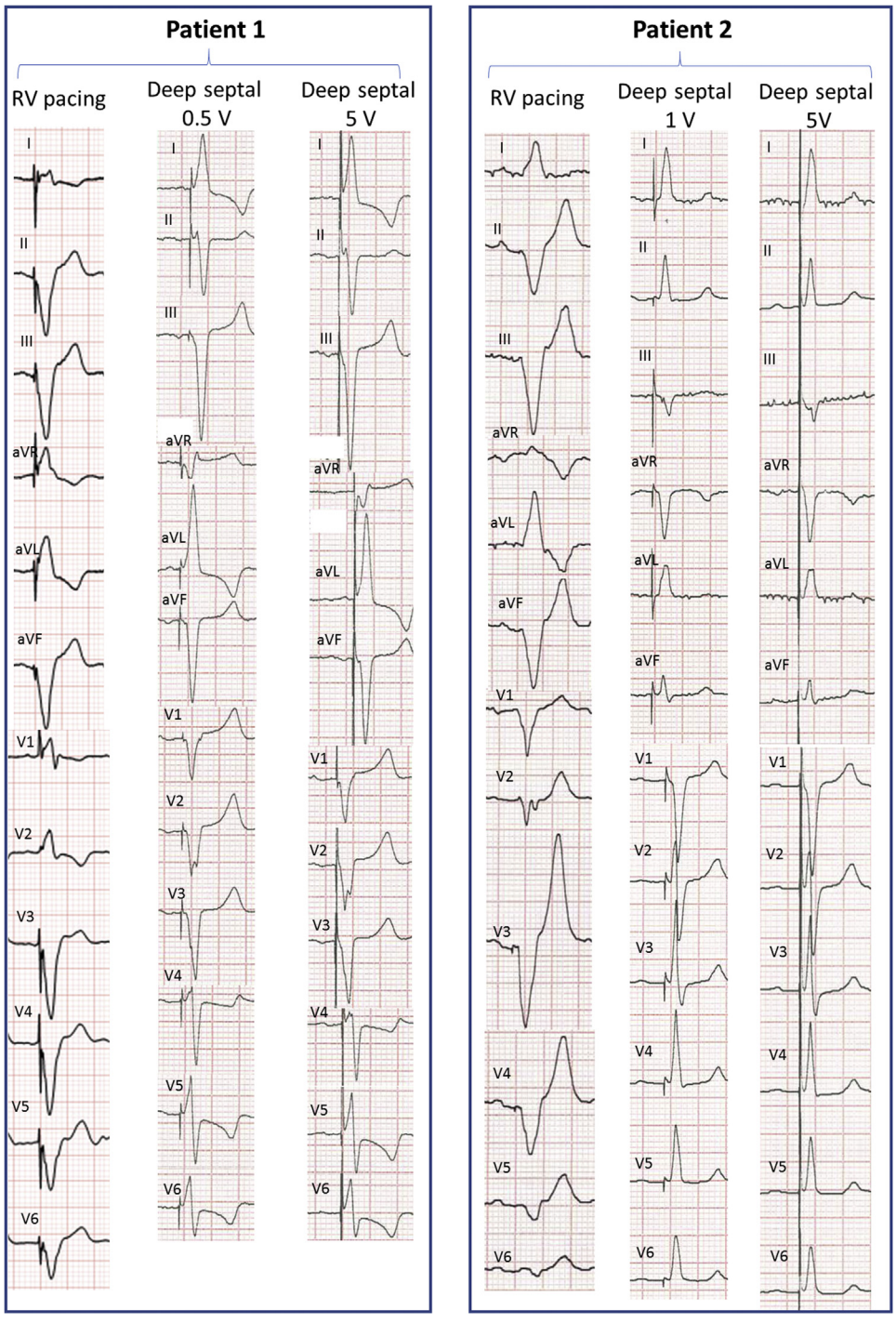

Figure 2 Electrocardiograms during right ventricular pacing and deep septal pacing at different outputs in the 2 patients.

is a simpler technique and might portend higher probability of success. For these reasons, if deep septal pacing could demonstrate similar clinical benefit as compared to LBBP, it might be considered not only as an alternative to failed LBBP but also as a reasonable first-line option; in this case the enhanced simplicity of the technique might allow a more widespread diffusion.

The capture of the physiologic conduction system allows great intraventricular and interventricular synchrony. However, classical CRT has achieved excellent results by producing a relatively narrow paced QRS despite the absence of any capture of the physiological conduction system. In this sense, deep septal pacing might be of clinical utility if a relatively narrow paced QRS can be achieved, as happened in the cases reported here.

In these 2 patients PICM occurred early after initial pacemaker implant, as already described in prior reports on PICM.[11] The second patient almost normalized the LVEF with deep septal pacing, while the first one persisted with moderate LV dysfunction. This latter finding is in line with

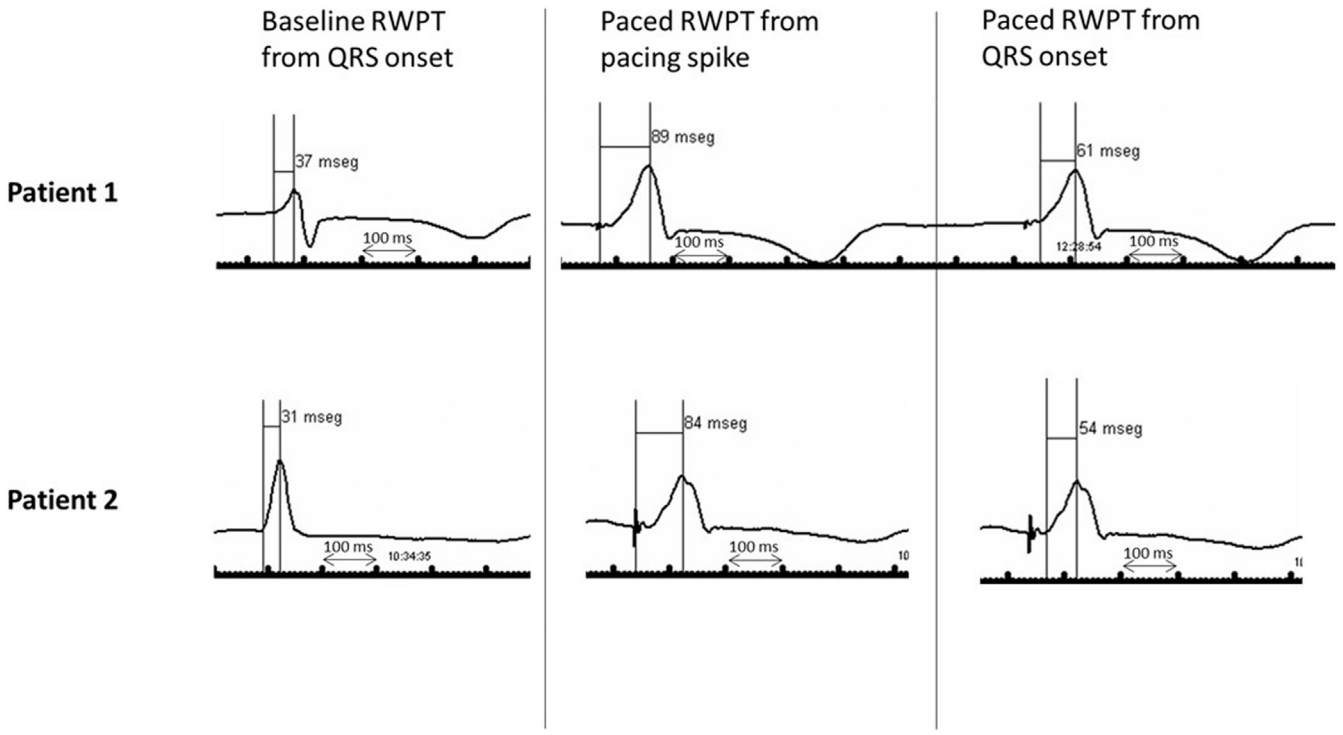

Figure 3 Evaluation of left ventricular activation time during spontaneous and paced QRS in both patients. RWPT = R-wave peak time, measured in lead V_6.

prior reports about the results of CRT upgrade in patients with PICM: in 1 large series, for example, the mean LVEF after upgrade was 45% and only 49% of patients achieved an LVEF improvement of >10%.[12] In addition, deep septal pacing was associated with a significant improvement of HF symptoms in the first patient (NYHA class III to NYHA class I).

The second patient also experienced an improvement in mitral regurgitation after upgrading to deep septal pacing, similarly to what has been recently reported for His bundle pacing.[13]

Indeed, patients with PICM may be good candidates for LBBP or deep septal pacing owing to the less robust evidence of conventional CRT in this subgroup. In addition, the cost-effectiveness of LBBP and deep septal pacing in these patients is further enhanced by the possibility to reuse the same generator. The findings of this report suggest that deep septal pacing may be a simple but effective option in patients with PICM.

Conclusion

Our initial experience suggests that deep septal pacing can produce a narrow paced QRS and might be considered as a potential option to revert PICM. Larger studies are needed to confirm this hypothesis.

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