A patient with an implanted dual-chamber pacemaker (DDDR) for sick sinus syndrome had a pulse generator exchange due to battery depletion. Apropos with the procedure, it was noted that the patient had continuous ventricular pacing via a pacing lead located at the right ventricular apex. In order to avoid possible deleterious effects of the iatrogenic dyssynchrony conferred by this kind of pacing, the algorithm of ventricular pace suppression function was activated in the new device that practically led to functional AAI pacing, deemed a more physiologic mode of pacing that could prevent the potential harmful effects of right ventricular apical pacing. 

**Case Report**

A 69-year-old lady was referred for pulse generator replacement due to battery depletion of a dual chamber pacemaker device (DDDR) implanted 7 years earlier for symptomatic sinus node dysfunction. The pacing leads had been implanted at conventional sites, the atrial lead in the right atrial appendage and the ventricular lead at the RV apex (Figure 1). At this time, the underlying rhythm was found to be sinus bradycardia at ~45-50 bpm, however, the pacemaker was functioning at a DDD mode with sequential atrial and ventricular pacing, with evident iatrogenic LBBB morphology of the paced QRS (Figure 2). Intraoperatively, measurements of the pacing and sensing thresholds were satisfactory for both leads. During programming of the new device (Enticos 4 DR, Biotronik) the algorithm of ventricular pace suppression function was activated and it allowed for functional atrial-based (AAI) pacing and native atrioventricular (AV) conduction in the form of incomplete right bundle branch block (Figure 3), thus entirely avoiding ventricular pacing with its potential harmful long-term effects. Information regarding the actual function of the previous device over the preceding years was unavailable, however, the LV ejection fraction (LVEF) measured on recent echocardiography was normal in this patient.

**Introduction**

Over the past several years, it has become abundantly clear that right ventricular (RV) apical pacing may have deleterious effects on left ventricular (LV) function due to the produced iatrogenic left bundle branch block (LBBB) (pacing-induced cardiomyopathy or PICM), similar to the effects conferred by the native LBBB which produces LV dyssynchrony leading to LV dysfunction and ultimately to heart failure. Thus, for prophylaxis of PICM it is prudent to activate the algorithms borne by current devices that allow for minimized ventricular pacing. A patient where such an algorithm was utilized is herein presented.
The incidence of pacemaker-induced cardiomyopathy (PICM), usually defined as a ≥10% decrease of a previously normal left ventricular ejection fraction (LVEF) resulting in LVEF <50%, ranges from 12% to 22% in recent studies of patients who are paced via a lead positioned at the RV apex.\(^8\)\(^{-}\)\(^{10}\) An analysis of 823 consecutive patients receiving a permanent pacemaker (PPM) for complete heart block (CHB) with LVEF >50%, indicated that 101 (12.3%) developed PICM over a mean follow-up of 4.3 ± 3.9 years, with post-PPM LVEF being 33.7% ± 7.4% in patients with PICM vs 57.6% ± 6.1% in patients without PICM (p < 0.001).\(^8\) In multivariable analysis, lower pre-PPM LVEF (hazard ratio - HR 1.047 per 1% LVEF decrease; p = 0.042) and RV pacing % both as a continuous (HR 1.011 per 1% RV pacing; p = 0.021) and as a categorical (<20% or ≥20% RV pacing) (HR 6.76; p = 0.002) variable were independently associated with PICM. The authors concluded that PICM is not uncommon in patients receiving PPM for CHB with preserved LVEF and is strongly associated with RV pacing burden >20%.

According to data from another retrospective report of 1,750 consecutive patients undergoing pacemaker implantation with normal baseline LVEF, the incidence of PICM (defined as >10% decrease in LVEF resulting in LVEF <50%) was 22.8% with 42 of 184 patients meeting study criteria, with decrease in mean LVEF from 62.1% to 35.3% over mean follow-up 2.5 years.\(^{10}\) Longer follow-up paced QRS duration was associated with the presence of PICM (multivariate odds ratio 1.34 per 10 ms increase, p = 0.01). Paced QRS duration ≥150 ms was 95% sensitive for PICM. Only half of patients with PICM had heart failure signs or symptoms at the time of echocardiographic diagnosis. The authors concluded that patients with frequent RV pacing and paced QRS duration ≥150 ms should be screened by echocardiogram to assess for PICM.

PICM can be largely reversed by biventricular pacing.\(^6\)\(^{,}\)\(^{11}\) However, prophylaxis is always better and easier than treatment. Studies comparing alternate site pacing with RV apical pacing have indicated that the adverse effects of ventricular pacing are largely attenuated with alternate site pacing, although this has not been conclusively settled as yet.\(^5\)\(^{-}\)\(^{12}\)\(^{-}\)\(^{14}\) Of course, biventricular pacing is always superior to other modes of pacing.\(^{15}\)\(^{-}\)\(^{17}\)

However, for patients who already have a conventional pacemaker with the RV lead implanted at the RV apex, the best initial approach is to minimize ventricular pacing, if at all possible, i.e. at least in those without permanent AV block. Thus, this is possible practically in all patients with sinus node dysfunction and normal AV conduction and in the majority of patients with intermittent AV block. Avoidance of or minimized ventricular pacing can be managed with programming of the device to a functional AAI pacing mode. This is practically effected with use of various algorithms available in all current devices.\(^18\)

In the present case, ventricular pacing could be entirely avoided with use of the ventricular pace suppression function of the device, a dual- to atrial single-chamber mode switch [DDD(R) to ADI(R)] algorithm automatically activated via a series of specific AV conduction tests (Fig. 3).\(^{19}\) This function entails searching for the presence of underlying intrinsic ventricular activity every 30 seconds and then at progressively longer intervals (up to 20 hours) if there is no native AV conduction. The device does this by prolonging its AV delay to 450 ms for 8 cycles. The pacemaker switches from DDD(R) to
ADI(R) mode if 1 intrinsic ventricular event is sensed during this prolonged AV delay over 6 consecutive cycles. During this search, no blocked P wave is allowed as ventricular pacing occurs after 450 ms. However, this recurring prolongation of the AV delay has its own inherent limitations as it may lead to the occurrence of pacemaker syndrome and/or pacemaker mediated tachycardia and/or run the risk of 2:1 AV conduction for a few cycles during exercise; in addition. To avoid too many switches between DDD(R) and ADI(R) mode, the ventricular pace suppression function is halted for 20 hours if more than 15 switches/hour have occurred within the last 24 hours.

There are certain differences in the algorithms of various device manufacturers designed to reduce RV pacing, however, all of them allow for pacing at a prolonged AV delay with its inherent limitations as described above emanating from AV decoupling or desynchronization.20 The advantage of the present device algorithm relates to avoidance of AV block or long pauses by applying DDD pacing when the AV delay is >450 ms but pacing mode does not revert to DDD pacing when the AV delay remains <450 ms. Furthermore, the algorithm remains active and periodically searches for presence of AV conduction compared to other algorithms that may be deactivated after a certain period of time elapses without detection of native AV conduction.

REFERENCES