

Continuous resetting of reentrant idioventricular rhythm with biventricular pacing: A cause of erroneous assumption of 100% pacing



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Introduction

Cardiac resynchronization therapy (CRT) is an established treatment for electrical dyssynchrony in patients with heart failure with reduced ejection fraction. Resynchronization of the left ventricular (LV) wall motion using biventricular (BiV) pacing often improves cardiac performance and promotes LV reverse remodeling. Therefore, a high percentage of BiV pacing is essential to maximize the benefit of CRT.¹ Even a small amount of ineffective BiV pacing increases the incidence of death, hospitalizations for heart failure, and ventricular tachyarrhythmias.² Although various algorithms allow us to achieve a high percentage of BiV pacing, the presence of a pacing stimulus does not always imply effective BiV pacing.³ Atrial tachycardia, atrial fibrillation, premature ventricular contractions, loss of LV capture, and fusion with intrinsic conduction are representative causes of ineffective BiV pacing.⁴ However, little has been described regarding cases where a reentrant idioventricular rhythm below the programmed lower rate limit of BiV pacing causes BiV pacing failure.

Case report

An 81-year-old man diagnosed with idiopathic dilated cardiomyopathy was admitted to our hospital because of worsening heart failure. Regardless of the introduction of the guideline-directed medical therapy and implantation of a CRT defibrillator (CRT-D) device, his cardiac function was still severely impaired (ie, LV ejection fraction of 15%), and his functional status had gradually deteriorated from New York Heart Association class III to ambulatory class IV.

KEYWORDS Biventricular pacing; Cardiac resynchronization therapy; Entrainment; Idioventricular rhythm; Reentry (Heart Rhythm Case Reports 2022;8:730–734)

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KEY TEACHING POINTS

- An idioventricular rhythm, defined as a regular ventricular arrhythmia with a rate of less than 100 beats per minute, usually occurs as a result of enhanced automaticity. However, a reentrant mechanism should also be considered, especially in patients after ventricular tachycardia ablation.
- A reentrant idioventricular rhythm can be manifestly entrained by biventricular pacing when the pacing rate is faster than the heart rate of an idioventricular rhythm. It can be a cause of ineffective biventricular pacing.
- It is difficult to identify fusion of an idioventricular rhythm and biventricular pacing with telemetry. The surface electrograms, including the 12-lead electrograms and Holter recordings, can help identify the fusion of the two.

On admission, the electrocardiogram (ECG) monitoring revealed repetitive episodes of ventricular tachycardia (VT) with a tachycardia cycle length of 380 ms. Although antitachycardia pacing reverted the VT to sinus rhythm, the VT caused chest discomfort and hemodynamic instability. Introduction of intravenous amiodarone and general anesthesia could not suppress the VT storm. After obtaining informed consent, we performed a catheter ablation of the VT storm. Although the inducibility of the nonclinical VT remained at the end of the session, he was successfully freed from the hemodynamically unstable VT storm condition (partial procedural success).

However, a hemodynamically stable idioventricular rhythm with heart rate of <100 beats per minute (bpm) sometimes occurred thereafter. As a temporary measure, we introduced overdrive BiV pacing with a ventricular rate of 110 paces per minute (ppm) to suppress the slow ventricular arrhythmia. After the modification of the device

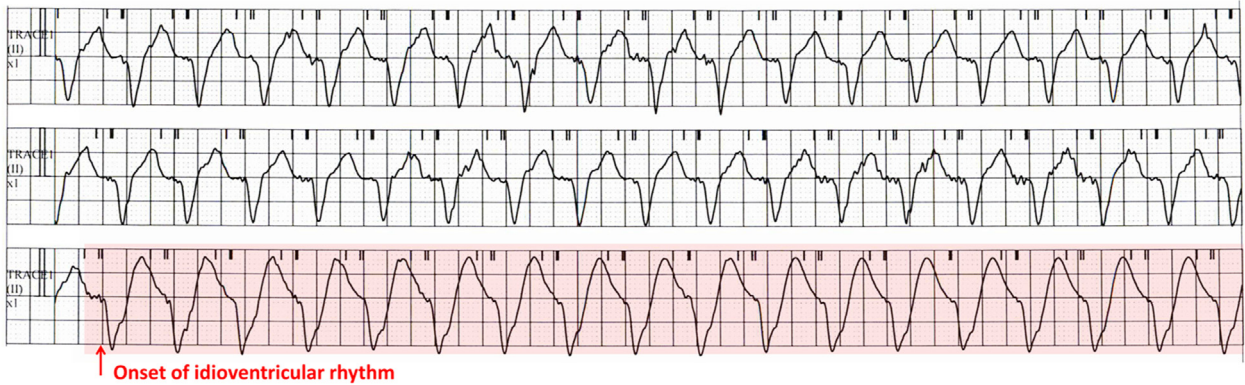
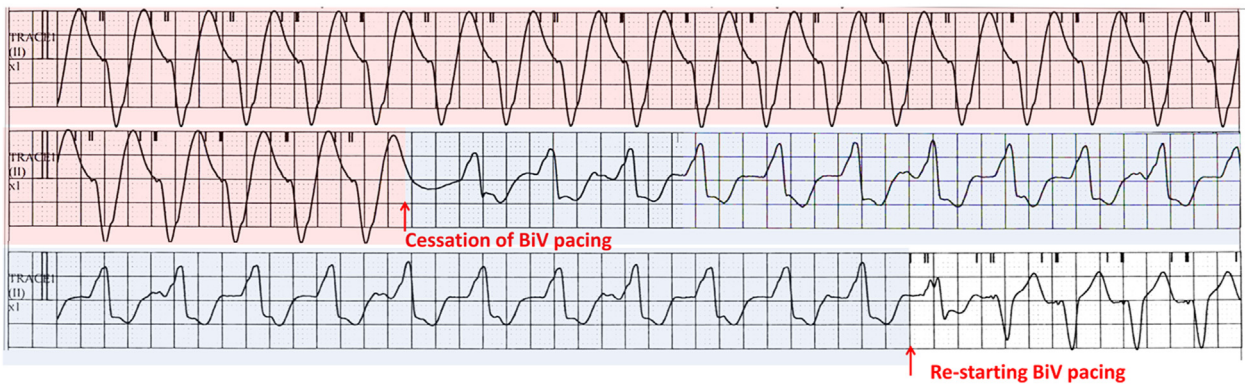
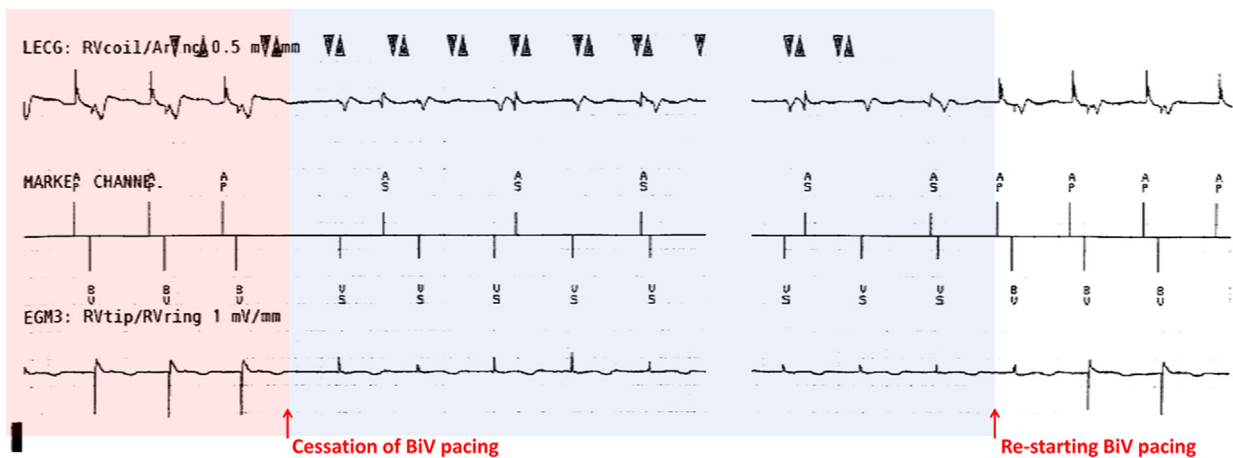
A Occurrence of an idioventricular rhythm during BiV pacing**B Manifestation of an idioventricular rhythm by cessation of BiV pacing****C Intracardiac electrograms during cessation of BiV pacing**

Figure 1 Continuous electrocardiac monitor recordings during the occurrence of reentrant idioventricular rhythm and its termination. **A:** During the occurrence of the reentrant idioventricular rhythm, a change in the QRS complex morphology indicated the onset of the reentrant idioventricular rhythm (85 beats/min). We could identify a constant fusion with the same ventricular rate as the biventricular (BiV) pacing (110 beats/min). Manifest entrainment of the idioventricular rhythm by BiV pacing is a plausible explanation. **B:** After cessation of the BiV pacing, the QRS morphology abruptly changed to a ventricular arrhythmia-alone morphology. Reintroduction of the BiV pacing changed the QRS morphology to a narrow one, which indicated the termination of the idioventricular rhythm and subsequent effective BiV pacing. **C:** The intracardiac electrograms before cessation of and after reintroduction of the BiV pacing are totally the same. Identifying fusion from the telemetry information alone is sometimes difficult.

setting, the ventricular arrhythmia subsided, and the patient appeared stable within a few days. However, the ECG monitor sometimes displayed wide QRS complexes regardless of the fixed pacing rate of 110 ppm (Figure 1A).

The telemetry checkup revealed no VT / ventricular fibrillation episodes, and the cumulative percentage of ventricular pacing was 97% with a ventricular sense response of 0% since the last CRT-D setting. The 12-lead ECG revealed

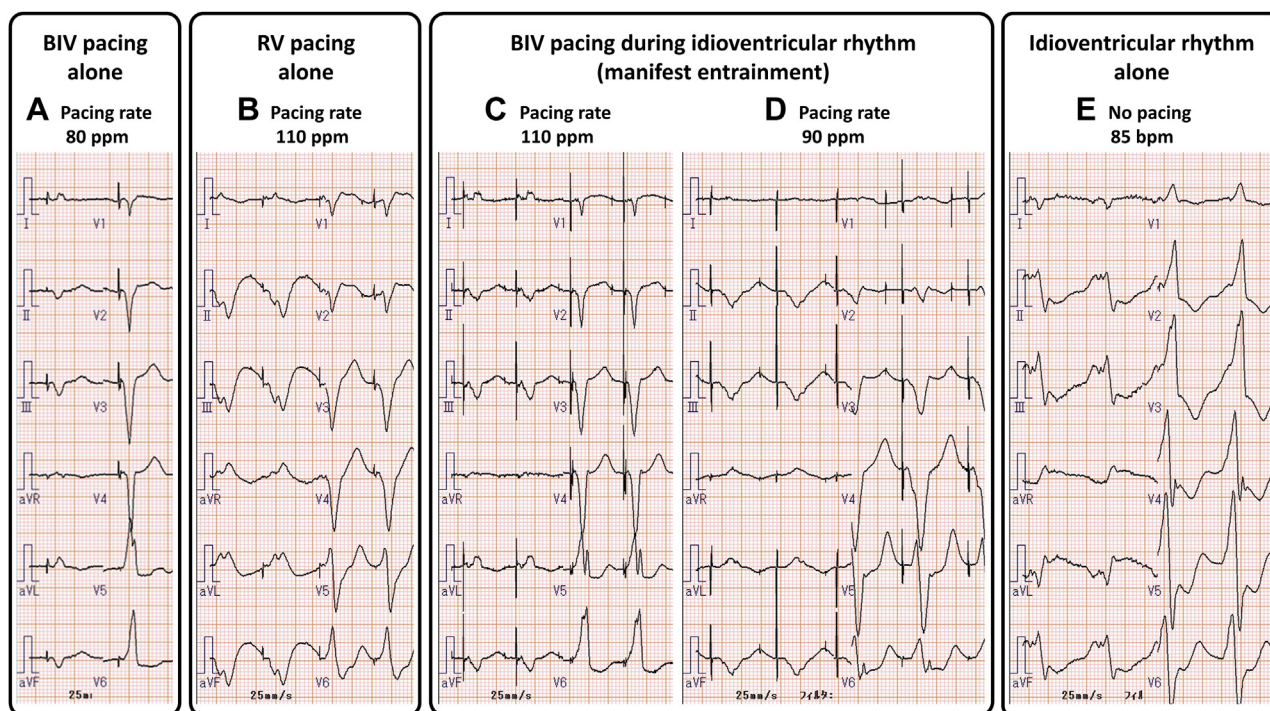


Figure 2 Twelve-lead electrocardiograms during pacing and an idioventricular rhythm. The QRS complex morphology and width completely differ during the biventricular (BiV) pacing (A) and right ventricular (RV) pacing (B) at baseline. C: During widening of the QRS complex morphology on the monitored electrograms, fused QRS morphologies between the BiV pacing and idioventricular rhythm are observed, which is consistent with constant fusion. D: Although rate-dependent changes in the intraventricular conduction or AV nodal conduction could not be completely ruled out, the change in the QRS morphology by the reduction in the BiV pacing rate is consistent with progressive fusion. The QRS complex morphology during constant and progressive fusion assumes an intermediate configuration between the one during BiV pacing alone (A) and that of the idioventricular rhythm alone (E). These findings suggest that this idioventricular rhythm has a reentrant mechanism. bpm = beats per minute; ppm = paces per minute.

that the QRS complex morphology differed from the one during RV-only pacing (Figure 2), which suggested no signs of LV pacing failure. Even during the wide QRS morphology, both the atrial and BiV pacing appeared to work correctly. However, cessation of the BiV pacing during the wider QRS morphology proved that this phenomenon was a continuous resetting of the idioventricular rhythm by the BiV pacing (Figure 1B). The ventricular arrhythmia was not recorded because the ventricular rate was 85 bpm, and it was below the lower rate of 110 ppm. We adjusted the CRT-D settings and changed the lower rate from 110 ppm to 90 ppm, and the 12-lead ECG revealed constant and progressive fusion of the idioventricular rhythm by the BiV pacing (Figure 2). We finally set the lower rate at 80 ppm to unmask this slow ventricular arrhythmia. Then, we administered sotalol in addition to amiodarone and increased the dosage of heart failure medications, including beta blocker and sacubitril/valsartan. Those interventions might have reduced the VT episodes; however, they also slowed down the arrhythmia rate with its tachycardia cycle length of 700–750 ms (80–86 bpm). We sometimes identified this idioventricular rhythm entrained by the BiV pacing from the 12-lead ECG. Although a quantitative analysis of the cardiac output was not performed during the reentrant arrhythmia, the patient complained of fatigue and difficulty breathing during this arrhythmia. He received anti-tachycardia pacing and/or cardioversion each time with the

occurrence of this reentrant idioventricular rhythm both during the hospitalization and at the outpatient clinic. He died 4 months after the second session owing to further worsening of heart failure.

Discussion

This case highlights the importance of recognizing a reentrant idioventricular rhythm as one of the important causes of ineffective BiV pacing. Because the arrhythmia mechanism was reentry, the idioventricular rhythm was entrained by BiV pacing, and the 12-lead ECG with different BiV rate exhibited constant and progressive fusion. Importantly, regardless of the occurrence of reentrant arrhythmia, the telemetry checkup found no abnormal findings, with a correct atrial pace and BiV pacing.

Slow VT (typically defined as VT with its rate of 100–150 bpm) is common in implantable cardioverter-defibrillator recipients and can be a cause of symptomatic deterioration in patients with heart failure.⁵ Conversely, the incidence of idioventricular rhythm (not meeting the criteria for VT owing to its rate of <100 bpm) has not been well described, especially in the setting of heart failure and post-VT ablation. Usually, abnormal automaticity in the His-Purkinje fibers and/or myocardium owing to ischemia and reperfusion injury is the likely mechanism of the idioventricular rhythm.

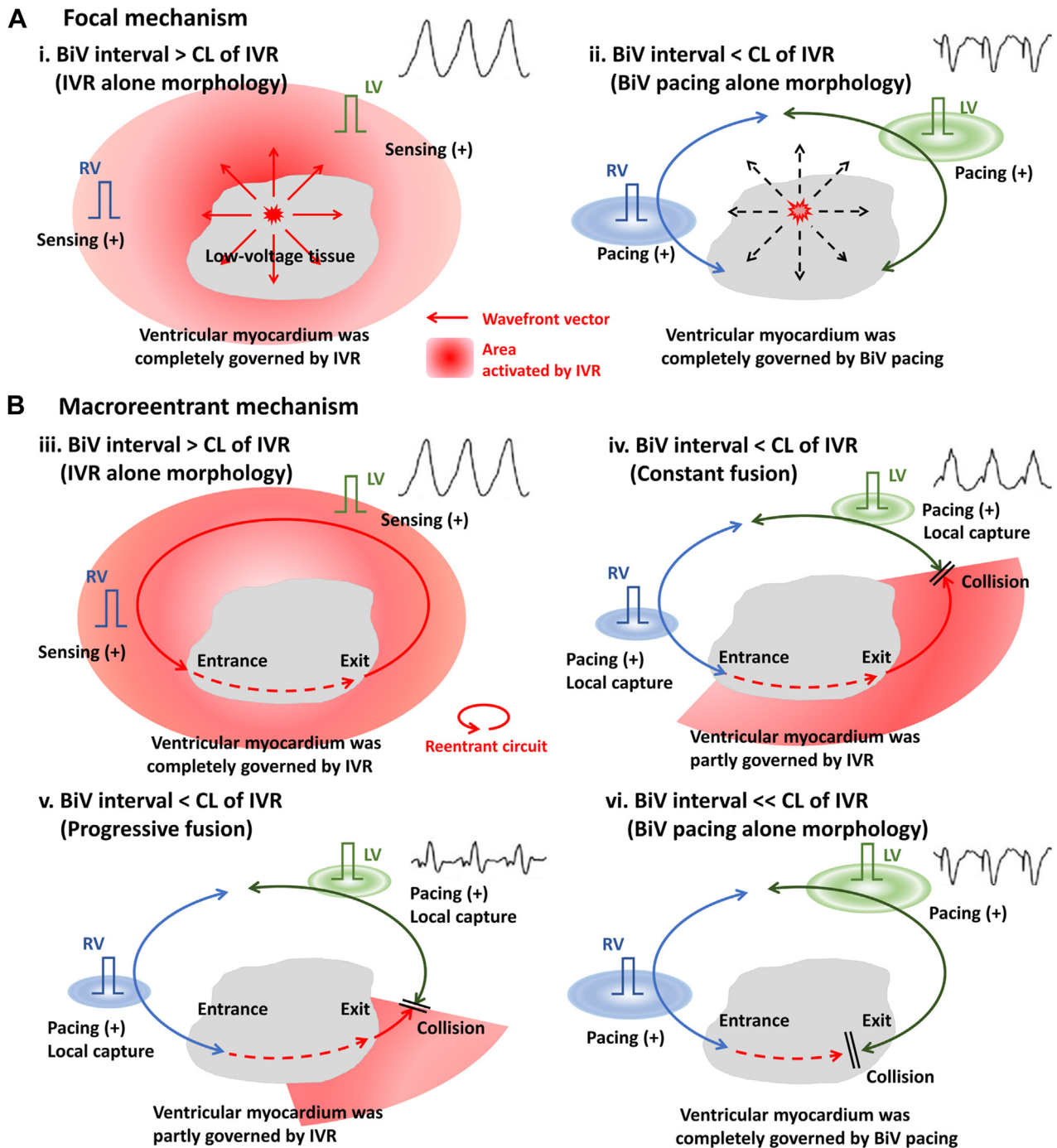


Figure 3 Schematic representation of a different response during the occurrence of an idioventricular rhythm (IVR) under biventricular (BiV) pacing. **A:** When an IVR has a focal mechanism, ventricular myocardium is activated by either the IVR alone (i) or BiV pacing alone (ii). The occurrence of QRS fusion is quite rare because it needs the same and stable interval between the IVR and BiV pacing. The situation is easily resolved by an increase in the BiV pacing rate. **B:** Conversely, when an IVR has a macroreentrant mechanism, the ventricular myocardium can be activated by the IVR alone, BiV pacing, or a combination of the two. When the heart rate of the IVR is faster than the BiV pacing rate, the ventricular myocardium is activated by the IVR alone (iii). However, when the BiV pacing rate is faster than the heart rate of the IVR, constant fusion can be seen by producing a collision site in the outer-loop myocardium (iv). An increase in the BiV pacing rate would produce a greater penetration of the pacing wavefront and result in a shift in the collision site, which would modify the QRS morphology (progressive fusion) (v). A further increase in the BiV pacing rate would either result in a wavefront collision within the slow conduction zone or terminate the reentrant IVR, which would produce the BiV-pacing-alone QRS morphology (vi). bpm = beats per minute, CL = cycle length.

However, in this patient who received VT ablation, the idioventricular rhythm had a reentrant mechanism that may be involved in a remnant heterogeneous tissue (a mixture of surviving normal tissue and scar) as a critical isthmus of the

circuit. Importantly, when the ventricular rate of this idioventricular rhythm is below the lower rate limit of the pacemaker, there can be 2 response patterns according to the mechanism of the tachycardia (Figure 3). When it has a focal mechanism

(automaticity, triggered activity, or microreentry), constant fusion is theoretically quite rare because it needs the same interval between the ventricular arrhythmia and BiV pacing. Conversely, when it has a macroreentrant mechanism, constant fusion is more frequent because it requires not the same interval but a similar interval between the ventricular arrhythmia and BiV pacing. In our case, the heart rate of the ventricular arrhythmia (85 bpm) differed from the one under BiV pacing (110 bpm). The constant and progressive fusion of the two proved that the arrhythmia mechanism was reentry (Figure 2). The extent of antidromic penetration by BiV pacing determines the degree of QRS fusion.

An idioventricular rhythm is generally transient and hemodynamically well tolerated owing to its slow ventricular rate. However, in patients with severe myocardial dysfunction, an idioventricular rhythm may lead to hemodynamic instability owing to the loss of AV synchrony. In the present case, the patient complained of fatigue and difficulty in breathing during the loss of BiV pacing owing to the idioventricular rhythm. After the termination of this idioventricular rhythm, his general condition abruptly improved, which suggested that the idioventricular rhythm was directly associated with his symptoms. There are no established methods as to how to prevent a reentrant idioventricular rhythm during BiV pacing. Although repetitive VT ablation procedures may be a promising method, the elimination of all reentrant circuits is sometimes difficult in patients with extensive myocardial damage. Up-titrating antiarrhythmic drugs or heart failure medications can be an alternative option. However, a low cardiac output and low blood pressure often make it impossible to achieve that. Additionally, these medications have the potential to further decrease the heart rate of arrhythmias. Increasing the lower rate of the BiV pacing may prevent the occurrence of this arrhythmia by expanding the antidromic penetration and inducing wavefront collisions of the pacing and idioventricular rhythm within the slow conduction zone (Figure 3). However, it may increase myocardial oxygen consumption and adversely affect the long-term prognosis in patients with end-stage heart failure. An alteration in the V-V timing can also prevent the occurrence and/or continuation of the reentrant idioventricular rhythm because of the change in the wavefront collision site. However, it may prevent an optimal QRS width and the resolution of dyssynchronous LV wall movements.

Finally, although the identification of this reentrant idioventricular rhythm is important, it is challenging, especially in asymptomatic cases. A decrease in the cumulative percentage of BiV pacing may be a clue to identifying an idioventricular rhythm above the lower limit of the BiV pacing. However, an idioventricular rhythm below the lower limit

of the BiV pacing is difficult to identify with telemetry. The percent ventricular (%V) pacing reported by CRT devices simply indicates the number of paces the device has delivered, and it is not always equal to the proportion of pacing that has captured the LV effectively. As in this case, when the arrhythmia mechanism is reentry, continuous resetting of the idioventricular rhythm with 100% BIV pacing can occur. The Medtronic EffectivCRT diagnostic algorithm (Medtronic Inc, Minneapolis, MN) evaluates whether there is LV capture based on an initial negative deflection of the intracardiac ECG immediately following the pacing spike.³ Such an algorithm may be useful for detecting very slow ventricular arrhythmias by evaluating differences between the %V pacing and percentage of effective LV capture; however, it is not a feature with which all CRT models are equipped. Manual detection based on the symptoms and conventional surface ECG and Holter recordings could help identify reentrant idioventricular rhythm in both inpatient and outpatient care settings.⁶

Conclusion

An idioventricular rhythm, defined as a regular ventricular arrhythmia with a rate of less than 100 bpm, usually occurs as a result of enhanced automaticity. However, a reentrant mechanism should also be considered, especially in patients after VT ablation. We need to acknowledge that the reentrant idioventricular rhythm can be manifestly entrained by BiV pacing and can be a cause of ineffective BiV pacing. Careful observation of the surface ECG (ie, fused QRS morphology) helps identify the occurrence of this slow reentrant arrhythmia.

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