IMAGES IN CARDIOLOGY

Chronic Atrial Overdrive Pacing to Suppress Recurrences of Atrial Fibrillation

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Abstract

A case of a patient with persistent atrial fibrillation is presented who was electrically cardioverted and was subsequently maintained on chronic overdrive atrial pacing and remained free of arrhythmia recurrences. ***Rhythmos 2021; 16(4):82-83.***

Key Words: atrial fibrillation; cardiac pacing; implantable cardioverter defibrillator; coronary artery disease

Abbreviations: AF = atrial fibrillation; AV = atrioventricular; EGM = electrogram(s); ICD = implantable cardioverter defibrillator; VAs = ventricular arrhythmias

A 74-year-old gentleman, ex-smoker, with history of type 2 diabetes, hypercholesterolemia and positive family history of coronary artery disease and prior (anterior) myocardial infarctions, developed severe left ventricular dysfunction with a left ventricular ejection fraction of ~30%, for which he received an implantable cardioverter defibrillator (ICD) for primary prevention of sudden cardiac death. Three years later, he developed persistent atrial fibrillation (AF). He was placed on oral anticoagulation therapy and was scheduled to undergo electrical cardioversion. Before cardioversion and after he had been on anticoagulation for 3 weeks, he also received a 2-week course of oral amiodarone therapy prior to cardioversion, which he continued afterwards.

Electrical cardioversion was successfully performed via the ICD device with the patient put under brief sedation with use of aliquots of intravenous midazolam (1.5 mg) and propofol (30 mg). Recording of intracardiac electrograms (EGMs) via the ICD device displayed the rapid AF waves (atrial sensed – AS electrograms) and the controlled ventricular rate (ventricular sensed – VS electrograms) (**Figure** 1). After having a 20-joule shock delivered via the device (not shown), AF was successfully converted into sinus rhythm at 58 bpm (**Figure** 2). He was subsequently re-programmed to functional atrial pacing (DDD mode with long atrioventricular delay) at a higher rate, initially at 70 ppm (**Figure 3**) and before discharge at 80 ppm (not shown).

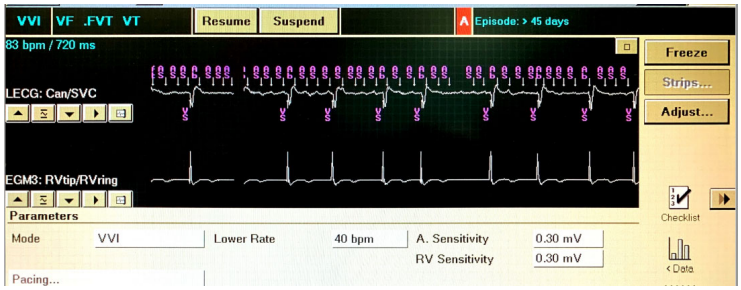

Figure 1

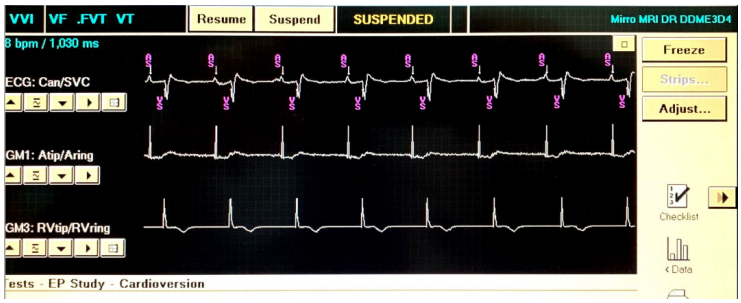

Figure 2

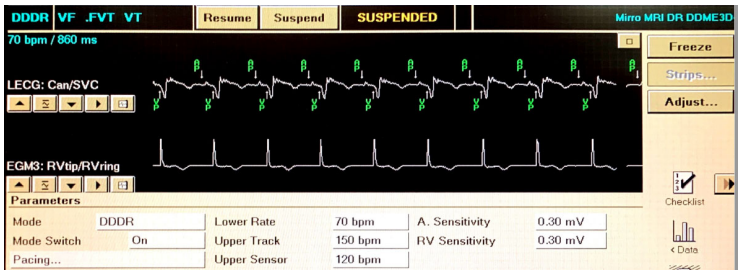

Figure 3

He was discharged on triple therapy (anticoagulation with apixaban, rate control with a beta-blocker, and prophylaxis with amiodarone). He was maintained at higher atrial pacing rate (80 ppm) for the first three months and then gradually reduced to 70 ppm without further arrhythmia recurrences over the ensuing 1 year.

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Overdrive pacing has been an effective mode in controlling ventricular arrhythmias (VAs).[1, 2] In analogy, overdrive atrial pacing could effectively control atrial arrhythmias, like atrial tachycardias and AF.[3] In most cases, particularly in patients with VAs, this approach has been employed as a temporizing measure;[2] however, permanent or chronic overdrive atrial pacing for the prevention of atrial arrhythmias has been tested in several studies.[4] The role that atrial pacing therapy plays on the AF recurrences and arrhythmia burden remains important, albeit not fully elucidated.[4] The optimal pacing site also remains controversial.

Pacing modes using atrial preference pacing algorithms have been reported to significantly reduce the

AF burden, regardless of the site of atrial stimulation (Bachmann's bundle or right atrial appendage), attributed to the high percentage of atrial pacing attained by the atrial overdrive algorithm, that may prevent the relative bradycardia and reduce the number of premature atrial contractions responsible for the reentry that may predispose to and initiate AF.[5]

Alternate site pacing has been suggested to decrease AF recurrences; however, this pacing approach has yielded conflicting and controversial results, at least for the low atrial septal sites or sites in juxtaposition to the coronary sinus; dual-site pacing has been suggested as a better approach.[6-9] However, pacing at a high atrial septal site (Bachmann's bundle) has been proposed as a promising approach.[10, 11]

Patients with AF who may be candidates for preventing pacing could be divided into three groups: (1) patients with sick sinus syndrome and AF, (2) patients with frequent episodes of paroxysmal AF, (3) patients with drug refractory AF.[4] The first patient group seems to be most amenable to an atrial-based pacing approach that seems to effectively reduce both paroxysmal AF and progression to chronic AF. The choice of optimal site or between single- or dual-site has not been determined. Perhaps, the interatrial septum or the Bachmann bundle may have the advantage of a single lead and greater efficacy for AF prevention.[11] Importantly, the implanted dual-chamber pacing system should be programmed with long paced AV intervals to allow intrinsic ventricular activation (functional AAI pacing mode).

What we have empirically applied in this and many other patients on a routine basis is an initial fixed high atrial rate pacing at 80-85 ppm for the first 3-6 months and then at lower rates (70-75 ppm) for all our patients with paroxysmal or permanent AF who are fitted with a pacing device (either a permanent pacemaker or an ICD). We always try to program the device in a functional AAI mode (relatively long AV delay), when possible, to avoid potential deleterious effects of ventricular pacing. Anecdotally, this seems to work, like in the present case, however, a systematic approach and a prospective study is needed to further evaluate such an approach. Nevertheless, one may argue that it was amiodarone that contributed to the favorable course of this patient rather than the mode of pacing, which is a legitimate argument and remains a moot point until further tested in a systematic approach.

We have observed that this particular pacing approach has been most rewarding when combined with atrial pacing effected via a pacing lead placed at a high septal position (Bachmann's bundle); we have applied such pacing with use of a thin and compact, active fixation and steroid-eluting pacing lead and a steerable guiding catheter system.[12] Again, this needs further studies to corroborate or refute our clinical observations.

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