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Revisiting Atrioventricular Nodal Ablation and Cardiac Pacing of Atrial Fibrillation in a Patient with Dextrocardia

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Conflict of interest:	None declared
Patient:	Female, 77
Final Diagnosis:	Tachycardia induced cardiomyopathy
Symptoms:	Shortness of breath
Medication:	-
Clinical Procedure:	-
Specialty:	Cardiology
Objective:	Unusual clinical course
Background:	Poorly controlled ventricular rate associated with atrial fibrillation (AF) leads to tachycardia-induced left ven-
	tricular dysfunction. Atrioventricular (AV) nodal ablation and cardiac pacing is the standard of care in refractory
	congestive heart failure (CHF) due to AF with moderate to rapid ventricular response that failed conventional
	medical therapy. If the patient is not a candidate for AF ablation with pulmonary vein isolation and elimination
	of AF foci, this is an effective approach, but it does have some challenges when done in a patient with dextro-
6 D I	cardia and situs inversus.
Case Report:	Our patient was a 77-year-old woman with dextrocardia and situs inversus, with a history of permanent AF
	due to severe coronary artery disease (CAD), who suffered from recurrent CHF exacerbations from permanent AF with moderate to rapid ventricular response with underlying hypertensive cardiovascular disease. She was
	a poor candidate for pulmonary vein isolation because of her permanent AF status and high risk of recurrence.
	She underwent a technically challenging AV nodal ablation with cardiac pacing due to the complex anatomy,
	with drastic improvement of symptoms within the next 24 h.
Conclusions:	AV nodal ablation with cardiac pacing is the standard of care in patients with refractory AF with moderate to
	rapid ventricular response who have failed medical therapy and are not candidates for pulmonary vein isolation.
MeSH Keywords:	Atrial Fibrillation • Cardiac Pacing, Artificial • Catheter Ablation
Full-text PDF:	https://www.amjcaserep.com/abstract/index/idArt/908654





Background

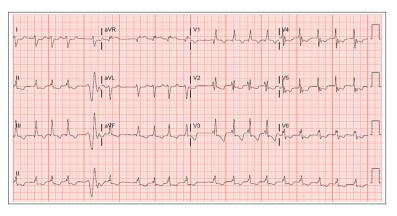
Atrial fibrillation (AF) is a known risk factor for heart failure, its exacerbation, and subsequent poor quality of life [1]. Poorly controlled ventricular rate associated with AF leads to tachycardia-induced left ventricular dysfunction mainly due to relative myocardial ischemia and subsequent depressed contractility, activation of the sympathetic nervous system, renin-angiotensin system activation, and depletion of high-energy phosphate stores [2]. In patients refractory to optimal medical therapy for rate control, atrioventricular (AV) nodal ablation with permanent pacemaker (PPM) implantation is an effective strategy for symptomatic relief [2]. Maintenance of controlled ventricular rate helps to avoid frequent exacerbations of CHF and tachycardia-mediated cardiomyopathy, and is also associated with improved quality of life [2]. In a study enrolling 600 patients, most had restoration of LV function and even remodeling of LV on elimination of tachycardia [3].



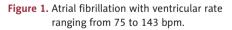
Figure 2. Chest X-ray revealed vascular congestion and dextrocardia. (Note the right sided aortic arch with the apex in the right hemi-thorax and higher left sided hemi-diaphragm).

Case Report

Our patient was a 77-year-old woman with a history of permanent AF due to severe coronary artery disease (CAD) status after coronary artery bypass graft surgery. Due to the history of hypertensive cardiovascular disease and permanent AF with moderate to rapid ventricular response, the patient suffered from recurrent CHF exacerbations and reduced ejection fraction of 30%. She presented to the Emergency Department with worsening shortness of breath and increased palpitations for 1 day. She had no other associated symptoms. On examination, she was afebrile, with blood pressure of 108/55 mmHg, respiratory rate 20/min, and irregular radial pulse with varying rate of 100-150 bpm. Her clinical symptoms were consistent with CHF with associated jugular venous distention of 9 cm of H₂O, positive hepato-jugular reflux, bibasilar faint crackles on auscultation, and bilateral pitting pedal edema up to the level of the mid-shin. Her 12-lead EKG revealed AF with ventricular rate ranging from 75 to 143 bpm (Figure 1) despite therapy with large doses of calcium channel blockers, beta-blockers,



and digoxin. Her chest x-ray showed vascular congestion and dextrocardia (Figure 2) and this could be compared with her chest X-ray 1 month prior to admission (Figure 3). Brain natriuretic peptide was elevated to 2539 pg/mL, while troponin I was negative and her electrolytes were normal. She was a poor candidate for pulmonary vein isolation because of her permanent AF status and high risk of recurrence. She underwent AV nodal ablation and cardiac pacing to control recurrent episodes of AF with rapid ventricular rate, which had resulted in tachycardia-mediated cardiomyopathy and refractory CHF. Complete AV block was achieved after application of 2 radiofrequency energies over the anatomic AV node. A PPM was programmed to VVIR at the rate of 90 bpm (Figure 4). Within 24 h after ablation, her shortness of breath dramatically improved (Figure 5). She was discharged home on warfarin, metoprolol succinate, lisinopril, spironolactone, and furosemide.



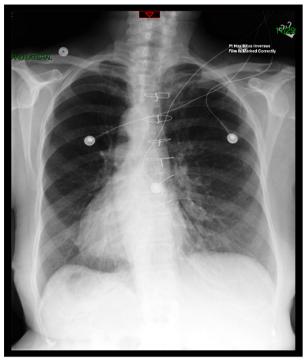


Figure 3. Chest X-ray 1 month prior to admission showing situs inversus, as noted by the apex pointing towards the right and bubble gas under the right hemi-diaphragm.



Figure 5. Chest X-ray showing significant resolution of pulmonary vascular congestion after placement of pacemaker.



Figure 4. Atrial-sensed ventricular paced rhythm after AV nodal ablation and PPM placement.

Discussion

AF is the most common cardiac dysrhythmia, and its prevalence is increasing, with 5.6 million U.S citizens expected to be affected by the year 2050 [4]. The lifetime risk of developing AF between 40 and 95 years of age was 26% in men and 23% in women, as reflected in the Framingham Heart Study [5]. Studies have shown that the most common cardiovascular reason of mortality in AF is heart failure, followed by stroke and other systemic embolizations [6]. One of the causes of recurrent CHF is tachycardia-induced cardiomyopathy. In some patients with AF, ventricular response cannot be controlled, likely because of greatly enhanced AV nodal conduction. Conventional medical therapies, including beta-blockers, non-dihydropyridine calcium channel blockers, digoxin, and anti-arrhythmic drugs, may fail to control the ventricular rate. To avoid enhanced AV node conduction, ablation was done in our case and ventricular rate control was achieved by cardiac pacing. AV nodal ablation was found to be effective for rate control in 97.4% of 646 patients in the 1998 NASPE Prospective Catheter Ablation Registry [7], while in the Ablate and Pace trial, this procedure was successful in 155 out of 156 patients [8]. AV nodal ablation is also extremely effective in alleviation of symptoms and improvement of quality of life due to uncontrolled AF. This was evident in our case as well. In a study of 107 patients, AV nodal ablation significantly reduced physician visits (5 vs. 10 prior to ablation), hospital readmissions (0.17 vs. 2.8 prior to ablation), and CHF exacerbation (8 vs. 18 prior to ablation) [9]. A meta-analysis of 21 studies including 1181 patients showed significant improvement in 19 different measures evaluated, including ventricular function, exercise capacity, quality of life, and healthcare use [10]. However, despite improvement in symptoms and quality of life, studies have failed to reveal any positive impact on overall survival. In a study conducted at the Mayo Clinic, 350 patients who underwent AV nodal ablation and PPM placement did not have better 3-year survival [11]. With ventricular pacing, there is evidence of ventricular dyssynchrony and hence some electrophysiologists prefer biventricular pacing, but in our case, the patient wanted a simpler approach, with an understanding that a cardiac pacing device upgrade could always be done. Furthermore, in the absence of standard data on biventricular pacing in dextrocardia patients, a single-chamber pacemaker was the safer option in our case.

Patients with AV nodal ablation need to have a functioning PPM placed prior to the ablation procedure, and its choice generally depends on the patient's clinical profile. In patients with chronic atrial fibrillation, a single-chamber ventricular pacemaker is sufficient. In these patients, ventricular rate does not respond to increased demand. Thus, as evident in our case, rate-adaptive programming (e.g., VVIR) is used. Patients with paroxysmal AF have an intact AV synchrony during periods of sinus rhythm; therefore, dual-chamber pacemakers are used (e.g., DDDR). These devices almost always have automatic mode-switching capabilities to prevent rapid ventricular pacing during periods of AF. In some patients, there have been

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incidences of post-ablation ventricular fibrillation (VF) and sudden cardiac death (SCD). In a review of 334 patients with recent AV nodal ablation, 2.7% were found to have SCD and most were within 4 days of the procedure. A pacing rate of 90 after the ablation has been found to be most effective in decreasing sympathetic activity, which might contribute to the reduction in VF and SCD [12]. As the AV nodal ablation does not prevent fibrillation of atria, the risk of thromboembolism is not decreased. Thus, there is a need for long-term anticoagulation similar to that in patients who have their ventricular rate controlled pharmacologically.

Conclusions

In patients with refractory CHF due to AF with moderate to rapid ventricular response and who failed conventional medical therapy, AV nodal ablation and cardiac pacing is the standard of care. If the patient is not a candidate for AF ablation with pulmonary vein isolation and elimination of AF foci, this is an effective approach. Although it is a commonly practiced technique among electrophysiologist dating back over 30 years, it is still relevant in the current scenario. If the ejection fraction fails to improve in these patients, left ventricular functioning can be enhanced by biventricular pacing.

Conflict of interest

None.

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