Dual Atrioventricular-Nodal Physiology, Elicited by Pacing and Leading to a Reversible Cardiomyopathy

Atrioventricular nodal re-entry tachycardia is the most common form of regular paroxysmal tachycardia in adults. This tachycardia is a re-entrant rhythm that uses the anatomic location of the atrioventricular node and its surrounding perinodal atrial tissue. The simplest concept underlying the AV-nodal physiology that allows re-entry involves the postulated existence of 2 AV-nodal pathways that have different conduction velocities and refractory periods. Mendez and Moe proposed that an atrio-nodal connection uses fast and slow pathways (termed alpha and beta, respectively) that connect in the distal node and functionally form a circular re-entry loop.

Two leading theories regarding the substrate for tachycardia are 1) the presence of dual AV-nodal pathways that set up a re-entrant tachycardia mechanism and 2) the presence of differential inputs to the AV node, with physiologically variable tissue in and surrounding the node, leading to differential excitability. Atrioventricular nodal re-entry tachycardia has been noted to occur in the pediatric and adult populations, but to varying extents. Studies have shown a positive correlation between the aging process and impairment of AV conduction secondary to degeneration of the AV conduction axis. However, a negative correlation between age and the presence of dual AV-nodal pathways has been described, despite the greater incidence of ectopic beats with older age.

Here, we present the case of a 64-year-old man who developed a tachycardia-induced cardiomyopathy with a perpetual dual response to the stimulus from his permanent pacemaker. The tachycardia displayed characteristic dual atrioventricular-nodal physiology that was suppressed by amiodarone therapy, leading to a reversal of the cardiomyopathy. We discuss the mechanisms that surround such phenomena.
fluids. His current medications included metoprolol, levothyroxine, prednisone, and digitalis. Physical examination revealed an irregular cardiac rhythm and a heart rate of 100 beats/min. His vital signs, including blood pressure and pulse oximetry, were normal. An electrocardiogram (ECG) showed atrial pacing with group bearing. Pacemaker interrogation confirmed a tachycardia with 1 atrial stimulus for every 2 ventricular signals, consistent with dual AV-nodal physiology and a perpetual dual response (Fig. 1). Noninvasively programmed stimulation through the patient’s device evoked this response over a wide excitable gap, with atrial cycle lengths shorter than 500 ms. Echocardiography showed moderate-to-severe left ventricular systolic dysfunction (estimated ejection fraction, 0.30–0.40). After a discussion of the risks and benefits of various treatments, the patient chose pharmacologic therapy with amiodarone. After a 5-g oral load of amiodarone was administered, the dual response was suppressed, and the patient was discharged from the hospital on a daily maintenance dose of 200 mg of amiodarone. Follow-up echocardiography after 3 months revealed normal left ventricular systolic function. Subsequently, diagnostic electrophysiologic testing was performed, and the slow pathway was modified through the delivery of radiofrequency energy. Through 1 year of follow-up, the patient exhibited no documented supraventricular tachycardia, mode switch, or dual response after the amiodarone was discontinued.

Discussion

The common form of AVNRT begins when a normal sinus beat enters the AV node and the impulses travel down the fast and slow pathways. The impulse traveling down the fast pathway reaches the bundle of His, first creating a refractory period. The impulse traveling down the slow pathway is extinguished when it meets the area of the final common pathway that has become refractory from the impulse that had traveled down the fast pathway. If the fast pathway has recovered its excitability by the time the slow-pathway impulse reaches the distal junction of the 2 pathways, the impulse may be able to conduct itself retrograde up the fast circuit, becoming repetitive with anterograde conduction down the slow pathway—resulting in the sustained tachycardia that is termed AVNRT. This type of pathway occurs in 80% of patients with AVNRT. In about 20% of those patients, the re-entrant circuit is atypical. In the atypical circuit, the fast–slow pathway involves anterograde conduction down the fast pathway and retrograde conduction up the slow pathway. Our patient had an unusual form of tachycardia in that there was no re-entry loop. The differential conduction down the fast and slow pathways led to a dual response for every atrial signal, and, consequently, incessant tachycardia. In fact, with a perpetual dual response, the existence of AVNRT is impossible.

The differential diagnosis was limited by device-induced intracardiac ECG results; that is, the ECG results could have been produced by atrial premature beats, with the P wave hidden by the previous QRS. This was satisfactorily ruled out, because the intracardiac electrograms proved 1 atrial electrogram for 2 ventricular responses. Although this effect certainly could have been junctional premature beats, this seemed unlikely, because the 2nd beat was directly related to atrial cycle length, which would be unusual for junctional automaticity. An increase in the pacemaker rate confirmed the presence of a wide excitable gap. The phenomenon was present with atrial-sensed beats of 40 beats/min through and including the paced rates of more than 100 beats/min. Accordingly, the diagnosis of a perpetual dual response was confirmed during pacemaker interrogation.

Our patient’s cardiomyopathy was most likely reversed due to the electrophysiologic effects of amiodarone on the myocardial conduction system and the cessation of the perpetual dual response. Although amiodarone does display Vaughn-Williams Class I, II, and IV antiarrhythmic effects, it is classified as a Class III potassium-channel-blocking antiarrhythmic agent.9 Systolic dysfunction secondary to tachyarrhythmias (atrial and ventricular in origin) is referred to as tachycardia-induced cardiomyopathy. This cardiomyopathy has been described most frequently in the context of uncontrolled atrial fibrillation. A specific diagnosis is reached.
upon a reversal of systolic dysfunction after control of the heart rate is achieved, which occurred in our patient: his cardiomyopathy was reversed after therapy with amiodarone, as evidenced by an increase in left ventricular ejection fraction upon follow-up echocardiography.

In general, it is difficult to reach a definitive diagnosis of tachycardia-induced cardiomyopathy on the basis of measurable values, because such conditions as dilated cardiomyopathy present with symptoms and findings that are similar upon diagnostic evaluation. Tachycardia-induced cardiomyopathy, to the contrary, is reversible after control of the causal arrhythmia, and studies have shown an excellent prognosis after reversal of the arrhythmia. In our patient, confirmation of the reversibility via pharmacotherapy was compelling enough for us to suggest that the patient undergo radiofrequency ablation, an invasive electrophysiologic procedure that he did not initially favor. The antiarrhythmic medications amiodarone and dofetilide are believed to be safe in patients who have heart failure and tachyarrhythmia. Amiodarone was chosen because of its efficacy, and with the hope that, upon proof of the reversal of the cardiomyopathy to the patient, he would then undergo the radiofrequency ablation.

Atrioventricular nodal re-entry tachycardia has been observed as a nonpulmonary-vein trigger for atrial fibrillation. Because there was no further evidence of mode-switching or atrial tachyarrhythmia in our patient, it can be postulated that amiodarone, followed by slow-pathway ablation, also had an impact on the atrial fibrillation. This suggests that the mechanism has an effect beyond rapid atrial rates and decreased atrial refractoriness. Perhaps an atrial myopathy or atrial stretch made our patient’s atrium more vulnerable. Changes in vagal tone may also have played a role.

Conclusion
Dual AV-nodal physiology can occur in the presence of normal or abnormal myocardial substrate. Consequent effects of this physiology can lead to atrial fibrillation or to a tachycardia-induced cardiomyopathy that can predispose patients to structural and arrhythmogenic myocardial changes. The pathophysiology underlying AVNRT often displays dual AV-nodal physiology that indicates the presence of 2 distinct electrophysiologic pathways. The termination of tachycardia by use of the Class III antiarrhythmic agent amiodarone acts to prolong action-potential duration, thus terminating the re-entrant rhythm. As was seen upon echocardiography, our patient’s tachycardia-induced cardiomyopathy was reversed after amiodarone therapy. An anatomic and physiologic understanding of the myocardial conduction system, specifically of the AV node, may enable clinicians to tailor the use of antiarrhythmic pharmacotherapy to the circumstances of individual patients.

References