Chronic Nonparoxysmal Junctional Tachycardia*

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We describe an adult with chronic (three years' duration) acquired nonparoxysmal junctional tachycardia, a previously undescribed rhythm. Ambulatory monitoring revealed junctional rates ranging from 75 to 110 beats/min. Electrophysiologic studies demonstrated intact atrioventricular and ventriculoatrial conduction with a normal H-V interval (43 msec) and narrow QRS. Underlying sinus node function appeared to be normal (recovery time of 900 msec). Junctional rate increased with administration of atropine and isoproterenol, suggesting that the junctional pacemaker was located in the proximal His bundle. Electrocardiographic and electrophysiologic observations suggested that this case of chronic nonparoxysmal junctional tachycardia was benign, not necessitating therapy.

Nonparoxysmal junctional tachycardia is usually a self-limited rhythm, associated with acute myocardial infarction, digitalis toxicity, acute rheumatic carditis, cardiac surgery, or acute electrolyte imbalance. Rarely, this arrhythmia may occur in a chronic congenital form in children. To the best of our knowledge, chronic nonparoxysmal junctional tachycardia has not been previously described in an adult.

In this report, we describe a patient with chronic nonparoxysmal junctional tachycardia associated with chronic obstructive lung disease.

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CASE REPORT

The patient, a 73-year-old woman, was admitted to the University of Illinois Hospital because of respiratory insufficiency. She was known to have had severe obstructive lung disease for three years. She had no history of angina, myocardial infarction, valvular heart disease, or hypertension.

On admission, she was in moderate respiratory distress with labored respirations at a rate of 34/min. The pulse rate was 80 beats/min, and blood pressure was 170/78 mm Hg. There was a prolonged expiratory phase of respiration, and early to mid-inspiratory crackles over both lower lung fields. Cardiac examination results were normal.

A chest roentgenogram revealed a normal cardiac silhouette. There was no laboratory evidence of acute myocardial injury. Serum electrolyte levels were within normal limits, and Digoxin was not detectable in the serum. Arterial blood gas analysis revealed PaO₂ of 60 mm Hg, a PaCO₂ of 55 mm Hg, and a pH of 7.37 while breathing room air (these values subsequently improved with supplemental oxygen therapy and bronchodilatation). Pulmonary function revealed a severe obstructive defect (first-second forced expiratory volume = 0.63 L, 36 percent of predicted; total lung capacity = 3.2 L, 111 percent of predicted; and residual volume = 2.21 L, 140 percent of predicted).

Gated nuclear angiography revealed normal left and right ventricular ejection fractions. Right heart catheterization revealed a cardiac output of 2.7 L/min, pulmonary artery pressure of 34/17 mm Hg, and pulmonary artery occluded mean pressure of 14 mm Hg, while right atrial and ventricular pressures were within normal limits.

Review of ECGs

All resting 12-lead ECGs obtained during hospitalization showed nonparoxysmal junctional tachycardia, with rates ranging from 71 to 83 beats/min (Fig 1). Usually there was 1:1 retrograde atrial capture with a constant interval between QRS complexes and retrograde P waves (Fig 2A). Occasionally, the intervals between QRS complexes and P waves prolonged, until ventricular echo beats occurred (Fig 2B). Occasional premature junctional extrasystoles were also observed. Electrocardiographic records obtained three years earlier revealed similar rhythms.

Twenty-four hour ambulatory electrocardiography demon-
strated nonparoxysmal junctional tachycardia as the dominant rhythm, with rates ranging between 75 and 110 beats/min during the day and between 80 and 100 beats/min during the night.

Electrophysiologic Observations

Electrophysiologic evaluation demonstrated a junctional rhythm with a rate of 77 beats/min, an H-V interval of 43 msec, and intact retrograde conduction to the atria (Fig 3). Atrial and ventricular pacing demonstrated atrioventricular and ventriculotriatal conduction to be intact at rates of 160 beats/min. Extrastimulus testing from the right atrium revealed a continuous A-V nodal conduction curve and normal refractory periods. Extrastimulus testing from the right ventricle revealed a continuous ventriculotriatal conduction curve. No ventricular echo beats were observed during this study.

Following incremental atrial pacing, the escape beat was sinus for two or three beats before junctional rhythm resumed. The sinus escape intervals of 780 to 1000 msec were within the normal range for sinus node recovery time. Following atrial extrastimuli, the recovery beats were of sinus origin (with escape times of 610 to 1,240 msec). Following ventricular extrastimuli, the recovery beats were usually of junctional origin (with escape times of 820 to 950 msec) and occasionally of sinus origin (with escape times of 500 to 530 msec).

Following intravenous administration of 1 mg of atropine sulfate, the junctional rate increased to 104 beats/min, with abolition of premature His bundle beats. Following termination of atrial pacing, the escape beats were always of junctional origin, with escape intervals ranging from 600 to 700 msec, except for one instance when the sinus escaped at 550 msec. Following atrial extrastimuli, the recovery beats were usually junctional (with escape intervals of 610 to 650 msec) and occasionally sinus (with escape intervals of 320 to 800 msec). Following ventricular extrastimuli, the recovery beats were always junctional, with escape times of 690 to 760 msec. During infusion of isoproterenol (maximum infusion rate of 3.3 µg/min) on a different day, the junctional rate increased from 52 to 100 beats/min. Pacing studies were not performed during isoproterenol administration.

**DISCUSSION**

Nonparoxysmal junctional tachycardia is usually a self-limited rhythm, associated with acute myocardial infarction, digitalis toxicity, acute rheumatic carditis, cardiac surgery, or acute electrolyte imbalance. Rarely, this arrhythmia may occur in a chronic congenital

**FIGURE 2.** ECG rhythm strips (lead 2) showing two patterns of retrograde conduction during nonparoxysmal junctional tachycardia. 
*(Panel A)* Retrograde P waves have a constant relationship to QRS complexes. 
*(Panel B)* Gradual prolongation of interval between QRS complexes and retrograde P waves, until (narrow QRS) ventricular echo beat occurs (arrows).

**FIGURE 3.** Intracardiac recording during nonparoxysmal junctional tachycardia. Shown are surface ECG leads 1, 2, 3, and V_{r}, as well as high right atrial (HRA) and two His bundle (HBE) electrograms. Time lines at 1-sec intervals. Each QRS complex preceded by His bundle depolarization (H) with H-V interval of 40 msec. Atrial depolarization (A) is noted in the terminal part of each QRS complex (HA interval = 80 msec). (Transient right bundle-branch block was catheter induced).
form in children. To the best of our knowledge, chronic nonparoxysmal junctional tachycardia has not been previously described in an adult.

The patient we describe is known to have had nonparoxysmal junctional tachycardia for at least three years. Moreover, none of the usual conditions associated with nonparoxysmal junctional tachycardia was identified in our patient, although she has severe chronic obstructive pulmonary disease with little evidence of chronic cor pulmonale. The cause-and-effect relationship of these two conditions is difficult to establish.

There are limited data concerning the electrophysiologic characteristics of junctional rhythms in man. Most observations have been in patients with A-V block and A-V junctional escape, or in patients with sinus node dysfunction and junctional escape. In these patients, the QRS complex, which may be narrow or wide, is preceded by a His bundle deflection with an H-V interval that is either normal or prolonged, with escape rates in the range of 15 to 61 beats/min. The location of the junctional escape pacemaker can be inferred from the escape rate and from the response to cholinergic blockade with atropine or to sympathctic stimulation with isoproterenol. The proximal His bundle would appear to have escape rates of 45 to 60 beats/min and significant increase in automaticity with either vagolyis or catecholamines. In contrast, the distal His bundle would appear to have escape rates of 35 to 45 beats/min and no significant response to vago-lysis, although automaticity may increase with catecholamines.

The junctional pacemaker in the present case had several interesting characteristics. First, under both basal and ambulatory conditions, junctional automaticity was greater than sinus automaticity, hence the occurrence of chronic nonparoxysmal junctional tachycardia. Second, escape rates, observed following atrial and ventricular stimulation, were shorter for the sinus node than the junction, suggesting that if the junction should slow, the sinus node would probably be available. The responses to atropine and isoproterenol suggest that the junctional pacemaker in this patient was probably in the proximal His bundle (vide supra). It was of interest that the junctional pacemaker had a greater sensitivity than the sinus node in regard to automaticity, since neither atropine nor isoproterenol established sinus tachycardia, but resulted in faster nonparoxysmal junctional tachycardia.

The clinical significance of nonparoxysmal junctional tachycardia in our case is its chronicity and benign course. When seen in association with acute myocardial infarction, digitalis toxicity, acute rheumatic carditis, and acute electrolyte imbalance, nonparoxysmal junctional tachycardia often implies a serious prognosis. However, our patient has done relatively well with this stable rhythm for at least three years. Chronic loss of atrial contribution to stroke volume did not appear to be a significant problem in our patient.

The future course of our patient's arrhythmia is unknown. The sinus node appears intact from electrophysiologic study. Therefore, slowing of junctional rate in the future would appear to be of no major threat to our patient. Acceleration of the junctional rate is a potential risk in our patient, although the stability of nonparoxysmal junctional tachycardia over three years does not suggest that this will occur.

The importance of this case is the recognition that nonparoxysmal junctional tachycardia can occur in the absence of acute myocardial infarction, digitalis toxicity, acute rheumatic carditis, cardiac surgery, or acute electrolyte imbalance, and that this rhythm can exist in a chronic form with a relatively benign course. The clinical and electrophysiologic observations that we made in regard to this patient support a conservative approach to management.

REFERENCES