Catecholamine-dependent Atrioventricular Nodal Reentrant Tachycardia

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SUMMARY An unusual case of atrioventricular nodal reentrant tachycardia precipitated by ethanol ingestion is presented. Programmed atrial and ventricular stimulation failed to induce the tachycardia during control conditions or after atropine administration. This failure to induce tachycardia was related to the absence of ventriculoatrial conduction. A low-dose isoproterenol infusion allowed induction of atrioventricular nodal reentrant tachycardia by the enhancement of ventriculoatrial conduction. This report suggests that programmed stimulation during isoproterenol infusion can be used to induce paroxysmal supraventricular tachycardia in suspected cases in whom induction during control conditions or after atropine administration is not possible.

ELECTROPHYSIOLOGIC studies and programmed atrial and ventricular stimulation have been widely used to evaluate patients with paroxysmal supraventricular tachycardia due to reentry within the atrioventricular (AV) node. A critical AH interval prolongation and a critical balance between conduction and refactoriness of the reentrant pathways are required for the initiation of the tachycardia. In most cases those conditions can be achieved and tachycardia can be initiated by programmed atrial stimulation or by rapid atrial pacing. In some, initiation and sustainment of the tachycardia can only be achieved after a small dose (0.5 mg) of atropine. We recently observed an unusual case of paroxysmal supraventricular tachycardia associated with alcohol ingestion. Paroxysmal supraventricular tachycardia in this case could not be induced during repeated electrophysiologic studies before and after the administration of atropine. However, sustained AV nodal reentry could be induced during infusion of a small dose of isoproterenol. This report describes the mechanism by which isoproterenol permits induction of the tachycardia.

Case Report

The patient was a 79-year-old man with recurrent palpitations that only occurred within a few hours after ingestion of moderate-to-heavy amounts of ethanol. The patient was admitted in 1979 to the VA Medical Center in Brooklyn, New York, for ethanol intoxication. A routine ECG at the time of admission revealed a supraventricular tachycardia at a rate of 150 beats/min. The patient underwent electrophysiologic studies 4 days after admission. Atrial stimulation failed to induce either atrial echo beats or supraventricular tachycardia during control conditions or after i.v. atropine (0.5 mg). No ventriculoatrial conduction could be demonstrated during the studies. In February 1982, the patient was readmitted for palpitation, which again occurred about 2 hours after ingestion of ethanol.

Rhythm strips taken at the time of admission (fig. 1) revealed sinus tachycardia of 118 beats/min and paroxysms of supraventricular tachycardia initiated by a premature atrial beat followed by a prolonged PR interval of 360 msec. The tachycardia persisted for about 5 minutes at a rate of about 150 beats/min. After spontaneous termination of the tachycardia (fig. 1C), a ventricular escape beat was noted. For a few seconds, the sinus rate after the tachycardia was slower than the sinus rate before the tachycardia, probably because of overdrive suppression of the sinus node by the tachycardia. The patient underwent electrophysiologic studies 3 days after admission, when there were no signs of either alcohol intoxication or alcohol withdrawal.

Electrophysiologic Studies

At the time of the study, the patient had not taken any cardioactive medication for at least 48 hours. The nature of the study was explained and informed consent was obtained. Three quadripolar electrode catheters (interelectrode distance 1 cm) were introduced percutaneously into the right femoral vein and the brachial vein and positioned under fluoroscopy in the region of the middle right atrium, His bundle and right ventricular apex. Programmed atrial and ventricular stimulations were performed during control conditions, after 0.5 mg of i.v. atropine sulfate (which was repeated twice at 10-minute intervals to a total dose of 1.5 mg), and during i.v. isoproterenol administration (0.5 µg/min) initiated about 60 minutes after the last dose of atropine sulfate, when the heart rate had returned to the control level.

Table 1 shows the electrophysiologic findings. The control resting sinus rate was 65–75 beats/min; after atropine, the sinus rate reached a maximum of 90 beats/min, and during isoproterenol infusion reached 100 beats/min. The spontaneous AH intervals during control, after atropine and during isoproterenol administration were 160, 150 and 150 msec, respectively; the HV intervals remained constant at 45 msec. During control conditions, incremental atrial pacing resulted in progressive prolongations of AH intervals from 180 to 470 msec until AV nodal Wenckebach periodicity occurred at a pacing cycle length of 510 msec. After 1.5 mg of atropine (total dose) had been given, incremental atrial pacing resulted in a maximum AH interval of 400 msec before AV nodal Wenckebach periodicity occurred at a pacing cycle length of 490 msec.

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No atrial echo beats or sustained AV nodal reentrant tachycardia could be induced during control conditions or after atropine administration. During isoproterenol administration, incremental atrial pacing resulted in AV nodal Wenckebach periodicity at a pacing cycle length of 460 msec and a maximal AH interval of 380 msec. Constant atrial pacing at this time produced also sustained AV nodal reentrant tachycardia preceded by critical AH intervals between 315 and 380 msec.

Figure 2 shows the results of constant atrial pacing at a cycle length of 480 msec during control, after atropine and during isoproterenol administration. During control conditions, the last of a series of paced atrial beats resulted in an AH interval of 460 msec, but induced no reentrant tachycardia. After a total of 1.0 mg of atropine, the last atrial beat resulted in an AH interval of 350 msec and no reentrant tachycardia. After atropine, the sinus beat escaped 860 msec after the last paced atrial beat; in contrast, during control, the sinus escape interval was 1180 msec. During isoproterenol infusion (0.5 μg/min), a short burst of atrial pacing at a cycle length of 480 msec resulted in a supraventricular tachycardia with a cycle length of 440 msec, preceded by an AH interval of 350 msec. During the tachycardia, atrial electrograms showed low-to-high activation and occurred within and immediately after the ventricular complexes. The His-to-atrial echo interval (measured from the His bundle deflections to the atrial echo recorded in the middle right atrium) was 80 msec.

Figure 3 shows that the tachycardia could be terminated by a single atrial premature depolarization induced 320 msec after the last atrial electrogram during the tachycardia. After the tachycardia, a sinus beat escaped 670 msec after the atrial premature depolarization. The initiation of the tachycardia depending on a critical AH interval, the termination of the tachycardia by a single atrial premature depolarization and the relationship between the time of activations of the atria and ventricles suggest that the tachycardia was due to reentry within the AV node (type I).

Figure 4A shows plots relating coupling intervals of premature atrial depolarizations (A1, A2) delivered after eight paced atrial beats at a cycle length of 600 msec and the resulting H1, H2 intervals. Discontinuous AV nodal conduction curves were observed during control, after atropine (1 mg total dose) and during i.v. isoproterenol infusion. The effective and functional refractory periods of the fast AV nodal pathway during control were 520 and 545 msec, after atropine 480 and 520 msec, and during isoproterenol infusion 480 and 520 msec.

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Abbreviations: ERP = effective refractory period; AV = atrio-ventricular; FRP = functional refractory period.
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Figure 2. Effects of constant atrial pacing at a cycle length of 480 msec during control conditions (A), after atropine (B), and during isoproterenol infusion (C). The last three stimulated beats are shown in each panel. Tracings from ECG leads I, II and V1 and from the mid-right atrial (RA) and His bundle (HB) areas are shown. Thick arrows indicate pacing artifacts; A = atrial electrogram; AE = atrial echo; H = His bundle deflections; V = ventricular electrogram. Thin arrows indicate the direction of conduction. Note the failure of atrial pacing to induce paroxysmal supraventricular tachycardia during control and after atropine and the initiation of paroxysmal supraventricular tachycardia by atrial pacing during isoproterenol infusion. The longest AH interval during control is 460 msec, after atropine 350 msec, and during isoproterenol infusion 350 msec. During paroxysmal supraventricular tachycardia HA_E interval (measured from the His bundle deflection to the AE recorded in RA tracing) was 80 msec and AE_H was 360 msec.

510 msec, respectively. The effective and functional refractory periods of the slow pathway were 460 and 620 msec during control, 420 and 615 msec after atropine, and 410 and 610 msec during isoproterenol. During isoproterenol infusion, atrial premature depolarizations delivered at A1A2 coupling intervals of 410–430 msec induced the tachycardia.

The AH intervals that initiated the tachycardia during isoproterenol infusion were shorter than the maximal AH intervals during control and after atropine.

Figure 3. Termination of paroxysmal supraventricular tachycardia by a single atrial premature depolarization (Ap), which induced block in retrograde conduction. Abbreviations and symbols are as in figure 2.
vascular pacing at a cycle length of 440 msec, the cycle length of the AV nodal reentrant tachycardia. During control and after atropine administration (1.5 mg), no ventriculoatrial conduction could be seen. The atrial cycle length was 720–810 msec during control and 640–680 msec after atropine. Isoproterenol infusion resulted in 1:1 ventriculoatrial conduction with a ventriculoatrial interval of 130 msec.

Figure 4B shows plots relating coupling intervals of premature ventricular stimuli (S1S2) delivered after eight regular ventricular stimuli at a cycle length of 600 msec and the resulting A1A2 and H1A2 intervals. During control conditions, ventriculoatrial conduction did not occur. After atropine (total 1.5 mg), a premature ventricular stimulus at an S1S2 of 510 msec resulted in ventricular capture and ventriculoatrial block. The block probably occurred in the AV node, because more premature ventricular stimuli with an S1S2 of 270–290 msec resulted in ventricular capture and appearance of His bundle deflection after the ventricular complexes. The effective refractory period of the right ventricle after atropine administration was 260 msec. During isoproterenol infusion, progressively premature ventricular complexes resulted in ventriculoatrial conduction until the right ventricular effective refractory period was reached at an S1S2 of 210 msec. Thus, the effective refractory period of the ventriculoatrial conduction system was 510 msec after atropine and was less than 210 msec during the isoproterenol infusion. The functional refractory period of the ventriculoatrial conduction system was 570 msec after atropine and 370 msec during the isoproterenol infusion. S1S2 intervals of 270–230 msec resulted in V1H1 intervals of 150–210 msec and H1A2 intervals of 70–80 msec. During progressively shorter S1S2 intervals, S1A1 intervals prolonged from 120 to 280 msec.

Discussion

The present case is unusual in two respects: (1) AV nodal reentrant tachycardia occurred only after ingestion of ethanol. (2) During electrophysiologic studies, tachycardia could not be induced during control or after administration of 1.5 mg of atropine, but could be induced during infusion of a low dose of isoproterenol (0.5 μg/min).

Increased blood ethanol concentrations are associated with increased sympathetic responses, probably indirectly through an increase in blood acetaldehyde concentrations. The paroxysmal supraventricular tachycardia in this case occurred at the time ethanol caused an increase in his sympathetic tone, reflected by the presence of sinus tachycardia. Various arrhythmias associated with ethanol ingestion, including paroxysmal atrial tachycardia and junctional tachycardia (“holiday heart syndrome”), have been reported; however, the electrophysiologic mechanisms in these cases have not been studied. Before we conducted the most recent electrophysiologic studies, we had postulated that catecholamines might play important role in this case. This postulation was based on the history obtained from the patient and the failure to elicit supraventricular tachycardia in the previous electrophysio-
logic studies despite the administration of atropine. This case also stresses the importance of the presence of ventriculoatrial conduction as a prerequisite for the induction of AV nodal reentrant tachycardia. It is doubtful whether paroxysmal supraventricular tachycardia due to AV nodal reentry or reentry using the accessory pathway can occur in the absence of ventriculoatrial conduction. The absence of ventriculoatrial conduction in humans without AV bypass is frequently related to retrograde AV nodal block. The site of ventriculoatrial block during control and after atropine in this case was also probably in the AV node, because during S1S2 ventricular stimulation, His bundle deflections not followed by atrial electrograms could be seen after the premature ventricular complex. Possibly, in patients with AV nodal reentry the retrograde limb of the AV nodal reentrant pathway is used for ventriculoatrial conduction during ventricular pacing. In support of this belief is the fact that H1A1 intervals during S1S2 ventricular stimulation in those patients are identical to H1A2 intervals during AV nodal reentrant tachycardia. Thus, isoproterenol probably improved ventriculoatrial conduction by improving conduction through the retrograde limb of the reentrant pathway. By doing so, isoproterenol also allowed induction of AV nodal reentrant tachycardia.

The present report illustrates the importance of ventriculoatrial conduction as a prerequisite of paroxysmal supraventricular tachycardia due to AV nodal reentry. A small dose of isoproterenol can be used in cases of paroxysmal supraventricular tachycardia that cannot be induced at the time of electrophysiologic studies during control conditions or after atropine and that do not exhibit good ventriculoatrial conduction. To our knowledge, such practice using isoproterenol infusion during induction of paroxysmal supraventricular tachycardia is uncommon and probably should be used more frequently.

References
Catecholamine-dependent atrioventricular nodal reentrant tachycardia.
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