Hyperventilation Facilitates Induction of Supraventricular Tachycardia: A Novel Method and the Possible Mechanism

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Hyperventilation and Supraventricular Tachycardia. Introduction: Hyperventilation has been demonstrated to alter autonomic function. Sympathomimetic drugs (isoproterenol) and parasympatholytic drugs (atropine) may be needed to facilitate induction of supraventricular tachycardia (SVT). The aim of this study was to test the clinical utility and mechanisms of hyperventilation to facilitate SVT initiation.

Methods and Results: Fourteen patients with clinically documented SVT (9 AV nodal reentrant tachycardia and 5 AV reciprocating tachycardia) but noninducible during baseline electrophysiologic study were included. Immediately after hyperventilation test (at least 30 respirations/min) for 2 minutes, systolic blood pressure, sinus cycle length, anterograde and retrograde 1:1 conduction, and induced SVT were measured. Arterial blood gas, pH, and heart rate variability before and after hyperventilation were measured. Seven of nine patients with AV nodal reentrant tachycardia and 3 of 5 patients with AV reciprocating tachycardia could be induced immediately after the hyperventilation test. After hyperventilation, anterograde AV and retrograde VA 1:1 conduction were improved, sinus cycle length was decreased, and heart rate variability were decreased in both groups.

Conclusion: Hyperventilation can facilitate induction of SVT. Improvement of conduction properties and changes of autonomic function are the possible mechanisms. (J Cardiovasc Electrophysiol, Vol. 12, pp. 1242-1246, November 2001)

hyperventilation, supraventricular tachycardia, autonomic function, atrioventricular nodal reentry, atrioventricular reentry

Introduction

Several investigators reported that sympathomimetic or parasympatholytic agents can facilitate induction of supraventricular tachycardia (SVT) during electrophysiologic study.1-4 Furthermore, increases in sympathetic tone and withdrawal of parasympathetic tone, or both, can facilitate spontaneous onset of tachycardia, for example, during exercise. Thus, the effects of autonomic tone on cardiac tissue may play an important role in the occurrence of arrhythmias.5 Hyperventilation can induce ST-T wave changes in patients without heart disease and produce coronary artery spasm in patients with variant angina,6-10 effects related in part to changes of autonomic function. However, the effects of hyperventilation on sinus and AV nodal function and inducibility of SVT have not been reported. The aim of this study was to test the hypothesis that hyperventilation can facilitate induction of SVT by alterations of the sympathovagal balance on AV nodal conduction.

Methods

Patient Characteristics

Fourteen patients (8 men and 6 women, mean age 35 ± 13 years) with clinically documented SVT that could not be induced during baseline electrophysiologic study were included. Nine patients had AV nodal reentrant tachycardia (AVNRT) and 5 had Wolff-Parkinson-White (WPW) syndrome and AV reciprocating tachycardia (AVRT). None of the patients had hypertension, coronary artery disease (all had a normal treadmill test and no symptoms), diabetes mellitus, or other systemic diseases. No patient had a history of initiation of tachycardia after hyperventilation.

Baseline Electrophysiologic Study

Each patient gave informed written consent for the electrophysiologic study. All antiarrhythmic drugs were discontinued for at least five half-lives before study.5,11,12 The patients received no sedative or analgesic premedication. Three multipolar, closely spaced (2-mm) electrode catheters (Mansfield Scientific, Mansfield, MA, USA) were inserted percutaneously into the right and left femoral veins and positioned in the high right atrium, His-bundle area, and right ventricle. The catheters were used for recording and for programmed atrial and ventricular stimulation. One or-
thogonal electrode catheter (12 poles, distal 3 cm free of electrode; Mansfield Scientific) or one decapolar electrode catheter (5-mm spacing; Daig, Minneapolis, MN, USA) was inserted percutaneously into the right internal jugular vein and positioned in the coronary sinus to record electrical activity around the posteroseptal and coronary sinus area. Intracardiac electrograms were filtered at 30 to 500 Hz and simultaneously displayed with surface ECG leads I, II, and V₁ on a multichannel oscilloscope (Cardiolab, Prucka Engineering, Houston, TX, USA). Electrical stimulation was performed with a programmable stimulator (Bloom Associated Ltd., Narbeth, PA, USA) that delivered rectangular pulses of twice diastolic threshold and a pulse duration of 2 msec. All stimuli were delivered through an isolation unit, and the electrical equipment was grounded.

Baseline electrophysiologic study protocols for AVNRT and AVRT were described previously, including (1) determination of effective refractory periods (ERP) of the right atrium, AV node (fast and slow pathways in the anterograde and retrograde directions), and right ventricle at pacing cycle lengths of 500 and 600 msec; (2) atrial and ventricular decremental pacing to the points of AV and VA conduction block; and (3) induction of tachycardia using decremental atrial or ventricular pacing, single and double extrastimulation from the right atrium or right ventricle. Diagnosis of AVNRT and AVRT was made by classic criteria and by excluding atrial tachycardia.5,11,12

**Hyperventilation Test**

Hyperventilation test13,14 was performed after baseline electrophysiologic study in the electrophysiology laboratory. All subjects were asked to breathe rapidly (at least 30 respirations/min) for 2 minutes. ECG and blood pressure by femoral arterial line were monitored continuously. Arterial blood gas and pH value were checked at baseline, during 2 minutes of hyperventilation, and immediately after hyperventilation. The first hyperventilation test was done to analyze heart rate variability (HRV). No electrophysiologic study was done at this time. After the first hyperventilation test, a 10-minute waiting period was allowed for conditions to return to the control state. Then, a second hyperventilation test was performed, and electrophysiologic study was performed immediately after the hyperventilation test to evaluate sinus cycle length (SCL), AV and VA 1:1 conduction, and inducibility of SVT. ERP measurements were not evaluated.

**Frequency-Domain Measurements of HRV**

Power spectrum analysis of HRV was performed using fast Fourier transformation on each 2-minute segment of recording before and after the first hyperventilation test. Power spectrum was quantified by the area (power) in two frequency bands: (1) low-frequency (LF) power between 0.08 and 0.15 Hz; and (2) high-frequency (HF) power between 0.15 and 0.45 Hz, via modification of our computer algorithms in a continuous on-line and real-time analysis of heart rate signals designed for rats.15,16 Additionally, the LF to HF (L/H) ratio was calculated simultaneously. This software has been verified.15,16

**Statistical Analysis**

Statistical significance of differences between mean values was tested with paired and unpaired t-tests as appropriate. P < 0.05 was considered statistically significant.

**Results**

**Patients with AVNRT**

All patients had anterograde dual AV nodal pathway physiology, but none had retrograde dual AV nodal pathway physiology. At baseline electrophysiologic study, the shortest pacing cycle lengths with 1:1 conduction over the anterograde fast and slow pathways were 378 ± 51 msec and 368 ± 51 msec, respectively. Retrograde 1:1 conduction was over the fast pathway with a minimal pacing cycle length of 440 ± 91 msec. No patient had tachycardia induced at baseline study. Two patients had two AV nodal reentrant echoes with anterograde block, and seven patients had one AV nodal reentrant echo with anterograde block.

After 2 minutes of hyperventilation, significant improvement of 1:1 conduction occurred over the anterograde fast pathway (378 ± 51 msec vs 311 ± 40 msec; P < 0.001), anterograde slow pathway (368 ± 51 msec vs 301 ± 40 msec; P < 0.001), and retrograde fast pathway (440 ± 91 msec vs 319 ± 49 msec; P < 0.001) (Fig. 1). Seven of nine patients had tachycardia induced with rapid atrial pacing after hyperventilation. Systolic blood pressure (SBP) was not significantly changed (127 ± 19 mmHg vs 133 ± 20 mmHg; P > 0.05) after hyperventilation; however, SCL was significantly shortened from 695 ± 140 msec to 543 ± 40 msec (P < 0.001) (Fig. 1). Arterial blood gas showed a significant increase of pH value (7.38 ± 0.03 vs 7.60 ± 0.02; P < 0.001). Furthermore, HRV analysis showed a significant decrease of LF (364 ± 196 msec² vs 12.8 ± 15.7 msec²; P < 0.01), HF (177.2 ± 92.2 msec² vs 11.2 ± 14.7 msec²; P < 0.01), and L/H ratio (2.2 ± 0.2 vs 1.1 ± 0.4; P < 0.005) (Figs. 2 and 3).

**Patients with AVRT**

All of the patients had a single accessory pathway with bidirectional conduction. At baseline electrophysiologic
study, the shortest pacing cycle length with 1:1 anterograde conduction over the accessory pathway was 342 ± 26 msec. Anterograde AV nodal conduction properties could not be measured because atrial pacing and extrastimulus exclusively conducted over the accessory pathway. In all patients, when the premature atrial beats were blocked over the accessory pathway, they also could not conduct over the AV node. The shortest pacing cycle length with 1:1 retrograde conduction over the accessory pathway was 294 ± 28 msec. No patients had tachycardia or echo beats induced at baseline study. After 2 minutes of hyperventilation, significant improvement of accessory pathway 1:1 anterograde conduction (342 ± 26 msec vs 316 ± 32 msec; P = 0.01) and retrograde conduction (294 ± 28 msec vs 262 ± 16 msec; P = 0.005) was demonstrated (Fig. 4). Three of five patients had tachycardia induced with rapid atrial pacing. SBP was not significantly changed (125 ± 11 mmHg vs 131 ± 14 mmHg; P > 0.05) after hyperventilation; however, SCL was significantly shortened from 717 ± 58 msec to 598 ± 45 msec (P < 0.001) (Fig. 4). Arterial blood gas showed significant increase of pH value (7.37 ± 0.04 vs 7.59 ± 0.03; P < 0.001). Furthermore, HRV analysis showed a significant decrease of LF (456.5 ± 160.9 msec² vs 27.5 ± 23.4 msec²; P < 0.01), HF (196.1 ± 92.6 msec² vs 18.8 ± 16.7 msec²; P < 0.01), and L/H ratio (2.55 ± 0.64 vs 1.38 ± 0.43, P < 0.05) (Fig. 2).
Comparison of Changes of AV Node and Accessory Pathway After Hyperventilation

Improvement of anterograde and retrograde conduction properties were more significant in the AV node than accessory pathway (anterograde, −18% ± 8% vs −8% ± 4%, P < 0.05; retrograde −25% ± 15% vs −11% ± 3%, P < 0.05) conduction after hyperventilation. However, there were no significant differences in ΔSCL, ΔSBP, and ΔL/H ratio between the two groups (Fig. 3).

Noninducibility of SVT after Hyperventilation

Seven of nine patients with AVNRT and 3 of 5 patients with AVRT could be induced after hyperventilation (Fig. 5). For AVNRT, initiation of tachycardia was facilitated by improved anterograde slow pathway conduction and retrograde fast pathway conduction. In AVRT, initiation of tachycardia might be facilitated by better anterograde AV node conduction than anterograde accessory pathway conduction. AVNRT could not be induced at baseline study or after hyperventilation in two patients, who had one or two induced echo beats with anterograde block in the slow pathway after hyperventilation. AVRT could not be induced at baseline study and after hyperventilation in two patients, who also also had one or two echo beats with anterograde conduction block in the AV node after hyperventilation.

Discussion

Main Findings

To our knowledge, this is the first study demonstrating that hyperventilation can alter the electrophysiologic properties of AV nodal and accessory pathway conduction and facilitate induction of SVT. Further, hyperventilation has a significant effect on HRV.

Hyperventilation and Autonomic Tone

HF power reflects modulation of vagal tone, primarily by breathing, and the amplitude of HF power is measurably influenced by tidal volume and frequency of breathing. Hayano et al. estimated that LF power is approximately two thirds dependent on vagal tone and only one third on nonvagal influences. The L/H ratio is an indicator of sympathovagal balance. During hyperventilation, LF and HF components were decreased and the L/H ratio also was decreased; thus, it may be caused by vagal withdrawal and sympathovagal imbalance.

Relation Between Autonomic Tone and SVT

Although isoproterenol can facilitate induction of sustained AVNRT, induction of tachycardia can still be difficult in some patients. Several investigators reported that some AVNRT could be induced after intravenous infusion of atropine. They also demonstrated that atropine could improve retrograde fast pathway conduction. Thus, both enhanced sympathetic and reduced parasympathetic tone may allow AVNRT to be induced at electrophysiologic study.

Spectral analysis of HRV can, in part, separate parasympathetic from sympathetic drive to the heart. In this study, we demonstrated that heart rate increased consistent with withdrawal of vagal tone and/or increase of sympathetic tone and that the decrease in HF is consistent with withdrawal of vagal tone. Failure of the L/H ratio to change in the expected direction suggests that the effect of hyperventilation on sympathetic tone and parasympathetic/sympathetic balance is complex. These changes resulted in better anterograde and retrograde AV node conduction and initiation of AVNRT in 7 of 9 patients who were not inducible before hyperventilation. These changes also resulted in better anterograde AV node conduction than anterograde accessory pathway conduction and initiation of AVRT in 3 of 5 patients who were not inducible before hyperventilation.

In our study, patients did not have a history of hyperventilation preceding onset of tachycardia. However, this certainly could facilitate induction of paroxysmal SVT in some patients and may lead to misdiagnosis of a panic disorder.

Comparison Between AVNRT and AVRT

Although this study showed no significant differences of ΔL/H ratio and ΔSCL between the two groups after hyperventilation, the improvement of AV nodal conduction in patients with AVNRT was more significant than improved accessory pathway conduction in both the anterograde and retrograde direction. This was likely due to the more pronounced effect of parasympathetic tone on AV nodal tissue than the accessory pathway. Thus, the greater effect of vagal withdrawal showed more significant improvement in anterograde and retrograde conduction of AV node than in the accessory pathway conduction after hyperventilation. Analysis of AV node conduction changes between patients with AVRT and AVNRT could not be made, because accessory pathway conduction precluded accurate determina-
tion of AV node conduction in patients with WPW syndrome.

Study Limitations

It is clear that hyperventilation facilitates AV node and accessory pathway conduction. Although changes in HRV suggest a major effect of hyperventilation is parasympathetic withdrawal, this is measured by sinus nodal automaticity. Because automatic perturbations can differ in the sinus and AV nodes, it is not known whether the primary effect on the AV node was vagal withdrawal, sympathetic increase, or both. A second limitation is the possibility of spontaneous variability in induction of paroxysmal SVT. We doubt this was a significant factor in our study, because aggressive attempts were made to induce paroxysmal SVT before hyperventilation, and only a short time elapsed from control to hyperventilation study.

Conclusion

This study demonstrated that hyperventilation facilitates induction of SVT by altering AV nodal conduction. Hyperventilation test is a simple maneuver and can be attempted before routine use of isoproterenol or atropine. Hyperventilation may facilitate spontaneous initiation of SVT, especially in patients with panic disorders.

References