An Electrophysiologic Study of Swallowing-Induced Tachycardia*

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Electrophysiologic studies were performed on a 73-year-old man with swallowing-induced supraventricular tachycardia, in order to define the characteristics of this unique dysrhythmia in this patient. Swallowing reliably provoked an automatic atrial focus type of atrial tachycardia, which usually changed into an atrioventricular nodal reentrant tachycardia when a critical delay in atrioventricular nodal conduction (atrio-His interval ≥ 340 msec) was achieved. The atrioventricular nodal reentrant form of tachycardia did not occur spontaneously. The ease of induction and the duration of the episodes of supraventricular tachycardia were facilitated with the intravenous administration of atropine and ouabain and were decreased with administration of procainamide hydrochloride.

Only eight case reports1-8 of deglutition-induced tachycardia have appeared since Sakai and Mori8 first described the entity in 1926. The proposed theoretic mechanisms range from a vagovagal reflex arc to mechanical stimulation of the left atrium by a transiently distended esophagus. An electrophysiologic study (with pharmacologic manipulations) performed on a patient with deglutition-induced tachycardia forms the basis of this report.

Case Report

A 73-year-old man described a 30-year history of infrequent palpitations, which were documented to be secondary to paroxysms of supraventricular tachycardia and were generally well controlled with therapy with quinidine sulfate. More recently, the tachyarrhythmia was precipitated only by swallowing solids or liquids. The therapy with quinidine sulfate was changed to oral administration of propranolol (40 mg every six hours), with little or no improvement. The cardiac physical findings were a nonejection middiastolic click and a grade 2/6 mid to late systolic apical murmur.

On admission, the chest x-ray film, electrocardiogram, and echocardiogram were within normal limits. Continuous 24-hour ambulatory electrocardiographic monitoring performed prior to admission demonstrated premature atrial contractions with varying coupling intervals between the P wave and the premature atrial contraction and paroxysms of supraventricular tachycardia consistently initiated by swallowing solids or liquids. The results of cardiac fluoroscopic studies with a barium esophagogram were normal. The patient noticed a short period of palpitations five seconds after the

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PAC coupling interval range of 400 to 750 msec initiated the type 1 tachycardia. This tachycardia was further characterized by a "warm-up" period and by an A-A interval of 370 msec. The P waves of type 1 supraventricular tachycardia (P' in Fig 1 and 2) were upright in leads I, aVF, and V; and the sequence of atrial activation was high right atrium (HRA), left atrium (LA), and low right atrium (LRA), with conduction times for HRA-LRA interval of 10 msec and for the HRA-LRA interval of 45 msec (Fig 2). The type 1 supraventricular tachycardia could not be initiated by the placement of atrial premature depolarizations (extrastimuli) and either ended spontaneously after a short salvo of five to ten beats or evolved into type 2 supraventricular tachycardia.

Type 2 supraventricular tachycardia was initiated by type 1 when the A-H interval increased to 340 msec or greater (Fig 2). The A-A interval of type 2 was 400 msec, with a constant A-H interval (360 msec). The sequence of atrial activation of this dysrhythmia was low right atrium to left atrium and high right atrium (conduction interval of 40 msec), with the left atrial and high right atrial deflections occurring at the same time. Type 2 supraventricular tachycardia did not develop spontaneously and was related to swallowing only by evolving from type 1. Placement of atrial premature depolarizations in an A-S 3 range of 380 to 400 msec (with an A-H interval ≥ 340 msec) also initiated type 2. In contrast to type 1 supraventricular tachycardia (which either stopped spontaneously or evolved into type 2), type 2 persisted until it was converted to sinus rhythm with carotid massage, atrial pacing, or the placement of atrial premature depolarizations.

The electrophysiologic studies were repeated after the intravenous administration of each of the following three drugs: atropine (1 mg); propranolol hydrochloride (350 mg); and ouabain (0.5 mg). Administration of atropine dramatically increased the frequency of supraventricular tachycardia (type 1 to type 2) and, in fact, the supraventricular tachycardia occasionally began spontaneously (ie, without swallowing). After the basic cycle length returned to baseline values (45 minutes), propranolol was administered. Within three to five minutes, the frequency of type 1 supraventricular tachycardia (hence, type 2) decreased dramatically; and over the ensuing hour, only one episode of three successive atrial beats of type 1 were noted. Swallowing increasing amounts of liquids did not elicit the supraventricular tachycardia. Administration of ouabain (90 minutes after administration of propranolol) significantly increased the frequency and ease of induction of type 1 supraventricular tachycardia, which evolved into type 2 in most instances.

**DISCUSSION**

The electrophysiologic study demonstrated that this patient's swallowing-induced tachycardia was a composite of two types of supraventricular tachycardia. The mechanism of type 1 supraventricular tachycardia is most compatible with an automatic atrial focus on the basis of (1) a variable coupling interval between the A wave and the premature atrial contractions, (2) the presence of a "warm-up" period of the first two to three beats, and (3) the inability to initiate or terminate the tachycardia with the placement of premature atrial depolarizations. In addition, the atrioventricular nodal echo beats (type 2) depolarized the automatic focus (type 1), causing a reset of the focus (compensatory

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Figure 1. Intracardiac and esophageal electrograms, scalar leads I, aVF, and V of ECG, direct blood pressure recording, and diagram of type 1 supraventricular tachycardia. Tachycardia was initiated by premature atrial contraction occurring 750 msec after previous atrial beat. After three beats of progressively decreasing A-A intervals, A-A interval of the supraventricular tachycardia (paroxysmal atrial tachycardia) becomes 370 msec, HRA, LA, and HBE; Electrograms of high right atrium, left atrium, and His bundle, respectively; H, His bundle; P and P', P waves of sinus rhythm and of type 1 supraventricular tachycardia, respectively; A and A(I), A waves of sinus rhythm and of type 1 supraventricular tachycardia, respectively; LRA, low right atrium; V, ventricle.

Barium cleared the esophagus. Cardiac catheterization demonstrated normal coronary arteries and left ventricle, with mild prolapse of the mitral leaflets.

Electrophysiologic studies were performed using standard techniques. Simultaneous recordings of high and low right atrial and left atrial electrical events, bundle of His and ventricular deflections, three scalar electrocardiographic leads, and a direct measurement of blood pressure (right femoral artery) were obtained throughout the period of study. Times for atrial conduction were slightly prolonged, with a right intra-atrial conduction time of 55 msec and an interatrial conduction time of 75 msec. The atrio-His (A-H) and His-ventricle intervals were normal. The times for sinoatrial conduction and for recovery were within normal limits. The effective atrial refractory period (A-A interval = 800 to 900 msec) was 220 msec. Plots of the atrioventricular nodal conduction time and the refractory period did not demonstrate the presence of dual atrioventricular nodal pathways.

Two types of paroxysmal supraventricular tachycardia were found and will be arbitrarily designated as type 1 and type 2. Type 1 (Fig 1) started 10 to 15 seconds after swallowing 5 ml of liquid or more and occasionally started after dry swallowing. This type did not occur spontaneously. A premature atrial contraction (PAC) occurring in the A-
pause), accounting for the total suppression of the focus during the type 2 supraventricular tachycardia (despite the longer cycle length of the reentrant dysrhythmia). The sequence of atrial activation, the atrial conduction intervals, and the P-wave configuration of type 1 supraventricular tachycardia suggest that the focus was in the high right atrium or high interatrial septum. Exploration with the esophageal catheter during type 1 supraventricular tachycardia did not reveal a left atrial deflection occurring earlier than 10 msec after the high right atrial depolarization. The type 2 supraventricular tachycardia fulfills the criteria for an atrioventricular nodal reentrant mechanism, with induction and termination by a single premature atrial depolarization, initiation only after a critically prolonged A-H interval, a constant and prolonged A-H interval during the supraventricular tachycardia, and a sequence of atrial activation of low right atrium to high right atrium and left atrium.

The potential mechanisms by which deglutition may provoke supraventricular tachycardia have been reviewed in other reports. In contrast to the case reported by Engel and colleagues, mechanical stimulation of the left atrium by transient distention of the esophagus is an unlikely mechanism in our patient for the following reasons: (1) the focus appeared to be located in the high right atrium or interatrial septum (areas usually not adjacent to the esophagus); (2) the left atrium was of normal size, and no atrial indentation of the esophagus was noted during fluoroscopic examination via a barium swallow; (3) the supraventricular tachycardia developed five seconds or more after the barium cleared the esophagus; and (4) the tachyarrhythmia could occasionally be precipitated by a dry swallow and occurred spontaneously after administration of atropine.

A complete vagovagal reflex arc is also an unlikely mechanism in our patient, since administration of atropine dramatically increased the frequency of supraventricular tachycardia, indicating that efferent vagal stimulation was not necessary for the initiation of the tachyarrhythmia and, in fact, may have played an inhibiting role. On the other hand, the importance of the influence of the sympathetic nervous system is also in question because of the failure of our patient's condition (and those in other reports) to respond to administration of varying doses of propranolol.

While it is apparent that the mechanism of deglutition-induced tachycardia remains elusive, it is noteworthy that administration of quinidine and procainamide provides the most successful control of this unique disturbance in rhythm (six of nine patients) in the few reports available, suggesting a common mechanism in these patients. An ectopic atrial focus (probably automatic) easily provoked by "autonomic imbalance," a pharmacologic effect, or mechanical stimulation is a favorable hypothesis. Additional electrophysiologic stud-
ies will have to be performed in patients with deglutition-induced tachycardia in order to determine whether this hypothetic mechanism is correct.

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CLEFT TONGUE AND ULCERATION OF HARD PALATE

Complications of Oral Intubation

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In a prospective study of the complications of endotracheal intubation and tracheotomy, we encountered an unusual complication of oral intubation. This report describes a 32-year-old man who sustained laceration and cleft of the tongue and ulceration of the hard palate as a result of the use of an oral airway in conjunction with oral intubation.

Oral complications of endotracheal intubation are common and include injury to the teeth, lips, gums, tongue, and mucosa.¹ Most of these injuries result from traumatic insertion of the oral endotracheal tube, but some may occur after initial placement of the tube.² Oral airways are widely used to facilitate oral care and to prevent trauma to the soft tissues of the mouth during orotracheal intubation; however, these devices may themselves be hazardous. We report a case of laceration and cleft of the tongue and ulceration of the hard palate from prolonged use of an oral airway, in order to make the reader aware of these potential complications of oral intubation.

CASE REPORT

A 32-year-old man was admitted to Colorado General Hospital, Denver, after sustaining an injury to the brain stem in an automobile accident. On admission, he was comatose and decerebrate and had acute respiratory failure. An oral endotracheal tube (Portex; 9.0 mm in internal diameter) was inserted without trauma, and a curved polyethylene Berman-type oral airway (length, 9.0 cm; width, 2.1 cm; and thickness, 0.8 cm) was placed in the mouth and secured with tape. Decerebrate rigidity with tense contraction of the jaw increased during the first 48 hours of hospitalization.

Five days after admission, brisk oral bleeding developed from a 2.5-cm through-and-through laceration of the tongue, which had been trapped between the lower incisor teeth and the oral airway. A tracheostomy was immediately performed, and the oral airway was left in place to facilitate oral care and to prevent biting of the tongue.

On the 34th day of hospitalization, massive hemorrhage occurred from an erosion of the stoma of the tracheostomy into the inferior thyroid artery. An oral endotracheal tube (Portex; 9.0 mm in internal diameter) was again inserted and left in place, along with an oral airway, for eight days before cricothyroidotomy was performed.

On the 45th day of hospitalization, rigidity of the jaw abated, and a 3.0-cm cleft on the left side of the tip of the tongue, distal to the previous laceration, was discovered (Fig 1A). Pressure necrosis on both sides of the cleft was apparent. The cleft coincided with the position of the oral airway

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