Intrapleural Positioning of Esophagus for Treatment of Swallowing-Induced Arrhythmia*

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Swallowing-induced atrial tachycardia is a rare phenomenon generally assumed to be caused by abnormal parasympathetic reflexes arising in the esophagus or pharynx. We describe a patient with intractable swallow tachycardia. Since certain features of the case suggested a mechanical rather than a reflex mechanism, he was treated by intrapleural repositioning of the esophagus to effect physical separation of esophagus and left atrium. Ten months after an uncomplicated procedure, the patient remains asymptomatic and free of arrhythmia.

Despite the anatomic relationship between left atrium and the esophagus, swallowing-induced tachyarrhythmia is a rare phenomenon. We report here a case of incapacitating, swallowing-induced atrial tachycardia treated successfully by repositioning the mid-portion of the esophagus in the right pleural space. We believe this to be the first such case reported.

Case Report

Because of symptomatic, poorly controlled ventricular arrhythmia and gradually increasing effort fatigue and dyspnea, three years after anterior wall infarction, this 48-year-old man underwent left ventricular aneurysm resection and aortocoronary bypass grafting in April 1976. Except for recurring pain, believed to be of pleural or pericardial origin and requiring intermittent steroid therapy for several months, surgery was uncomplicated. Exercise tolerance improved, and minor palpitations recurred infrequently.

He experienced one episode of near syncope in January 1977, and because of unifocal ventricular premature beats, was given propranolol. This symptom recurred a few months later while the patient was drinking beer, but no arrhythmia was demonstrable subsequently.

In September 1978 he was readmitted with a two-week history of brief episodes of palpitation, dizziness, and near syncope associated with swallowing food or fluids. Monitoring revealed no arrhythmia except in association with swallowing, at which time he had irregular atrial tachycardia lasting from 1 to about 4 sec, with ventricular response rates frequently exceeding 200 beats per minute.

Results of barium swallow, cine-esophagogram, and esophagogastrosopy disclosed no esophageal pathology. Manipulation of the endoscope in the mid-esophagus triggered the identical arrhythmia.

Cine-radiographic study was undertaken, during which the patient swallowed barium-soaked marshmallows (difficult objects for the esophagus to handle). When the marshmallow bolus reached the level of the carina, the tachycardia was readily demonstrated fluoroscopically and could be seen to terminate when the bolus passed the level of the left atrium. Simultaneous right atrial electrogram and surface ECG confirmed an irregular atrial tachycardia with variable conduction to the ventricles. The configurations of the atrial depolarizations were similar to each other and to those associated with sinus beats. Five swallowing maneuvers and five paroxysms of atrial tachycardia were observed. In all, the arrhythmias were similar (Fig 1). In the first, eight of 11 atrial depolarizations were conducted to the ventricles. In the second, third, and fourth episodes, 15, 16, and 13 atrial depolarizations conducted to the ventricles in 9, 11, and 8 cases, respectively. In the fifth episode, 2:1 AV block was present. In all cases tachycardia ended with a nonconducted atrial premature beat followed by longer P-P interval than those associated with sinus beats, and then sinus rhythm ensued.

Digoxin therapy was initiated and at a dose of 0.5 mg daily; the patient had complete remission of symptoms and abolition of swallowing-induced tachycardia for several months, but in March 1979 it occurred with increasing frequency, and episodes of near syncope precipitated readmission. His arrhythmia appeared precisely similar to previously, but was occurring on a therapeutic dose of digoxin sufficient to produce a blood level of 2.3 ng/ml. In order to increase the degree of A-V block, propranolol was added to the regimen without effect. Attempts to suppress the ectopic focus with quinidine were unsuccessful because of gastrointestinal intolerance to the drug. The patient was already receiving procainamide.

Since these methods had been unsuccessful, we decided to attempt a surgical solution. With the patient in the left lateral decubitus position, a right posterolateral thoracotomy was performed through the sixth interspace. The mediastinal pleura was incised longitudinally over the esophagus. The azygos vein was ligated and divided, and the entire esophagus mobilized and raised from its bed, leaving some vascular pedicles intact. The mediastinal pleura was then reapproximated medial to the esophagus, forming a hammock for the intrapleural esophagus. The esophagus was thus displaced to the right and prevented from returning to its former position.

![Figure 1. Simultaneous surface ECG (above) and right atrial electrogram (below). Paroxysm of irregular atrial tachycardia associated with swallowing. Complexes marked with arrows are blocked P waves. Time line markings are 1 sec apart.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21199/ on 06/21/2017)
by the intact mediastinal pleura. A chest tube was inserted and the wound closed. No operative arrhythmia was encountered.

Except for incisional discomfort, the postoperative course was uncomplicated. Ten months later the patient remains free of symptoms and has no demonstrable arrhythmia. When the transient postoperative dysphagia subsided, a repeat of the swallow of barium-soaked marshmallow demonstrated the new position of the esophagus without obstruction or kinking (Fig 2 and 3). More importantly, despite several attempts, no arrhythmia could be provoked.

**DISCUSSION**

Swallowing-induced atrial tachycardia is an uncommon entity first described by Sakai1 in 1926. Most of the cases described have been associated with esophageal disease or dysfunction,2-4 and most have had no clinical evidence of underlying cardiac disease.2-5

The cause of these tachyarrhythmias has generally been postulated to be a reflex originating in the pharynx or esophagus; however, proof of this reflex mechanism is still lacking. The apparent coincidence, in our case, of a nonreciprocating atrial tachycardia triggered by a bolus of food just as it reached the level where the left atrium and esophagus are virtually in contact suggests another possible cause. We believe that local mechanical stimulation of the left atrium might account for this phenomenon, although the reasons for sensitivity of the left atrium to this type of stimulation are not clear. Endocardial mapping studies were not done, but might be of interest in confirming the left atrial origin of the arrhythmia. It is interesting that Cohen et al6 likewise described a patient in whom the arrhythmia could be triggered by manipulating, either by esophageal balloon or by swallowed marshmallow, the segment of esophagus just below the level of the carina.

The fact that an operation that physically separated left atrium and esophagus, with care to preserve vascular and nerve supply to the esophagus, appears to have abolished the arrhythmia also favors our hypothesis. Kalloor et al3 describe such a patient treated by circular myotomy of the esophagus in an attempt to produce denervation. Although they describe the patient as improved, he was still troubled by occasional palpitations related to swallowing despite a vigorous attempt at denervation of the esophagus.

Unlike the described cases of circular esophageal myotomy, dysphagia was not a problem after the first three to four postoperative weeks. The success of surgery in this case suggests that this method of treatment be considered in patients with intractable swallowing-induced atrial arrhythmia when suppression by anti-arrhythmic agents and pharmacologic blockade of the A-V node has been unsuccessful in alleviating symptoms.

**REFERENCES**

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