A 56-year-old man with gastroesophageal reflux disease had severe but transient palpitations during swallowing with every meal. He continued to have symptoms despite cessation of alcohol and caffeine consumption. Although he was occasionally able to reproduce his symptoms with dry swallowing, they were more prominent after swallowing solid food or carbonated beverages. He reported no palpitations unrelated to deglutition. A 24-hour Holter monitor revealed numerous runs of atrial tachycardia (AT) during meals with varying atrial cycle lengths between 320–500 ms (Figure 1). An echocardiogram was normal.

He was initially placed on β-blockers, which had minimal therapeutic effect and were not tolerated due to fatigue. He was then placed on flecainide, which reduced but did not abolish the arrhythmias. Because of his daily symptoms despite antiarrhythmic medications, he underwent an invasive electrophysiology study. During electrophysiology study, an AT with varying cycle lengths between 300–510 ms was reproducibly induced with swallowing and was found to arise from the left atrium. A circular mapping catheter was placed in the right superior pulmonary vein (Figure 2). During direct intracardiac echocardiographic visualization of the esophagus, AT was induced during swallowing of a carbonated beverage and was coincident with early electric activation from the circular mapping catheter (online-only Data Supplement Video, Figure 3A and 3B, and Figure 4). The esophagus was noted to course close to the right pulmonary veins (Figure 5). The right superior pulmonary vein was circumferentially isolated with radiofrequency ablation (Figure 5), confirmed by pulmonary vein entrance and exit block. No vagal response was elicited during right superior pulmonary vein isolation.

After ablation, the right superior pulmonary vein remained quiescent during deglutition and no atrial arrhythmias were inducible with swallowing or programmed stimulation. After more than 12 months of follow-up, the patient remains completely free of palpitations during swallowing. A follow-up 24-hour Holter monitor revealed sinus rhythm with no atrial premature contractions or AT.

Discussion

Deglutition-induced AT is rare, with a reported prevalence of 0.6% among patients presenting with paroxysmal atrial arrhythmias. The arrhythmia occurs most often in men without structural heart disease.¹ Our case illustrates the utility of intracardiac echocardiography in elucidating the temporal sequence and mechanism of deglutition-induced AT. Our patient swallowed a carbonated beverage to enhance visualization of the esophageal lumen. We found that AT was reproducibly induced after the liquid had reached the esophagus at the level of the left atrium but before the onset of esophageal distension by secondary peristalsis.
Several mechanisms for deglutition-induced AT have been proposed, which include induction of vasovagal and adrenergic reflexes by the esophagus and mechanical stimulation of the left atrium by a distended esophagus. The former hypothesis is supported in our case because AT was initiated in our patient during primary peristalsis before the occurrence of esophageal distension. Primary peristalsis is mediated by centrally activated firing of vagal efferent fibers; activation of vagal reflexes may in turn lead to pulmonary vein firing. A prior study using esophageal balloon dilation that showed that direct mechanical stimulation of the left atrium did not consistently result in induction of atrial arrhythmias.

Medical therapy with agents such as β-blockers, calcium channel blockers, and antiarrhythmic drugs can be used for debilitating symptoms of deglutition-induced AT but may have limited efficacy. Alternatively, catheter ablation of the arrhythmogenic source, presumably the right superior vein due to vagally mediated AT in our study, can offer a curative therapy.

Disclosures

None.

References


Key Words: atrial tachycardia □ esophagus □ intracardiac echocardiography
Deglutition-Induced Atrial Tachycardia: Direct Visualization by Intracardiac Echocardiography
James E. Ip, Bruce B. Lerman and Jim W. Cheung

Circ Arrhythm Electrophysiol. 2012;5:e36-e37
doi: 10.1161/CIRCEP.111.969626

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circep.ahajournals.org/content/5/2/e36

Data Supplement (unedited) at:
http://circep.ahajournals.org/content/suppl/2012/04/18/5.2.e36.DC1

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation: Arrhythmia and Electrophysiology can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation: Arrhythmia and Electrophysiology is online at:
http://circep.ahajournals.org/subscriptions/
SUPPLEMENTAL MATERIAL

Online Video Legend.

Intracardiac echocardiography of left atrium and esophagus during deglutition. Electrograms from the lateral right atrium are listed as poles 20B 19-20 (high right atrium), 17-18, 15-16, 13-14 and 11-12 (low right atrium). Electrograms from the coronary sinus are listed as poles 20B 9-10 (proximal), 7-8, 5-6, 3-4, and 1-2 (distal). At the start of the video, the position of esophagus posterior to the left atrium (LA) is noted during sinus rhythm. The video is then slowed down to half-speed as patient swallows a carbonated beverage. During deglutition, acoustic artifact from the soda bubbles is seen within the esophagus which is quickly followed by the onset of atrial tachycardia. Peristaltic contraction is then seen following termination of the atrial tachycardia.