

## The Novel Use of an Esophageal Deviator System to Displace the Phrenic Nerve during Ablation of a Focal Right Atrial Tachycardia

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### Abstract

Atrial tachycardia is a sub-type of supraventricular tachycardia (SVT) that can be seen in patients with both structurally normal and abnormal hearts. In contrast to other types of SVTs, an atrial tachycardia does not require an accessory pathway or the atrioventricular node for maintenance.

**Keywords:** atrial tachycardia; esophageal deviator; phrenic nerve

### Introduction

Atrial tachycardia is a sub-type of supraventricular tachycardia (SVT) that can be seen in patients with both structurally normal and abnormal hearts. In contrast to other types of SVTs, an atrial tachycardia does not require an accessory pathway or the atrioventricular node for maintenance. It typically arises from any ectopic site within atrial myocardium and typically the atrial rate ranges between 150-250 beats per minute; in addition P waves may appear either similar or dissimilar to sinus P wave morphology.

Most focal atrial tachycardias arise from within the right atrium, and more specifically they are most commonly observed along the superior to mid portion of the crista terminalis. Less commonly they arise from structures of the left atrium that include the left atrial appendage, septal portion, or the pulmonary veins. Majority of focal atrial tachycardias are amenable to catheter ablation for long term cure and additionally, during ablation of anterior right atrial foci, care must be taken to avoid causing injury to the phrenic nerve. The case below highlights the novel use of an esophageal deviator to shift the phrenic nerve away from a site of ablation during atrial tachycardia.

### Case

The patient is a 59 year old female with past medical history of migraine disorder, vitamin D deficiency, and paroxysmal SVT who presented as an outpatient with complaints of severe intermittent palpitations associated with dizziness and chest discomfort. A 12 lead ECG showed a narrow-complex tachycardia where the differential diagnosis included focal atrial tachycardia versus AVNRT with baseline first degree AV delay. She was not keen on taking medications and subsequently presented for an elective EP study with attempted induction and possible catheter ablation for SVT.

Using the modified Seldinger technique, two (6F, 8F) sheaths were placed percutaneously in the right femoral vein, and two (7F, 6F) sheaths in the

left femoral vein. Quadripolar catheters were advanced to the locations of the high right atrium (HRA), HIS bundle, and RV apex under fluoroscopic guidance. A deflectable decapolar catheter was positioned in the coronary sinus.

Intracardiac electrograms were recorded from the right atrium, right ventricle and coronary sinus. Pacing maneuvers were performed from the right atrium, right ventricle and coronary sinus. Isoproterenol was used for induction of SVT and maximum dose was 2mcg/minute. During isoproterenol infusion the patient's heart rate increased >20% from baseline.

AV nodal conduction was normal with AV nodal Wenckebach cycle length= 470msec. Right atrial pacing revealed normal AV nodal physiology with no evidence of dual AV nodal pathways. No echo beats were noted. Antegrade AV nodal ERP = 600msec/430msec. RV pacing showed that VA conduction was not present when assessed at baseline and was not present during isoproterenol.

With programmed stimulation using single extra-stimuli from the HRA and CS positions during isoproterenol infusion (450/380ms pacing CL), a sustained narrow-complex, long-RP (VA time >70msec) tachycardia was induced. Features included dissociation of ventricular electrograms from atrial electrograms and a VAAV response to ventricular overdrive pacing that suggested a diagnosis of atrial tachycardia.

The 8F right femoral vein sheath was exchanged for an 8.5F long SRO sheath over a wire using fluoroscopic guidance and advanced to the RA. A 20-pole Pentaray mapping catheter was inserted through the SRO sheath to the RA. The Biosense Webster CARTO3 3D electroanatomical mapping system was used to define right atrial geometry. A high-density electroanatomic activation map of the RA was created (<70% of tachycardia CL) with earliest activation at the superior-posterior RA, suggesting a focal AT exit site at this location. The Pentaray mapping

catheter was exchanged for a 3.5mm deflectable Thermocool Smarttouch irrigated ablation catheter that was advanced to the site of earliest activation.

Prior to ablation, high amplitude (20mA @ 2msec) pacing was performed to assess for phrenic nerve/diaphragmatic capture, and revealed extensive phrenic nerve capture overlying the site of intended ablation. Given the nearly exact proximity of the phrenic course over that planned ablation site, a decision was made to abort the ablation procedure due to high risk of phrenic nerve injury/paralysis with ablation at this site. The patient was then planned for a repeat EP study in the future with consideration to use cryoablation with an epicardial balloon to avoid phrenic nerve injury.

The patient then presented again to the EP lab approximately 3 months later for a redo EP study and possible catheter ablation. Using the modified Seldinger technique, an 8F and 7F sheath were inserted percutaneously into the right femoral vein. A decapolar catheter was advanced to the coronary sinus and a thermocool force sensing irrigated ablation catheter was advanced to the right atrium.

Isoproterenol was started and burst pacing maneuvers including premature extrastimuli were performed to induce atrial tachycardia that showed 1:1 AV conduction and with a cycle length of 380 - 400 msec. Activation pattern occurred from proximal to distal CS pole and the tachycardia would terminate spontaneously each time with a ventricular electrogram. Ventricular overdrive pacing showed a VAAV response similar to the prior EP study.

The ablation catheter was advanced to the high posterior wall and high output pacing was used to perform mapping of the course of the phrenic nerve. During tachycardia activation mapping was performed and showed the earliest atrial activation in the high posterior wall that was unchanged from the prior EP study on June 2018 (pre-CS time of -65 msec). That focal area showed fractionated atrial electrogram and was directly over the area with phrenic nerve stimulation.

A 6F sheath was then inserted percutaneously into the left femoral vein and a diagnostic catheter was advanced to the SVC-RA junction where phrenic nerve stimulation was able to be performed with high output pacing. The 8F sheath was exchanged for an SR0 sheath and a Pentaray mapping catheter was advanced to the right atrium. Another activation map was performed during atrial tachycardia and showed earliest activation again at the high posterior wall.

Prior to the consideration of the use of an epicardial balloon, the decision was made to use an Esosure esophageal deviator system. It was placed within the esophagus using fluoroscopic guidance and then manipulated to shift the esophagus to an extreme leftward direction

High output pacing was again performed and this time there was no evidence of phrenic stimulation using both manual palpation of the diaphragm and fluoroscopy. Radiofrequency ablation lesions were then delivered to the focal area of earliest activation. Multiple ablation lesions were applied during sinus rhythm and also over adjacent areas where fractionated electrograms were recorded. Subsequently no further arrhythmia was inducible with pacing maneuvers while in isoproterenol.

## Discussion

The majority of focal atrial tachycardias arise from within the right atrium, and very commonly from the area of the crista terminalis. As a result during catheter ablation of a focal exit site, care must be taken to avoid causing local injury to the phrenic nerve that can result in complications such as diaphragmatic paralysis.

After activation mapping of a focal right atrial tachycardia is completed, high output pacing can then be performed over the area of intended ablation to assess for phrenic nerve capture (using fluoroscopy and manual palpation over the diaphragm). In most cases radiofrequency ablation may have to be deferred if it can cause local injury to the phrenic nerve and other modalities may need to be explored (i.e. cryoablation, epicardial cryoballoon, etc.). However in this case we illustrated the use of an Esosure esophageal deviator system in shifting the esophagus to an extreme leftward direction that also displaced the phrenic nerve from a planned ablation site in the right atrium.

The phrenic nerve originates from the cervical spinal roots (C3, C4, and C5) and the right phrenic nerve enters the thorax via the superior thoracic aperture. It is located usually posterior to the right subclavian vein and courses adjacent to the esophagus. Distally it overlies the pericardium of the right atrium and then innervates the inferior surface of the diaphragm. Thus manipulation of the esophagus can also result in concomitant displacement of the right phrenic nerve.

## Conclusion

Focal right atrial tachycardias very commonly arise from the region of the crista terminalis and are amenable to long term cure via catheter ablation. However a potentially serious complication involves local damage to the phrenic nerve during ablation especially over anterior or lateral sites. We present a case report that illustrates the novel use of an esophageal deviator in displacing the phrenic nerve over a planned ablation site. Following ablation there was no evidence of phrenic nerve trauma and the patient remained asymptomatic over long term follow-up.

## Disclosures:

Asad Mohammad D.O.: None

Devendra Mehta M.D. Ph.D.: None

Jay Doshi M.D.: None

## References:

1. Miyazaki, S., Iesaka, Y. (2012) Phrenic Nerve and Esophageal Injury during Catheter Ablation of Atrial Fibrillation. *J Atr Fibrillation*. Feb-Mar; 4(5); 485.
2. Anatomic Relations Between the Esophagus and Left Atrium and Relevance for Ablation of Atrial Fibrillation. Damian Sánchez-Quintana, MD, PhD. *Et. Al. Circulation*. 2005;112:1400-1405.
3. Mechanical displacement of the esophagus in patients undergoing left atrial ablation of atrial fibrillation. Aman Chugh, MD, Fred Morady, MD. et al. *Uni. of Michigan. Heart Rhythm Journal*. Heart Rhythm Journal Vol. 6, Issue 3, Pgs. 319-322 (March 2009)



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