INNOVATIONS™ in Cardiac Rhythm Management

LETTER FROM THE EDITOR IN CHIEF

Dear Readers,

Within this month's letter, I would like to provide commentary on the devastating complications associated with catheter ablation of atrial fibrillation (AF), namely that of an esophageal injury. You will find an excellent review of strategies to prevent esophageal injury within AF ablation procedures by Dr Buch and colleagues from the UCLA Cardiac Arrhythmia Center within the *Innovative Techniques* section of this issue. I highly recommend this manuscript to all of our readers, as this is a critically important topic to every provider that cares for AF patients.

Upon reading this article, I could not help but to reflect on my own experience with an atrioesophageal fistula following AF ablation in 2004. In early 2004, Drs. Pappone and Morady reported the first series of two patients that developed an atrioesophageal fistula following AF ablation.[1] At the time, we really did not have a strong understanding of the pathogenesis associated with this condition, or for that matter, how to prevent it from occurring. It is a shame that we had not fully learned from surgeons who had experienced this complication from radiofrequency MAZE procedures years before 2004. I have also observed that, in general, physicians that are most concerned with preventing this complication are those who have managed an esophageal injury complication in the past.

Reflecting back to early 2004, I felt I must have been doing something right. I had performed AF ablation for more than five years without the slightest hint of esophageal injury. This was until July of 2004, when during that month I had two esophageal injuries occur following AF ablation procedures. Both cases presented to their local emergency rooms in August 2004 with severe chest discomfort, dysphagia and fever.

In July of 2004, we (the AF ablation community) did not employ the same esophageal protection strategies that we do today. At that time, our center was using the eight mm tip ablation catheter and ablating at 70 W, with a maximum temperature of 55 degrees for 30 seconds on the posterior wall. As I look back on this now, I realize that we were fortunate not to have had more esophageal injury complications.

Unfortunately, the first patient who presented with severe chest discomfort, dysphagia, and fever two weeks following her AF ablation procedure did not do well. Despite surgery to repair an atrioesophageal fistula and many other interventions, she ultimately passed away after care was withdrawn following massive and overwhelming sepsis. Fortunately, our second patient that presented just one week later was treated earlier in the disease process. Temporary esophageal stenting, broad spectrum antibiotics (including anti-fungal agents) and total parental nutrition were well received, and the patient eventually had a complete recovery with no long-term complications. This was also the first reported case study that utilized temporary esophageal stenting to treat an esophageal perforation following AF ablation.[2]

Our approach has undergone considerable change since August of 2004. The most notable change is the energy delivery to the posterior wall, which has been decreased dramatically. While I still ablate at 50 W with the irrigated tip ablation catheter, this is conducted in the temperature control mode, limiting to just 42 degrees. Thus, 50 W is rarely achieved on the posterior wall where catheter contact is generally very good. I believe the most important change is that I only ablate for two to four seconds on the posterior wall. The energy delivered is a function of the power multiplied by the time, this ablation strategy (100–200 Watt seconds of total energy delivery), from a safety standpoint, delivers much less energy than the Heart Rhythm Society AF Ablation expert consensus document, which recommended 25–35 W for 10–20 seconds (total energy delivered: 250 to 700 Watt seconds).[3]

The rationale behind this approach is that thermal injury to the esophagus occurs from conductive heating, which is a time dependent process that transfers energy deep enough to injure the esophagus. Thus, by utilizing high power radiofrequency applications for a very short period of time, we can heat the local myocardial tissue enough to adequately ablate the local myocardial tissue and not apply the energy long enough to allow conducting heating deeper to the esophagus. In addition, as it takes two to three minutes for the esophagus to cool following each

radiofrequency application, we do not ablate in the same area on the posterior wall until two to three minutes had passed since the previous application.

From the fall of 2004, until January of 2012 I thought we had completely figured out the proper strategy for avoiding esophageal complications. In addition to utilizing this ablation strategy of minimizing energy delivery to the posterior wall, as described above, each of our patients is placed on high dose omeprazole and sucralfate suspension therapy for one month following their AF ablation procedure. During this seven and a half year span I had personally performed more than 2,000 AF ablation procedures without esophageal injury. In fact, we had not even witnessed esophageal mucosal erythema in the patients who had an endoscopy performed for various reasons immediately following their ablation procedure. However, in January of 2012, I was humbled by an esophageal ulceration following AF ablation.

The patient was a 58 year old woman with highly symptomatic paroxysmal AF refractory to flecainide anti-arrhythmic drug therapy. She also had a past medical history significant for obesity, gastric bypass surgery, a moderately sized hiatal hernia, and a history of peptic ulcer disease with prior esophageal ulceration from this condition. She underwent her first AF ablation procedure on September 9, 2011. Due to symptomatic AF recurrences despite antiarrhythmic drug therapy, she underwent a second AF ablation procedure on December 29, 2011. As with all of our AF ablation cases, energy delivery to the posterior was much lower than what is recommended with the HRS AF Ablation Expert Consensus Statement.[3] The esophagus was mapped, and there was no rise in the esophageal temperature at any time during the case. As with all of our patients, she was discharged home on high dose omeprazole and sucralfate suspension for one month. She did well until she had presented to the hospital with progressive severe substernal chest discomfort, dysphagia, nausea, and vomiting.

Even though I was not on call the Sunday that she was admitted to the hospital, the fear of a potential esophageal injury kept me in the facility for most of the day. Fortunately, a stat CT scan with oral contrast showed no esophageal perforation, no mediastinal extravasation of contrast, and no esophageal wall thickening. The study did show that she had a moderately sized hiatal hernia and prior gastric bypass surgery. I spent a good amount of time discussing the case with our radiologist, both before and after the scan, understanding that our radiology colleagues may miss the subtle findings of an esophageal injury following ablation. Relived that the CT scan did not show any esophageal injury, I next contacted my gasteroenterology (GI) colleague to arrange for an emergent endoscopy. After educating my GI colleague on the potentially devastating complication of air insufflation with an endoscopy if an atrioesophageal fistula is present, an endoscopy was completed without air insufflations, which showed a 0.5 by 0.3 cm ulcer on the anterior portion of the esophagus immediately adjacent to the left atrium.

I then called my Thoracic Surgery colleague and asked him to see the patient as soon as possible. Both my GI and Thoracic Surgery colleagues recommended continuation of a proton pump inhibitor and sucralfate suspension, nothing by mouth with total parental nutrition, and broad spectrum antibiotics including an anti-fungal agent. Fortunately, four days later the ulcer was completely healed and her symptoms had nearly resolved.

This case was very instructional, considering the fact that there was delivery of far less energy to the posterior wall than what is recommended by the HRS AF Ablation Expert Consensus Statement, and without any esophageal temperature rise during the case, esophageal injuries can still occur. It should be emphasized that we have since shown, especially with a hiatal hernia present, any ablation on the posterior wall of the left atrium, regardless of where the esophagus is ''mapped,'' could potentially result in an esophageal injury.[4]

So how has this recent case affected our approach to ablation of AF? Well, our approach is basically the same, but we have become even more vigilant than before. Our approach to minimize the risk of esophageal injury following AF ablation is listed below:

1. Limit any posterior wall radiofrequency application to a maximum energy delivery of 200 Watt seconds (Energy = Power × Time).
2. Wait two to three minutes before ablating again in the same posterior wall area.
3. Any posterior wall location, regardless of esophageal mapping, could be at risk of injuring the esophagus.
4. Excessive catheter contact on the posterior wall significantly increases energy delivery.
5. Treat all patients with high dose omeprazole and sucralfate suspension for one month following ablation.
6. Closely follow all patients for the first month following ablation for possible esophageal injury.

I hope that you will enjoy this month's issue of the Journal, and that we can continue to be an important aspect of your ongoing education. As always, it is great to hear from you. Please feel free to email me at any time with your thoughts and suggestions.

Sincerely,

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