

Esophageal Heating Is Not Limited to Left Atrial Ablation

W. Kevin Tsai, MD; Jacob Koruth, MD; Vivek Y. Reddy, MD

A 36-year-old man with a history of cardiac arrest status post dual-chamber implantable cardioverter-defibrillator underwent pulmonary vein (PV) isolation for paroxysmal atrial fibrillation (AF). During the first procedure, esophageal temperature rises ($\geq 38.5^{\circ}\text{C}$) were noted during posterior left atrial radiofrequency ablation (red dots; Figure 1A), but PV isolation was eventually achieved. However, the patient developed recurrent AF and underwent a redo procedure under general anesthesia. Two transeptal punctures were performed under fluoroscopy and intracardiac echo (AcuNav; Siemens, Mountain View, CA) guidance. A long 8-Fr (SL-1; St Jude Medical, St Paul, MN) and a deflectable sheath (Agilis; St Jude Medical) were advanced to the left atrium (LA). In both procedures, a multisensor esophageal temperature probe (Circa S-Cath; Circa Scientific, Park City, UT) was inserted into the esophagus to the level of the PVs and used to record the intraluminal esophageal temperature continuously. All 4 PVs were found to be persistently isolated. The level of PV isolation was extended to include the posterior wall using an externally irrigated 3.5-mm tip quadripolar ablation catheter (Thermocool; Biosense Webster Inc, Diamond Bar, CA). Esophageal temperature rises were seen again during ablation of roof and posterior lines (red dots; Figure 1D). Burst atrial pacing–induced sustained AF and subsequent electric cardioversion were followed by premature atrial complexes that triggered AF (Figure 1B). These were mapped to the posterior right atrial (RA) wall near the inferior vena cava. During ablation (25 W) in this region (Figure 1C), multiple esophageal temperature rises $>38.5^{\circ}\text{C}$ were noted. The trigger disappeared with ablation, and nothing further could be induced even with isoproterenol infusion (20 $\mu\text{g}/\text{min}$). The patient had no recurrent AF off of antiarrhythmic drugs at 6 months of follow-up.

Discussion

Atrioesophageal fistula is a feared complication of AF ablation and has been reported after ablation of the posterior LA.^{1,2} Despite its low incidence (0.03–0.1%), this complication can be devastating, with a resultant mortality $>75\%$.³ In addition to limiting the power and duration of radiofrequency applications on posterior LA, real-time luminal esophageal temperature (LET) monitoring with a probe has been shown to minimize the risk of esophageal injury when using a strategy of cessation of radiofrequency ablation once a prespecified rise in LET is observed.⁴ Although esophageal temperature rises are of

common occurrence during posterior LA ablation, LET monitoring is rarely performed when ablating the posterior RA wall. As demonstrated fortuitously in this case, significant esophageal temperature ($\approx 39^{\circ}\text{C}$) rises can be seen during ablation of posterior RA. Esophageal injury was not assessed by endoscopy in this patient; however, the potential for thermal injury to the esophagus in the setting of prolonged ablation exists, especially when one is not monitoring the esophageal temperatures. Although the actual risk of such injury culminating in atrioesophageal fistula formation is difficult to estimate, it can add to injury already created during posterior LA ablation. Figures 1D, 1E, and 2 show the anatomic relationships between the left and right atria, ablation lesions, and the esophagus: 11 mm between the ablation site in the RA and the lateral wall of the esophagus. It is also worth noting that the multisensor esophageal temperature probe used in this case provided additional spatial sampling. Given the proximity of the RA ablation site and the esophagus in combination with increased spatial sampling, this can explain the detection of esophageal temperature rise during ablation of the RA, which may otherwise be missed by using a traditional single-sensor probe. The incidence of esophageal temperature rise during posterior RA ablation needs to be prospectively determined, and LET monitoring during posterior RA ablation should be considered.

Disclosures

None.

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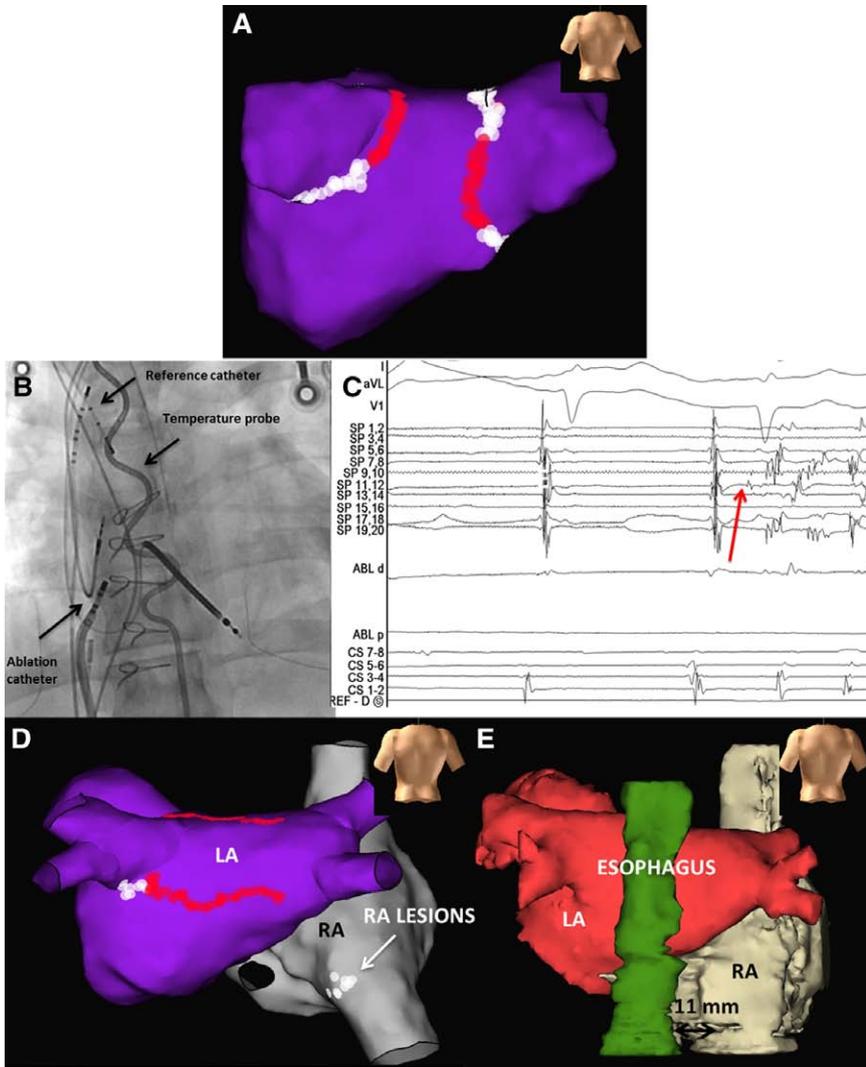


Figure 1. **A**, Initial pulmonary vein (PV) isolation with esophageal temperature rises (red lesions) seen on posterior wall. **B**, Fluoroscopic (anteroposterior) view of ablation catheter at site of triggering atrial premature complexes, with respect to the esophageal temperature probe. **C**, Intracardiac electrogram of atrial fibrillation trigger demonstrated on multipolar mapping catheter (spiral [SP] 11, 12). **D**, 3-Dimensional map of right atrium (RA), left atrium (LA), and ablation lesions (posterioranterior view). Extension of PV isolation with posterior wall isolation (esophageal temperature rise at red lesions). **E**, Relationships between RA, LA, and the esophagus (PA view) of CT reconstruction.

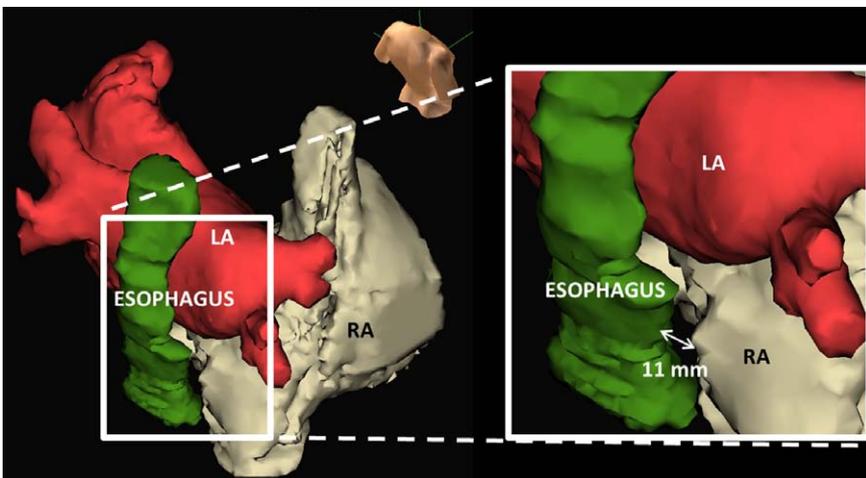


Figure 2. Relationships between right atrium (RA), left atrium, and the esophagus (right lateral view with posterior cranial angulation) of CT reconstruction with distance between the ablation site of the RA and esophagus noted.

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