

Limitations of Esophageal Temperature-Monitoring to Prevent Esophageal Injury During Atrial Fibrillation Ablation

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A number of complications have been associated with ablation of atrial fibrillation (AF), including arterial thrombo-embolism, pulmonary vein stenosis, phrenic nerve injury, and pericardial tamponade.¹⁻⁴ Esophageal injury, manifested as esophageal perforation or left atrial-esophageal fistula, has been reported after catheter or surgical ablation of AF using radiofrequency (RF) current⁵⁻⁸ and catheter ablation using high-intensity focused ultrasound (HIFU).⁹ Left atrial-esophageal fistula usually is associated with a very high morbidity and mortality, including air embolism and sepsis.

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Esophageal injury during RF ablation in the left atrium is thought to be thermal injury.¹⁰⁻¹² In this issue of *Circulation: Arrhythmia and Electrophysiology*, Singh et al¹³ sought to determine whether the risk of esophageal injury would be reduced by measuring the luminal esophageal temperature (LET) during ablation and maintaining the LET below 38.5°C. The LET was measured using a 9Fr flexible temperature probe (with a single thermocouple) in the esophagus. The temperature probe was maneuvered in cranial-caudal direction to position the thermocouple close to the ablation catheter tip in the left atrium.

We agree in principal with the authors' conclusion that esophageal temperature-monitoring may reduce the risk of esophageal injury during AF ablation. An esophageal ulcer was observed by endoscopy 1 to 3 days postablation in 4 of 67 (6%) patients with LET-monitoring (and discontinuing RF application at LET \geq 38.5°C) compared to 5 of 14 (36%) patients without LET-monitoring. Importantly, in patients with LET-monitoring, this study showed no significant difference between patients with and without an esophageal ulcer and the maximum LET during ablation or the number of RF applications producing an increase in LET to \geq 38.5°C. This study did not identify a safety LET cut-off, below which an esophageal ulcer would not occur. There was also no

relationship between the average RF power and the maximum LET.

The lack of relationship between the LET and the occurrence of esophageal ulceration in this study may relate to the inability of currently available probes (a single thermocouple within the esophagus) to locate and measure the highest esophageal temperature during left atrial ablation. This problem was identified in a canine model of esophageal injury where RF, HIFU, and cryo-ablation were performed in the left atrium adjacent to the esophagus.^{12,14,15} An air-filled balloon (20 to 25 mm diameter) was positioned in the esophagus to move the esophagus close to the posterior left atrium. Temperature was measured along the esophageal balloon by 7 thermocouples (2 mm separation) facing the left atrium and the ablation catheter. Esophageal ulceration occurred consistently when the maximum LET was \geq 49.6°C for RF energy and HIFU energy.^{12,14} Lower LET was not associated with ulceration. During cryo-ablation, esophageal ulceration occurred consistently when the minimum LET was \leq 1.3°C.¹⁵ Esophageal ulceration was associated with transmural atrial and esophageal necrosis with all energy sources. The size (area) of esophageal ulceration was significantly related to the maximum LET for RF energy and HIFU and the minimum LET for cryo-ablation. These data support thermal injury as a mechanism of esophageal injury (ulceration) during AF ablation.

In these canine studies, there was a steep gradient of LET, such that the highest LET (RF and HIFU) or lowest LET (cryo-ablation) was recorded in only a very small area. The small area of highest (or lowest) LET would usually be missed by a single thermocouple as used in the study by Singh et al,¹³ especially because the thermocouple is not likely to be located against the anterior wall of esophagus, facing the left atrial ablation site. This probably explains the lack of the relationship between LET and esophageal injury in their study.

An interesting observation in their study was the difference in the use of general anesthesia between the groups with and without LET-monitoring (13% versus 43%). The authors did not mention whether general anesthesia was associated with a higher incidence of esophageal ulceration, but this might be suspected because there was a higher incidence of both anesthesia use and esophageal ulceration in the group without LET-monitoring. In our experience, there is usually very little movement of the esophagus during general anesthesia in the presence of a paralytic agent. Esophageal movement is greater under conscious sedation and the esophagus may contract in response to heat. In addition, an RF application

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close to the esophagus may be discontinued early because of esophageal pain in the nonanesthetized patient. The esophageal movement or pain response may lead to a lower risk of esophageal injury in patients without general anesthesia. We found a higher incidence (38%) of asymptomatic esophageal ulceration by endoscopy 1 day postablation in patients who underwent AF ablation using general anesthesia with a paralytic agent (vecuronium),¹⁶ despite limiting RF applications to 15 to 25 watts for 30 seconds in areas close to the esophagus (<1.5 cm) and terminating the RF application immediately for LET increases of just 0.2°C from baseline (eg, 37.0°C to 37.2°C). LET was measured using an esophageal temperature probe with a single thermocouple similar to the study of Singh et al.¹³ As in their study, we found no significant difference in the maximum LET or the number of RF applications associated with LET increase ($\geq 0.2^\circ\text{C}$) between patients with and without esophageal ulceration. There was also no relationship between the maximum LET and the size of the esophageal ulcer,¹⁶ because we were unable to measure the true highest LET.

Singh et al¹³ found that 34% of RF applications producing LET $\geq 38.5^\circ\text{C}$ were at a power of ≤ 20 watts, and 8% were at a power of ≤ 10 watts. The increase in LET at low RF power may be related to greater electrode-tissue contact force.¹³ We found in the canine model that LET and the size of esophageal ulcer increase significantly with increasing contact force at constant RF power.¹⁴ Contact force appears to be a major determinant of lesion formation. Large lesions are produced at high contact force and lower RF power.

Although the clinical studies are less definitive, the animal studies strongly support a role for measuring esophageal temperature to prevent transmural esophageal injury during AF ablation. However, to be effective, it would be necessary to measure the true highest (or lowest with cryo-ablation) LET. A thermocouple or thermistor would have to be positioned against the anterior esophageal wall, directly opposite the ablation site in the left atrium for each RF application. One approach would be to place multiple closely spaced temperature sensors on an esophageal balloon.¹² The inflated balloon would position the temperature sensors appropriately. However, the inflated balloon would also push the esophageal wall against the left atrium. The shorter distance and increased contact force might increase the risk of esophageal injury.¹⁴ Some investigators are exploring the use of cooling the esophageal balloon to protect against thermal injury.^{17–19} Noncontact temperature probes, which would not compress the esophagus against the left atrium, are also under development.

Although asymptomatic esophageal ulcers (transmural esophageal necrosis) occur frequently,^{13,16} the catastrophic development of a left atrial-esophageal fistula is relatively infrequent and usually present 2 to 4 weeks after ablation. In the canine model, progression of esophageal ulcer size and the development of left atrial-esophageal fistulae were associated with reflux esophagitis and relaxation of the gastroesophageal sphincter, which might be due to damage to the peri-esophageal vagal plexus during ablation.¹² These observations support the authors' use of a proton pump inhibitor to facilitate ulcer healing.¹³ We favor the use of a proton pump

inhibitor beginning 4 to 7 days before ablation and continuing until 4 to 6 weeks after ablation. In patients with an esophageal ulcer, the addition of cytoprotective agents (such as sucralfate) may be beneficial to prevent fistula formation.

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