Deliberate Epicardial Catheter Ablation Lesions
Making Outside Contact

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One of the major limitations of complex catheter ablation procedures is the inability to ensure predictable effective lesion creation at a large number of sites, including linear or contiguous applications used for isolating pulmonary venous antra or for interrupting ventricular tachycardia.1-3 This is of particular importance for arrhythmias that have a deep intramyocardial or epicardial substrate, most frequently in non-ischemic cardiomyopathies.4,5 Multicenter experiences with catheter access to the pericardial space have increased substantially during the past decade,6 increasing our understanding of the associated risks and limitations. We still need better understanding of the influence of epicardial fat on mapping and ablation, the risk of collateral injury to coronary arteries, the phrenic nerve or other extracardiac structures, and epicardial lesion creation using radiofrequency energy.

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Irrigation of the catheter tip permits greater power delivery and the production of larger lesions but uncouples the temperature of the tip from that of the tissue, removing important biophysical feedback and hence lesion predictability.7,8 This trade-off of safety for effectiveness requires greater care by physicians to deliberately deliver ablation lesions where they are needed and to purposely avoid damage to nontarget tissues. The advent of clinically available catheter tip contact force-sensing technology has opened a new window for the delivery of radiofrequency ablation and returns a degree of control over lesion creation. In endocardial studies, contact force has demonstrated greater correlation with lesion size than other frequently used clinical markers, such as tip temperature, electrogram amplitude, or impedance changes, and has influence on lesion size similar to that of delivered power.9-14

The potential benefit of contact force sensing in the epicardial space has had relatively little attention. In this issue of Circulation: Arrhythmia and Electrophysiology, Wong et al15 systematically studied the effect of this technology on epicardial tissue, including myocardium (directly on or through fat tissue) and coronary arteries (directly on or adjacent to), and directly on phrenic nerve in an in vivo animal model. The catheter was positioned parallel to the tissue to mimic clinical conditions, and power and duration were held constant at 30 W for 30 seconds while force was varied from 5 to 70 g.

The relationship between contact force and lesion depth and volume was approximately linear as was the inverse effect of increasing thickness of epicardial fat, which predictably and reproducibly reduced lesion depth. A novel finding is that lesion attenuation by fat can be partially overcome by increasing contact force. For each 1 mm of fat thickness, there was a 0.7-mm reduction in lesion depth, but for each additional 10 g of force, there was a 0.6-mm increase in lesion depth. Understanding the strength of the influence of the thickness of the fat layer on lesion depth is somewhat limited by the systematic application of higher contact force at sites with thicker fat layers in these experiments, but the observation that deeper lesions still occurred with higher force suggests that when ablation is limited by fat, deliberately increasing contact may be helpful.

Not surprisingly, the corollary observation was that increased contact force also increased collateral injury over the phrenic nerve and when on or adjacent to coronary arteries. The direct application of radiofrequency energy over the phrenic nerve resulted in paralysis in almost all cases with ≥20 g contact force. Paralysis was less frequent when 10 g of force was applied, and injury was avoided with 5 g of force. Histological evidence of coronary arterial injury was seen when radiofrequency energy was delivered directly over a coronary artery, even with as little as 10 g of force and at higher forces when delivered adjacent to an artery. Steam pops were observed rarely at 5 or 10 g contact force but frequently at 20 g (11%), 40 g (12%) and 70 g (33%).

The results of this study extend prior observations of the importance of contact force on the endocardium to the epicardium in an in vivo setting. Prior study has compared endocardial and epicardial ablation using an irrigated contact force-sensing catheter in the ventricles of 20 sheep,16 with power limited to 30 W for 60 seconds. It was found that although contact force was greater on the endocardium than epicardium (39 g versus 21 g), the latter was associated with creation of significantly larger lesions, presumably because of differential cooling by the flow of blood or different catheter orientation. In the study by Wong et al,15 lesion depth increased by ≈1 mm, with each doubling of force in the absence of epicardial fat reaching 7 mm depth in the presence of 70 g of force. This result is difficult to compare directly with previous studies because of the differences in animal models, tissue type, (atrium or ventricle or nonheart tissue), as well as power and duration variations,9-14 but the strength of the relationship is likely to be robust and applicable in humans. This study would have been strengthened...
by reporting the relationship between contact force and other potential predictors of lesion size, such as impedance changes or electrogram amplitude/pacing threshold. It remains to be seen what effect myocardial scar might have on all of these relationships.

Avoidance of injury may be a significant benefit of force-sensing monitoring. Within the left atrium, perforation and esophageal injury may both relate to excess tip-tissue force. In the pericardial space, however, collateral ablation injury to lung, phrenic nerve, and coronary arteries are of greatest concern. The findings in the current study extend the prior observation that contact force is an ablation parameter that can be varied just like power and duration. They include the observations that excessive force increases the likelihood of steam pops with myocardial crating, as well as the likelihood of histologically evident coronary arterial injury and phrenic nerve interruption when applied adjacent to or on top of those structures. Despite the absence of acute coronary occlusion in this study, evidence of arterial injury was evident, even with relatively low force over a coronary artery. Presumably, the rapid flow of blood through a normal coronary artery is protective; however, it is uncertain what long-term effects could follow arterial injury, which may not be clinically obvious acutely. Similarly, these findings cannot be extrapolated to smaller coronary arteries, which may have lesser intrinsic protection; thus, it cannot be suggested that it is safe to ablate on or very near coronary arteries. However, it remains to be determined whether simply directing contact force away from the parietal pericardium can be adequate protection for the phrenic nerve. Phrenic nerve injury may have less severe consequences than coronary injury, but it should still be assiduously avoided. A technique for its protection that could replace cumbersome methods (such as instillation of saline/air or the introduction and inflation of a balloon) would be beneficial.

The information gained from this study is helpful; incorporation of knowledge of contact force during epicardial ablation may improve both efficacy and safety. The rules of thumb for lesion depth/size derived in this study may not apply directly in humans, but the relationships between force and size are likely to be preserved. Similarly, the amount of force that avoids collateral injury may not accurately reflect what can be expected in humans. Understanding the linear relationship between force and lesion size is likely to be helpful, however, and may help improve ablation efficacy and reduce complications of epicardial ablation.

This study is a step toward a more deliberate ablation procedure for VT (ventricular tachycardia). The performance of a substrate-based ablation for unmappable VT is challenged by the lack of clear end points for radiofrequency delivery at target sites and the consequent need to target ancillary sites that may be unnecessary. The ability to create more predictable lesions, or alternatively to assess lesion size, may permit more specific energy delivery, potentially achieving both greater efficacy and lesser chance of collateral injury.

Disclosures

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References


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