

EDITORIAL COMMENT

Eliminating Coagulum Formation With Charge Delivery During Radiofrequency Ablation

Negative May Be a Positive!*

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Since its introduction into clinical practice in the 1980s, radiofrequency (RF) catheter ablation has seen a meteoric rise in the treatment of cardiac arrhythmias owing to its safety and efficacy profile and ability to precisely target arrhythmogenic tissue. Driven by seminal observations by Haines (1), Wittkamp et al. (2), and Nakagawa et al. (3), among many others, the past 3 decades have seen remarkable evolution in catheter design allowing more effective and reliable energy delivery to target tissue. Permanent tissue destruction is a fundamental goal of RF ablation and reliably occurs when tissue temperature of $>50^{\circ}\text{C}$ can be achieved (4). There are a plethora of factors that influence lesion formation; however, lesion size is fundamentally governed by extent of tissue heating, which occurs via resistive and conductive means (5).

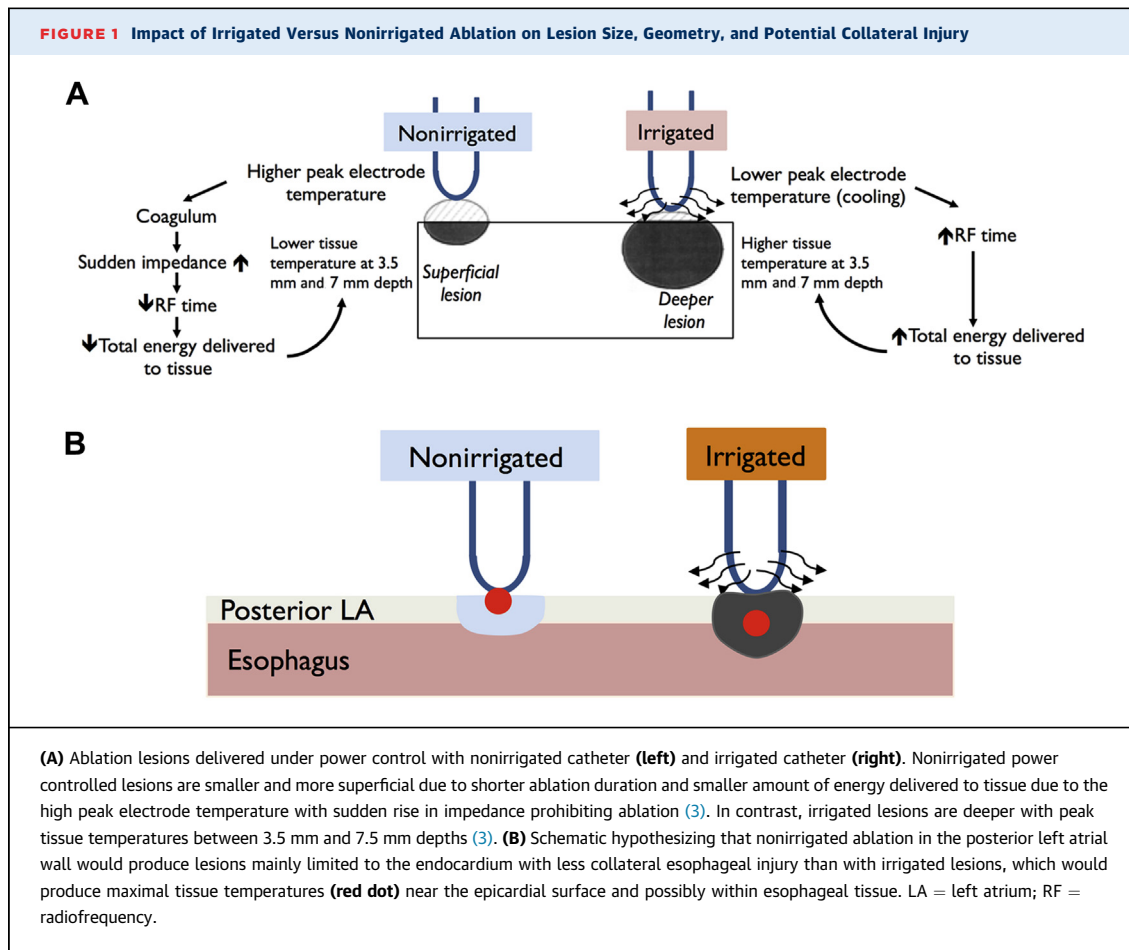
When comparing solid-tip versus irrigated catheters, there are key biophysical differences that must be appreciated (Figure 1A) (3). First, with solid-tip catheters lesion width and depth increase linearly with electrode-tissue interface temperature up to 90°C (6). With electrode-tip interface temperature of 80°C to 100°C , coagulum forms on the catheter tip due to the boiling of plasma and adherence of denatured

plasma proteins. This results in a reduction in the surface area at the catheter tip, a larger local current density, thrombus formation, and progression of the coagulum, followed by a sudden rise in the impedance (6). This phenomenon frequently limits the duration of RF delivery and thus extent of energy delivery to target tissue (3). Without irrigation, tissue temperature is highest just beneath the endocardial surface with a steady gradient to lower temperatures in the deeper layers. If power or lesion duration is limited to avoid high temperatures at the catheter tip-tissue interface, smaller, superficial lesions will result (3). In contrast, irrigation cools the tip-tissue interface, thus avoiding coagulum formation and allowing higher power settings for longer duration. Importantly, surface cooling changes lesion geometry so that a smaller surface area is exposed to tissue temperatures exceeding 50°C and maximal lesion width is found 1 mm to 3.5 mm beneath the endocardial surface (Figure 1A) (3,7,8). Contrary to common belief, irrigated lesions are smaller than nonirrigated lesions at the same power setting and duration (3).

Given the need to create deeper, transmural lesions and the risk of thrombus formation at higher power settings, nonirrigated ablation catheters have been supplanted largely by open-irrigated catheters. However, the risk of clinical thromboembolic events such as stroke or transient ischemic attacks has not been entirely eliminated with the use of open-irrigated catheters despite systemic anticoagulation periprocedurally and additional heparinization intra-procedurally: for example, stroke is reported to occur with a frequency of $\sim 0.6\%$ to 2.5% in atrial fibrillation ablation literature (9,10). Even more compelling are data that asymptomatic thromboembolic events detected by brain magnetic resonance imaging can

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occur in a substantial proportion of patients (14%) undergoing catheter ablation for atrial fibrillation (11), some of whom may experience subtle neurocognitive dysfunction (12). It is entirely possible that many such events may be a result of “soft thrombus” formation, which can develop as a result of denaturation and aggregation of plasma proteins at blood temperatures between 50°C and 80°C. Soft thrombus is poorly adherent to the ablation electrode and is not associated with an impedance increase. In contradistinction, “char formation” can be detected by impedance increase (13). Clearly, thrombus formation remains a significant contemporary challenge during catheter ablation of cardiac arrhythmias.

Within the context of this discussion, the contribution by Lim et al. (14), in this issue of *JACC: Clinical Electrophysiology*, unveils an important novel method of reducing thrombus formation via continuously delivering a negative charge at the catheter tip. The premise for this design is that fibrinogen, which is a fundamental building block for thrombus, is negatively charged and preferentially binds to positively charged surfaces, which then induces conformational

changes in its molecular structure that further invokes a prothrombotic cascade (15).

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The investigators designed a system wherein high-impedance, negative direct current is delivered from a 9-V battery to the ablation catheter in parallel to the signal-processing pathway in a canine model. After identifying that a minimum negative current of 100 μ A was required to eliminate coagulum, the investigators performed ablation using 2 solid-tip catheters and 1 closed-loop irrigation catheter to assess the extent of thrombus formation on the catheter tip using direct inspection, intracardiac echo, and scanning electron microscopy. One-half of the 110 lesions were delivered using negative charge and the other one-half without charge in a nonrandom fashion. Remarkably, negative charge application showed an “all or none” response with negatively charged applications being completely devoid of any coagulum macroscopically, whereas ~91% of the uncharged catheter tip surface was coated with coagulum; these findings were confirmed on scanning electron microscopy. Importantly, negative

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