Editorial Comment

Temperature Response in Radiofrequency Catheter Ablation

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The ablative effect of radiofrequency (RF) energy is based on resistive heating of tissue adjacent to the delivery electrode. Generally, the amount of heat generated relates to the amount of power delivered. Thus, under controlled experimental conditions, delivered power can be used as a parameter to control the amount of myocardial damage. However, in the clinical situation, a large proportion of power delivered is dissipated and lost in circulating cavitory blood, and the amount of power that is effectively used for heating myocardial tissue will vary depending on the quality of electrode-tissue coupling. Several studies including the article by Langberg et al in this issue of Circulation have now demonstrated that temperature measurement yields better control over the creation of RF lesions.

Some basic understanding of the mechanism of tissue heating by high-frequency power dissipation is necessary to appreciate the value and limitations of these techniques. The amount of power, expressed in watts, dissipated in a patient during RF ablation equals the square of total current (I) multiplied by total resistance (R): watts = I²×R. Similarly, in a three-dimensional medium, the amount of power dissipated per unit volume (w) equals the square of current density (i) multiplied by the specific resistance (ρ) of the medium: w = i²×ρ. This implies that power dissipation and, thus, heat generation are not evenly distributed but rather concentrated in area(s) with the highest current density, thus near the ablation electrode(s). With RF delivery in the unipolar mode, current density decreases approximately with the square of the distance from the ablation electrode. This, in combination with the equation above, thus results in a power dissipation per unit volume that decreases with the fourth power of the distance from the ablation electrode.

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With this dramatic decay in dissipated power per unit volume, it is clear that direct resistive heating only plays a major role in very close vicinity to the ablation electrode. Beyond that first layer, other heat transfer mechanisms, predominantly conduction in myocardium, are of major importance for myocardial temperature elevation.

Differences in the relative contribution of these two heating mechanisms in myocardial tissue at various distances from the ablation electrode as explained above might lead to quantitative and qualitative differences in the temperature response at different sites in the electrode-interface-myocardium trajectory: within the ablation electrode, at the electrode-tissue interface, and within myocardial tissue at various distances from the ablation electrode. It therefore is of interest to review the literature with regard to the temperature responses of these three elements during RF ablation.

Ablation Electrode Temperature

In an in vitro experiment, Blouin et al simultaneously measured the steady-state temperature at the inside of the ablation electrode as well as at the electrode-tissue interface during RF ablation. Their study demonstrates a close to linear relation between both temperatures at various power levels under controlled electrode-tissue contact conditions with a consistently lower temperature within the ablation electrode. This linear relation between both temperatures can be expected from the basic principles of heat conduction from electrode-tissue interface via ablation electrode to circulating blood. Temperature gradients along this route will be generally proportional to the amount of heat flow and thus to the total temperature difference between tissue and blood. With stable electrode tissue coupling, the temperature rise of the metal electrode itself can thus be expected to be a certain percentage of the total temperature rise at the electrode-tissue interface.

Electrode-tissue Interface Temperature

Langberg et al and several other investigators studied the electrode-tissue interface temperature under in vitro and in vivo conditions. Hindricks et al studied the predictability of lesion size from electrode-tissue interface temperature in vitro and in canines. With RF power ranging from 3 to 25 W, the temperature at the interface sharply increased within 2–5 seconds to a maximum ranging between 67°C and 106°C.

Also, Haines et al measured the interface temperature in a canine model, again both in vitro and in vivo, demonstrating that coagulum formation occurs at interface temperatures around 100°C. Measurement of electrode and interface temperature can be used to avoid coagulum formation and, more important, to discrimi-
nate between incorrect target sites and insufficient tissue heating during RF applications.

An important finding by Langberg et al is a large spread in interface temperature and the absence of a relation between temperature response and ST segment elevation before ablation at a nominal power setting, suggesting that electrode-tissue coupling is difficult to control, at least in the setting of catheter ablation of accessory atrioventricular pathways. They found a positive dose–response relation between applied power and interface temperature at each individual target site but no significant relation between power and achieved steady-state temperature when all applications at four power settings were compared. However, this conclusion may not be justified. The four different groups are not comparable; part of their sequences of increasing power deliveries were interrupted, for example, because of coagulum formation, which only occurs at high temperatures, and maximum power was only delivered at sites with relatively low temperatures.

**Myocardial Temperatures**

Haines et al measured the radial temperature gradients in perfused and superfused myocardial tissue in vitro with a stabilized catheter tip temperature of 80°C. With increasing distance from the ablation electrode, myocardial temperature decreased in a hyperbolic form. This temperature gradient was unaffected by the rate of coronary perfusion, indicating relatively little contribution of convective heat transfer and a dominant role of heat conduction within the myocardium.

More important with respect to the value of electrode tip and electrode-tissue interface temperature is their observation that the rate of rise of tissue temperatures measured at a distances of a few millimeters from the ablation electrode was rather slow; steady-state temperatures were reached after only 2 minutes.

We recently reported on in vivo epicardial temperature measurements at various distances from the endocardial ablation electrode during RF ablation. Temperature gradients and the rate of rise of local tissue temperatures were determined. We also found a very slow rate of rise of tissue temperatures. After 60 seconds, steady-state temperatures had not yet been reached; only 75% of the temperature rise after 60 seconds was reached after 30 seconds, which indicates an even slower temperature rise in the myocardium under in vivo conditions. Both studies thus demonstrate that during RF catheter ablation, steady-state myocardial temperatures are reached after only a few minutes.

This slowly rising myocardial temperature contrasts with temperature data obtained from the electrode-tissue interface as obtained by Langberg et al and others. Electrode-tissue interface and probably also tip temperature stabilize within a few seconds. This implies a difference in response time of approximately a factor of 60 between the interface, which is heated resistively, and myocardium, which is heated by conduction predominantly.

With an hyperbolic decay of steady-state myocardial temperature with distance, there can be expected to be a strong relation between steady-state interface and myocardial temperature and thus also between interface temperature and lesion size as demonstrated by several investigators. However, major quantitative and qualitative differences exist between both the steady-state value and the time course of interface and myocardial temperatures. Temperature within the accessory atrioventricular pathway depends strongly on its distance from the ablation electrode and will be considerably lower than at the electrode-tissue interface. The rate of rise of interface and myocardial temperature differs with an approximate factor of 60. Both factors limit the value of interface temperature measurement for the response of accessory pathways and other arrhythmogenic substrates located at some distance from the ablation electrode.

Langberg et al associate the fast response of electrode-tissue interface temperature of a few seconds to the clinically observed equally fast response of the accessory pathway to RF catheter ablation. However, as discussed above, the approximately 60 times slower response of myocardial temperature makes it highly unlikely that both phenomena are directly related. Assuming that only temperature and not the high-frequency currents themselves is the basis for the electrophysiological effects of RF energy, the transient effects that are often observed during RF ablation prove that conductive structures can be affected at a temperature level below the level of permanent ablation. With a slowly rising temperature within the myocardium, pathway interruption will be observed much earlier than the moment at which permanent damage is achieved. The delays between RF onset, transient block, and permanent damage then depend on the rate of rise of local temperature, the levels of both critical temperatures, and, of course, on the maximum tissue temperature, the latter being hyperbolically related to the steady-state temperature at the electrode-tissue interface.

These considerations do not change the fact that tip and electrode-tissue interface temperatures are good indicators for the quality of tissue contact. However, both methods have their limitations. As demonstrated in the study by Blouin et al, tip temperatures are consistently lower than electrode-tissue interface temperatures.

An obvious limitation of electrode-tissue interface temperature measurement as described by Langberg et al and others is the importance of tip orientation for the reliability of recorded temperature. In addressing this issue, Langberg et al discuss that it did not appear to be a major limitation in their study. However, in RF catheter ablation of ventricular tachyarrhythmias, the endocardial surface in border zones of infarcted areas may be rather smooth, and it may often be difficult and sometimes impossible to obtain a catheter orientation with the thermistor in contact with endocardium.

Both interface as well as the tip temperature obtained with a nominal power setting will depend on the quality of electrode-tissue contact and thus can serve to judge contact quality. Which one of both methods is clinically most useful will have to be evaluated in further studies. The most important property of ablation catheters remains their mechanical handling characteristics and, especially, steerability. As long as these characteristics remain undisturbed, tip and/or interface temperature is an additional advantage enabling better judgment about catheter position and power delivery and may thus help to improve ablation procedures.
References

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