Brief Rapid Communications

Observations on Electrode-Tissue Interface Temperature and Effect on Electrical Impedance During Radiofrequency Ablation of Ventricular Myocardium

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The purpose of this study was to correlate changes in electrical impedance with the electrode-tissue interface temperature and to characterize the associated events occurring at the catheter tip electrode. In a canine model, lesions were created in vitro (n=49) and in vivo (n=31) and radiofrequency power settings were varied. Electrode-tissue interface temperature, delivered current, and voltage were recorded, and impedance was calculated. A sudden rise in electrical impedance was seen in only two of 17 ablations in vitro and in one of 16 ablations in vivo with a peak electrode-tissue interface temperature of less than 100° C compared with 29 of 32 ablations in vitro (p=0.0001) and 12 of 15 ablations in vivo with a temperature of more than 100° C (p=0.0001). This phenomenon was associated with the observation of boiling and popping at the tip in in vitro preparations and tissue avulsion and thrombus formation on the catheter tip in in vivo studies. The lesion size was directly proportional to the peak temperature for all ablations but not to the peak power, current, or voltage during radiofrequency catheter ablation in the heart. Maintaining electrode-tissue interface temperature at less than 100° C during radiofrequency catheter ablation in the heart may avoid the complications associated with the sudden rise in electrical impedance. (*Circulation* 1990;82:1034–1038)

Catheter ablation has been proposed as a valuable adjunct in the treatment of symptomatic sustained tachyarrhythmias.[1,2] Transcatheter radiofrequency energy ablation causes myocardial injury by direct electrical (resistive) tissue heating as well as by passive (conductive) heating of contiguous tissue.[3] Although it is relatively safe, the formation of coagulum and thrombus on the electrode surface[4] as well as occasional adherent tissue on catheter removal have been observed with radiofrequency ablation. Coincident with these findings is a sudden rise in electrical impedance and a resultant rapid decline in radiofrequency current.[4,5] The impedance rise generally occurs at higher levels of delivered radiofrequency power,[5,6] but the actual causative factor for this phenomenon has not been elucidated.

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In the present study, it was hypothesized that a sudden rise in electrical impedance during radiofrequency catheter ablation might be due to elevation of the electrode-tissue interface temperature above the boiling point, with resultant boiling of plasma and adherance of denatured plasma proteins to the electrode. If the electrode-tissue interface temperature is maintained at less than 100° C for the duration of energy delivery, then boiling with its associated impedance rise should not occur. Thus, the purpose of this study was to assess the effect of electrode-tissue interface temperature during radiofrequency catheter ablation on the electrical impedance of the system.

Methods

In Vitro Preparation

The preparation for an isolated perfused and superfused canine right ventricular free wall has been described in detail elsewhere.[3] In brief, five mongrel dogs (10–35 kg) were anesthetized, and the beating heart was rapidly excised and cooled in iced 0.9% saline at 4° C. The right ventricle with an intact coronary artery pedicle was dissected free from the remainder of the heart and transferred to a warmed (36.5±0.5° C) tissue chamber. The right coronary artery was cannulated, and the specimen was perfused

and superfused with Krebs-Henseleit buffer. During the course of the experiment, tissue temperature was continuously monitored. Radiofrequency lesions were created with a 7F bipolar central lumen electrode catheter attached to a fulcrum device that maintained a constant 196 N (20 g) electrode-tissue contact force.

In Vivo Preparation

Eight mongrel dogs weighing 10–35 kg were anesthetized with pentobarbitol sodium (30 mg/kg), intubated, and ventilated with a dual-phase control respirator pump (Harvard Apparatus, South Natick, Mass.). The right femoral artery was cannulated, and a 7F central lumen bipolar temporary pacemaker wire (Bard Electrophysiology, Billerica, Mass.) was positioned within the left ventricle for subsequent radiofrequency lesion production. The catheter position was documented before each ablation with biplane cinefluoroscopy. After a 2-day recovery period, the animal was euthanized, and the heart was examined grossly and histochemically.

Radiofrequency Lesion Production

All radiofrequency lesions were produced with a Radionics RFG-3AV lesion generator, Burlington, Mass., which produces a continuous 500-kHz sine wave output. For both in vitro and in vivo preparation, the active electrode was the tip electrode of the ablation catheter. The indifferent electrode was a 30-cm^2 aluminum plate on the floor of the tissue bath for the in vitro experiments and a 110-cm^2 cutaneous patch electrode placed on the left lateral thorax of the in vivo preparation. During lesion production, electrode-tissue interface temperature was continuously monitored with a temperature-sensitive fiberoptic probe connected to a Luxtron Model 750 Fluoroptic Thermometer, Mountain View, Calif. This probe was advanced through the central lumen of the ablation catheter until firm contact with the tissue was made. Radiofrequency power output was selected at 20% to 100% of the maximum generator output. Actual delivered root mean squared (RMS) current and voltage were continuously recorded on a strip-chart recorder. Impedance and RMS power were calculated at 1-second intervals from the mean measured current and voltage values during that interval. Mean RMS power was the sum of interval power calculations divided by the number of intervals. Peak power was defined as the maximal value recorded prior to the observed impedance rise. An impedance rise was defined as a sudden increase in calculated impedance exceeding a threshold rate of change of 5% per second. The duration of radiofrequency energy delivery chosen was 90 seconds because a steady-state tissue temperature rise is approached at this time.[3]

Lesion Size Determination

The intact hearts from the in vivo experiments were closely examined, and the lesions were identified by comparing their gross location with the location of the ablation catheter tip on the cinefluorograms. The lesions were inspected for evidence of adherent fibrin or thrombus. Lesions from both preparations were bisected and stained with nitro blue tetrazolium (NBT).[7] Lesion width and depth were then recorded.

TABLE 1. Peak Electrode-Tissue Interface Temperatures and Delivered Power of Observed Patterns of Radiofrequency Ablation In Vitro

Pattern	Temperature range (° C)	Mean peak temperature (° C)	Mean peak power (W)
No boiling	43.6–100.7	81±18	2.2±1.2
Smooth boiling	99.5–113.0	104±5	8.5±5.0
Sudden boiling	99.7–114.3	105±4	17.7±10.8
Popping	102.6–116.8	109±5	19.2±7.8

Statistics

Raw data were stored in a computerized data base. Categorical data were compared in frequency tables, and significance was tested with Fisher's exact statistics. Continuous data were presented as mean±SD. Regression lines were determined by least-squares analysis, and goodness-of-fit was determined by analysis of variance and calculation of a multiple correlation coefficient.

Results

In Vitro Observations

A total of 49 lesions were made with the power output ranging from 10% to 100% and measured mean powers ranging from 0.2 to 52.0 W. During radiofrequency ablation in vitro, four general patterns emerged. No boiling was seen at the electrode tip in 14 ablations. A pattern of smooth boiling was observed during six ablations as the electrode-tissue interface temperature reached approximately 100° C. During 18 ablations, the electrode-tissue interface temperature achieved or exceeded the temperature of boiling; then, sudden boiling was observed, and the temperature rapidly returned to approximately 100° C. Finally, 11 ablations followed a pattern similar to the sudden boiling group but manifested audible popping at the electrode tip with subsequent boiling. The peak electrode-tissue interface temperatures and measured power delivered for the various patterns are presented in Table 1.

During 17 of the 49 ablations, the peak electrode-tissue interface temperature achieved was less than 100° C. An impedance rise was observed in only two ablations (11.8%) in this group (peak temperatures, 99.7° and 99.6° C, respectively). A pattern of smooth or sudden boiling was observed in four ablations (two each) with peak temperatures ranging from 99.6° to 99.9° C. Of the remaining 13 ablations whose electrode tips did not exceed 99.5° C, none demonstrated an impedance rise or visible boiling. Thirty-two of the 49 ablations had a peak electrode-tissue interface temperature of more than or equal to 100° C. Unlike the former group, a significant impedance rise was

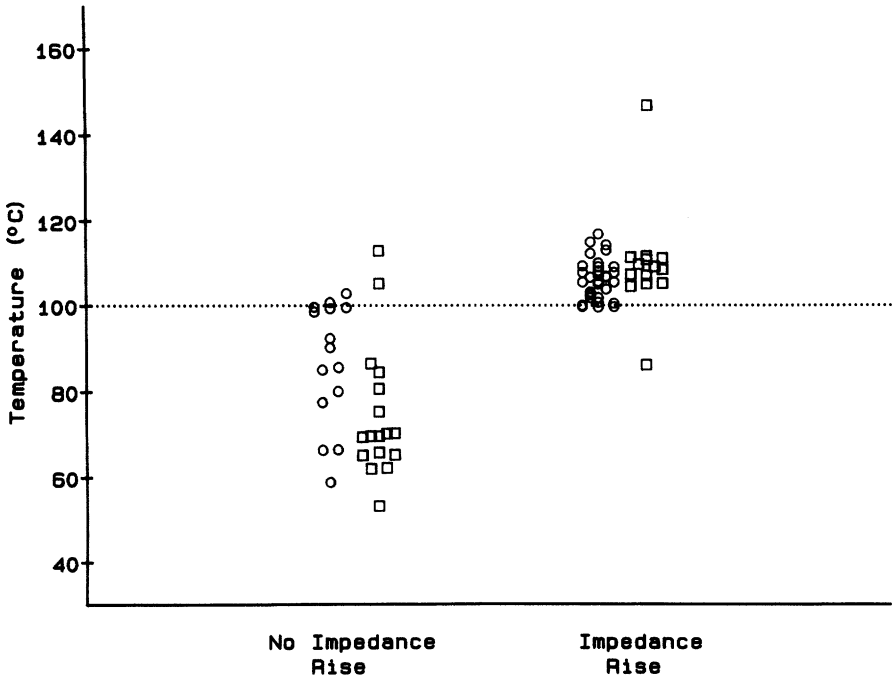

FIGURE 1. *Scatterplot of peak electrode-tissue interface temperature recorded during lesion production from all in vitro (○) and in vivo (□) ablations in experiments with and without a significant rise in electrical impedance. Note strong association of impedance rise with a peak temperature exceeding 100° C.*

measured in 29 of 32 ablations (91%, $p=0.0001$) (Figure 1). Only one of 32 ablations showed no boiling at the electrode tip (peak measured temperature, 100.7° C). Four ablations had smooth boiling, 16 had sudden boiling, and 11 had popping at the electrode tip. In all cases, the sudden impedance rises observed during ablation in vitro were temporally associated with the observations of boiling or popping at the electrode tip.

In Vivo Observations

A total of 31 in vivo lesions were produced with the power output ranging from 35% to 80% and measured peak powers ranging from 4.8 to 20.0 W. The peak electrode-tissue interface temperature did not exceed 100° C in 16 ablations. Of those, 15 showed no impedance rise. By comparison, 15 ablations had a peak temperature of more than 100° C (range, 105–145° C), of which 12 (80%) showed a marked impedance rise ranging between 91% and 400% per second ($p=0.0001$) (Figure 1).

Examination of the electrode catheters after lesion production revealed charring, coagulum, or clot adherent to all electrodes in which an impedance rise was observed. In two cases, a small amount of adherent myocardium was grossly visible at the catheter tip. These findings were not observed in cases without an impedance rise. Upon gross inspection of the endocardial surface at necropsy, fibrin or clot was adherent to the surface of the lesion in eight of 13 cases where an impedance rise was observed compared with two of 18 where there was no rise in impedance ($p=0.005$).

Univariate analyses were performed to quantify the association between radiofrequency-induced lesion size and measurements of energy delivery to the myocardium in vivo. No significant relation was seen between lesion depth and mean delivered current ($p=0.38$, $r=0.16$), voltage ($p=0.12$, $r=0.28$), power ($p=0.63$, $r=0.09$), or peak power before impedance rise ($p=0.28$, $r=0.20$). However, a strong correlation was found between both peak and mean electrode-tissue interface temperature, and lesion size (Figure 2).

Discussion

In the present study, an in vitro system of isolated canine myocardium was used to directly observe the events occurring at the electrode-tissue interface during radiofrequency ablation to elucidate their association with changes in electrical impedance. This protocol demonstrated a close association between sudden boiling, sometimes with audible popping, at the electrode-tissue interface and a rise in electrical impedance. As one would anticipate, boiling only occurred when the electrode-tissue interface temperature approached or exceeded 100° C. Because an in vitro system with crystalloid superfusate might not accurately represent in vivo phenomena, a similar protocol was repeated in an intact animal preparation. In these experiments, a powerful association was also seen between an electrode-tissue interface temperature exceeding 100° C and an electrical impedance rise. It is likely that catheter-tip boiling, similar to that seen in vitro, occurred at the electrode catheter tip when the boiling point was exceeded.

The pathological findings of charring and thrombus found in these cases offer insight into the mechanism of the electrical impedance rise in vivo during radiofrequency ablation. When the electrode-tissue interface temperature exceeds 100° C, tissue contig-

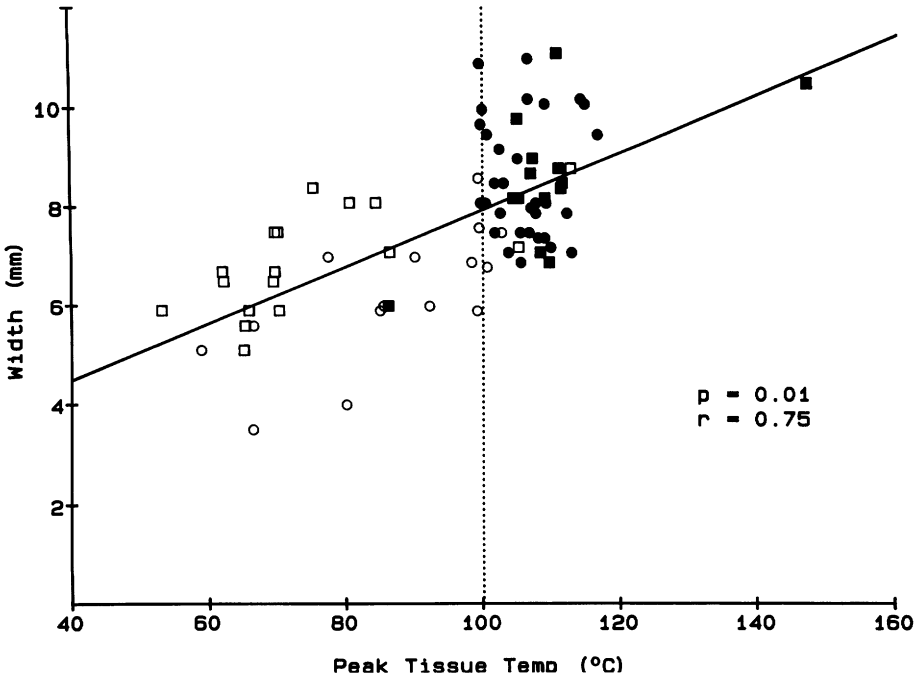

FIGURE 2. *Scatterplot of lesion width versus peak electrode-tissue interface temperature for all in vitro (□) and in vivo (○) lesions. Lesion size from radiofrequency ablation was directly proportional to peak electrode-tissue interface temperature measured during radiofrequency energy delivery irrespective of the occurrence of a rise in electrical impedance; ablations during which an impedance rise was observed are represented by solid symbols.*

uous to the electrode desiccates, and plasma proteins denature to form an insulating layer on the surface of the electrode, generally referred to as "coagulum." Because tissue heating by radiofrequency energy is proportional to the square of current density in tissue,[8] a decrease in delivered current secondary to the insulating effects of coagulum results in a marked decrease in heating. In addition, the denatured proteins and dessicated tissue at the site of ablation are probably thrombogenic and create a nidus from which loosely adherent thrombus might embolize.

The ultimate size and shape of the lesion resulting from radiofrequency ablation may be markedly affected by changes in electrical impedance. Lesion dimension has been demonstrated to be directly proportional to the electrode-tissue interface temperature during radiofrequency catheter ablation.[3] It has been suggested that other parameters of radiofrequency energy delivery, such as current or power, should also be predictive.[9] However, the present study demonstrates that once an impedance rise has occurred, the electrical measurements are no longer useful in predicting lesion size. Only the temperature measurements were predictive of lesion size. Because a practical upper limit of radiofrequency heating exists as the temperature approaches 100° C, a theoretical maximum lesion size exists for any given electrode size and shape. For a standard 6F catheter, this is estimated to be 7.5 mm in depth. To further increase the size of radiofrequency-induced lesions and improve the efficacy rate of radiofrequency ablation, the final parameters that may be adjusted are the electrode size and surface area. A larger electrode tip will increase the lesion size proportional to the electrode radius[10] and to the electrode-tissue interface temperature.

Radiofrequency catheter ablation of the foci of arrhythmias continues to be a promising modality in the treatment of arrhythmias in a variety of locations with varying mechanisms. To optimally control the energy delivery, electrode catheters should incorporate thermometry so that the amount of radiofrequency heating in the myocardium may be accurately gauged. With this technique, boiling at the catheter tip may be prevented. This will in turn prevent a rise in electrical impedance and may decrease the chance of thrombus formation. New catheter designs incorporating larger-tip electrodes with temperature monitors will allow for more predictable and safe but larger and more effective myocardial lesion production.

References

1. Scheinman MM, Morady F, Hess DS, Gonzalez R: Catheter induced ablation of the atrioventricular junction to control refractory supraventricular arrhythmia. *JAMA* 1982; 248:851–855
2. Critelli G, Perticone F, Coltorti F, Monda V, Gallagher JJ: Antegrade slow bypass conduction after closed-chest ablation of the His bundle in permanent junctional reciprocating tachycardia. *Circulation* 1983;67:687–692
3. Haines DE, Watson DD: Tissue heating during radiofrequency catheter ablation: A thermodynamic model and observations in isolated perfused and superfused canine right ventricular free wall. *PACE* 1989;12:962–976
4. Jackman WM, Kuck K-H, Naccarelli GV, Carmen L, Pitha J: Radiofrequency current directed across the mitral annulus with a bipolar epicardial-endocardial catheter electrode configuration in dogs. *Circulation* 1988;78:1288–1298
5. Ring ME, Huang SKS, Gorman G, Graham AR: Determinants of impedance rise during catheter ablation of bovine myocardium with radiofrequency energy. *PACE* 1989; 12:1502–1513
6. Grogan EW Jr, Nellis SH, Subramanian R: Impedance changes during catheter ablation by radiofrequency energy: A potential method for monitoring efficacy of lesion generation (abstract). *J Am Coll Cardiol* 1987;9:95A

7. Gottlieb GJ, Kubo SH, Alonso DR: Ultrastructural characterization of the border zone surrounding early experimental myocardial infarcts in dogs. *Am J Pathol* 1981;103:292–303
8. Erez A, Shitzer A: Controlled destruction and temperature distributions in biological tissues subjected to monoactive electrocoagulation. *J Biomech Eng* 1980;102:42–49
9. Wittkampf FHM, Hauer RNW, Robles de Medina EO: Control of radiofrequency lesion size by power regulation. *Circulation* 1989;80:962–968
10. Haines DE, Watson DD, Verow AF: Electrode radius predicts lesion radius during radiofrequency energy heating. Validation of a proposed hemodynamic model. *Circ Res* (in press)

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