Control of Radiofrequency Lesion Size by Power Regulation

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The influence of power and exposure duration on lesion size in radiofrequency catheter ablation was investigated in 15 closed-chest dogs. Radiofrequency energy was delivered to the right ventricular endocardium between the tip of a standard 6F electrode catheter and a large external surface electrode. A total of 102 ablations were performed at power levels of 0.3–9.3 W and durations of 5, 10, 20, 30, and 60 seconds. At necropsy 1 week later, well-demarcated homogeneous lesions were found when power had exceeded a threshold level that decreased from 1.8 W at 5 seconds to 0.7 W at 60 seconds. Lesion size ranged from 0 to 7.5 mm in depth and 0 to 9 mm in diameter. For the 5, 10, and 20 second ablations, lesion size was determined by exposure duration and power level. However, after a 20 second exposure, lesion size had reached maturity and was related to delivered power only. Therefore, a gradual, controlled growth of the lesion can be obtained by a stepwise increase of the radiofrequency power level with ample exposure duration at each level to allow for stabilization. At levels exceeding 7 W, the formation of a thin insulating layer of blood coagulum on the electrode surface caused an abrupt increase of impedance within approximately 30 seconds. Therefore, lesion size is limited to 8.5 mm in radiofrequency ablation with a standard 6F endocardial electrode catheter. (Circulation 1989;80:962–968)

Closed-chest endocardial DC shock ablation of conduction pathways and arrhythmogenic sites has been used successfully in patients with cardiac arrhythmias.1–6 However, important limitations of this technique, including variability of the size of the ablation lesion at equal energy setting,7 production of a pressure wave,8 proarhythmic effects,4,5,9–11 and incapability of the catheter to withstand high voltage and current,12,13 warrant further evaluation of other energy sources.

By using radiofrequency (RF) alternating current, most of these limitations may be avoided.14–16 With RF catheter ablations in dogs, relatively small, well-demarcated lesions have been observed.14,15,17–20 This holds promise, particularly for ablation of accessory pathways in close proximity to critical structures like the atroventricular (AV) node or His bundle. Modification rather than ablation of the AV node may prevent intranodal reentrant tachycardia with preservation of AV conduction. These applications require that the size of the lesion be controllable. However, in the in vivo setting, the relation of lesion size to physical parameters is controversial.14,19–24

In this study, we report the influence of RF power, exposure duration, and delivered energy on lesion extension.

Methods

Ablation

Fifteen beagles weighing 12–17 kg were anesthetized with methadon:droperidol (20–25 mg i.v.). After endotrachial intubation, ventilation was maintained by a Bird respirator (nitrous oxide:oxygen, 1:1). Anesthesia was maintained by a methadon:droperidol mixture. The dogs were placed on their right side, and a large metal electrode covered with conductive gel was positioned underneath the shaved chest.

Under sterile conditions, two 6F bipolar USCI electrode catheters were introduced into the right femoral artery and vein and advanced to the left and right ventricular cavities, respectively, under fluoroscopic guidance. One of these catheters was
placed in a stable apical position in the left chamber to use its tip electrode as a reference marker to control the position of the heart within the biplane roentgenographic system. The tip electrode of the other catheter, to be used for ablation, was placed in a stable position against the right ventricular free wall. Standard surface electrocardiographic leads I, II, and III and a unipolar electrogram (filter frequency, 0.1–1,000 Hz) derived from the tip electrode at the ablation site were recorded using a multichannel Siemens ink-jet recorder. Close contact between the ablation electrode and the endocardial ventricular wall was ensured by fluoroscopic inspection, high rate of rise of the unipolar electrogram voltage (dV/dt), and the appearance of ST segment elevation in this electrogram. The end-diastolic position of the tip electrodes of both catheters was subsequently marked on frontal and lateral fluoroscopic image-intensifier screens.

RF power was generated from a SSE-2 Valleylab electrosurgical generator that delivers a 500-kHz continuous waveform on its bipolar output. A specially designed and calibrated insulation circuit and a storage oscilloscope were used to monitor and measure RF voltage and current during the ablation procedures. During RF exposure, the electrode resistance was 163 ± 18 Ω. A dummy load of 150 Ω was used to set the generator at approximately the desired power level. Subsequently, the distal electrode of the ablation catheter (surface area, 11 mm²) and the large surface electrode were connected to the generator. RF ablation was then performed at the desired exposure duration while recording the surface electrocardiogram. Only a single RF pulse was delivered at each ablation site. After each ablation, mean delivered RF power was calculated from the mean voltage and current amplitudes. The measurements were calibrated by measuring effective voltage and current and their peak-to-peak amplitudes at various output levels using the 150 Ω dummy load. During RF ablation, an approximately 10° phase shift was observed between voltage-current waveforms at power levels of less than 10 W. However, the same phase shift was also present with the equipment and electrode catheter connected to the 150 Ω dummy load. Thus, the observed phase shift was caused by the connected equipment and wiring and not by the tissue-electrode combination. Exposure durations were set at 5, 10, 20, 30, or 60 seconds, and selected RF power levels were between 0.5 and 10 W. Exposure duration was timed by hand and could be verified by the duration of low-amplitude noise interference on the electrocardiogram. A few 5 second exposures that deviated more than 10% from the intended duration were excluded from this study.

The ablation protocol was as follows: 1) adjustment of RF generator with the 150 Ω dummy load, 2) marking of the position of the tip electrodes of both catheters on the frontal and lateral image-intensifier screens, 3) RF exposure via the tip electrode of the right ventricular catheter, 4) fluoroscopic control of both catheter positions, 5) calculation of mean delivered RF power, and 6) repositioning of the right ventricular electrode catheter to a different site with an estimated distance between subsequent sites of at least 1.5 cm and repetition of the sequence.

At the end of the procedure, the coordinates of the ablation site were measured from the frontal and lateral screens to compute the three-dimensional position of the ablation site as previously described. In the first 10 dogs, the desired RF power level and exposure duration were randomly selected for each ablation site and animal.

In the last five animals, the procedure was slightly different. In 45 of the 61 ablations in these dogs, RF power level and exposure duration were randomly chosen from two selected power levels of 4 and 6 W and all five exposure durations. The remaining 16 procedures were identical to those of the first series.

Pathologic Examination

One week after the ablations, the dogs were anesthetized and killed by exsanguination. With the cadaver placed on its right side, the thorax was opened and three sutures were placed on the heart to indicate its orientation in the thorax. Then, the heart was excised and fixed in formalin. One to 2 weeks later, the heart was carefully sectioned parallel to its base. After visual inspection and palpation, the ablation sites were marked. Identification of the individual lesions was facilitated by interactive computerized visualization of the calculated three-dimensional ablation sites (Figure 1, top panel) on a Macintosh SE computer and verified by comparing their relative positions and mutual distances with the calculated values. For five of the 15 hearts in which more than eight right ventricular ablations were performed, a wire skeleton was constructed as an additional tool to identify the individual lesions (Figure 1, bottom panel). After sectioning through the lesion area, the depth, length, and width of the lesions were measured. Extension of the lesion was always verified by histopathologic examination. The diameter of the lesion was defined as (width + length)/2 and lesion size as (width + length + depth)/3. The lesions that could not be identified by either of the above methods or careful cutting in the vicinity of the calculated location were assigned a size of zero. Lesions were found at all ablation sites where RF power had exceeded 2.4 W. Special attention was paid to the extension of the lesion in a transmural direction. Histologic sections of the lesions were stained with hematoxylin and eosin, and histopathology of the lesion was studied.

Statistical Analysis

The influence of exposure duration and RF power level on lesion size was examined by nonlinear regression and analysis of variance. Test results
Nonsustained ventricular tachycardia (four to 10 complexes) occurred three times within a few minutes after RF exposure.

During the first 20 seconds of RF exposure, we noticed a 10–20% decrease in impedance. Thereafter, both current and voltage amplitudes remained stable.

After RF exposure exceeding 4 W and 10 seconds duration, the distal electrode was always found to adhere to the endocardial surface. With gentle traction, the catheter could easily be removed. After rinsing with water, visual inspection of the catheter did not show any myocardial tissue residues on the electrode surface.

A sudden increase of impedance was observed at 7 W after 23 and 32 seconds and at 10 and 13 seconds during delivery of 9.3 and 9.1 W, respectively. In these cases, electrode impedance rose to approximately 1,000 Ω and the procedure was immediately terminated and the catheter removed. The distal electrode was then found to be covered with a thin layer of coagulum that was firmly attached to the metal surface. This coating was not observed in the absence of a sudden increase of impedance and never occurred when ablations had been performed with less than 7 W. Visual inspection of the ablation catheter never revealed signs of electrode or insulation damage.

No complications were seen during or after ablation. All dogs survived for 1 week.

Lesion Size

At the endocardial side, a discolored area marked the ablation sites. All lesions were found in areas in which the gross anatomic structure or small niches had obviously trapped the distal catheter electrode. In 15 cases, the lesion area was covered with a thin fibrin thrombus. Histologic sections of the lesions revealed a sharply demarcated homogeneous area of coagulation necrosis surrounded by a thin layer of granulation tissue. The lesions were located mainly subendocardially (Figure 2). Fourteen of the 102 lesions were transmural. There were no perforations.

Lesion size ranged from 0 to 9 mm in diameter and from 0 to 7.5 mm in depth. Nontransmural lesions were ellipsoidal in shape with a diameter-to-depth ratio of 1.2±0.3. In the 14 transmural lesions, this ratio was 1.5±0.3.

Relation of Lesion Size to RF Power and Ablation Duration

In Figures 3 and 4, lesion size is plotted against delivered RF power. Transmural lesions in which lesion depth is limited by wall thickness and lesions after RF exposures that were terminated because of the abrupt impedance rise were not used for further analysis. Figures 3 and 4 show the influence of RF dosimetry on lesion size of the remaining 86 lesions.

The minimum power level at which a lesion was observed was 1 W at 60 second exposure duration.
FIGURE 2. Microphotograph of an almost transmural, well-demarcated right ventricular radiofrequency lesion after 1 week. Central necrotic area is surrounded by a thin layer of granulation tissue.

(Figure 4). Sixteen RF exposures between 0.3 and 2.4 W did not create any detectable lesion (Figures 3 and 4). Thus, a certain minimal power level (ablation threshold) is required for production of RF lesions. We defined this threshold as the maximal power at which no lesion was created. At 5, 10, 20, 30, and 60 second exposure duration, this threshold was 1.8, 1.8, 1.1, 1.0, and 0.7 W, respectively.

Of 45 exposures that were intended to be at 4 or 6 W, 36 deviated less than 5% from the desired power level. These 36 lesions were used to investigate the influence of RF power and exposure duration on lesion size. At each experimental condition, three or four lesions were available for this analysis.

The 6 W lesions were significantly larger than those at 4 W. At 4 and 6 W, the 5- and 10-second lesions were significantly smaller than after longer exposure durations. However, the difference between the 20, 30, and 60 second lesions was not significant at both power levels.

On theoretic grounds, one can believe that the rate of growth at a particular time is directly proportional to the amount of growth yet to be achieved.

FIGURE 3. Plot of lesion (L) size, after 5 and 10 seconds of radiofrequency (RF) exposure, against delivered power. (Compare with Figure 4.)

FIGURE 4. Plot of lesion (L) size after 20, 30, and 60 seconds of radiofrequency (RF) pulses against delivered power. (Compare with Figure 3.) At a similar power setting, RF pulses of 20 seconds and longer create larger lesions than with shorter exposures. However, the size of the lesion is similar after 20, 30, and 60 seconds.
TABLE 1. Relation Between Lesion Size and Exposure Duration at 4 and 6 W

<table>
<thead>
<tr>
<th>Power (W)</th>
<th>n</th>
<th>(A_1) (mm)</th>
<th>(A_2) (mm)</th>
<th>(b) (sec \cdot 1)</th>
<th>(R)</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>16</td>
<td>4.5±0.3</td>
<td>4.8±1.4</td>
<td>0.11±0.05</td>
<td>0.86</td>
</tr>
<tr>
<td>6</td>
<td>20</td>
<td>6.4±0.3</td>
<td>6.5±1.2</td>
<td>0.11±0.03</td>
<td>0.91</td>
</tr>
</tbody>
</table>

\(L\), lesion size expressed in \((\text{length}+\text{width}+\text{depth})/3\) in millimeters; \(R\), correlation coefficient; \(t\), exposure duration; \(A_1\), \(A_2\), \(b\), expressed as estimates±SEM.

Nonlinear regression analysis: \(L=A_1-A_2\cdot e^{-bt}\). Nonlinear regression analysis of the results shown as Figure 5 requires that Equation \(L=A_1-(1-e^{-bt})\) is modified to \(L=A_1-A_2\cdot e^{-bt}\) because \(A_1\) and \(A_2\) must be investigated as two independent parameters. From this data, the time constant of lesion growth \((1/b)\) was calculated to be approximately 9 seconds for both power levels.

Discussion

Relation Between RF Power, Exposure Duration, and Size of Lesion

For all exposure durations, lesion size increased with increasing power. Comparison of lesion size at different RF exposure durations showed rapid growth of the lesion during the initial period with a time constant of approximately 9 seconds (Figure 5). After 20 seconds of exposure, time and, thus, energy no longer played a role in determining the size of the lesion. With these longer RF exposures, only the amount of delivered power determined lesion size. The threshold for ablation was then approximately 0.7 W.

We did not investigate the influence of the shape and size of the ablation electrode and the intensity of the electrode wall contact, but one can expect that both parameters will influence the relation between delivered power and lesion size. Thus, for situations that differ from our experimental conditions, the influence of delivered power and exposure duration on the size of the lesion should be reestablished. However, the main findings, being a threshold level for tissue damage, the time course of lesion growth, and, thus, the control over lesion size by power setting, will keep their merit.

Our observations appear to be in contrast to the findings of several authors. Hoyt et al.19 found that power, exposure time, and delivered energy were the most significant factors influencing the amount of myocardial damage. A linear relation between energy and the volume of myocardial necrosis was found. However, only exposures shorter than 20 seconds were used. Because we demonstrated stabilization of lesion size after 20 seconds, no relation between energy and lesion size exists at longer exposure durations. In addition, no such relation was found after in vivo RF ablation of the atrioventricular junction by multiple successive 10 second pulses of increasing power.14 This may be explained by the fact that with intervals of 10–15 minutes between successive exposures, each subsequent pulse has to heat up the already ablated area before lesion enlargement can be obtained.

Controversial results about the relation between delivered RF energy and lesion extension in closed-chest RF ablation of the atrioventricular junction or ventricular or atrial myocardium were reported by several other authors.14,19–24

Results of RF ablations at power levels up to 500 W and shorter exposure durations have also been described,17,18,23,26 but the resulting lesions were comparable to or smaller than those obtained in our study. Mitsui et al.26 using power settings of 7.5–500 W for 0.5 seconds for His bundle ablation, reported a maximal lesion size of 3 mm wide and 2.5 mm deep. Huang et al.18 obtained lesions with approximately the same maximum dimensions as in our study by presumed delivery of 10, 20, and 30 W during 10 seconds, although actual delivered RF power was not reported. Our study indicates that if delivered voltage and current are not monitored, an abrupt increase of impedance, resulting in diminished RF power and energy delivery, will remain unnoticed.20,27 Especially during application in close proximity to the normal AV conduction system, a low power level and a relatively long exposure

![Figure 5. Plot of lesion (L) size after 4 and 6 W±5% exposure increases with increasing exposure duration with a time constant of approximately 9 seconds and stabilization of lesion size occurs after approximately 20 seconds. Vertical bars indicate standard deviation.](image-url)
duration may be preferable; this allows careful monitoring of conduction delay and timely interruption of RF delivery.

Lesion Shape

All lesions were located predominantly intramurally with little endocardial damage (Figure 2). We believe that the mainly subendocardial location of the lesions can be explained by the intramyocardial temperature profile due to the cooling effect on the endocardium by circulating intracavitary blood.

Histopathology

All lesions were sharply demarcated. These findings are in variance with those obtained after DC shock ablation. One week after a 30–250 J DC shock in dogs, an irregular border zone surrounded by small islands of granulation tissue embedded within viable myocardium has been observed. Homogeneity of the lesions created by RF ablation may be important in avoiding late arrhythmogenicity. Notwithstanding catheter adherence to the endocardium after RF exposure above 4 W and 10 seconds, we did not observe any perforations. However, electrode adherence will depend on the surface structure of the ablation electrode; thus, additional research is needed to investigate the safety of electrodes with different surface properties.

Electrode Catheter Integrity

Visual inspection of the tip electrode and the catheter insulation and comparison of the electrical resistance of the catheters after ablation with newly manufactured catheters did not reveal damage to the ablation electrode, the catheter insulation, or the conductor. In our study, the maximal peak-to-peak amplitude of the RF signal was 106 V and 0.7 A. The maximal voltage across the catheter insulation and the effective current through the catheter were, therefore, only 53 V and 0.25 A, respectively.

Clinical Implications

Our results, and those of others, demonstrate that RF catheter ablation can safely be used to create small, well-circumscribed homogeneous lesions. Except for the occurrence of brief episodes of nonsustained ventricular tachycardia shortly after RF exposure, no complications have been observed during the 1 week survival period. Within 20 seconds, the ablation lesion reaches maturity; thereafter, its size is determined only by the RF power level. Therefore, controlled growth of the lesion can be obtained by an uninterrupted, gradual, stepwise increase of the RF power level. Amelee exposure duration is needed for stabilization of the lesion size at each level. The absence of shock waves during RF ablation and the adherence of the electrode to the endocardial surface enable repeated exposures at the same location, if necessary.

Control of lesion extension and the possibility to monitor AV conduction during ablation may be beneficiary for safe use in the interruption of anomalous pathways, particularly those in close proximity to the AV node and His bundle. Modification of AV nodal function in case of intranodal reentrant pathways is also a potentially attractive application of this technique.

We reached four conclusions. First, at any given power below 7 W, RF lesions are full grown within 20 seconds. Second, above 20 seconds of exposure, lesion size is determined by power and not by total delivered energy. Third, controlled growth of the lesion can be achieved by a gradual increase of RF power. Finally, at levels exceeding 7 W for 30 seconds, effective energy transfer to the myocardium is limited by the formation of an insulating layer of debris at the electrode surface.

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