**Microwave Ablation of the Atrioventricular Junction
In Vivo and Ventricular Myocardium
In Vitro and In Vivo**

Effects of Varying Power and Duration on Lesion Volume

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SUMMARY

Catheter ablation in ventricular tachycardia has achieved only limited success using direct current (DC) and radiofrequency (RF) energy, due to either high complication rates or a limited lesion size. Microwave energy represents a possible alternative source of energy for percutaneous ablation of the ventricular myocardium. However, an optimal method for titration of the dose of microwave energy to achieve the desired lesion volume has not yet been established. The safety and efficacy of microwave ablation of the atrioventricular (AV) junction were studied in 11 dogs *in vivo*. The relationship between lesion size following microwave ablation and the power output of or exposure duration to microwave energy was also examined at disparate sites in each superfused left ventricular epicardium *in vitro*. To observe the pathologic changes in the myocardium after microwave ablation, microwave ablation of the endocardium of the left ventricle was carried out in 9 dogs *in vivo*. Complete AV block was achieved in 10 of the above 11 dogs with a mean of 5 applications of microwave energy. The lesion volume *in vitro* demonstrated a parallel increase with power ($r=0.76$) and duration ($r=0.81$). The mean lesion volume at 30 sec was: at 10W, 0.8 ± 1.6 ; 20 W, 34.7 ± 10.3 ; 30 W, 34.7 ± 22.4 ; 40 W, 64.7 ± 64.4 ; 50 W, 87.2 ± 42.3 ; 60 W, 85.8 ± 38.1 ; 70 W, 124.7 ± 36.5 ; 80 W, 134.2 ± 49.0 mm³. The mean lesion volume at 80 W was: at 15 sec, 32.6 ± 37.8 ; 30 sec, 101.2 ± 46.4 ; 60 sec, 180.6 ± 80.1 ; 120 sec, 291.8 ± 122.7 ; and 180 sec, 459.3 ± 204.6 mm³. The ablated lesions showed discrete, homogeneous coagulation necrosis with sharp margins from the adjacent normal myocardium. Microwave energy may thus be more effective than RF energy, and have a lower risk of complications and arrhythmogenesis than DC energy when used for ablation in ventricular tachycardia. (Jpn Heart J 35: 175–191, 1994)

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CATHETER ablation has become an attractive approach for the nonpharmacological treatment of selected patients with drug refractory arrhythmia.^{1,2)} Catheter ablation techniques utilizing direct current (DC) shocks and radiofrequency (RE) energy have been employed in large numbers of patients.³⁾ However, high voltage DC application has limitations in its usefulness because of severe complications including the rupture of cardiac structures due to barotrauma,⁴⁾ the development of late arrhythmias or sudden death postulated to be due to heterogeneity of tissue injury,⁵⁾ and variability in the size of the ablation lesion at an equal energy setting.⁶⁾ RF energy delivery through an electrode catheter has recently been reported to yield a high success rate with few complications for elimination of the anatomical substrate responsible for supraventricular tachycardia in patients with the Wolff-Parkinson-White syndrome,⁷⁻⁹⁾ atrioventricular (AV) nodal reentry¹⁰⁻¹²⁾ and atrial flutter.^{13,14)} The advantages of RF energy include the absence of barotrauma or sparking, no need for general anesthesia, a lower risk of arrhythmogenesis because of the homogeneous nature of the lesion,¹⁵⁾ and the feasibility of controlling the energy delivery.¹⁶⁾ The limitations of RF energy delivery include the requirement for a large surface area electrode, a rise in impedance due to coagulum formation on the catheter tip and tissue vaporization, the critical nature of the catheter-tissue contact, a smaller lesion size, and limited penetration of scar tissue.^{8,9,15)} The latter is of particular importance in patients suffering from coronary artery disease with endocardial scarring.¹⁷⁾ A high success rate could thus not be achieved in patients with ventricular tachycardia (VT)¹⁸⁻²⁰⁾ except for VT due to bundle branch reentry,^{21,22)} or idiopathic (right ventricle outflow, or left ventricular septum) VT.^{23,24)} Failure to cure VT by using RF energy delivered through a catheter in patients with coronary artery disease may be due to the small lesion size and the shallow depth created by the RF,^{25,26)} since the location of the VT foci or pathways may exist at mid-myocardial or subepicardial sites.

Alternative energy sources such as laser energy and microwave energy could therefore be preferable for curing VT by catheter ablation. Lasers offer a unique source of energy for ablation. Of the currently available energy sources, Nd: YAG photocoagulation alone appears to be capable of reaching deep intramural or epicardial sites of reentry from the endocardial surface. Experimental studies have indicated a strong theoretical potential for the Nd: YAG laser catheter ablation of VT^{27,28)} and successful intraoperative ablation of VT has been performed.²⁹⁾ However, problems do exist, and are principally related to the

development of effective catheter systems for endocardial mapping and incorporation of the conduit for the energy source within the catheter. One difficulty more unique to laser photocoagulation concerns the importance of the directionality or fiber orientation to the surface, so that clinical application awaits solutions to the several unresolved technical problems of effective catheter design.³⁰⁾

Microwave energy like RF energy has been employed clinically for interstitial hyperthermia.³¹⁾ A few recent *in vitro* studies have demonstrated that a microwave source is capable of creating a larger volume of lesion tissue than an RF electrode.³²⁻³⁴⁾ However, an optimal method for titration of the microwave dose to achieve the desired lesion volume has not yet been established. In the present study, we examined the safety and efficacy of microwave energy for ablation of the AV junction, the influence of the delivered power and exposure time on the lesion size in an animal model, and the pathologic changes in the AV junction and ventricular muscles following microwave ablation.

METHODS

The catheter used to perform the ablation consisted of a coaxial transmission line terminating in a helical coil antenna. The coaxial cable had an impedance of 50 ohms, was 2.3 mm in diameter and 100 cm in length. The distal 6 mm of the cable was stripped of its outer conductor, exposing the dielectric. A 5 mm helical coil was connected to the central conductor at the tip. The coil consisted of six turns of copper magnet wire wound around the dielectric with a 1 mm spacing between the turns. A 1 mm gap remained between the proximal end of the coil and the distal extent of the outer conductor.

For recording intracardiac electrograms, an electrode was attached to the distal end of the central conductor and a proximal ring electrode to the outer conductor at the base of the antenna. Bipolar signals could then be recorded from the inner and outer conductors of the coaxial cable. A 5 mm helical coil with only a proximal ring electrode at a spacing of 5 mm from the tip connected to the outer conductor at the base of the antenna, was also used to record unipolar signals from the outer conductor of the coaxial cable, and an indifferent electrode was placed in the right leg.

A commercial microwave power source designed for gastroenteric application with an unmodulated, sinusoidal 2450 MHz microwave source (Model HSE-8M and HS-15M, Heiwa Denshi Industry Corp., Japan) was employed.

Experimental protocol: Eleven adult mongrel dogs weighing between 10 and 21 kg were studied. The dogs were anesthetized with pentobarbital sodium (30 mg/kg body weight, intravenously). The animals were then intubated and ventilated with a Harvard Apparatus volume ventilator (Model 613, Harvard Appara-

tus Corp. Inc.). The femoral arterial pressure and surface electrocardiogram (ECG) leads I, II and V₁ were continuously monitored and recorded on a Polygraph MIC-9800 (CA-202, Fukuda Denshi Corp. Tokyo).

Microwave ablation of the AV junction *in vivo*: A microwave antenna catheter (helical antenna) was introduced into the femoral vein and advanced under fluoroscopic guidance to the level of the tricuspid annulus. It was manipulated so as to record the largest bipolar His bundle electrogram. Although this site would probably be too distal for effective ablation of the AV junction with an RF current, it represented an appropriate site for microwave ablation of the AV node because the microwave field produced by the helical coil was proximal to the catheter tip with maximal heating halfway between the two recording electrodes. After the ablation catheter had been positioned, it was disconnected from the recorder and connected directly to the output of the microwave power supply. Continuous monitoring of the surface ECG and arterial pressure was performed throughout.

The ablation protocol varied according to the electrophysiological response to each application of energy. The power was set at between 40 and 90 Watts (W) for 30 sec. When an accelerated junction rhythm and AV block developed during energy delivery, the energy application was continued until complete AV block persisted (usually 30–90 sec following the development of AV block). If application of the microwave energy did not produce even a transient AV block, the antenna catheter was repositioned before the next ablation. All animals were monitored for 3 to 4 hours after the ablation.

When microwave ablation was performed using a unipolar microwave antenna catheter, a hexapolar system with a 2 mm space (USCI Corp.) was manipulated to record the shortest AH interval and A/V ratio ≥ 1 with the distal and fourth electrodes of the catheter. A unipolar electrogram employing the fourth electrode at the same position was also recorded. A 5 mm helical coil with a 5 mm space between the tip and recording electrode was then positioned at the same place so as to record a similar unipolar electrogram to that before. The microwave ablation protocol was carried out with the microwave antenna catheter described earlier. Microscopic examinations were later performed on the dog.

Microwave ablation of the myocardium *in vitro*: Animals were sacrificed at 3–4 hours after completion of the AV junctional ablation procedure. The hearts of the dogs were placed in a container filled with lactate Ringer's solution (Na⁺ 130 mEq, Cl⁻ 109 mEq, K⁺ 4 mEq, lactate⁻ 28 mEq, Ca²⁺ 3 mEq). To assess the integrity of the duration and power of microwave ablation, microwave ablation using a 5 mm helical antenna catheter was performed on the surface of the left ventricular epicardium for the desired exposure duration and with the desired

power delivered. Only a single microwave application was made at each ablation site. Exposure duration settings of 15, 30, 60, 120 and 180 sec with a constant selected power (80 W), and power delivery settings of 10 to 80 W, at 10 W intervals, with a constant time (30 sec) were utilized. The lesion perimeters were clearly demarcated by discoloration. The dimensions of each lesion, including its diametric length, width and depth, were measured. Taking the morphology of the ablation lesion as approximating to a rotated ellipsoid, the lesion volume was estimated as $1/6\pi \times \text{length} \times \text{width} \times \text{depth}$.

Microwave ablation of the myocardium *in vivo*: In order to examine the safety *in vivo* and the histologic changes of the lesions in the ventricular myocardium caused by microwave ablation, microwave energy was applied to the left ventricular endocardium in 9 dogs weighing between 14.5 and 22 kg. A 10 mm helical antenna catheter was used for the microwave ablation. The microwave antenna catheter (helical antenna) was introduced into the left carotid artery with a long sheath (9F, 23 cm) and advanced to the left ventricle under fluoroscopic guidance. It was manipulated to a position in the endocardium at the septum, apex or lateral wall (all of 20 ablated sites). The power output was set at between 60 and 70 W, and the exposure duration at between 120 and 180 sec. Only the same microwave power and applied duration were utilized in each dog. The lesion perimeters were clearly demarcated by discoloration and the dimensions of the lesions were measured by the method described above. The canine heart was then fixed in 10% formalin. The ventricular wall was sectioned through the ablation lesions, and the slices were stained with hematoxylin-eosin and azan.

Statistical analysis: All data are expressed as the mean $\pm$ SD. Differences in the lesion dimensions and volume among the groups at the same power and after the same duration were evaluated by Student's t test. The relation between lesion size after microwave ablation and the microwave power or exposure duration was analyzed by linear regression analysis. Differences were defined as significant when the p values were <0.05 .

RESULTS

AV junctional ablation: The effects of microwave ablation with a bipolar antenna catheter are illustrated in Figure 1. Microwave application was performed at the site where a His bundle electrogram was clearly recorded in the bipolar mode under fluoroscopic guidance. Complete AV block was induced at 9 sec after the onset of microwave energy delivery. The arterial blood pressure underwent no change before and after ablation. The QRS complex in the surface ECG and His bundle electrogram after ablation revealed complete AV block and the escape rhythm was derived from the proximal site to the His bundle.

Figure 1. Microwave ablation of the AV junction. A: His bundle electrogram recording with a bipolar antenna electrode before ablation. B: Complete AV block induced at 9 sec after the onset of microwave energy delivery. C: His bundle electrogram after microwave ablation. Note that the site of AV block was proximal to the His bundle. I, II, V₁=surface ECG I, II, V₁; HBE=His bundle electrogram; Aop=arterial blood pressure.

Figure 2 shows bipolar and unipolar His bundle electrograms at the same position recorded using a hexapolar catheter with a 2 mm spacing (left panel). A unipolar His bundle electrogram recorded with a unipolar antenna catheter is shown in the right panel of Figure 2. The unipolar antenna catheter was placed in the same position as the hexapolar catheter under fluoroscopic guidance. The unipolar His bundle electrogram exhibited a similar configuration to that recorded with the hexapolar catheter. Microwave energy (70 W) was delivered to that site and an accelerated junctional rhythm was noted at 11 sec after the onset

Figure 2. His bundle electrogram recordings using bipolar and unipolar modes before ablation. A: The His bundle electrogram was recorded using bipolar electrodes (1, 4) of a hexapolar catheter with a 2 mm spacing (USCI). Note that the A wave is larger than the V wave, and the His bundle wave is very small but can be seen. Below this electrogram, a His bundle electrogram recorded with a unipolar electrode (4, indifferent electrode) of the same hexapolar catheter is shown. Note that the A wave recorded with the bipolar electrodes is larger than that recorded with the unipolar electrode of the same catheter at the same position. B: The His bundle electrogram was recorded using a unipolar antenna electrode with a 5 mm helical coil at the same position as the hexapolar catheter under fluoroscopic guidance. The His bundle electrogram shows a similar configuration to that recorded with the unipolar electrode of the hexapolar catheter.

of power. Complete AV block was then achieved at 26 sec after the onset of microwave power. The arterial blood pressure remained the same before, during and after the ablation (Figure 3). Figure 4 shows the surface ECG, arterial blood pressure and His bundle electrogram recorded after the microwave ablation. Complete AV block was induced and the escape rhythm was derived from the proximal site to the His bundle.

Complete AV block with microwave energy was achieved in 10 of the 11 dogs. The conditions for inducing complete AV block are summarized in the Table. A microwave energy of 40–90 W was applied for 30–60 sec. Persistent complete AV block was achieved with a mean of 5 (range, 1–10) applications of microwave energy. All dogs remained in complete AV block during the 3–4 hour monitoring period after ablation. The escape rhythm rate was 68 ± 20 beats/min, excluding 2 dogs in which xylazine was used for sedation.

Figure 3. Surface ECG and arterial blood pressure recordings during microwave ablation in another dog. Note that an accelerated junctional rhythm appeared 11 sec after the onset of power delivery, and complete AV block resulted 26 sec after the onset of power. Arterial blood pressure did not change during or after ablation.

Figure 4. Surface ECG, arterial blood pressure and His bundle electrogram after ablation. Note that complete AV block was induced and the escape rhythm was derived from the proximal site to the His bundle because the His bundle wave preceded the V wave.

Table. Conditions for Successful Microwave Ablation of the AV Junction in the Dog

Animal no.	No. of applications	Ablation conditions		His bundle electrogram					Escape rhythm	
		Power (Watt)	Duration (sec)	Electrode	AH (msec)	HV (msec)	A/V	Amplitude (μ V)	Origin position	Beats (min)
1	7	40	30	bipolar	58	40	1.00	74	AV junction	38
2	8	45	30	bipolar	58	38	0.55	96	AV junction	88
3	10	60	60	unipolar	#	#	#	#	ventricle	71
4	15	failure	failure	unipolar	—	—	—	—	—	—
5	8	70	60	unipolar	#	#	#	#	ventricle	89
6	7	70	60	unipolar	#	#	#	#	ventricle	42
7	8	80	60	unipolar	54	24	<1.0	$\pm$	AV junction	76
8	4	80	60	unipolar	97	26	0.13	$\pm$	AV junction	21*
9	1	70	60	unipolar	88	32	1.01	$\pm$	ventricle	20*
10	4	70	60	unipolar	65	28	0.49	$\pm$	AV junction	71
11	8	90	120	unipolar	58	32	0.32	$\pm$	AV junction	78

AV junction=atrioventricular junction; #=not recorded; *=xylazine was used for sedation.

Figure 5. Right atrial and right ventricular view of a canine heart showing the effects of microwave antenna catheter ablation in the AV nodal area. The arrow points to a well-demarcated lesion at the AV junction. The dog was killed 4 hours after ablation. Complete AV block was achieved after 8 applications of microwave energy using a 5 mm helical coil antenna catheter. RA=right atrium; RV=right ventricle; TV=tricuspid valve annulus; S=septal valve; CS=coronary sinus; ABL=ablated site.

The macroscopic and microscopic findings for the AV junction are illustrated in Figures 5 and 6, respectively. An area of well-demarcated oval coagulation necrosis was observed at the AV junction without destruction of the surrounding normal tissue and valves in all animals (Figure 5). As shown in Figure 6, microscopic examination of the lesions revealed extensive necrosis involving the

Figure 6. Low power photomicrograph of the AV junction. The borders of the areas of extensive necrosis are quite distinct from the normal tissue (arrowheads). The AV node, right atrial endocardium and interventricular septum are involved. AS=atrial septum; RA=right atrium; LA=left atrium; LV=left ventricle; TV=tricuspid valve; N=AV node; IVS=interventricular septum; AL=ablated lesion; NT=normal tissue. (Hematoxylin-eosin stain. Original magnification, $\times 10$)

AV junction, right atrial endocardium and summit of the interventricular septum. A discrete boundary between the necrotic tissue and the normal tissue was noted. The structure of the myocardial fibers was destroyed and the interstitium appeared congested in the capillaries under a high power microscope.

Relation between microwave energy power and lesion size *in vitro*: The effect of the delivered power of the microwave on lesion size *in vitro* is illustrated in Figure 7. Increasing the power (from 10 to 80 W) at a fixed duration (30 sec) resulted in a parallel increase in ventricular lesion volume. The lesion volume varied directly with power from 10 to 80 W ($r=0.76$); at 10 W, 0.8 ± 1.6 ; 20 W, 34.7 ± 10.3 ; 30 W, 34.7 ± 22.4 ; 40 W, 64.7 ± 64.4 ; 50 W, 87.2 ± 42.3 ; 60 W, 85.8 ± 38.1 ; 70 W, 124.7 ± 36.5 ; and 80 W, 134.2 ± 49.0 mm³. A significant increase in lesion volume was observed from 10 to 50 W. However, there were no significant increases in lesion volume over 50 W under the present experimental condi-

Figure 7. Effect of microwave power on lesion size. The lesion size increased as the power was increased from 10 to 50 W. The rate of increase diminished above 50 W. See text for details.

tions.

Relation between microwave exposure duration and lesion size *in vitro*:

The relationship between the duration of microwave delivery at a fixed power output (80 W) and lesion size is shown in Figure 8. The lesion volume varied directly with microwave exposure duration from 15 through 180 sec ($r=0.81$). Increasing the exposure duration yielded a significant increase in lesion volume of the left ventricular myocardium (15 sec, 32.6 ± 37.8 ; 30 sec, 101.2 ± 46.4 ; 60 sec, 180.6 ± 80.1 ; 120 sec, 291.8 ± 122.7 ; and 180 sec, 459.3 ± 204.6 mm³). In contrast to the lesion volume, the lesion surface area (based on length and width) exhibited a significant increase only at durations of 15, 30 sec and 60 sec. There was no significant difference in lesion surface area among the different exposure durations above 60 sec, although the lesion depth did increase with increasing exposure duration above 60 sec. The depth of the ablation lesions in the ventricular myocardium revealed a significant increase with increasing exposure duration to ablation from 15 through 180 sec ($r=0.93$). The depths were at 15 sec, 0.9 ± 0.5 ; 30sec, 2.6 ± 0.7 ; 60 sec, 3.8 ± 0.8 ; 120 sec, 5.1 ± 0.6 ; and 180 sec, 7.1 ± 1.1 mm.

Microwave ablation of the left ventricle *in vivo*: The lesion size ranged from

Figure 8. Effect of exposure duration on lesion size. The lesion size increased through 30 to 180 sec (n=5). Note that the increase in lesion depth was more closely related to the exposure duration than was the lesion length or width. See text for details.

Figure 9. Left ventricle view of a microwave ablated lesion obtained high energy setting (70 W, 180 sec) *in vivo*. Note the transmural involvement of the left ventricle and the well-defined margins of the ablated lesion of the epicardium.

Figure 10. Low power photomicrograph of the ablated lesion of the left ventricle. There is a well-defined margin (arrowheads) between the ablated lesion and the adjacent normal myocardium. AL=ablated lesion, NM=normal myocardium. (Hematoxylin-eosin stain. Original magnification, $\times 10$)

8 to 14 mm (10.9 ± 3.2 mm) in diameter and from 5 to 10 mm (7.1 ± 2.2 mm) in depth. The lesion volume varied directly with the power delivered (259.5 ± 70.5 mm³ at 60 W and 120 sec; 495.3 ± 300.4 mm³ at 70 W and 120 sec, 357.3 ± 166.2 mm³ at 60 W and 180 sec; and 467.2 ± 530.6 mm³ at 70 W and 180 sec). The lesions were ellipsoidal in shape with a diameter-to-depth ratio of 1.59 ± 0.43 . The lesion depth involved an average of 1/2 to 3/4 of the thickness of the left ventricular myocardium. Transmural extension of left ventricular lesions was observed in the apical area of the left ventricle in 5 ablation sites using 70 W and 120 to 180 sec (Figure 9). Microscopically, the microwave ablated lesions showed homogeneous coagulation necrosis with sharp margins from the adjacent normal myocardium (Figure 10).

DISCUSSION

High energy direct current pulses have been employed as an energy source clinically since 1982.³⁵⁾ A high energy electrical discharge (100–300 J) from a conventional defibrillator to a catheter electrode usually gives rise to an instantaneous damped monophasic sinusoid potential at a peak voltage of 2000 to 3000 V at the electrode surface. This condition results in an explosive flash and spark (fulguration) and produces a shock wave of >1 to 2 atm pressure. The combination of sparking and barotraumatic influences may disrupt the cellular integrity and contribute to several untoward effects, including perforation, hypotension, focal hypokinesia or dyskinesia, arrhythmias and damage to the catheters.³⁶⁾ An-

other problem is that although Hauer et al⁶⁾ demonstrated that lesion size increased with increasing DC energy, the size of the ablation lesion varied at an equal energy setting, and the lesions exhibited a very irregular border zone surrounded by small islands of granulating tissue embedded within viable myocardium. Such inhomogeneity could play a role in late arrhythmogenesis.

Although RF current ablation gave rise to little ventricular dysfunction and less ventricular arrhythmia and induced more discrete and homogeneous necrosis as compared to DC ablation, the lesion size induced by the RF current was smaller. RF catheter ablation employing 300 to 750 kHz continuous unmodulated sinusoidal waveforms with a peak voltage of <100 V can achieve highly localized endocardial necrosis without explosive gas formation or barotraumatic side effects.³⁷⁾ The RF current is delivered between an electrode catheter inside the heart and a large patch electrode on the body surface. During RF catheter ablation, the electrode is in direct contact with the tissue and the current flows directly from the catheter electrode tip into the low resistance and moist tissue, resulting in an acceleration of ions and resistive heating of the electrode-tissue interface. Further, the heat arising in the tissue slowly drives the extracellular and intracellular water out of the tissue, leading to desiccation and coagulation necrosis.³⁸⁾ Since the power density, which corresponds to the amount of resistive heating for any given volume of tissue, falls as $1/r^4$ with distance from the surface of the electrode, only a small rim of tissue around the catheter tip is heated directly by the current. The majority of the thermal injury is mediated by the conduction of heat away from the area of electrode contact into the surrounding tissue. The temperature gradient seen during RF ablation is thus accurately modeled as a heat source with a diameter only slightly larger than the ablating electrode.^{16,39)} RF-induced lesions increase rapidly in size during the initial 30 sec of heating, but the rate of increase subsequently diminishes because effective energy transfer to the myocardium is limited by the formation of an insulating layer of debris at the electrode surface.¹⁶⁾ Moreover, following an increase of output power in RF ablation, the temperature rises (exceeding 100°C) at the electrode-tissue interface because the increase in power density results in the formation of coagula that coat the ablating electrode, so insulating the electrode and thereby causing an abrupt rise in impedance which limits the current flow and prevents lesion expansion.^{16,40,41)} Electrode size should influence lesion size in RF ablation. Langberg et al³⁹⁾ demonstrated that increasing the electrode length from 2 to 4 mm resulted in a more than doubled lesion volume, while increasing the electrode length beyond 4 mm produced progressively smaller lesions because of the decreased current density at the electrode-tissue interface.

In contrast to the heating associated with RF currents, the heating that accompanies microwaves is induced by the propagation of the electromagnetic

field that is radiated by the microwave antenna into the surrounding tissue. The electromagnetic field raises heat from the energy of the dielectric molecules through which the field passes by both conduction and displacement currents.⁴²⁾ While maintaining alignment with the alternating electric field, neighboring energized dipole molecules collide with each other producing heat as a result of friction. Since this field heats through molecular interactions in the nearby tissue and is not dependent on current flow from the ablation catheter to tissue and direct electrode contact, coagulation necrosis of the myocardium adjacent to the catheter does not limit the energy transfer, and the catheter contact pressure and tissue desiccation do not limit the lesion formation.^{32,43)} In the present study, the volume and depth of the ablation lesions obtained with microwave energy increased markedly with increasing power and duration of the delivered energy, and could be well controlled. Although microwave ablation of the ventricular myocardium led to lesion volume expansion through 180 sec, the lesion depth increased more closely with the duration of delivered energy than did the lesion surface area. This phenomenon may derive from the effect of the angle between the microwave antenna and tissue surface, since Wonnell et al⁴³⁾ have demonstrated that the heating pattern from a microwave antenna dropped off both radially and axially, but much less abruptly than that from an RF electrode. This characteristic of microwave ablation may be suitable for eliminating VT foci due to prior myocardial infarction. Furthermore, the pathologic changes of ablated lesions obtained with microwave energy revealed that the lesions showed discrete, homogeneous coagulation necrosis with sharp margins from the adjacent normal myocardium on low power photomicrography of sites of microwave ablation. There would thus be a lower risk of arrhythmogenesis after ablation.

Conclusion: Our results suggest that microwave energy may be more useful for the ablation of drug refractory VT than either direct current or RF energy. Further studies are needed to establish the safety of applying microwave ablation to the ventricular myocardium, to elucidate the effects of microwave ablation for VT *in vivo*, and to develop a steerable and flexible catheter using a coaxial cable.

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