Flow Effect on Lesion Formation in RF Cardiac Catheter Ablation

Hong Cao, *Student Member, IEEE*, Vicken R. Vorperian, Supan Tungjitkusolmun, *Member, IEEE*, Jang-Zern Tsai, *Student Member, IEEE*, Dieter Haemmerich, *Student Member, IEEE*, Young Bin Choy, *Student Member, IEEE*, and John G. Webster*, *Life Fellow, IEEE*

***Abstract*—This study investigated the flow effect on the lesion formation during radio-frequency cardiac catheter ablation in temperature-controlled mode. The blood flow in heart chambers carries heat away from the endocardium by convection. This cooling effect requires more power from the ablation generator and causes a larger lesion. We set up a flow system to simulate the flow inside the heart chamber. We performed *in vitro* ablation on bovine myocardium with three different flow rates (0 L/min, 1 L/min and 3 L/min) and two target temperatures (60 °C and 80 °C). During ablation, we also recorded the temperatures inside the myocardium with a three-thermocouple temperature probe. The results show that lesion dimensions (maximum depth, maximum width and lesion volume) are larger in high flow rates ($p < 0.01$). Also, the temperature recordings show that the tissue temperature rises faster and reaches a higher temperature under higher flow rate.**

***Index Terms*—Ablation, cardiac catheter ablation, convection film coefficient, lesion dimension, RF cardiac catheter ablation, temperature recording.**

I. INTRODUCTION

SINCE the 1980s, radio-frequency (RF) catheter ablation has proven a successful cure for some cardiac arrhythmias, such as atrioventricular node reentrant tachycardias, accessory pathways and fascicular ventricular tachycardias, with a success rate of about 95% [1]–[3]. Its efficacy, controllability, minimal invasiveness, and relatively low cost make it a preferable method for treating cardiac arrhythmias. It is also a promising method for curing ventricular tachycardias [3]–[5]. During catheter ablation, an electric current with a frequency between 300 kHz and 1 MHz is applied between the catheter electrode (~2.6 mm in diameter) in contact with the endocardium and a rectangular (~15 cm × 9 cm) dispersive electrode attached at the back of the patient. Thermal myocardial injury occurs as a result of direct resistive heating (Joule effect) at the catheter-tissue electrode interface and passive heat conduction to deeper tissue. When the tissue temperature reaches 50 °C [6], irreversible myocardial injury occurs. The cells lose electrical excitability and the reentrant pathways are interrupted. 50 °C is usually considered as the threshold for lesion formation.

Most RF ablation units work in two modes: power-controlled mode or temperature-controlled mode. In power-controlled ablation, a constant power is applied. The ablation generator adjusts the delivered current to maintain a constant delivered power even though the impedance may change during ablation. In temperature-controlled mode, an insulated thermistor is located at the tip and reads the temperature at the point of contact in the endocardium. The ablation generator first applies incremental power to raise the temperature at the catheter tip. Once the catheter tip temperature reaches the preset target temperature, the ablation generator reduces the delivered power and adjusts the power level to keep the catheter tip temperature at the target temperature. To ensure safety, there are limits of allowed maximum power and allowed maximum catheter tip temperature to avoid charring of endocardium, which causes unnecessary damage to the heart. Since catheter ablation is a heating process, temperature-controlled mode ablation provides better control of lesion size than power-controlled mode ablation [7], [8]. Physicians empirically set different target temperatures, generally >55 °C. In isolated tissue preparation models, a higher preset target temperature results in a larger lesion [7], [9].

In addition to the target temperature, the blood flow in the heart chambers also plays an important role in lesion formation in temperature-controlled catheter ablation. The blood flow carries away heat from the endocardium and from the catheter tip by convection. It has a cooling effect on the myocardium and affects the final lesion size [9], [10]. For a normal heart, the cardiac output is about 5 L/min. But at different locations inside heart chambers, the flow rates are significantly different. The flow rate at the surface of mitral or tricuspid valves is much higher (~5 L/min) than the flow rate underneath the valves (near 0). So the cooling effect due to blood convection differs at different ablation locations. Even though the tip temperature is set at the same temperature, it results in different lesion sizes at different locations.

The convective cooling effect should also be considered for new catheter design for treatment of ventricular tachycardia

Manuscript received March 2, 2000; revised January 8, 2001. This work was supported by the National Institutes of Health (NIH) under Grant HL56143. *Asterisk indicates corresponding author*.

H. Cao, J.-Z. Tsai, and Y. B. Choy are with the Department of Electrical and Computer Engineering, University of Wisconsin-Madison, Madison, WI 53706 USA.

V. R. Vorperian is with the Department of Medicine, University of Wisconsin-Madison, Madison, WI 53792 USA.

S. Tungjitkusolmun is with the Department of Electronics, Faculty of Engineering, and the Research Center for Communications and Information Technology, King Mongkut's Institute of Technology Ladkrabang, Chalongkrung Road, Ladkrabang, Bangkok 10520, Thailand.

D. Haemmerich is with the Department of Biomedical Engineering, University of Wisconsin-Madison, Madison, WI 53706 USA.

*J. G. Webster is with the Department of Biomedical Engineering, University of Wisconsin-Madison, 1415 Engineering Drive, Madison, WI 53706 USA (e-mail: webster@engr.wisc.edu).

Publisher Item Identifier S 0018-9294(01)02458-2.

(VT). The reentrant pathway of VT is deep in the myocardium in some cases. It is desirable to obtain a large lesion volume to cure VT. Recently, several research groups proposed large-tip electrodes [11] and saline-irrigated electrodes [12] for VT catheter ablation. These methods are based on the concept of increasing the cooling effect at the endocardial surface so that more power can be delivered to create a deeper and larger lesion.

The goal of this study was to determine the flow effect on the lesion dimensions during catheter ablation. We set up a circulation system to simulate the flow rate inside the heart chambers and carried out *in vitro* catheter ablation on bovine myocardium. We also used a thermocouple probe to record the temperature change inside the myocardium during ablation to help us understand the heating process during ablation.

II. Model and Numerical Simulation

RF ablation lesion formation depends on the temperature distribution inside the tissue, which, in turn, depends on the local electric current distribution during ablation and heat conduction from or to other regions. The temperature change during ablation inside myocardium is governed by the bio-heat equation

$$\rho c \frac{\partial T}{\partial t} = \nabla \cdot k \nabla T + J \cdot E - Q_h \qquad (1)$$

where

- T temperature of myocardium (°C);
- ρ density (kg/m^3);
- c specific heat (J/kg·°C);
- k thermal conductivity (W/m·°C);
- J current density (A/m^2);
- E electric field intensity (V/m);
- Q_h heat loss due to blood perfusion in the myocardium (W/m^3).

Q_h is neglected since it is small during the normal ablation period of about 60 s [8]. At the blood catheter, and the blood tissue interfaces, there is heat exchange due to the blood flow convection

$$k \frac{\partial T}{\partial n} = h_b (T - T_{\mathrm{bl}}) \qquad (2)$$

where h_b is the convective film coefficient due to the blood flow (W/m^2·°C). T_{bl} is the temperature of blood, which is considered 37 °C due to blood circulation. The electric field can be calculated from Laplacian equation

$$\nabla \cdot \sigma \nabla V = 0 \qquad (3)$$

where σ is electric conductivity (S/m). RF catheter ablation is a thermal-electrical process and it is not practical to solve the coupled problem analytically. The finite element method (FEM) is usually used to solve it numerically [9], [13]–[17].

Due to the geometry of the ablation setup (small hemisphere catheter electrode and large dispersive pad), the current density decreases as a function of distance from the catheter surface and is proportional to $1/r^2$. Hence, the power density decreases at a faster rate of $1/r^4$. As a result, only a thin (1–2 mm) rim of tissue adjacent to the catheter tip is heated directly by the applied RF power. The majority of thermal injury is produced by heat conduction from the high-temperature rim to the surrounding myocardium [8].

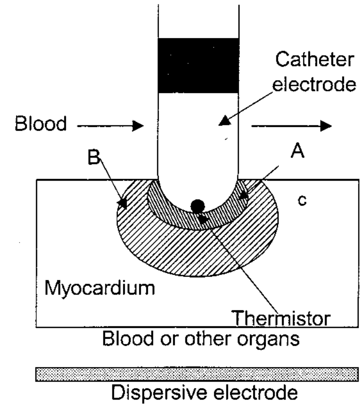


Fig. 1. Simplified model of cardiac catheter ablation. The catheter is in perpendicular contact with the myocardium. Current is applied between the catheter electrode and the dispersive electrode. The myocardium in region A is heated by the Joule effect and heat conduction. The myocardium in region B is mainly heated by the conduction from region A. Blood flowing around the myocardium and catheter cools them by convection.

The blood remains at 37 °C during ablation. The temperature difference between the ablated myocardium and blood flow in the heart chamber causes heat transfer from the endocardium to the blood by heat convection. It has a cooling effect on the catheter surface and endocardial surface. As heat is carried away from these surfaces, the temperature at the endocardial surface is lower than the temperature inside the myocardium. The lesion diameter on the surface is smaller than the maximum lesion diameter inside the myocardium as shown in Fig. 1.

The convection film coefficient is dependent on flow around the catheter. High flow results in a high film coefficient and more heat is carried away by convection. Then it takes more power to maintain the catheter at a target temperature under high flow conditions. The ablation generator delivers more electric current to the catheter. This has several effects on lesion formation.

1) It increases the current density and Joule heat generation inside the myocardium. The myocardium rises to a higher temperature and more myocardium exceeds the temperature threshold to form a larger lesion.
2) The directly heated rim rises to a higher temperature and becomes larger. It conducts more heat to the surrounding tissue to a larger boundary due to the higher temperature gradient. Thus, it raises the myocardial temperature in other regions and causes a larger lesion.
3) Higher delivered power causes the myocardial temperature to rise faster, especially in the directly heated rim. It has more time to conduct heat into the surrounding myocardium and reaches a larger boundary. This also increases the lesion dimensions.

We use FEM to simulate temperature-controlled mode catheter ablation with a target temperature of 60 °C [16]. For low flow of 1 L/min in Fig. 2(a), the maximal diameter of lesion

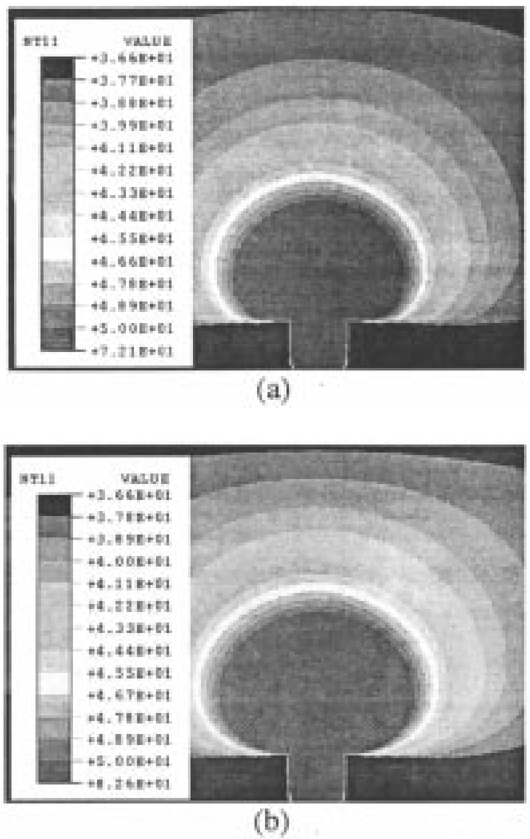


Fig. 2. FEM results of temperature-controlled catheter ablation at 60 °C with flow rate at (a) 1 L/min and (b) 3 L/min. The pictures show temperature distribution at the end of the ablation at the center cross section of the myocardium. The oval dark gray represents the lesion. The lesion size is larger under high flow than under low flow.

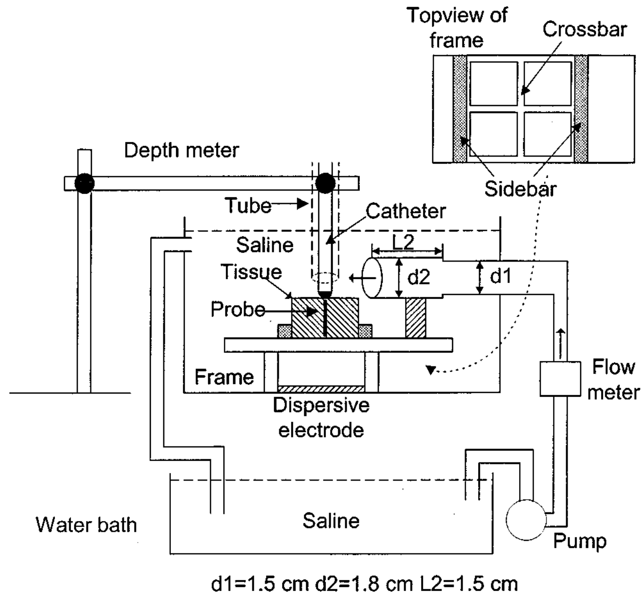


Fig. 3. The ablation system consists of a plastic container and a 12 L constant temperature water bath. The flow rate is regulated by the flow meter 0 L/min to 6 L/min. The myocardium sits on a Plexiglas frame attached to the bottom of the container. The catheter is fixed to a depth meter so that the insertion depth is well controlled.

is 7.2 mm and maximal depth is 5.2 mm. For high flow of 3 L/min in Fig. 2(b), the maximal diameter of lesion is 8.3 mm and maximal depth is 6.3 mm. Fig. 2 shows that the lesion is larger for high flow than for low flow.

III. Experimental Method

Fig. 3 shows the flow system that simulates the different flow conditions inside the heart chamber [18] for *in vitro* catheter ablation on bovine myocardium. The myocardium block sits on a Plexiglas frame immersed in a plastic container filled with 0.5% saline. The catheter, attached to a depth meter, contacts the tissue surface at a normal angle. A pump circulates the saline between the plastic container and a 12 L water bath (Model 180, Precision Scientific, Winchester, VA) to keep the saline at 37 ± 1 °C. A flow meter (7200 series, King Instrument, Huntington Beach, CA) regulates the flow rate from 0 L/min to 6 L/min. The hose injecting saline to the tissue has an 18 mm inside diameter, which corresponds to 254 mm^2 area and is comparable to the areas of the mitral and tricuspid valves. The bottom of the injecting hose aperture and the surface of myocardium are at the same level. The distance from the hose to the catheter is 20 mm. Assuming laminar flow, the maximum flow velocity at the injecting hose is 13 cm/s and 39 cm/s at the flow rates of 1 L/min and 3 L/min. We injected one drop of blue dye from the pump and used a SONY MVC-FD95 digital camera with motion picture capability to take pictures of the blue dye wavefront from the injecting hose at 25 frame/s. It shows the maximum flow velocities around the catheter are 10 cm/s and 30 cm/s. These velocities are comparable to regional blood velocity measurements at intracardiac ablation sites in the beating heart made by a doppler transducer [16].

We built a 3-thermocouple probe to measure the temperature change inside the myocardium during ablation. The IT-21 copper/constantan T-type thermocouple (Physitemp Instrument Inc., Clifton, NJ) has a time constant of 0.08 s. The thermocouple is 0.23 mm in diameter without Teflon coating and 0.41 mm with Teflon coating. We use a silver wire with Teflon coating (1 cm long and 0.25 mm in diameter with Teflon coating) as a shaft for support. The Teflon coating, as an insulator, minimizes electrical and thermal disturbance inside the issue. We group three thermocouples and the silver wire together as a probe. The thermocouple tips are 0.9, 2.0, and 3.0 mm from the shaft tip. The probe has a diameter of 0.55 mm. We introduce the probe into the tissue so that the probe tip contacts the catheter tip. In this way, we know the distances of the thermocouple from the catheter tip.

Fig. 4 shows our measurement system with the built-in thermistor at the tip of the catheter and the 3-thermocouple probe. A 12-bit analog-to-digital converter (DI-220 by Data Instruments, Akron, OH) samples the data at ten samples/s for each channel. A LabVIEW program collects the sampled data from four channels (one for the thermistor and three for the thermocouples) and saves data to storage. It converts the voltage to temperature using a calibration curve for each channel and displays the temperature changes in real-time on an IBM PC compatible 333-MHz Pentium laptop computer. With calibration, we can measure the temperature with an accuracy of 0.1 °C.

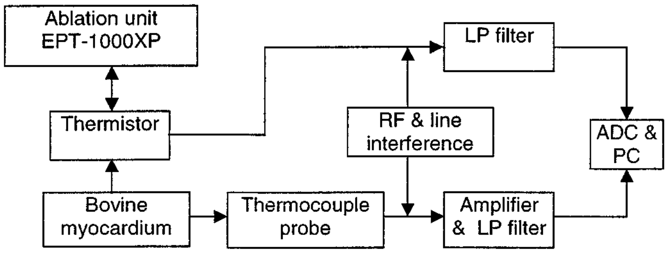


Fig. 4. The temperature measurement system contains three thermocouples inside the myocardium and a thermistor at the catheter tip.

We obtained fresh bovine heart from the local butcher shop. We cut the myocardium into 2.5 cm $\times$ 3.0 $\times$ 1.8 cm blocks. We inserted the thermocouple probe from the backside of the tissue block and placed it in the Plexiglas frame. We aligned the catheter electrode and the probe and inserted the catheter 4 mm into the myocardium for ablation. The myocardium deformed into a bowl shape as shown in Fig. 5(a). The catheter electrode surface was not totally in contact with the myocardium and about 1.5 mm was still exposed to saline.

After ablation, we dissected the myocardium through the center and then immersed it in p-nitro blue tetrazolium solution for about 10 min to distinguish the viable (blue) and nonviable (pale) tissue. The staining solution changes the color of normal cardiac tissues into dark blue while keeping the color of lesions pale. We then used a digital camera to take pictures of lesions with a ruler scale on the lesion surface as shown in Fig. 5(b). Six observers measured the dimensions independently without knowing the ablation or the flow settings. Their measurements were averaged to yield the dimension for each lesion so that we could avoid subject bias and determine the variability of the measurement. Assuming the lesion is a partial oblate ellipsoid, we derived the ablation volume using calculus as

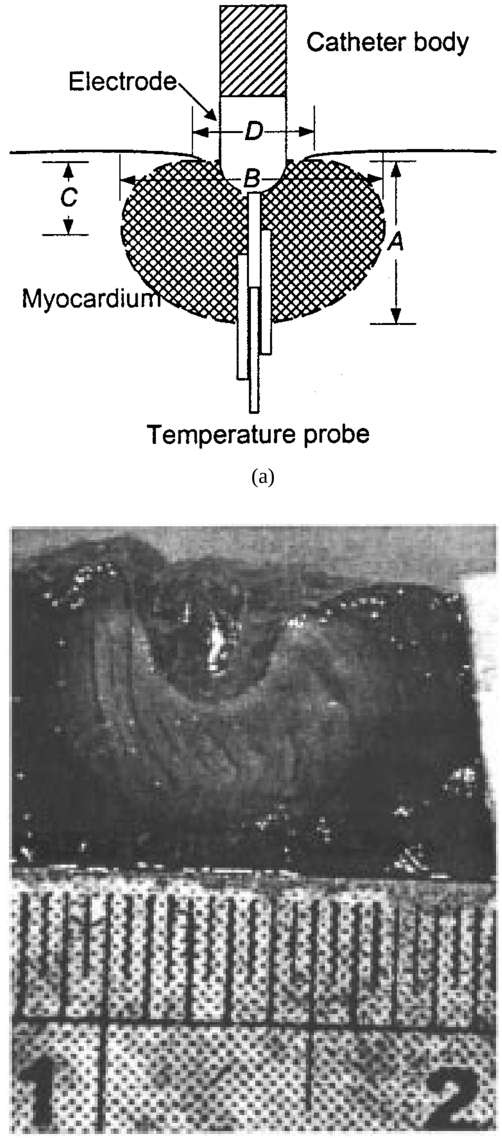
$$V_L = \frac{2}{3}\pi\left(\frac{B}{2}\right)^2 A + \frac{1}{3}\pi\left(\frac{D}{2}\right)^2 C \tag{4}$$

where

- A maximal depth;
- B maximal diameter;
- C depth at maximal diameter;
- D lesion surface diameter as shown in Fig. 5(a).

The influence of convective cooling was assessed by the t-test using Microsoft Excel. Values of $p < 0.05$ were considered statistically significantly different.

We used an EPT-1000XP ablation unit and a Blazer II 7-Fr catheter with a 2.6-mm diameter and 4-mm length electrode, both from EP Technologies (San Jose, CA). We performed *in vitro* ablation on bovine myocardium tissue with three different flow rates (0, 1, and 3 L/min) and two target temperatures (60 °C and 80 °C). For each condition, we performed six to eight ablations. The ablation unit automatically recorded the delivered power, impedance, and catheter tip temperature during ablation for analysis. We also performed temperature recording during ablation to analyze the temperature change inside the myocardium during ablation.



(a)

(b)

Fig. 5. (a) Myocardium deformation during ablation. (b) Sample of ablated myocardium after p-nitro tetrazolium blue staining. This case is 60 °C with 3 L/min. The smallest unit on the ruler is 0.5 mm. The ablation causes an electrode shaped hole in myocardium. The hole is not 4 mm deep because the myocardium bounces back a little after the catheter is removed.

IV. Results and Discussion

A. Lesion Dimension Versus Flow Rate

Table I shows the dimensions of the six lesion groups. Note that the dimensions (maximum depth, maximum diameter, and volume) of higher target temperature (80 °C) are larger than those of the lower target temperature (60 °C). This shows that the target set temperature plays a critical role in lesion dimension.

Table I also shows that at the same target temperature setting, the lesion dimension grows significantly $(p < 0.02)$ as the flow rate increases for both 60 °C and 80 °C cases. The averaged delivered power increases dramatically with flow as we analyzed in Part II. In the case of 80 °C target temperature and 3 L/min

TABLE I
LESION DIMENSION VERSUS FLOW AT 60 °C AND 80 °C

T	60 °C			80 °C		
Flow rate	0 L/min	1 L/min	3 L/min	0 L/min	1 L/min	3 L/min
Samples	6	8	8	8	8	8
MP (mm)	2.14±0.54	3.58±0.69	4.99±0.72	5.97±0.40	6.72±0.49	7.34±0.36
MD (mm)	5.88±0.27	6.38±0.46	8.17±0.63	9.04±0.56	12.0±1.1	13.5±0.4
V (mm^3)	41.3±6.9	85.5±22.7	188±46.1	278±38.4	549±107	743±67.1
P (W)	3.83±0.75	7.25±1.03	11.9±2.4	9.6±0.5	18.7±2.5	42.5±5.7
Z (Ω)	115±3	110±3	100±3	91±1	99.5±5.4	105.8±5.3

flow rate, we reached the system power limit of 50 W. The ablation generator could not provide enough power to maintain the catheter tip at the target temperature of 80 °C. During ablation, the average tip temperature was 73 °C, well below the target temperature. If we could have overcome the power limit, we could have delivered more power to the electrode and caused an even larger lesion. In clinical applications, we note that although the temperatures are the same, the final lesions may be different at various locations, dependent on the local blood flow. At high flow rate locations (such as near the mitral and tricuspid valve surface), even the low-temperature setting can cause a larger lesion. Also if we introduce high flow during ablation, we can achieve a larger lesion than in normal conditions. Thus, the saline-irrigated electrode is helpful to create larger lesions for VT, as shown by Nakagawa *et al.* [12].

The myocardial resistivity reduces 2%/°C as temperature increases [19]. Hence, the impedance between the electrode and the grounding pad decreases as more myocardium is heated up. We expect the average impedance during ablation to decrease as the lesion dimension increases and temperature increases. This is true for the 60 °C cases as shown in Table I. The 60 °, C 3 L/min case has the largest delivered power and lesion dimension in the three cases, and its average impedance is the lowest during ablation. For the 80 °C cases, we found there was slight tissue dehydration for the 1-L/min case and significant tissue dehydration at 3 L/min on the surface of the myocardium. When the temperature is high enough, such as 80 °C, it causes tissue dehydration in the myocardium. The dehydrated tissue is an electrical insulator and it results in higher impedance. This explains why the average impedance is the highest for 3 L/min at 80 °C cases, compared to the 60 °C cases.

B. Temperature Recording

We recorded the catheter tip temperature and myocardial temperature at three locations (0.9, 2.0, and 3.0 mm from the catheter tip) during ablation and after the ablation stopped. Fig. 6 shows typical recordings for each group. The patterns of temperature change during ablation are similar except for the 80 °C, 3 L/min case. The catheter tip temperature $(T0)$ rises quickly at the beginning of ablation and reaches the target temperature in about 3–5 s. Then it fluctuates around the target temperature due to the power adjustment of the ablation unit.

The TC1, TC2, and TC3, three temperatures from thermocouples at fixed locations, increase as the flow rate increases. Table II shows the time (t_{50}) for which they reach 50 °C, the lesion threshold and the maximum temperature (T_m) at the end of ablation. It shows that the higher blood flow causes the myocardial temperature to rise faster and reach a higher temperature, just as we analyzed in Part II. Taking 60 °C as an example, in the 0 L/min cases, TC2 merely reaches 50 °C around 50 s and TC3 never reaches that. With only 10 s above the lesion threshold, the tissue gets ablated and the lesion boundary just reaches TC2 (2.0 mm from the catheter tip), which is close to the maximum depth for the 0 L/min case (2.14 mm as in Table I). As for the 1 L/min case, TC3 (3.0 mm from the catheter tip) reaches 50 °C 46 s after ablation with a maximum temperature of 51 °C. So the lesion boundary is a little bit outside this position (3.0 mm) at 3.6 mm. In the 3 L/min case, TC3 reaches the threshold much earlier and has a higher maximum temperature. This causes a larger lesion (5.0-mm depth) than in the 1 L/min case.

Fig. 6(c) (60 °C, 3 L/min) and 6(e) (80 °C, 1 L/min) also show that T_m of TC1 is much higher than the catheter tip temperature. TC1 is higher than the target temperature for about 40 s in 6(c). In 6(e), it is higher for about 30 s. As discussed in the numerical analysis in [9], the highest temperature point during ablation is in the myocardium, about 0.5–1 mm underneath the catheter tip, instead of at the catheter tip. It indicates that the temperature monitoring is especially important at high flow locations such as at the mitral valve and tricuspid valve. The myocardial temperature inside may be several degrees higher than the catheter tip temperature. Physicians should not set the target temperature too high (say 80 °C), which may cause charring inside the myocardium.

C. 80 °, C 3 L/min Case

The ablation at 80 °C and 3 L/min is different from other cases. Fig. 6(f) shows the catheter tip temperature rises at first to 80 °C. Then it keeps decreasing although the myocardial temperature (TC1, TC2, and TC3) increases. We noted that the ablation generator kept increasing the delivered power until it reached the maximal allowed power (50 W) and the impedance was higher than other 80 °C cases during ablation. Also during ablation, the system impedance first went down as the ablation started because of tissue heating. Then it started to increase after the catheter reached 80 °C as shown in Fig. 7(d). Visual inspection showed that there was tissue dehydration at the catheter tip.

For normal cases, the electric current dissipates into the myocardium uniformly as shown in Fig. 7(a). The myocardium underneath the thermistor is heated by local Joule heat during the ablation. The ablation generator adjusts delivered power

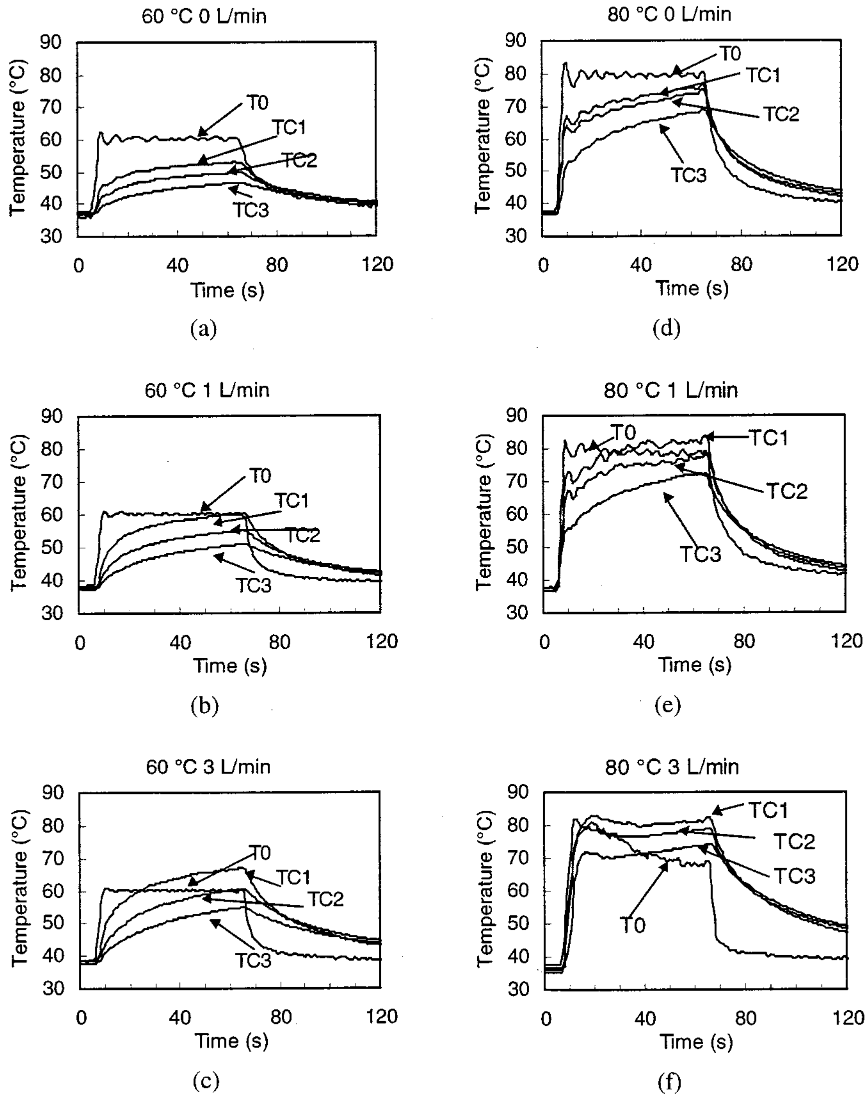


Fig. 6. Temperature recordings during catheter ablation. $T0$ is catheter tip temperature. TC1, TC2, and TC3 are temperature recordings of thermocouples, which are 0.9, 2.0, and 3.0 mm from the catheter tip.

TABLE II
TIME FOR THE THREE THERMOCOUPLE TEMPERATURES TO REACH THE 50 °C THRESHOLD

	0 L/min		1 L/min		3 L/min	
60 °C	t_{50}	T_m	t_{50}	T_m	t_{50}	T_m
TC1	20±2	53±2	8±1	60±3	8±1	67±5
TC2	50±4	~50*	20±2	55±2	16±1	60±3
TC3	NA	46±1	46±4	51±2	30±2	55±3
80 °C	t_{50}	T_m	t_{50}	T_m	t_{50}	T_m
TC1	7±0.9	76±2	6±0.3	83±2	5±0.3	81±4
TC2	7±1	73±2	7±0.4	78±3	6±0.5	71±2
TC3	9±1	68±1	7±1	72±2	7±1	64±2

to maintain the myocardium there to the target temperature. Hence, the delivered power decreases after the target temperature is achieved [Fig. 7(b)]. Also the impedance decreases as the myocardium surrounding the catheter electrode is heated up.

For the 80 °, C 3 L/min case, the high power (50 W) causes high current density and Joule heat generation at the tissue just around the catheter electrode. The myocardium at the interface reaches a high temperature and causes a thin layer of tissue dehydration around the location of thermistor, where the highest temperature is. The dehydrated myocardium is an electrical insulator and, thus, the impedance increases. The electric current bypasses that region to heat the myocardium underneath [Fig. 7(c)]. The dehydrated myocardium is also a thermal insulator and isolates the thermistor from the heated myocardium underneath. The thermistor temperature starts decreasing through the heat leakage to the catheter electrode. The catheter electrode is at a lower temperature due to convection and, thus, the thermistor temperature keeps decreasing during ablation. As a result, the control system assumes the catheter tip temperature is lower than the target temperature and increases the delivered power from the ablation generator. The tissue thermocouple temperatures TC1, TC2, and TC3 rise faster to a higher plateau compared with the correspondents in the 1 L/min

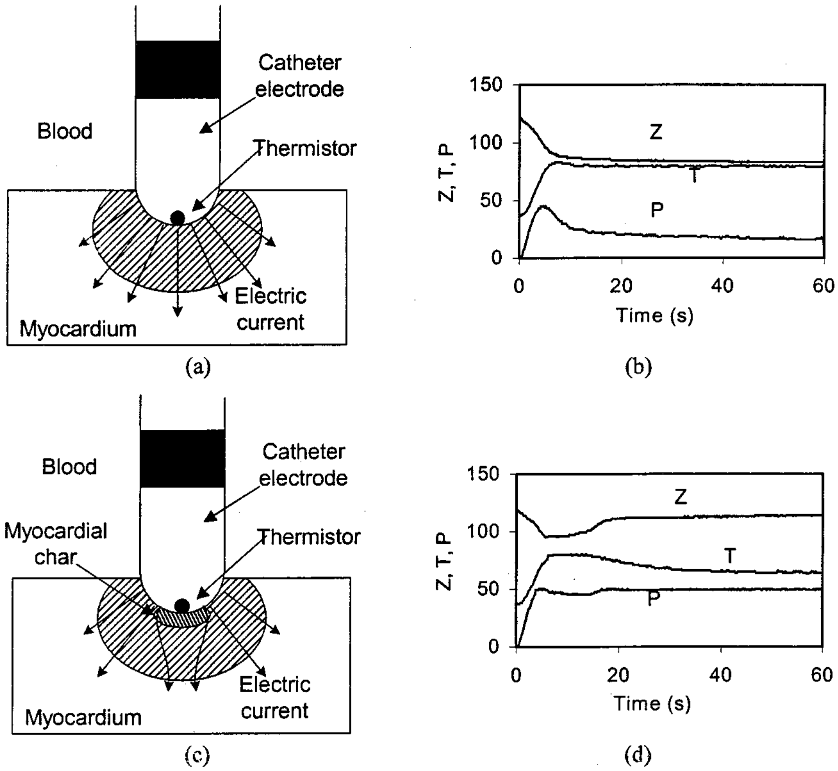


Fig. 7. Electric current pattern, catheter tip temperature, impedance, and power during ablation. (a) and (b) are for 80 °C, 1 L/min. (c) and (d) are for 80 °C, 3 L/min. Other cases are similar to (a) and (b). In (c), the myocardium char blocks the catheter tip. It increases the impedance and decreases catheter tip temperature during ablation.

case because of thermal conduction and the high power output (average 42.5 W) results in the largest lesion, although the tip temperature $(T0)$ is low.

V. Conclusion

This study investigated the blood flow effect on lesion formation during RF cardiac catheter ablation. With lesion measurement and temperature recording inside the myocardium, we demonstrated that lesion dimensions and tissue heating are dependent on local flow conditions for a given tip temperature during temperature-controlled RF ablation. In general, with high flow rates and local blood velocities, lesions are larger and tissue temperatures rise faster compared with low local flow rates. Moreover, the recorded catheter tip temperature, which is the only available temperature information to the operator in the clinical setting, does not always reflect these findings.

References

[1] S. K. S. Huang and D. J. Wilber, Eds., *Radiofrequency Catheter Ablation of Cardiac Arrhythmias: Basic Concepts and Clinical Applications*, 2nd ed. Armonk, NY: Futura, 2000.

[2] D. P. Zipes, *Catheter Ablation of Arrhythmias*. Armonk, NY: Futura, 1994.

[3] F. Morady, “Drug therapy: Radio-frequency ablation as treatment for cardiac arrhythmias,” *N. Eng. J. Med.*, vol. 340, no. 7, pp. 534–544, Feb. 1999.

[4] D. Panescu, “Intraventricular electrogram mapping and radiofrequency cardiac ablation for ventricular tachycardia,” *Physiol. Meas.*, vol. 18, pp. 1–38, Feb. 1997.

[5] H. Nakagawa, K. J. Beckman, and J. H. McClelland, “Radiofrequency catheter ablation of idiopathic left ventricular tachycardia guided by a Purkinje potential,” *Circulation*, vol. 85, pp. 1666–1674, Dec. 1992.

[6] S. Nath, C. Lynch, J. G. Whayne, and D. E. Haines, “Cellular electrophysiological effects of hyperthermia on isolated guinea pig papillary muscle implications for catheter ablation,” *Circulation*, pt. 1, vol. 88, pp. 1826–1831, Oct. 1993.

[7] D. E. Haines, “The biophysics of radiofrequency catheter ablation in the heart: The importance of temperature monitoring,” *PACE*, vol. 16, pp. 596–591, Mar. 1993.

[8] D. E. Haines and D. D. Watson, “Tissue heating during radiofrequency catheter ablation: A thermodynamic model and observation in isolated perfused and superfused canine right ventricular free wall,” *PACE*, vol. 12, pp. 962–976, June 1989.

[9] D. Panescu, J. G. Whayne, S. D. Fleischman, M. S. Microtznik, D. K. Swanson, and J. G. Webster, “Three-dimensional finite element analysis of current density and temperature distributions during radio-frequency ablation,” *IEEE Trans. Biomed. Eng.*, vol. 42, pp. 879–890, Sept. 1995.

[10] H. H. Peterson, X. Chen, A. Pietersen, J. H. Svendsen, and S. Haunsø, “Lesion dimensions during temperature-controlled radiofrequency catheter ablation of left ventricular porcine myocardium: Impact of ablation site, electrode size and convective cooling,” *Circulation*, vol. 99, pp. 319–325, Jan. 1999.

[11] J. J. Lamberg, M. Gallagher, S. A. Strickberger, and O. Amirana, “Temperature-guided radiofrequency catheter ablation with very large distal electrodes,” *Circulation*, vol. 88, pp. 245–249, July 1993.

[12] H. Nakagawa, W. S. Yamanashi, J. V. Pitha, M. Arruda, X. Wang, K. Ohtomo, K. J. Beckman, J. H. McClelland, R. Lazzara, and W. M. Jackman, “Comparison of *in vivo* tissue temperature profile and lesion geometry for radiofrequency ablation with a saline-irrigated electrode versus temperature control in a canine thigh muscle preparation,” *Circulation*, vol. 91, pp. 2264–2273, Apr. 1995.

[13] S. Labonté, “Numerical model for radio-frequency ablation of the endocardium and its experimental validation,” *IEEE Trans. Biomed. Eng.*, vol. 41, pp. 108–115, Feb. 1994.

[14] Z. Kaouk, A. Khebir, and P. Savard, “A finite element model of a microwave catheter for cardiac ablation,” *IEEE Trans. Microwave Theory Tech.*, vol. 44, pp. 1848–1854, Oct. 1996.

[15] K. Hynynen, J. Dennie, J. Zimmer, W. Simmons, D. He, F. Marcus, and M. Aguirre, "Cylindrical ultrasonic transducers for cardiac catheter ablation," *IEEE Trans. Biomed. Eng.*, vol. 44, pp. 144–151, Feb. 1997.

[16] S. Tungjitkusolmun, V. R. Vorperian, H. Cao, J.-Z. Tsai, and J. G. Webster, "Guidelines for predicting lesion size at several endocardial locations during radio-frequency ablation," *IEEE Trans. Biomed. Eng.*, pp. 194–201, Feb. 2001.

[17] M. K. Jain and P. D. Wolf, "Temperature controlled and constant-power radio-frequency ablation: What affects lesion growth?," *IEEE Trans. Biomed. Eng.*, vol. 46, pp. 1405–1412, Dec. 1999.

[18] H. Cao, V. R. Vorperian, J.-Z. Tsai, S. Tungjitkusolmun, E. J. Woo, and J. G. Webster, "Temperature measurement within myocardium during *in vitro* RF catheter ablation," *IEEE Trans. Biomed. Eng.*, vol. 47, pp. 1518–1524, Nov. 2000.

[19] K. R. Foster and H. P. Schwan, "Dielectric properties of tissues and biological materials: A critical review," *CRC Crit. Rev. Biomed. Eng.*, vol. 17, pp. 25–104, Jan. 1989.

Hong Cao (S'97) received the B.S.E.E. and M.S.E.E. degrees from Nanjing University, Nanjing, China, in 1992 and 1995, respectively. He is currently a Ph.D. candidate in the Department of Electrical and Computer Engineering at the University of Wisconsin-Madison.

His research interests include temperature measurement inside myocardium during RF catheter ablation and optimization of catheter design. He is contributing author to *Minimally Invasive Medical Technology* (J. G. Webster, Ed., Bristol, U.K.: IOP, 2001).

Vicken R. Vorperian received the B.S. degree in biology and chemistry in 1979 and the M.D. degree in 1985, both from the American University of Beirut, Lebanon. He then completed the following postgraduate medical clinical training programs: internship and residency in the specialty of internal medicine from 1985–1988 at the Sinai Hospital of Baltimore, Baltimore, MD; fellowship in the subspecialty of cardiology from 1988–1990 at the Brooklyn Hospital, Brooklyn, NY; fellowship in cardiac arrhythmias and electrophysiology at the Vanderbilt University, Nashville, TN; fellowship in cardiac pacing and catheter ablation at the University of Michigan, Ann Arbor, MI. He received his Board Certification in the specialty of Internal Medicine in 1988, in Cardiology in 1991, and in Clinical Cardiac Electrophysiology in 1994 from the American Board of Internal Medicine.

He is an Associate Scientist in the cardiac RF ablation project in the Department of Biomedical Engineering. He holds the title of Clinical Associate Professor of Medicine in the section of Cardiology, Department of Medicine, University of Wisconsin-Madison and is an Attending Electrophysiologist in the Cardiac Electrophysiology Laboratory at the University of Wisconsin Hospital-Madison. He is also a Member of the Arrhythmia Consultants of Milwaukee S.C., a private practice group in cardiac arrhythmias and clinical cardiac electrophysiology. His research interests in cardiac electrophysiology include the design of experimental setups to address questions about different aspects of RF catheter ablation of arrhythmias in the clinical setting. His research interests also include the clinical and basic electrophysiologic properties of newer and developmental antiarrhythmic agents.

Supan Tungjitkusolmun (S'96–M'00) was born in Bangkok, Thailand, on December 5, 1972. He received the B.S.E.E. degree from the University of Pennsylvania, Philadelphia, PA, in 1995, and the M.S.E.E. and Ph.D. degrees from the University of Wisconsin, Madison, WI, in 1996, and 2000, respectively.

He is on the faculty of the Department of Electronics Engineering, King Mongkut's Institute of Technology Ladkrabang (KMITL), Bangkok, Thailand. His research interests include finite element modeling, radio-frequency cardiac ablation, and hepatic ablation. He is contributing author to *Design of Pulse Oximeters* (J. G. Webster Ed., Bristol, UK: IOP, 1997) and to *Minimally Invasive Medical Technology* (J. G. Webster, Ed., Bristol, U.K.: IOP, 2001).

Dr. Tungjitkusolmun was a recipient of the Royal Thai Government Scholarship from 1990 to 2000. He is a member of Tau Beta Pi, Eta Kappa Nu, and Pi Mu Epsilon.

Jang-Zern Tsai (S'97) was born in Chia-Yi, Taiwan, in 1961. He received the B.S.E.E. degree from National Central University, Chung-Li, Taiwan, in 1984, and the M.S.E.E. degree from National Tsing Hua University, Hsinchu, Taiwan, in 1986. In 1986, he joined the Electronics Research and Service Organization (ERSO) of the Industrial Technology Research Institute (ITRI) as an integrated circuit applications engineer. In 1990, he joined the Computer and Communication Research Laboratories (CCL) of ITRI and worked as a Hardware Engineer on digital recording technology. In 1994, he joined the Accton Technology Corporation as a Computer Network Software Engineer. All companies are located in Taiwan. He is currently a Ph.D. student in the Department of Electrical and Computer Engineering, University of Wisconsin at Madison, WI, doing research on radio-frequency cardiac ablation. His current research interest is myocardial resistivity measurement.

Dieter Haemmerich (S'00) was born in Vienna, Austria on May 22, 1971. He received the B.S.E.E. degree from the Technical University of Vienna in 1997 and the M.S.B.M.E. from the University of Wisconsin, Madison, in 2000. He is currently a Ph.D. degree candidate in the Department of Biomedical Engineering, University of Wisconsin, Madison.

His research interests include finite element analysis of radio-frequency ablation and tissue impedance measurement.

Young Bin Choy (S'00) was born in Seoul, Korea, on Aug. 17, 1976. He received the B.S. degree in 1999 from the School of Electrical Engineering of Seoul National University. He received the M.S.E.E. degree from the University of Wisconsin-Madison in 2000, where he is currently a Ph.D. degree candidate.

He was an Engineer in LG Corporate Institute of Technology from January 1999 to June 1999. He was a Teaching Assistant in the Medical Instrumentation Laboratory at the University of Wisconsin-Madison, in fall, 2000. His research is measuring the mechanical compliance of the endocardium to determine the relation between the penetration depth of the RF cardiac catheter ablation electrode and the force.

John G. Webster (M'59–SM'69–F'86–LF'97) received the B.E.E. degree from Cornell University, Ithaca, NY, in 1953, and the M.S.E.E. and Ph.D. degrees from the University of Rochester, Rochester, NY, in 1965 and 1967, respectively.

He is Professor of Biomedical Engineering at the University of Wisconsin-Madison. In the field of medical instrumentation he teaches undergraduate and graduate courses, and does research on RF cardiac ablation.

He is author of *Transducers and Sensors, An IEEE/EAB Individual Learning Program* (Piscataway, NJ: IEEE, 1989). He is coauthor, with B. Jacobson, of *Medicine and Clinical Engineering* (Englewood Cliffs, NJ: Prentice-Hall, 1977), with R. Pallás-Areny, of *Sensors and Signal Conditioning*, 2nd ed. (New York: Wiley, 2001) and with R. Pallá-Areny, of *Analog Signal Processing* (New York: Wiley, 1999). He is Editor of *Encyclopedia of Medical Devices and Instrumentation* (New York: Wiley, 1988), *Tactile Sensors for Robotics and Medicine* (New York: Wiley, 1988), *Electrical Impedance Tomography* (Bristol, UK: Adam Hilger, 1990), *Teaching Design in Electrical Engineering* (Piscataway, NJ: Educational Activities Board, IEEE, 1990), *Prevention of Pressure Sores: Engineering and Clinical Aspects* (Bristol, UK: Adam Hilger, 1991), *Design of Cardiac Pacemakers* (Piscataway, NJ: IEEE Press, 1995), *Design of Pulse Oximeters* (Bristol, UK: IOP Publishing, 1997), *Medical Instrumentation: Application and Design*, Third Edition (New York: Wiley, 1998), *Handbook of Measurement, Instrumentation, and Sensors* (CRC Press, Boca Raton, FL, 1999), *Encyclopedia of Electrical and Electronics Engineering* (New York: Wiley, 1999), *Mechanical Variables Measurement* (CRC Press, Boca Raton, FL, 2000), and *Minimally Invasive Medical Technology* (Bristol, U.K.: IOP, 2001). He is Coeditor, with A. M. Cook, of *Clinical Engineering: Principles and Practices* (Englewood Cliffs, NJ: Prentice-Hall, 1979) and *Therapeutic Medical Devices: Application and Design* (Englewood Cliffs, NJ: Prentice-Hall, 1982), with W. J. Tompkins, of *Design of Microcomputer-Based Medical Instrumentation* (Englewood Cliffs, NJ: Prentice-Hall, 1981) and *Interfacing Sensors to the IBM PC* (Englewood Cliffs, NJ: Prentice Hall, 1988), and with A. M. Cook, W. J. Tompkins, and G. C. Vanderheiden, of *Electronic Devices for Rehabilitation* (London: Chapman & Hall, 1985).

Dr. Webster has been a Member of the IEEE-EMBS Administrative Committee and the NIH Surgery and Bioengineering Study Section. He is a Fellow of the Instrument Society of America, the American Institute of Medical and Biological Engineering, and the Institute of Physics. He is the recipient of the AAMI Foundation Laufman-Greatbatch Prize, the ASEE/Biomedical Engineering Division, Theo C. Pilkington Outstanding Educator Award and the ASEE/Engineering Libraries Division, Best Reference Work Award.