Laser probe ablation of normal and atherosclerotic human aorta in vitro: a first thermographic and histologic analysis

ASHLEY J. WELCH, PH.D., ARLENE B. BRADLEY, M.D., JORGE H. TORRES, M.D., MASSoud MOTAMEDI, M.S., JOHN J. GHIDONI, M.D., JOHN A. PEARCE, PH.D., HANY HUSSEIN, PH.D., and ROBERT A. O’ROURKE, M.D.

ABSTRACT The metal-tipped optical fiber or “laser probe” has been extensively studied in animal preparations in vivo and in human clinical trials of revascularization. The aim of this study was to evaluate the thermal characteristics of laser probe tissue ablation and to contrast the vascular tissue response to exposure to the laser probe and bare optical fiber. A 2 mm laser probe was heated with up to 4 W of argon-ion laser irradiation and applied to six postmortem strips of human nonatherosclerotic aorta as well as to five atherosclerotic aortic specimens. Surface temperature maps of the laser probe and of the vascular tissue in air were obtained via 8 to 12 μm thermographic imaging. Laser probe temperature was additionally monitored via thermocouples. Two strips each of normal and diseased aorta were irradiated directly with the bare optical fiber. Thus a total of 43 laser probe application sites and 19 bare fiberoptic laser irradiation sites on a total of 15 aortic strips were analyzed both thermographically and histologically. Based on measured temperature rises and histologic findings, the following observations were made: (1) The laser probe heats initially at its tip and attains a uniform surface temperature distribution within 5 sec. The steady-state temperature attained by the probe is inversely related to the thermal conductivity of the surrounding media. In all media studied, probe temperature increases linearly with applied laser energy. (2) Tissue ablation starts at temperatures greater than 100°C, and ablation temperatures typically exceed 180°C. Adventitial temperatures during laser probe application may reach 70°C. Tissue ablation is enhanced both by greater laser energy deposition in the probe and by higher force at which the probe is applied to tissue. (3) Ablation of fibrofatty atheromata is more extensive than of nonatherosclerotic aortic tissue. This may be due to the lower thermal conductivity of atheromatous tissue. (4) In contrast to direct argon-ion laser ablation of aortic tissue, laser probe–mediated ablation occurs in a controlled fashion, is not associated with extensive subintimal dissections, and allows uniform conduction of heat to tissue as reflected by essentially “isothermal” injury lines.


ATHEROSCLEROTIC arterial occlusive disease continues to constitute the major cause of morbidity and mortality in the United States. Over the past 4 years, substantial work has been done in an attempt to develop a laser technology that can be applied to the percutaneous revascularization of obstructive peripheral vascular and coronary artery disease. In contrast to conventional bypass surgery or balloon angioplasty, laser angioplasty has the potential benefit of allowing the actual removal of obstructing atheromatous plaque or thrombus. This might permit the more effective percutaneous transluminal revascularization of severely stenosed, diffusely diseased, or totally occluded peripheral and coronary arteries.

To date, optical fiber–mediated laser applications to vascular tissue have been complicated by a high incidence of vessel perforation coupled with a limited reduction in stenosis severity. However, recently the “laser probe,” i.e., a quartz optical fiber capped with a tapered metallic tip, has been shown to be more effective in restoring vascular luminal patency. In contrast to a regular optical fiber that transmits the laser beam directly to tissue, the laser probe used in our
investigations confines the laser energy to the metal tip as heat. This allows the controlled conductive transfer of heat to the vessel wall. Although the laser probe has been used extensively in short- and long-term animal experiments and has more recently been introduced in the treatment of human peripheral and coronary arterial disease, thermal analysis of laser probe tissue ablation has not been described to date. The aims of this study were therefore to (1) determine the thermal characteristics of the metal-tipped laser probe, (2) measure the temperature changes and the extent of histologic damage produced by the heated probe on normal and atherosclerotic aortic tissue, and (3) contrast the effects of the laser probe as a contact heat source with those of direct laser irradiation on the same types of tissue.

**Methods**

**General procedure and experimental configuration.** Normal and atherosclerotic human aortic tissue was obtained without identifiers from the office of the medical examiner of Travis County and the autopsy service of the University of Texas Health Science Center at San Antonio. The tissues were used immediately or stored in an iced normal saline bath for 24 hr before an experiment. Fifteen vessel strips (eight without and seven with atheromatous disease) were used in this study. In the diseased aortas, only white, firm plaques were selected for laser probe application. Complex, grossly calcific, and ulcerated atheromata were excluded. Calcified plaques were avoided in this study in view of their histologic heterogeneity.

In all experiments, a Spectra-Physics argon-ion laser (Series 2000, Model 2020-05) with primary wavelengths of 488 and 514.5 nm was used to heat the probes or to directly irradiate the tissue at a 700 μm spot size. Laser power was measured with a Model RT-150C power meter from Laser Precision Corp.

The laser probes were either purchased or supplied by Trimedyne, Inc. The probes had a 300 μm silica core optical fiber and a 2 mm wide metallic ellipsoid tip. The bare optical fiber used in the experiments had characteristics identical to the metal-capped fibers and were supplied by the same company. All fibers and laser probes had a standard proximal coupling facilitating fiber exchange without realignment of the optics. The power delivered to the laser probe was determined by measuring the power transmitted to the distal end of an identical bare optical fiber. The laser power delivered ranged from 0.5 to 4 W. The responses of four probes were tested initially in three different media (air, water, and glycerin) at identical energies to ensure the reproducibility of probe behavior.

The experimental configuration is illustrated in figure 1. A moistened strip of aorta measuring 1 × 8 cm was placed horizontally with its intimal surface exposed on a flat sheet of cork mounted on a movable ruler. Both direct laser irradiation and the laser probe were directed vertically at the vessel surface. The laser probe, suspended at a known weight, was allowed to rest freely on the intimal vessel surface at constant force. To study the effects of pressure during application of the laser probe, suspension devices of different weights were used to alter the force applied.

**Correlation of probe and tissue temperatures with histologic results.** Laser probe temperature was measured via two methods: thermocouple measurements and thermography. In the first instance, a 127 μm wire Chromel-Alumel thermocouple with a 350 μm bead was placed into the guidewire channel. The distal opening of the channel was sealed with Omega high-temperature cement, and, where appropriate, the proximal opening of the channel was sealed as well. The reference temperature for the thermocouple was obtained with another Chromel-Alumel thermocouple placed in an ice-water bath. A Tektronix AM 502 differential amplifier amplified the electrical signal derived from the thermocouples for recording. Simultaneously, a Tektronix DM 501A digital multimeter was used to corroborate the maximum voltages recorded. Based on the high thermal conductivity of the probe supplied by the manufacturer (0.172 W/cm − °C), we made the assumption that laser probe temperature was uniform throughout. This assumption was vali-

![FIGURE 1. Experimental configuration to determine simultaneous laser probe and tissue surface temperatures for probe applied vertically to tissue at constant pressure.](image-url)
dated by comparing thermocouple measurements of laser probe temperature with laser probe temperature maps obtained via the thermal camera. The thermocouples (Omega Engineering, Inc.) generate a voltage difference of 0.040 mV for every 1°C change in temperature. In our previous experience their readings have been found to be accurate and reproducible.

Thermographic imaging was used to determine the surface temperature of both the laser probe and the tissue in air. An 8 to 12 μm thermal camera (Model 525 Inframetrics, Inc.) was used to detect the thermal radiation. For calibration, two black bodies of known temperature were placed in close proximity to the object being monitored. Images of the measured surface radiosity were recorded on videotape at 30 frames/sec during laser application, digitized on a programmed Intel computer, and processed to obtain the final thermographs. Thermographic imaging of the laser probe required covering the metal with black paint to render its thermal emissivity sufficiently close to 1.0. On the other hand, vascular tissue has thermal emissivity values close to 1 (0.93 to 0.99 according to our measurements) requiring no further manipulations.

After tissue ablation, the samples were examined under a dissecting surgical microscope to determine the diameters and depths of the lesions. The samples were fixed in a solution of buffered formalin for subsequent preparation of hematoxylin and eosin–stained sections and histologic examination.

To determine tissue-temperature profiles, tissue temperature was plotted as a function of the distance from the edge of the probe delineated in the thermographic image. Actual distances from the probe were determined with reference to the known separation between the black bodies. In view of the shrinkage that occurs during tissue fixation for histologic study, the crater dimensions determined under the dissecting surgical microscope after probe application were compared with the dimensions observed on the histologic sections for calculations of correction factors.

Forty-three laser probe applications and 19 direct laser irradiation sites on a total of 15 aortic strips were analyzed both thermographically and histologically. Because of the complexity of thermographic image processing, only a limited number of thermographs could be analyzed per experiment, and statistical analysis of the results was therefore not feasible.

Results

Thermal characterization of the laser probe

Thermal response of the laser probe. The argon-ion laser was used in all the experiments. Distribution of temperature of the laser probe surface after 1.4 W of argon-ion laser irradiation of the probe for 2 sec in air is shown in figure 2. At this power, the maximum temperature of the laser probe surface was 129.5°C at 2 sec and 225°C at 5 sec. Interestingly, the temperature rose sooner and higher at the tip of the probe, decreasing toward the probe neck, as seen in figure 2 at 2 sec. At this power, within 5 sec the temperature distribution of the probe was nearly uniform.

Temperature measurements of the probe by the thermal camera were compared with those obtained by thermocouple for five different laser powers. Excellent agreement between the two measurement systems is noted in table 1. The two measurement methods correlated within 10°C. Three determinations were done for each power and exposure setting with nearly identical results.

The rate and extent of temperature elevation of the probe was tested in five different media: air, water, glycerin, blood, and tissue (beef steak) for differing laser powers at fixed exposure durations. Two hundred milliliters of water, glycerin, and blood were used. Blood was obtained from a local blood bank and had a hematocrit of 45%. A plot of probe temperature (°C) vs power (W) delivered to the probe in 5 sec pulses is presented in figure 3. Clearly, probe temperature increased nearly linearly with delivered laser power. The probe temperature attained at 5 sec decreased as the thermal conductivity of the surrounding media (table 2) increased. During the 5 sec pulse, probe temperature achieved steady state only in water. The temperatures reached by the probe in blood exceeded those in water. This difference was more marked as laser power applied to the probe exceeded the range shown here.

Simultaneous laser probe and tissue temperatures. Probe and tissue (normal aorta) temperatures as a function of exposure time varying from 1 to 5 sec at constant laser

TABLE 1

<table>
<thead>
<tr>
<th>Power at tip (W)</th>
<th>Exposure time (sec)</th>
<th>Temperature sensed by thermocouple (°C)</th>
<th>Temperature detected by camera (°C)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.21</td>
<td>90</td>
<td>152</td>
<td>150–160</td>
</tr>
<tr>
<td>0.35</td>
<td>90</td>
<td>222</td>
<td>225–237</td>
</tr>
<tr>
<td>0.72</td>
<td>5</td>
<td>130</td>
<td>130–140</td>
</tr>
<tr>
<td>1.05</td>
<td>5</td>
<td>175</td>
<td>170–180</td>
</tr>
<tr>
<td>1.40</td>
<td>5</td>
<td>219</td>
<td>216–228</td>
</tr>
</tbody>
</table>
power (4 W) are shown in figure 4. The points correspond to five different applications of the probe in 1 to 5 sec pulses. Clearly, both probe and tissue temperature increased linearly with exposure time. However, the peak laser probe temperature attained was 410° C in contrast to the peak tissue temperature of only 184° C. Probe temperatures consistently exceeded tissue temperatures. The probe was noted to adhere to tissue between probe temperatures of 100° and 180° C.

Thermal and histologic findings during ablation of aorta

Correlation of histologic thermal injury with tissue temperatures. The histologic response of normal vessel wall to thermal injury has been previously described and generally consists of sharply demarcated zones as shown in figure 5: (1) normal aortic media, (2) a narrow zone of “coagulation” with increased eosinophilia, cellular shrinkage, and pyknotic nuclei, (3) an extensive zone of tissue vacuolization, (4) shrinkage, “melting” of surface elastic laminae, (5) precarbonization and carbonization at the leading edge of tissue evaporation, and (6) tissue loss caused by ablation.

Tissue-temperature correlations were made for 22 histograms of laser probe applications to normal aorta and the corresponding thermographic images. In general, at temperatures greater than 60° C, changes consistent with gradual dehydration and cellular shrinkage (“coagulation”) were seen. Vacuolization of tissue with production of multiple small cavities occurred at temperatures exceeding 120° C. Also, loose basophilic extracellular material accumulated adjacent to elastic laminae. Ablation occurred at temperatures exceeding 180° C.

Interestingly, tissue temperatures greater than 50° C—a temperature associated with protein denaturation—were seen at radial distances exceeding 1.0 mm from the edge of probe.

A distinct difference in the ablation pattern between plaque and normal aortic tissue is illustrated in figure 6, which was obtained after exposure to fibrofatty plaque to the laser probe (4 W, 10 sec). Penetration of the probe from the thickened, diseased neointima into normal media caused a visibly different ablation pattern of fibrofatty atheroma relative to the nonatherosclerotic aortic tissue. A trend toward more effective laser probe ablation of fibrofatty plaque vs nonatherosclerotic aortic tissue is also evident in table 3.

Adventitial surface temperature during laser probe application. In these experiments the laser probe was sandwiched between the intimal surfaces of two strips of normal aortic tissue to simulate intravascular insertion of the probe. Laser probe temperature was monitored by a thermocouple in the guidewire channel, whereas thermographic imaging was performed of the corresponding adventitial vessel surface. The temperature image obtained after applying argon-ion laser irradiation (4 W, 10 sec) to the laser probe is shown in figure 7. The adventitia attained a temperature of 70° C in a sample with a wall thickness of 1.3 mm. Only minimal coagulative damage was seen in the areolar adventitial tissue. Applying the probe under identical conditions (4 W) for only 5 rather than 10 sec resulted in an adventitial temperature of only 45° C (vs 70° C).
Histologic features of aortic tissue ablation

Relationship between the energy delivered to the probe and tissue ablation. Increasing the energy deposited in the metal probe by applying the laser probe perpendicularly to the vessel intima at increasing power for fixed 5 sec pulses resulted in greater depth of the ablation craters. Similar results were seen upon increasing the exposure duration to the probe at a fixed irradiation power.

Effect of the force applied to the probe on tissue ablation. To examine whether increasing the force applied by the laser probe on tissue would increase its effectiveness in ablating tissue, the probe was applied with forces of two different magnitudes, 95 and 182 millinewtons, to nonatherosclerotic as well as to atheromatous aortic tissue. In all instances, 4 W was delivered to the probe for 10 sec. It is apparent from table 3 that increasing the applied force resulted in both a larger crater diameter as well as a greater crater depth. Histologically, the effect of pressure could also be observed. Whereas the process of vascuolization in figure 8, A, in the absence of applied force resulted in virtually parallel residual elastic laminae, the application of force to the probe in figure 8, B, has in contrast caused marked compression of the residual elastic laminae against one another. This collapse is most marked at the tip of the probe.

Histologic comparison of bare optical fiber- vs laser probe-mediated tissue ablation. Continuous-wave laser ablation of tissue occurs via different mechanisms than metal probe ablation. Nevertheless, it was of interest to contrast the histologic findings obtained after tissue irra-
TABLE 3
Effect of the force applied to the probe on tissue ablation

<table>
<thead>
<tr>
<th>Type of tissue</th>
<th>Tissue thickness (mm)</th>
<th>Probe temp. (°C)</th>
<th>Crater diameter (mm)</th>
<th>Crater depth (mm)</th>
<th>Force = 182 mN</th>
<th>Force = 95 mN</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>1.6</td>
<td>527</td>
<td>2.0</td>
<td>1.5</td>
<td>464</td>
<td>1.7</td>
</tr>
<tr>
<td>Normal</td>
<td>1.8</td>
<td>503</td>
<td>2.2</td>
<td>1.8</td>
<td>452</td>
<td>1.5</td>
</tr>
<tr>
<td>Fibrous plaque</td>
<td>4.8</td>
<td>485</td>
<td>2.5</td>
<td>3.0</td>
<td>497</td>
<td>2.2</td>
</tr>
<tr>
<td>Fibrous plaque</td>
<td>3.0</td>
<td>477</td>
<td>2.6</td>
<td>2.0</td>
<td>421</td>
<td>1.7</td>
</tr>
<tr>
<td>Fibrous plaque</td>
<td>4.0</td>
<td>494</td>
<td>3.2</td>
<td>2.0</td>
<td>526</td>
<td>2.2</td>
</tr>
</tbody>
</table>

*The probe was applied perpendicularly at 4 W for 10 sec with two different forces.

diation with a bare fiberoptic and with the laser probe. In all instances, identical laser power (4 W) was applied to the optical fibers. In figure 9, A, the bare optical fiber (700 μm spot size) was used to ablate nonatherosclerotic aorta with a 4 sec pulse of argon-ion laser irradiation; in figure 9, B, the metal probe was applied horizontally with no force other than gravity for 10 sec. Despite the deposition of less radiant energy (16 J) in tissue with direct irradiation, the resulting ablation crater was clearly larger. Interestingly, the radial thermal injury zones surrounding the ablation crater showed a progressive attenuation with depth and there was minimal thermal damage at the crater base. In contrast, the laser probe (40 J) caused less ablation, and the thermal injury zones surrounding the laser probe crater were of uniform thickness.

One other difference was apparent with these low-power, long-pulse argon-ion laser irradiations of aortic tissue. Irradiation of nonatherosclerotic aorta with a bare optical fiber resulted in the explosive, fragmented, and split appearance of the superficial aortic tissue with extensive lateral subintimal dissections. This phenomenon is clearly seen in figure 9, A. In contrast, the injury resulting from metal probe application is limited and focally controlled with no evidence of vessel dissection.

Discussion

Although the laser probe is the only laser catheter to date that has been extensively tested in animal preparations and in human trials of peripheral and coronary revascularization, few data are available concerning the thermal characteristics of the laser probe ablation process.

In this study we found that laser probe temperature typically exceeded adjacent tissue temperatures. In contrast to direct, fiberoptic-mediated argon-ion laser ablation of aortic tissue, laser probe ablation occurred in a controlled fashion, not associated with subintimal dissections, and with uniform conduction of heat to tissue as reflected by essentially “isothermal” injury lines. Tissue ablation required temperatures exceeding 180° C, and adventitial temperatures during thermal ablation reached 70° C with the probe held in a fixed position. The deposition of more energy in the probe as well as the application of the heated probe at higher force effected greater tissue ablation. At identical laser variables, ablation of atheromata was more extensive than that of nonatherosclerotic aortic tissue.

Thermographic imaging illustrated that initial probe heating occurred at the tip where the laser beam exiting from the optical fiber first impinged on the metal. Heat was then rapidly conducted to the rest of the probe, resulting in a nearly uniform surface temperature after 5 sec of laser activation. The temperature of the probe increased linearly with laser energy, indicating that...
laser light deposition in metal dominated the heat transfer processes. Final probe temperature, however, was clearly inversely related to the thermal conductivity of the surrounding medium. Thus the laser probe attained the highest temperature in air, which is an excellent thermal insulator with low thermal conductivity. In contrast, the laser probe achieved the lowest temperature when immersed in water, which has much higher
thermal conductivity. This finding is rather puzzling in view of the fact that tissue ablation would hardly be expected to occur at temperatures lower than 90°C in vivo. Of interest here is the observation that the laser probe in blood attains temperatures higher than those in water. In a separate study with an Nd-YAG laser at higher powers (10 to 14 W), an even larger difference between probe temperatures in blood and in water was noted. In blood, probe temperatures up to 800°C were obtained, whereas in water temperatures never ex-

FIGURE 9. Hematoxylin and eosin–stained section of nonatherosclerotic aorta irradiated with 4 W argon-ion laser for 4 sec via a 300 μm optical fiber, 700 μm spot size in air (A). Normal aorta, exposed to the laser probe heated by 4 W of argon irradiation for 10 sec in air (B).
ceed 100°C for identical laser power-pulse levels. In blood, the hot laser probe was rapidly covered with carbonized debris, presumably derived from disintegrated cellular and proteinaceous elements. This carbonized coating may act as an insulating surface, allowing the probe to reach significantly higher temperatures despite immersion in air. (According to the manufacturer of the probes, similar probe temperatures are expected with the use of an argon laser and a Nd-YAG laser set at the same power.)

The prototype metal tips we used detached themselves from the optical fibers at powers exceeding 20 W in air. Furthermore, the optical fibers were occasionally damaged at probe temperatures exceeding 600°C. Laser probes currently used incorporate safety wires to prevent the intravascular loss of probes secondary to inadvertent overheating.

Probe temperatures persistently exceeded tissue temperatures. The heat loss that accompanies the change in tissue phase may, in part, account for the temperature differences between the laser probe and the surrounding vascular tissue. Poor thermal contact between the laser probe and tissue, the inability to measure the tissue temperature directly in contact with the probe, and the thermal insulation of tissue via escaping vapors may constitute other reasons for this difference.

This is the first published study that correlates a thermographic analysis of vascular surface temperatures during ablation with histologic findings. Vacuolation, and thus presumably evaporation of cellular water contents, was found to begin at approximately 120°C. More importantly, significant tissue volume loss, i.e., ablation, occurred only at temperatures exceeding 180°C, clearly in excess of 100°C, the boiling point of water. Similar observations have been made previously during direct laser beam irradiative ablation of vascular tissue. One explanation for these high ablative tissue temperatures is that tissue ablation is a nonequilibrium event inasmuch as the target tissue components are superheated at a rate exceeding the maximum for which thermodynamic equilibrium can be maintained. Thus the threshold temperature required for ablation would be expected to increase in proportion to the rate of heat conduction to the tissue. Furthermore, higher ablation temperatures would result in faster rates of ablation.

We observed that at probe temperatures between 100°C and 180°C, the metal tip adhered to tissue. Tissue adhesion of the probe in vivo has also occasionally been noted in work by other investigators. It is conceivable that at probe temperatures exceeding 180°C, as tissue ablation is initiated, a layer of fluid and gaseous ablation products surrounds the probe and effectively protects it from adhesion and consequent tissue avulsion.

Plots of tissue surface temperature as a function of radial distance from the edge of the probe showed that temperatures in excess of 50°C persisted at a distance greater than 1.0 mm from the laser probe. Given that irreversible denaturation of protein can occur at those temperatures, this radial conduction of heat may be important with respect to the lateral spread of thermal injury, particularly during prolonged stationary applications of the laser probe in vivo. In that respect, it was also of concern to find that adventitial tissue temperatures for 5 to 10 sec applications of the laser probe to the intimal surface reached 45° to 70°C. Even though the histologic changes observed consequent to our application of the laser probe in vitro were unimpressive, this does not exclude the evolution of a significant cellular response within the first 24 to 72 hr after the thermal insult in vivo. These observations emphasize the necessity of not allowing the heated laser probe to remain in stationary contact with the vessel wall (or the guiding catheter) for any period of time to avoid uncontrolled lateral and transmural conduction of heat with potentially adverse short- or long-term consequences. Rather, continuous intravascular movement of the hot probe would be expected to have a protective effect in vivo.

In this study, the laser probe seemed to be more effective in ablating fibrofatty plaque than nonatherosclerotic aorta. Although the number of samples tested was small, there was a tendency for more effective laser probe ablation of fibrofatty plaque compared with normal aortic tissue. Lower thermal conductivities of fibrous and fatty plaque relative to normal aorta, particularly at temperatures exceeding 60°C, have been demonstrated. By spatially confining laser probe heat and retarding its conductive transfer to tissue, the lower thermal conductivity and diffusivity constants of fibrofatty plaque relative to normal vessel wall might cause more extensive heating and evaporation of plaque. This difference in thermal properties would explain the clinical observation that the laser probe preferentially maintained an intravascular position during recanalization of total vessel occlusions.

The efficacy of tissue ablation can be enhanced by two mechanisms. First, increasing the irradiant energy deposited in the probe by increasing either laser beam power or pulse duration will result in higher laser probe temperature, greater conductive heat transfer to the tissue, and thus a faster rate of ablation. Second,
increasing the force applied to the hot probe enhances tissue ablation. Greater tissue ablation may simply be caused by lower thermal resistance with improved thermal contact of the probe with tissue. A contributing mechanism, as shown in figure 8, B may be compression of residual vacuolated tissue components resulting in the attendant loss of vacuolated cellular space. Whether other mechanisms contribute to the effect of force is not known.

Although both the continuous-wave visible and infrared lasers as well as the laser probe ablate tissue via evaporative heating,14 the mechanisms of ablation are quite different. Direct irradiation deposits the light energy in the tissue to be removed, whereas energy delivered to the laser probe primarily heats the metal probe. We were nevertheless interested in contrasting the tissue effects of similar argon-ion laser variables for direct tissue irradiation and for laser probe heating. It was clearly seen that the same amount of energy directly deposited in tissue (700 μm spot size) caused more ablation than when deposited in the metal tip for heating. Heat loss from the sides of the probe contribute to its lower efficiency. There may be additional heat loss because of the thermal resistance at the tissue-probe interface.

Direct tissue irradiation caused a progressive attenuation with depth of the radial injury zones surrounding the ablation crater. This observation can be readily explained by a number of mechanisms. First, the laser beam exiting the optical fiber has a divergence angle of approximately 15 degrees, resulting in progressive attenuation in power density with increasing distance from the optical fiber. Second, the absorptive and scattering properties of tissue will similarly result in an attenuation of power density with increasing distance from the optical fiber even in the absence of a divergence angle.11 In contrast, in view of the uniform temperature attained by the laser probe, it is not surprising to see the thermal injury zone surrounding the laser probe crater in figure 9, B, to be of uniform thickness, suggesting uniform radial heat conduction from the metal probe to the surrounding tissue.

Low-power, long-pulse direct argon-ion laser irradiation of tissue caused an explosive, fragmented, and split appearance of the superficial tissue, with extensive lateral subintimal dissections (figure 9, A). The argon-ion laser beam has a penetration depth of about 1.0 mm in normal aortic tissue. Thus at the argon-ion laser wavelength, subsurface temperatures may exceed surface temperatures of the irradiated tissue.11 This may cause greater nonequilibrium superheating of subsurface tissue components relative to the tissue surface, resulting in explosive evaporation of tissue with mechanical lateral stress tears. This uncontrolled ablation process might lead to progressive vessel dissection if performed in vivo, where arterial blood might enter and extend these cleavage planes. In contrast, the thermal injury after exposure of vascular tissue to the laser probe occurs in a controlled fashion without extensive dissection (figure 9, B). This finding can be explained on the basis that heat is not deposited below the surface but is rather transferred conductively into tissue.

To date, the confinement of laser irradiant energy in a metal tip for the controlled conductive heat transfer to tissue during evaporative ablation has been shown to be a promising alternative to direct laser beam ablation of tissue. In the future, it may constitute an important adjunct, or even substitute, for conventional balloon angioplasty. It will therefore be important to correlate the short- and long-term tissue responses in vivo to tissue probe application with temperature-time data. Such a study will have important implications regarding the definition of optimal laser application and catheter manipulation for safe and successful thermal peripheral and coronary angioplasty.

We thank Dr. Roberto J. Bayardo, Chief Medical Examiner of Travis County, Texas, for his assistance in conducting these experiments.

References


Laser probe ablation of normal and atherosclerotic human aorta in vitro: a first thermographic and histologic analysis.
A J Welch, A B Bradley, J H Torres, M Motamedi, J J Ghidoni, J A Pearce, H Hussein and R A O’Rourke

Circulation. 1987;76:1353-1363
doi: 10.1161/01.CIR.76.6.1353

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/76/6/1353