

# Laser Treatment of Leg Veins: Physical Mechanisms and Theoretical Considerations

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**Background and Objectives:** A discussion of laser treatment of leg veins is based on a review of the literature, theoretical analysis, and the clinical experiences of the authors. Theoretical computations are discussed within the context of clinical observations.

**Study Design/Materials and Methods:** A Monte Carlo model is used to examine volumetric heat production, fluence rate, and temperature profiles in blood vessels at 1,064 and 532 nm wavelengths with various beam diameters, vessel diameters, and pulse durations.

**Results:** Clinical observations, Monte Carlo results, and a review of the literature suggest that longer wavelengths and longer pulses durations favor vessel contraction over intraluminal thrombosis. Monte Carlo simulations show that longer wavelengths are more likely to uniformly heat the vessel compared to highly absorbing wavelengths. Methemoglobin production causes deeply penetrating wavelengths to generate more volumetric heat for the same input radiant exposure.

**Conclusions:** Clinical observations and models support the role of long wavelengths and long pulses in optimal clearance of most leg telangiectasias. *Lasers Surg. Med.* 36:105–116, 2005. © 2005 Wiley-Liss, Inc.

**Key words:** laser; leg; telangiectasia; model; light; mechanism

## INTRODUCTION

The trend in laser leg vein treatment has been toward longer wavelengths that allow for deeper penetration and greater sparing of the epidermis. A range of wavelengths from 532 to 1,064 nm has been applied [1–8]. Also, there has been renewed interest in intraluminal techniques, both with 800 and 940 nm, and more recently, 1,320 nm [9,10].

Sclerotherapy, the most common treatment for leg veins, is usually effective. However, cannulation of very small vessels (0.1–0.3 mm) can be challenging. Moreover, some patients are “needle-phobic.” Potential side effects of any leg vein treatment include pigmentary changes, cutaneous necrosis, pain, and telangiectatic matting. The likelihood of these adverse events in any individual is dependent on vein diameter, vessel color, location, intraoperative technique, and postoperative care.

Many clinicians restrict their practices to the treatment of spider and reticular veins. Spider veins are defined as 0.2–2-mm red and blue vascular ectasias. They are often

associated with larger reticular veins, defined as “non-bulging” subcutaneous veins ranging up to 5 mm in diameter [11]. Most smaller telangiectasias (0.2–1 mm) reside about 300  $\mu\text{m}$  below the skin surface [12]. These are the vessels that dermatologists treat most often and represent the most common complaint among patients with “leg veins.” They range in hue from dark blue to bright red. The bright red variants tend to be smaller (0.2–0.5 mm) than their blue counterparts, and the  $\text{PaO}_2$  is slightly higher. Deeper vessels appear bluer regardless of size or degree of oxygenation [13]. Laser treatment of leg veins has proved more challenging than facial telangiectasias. Compared to facial vessels, leg veins typically require more treatment sessions. Also, higher radiant exposures are required versus like-sized vessels on the face. Finally, the epidermis tends to be more sensitive to injury, even with equivalent constitutive or facultative pigmentation. On the legs, even when vessels are treated “appropriately” with laser, one often observes thrombosis and hyperpigmentation [14]. Moreover, particularly when using green/yellow (GY) sources, crusting and even scarring have been observed with radiant exposures that only slightly exceed the efficacy threshold.

The purpose of this study is to discuss laser treatment of leg veins, first by examining and comparing the results of a theoretical model with clinical observations. Next we analyze these results within the context of our clinical experiences and the literature.

Our clinical experience includes a recently completed study where we treated 23 patients with 0.2–1.6-mm diameter vessels located over the lateral thigh (in most cases) [15]. In the study, patients were treated once with a Nd YAG laser, usually with a 3-mm spot, and followed for 4 months. We also draw on our experience treating roughly 350 patients over 6 years using a PDL and/or KTP laser for smaller spider veins in lighter skinned patients.

We confine our discussions to laser irradiation applied from the surface and do not address newer intraluminal techniques. We limit our discussion to vessel diameters

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from 0.2 to 2 mm (those non-protuberant vessel that are often most visibly distressing to the patient). Treating larger vessels (>2 mm) is associated with higher incidences of thrombosis, hyperpigmentation, and pain. Also, in treating larger varicosities, there is often associated valvular incompetence (even without protruding vessels there may be incompetence of the superficial venous system) that might require non-invasive imaging to optimize outcomes [16,17].

## MATERIALS AND METHODS

### Monte Carlo Simulations

We used Monte Carlo simulations of light transport (MCML) [18,19] to model light distribution inside irradiated blood vessels and skin for various laser parameters and vessels. We considered in the computation a few realistic clinical scenarios. We divided laser interventions into two “extreme” portions of the wavelength range commonly applied to leg veins (choosing 1,064 and 532 nm as representative longer and shorter wavelengths, respectively). Then we considered two common types of vessels that present to a dermatology clinic. First we treated a 0.3-mm vessel diameter residing 0.3 mm under the skin. Then we treated a vessel 1.3 mm in diameter residing 1.3 mm in depth.

We used a semi-infinite two-layer geometry to simulate skin. The skin model comprised a top layer of 100- $\mu\text{m}$ -thick epidermis overlying a semi-infinite dermis. The blood vessels were inserted in the dermis at the desired depths. Laser beam energy was incident normally to the epidermis. The tissue volume was discretized into a three-dimensional Cartesian grid points and the fluence rate ( $\text{J}/\text{cm}^2$ ) and the volumetric heat production ( $\text{J}/\text{cm}^3$ ) were calculated at all the grid points. In all calculations, the input radiant exposure was  $1 \text{ J}/\text{cm}^2$ . The local volumetric heat production is the product of the local fluence rate and absorption coefficient ( $\mu_a$ ). The optical properties used in the simulations are shown in Table 1 [18,20,21].

## RESULTS

### Wavelength

Figure 1a,b shows the fluence rate distribution of a 3-mm beam diameter for 1,064- and 532-nm laser irradiations in dermis, respectively. The 1,064-nm wavelength penetrates deeper in the skin than 532 nm.

Figure 2a,b shows the distribution and magnitude of the volumetric heat production in a 1.3-mm diameter vessel residing 1.3 mm below the skin surface produced by 1,064- and 532-nm laser irradiations, respectively. The volumetric heat production inside a 1.3-mm vessel at 1,064-nm wavelength is more uniformly distributed than at 532 nm.

Figure 3a,b shows the distribution of the volumetric heat production in a 0.3-mm diameter vessel residing 0.3 mm below the skin surface produced by 1,064 and 532 nm, respectively. The volumetric heat production inside a 0.3-mm vessel at 1,064 nm is more uniformly distributed than at 532 nm.

Figures 2 and 3 also show that the volumetric heat production *magnitude* inside the 0.3- and 1.3-mm vessels at 532 nm is higher than at 1,064 nm.

### Beam Diameter

Figure 4 shows the distribution of fluence rate in dermis for 3-, 6-, 9-, 12-, and 15-mm beam diameters at the 1,064-nm wavelength. The optical penetration depth is proportional to the beam diameter. Larger spots penetrate deeper than smaller spots.

Figure 5a,b shows the volumetric heat production distribution in a 1.3-mm diameter vessel residing 1.3 mm below the skin surface produced by 1,064-nm irradiation with 3- and 6-mm beam diameters, respectively. The 6-mm beam diameter produces a more uniform volumetric heat production distribution with a higher magnitude than the 3-mm beam.

### Pulse Duration

Figure 6 shows the temperature distribution at the end of a 1,064-nm laser pulse inside a 1-mm diameter vessel residing 1 mm within the skin. The temperature rise was produced by laser pulse durations ranging between 3 and 1,000 milliseconds with equal input fluences. There is no significant difference in the temperature distribution within the vessel in the range of 3–100 milliseconds.

### Dynamic Changes in Blood Properties During Irradiation and Multiple Pulses

Figure 7a,b shows volumetric heat production in 1.3- and 0.3-mm diameter vessels residing 1.3 and 0.3 mm below the skin surface, respectively, produced by 532-nm irradiation and assuming 100% methemoglobin conversion. The model

**TABLE 1. Optical Properties Used in Simulation**

	1,064 nm			532 nm		
	Absorption coefficient ( $\mu_a$ ) (1/cm)	Reduced scattering coeff. ( $\mu_s'$ ) (1/cm)	Refractive index (n)	Absorption coefficient ( $\mu_a$ ) (1/cm)	Reduced scattering coeff. ( $\mu_s'$ ) (1/cm)	Refractive index (n)
Epidermis	1.34	7.32	1.37	11.3	41.3	1.37
Dermis	0.24	7.32	1.37	0.53	41.3	1.37
Blood (70% oxy-30% deoxy)	2.2	3.4	1.37	231	5.5	1.37
Met Hb	9	3.4	1.37	150	5.5	1.37

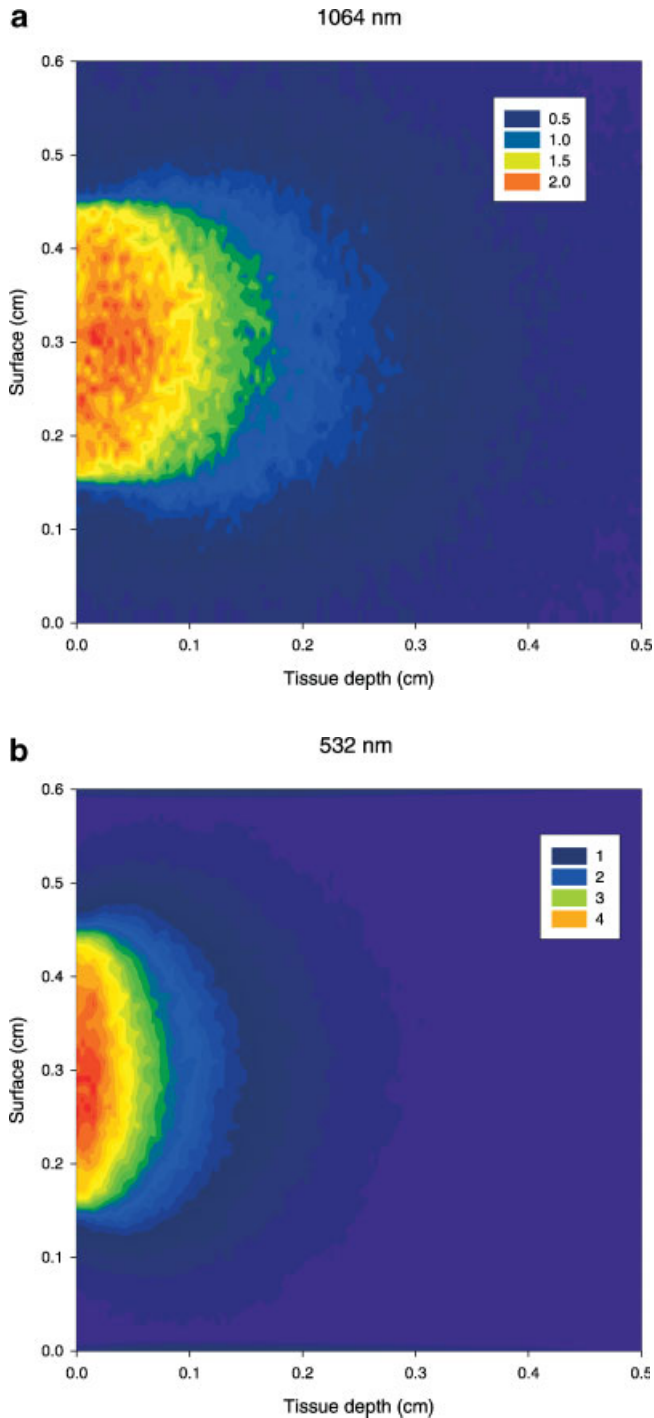


Fig. 1. It shows light distribution in bloodless dermis for 3-mm diameter: (a) 1,064- and (b) 532-nm laser beams.

shows similar volumetric heat production distribution and magnitude in the 1.3-mm vessel for methemoglobin and normal blood (see Figs. 2 and 3).

Figure 8a,b shows the volumetric heat production of 1,064 nm wavelength in 1.3- and 0.3-mm diameter vessels

residing 1.3 and 0.3 mm below skin surface, respectively. The model shows uniform and similar volumetric heat production *distribution* inside the 0.3-mm vessel using methemoglobin and normal blood properties. However, the *magnitude* of volumetric heat production is 3.5 times higher than with normal blood optical properties (see Figs. 2 and 3). On the other hand, the model showed a small difference in the volumetric heat production *distribution* inside the 1.3-mm vessel with methemoglobin versus normal blood optical properties.

## DISCUSSION

Figure 9 shows optical properties of blood, dermis, and light-skinned epidermis (with 4% volume fraction of the epidermis occupied by melanosomes), the skin constituents relevant to laser treatment of blood vessels, in the range of 500–1,100 nm [18,22]. According to Figure 9, selective targeting of blood vessels is possible in principle with all wavelengths in the range due to greater absorption by blood than by the surrounding dermis.

### Wavelength

The Monte Carlo computations show that a 1,064-nm beam penetrates deeper than 532 nm in both blood and skin. This result is expected because of lower scattering at the longer wavelengths, as shown in Figure 9. As a result, the 1,064-nm wavelength laser energy reaches deeper vessels and is more uniformly deposited in the vessels as shown in Figure 2. These results suggest that 1,064 nm is more effective than 532 nm in treating larger and deeper vessels. This is supported by multiple studies, as the 1,064-nm long-pulsed Nd:YAG laser (3–100-millisecond pulse duration) has demonstrated safety and efficacy in treating leg veins ranging from 0.2 to 3 mm in various skin types. Representative effective fluences are 60–200 J/cm<sup>2</sup> for larger (2–5 mm) vessels and larger spots (6–12 mm) [2,14,23–30].

As an alternative approach to deeply penetrating wavelengths, one can increase the radiant exposure with highly absorbing wavelengths (GY light). However, just increasing radiant exposure might lead to vaporization of blood in the top layer of the vessel followed by wall rupture and purpura. Clinically, we have observed that within the range of fluences tolerated by the epidermis, closure of leg veins greater than 1.3 mm is not usually achievable with the 532-nm laser. Most likely, insufficient heating of the deeper parts of the vessel accounts for this failure. In theory and in practice, one can optimize vessel closure with short wavelengths by employing: (1) efficient cooling, (2) long pulse durations (thus avoiding overheating of the superficial vessel wall while allowing for epidermal preservation), and (3) larger spots that allow for greater and more uniform penetration of the beam.

Figure 3 suggests that both 1,064 and 532 nm are suitable for treating smaller superficial vessels. Although 532 nm does not uniformly heat the whole vessel, it heats most of the circumference of the vessel wall. The 1,064 nm beam, on the other hand, heats the smaller vessel more uniformly, but because blood absorption at 1,064 nm is

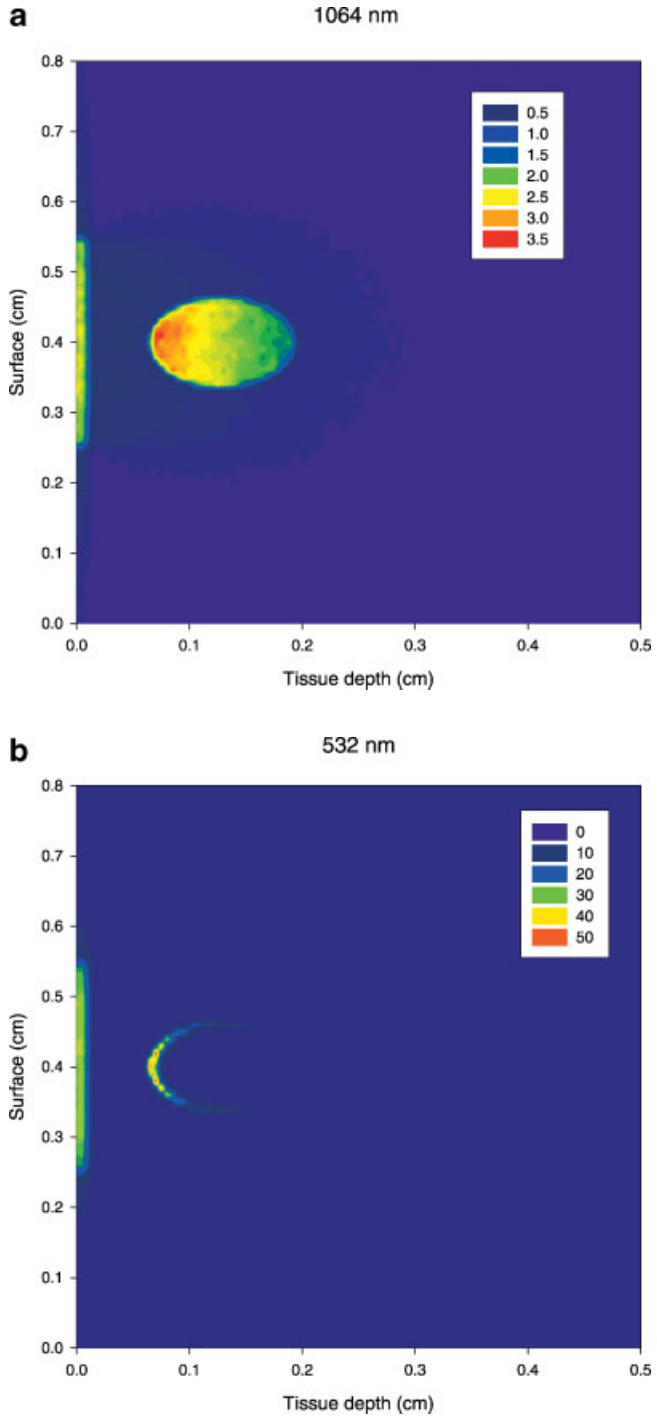


Fig. 2. It shows the volumetric heat production in a 1.3-mm diameter vessel residing 1.3 mm below the skin surface, produced by (a) 1,064 and (b) 532 nm.

significantly lower than at 532 nm ( $\mu_a$  of 2.2/cm vs. 231/cm), the 1,064-nm laser requires much higher radiant exposures. For example, with the 1,064-nm wavelength, smaller vessels (0.1–0.3 mm) have been successfully treated with smaller spots (i.e., 1.5 mm), but radiant exposures up to

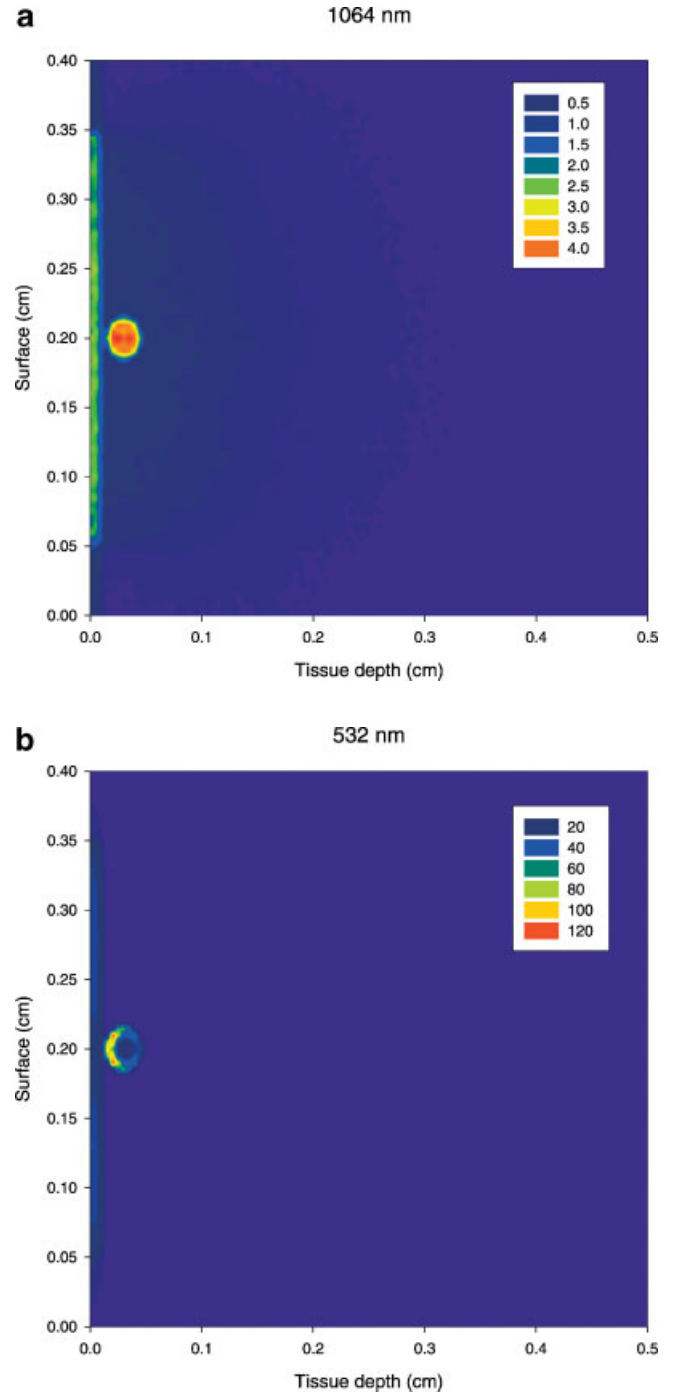


Fig. 3. It shows the volumetric heat production in a 0.3-mm diameter vessel residing 0.3 mm below the skin surface, produced by (a) 1,064 and (b) 532 nm, respectively.

500 J/cm<sup>2</sup> are required. With 532 nm, the same-sized vessel can be closed with a radiant exposure of only 15 J/cm<sup>2</sup> with the same 1.5-mm beam diameter.

Figures 2 and 3 also show that with the 532-nm wavelength, the volumetric heat production is higher when



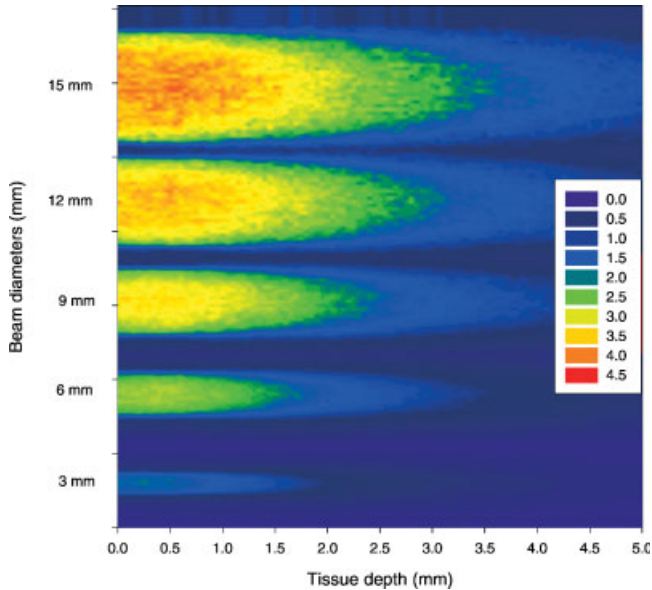


Fig. 4. It shows the distributions of fluence rate in dermis at 1,064-nm laser irradiation for 3-, 6-, 9-, 12-, and 15-mm beam diameters.

irradiating a smaller versus a larger vessel. This is due to the shallower penetration depth and high backscattering at this wavelength.

This finding is supported by our clinical observation that with the 532-nm wavelength, there is a need for higher radiant exposures when treating larger vessels and less when treating smaller vessels. For example, treating a 0.4-mm vessel with a KTP laser requires about  $15 \text{ J/cm}^2$  with a 2-mm spot, whereas treating a 1-mm vessel usually requires as much as  $20 \text{ J/cm}^2$ .

With the 1,064-nm wavelength, the modeling results and clinical observations are different. Figures 2 and 3 show that the volumetric heat production *magnitude* generated with the 1,064 nm wavelength in the 1.3-mm diameter and 0.3-mm diameter vessels is similar. However, clinical experience tells us that the 1,064-nm wavelength requires higher radiant exposures for treating smaller vessels compared to the treatment of larger vessels. For example, using a 2-mm spot, “closing” a 0.4-mm vessel requires about  $280 \text{ J/cm}^2$ , whereas a 1-mm vessel usually requires only  $200 \text{ J/cm}^2$ . We believe that the need for higher radiant exposures for effective vessel closure is because of the temperature-time dependent collagen shrinkage phenomena. Smaller vessels cool faster and therefore the temperature-time product is below that required for effective shrinkage. To compensate for the faster cooling of the vessel, the temperature must be higher, requiring higher incident radiant exposures.

Overall, one can optimize smaller vessel closure with longer wavelengths by employing: (1) shorter pulse durations (so long as the duration exceeds 20-millisecond pulses shorter than this were associated with greater purpura and less stenosis per unit input energy); (2) smaller spots

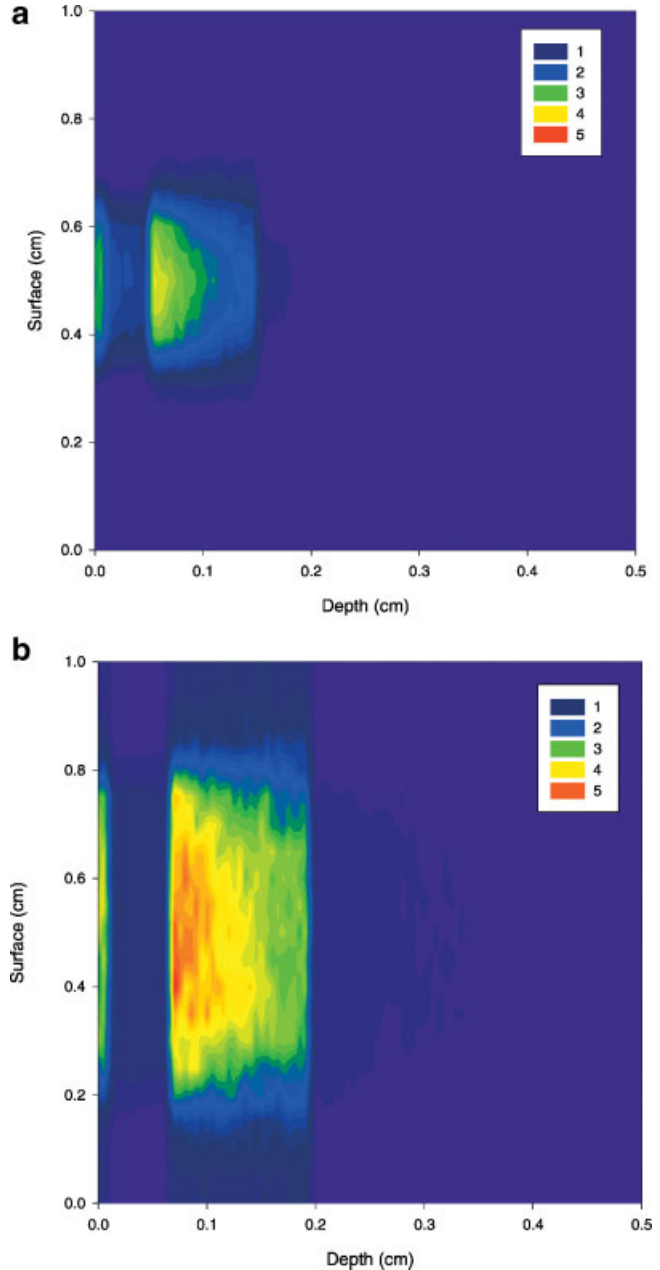


Fig. 5. It shows the volumetric heat production in a 1.3-mm diameter and 1.3-mm deep vessel, produced by (a) 3- and (b) 6-mm diameter laser beams.

that allow for higher fluences; and (3) efficient cooling for epidermal protection.

### Beam Diameter

Figure 4 shows that optical penetration depth is determined not only by wavelength but also by the incident laser beam diameter. Larger beam diameters penetrate the skin deeper than smaller beams and could translate into better vessel heating. This is illustrated by Figure 5a,b

that shows that a 6-mm laser beam heats a vessel more uniformly than a 3-mm beam. The larger beam also produces a larger volumetric heat production for the same incident radiant exposure, which means that higher radiant exposures will be required to achieve the desired end point with smaller beam diameters. Our clinical observations support the Monte Carlo computations. For example, we performed a leg vein study using three-spot sizes (6, 3, and 1.5 mm). For like-sized vessels ( $\sim 0.6$  mm), threshold radiant exposures for vessel stenosis were 160, 280, and  $400 \text{ J/cm}^2$ , respectively [15]. Because of deeper penetration, the larger beam diameters are more effective in reaching larger and deeper vessels. This is confirmed by our clinical observations. For example, we were able to treat a 2.5-mm vessel with a 6-mm spot at  $160 \text{ J/cm}^2$ . However, applying a 1.5-mm spot to the same vessel either resulted in localized vessel rupture at high fluences or a non-response with lower fluences.

These data might suggest that larger spots are “better” than smaller spots. However, when considering spot size within the context of pain, risk of dermal and epidermal injury, and efficacy, we suggest that spot size should be roughly equal to the vessel diameter (for deeply penetrating wavelengths). In this way, the maximum fraction of incident irradiation is absorbed by the vessel and not by dermis or other structures. Smaller spots tend to reduce pain because even though radiant exposure is high, the total laser energy is less, and the likelihood of catastrophic full-thickness ulceration decreases. With larger spots ( $>5$  mm), full thickness necrosis is observed with excessive fluences or pulse stacking. In these cases, an entire cylinder of tissue is heated. These 3–5-mm deep wounds inevitably heal with scarring (see Fig. 10).

### Pulse Duration

According to the principles of selective photothermolysis, laser pulse duration should be less than or equal to the vessel thermal relaxation time to maximize laser energy deposition within the vessel and to confine thermal damage to the vessel. Since the numerical values of the thermal relaxation times only provide “an order of magnitude” approximation, we solved numerically the conduction heat transfer equation with a finite-difference method. The model shows that the intravascular temperature profiles are similar for the range of pulse widths (3–100 milliseconds) used in commercially available lasers (see Fig. 6). However, clinically we have observed that longer pulses are more effective and/or cause less side effects, especially when treating larger diameter vessels. We found, for example, that 20–60-millisecond pulses consistently outperformed 3-millisecond pulses over a range of vessel diameters [15]. In the 3-millisecond sites, contraction was modest to non-existent, whereas longer pulses achieved fairly consistent vessel stenosis. The clinical responses were supported by histology, where the longer pulses resulted in perivascular collagen heating and vessel narrowing, whereas the 3-millisecond pulses showed thrombosis. Because the model did not show any significant difference in temperature distribution within the vessel

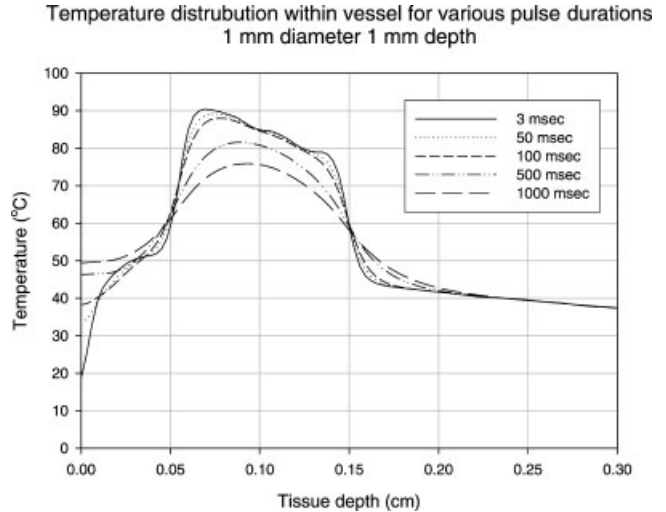


Fig. 6. It shows the temperature distribution at the end of a laser pulse inside a 1-mm diameter vessel residing 1 mm in skin produced by laser pulse durations ranging from 3 to 1,000 milliseconds with equal input radiant exposure (1,064 nm).

over the pulse duration range, we believe that more effective closure with longer pulses is due to the temperature-time nature of collagen shrinkage. It follows that for similar incident radiant exposures, longer pulse durations result in longer heating times that optimize collagen shrinkage and subsequent vessel closure [31]. In addition, longer pulses tend to spare smaller structures (i.e., melanosomes). Longer pulse durations might also allow for methemoglobin formation and subsequent increased efficacy [20,21,32].

The other approach, using higher radiant exposures with shorter pulse durations, leads to extreme localized heating and possible vessel rupture (as a localized steam bubble expands to fracture the wall) which is associated with an audible pop. Clinically, we have observed more purpura, thrombosis, and hyperpigmentation with shorter pulses [15]. Purpura (outside the vessel boundaries) caused by pulsed lasers can be an unacceptable side effect. Immediate purpura is due to laser-induced deoxygenation of blood. The causes of delayed purpura are more complex and most likely involve vasculitis, wherein inflammation results in sludging of the blood and extravasation of RBCs through leaky vessel walls. In post-treatment biopsies, coagulated red blood cells (RBCs) and fibrin thrombi have been observed in the vessels [33]. Immediate rupture and hemorrhage has been shown to be pulsewidth dependent, with longer pulses demonstrating a higher “hemorrhage threshold” [34].

### Epidermal Preservation

Figure 9 shows that longer wavelengths improve epidermal protection during treatment because of lower melanin absorption. Lower melanin absorption also reduces energy

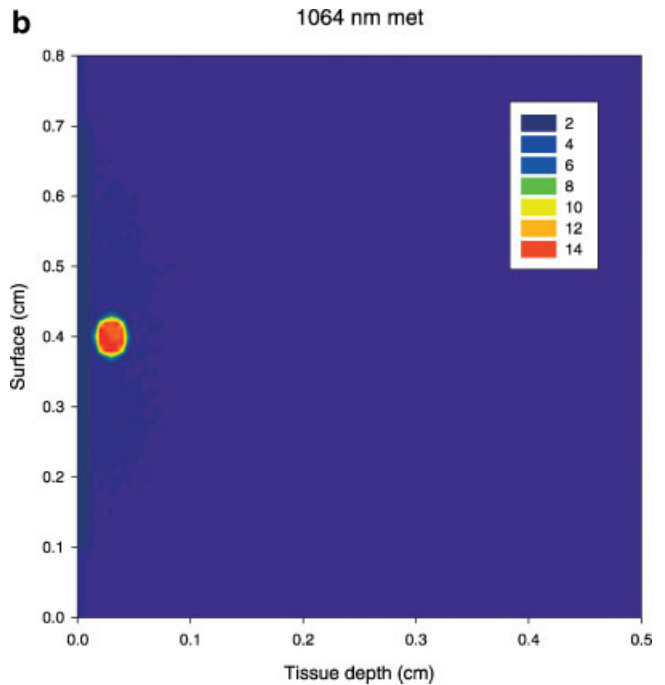
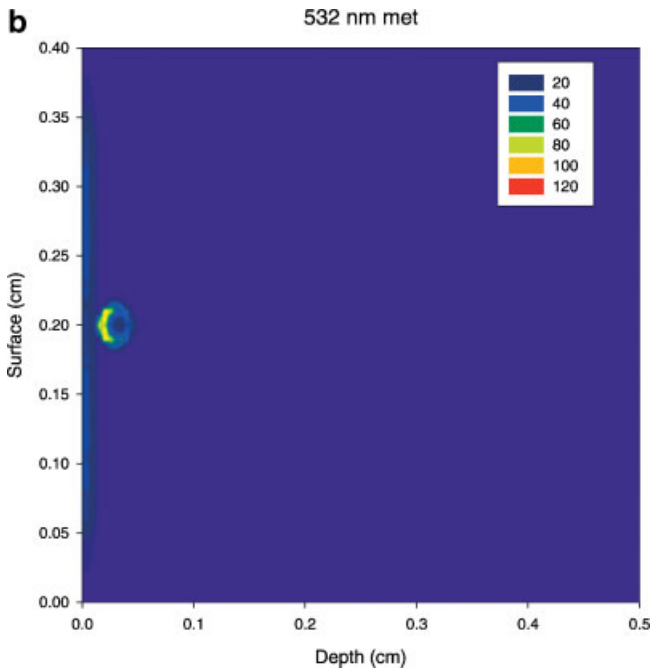
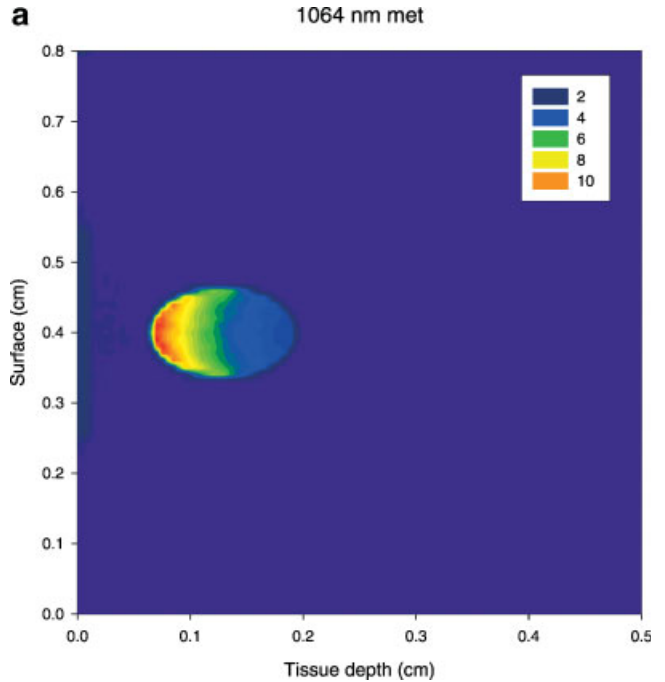
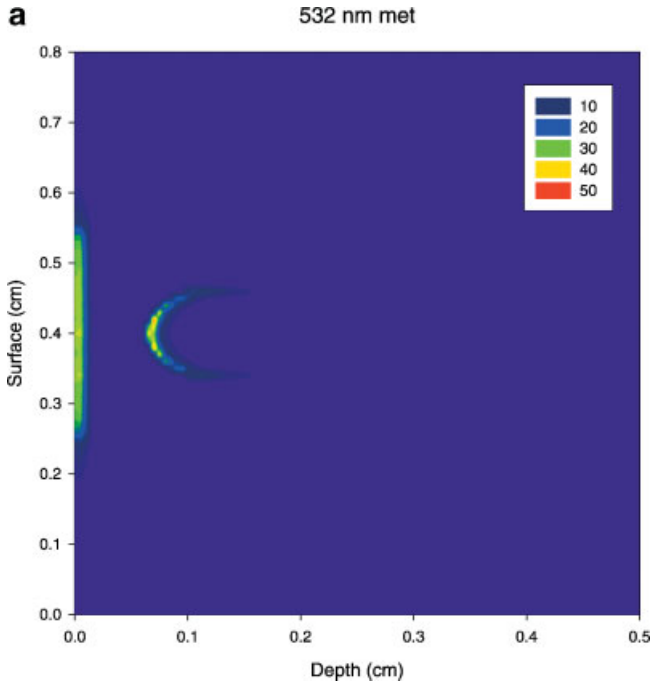


Fig. 7. It shows the volumetric heat production with methemoglobin for 532-nm light in (a) 1.3-mm diameter, 1.3-mm depth and (b) 0.3-mm diameter, 0.3-mm depth vessel, respectively (without methemoglobin counterpart is shown in Figs. 2 and 3).

Fig. 8. It shows the volumetric heat production of the 1,064-nm wavelength in 1.3-mm diameter and 0.3-mm vessels residing 1.3 mm and 0.3 mm below skin surface (without methemoglobin counterpart is shown in Figs. 2 and 3).

loss in the epidermis, increasing the overall efficiency. Epidermal cooling has increased the safety and efficacy of laser treatment. Techniques include contact cooling with a sapphire window or copper plate, convective air-cooling, and cryogen spray cooling. Also, cooling can be achieved

with cold gels and aluminum rollers. All of these methods achieve some degree of epidermal protection. When one examines heat transfer coefficients, cryogen spray cooling is optimal. However, given the actual cooling times (about 500 milliseconds, 2 seconds, and 50-milliseconds, for contact

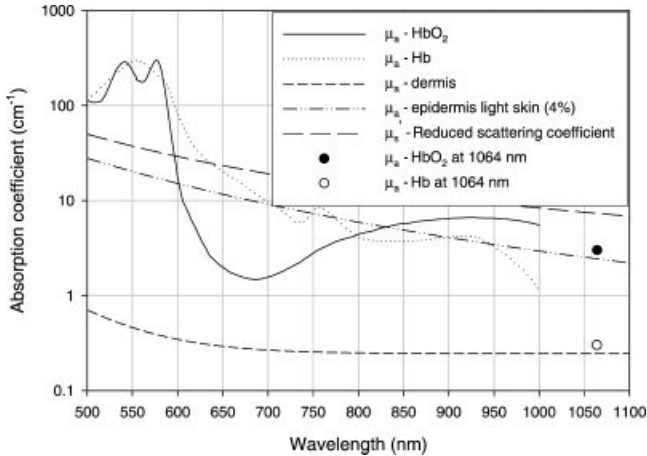


Fig. 9. It shows the optical properties of blood, dermis, and light-skinned epidermis at 500–1,100 nm.

cooling, cold air, and cryogen spray cooling, respectively), the total amount of heat extracted is actually fairly close in the practical application of these three cooling approaches. One can calculate the total heat extraction by calculating the product of the heat transfer coefficient and the cooling time.

### Dynamic Changes in Blood Optical Properties

Recently, Black and Barton have examined heat-induced chemical changes in blood. They found that with 1,064-nm irradiation, the RBC becomes spheroid and methemoglobin is formed, which has three times the absorption of oxyhemoglobin at 1,064 nm [20]. They concluded that any modeling of vessel heating was incomplete when dynamic changes occurring during irradiation were not considered. For example, due to methemoglobin production, during a pulse greater than 10 milliseconds, one can expect increased heating with less energy deposition near the end of the pulse than at the beginning of the pulse [20]. In support of this argument, Randeburg et al. [35] showed that gradual heating of vessels was more likely to lead to methemoglobin formation, whereas rapid heating was more likely to lead to hemoglobin denaturation. This study, plus our model results showing that at 1,064 nm the volumetric heat production *magnitude* was much higher inside vessels with methemoglobin, suggests that methemoglobin can play an important role during leg vein treatment and should be taken into consideration. For example, when using multiple pulses, a second pulse with similar radiant exposure might lead to an enhanced reaction versus the first pulse. With very high conversion to methemoglobin, however, high fluences might result in localized vessel overheating and wall rupture. Therefore, in applying a second pulse, energy should be significantly reduced in order to minimize side effects. At 532 nm, the modeling shows that methemoglobin does not have a significant effect on light distribution and volumetric heat production as at 1,064 nm. We assumed in our model 100% methemoglobin conversion in order to simplify the

calculation and to examine the extreme case of full conversion. However, on a practical level, it is unknown what proportion of blood is converted to methemoglobin as a function of temperature and time during exposures of up to 100 milliseconds. We need more information to fully integrate this effect into our mathematical modeling. At present, the *in vivo* role for methemoglobin is not completely known.

### Clinical Scenarios

**Scenario (1): Small vessels (0.2–1 mm)–short wavelength (GY).** With lower radiant exposures (and/or longer pulses), one observes transient bluing that represents cyanosis and small microcoagula (as the coagulum travels down the lumen). Within seconds [1], the vessel appears to re-perfuse and appears grossly normal, or [2] the bluing gives way to apparent vessel closure (possibly from a coagulum that becomes adherent to the vessel wall and stops local flow) [36]. With increasing radiant exposure, there is vessel constriction and an increasing number of microcoagula but sometimes a return of flow. With even higher radiant exposures, there is robust constriction and fixed coagula with no return of flow [36]. Sometimes a second pass is required to cause permanent closure after initial bluing of the vessel. Within this range of vessel diameters, complete stenosis is often observed in only the smallest vessels. The PDL is more likely to show persistent intraluminal bluing and spot-sized purpura compared to intense pulsed light (IPL) and KTP lasers. The severity of the purpura decreases as the macropulse width is increased. Patients with a slight tan and/or high levels of constitutive pigment tend to show epidermal damage with higher fluences, even with active cooling.

**Scenario (2): Smaller vessels (0.2–1 mm)–long wavelength.** With larger spots (4–6 mm) and small radiant exposures, one observes transient bluing or no effect at all. As the radiant exposure increases, smaller vessels show partial constriction but rarely full closure. At radiant exposures above 300 J/cm<sup>2</sup> and pulse durations in the 20-millisecond range, one reaches an epidermal and/or dermal damage threshold where full-thickness necrosis can be observed. Interestingly, pain is usually moderate until a point where the vessel appears clinically altered. Then pain tends to be severe, even for small vessels.

With a smaller spot (1.5 mm) and radiant exposures below 300 J/cm<sup>2</sup>, one sometimes observes bluing of the vessel. As the radiant exposure approaches 350–400 J/cm<sup>2</sup>, in the absence of compression, even very small vessels will close. Within minutes, linear urticaria surrounds the vessel. By this time that the vessel may partially reopen, leaving a pencil-thin blue line representing early thrombosis. Sometimes a second pass will reclose the vessel permanently.

**Scenario (3): larger vessels (1–2 mm)–long wavelength, and large spot.** Radiant exposures as small as 70 J/cm<sup>2</sup> will close vessels with a 5–6-mm spot. Pain is typically severe and proportional to the spot *area* and the vessel diameter. For a 3-mm spot, radiant exposures from 150 to 180 J/cm<sup>2</sup> are typically needed for vessel closure.



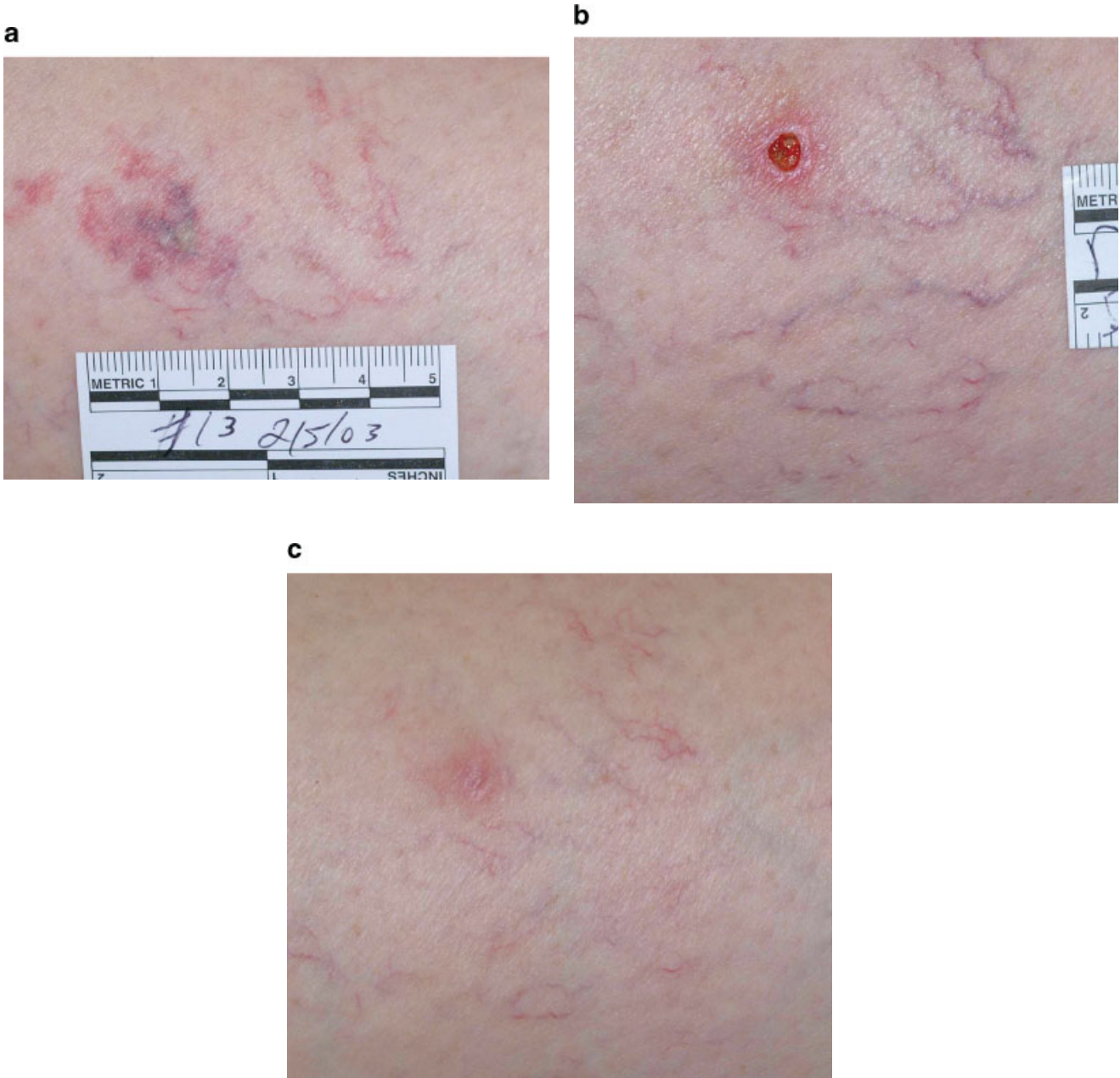


Fig. 10. It shows ulceration and healing after inadvertent stacking at one site: 1,064-nm pulse, 6-mm spot,  $200 \text{ J/cm}^2$ , convective cold air chilling, 2 pulses at 1 Hz. **a:** One day after treatment—note white color is harbinger of necrosis. **b:** Three weeks later, ulceration has occurred, **(c)** after 4 months, ulcer has healed with small scar.

Often the vessel will partially close. A second pass will either further close the vessel or achieve no further change (as if a solid thrombus has already formed). In this case, the vessel is no longer compressible. If one exceeds  $250 \text{ J/cm}^2$  with a 2-mm vessel, an audible pop is observed, followed by a palpable wheal and the near-immediate appearance of intravascular “purpura.” In this case, vessel rupture results in purpura that over 1–2 days extends beyond the linear profile of the vessel. These vessels often show

intravascular thrombosis lasting for weeks to months as well as delayed hyperpigmentation. Despite the side effects, these vessels usually eventually clear (see Fig. 11). However, clearance and the resolution of hyperpigmentation may require 6 months or longer.

In clinical practice, with vessels greater than 1 mm and using 1,064 nm, we have found it difficult to achieve complete and irreversible stenosis after one pass. More often there is immediate disappearance of the vessel

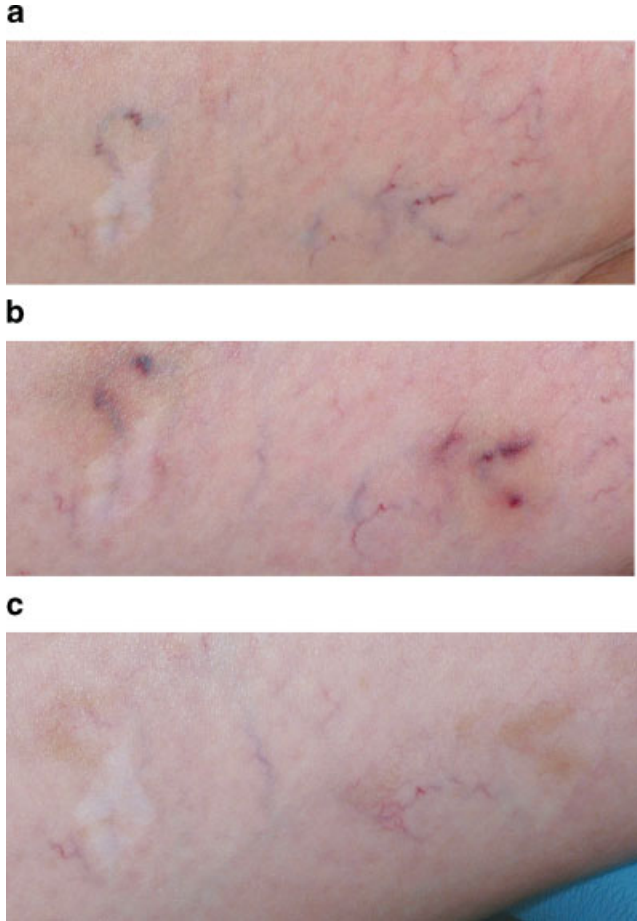


Fig. 11. **a:** Pretreatment inner thigh, **(b)** 1 week after treatment shows thrombosis, **(c)** 6 months later, vessels have largely cleared but some hyperpigmentation persists. (Settings: 260 J/cm<sup>2</sup>, 40 milliseconds, 3-mm spot, cryogen spray cooling, 1 pass).

followed by the emergence of a sliver-like thread within minutes of irradiation (much like a shrimp “vein,” see Fig. 12). By the following day (and often within 15 min of irradiation), in some cases there are portions of the vessel with obvious inflammation and intravascular thrombosis. Based on the literature and our clinical impressions, even without complete stenosis, there is damage to the wall and an adherent coagulum [32,36,37]. Over the following day, the thrombus begins to organize. The advantage of complete or almost complete irreversible stenosis is that the likelihood of thrombosis and dyspigmentation is reduced. The return of the threadlike blue strip followed by the emergence of a larger thrombus is likely the result of collagen shrinkage forces being overcome by the inflammatory reaction. Also, some of the constriction from the heated collagen may “relax” with cooling.

### Mechanisms For Vessel Clearance

From our clinical observations, theoretical analysis and literature review, the mechanisms for laser-induced vessel

clearance can be divided broadly into (1) heat-induced vessel contraction (Fig. 12a–c) and (2) intravascular thrombosis (whose immediate clinical endpoint is persistent bluing) (Fig. 12a–c). Other investigators using high-speed photography have suggested that vessel constriction is important for laser-induced vascular destruction [38]. Constriction results from direct heat-induced collagen shrinkage; however, some heat-induced vessel spasm might occur. Vessel walls contain fibrous collagens oriented circumferentially about the lumen. Collagen shrinkage is a temperature-time dependent phenomenon [39]. Lower temperatures require longer exposures for effective shrinkage and vice versa [31,40]. For very small spider veins, there is no true wall but only a fine lining of endothelial cells. In this case, perivascular collagen is heated and constricts the lumen [39].

The other mechanism for vessel clearance is intravascular thrombosis after thermal denaturation of the inner vessel wall [41]. The thermally damaged endothelium and perivascular tissue initiate a cascade of inflammation and wound healing, which results in replacement of the vessel lumen by fibrous tissue. Very large vessels tend to thrombose with either laser or sclerotherapy, and most side effects arise from thrombosis. Thrombosis has been called an “impediment” to vessel clearance in sclerotherapy [42].

With enhanced illumination (V300 polarized light source, Syris Scientific, Gray, ME), one can observe blood being squeezed out of the lumen by the laser pulse. The constricting wall pushes blood in both directions toward a more dilated adjacent lumen. Often, after a few minutes, the vessel appears to be partly refilled, most likely the result of incomplete or reversible stenosis. If coagulated blood is trapped in the lumen after treatment, marked inflammation accompanies the evolving thrombus. Thrombus resolution is often accompanied with purpura, hyperpigmentation, and tenderness.

Although vessel clearance proceeds through either constriction and/or thrombosis, the former is the optimal mechanism, as there is less likelihood for side effects. Most likely both mechanisms are operative in the clearance of many vessels. In any individual vessel, the “proportion” of constriction versus thrombosis responsible for final vessel clearance is dependent on vessel diameter, radiant exposure, wavelength, pulse duration, and spot size (vide infra) [32].

In summary, we suggest that long pulses and long wavelengths are the best approach for side effect free clearance of leg veins 0.2–2 mm in diameter. The only exception is treatment of very small vessels (0.1–0.6 mm) in extremely fair skin. In this scenario, vessel clearance with GY light is equivalent to 1,064 nm; however an advantage of GY light is that treatment tends to be less painful with equal spot sizes and equal immediate responses. Large spots, regardless of wavelength, result in more pain and a higher risk profile than smaller spots. Closure of smaller blood vessels, regardless of spot size, results in less pain than closure of large vessels.

Of the two primary clearance mechanisms, vessel contraction is favored over thrombosis. Shorter pulses

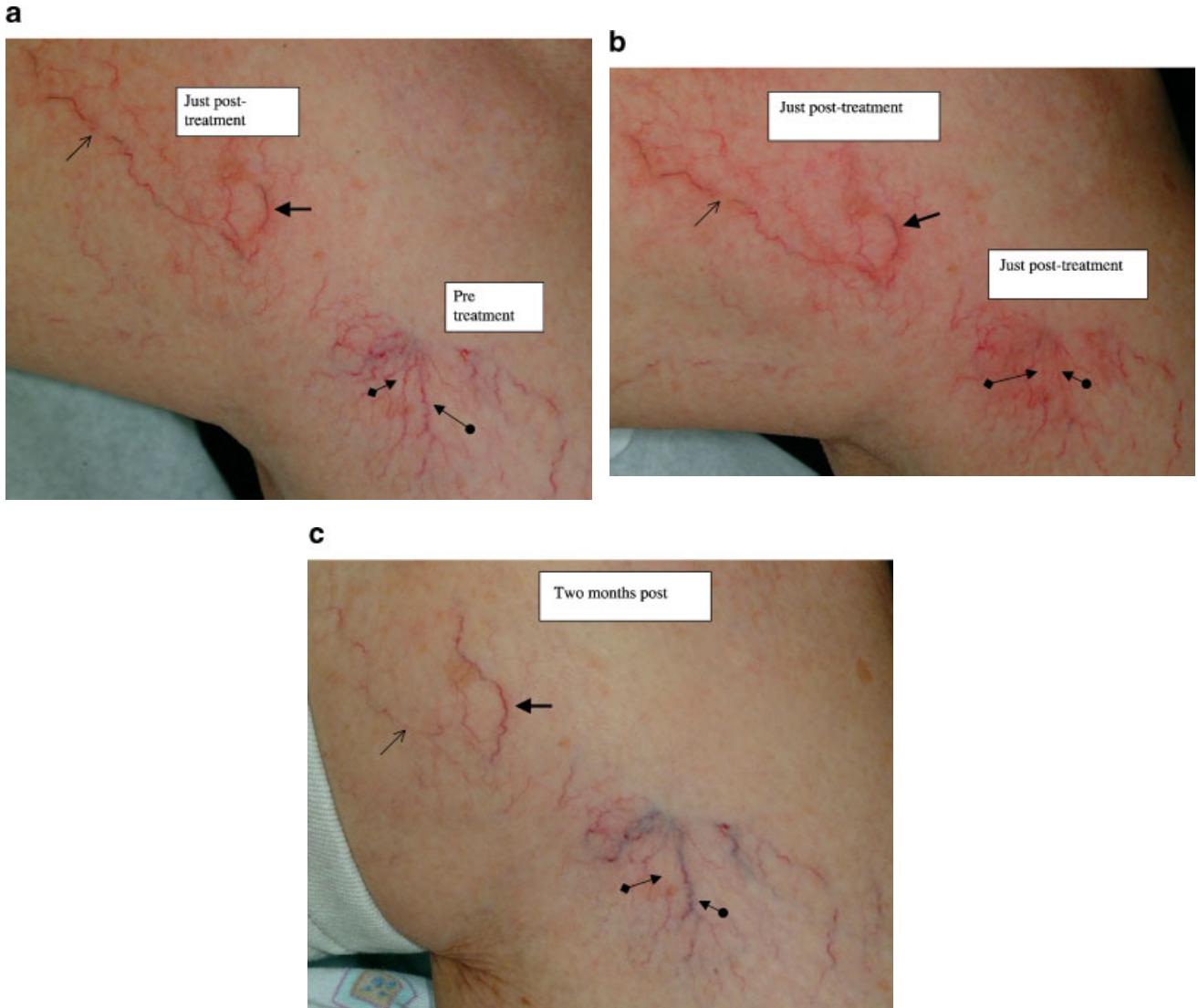


Fig. 12. **a:** Inner thigh: 3-mm spot, 280 J/cm<sup>2</sup>, 60 milliseconds, cryogen spray cooling; inferior portion is just before treatment while superior area is just after treatment—thin arrow at top shows immediate stenosis, thicker arrow at top shows early evolving thrombus and coagulum. At the bottom of photo, two arrows point to untreated vessels. Vessels at upper thigh before treatment were similar to the inferior vessels. **b:** Both areas just after treatment—at bottom of photo, both sites

(arrows) show partial vessel stenosis. **c:** Eight weeks later, variable responses are noted. Upper part of photo shows persistent closure where early stenosis was noted, whereas area that initially showed thrombosis has not cleared. Lower part of photo shows one cleared vessel and one vessel with delayed/persistent thrombosis (even though both showed early partial contraction, b).

favor wall rupture and thrombosis [38]. Longer pulses result in greater contraction in blood vessels over the range of diameters considered. Most likely the greater efficacy stems from longer heating times and possibly methemoglobin formation (vs. shorter pulses). “Slow” vessel heating appears to be the optimal mechanism for permanent closure, so parameters should be designed to optimize wall contraction [31]. Methemoglobin can play an important role at the 1,064-nm wavelength and should be considered when designing treatment parameters.

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