

Surgical Laser Management of Common Foot Disorders

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Laser surgery as utilized in podiatry has undergone a significant evolution since its inception in the 1970s. Historically, the first operating laser was the ruby laser constructed in 1960 by Theodore H. Maimon, a scientist associated with Hughes Aircraft Research Laboratories (1). The carbon dioxide (CO₂) laser, conceived by Patel in 1965 (2), has been and still is the most widely used laser for a variety of podiatric applications. The advantages of laser surgery have been well documented (3-5), whereas its risks and complications have received less attention (6-9).

The laser has been theorized as having the advantages of complete eradication of the target lesion via its photoablative properties. Associated with this function is wound sterilization (10) by which pathologic organisms or cells are destroyed, thereby precluding spread to adjacent tissues. Lanzafame et al. (11) stated, "Our work supports . . . that the CO₂ laser is effective in the reduction of local tumor recurrence. Furthermore, there was a significant delay in the appearance of recurrent tumor in the laser treated animals compared to their scalpel-treated counterparts. These favorable results . . . occur as a result of the no touch surgery, destruction of cells and cell nuclei in the beam path, and the sealing of small vessels and lymphatics."

The patient should experience less postoperative discomfort and edema because of the laser's pinpoint accuracy and minimal adjacent tissue damage. Thermal effects of the laser are responsible for

the coagulation of blood vessels and lymphatics, resulting in a bloodless field and sealing of nerve endings. According to McKenzie and Carruth (5), "laser seals nerve endings which contributes to the remarkable lack of post-operative pain." Many of these advantages are founded in scientific and clinical fact, whereas others are being questioned.

Other lasers, such as the neodymium:yttrium-aluminum-garnet (YAG) (12), argon (13), helium-neon (14), erbium:YAG (12), copper vapor (15), eximer (16, 17), and holmium:YAG (18), may have future podiatric applications. Generally, the most common podiatric usages for the CO₂ laser are the treatment of verruca (3, 19-25) onychocryptosis (3, 26-31), onychomycosis (3, 26, 29, 32-34), and neuroma (4, 35) and the excision of benign skin lesions such as porokeratosis (4, 36). Other less common uses are for heel fissures (36), burns (37-39), keloids (40, 41), ulcers (42, 43), and spider veins (41). Investigational laser applications include induction of cartilage regeneration (44). Other proposed uses are for soft tissue dissection (45) and bone and joint surgery (12, 16, 18, 46-56).

THE PHYSICS

The CO₂ laser emits a beam with a wavelength of 10.6 μ . This places the CO₂ light in the invisible mid-infrared portion of the electromagnetic spectrum. For this reason, a helium-neon laser, whose beam lies in the visible red part of the spectrum, is coaxially aligned with the CO₂

laser for use as a tracer or identification beam. At the above wavelength, the CO₂ laser energy will be absorbed by any tissue containing water.

Vaporization is the energy-dependent conversion of a liquid to a gas. Since the living cell is mostly water, the laser energy delivered to the tissues in the form of heat is rapidly absorbed by the intracellular water. This photoablative heat energy raises the basal body temperature of 37°C rapidly to the heat of vaporization, which is 100°C. As the intracellular fluid is heated by the absorbed laser radiation, the vapor pressure inside the cells rises. As soon as it surpasses the surrounding pressure, the cells expand until their membranes rupture and subsequently burst, thereby destroying them.

At 65°C biologic protein is denatured. Collagen is the predominant protein component of dermis. Certain levels of skin will therefore display a profound "shrinkage" effect when exposed to this sub-heat of vaporization laser energy, between 65°C and 99°C. This phenomenon is due to the denaturation of the skin's protein constitution (5, 57-59).

When all the water has been eliminated, continued irradiation rapidly increases the temperature of the residual material until a temperature between 300°C and 400°C is reached. At this point, the tissue blackens, becomes carbonized, and begins to outgas and smoke, leaving behind a black residue called carbon char. With continued re-lasing of this carbon char, the temperature could reach as high as 4000°C. A visible white hot spot of ultraviolet light will form. This phenomenon is known as carbon arcing. Its effects are reviewed in the section on laser safety.

The smaller particles of cellular debris are released into the air as the plume. Much controversy exists concerning the viability of cellular components within the plume and the results of long-term exposure to inhalation of these compo-

$$\begin{array}{c}
 \text{TISSUE EFFECT} = \\
 \boxed{\text{SPOT SIZE}} \cdot \boxed{\text{POWER}} \cdot \boxed{\text{TIME}} \\
 \text{(cm}^2\text{)} \quad \text{(Watts)} \quad \text{(sec)} \\
 \hline
 \text{POWER DENSITY (W/cm}^2\text{)} \\
 \hline
 \text{TOTAL ENERGY} \\
 \text{RADIANT EXPOSURE (joules)} \\
 \hline
 \text{FLUENCE (J/cm}^2\text{)}
 \end{array}$$

Figure 3.1. Laser power, beam spot size, and time are variables utilized in formulas and terminologies when expressing the laser's effect on tissue.

nents as well as gaseous and particulate matter debris. The plume's effects on surrounding tissues as related to the seeding of pathologic cells or cellular debris is also of concern.

The CO₂ laser is a class IV laser according to the American National Standards Institute (60). This is a laser or laser system that can produce a hazard not only from direct or specular reflections, but also from diffuse reflection. In addition, such lasers may produce fire and skin hazards (6). When used improperly, this laser can become a very destructive force.

The components of the laser that determine its effect upon the tissue are (a) power (wattage), (b) laser beam spot size, (c) handpiece distance from the tissue, (d) type of beam (focused or defocused), and (e) time. The power is set on the laser unit by the surgeon in accordance with the specific procedure being performed. The focal spot size is predetermined by the handpiece focusing lens. Actual spot size is determined by the handpiece distance from the tissue and whether the beam is focused (focal spot size) or defocused as it makes contact with the tissue. The volume of tissue within this spot is used in many mathematical equations for determination of laser power. Time is the most variable component in determin-

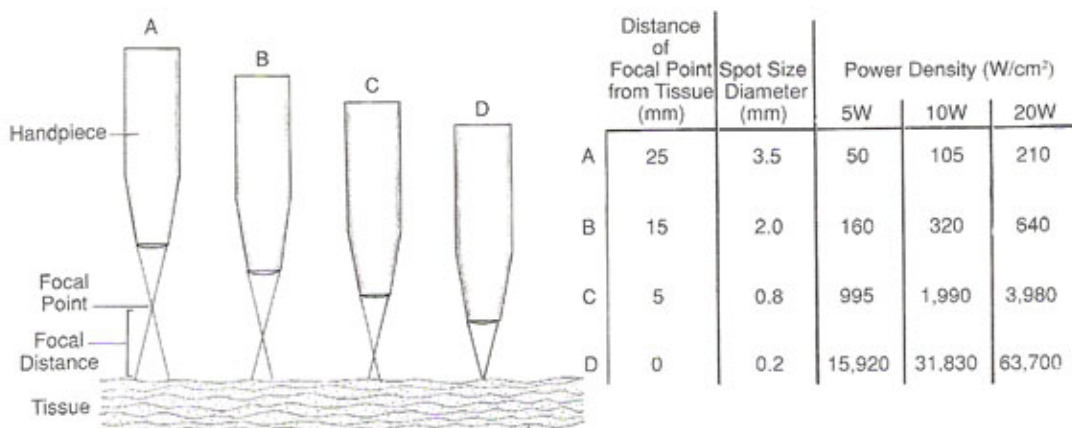


Figure 3.2. Diagrammatic representation of how variation of the laser handpiece distance (and fixed focal point distance) from the tissue relates to alterations in spot size diameter and ultimate power density. Different power settings are represented. Note that the closer the tissue is to the focal point, the smaller the spot size and therefore the greater the power density.

ing the laser's effect upon the tissue. When the beam is held stationary, the time the beam is in contact with the tissue is determined by the time the foot pedal is depressed, activating the laser unit. Time in contact with the tissue could also be varied by the movement of the handpiece itself. The more rapidly the laser beam is moved across the tissue surface, the shorter is the duration (exposure time) of the actual laser-tissue contact. Likewise, the slower the movement, the longer duration of contact.

Laser energy is measured at the tissue in terms of *power density*. The relationship of power and spot size come together in the formula for power density. Expressed in watts per centimeter squared (W/cm²), the power density (PD) is:

$$PD = \frac{(\text{power in watts}) \times 100}{(\text{spot size in mm})^2}$$

This formula, however, does not take into consideration the laser's effect on the tissue. When the variable of the laser beam's time in contact with the tissue (exposure time) is added, we measure this influence as *fluence*. The formula for fluence, expressed in joules per centimeter squared (J/cm²) is:

$$\text{Fluence} = \text{power density} \times \text{time}$$

Fluence is the energy per unit area. Total energy can be measured using the following formula:

$$\text{Total energy (J)} = \text{power (J/sec)} \times \text{time (sec)}$$

Since the total energy encompasses both the rate of flow of energy and the time of exposure, it helps determine the volume of tissue affected (Fig. 3.1).

The significance of the aforementioned variables is that they all play an integral part in the depth of penetration and subsequent tissue damage. For example, if the laser handpiece is held at the correct distance from the tissue—that is, at the laser beam's focal length (focused) with a 0.2-mm spot size and 10 W of power—the power density would be almost 32,000 W/cm². By altering the handpiece distance such that the spot size becomes 2.0 mm (defocused), the power density would be reduced to 320 W/cm² (Fig. 3.2). If time is now incorporated as either the duration of the interrupted or pulsed-mode impact bursts or the speed at which the continuous-mode beam is brushed across the tissue, the total energy delivered to the tissue will be affected. By

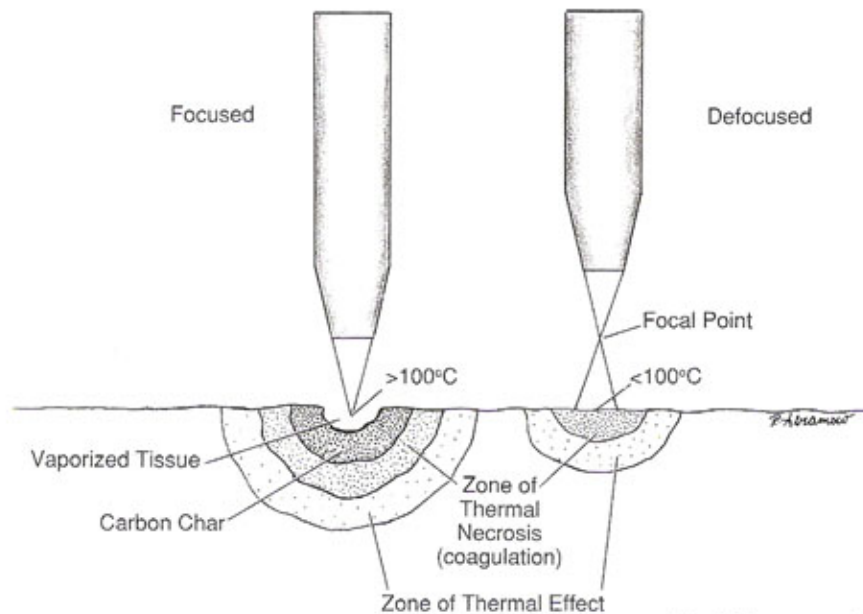


Figure 3.3. Levels of tissue effect based on focused and defocused beams. The higher power density produced by the focused beam produces tissue temperature in excess of 100°C, and therefore vaporization and charring. The defocused beam produces lower power density when the handpiece is moved away from the tissue, resulting in tissue temperature below 100°C. With a low power density only superficial necrosis (coagulation) occurs. The laser energy penetrates the tissue without vaporization or charring.

increasing the beam's time in contact with the tissue, the fluence is increased when using a low power density. By decreasing the time, a lower fluence results when using a high power density. Here, the laser beam's effect on the tissue as related to depth of penetration and tissue damage is altered by the power density and, more importantly, by the beam's duration of the application to the tissue (time).

When the laser burst impacts the tissue, a central carbonization zone or carbon char is formed, surrounded by a zone of thermal necrosis and then by a zone of thermal effect (Fig. 3.3). This central carbonization zone is the result of superheated cellular debris. The zone of thermal necrosis is a nonviable area surrounding the carbonization zone. It is the result of heat coagulation—that is, transmission of photocoagulative heat energy of such intensity as to render cell death. The outer zone of thermal effect is a viable cell zone. These cells

have been affected by the heat transmission, yet no destruction of cellular architecture has occurred. This zone will mend itself, usually within a short period of time.

When working with a focused laser beam at a high power density, the temperature at tissue level rises rapidly to 100°C and in most cases greatly exceeds this level. Tissue vaporization occurs, and the respective zones are apparent (Fig. 3.3). When the laser beam is defocused, in contrast, the same power is now spread over a larger surface, resulting in a lower power density. The tissue temperature in this case does not reach 100°C, so that vaporization and charring are absent, and tissue coagulation occurs over more superficial levels (Fig. 3.3).

Here, again, it is of utmost importance to appreciate the relationship of power density and time as these variables relate to tissue effect. A high power density and short duration of application of the laser beam will result in

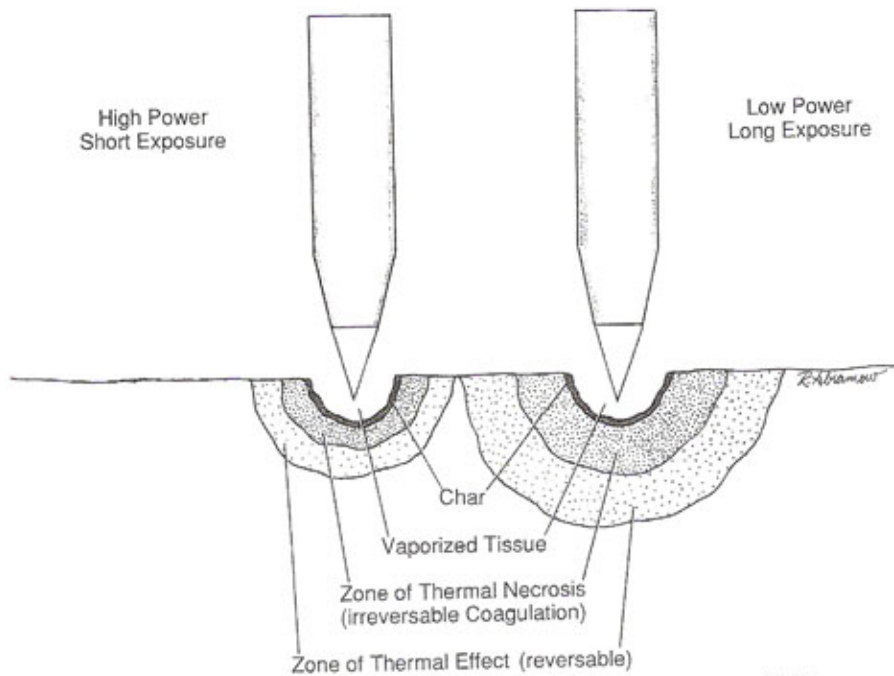


Figure 3.4. Tissue effect of high power setting and short tissue exposure compared to lower power setting and longer tissue exposure. Note similar amounts of tissue vaporization, yet significantly greater zones of thermal necrosis and effect with longer tissue exposure to the laser beam.

atraumatic photoablative effects with minimal zonal thermal necrosis, whereas a low power density and increased time may result in greater photoablative effect and greater zones of thermal necrosis and thermal effect. Either mode may produce similar vaporization zones, yet the less heat transfer through the tissue, the less thermal damage (Fig. 3.4). Proper understanding and application of these relationships will enable the laser surgeon to capitalize on the many advantages of CO₂ laser surgery. Thus, knowledge of the various combinations and integrations of all the aforementioned variable factors by the experienced laser surgeon would yield destruction of the lesion with minimal adjacent tissue damage, whereas, in inexperienced or unknowledgeable hands, utilization of laser technology in laser surgery would be prone to result in many complications caused by excessive tissue damage.