Dedicated to
Deborah, Lucius and Maria
ACKNOWLEDGEMENTS

There are many broad shoulders upon which this book stands. First of all, there are the pioneers of retinopathy treatment that have given us the tools that we have and the elegant studies that tell us how to use them. There are also the folks all around the world that are trying to provide even better therapies—they intuitively grasp the infinity of things not covered in Chapter 2. On a more personal level, I owe a great debt to all the attending physicians and ancillary staff at the Ohio State University, the USC/Doheny Eye Institute and the University of Iowa. They are not only busy performing all the tasks mentioned above; they also had to suffer through training me. Any bad advice found in this book, however, is something I made up and not something they taught me.

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Thanks to my editor and publisher, Lauren Fath, who had the yeoman’s work of making the text readable and did the infinite number of things required to shepherd a book into your hands. Also thanks to M. Walt Keys, who did the layout and design and found all the cool images at the beginning of each chapter (the symbolism of which, given the new treatments we will soon have, should be obvious). Their creativity, energy and enthusiasm made assembling the book effortless and a true pleasure. (Their contact info is in the front matter if you have a book in you that is hankering to get out.)

The marvey line art was provided by Roberta Sandy-Shadle and the photos in Chapter 7 were done by James Whitcraft—both at Indiana-Purdue University, Fort Wayne. Mike Neeson of Iridex helped out by confirming my memories of lasers of old, and Larry Hubbard of the Wisconsin Reading Center generously shared his Zen Master knowledge of retinopathy grading.

Thanks to my partner—Matt Farber—who actually saw the patients while I was locked in my office Photoshopping laser spots, and thanks to my exemplary office staff for keeping everything going when I wasn’t. Of course, there are no words to thank my wife and kids for doing all the real work while I alternately napped and typed on the couch. No soy nada sin ellos.

Finally, thanks to the referring doctors for entrusting me with their patients and especially thanks to the patients themselves, who extend the ultimate honor of entrusting us with their eyes.
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This book is designed to transfer useful skills for the clinical management of diabetic patients. It does not start with the fundamentals; instead, it is assumed that the reader has basic examination skills and is at least partially familiar with various tests, such as fluorescein angiography and optical coherence tomography.

Nor does this text offer an in-depth discussion of basic science or an exhaustive review of the available literature. Like the Basic and Clinical Science Course from the American Academy of Ophthalmology, there are some references given at the ends of most chapters for further information. However, if you want an in-depth look at the literature behind treating retinopathy, you are encouraged to review the sections on diabetes in any of the major ophthalmology texts, and in particular, Ryan’s retina text. The American Academy of Ophthalmology’s Focal Point from March 2003 on diabetic retinopathy by Drs. Fong and Ferris is also an excellent and succinct review.

Simply put, the goal of this book is to help make the trenches where most of us live a bit more comfortable.

The voice of this text is different from standard texts—something done in hopes of conveying useful information without too much tedium. However, as a wise person once said, “There is a fine line between clever and stupid.” If anything offends or interferes with the smooth download of information, let me know.

Also, there are no absolutes here. Once you think you know the best way to do anything, you have lost the ability to learn. Try these suggestions and techniques, and if they don’t work, throw them out. Run them by your mentors and your friendly neighborhood retinal specialists—get other opinions and synthesize a style of your own. I welcome any comments and/or complaints. If the gods of retina smile on this book, then perhaps there will be better editions with plenty of input from people way smarter than I am. My contact info is below.

Mostly, I hope that you can peruse these pages and find something that will help you to help patients who have one of the most prevalent and vicious causes of blindness on this planet.

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P.S. At various points in the text, there are unavoidable opportunities to harass our surgical colleagues who have mastered more refractively oriented procedures. Recognize that this is meant in good sport and, in truth, stems largely
from professional jealousy—they can actually understand things like high order aberrations and apodized lenses and they have patients who hug them after surgery.

Retina specialists do not generally get hugged by their patients. Moreover, the only bit of optics we understand is The Retina Refraction: room lights on—better one; room lights off—better two.

Onward…
A Tiny Bit of Statistics and a Big Pep Talk
First, some really big numbers: An estimated 20.2 million Americans have diabetes mellitus, and the number is expected to grow to over 30 million cases by the year 2025. Thanks to exports like the Great Western Lifestyle, the number of worldwide cases is expected to increase by 72%—to 333 million—by the year 2025. That is a lot of microaneurysms. By contrast, currently the number of patients blind from cataracts worldwide is estimated to be 18 million people. In other words, although a lot of ophthalmic effort is (correctly) directed towards decreasing the worldwide cataract burden, the number of patients at risk for vision loss from diabetes will soon be almost 20 times greater. Moreover, once a cataract is popped out, the job is done. Treating diabetes goes on forever for both the patient and physician—it ain’t one-stop shopping.

Diabetic blindness also tends to occur at a time when people are younger and more active in society; it is the leading cause of new blindness in patients under the age of 65. The rate of onset is variable, but after 20 years, about 60% of Type 2 and essentially all Type 1 diabetics will have some sort of retinopathy. You will spend a great deal of time caring for these patients. It may seem that the treatment of diabetic retinopathy has been tremendously streamlined with the help of large clinical trials with which you are no doubt familiar. However, the reality is that each patient you see presents an incredibly complex array of variables—social, emotional, physical and retinal. Addressing all of these variables requires a lot more than the ability to memorize the definition of clinically significant macular edema. It is axiomatic that we all went into ophthalmology to avoid dealing with the morass of an entire patient. Unfortunately, when it comes to treating diabetic retinopathy, your results are going to suck if you don’t start by understanding the entire patient. At the very least, recognize that by the time a diabetic needs your help, they are usually facing the risk of irreversible vision loss—real, life-changing, disabling vision loss—not Nerf vision loss that can be fixed with Lasik.

**Dharma break:** Each diabetic patient whose vision you save probably represents more quality-of-life units than a whole surgery schedule full of 20/30 glare cataracts. Think about it…

And the battle is bigger than just honing your clinical skills and trying to deal with the entire patient. At the risk of sounding hyperbolic, you also have to look at the society in which you function. It has been said that if patients are examined in a timely fashion and the standard treatment guidelines are followed, less than 5% of diabetics will develop severe vision loss. A huge part of your job lies in recognizing the importance of the first clause of that sentence: *if patients are examined in a timely fashion.* Not only do you need to develop the ability to treat these people, but you also have to be aggressive about getting them in to be seen. Far too many diabetics show up only when they start having symptoms, and this is just not the best way to keep people seeing.

Educate the patient and the patient’s physicians at every visit. Educate the
patient’s family about the importance of getting everyone in the family routinely checked for diabetes and getting anyone who is diabetic in for an annual exam.

Educate society. Give talks at local diabetes support groups. Offer to provide information for the health desk editors at local newspapers, magazines or TV stations. Get involved with efforts to provide universal coverage. Do the free clinic thing. Make general information slides for the local cinema multiplex so they can be interspersed with all of those fascinating questions about which actor said what in which movie. Whatever. Just get these people in.

(All this may not only help prevent blindness; it can also help build your reputation and your practice—a twofer! Watch the ethical ramifications, though. It is one thing to generate public service messages that help patients and their doctors. It is quite another thing to plant your smiling face on an ad that says you are the bronzed god or goddess of retinopathy. This is a test…)

Unfortunately, a large part of your diabetic-treating career will consist of taking care of sad cases that didn’t get in—hence the rationale for books like this. Helping a pair of eyes, and the patient attached to them, by slowing their descent into severe vision loss is still a good thing, but not a lot of fun. It is way better to body-slam the retinopathy before it can get to the fovea or up into the vitreous, but you can only do this if you see the patient before the real trouble begins. Aggressive monitoring and treatment can easily keep someone seeing until well after they leave the planet, which is something that you will hopefully be able to do many times and for many people before you hop off the globe, too.

References and Suggested Reading


Diabetic Retinopathy
Didn’t you read the intro? This is not a basic science text. If you want basic science, get a real textbook. Or go to ARVO. Sheesh...
ch. 3 / Know Your Weapons — Lasers and Their Ilk
PART A. Laser Physics for Wimps

This section really does not have a heck of a lot to do with patient care issues, but it is useful to have some idea about how the little demons inside the laser box do their thing. First of all, it is impossible to talk about lasers without rehashing the acronym. At some point in your career you may come across a pedant that uses your ability to regurgitate the meaning of “LASER” in order to determine whether you are a worthwhile physician. So never forget that laser stands for Light Amplification by Stimulated Emission of Radiation. But what does that mean?

The fundamental thing to remember is that the electrons orbiting the atomic nucleus want to ditch their extra energy and get to lower levels. This process results in the emission of photons.

It turns out that electrons can release photons of only certain wavelengths because electrons can only live in certain orbitals, which are determined by the atom in which the electron resides. If the electron falls to a lower orbit, the electron releases a photon whose energy corresponds exactly to the difference in energy between the two orbitals—no in-betweeners allowed. The electron can also be bumped up to a higher orbital if it happens to absorb a photon of the exact energy that matches the energy difference between the lower and upper orbitals. If you can bump a bunch of electrons up to a given orbital and then get them to drop back down to a lower level at the same time, you can—for instance—stop diabetic retinopathy.

Getting the electrons to do this involves a weird and mysterious variation of the whole bumping up and dropping down process (this is where the Stimulated Emission part comes from). It turns out that if a photon happens to have the same energy as the difference between the pumped-up orbit where an electron is and the next-lowest orbit, and if said photon happens to pass by one of these electrons—without hitting it—the photon will stimulate the electron to drop into the lower orbit and produce a second photon that is coherent (meaning the peaks and troughs of the waves of both photons occur at the same time). It is this rather amazing property that allows the production of laser light from a host of stimulated electrons. Furthermore, a given photon can stimulate a whole bunch of electrons as it whizzes by, and each photon released will go out and stimulate the release of even more photons (the Light Amplification part of LASER).

In 1917, Albert discovered that the oscillating field of the stimulating photon perturbs the electron’s field, which causes it drop to the lower energy level sooner than it otherwise would. It took several decades, however, to turn this theoretical knowledge into something that even an ophthalmologist could use.

Older lasers use some type of gas to provide a population of high-energy electrons for this process to occur. You can usually identify a gas ion laser because it tends to be large with big black cables running from the laser to the wall; many
such lasers are also water cooled, which adds a gurgling-broken-toilet ambi-
ence to the treatment experience. The gas molecules are “pumped” by either an
electric discharge or a powerful light source, which creates a large population of
high-energy electrons. There is also a fully reflective mirror at one end of the gas
tube and a partially reflective mirror at the other. This makes the photons bounce
back and forth a bit, which ensures that as many electrons as possible are
stimulated to drop to a lower orbit and release a photon. Only a small amount of
photons escape through the partially reflective mirror and this, in turn, produces
the laser light that you then put into a patient’s eye.

Nowadays, most lasers generate coherent light from a light emitting diode; such
lasers tend to be much smaller and look not unlike a home theatre amp, but with
fewer buttons. These diode, or solid-state, lasers are a bit more complicated to
explain. They involve things such as electrons moving from high-energy con-
duction bands to low-energy valence bands, skipping altogether the delightfully
named “forbidden region” of energy. This sets up a situation where stimulated
emission can occur in a chunk of matter that is much smaller than the gas tube
of an older laser. Electrical energy is used to shove electrons into the higher va-
lence levels and the release of photons is stimulated at the junction of the diode.
The diode itself is still sandwiched between mirrors, just like in a gas tube laser.
The whole process is far more efficient than in a gas laser, hence the lack of big
cables and pipes which made gas ion lasers unsuitable for use as laser pointers.

Lasers used to treat retinal diseases are known as continuous wave lasers
because the laser beam can be generated, well, continuously. The user sets
the actual duration of the beam, and the power output is relatively low, which
allows a gradual, controlled response in the target tissue. This is in contrast to
the “pulse lasers” that are used in ophthalmology—the neodymium: yttrium-alu-
minum-garnet laser (Nd: YAG) and the excited dimer laser (excimer). This type
of laser puts all of its energy output into a very brief period of time. Because the
energy is the power per unit of time, a laser pulse released in a very short time
can have a very high peak power, which, if focused in a small spot, can reach an
extremely high power density (irradiance) and can essentially be explosive.

The frequency of light generated by a laser depends on the substance being
used to generate the light. If the frequency produced is not ideal for the chosen
application, it can be changed by using either harmonic generation or organic
dyes. An organic dye laser can produce a spectrum of wavelengths, but such
lasers are inefficient—a lot of energy is lost when the primary laser is fired into
the dye to excite and lase its fluorescence spectrum in the dye laser cavity. Dye
lasers tend to be expensive and difficult to maintain, and you are not likely to
see such a laser nowadays.

Harmonic generation is a far more common technique for changing a laser’s
frequency. In this case, the laser light is passed through a special crystal that will
vibrate at the laser’s frequency and generate harmonics that are multiples of the
laser’s frequency. Such crystals are commonly used to double the frequency of the output of a YAG laser in order to produce a wavelength in the green spectrum (i.e., from 1064 to 532 nm). A typical diode green laser generates light in this fashion.

All of this is a horribly oversimplified explanation of one of the mainstays of retinal therapy. If you ever want to feel overwhelmed, pick up a bona fide textbook on lasers to get an idea of how complex they really are. Ultimately, we all have to be very grateful for the fact that there are plenty of good folks out there that actually understand this stuff on a fundamental level and are always working to give us better and better tools. This way we can concentrate on part B.

PART B. From Acronym to Verb: Lasering People

Once you manage to get your hands on a laser and point it at a patient, you can expect three types of tissue interactions, depending on the nature of the laser: photocoagulation, photodisruption and photoablation. These categories are a bit arbitrary because they are really part of a spectrum of how tissues respond to laser energy. It is convenient, though, to use these terms to distinguish the tissue effects of the different types of ophthalmic lasers. For instance, if you devote your life to fighting the demon scourge known as spectacles, you will depend on photoablation to provide your worldly needs. In this case, an excimer laser generates a wavelength of 193 nm (in the ultraviolet range), which can break chemical bonds. This allows very precise removal of tissue with only minimal damage to the surrounding structures. Photoablation is definitely a “now you see it, now you don’t” kind of thing.

When you perform a YAG peripheral iridectomy or capsulotomy, you will be depending on photodisruption. This is more of a mechanical effect that results from tightly focused, high-power laser light, which produces an explosively expanding vapor bubble of ionized plasma. This bubble then quickly collapses, producing acoustic shockwaves that happily blow apart the structure you are treating. This is very satisfying from a single-player-shooter point of view, but it is not particularly user-friendly when you want to treat something delicate and squishy like the retina.

Retinal laser treatment depends on the far more gentlemanly tissue effect known as photocoagulation. In this case, the laser literally cooks the tissue at a microscopic level. The resulting coagulation of proteins causes the desired effect—hopefully without any photoablative or photodisruptive pyrotechnics.

There is a fourth tissue interaction—photochemical—but you are unlikely to use it. In this case, a very low-power laser is used to activate a specific chemical to obtain the desired effect in the tissue. The use of a red laser to activate verteporfin (Visudyne) in order to treat neovascular age-related macular degeneration is the best example of this.
Regardless of how the laser is produced, there are certain variables that you need to intuitively understand if you are going to treat patients safely and effectively. The first one is the wavelength of the laser you use. Figure 1 is the classic display of how each laser color is absorbed in various ocular tissues. For a long time, people hoped that different colors would allow one to customize the treatment depending on the indication. For instance, you can see that yellow really nails one of the peaks of oxyhemoglobin relative to green, and if you ever have occasion to use a yellow laser, you can detect a significant difference in how, for instance, microaneurysms respond to a different wavelength (they tend to be very easy to pick off with the yellow—often with very little disruption of the retinal pigment epithelium and outer retina).

Perhaps more clinically significant is both the marked dropoff in hemoglobin absorption and the gradual dropoff in melanin uptake as you move into the red end of the spectrum. This explains in part why red and infrared burns require more power and tend to penetrate deeper into the more pigmented choroid. It also helps explain why the infrared diode laser in particular is so different to use relative to a green laser.

**Figure 1.** The absorption of different laser wavelengths by different substances in the retina, RPE and choroid. Note that, in general, the further you go toward red, the less the absorption—hence the need for more power and a resultant deeper burn with longer wavelengths. You can also see how yellow hits a peak of oxyhemoglobin absorption relative to green, which accounts for the difference in how microaneurysms are affected by each wavelength. Finally, you can see why it is a very bad idea to use blue light anywhere near the fovea, where xanthophyll pigment is found. (Data from Mainster MA. Wavelength selection in macular photocoagulation. Tissue optics, thermal effects, and laser systems. Ophthalmology 1986;93:952-8.)
However, although one does get different tissue responses depending on the wavelength, no one has proven that there is a huge difference in the ultimate treatment effect. Besides, you will basically be using whatever laser has been plopped in your clinic because there is no way you can go out and shop and compare with these enormously expensive devices. Fortunately, most of the studies on diabetic retinopathy were performed using some sort of green wavelength—usually argon green or its kissin' cousin diode green—and that is pretty much the standard color of laser found anywhere. Some places do have the fancy lasers that can generate different colors, and you should experiment with these colors for yourself. (Plus, there are few things cooler than the appearance of yellow or orange light coming from an ophthalmic laser.) Because multicolor lasers are as rare as Bugattis, though, the rest of this book will assume you have some sort of green laser to work with. Go Irish.

**You may read references to “argon blue-green.”** This is because 488 and 514 nm wavelengths were simultaneously available on older argon lasers. The 488 nm blue wavelength became déclassé and was eliminated because of increased uptake by xanthophyll pigment in the fovea and an increased risk of causing burns in yellowish nuclear sclerotic cataracts. (Look at the uptake of xanthophyll in Figure 1—this is not a subtle effect.) If you have an argon laser that actually has both lines don’t ever use it for treating the retina, unless the blue line has been properly suppressed. You might be able to sell it on eBay as an antique, though. Ask your chairman first.

The one thing to remember for sure with any wavelength is what you learned in second-grade science class: Black absorbs everything and white reflects everything. In other words, if you are treating happily in an area of the retina and you come upon a dark area like a nevus or a previous laser scar, you need to watch out, because you can get an explosive burn as the pigment sucks in the laser (higher absorption translates in higher photothermal elevation). Remember to Turn It Down When You Hit Brown (and Cut Way Back When You Hit Black). Alternatively, if you need to treat a very pale area, you will need to crank it up—but be super careful when you hit pigment again.

**FLUENCE**

Although wavelength is fun to theorize about, the power density, or irradiance, and the energy density, or fluence, of the laser beam are the most important concepts to master if you are going to be a safe and effective laserist. Here, for completeness, are the only formulas in the book:

\[
\text{Irradiance (W/cm}^2) = \frac{\text{Power (Watts)}}{\text{Spot Area (cm}^2)}
\]

\[
\text{Energy (Joules) = Power (Watts) } \times \text{ Time (Seconds)}
\]
We will try to stay away from the obligatory discussion of energy, work, radiometric terminology, etc. that often shows up at this point in real textbooks. The key thing is that your laser output has a certain level of mojo and you need to know exactly how to control it.

Look at the last equation for fluence. Note that going up or down on power (Watts) or on exposure duration (time) creates a linear increase or decrease in the energy delivered. This means that if you are getting a good burn and you decide to, say, double the exposure duration, then you have to decrease the power or you will really cook things. It is hard to imagine why on earth one would want to do this when one is getting a good burn, but this is always mentioned in basic laser texts and it does help ensure that you understand the relationship. The really significant thing is that the clinical effect tends to be very intuitive—a mild increase in the power or duration will give you a mild increase in your burn, and the same is true if you want to turn things down.

However, because we are dealing with a biological system and not a photometer, it turns out that the relationship between the energy delivered and the type of burn that you get is a bit more complex. The exact same energy can result in different burns because the burn depends on how the laser is absorbed and how the heat is transmitted by the tissue. In other words, fiddling with the laser power and duration generally results in a common-sense change in the degree of uptake—a little more time or power results in a little more burn and a lot more time or power results in a lot more burn. But don’t depend on this absolutely. Let’s take another colorful box break.

**Colorful Box Break:** Since you are basically using your laser to warm up the retina, you do need to be careful about using high powers at short duration, because the nice linear relationship breaks down and you can end up microwaving the proverbial poodle of urban legend.* Your “typical” laser burn is determined not only by the energy density but also by the rate of heat transfer out of the burn area. Unfortunately, heat transfer is governed by factors far more complicated than the weenie-pre-med-physics equation above. For instance, heat transfer explains why it is easy to get a burn in the retina but really hard to get a burn on a big blood vessel—the blood “carries” away the heat and you can’t get the vessel wall to cook easily. Because you should treat a big vessel exactly never, the real point of all this is that if you use a lot of power over a really short duration, there is not time for the heat to spread out and you can get a much hotter burn than you would expect if the response of the tissue were truly linear. To repeat: Stuffing a lot of power into a short duration can become explosive—you are going from a slow cook to a fast boil. This will be important in the next paragraph…

(*Yes, this is a sophomoric metaphor, but if sleazy skull imagery will help you remember this point it is worth it.)
THE EFFECT OF SPOT SIZE

Going back to the mini-equations above, note that the irradiance (or power density) and the fluence (or energy density) are an inverse function of the square of the spot size—i.e., a small change in spot size can make a big difference in the irradiance and in the fluence you pour into the retina if you don’t compensate by changing the power or duration. With a lot of energy delivered into a small spot, you can create a “YAG effect” because you will raise the temperature so fast and so high that the water in the tissue will actually boil. This is especially likely if you are also using a brief duration (less time for heat transfer, remember). The result is an explosively expanding bubble of water vapor that will cause a hole or hemorrhage or both. (Technically, it won’t be a true YAG photodisruptive effect—there won’t be any plasma formation—but the explosive vaporization of water in the tissue will have the same destructive physical effect, complete with a sickening pop-like sound in the patient’s head. You can really mess up an eye doing this—and lose lots of style points with your patients and colleagues. We will return to this concept several times in this book to be sure it sinks in—it has to be internalized to your lizard brain parts just like the mental switch that keeps you from engaging phaco when you are next to the posterior capsule.)

Anyway, if you make the spot size smaller—even if it is only a little bit smaller—you really have to be religious about decreasing the other parameters so that you don’t start punching holes in the retina. For instance, you might be using a strong power to cut through media opacities and you might also be using a short duration to try to make the laser less painful for the patient (no worries—much more on these techniques later). You might then decide to decrease the spot size in order to get an even better burn—a smaller spot will not spread out as much as a large spot if the view is hazy. If you do this, then you must cut back on power and work your way back up to a safe burn; otherwise, you will have increased irradiance and fluence by the square of the difference in spot size, and you will very likely cause a dangerously hot burn. Repeat: You must cut back on power and work your way back up to a safe burn if you decrease the spot size.

Also, remember that your spot size is not exclusively dependent on the setting you put on the slit lamp adapter. As we will see next chapter, each type of contact lens will minify or magnify the size of the actual spot projected on the retina. If you switch to a different contact lens, you might be shrinking the actual spot size without realizing it—thus dramatically changing how much power is focused onto the retina.

There are even more ways the spot size can change unintentionally. When you are working in the retinal periphery, your spot will sometimes shrink down as you treat through the edge of the patient’s lens. Or if you are doing a macular laser in an area of swollen retina, the thickened retina will tend to diffuse the beam, and when you move to an area of thin retina, your spot effectively shrinks. Or when you are starting your laser career, it may take way longer than you want to get anything into focus and you may decide to fire away before your focus is crisp because you are frustrated. If the gods of retina then suddenly put your aiming beam into perfect focus, your spot will shrink down and suddenly you will be
burning holes in important parts of your patient. Again, all this will be covered in greater detail in upcoming chapters—but the point is that your spot size may change whether you want it to or not, and you have to be ready to anticipate these changes and alter your parameters accordingly.

There is yet another way that the biology of lasering can get you into trouble even without using small spots, and this occurs when you are using powers, for whatever reason, that are causing very hot burns. In this case, the very center of the spot can get hotter than the periphery. Heat building up in the periphery of the burn can at least dissipate into untreated retina, but heat building up in the center of the burn is trapped and cannot spread out much. The result is a sudden hemorrhage at the center of the burn if the uptake increases even a little bit (such as when going into more pigmented areas). As will be discussed in upcoming chapters, it is unlikely that you would be trying to create such a hot burn to treat diabetic retinopathy. It is important to know all the ways that things can go bad, though.

Time for a Paragraph That Begins With the Phrase “The Bottom Line…”

The bottom line is that you will have three variables that you can control from the front panel of your laser and slit lamp adapter. The power and the duration are mostly linear and tend to be fairly forgiving if you make small adjustments at a time. Spot size, however, is the one variable that is truly exponential and you have to keep this in mind if you are switching to smaller spots. You must turn down the power and titrate back up. By the way, it may seem daunting when the process of lasering a retina is “unpacked” into all these component parts. One gets a sense that it will take about 30 minutes to line up each shot after tinkering with power, duration and spot size. Actually, there are many tricks to controlling these variables quickly and effortlessly and, well, you are just going to have to read the rest of the book to find out.

OK. Suppose all of these variables seem too confusing. Let’s get basic and remember what a burn is. The retina is normally a beautifully transparent structure. If the organization of the proteins and cells is disrupted then it begins to lose its transparency, in the same way the cornea begins to become cloudy when it swells. A mild burn means that, literally, the retinal proteins are gently cooked so that the retina becomes translucent—it gets a slight grayish color as light begins to be mildly scattered. You can still see choroidal details through a light gray burn. As the burn gets hotter there is more disruption of the protein matrix and there is more scattering of light and the retina gets whiter and whiter—the choroidal detail is masked by the opaque white retina. If you are treating a patient and suddenly your burns get very white, please stop immediately and adjust your settings—the easiest thing to do is turn down the power—so that you do not start blowing holes in things.
Laser in the Infrared

Infrared diode lasers tend to be cheaper and relatively bulletproof (their design is simpler than a frequency-doubled green diode laser and they require way less fuss than a gas laser). If cost or logistical considerations are important, you may have no choice but to use infrared. This could be problematic because infrared is much trickier to use. Appendix I talks a bit about the special needs of learning this wavelength—best to carve through the basic techniques covered in this book, and then you can read the stuff in the appendix to get ready to use infrared.

Micropulse and Other “No Touch” Lasers

There are lots of wondrous things to be found in the halls of diabetic retinopathy treatment. This might be one of them. Not a common technique, micropulse laser involves delivering only a fraction of the requested power over the duration of a burn. It does this by delivering laser energy in pulses rather than continuously, and it brings into the mix a cool new term: duty cycle. This is simply the percentage of time that the pulses are actually delivering laser power relative to the total time of the exposure (Figures 2 and 3). The pulsing keeps the temperature from building up in the same way it would with a continuous wave and allows a gentle subclinical effect without the creation of a visibly identifiable burn.

**Figure 2.** Schematic of the temperature rise associated with a continuous wave laser application. The yellow bar represents the duration of the laser pulse and the red represents the rise and fall of the temperature of the treated tissues.

**Figure 3.** Schematic of micropulse laser. The yellow bars represent the effect of “slicing” up the continuous wave laser into small segments, with the wider bars representing larger duty cycles (the laser is on for a greater percentage of the duration of the exposure). You can see that the temperature in the tissue can be finely controlled to create separate elevations or gradually converging and increasing elevations depending on the duty cycle setting. (Figures 2 and 3 courtesy of G. Dorn, Ph.D., Iridex Corporation.)
This approach is felt to create a very localized treatment effect—for instance, warming only the retinal pigment epithelium without affecting the underlying choroid or overlying retina.

How does it work? Well, since no one knows how any retinal laser really works, it is hard to say, but the philosophy would be that if a gnarly scar gets the job done, then perhaps gently heating cells without disrupting them might have some effect without necessarily causing permanent damage. There is a small literature suggesting that this approach can be effective, but at this point in time it is not clear how such techniques fit into the armamentarium—there are no large-scale controlled trials. There are other laser techniques being evaluated that are similar in philosophy—they all try to create a clinical effect without a destructive visible burn. The ultimate goal would be to use such techniques, perhaps in combination with pharmacologic treatments, in order to treat retinopathy without causing irreversible structural changes in the retina. It is worth keeping an eye on all these approaches—and if future studies demonstrate clear-cut efficacy, you can use this book for kindling or compost…

References and Suggested Reading


