

Special points of interest:

- New Laser Technology
- Retinal Venous Occlusion

# Eye Care Newsletter

Omni Eye Specialists • Madison Street Surgery Center  
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## *New Laser Technology for Creating LASIK Flaps*

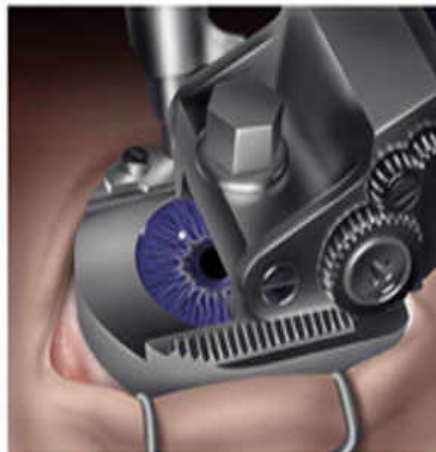
**Vandi Rimer, O.D.**

Lasik surgery was first patented in 1989. The FDA started studies at 10 different centers in the US with approval in 1998. Now there are over 700,000 LASIK procedures performed each year (wikepedia).

The instrument used to separate the layers of the cornea to create a “flap” is known as the microkeratome. It was patented by Jose Barrquer in 1950. This instrument was later used for LASIK procedures starting in 1989. The microkeratome is a mechanical oscillating blade that creates an incision as it passes over the cornea. There are now multiple models of the microkeratome and several manufactures.

Though rare, there are some potential complications that can occur with the microkeratome, such as the risk of a partial flap, hole in the flap, and other flap complications. A flap created using a microkeratome blade can sometimes make the flap thinner in the center and

thicker on the edges. There is also a wide variability in the thickness of the flap. This thickness is often unpredictable ranging from 90 microns to 180 microns with the same instrument.



There are several plate thickness selections such as 90, 160 and 180 microns – but each have variability as to the depth of the incision. The amount of suction applied to the eye also had an effect on the depth of the incision. This variability made it difficult to predict if a flap would be too thick or too thin. In some patients with a thin cornea, if a thick flap was

made – surgery may need to be aborted, as there wasn't enough residual thickness to proceed with the surgery. New advances in laser technology now allow the surgeon to use a laser to create the flap instead of the microkeratome blade device. This new technology is called the Femtosecond laser. (Nd:Glass) 1053 nm (near infrared) laser 60hz. The laser places millions of gas bubbles into the cornea at the rate of a femtosecond. One femtosecond equals one billionth of one millionth of a second; or 0.000000000000001 seconds. For context, a femtosecond is to a second, what a second is to about 31.7 million years. As millions of gas bubbles are placed into the cornea at a specific depth they expand and separate the collagen fibrils of the stromal layers in the cornea, thus creating a flap incision. The entire flap is created in 8-12 seconds.

Retinal venous occlusion is among the foremost causes of sudden painless loss of vision and the second most prevalent retinal vascular disease after diabetic retinopathy. The rubric retinal venous obstructive disease includes two primary manifestations: branch retinal vein occlusion (BRVO) and central retinal vein occlusion (CRVO). Although BRVO and CRVO differ in terms of pathophysiology and clinical course, the shared mechanisms for decreased vision include retinal edema, retinal hemorrhage, retinal ischemia, and vitreous hemorrhage.

Eighty percent of retinal vein occlusions are BRVO's<sup>1</sup>. Associations with BRVO include hypertension, cardiovascular disease, increased body mass index by age 20, and glaucoma<sup>2</sup>. The pathophysiology of BRVO typically originates in the retinal vascular anatomy wherein retinal arteries and veins share a common adventitial sheath. As arteriosclerotic changes develop in the arteries, secondary venous compression results in turbulent blood flow leading to endothelial damage and thrombus formation. Subsequent venous stasis leads to increased transmural vascular pressure and vascular permeability. Intraretinal hemorrhage ensues. Diagnosis of acute BRVO includes observation of intraretinal hemorrhage distal to the site of the arterial-venous crossing.

Central retinal vein occlusion involves venous thrombosis proximal to the insertion of the central retinal vein into the eye. Risk factors include systemic hypertension, diabetes, and glaucoma<sup>3</sup>. Hypercoagulable diseases, blood dyscrasias, and vasculitis have also been associated with CRVO. Increased venous pressure distal to thrombosis of the central retinal vein leads to diffuse intraretinal hemorrhage, retinal edema, and variable amounts of retinal ischemia.

The clinical sequelae of BRVO include spontaneous visual recovery, persistent macular edema (the macula is anatomic part of the retina encom-

passing the highest concentration of cone photoreceptors, responsible for fine visual acuity), and/or rarely ischemia associated with retinal neovascularization and possible vitreous hemorrhage. In contrast to BRVO, CRVO is less likely to spontaneously resolve and more often associated with retinal ischemia, retinal neovascularization, and vitreous hemorrhage. Imaging modalities such as optical coherence tomography (OCT) and intravenous fluorescein angiography (IVFA) may be used to quantify the degree of edema, ischemia, and neovascularization.

Visual decrease post vein occlusion is primarily caused by edema involving the macula. The standard of therapy for macular edema associated with BRVO, established by the Branch Vein Occlusion Study Group in 1984<sup>4</sup>, has been observation followed by macular laser if poor vision secondary to retinal edema persists. Macular laser increases vision by approximately two lines (on the eye chart) in 2/3<sup>rd</sup> of treated patients versus a similar visual improvement in 1/3<sup>rd</sup> of untreated patients. The Central Vein Occlusion Study established that macular laser does not improve vision loss caused by macular edema post CRVO<sup>5</sup>. There are no treatments for CRVO induced macular edema supported by randomized controlled clinical trials. The off-label use of intraocular steroid injection decreases the macular edema associated with vein occlusion, but the results are temporary and the treatment can have serious adverse ophthalmic sequelae including cataract and glaucoma<sup>6</sup>.

In the last year two new therapies for macular edema post vein occlusion have earned FDA approval. Ozurdex<sup>®</sup> is a biodegradable intraocular implant that slowly releases dexamethasone over the course of three to six months. By three months visual acuity improved by 3 lines in 20-30% of patients treated with Ozurdex versus 8-14% treated with sham injection. Visual improvement also oc-

curred more quickly in the treatment cohort<sup>7</sup>. However, the visual improvements lasted three months. The incidence of glaucoma and cataracts remained relatively low.

Ranibizumab (Lucentis<sup>®</sup>) is a monoclonal antibody fragment with anti-angiogenic properties caused by inhibition of vascular endothelial growth factor (VEGF). Serial intraocular injection of Ranibizumab is currently the standard of care for exudative age related macular degeneration. Recent studies have demonstrated the efficacy of Ranibizumab in treating macular edema caused by BRVO<sup>8</sup> and CRVO<sup>9</sup>. At six months serial injection of Ranibizumab resulted in a 3-line improvement in visual acuity in 55-61% of patients with macular edema after BRVO, versus 29% of control patients. Similarly, at six months serial treatments with Ranibizumab resulted in a 3-line improvement in visual acuity in 44-47% of patients with macular edema after CRVO, versus 17% of control patients. Serious adverse events associated with repeated intraocular injections include retinal detachment and endophthalmitis, though the incidence of these events is exceedingly rare. It is possible that continued injections are necessary to maintain visual gains.

Unilateral acute loss of vision requires urgent evaluation by a vitreoretinal specialist to rule out surgical and medical emergencies. In the context of venous occlusive disease newer treatment modalities offer more patients a chance of visual recovery. Once the diagnosis of venous occlusive disease is made the internist and ophthalmologist should endeavor to maximize medical management of associated risk factors. In those patients without typical vasculopathic risk factors a workup for vasculitis, blood dyscrasia, and hypercoagulable states may be indicated.

*See following page for references*

The advantages of laser technology compared to the blade is as follows:

Intralase IFS femtosecond laser

- Patient no longer has “fears” of a blade cutting on the eye
- Only flap creating technology that allows customized parameters
- Makes a consistent flap approx 110 microns thick every time
- Variability on flap thickness only 10 microns
- Less IOP/suction on the eye
- More corneal edema first few days
- Less risk of complications
- If suction loss – can re-applanate and start the procedure over
- Easier for patients with a small palpebral fissure

Microkeratome

- Makes a thicker flap that varies from 90 to 180 microns
- Meniscus flap, thicker at the edge thinner centrally
- Faster than IntraLase
- Higher IOP during the procedure than the IL
- Risk of buttonhole if suction loss
- May need to switch to PRK with a small palpebral fissure

Benefits of Intralase

- Increased quality of vision
- Reduced enhancement rates
- Faster recovery in corneal sensitivity may lead to an earlier reduction of postop dry eye
- Reduction of higher order aberrations
- Reduction in most serious, sight-threatening flap complications
- Consistently creates precise, accurate corneal flaps
- Femtosecond technology has revolutionized LASIK surgery and is far more superior to the mechanical microkeratome flap technology.
- IFS technology has taken flap technology to an even higher level with safer, faster, more precise flaps, a smoother stromal bed and it offers increased flap stability with the customized flap edge.



We are pleased to be one of only a few practices in the state of Colorado to offer the newest IFS – Femtosecond laser flap technology to our patients. We believe that blade free LASIK is becoming the standard of care and most patients choose this technology over the microkeratome. You can learn more about blade free LASIK surgery by going to our website at [www.spivack.com](http://www.spivack.com).

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*References for Retianl Venous Occlusion article*

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