update

RETINA VITREOUS RESOURCE CENTER

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Interchange

Dr. Norman D. Radtke

Dear Colleagues,

The *Update's* continuing mission is to provide you with the most in-depth and comprehensive review of diagnosis and management of vitreoretinal disorders. Our collective charge is to take the most innovative research findings from both laboratory and clinical trials and translate them into the most up-to-date care for our patients.

New therapies are evolving rapidly for the treatment of wet age related macular degeneration (ARMD); this issue evaluates the present status of these therapies. New information may change our evaluations in the coming weeks and months. We will continue to keep abreast of these developments, and we are only a phone call, an email, or a website away if you have any questions.

The accelerated pace of discoveries in the retinal area requires that they be rapidly translated into practice so our patients can benefit from these new understandings. Some of the most rapidly changing areas are anti-VEGF therapies and treatments to reduce the inflammatory aspect of ARMD and abnormal vessel formation. It is important to integrate systems that link research information to our practices.

Some studies have proven certain proposed therapies to be ineffective. We note some of these in the Update. We should call for all evaluations of therapy to be done in the context of a properly designed and executed clinical trial so that our experience, whatever the outcome, will provide useful information that can guide rational clinical care of patients.

To update you on our clinical research effort in patients with retinitis pigmentosa (RP) and dry ARMD, we have included our retinal transplantation data gathered since 2002 on patients with RP and dry ARMD. Our findings continue to evolve, emphasizing the assessment of basic research and clinical trials as they pertain to our clinical practice with patients.

I look forward to one-on-one and group discussions on how we can collectively find ways to provide the best eye care possible for our patients and to promote and support excellent eye care research.

Sincerely,

N.D. Radtke, M.D.

Norman D. Radtke, M.D., F.A.C.S.
Vitreoretinal Surgeon

Subject

Advances in retinal research and clinical application in retinal transplantation, photomicrochip implantation, macular degeneration, retinal vascular abnormalities, diabetic retinopathy, drugs affecting vision, and patient care.

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Improving Vision: Transplants and Technologies

Fetal-tissue transplant rescues woman's sight

Louisvillian part of a clinical trial

The operation

Figure 1: Article from Louisville's Courier-Journal *newspaper featuring a retinal transplantation patient. The article is available on our website (www.rvrc.com).*

What are the results of retinal transplantation in patients with retinitis pigmentosa (RP) and dry age related macular degeneration (ARMD) since 2002?

The table below (Figure 3) shows the current results for patients who received retinal transplants since 2002. Subjective comments from patients are also provided.

Patient EB: At six months, she first noted improved vision when she saw the pendulum swing on her grandfather clock across from her favorite chair. At four years after surgery, the patient noted that she could definitely see better with the eye that underwent operation. The vision in that eye was less cloudy than that of the unoperated eye. She could also read the Reader's Digest (large print edition) and read print on the computer with the operated eye which she could not read with the unoperated eye. She can now write checks, sew and thread a needle occasionally, and can paint ceramics.

Patient DS: She can now see more color and is better able to see light clothing in a dark closet which was black before. She relies on the operated right eye

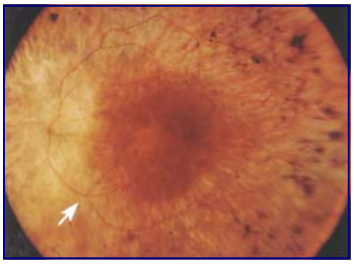

Figure 2: Retinitis pigmentosa patient EB. Preoperative photo prior to retinal transplantation. Visual acuity 20/800 ETDRS.

Retinal Transplantation Cumulative Results Since 2002

Patient ID	Diagnosis	Donor Tissue (gestational age)	Surgery Date	Pre-op Vision (ETDRS)	Post-op Vision 6 months	Post-op Vision 1 year	Post-op Vision Last exam of Post-Study Surveillance
EB	RP	Retina + RPE 11.5 weeks	02/02	20/800	20/400	20/160	4 years 20/200
CB	RP	Retina + RPE 10 weeks	03/03	LP (both eyes)	HM (both eyes)	HM (both eyes)	1 year HM
DS	RP	Retina + RPE 11 weeks	11/03	HM	20/640	20/400	1 year 20/400
RN	RP	Retina + RPE 12 weeks	02/04	20/640	20/640	20/600	1.5 years 20/640
RL	ARMD	Retina + RPE 11.5 weeks	03/04	20/640	20/240	20/400	1.5 years 20/240
DP	ARMD	Retina + RPE 15 weeks	09/05	20/400	20/240	To Be Determined	
EG	ARM	Retina + RPE 12-13 weeks	10/05	20/200	20/320	To Be Determined	
RS	RP	Retina + RPE 12-13 weeks	10/05	LP	LP	To Be Determined	
DD	RP	Retina + RPE 11-12 weeks	01/06	20/320	To Be Determined	To Be Determined	
LC	ARMD	Retina + RPE 12-13 weeks	03/06	20/400	To Be Determined	To Be Determined	

Figure 3: LP = Light Perception HM = Hand Motion
Pink shading indicates patients with visual improvement. Yellow shading indicates patients that are currently being evaluated.

now when she previously relied on the left unoperated eye. She sees more shapes in dark areas now and she sees her face better in the mirror now with the operated eye, which she could not do preoperatively. She sees the upper field better than before, which coincides appropriately with the transplanted retinal area of the eye.

Patient RN: Sees more clearly. At one year, patient could see objects at night in a car at a distance more clearly. At two years, patient could see more clearly with right eye—can't read or see TV features, but can see the screen clearer with the right eye.

Patient RL: She has noted subjective improvement where the operated eye is now her better eye. She sees her clock better, and when reading with her magnifier she sees better than she did before surgery.

Patient RS: At six months, he sees more light peripherally to the right than he did preoperatively. Corresponds to MP1 test.

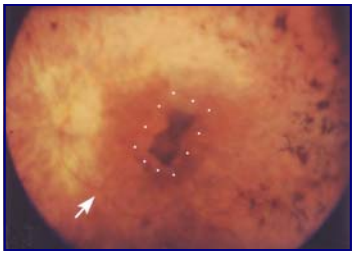

Figure 4: Retinitis pigmentosa patient EB two years after implantation of sheets of neural retina with RPE. Visual acuity 20/200 ETDRS at four years.

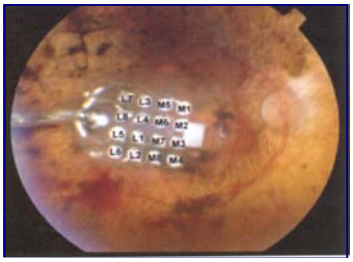

Figure 5: Photomicrochip array placed on the retina by Dr. Humayan.

Photomicrochip technology and artificial vision overview

All prostheses in development share the same principle: create phosphenes through stimulation at some point in the visual pathway with a device that produces an electrical or electric chemical signal. The most likely candidates for this type of treatment are patients with outer retinal degeneration as in retinitis pigmentosa and ARMD. Glaucoma and trauma could also be addressed with optic nerve and cortical prosthesis.

Location of the prosthesis in the eye has been studied in the following research centers: subretinal (Alan Y. Chow, M.D., Chicago; Joseph Reygo, M.D., Boston), epiretinal (Mark Humayan, Los Angeles—See Figure 5), suprachoroidal or transcorneal (Prof. Yagi Naguya, Japan; Yasuo Tano, M.D., Osaka, Japan).

Neurotransmitter and hybrid prosthesis work has also been done (Raymond Lezzi, M.D., Detroit; Danial Palanber, Ph.D., Palo Alto, CA).

What is the status of nanotechnology in ophthalmology?

According to Paul Sieving, M.D., Ph.D., director, National Eye Institute (NEI) in Bethesda, MD, the goals of NIH are to characterize the properties of molecules and nanomolecules, to understand the engineering principles that govern the interface between objects and biology, and to apply this knowledge through demonstration projects for repairing tissues, and ultimately to prevent and cure disease. The NEI has funded a number of applications that employ nanomedicine concepts including plasticity and regeneration of retinal synapses, subconjunctival routes to prolong corticosteroid repair, and effects of substratum topography on corneal epithelium. Nanotechnology yields promise for retinitis pigmentosa, age related macular degeneration, and ocular infections.

⚠ Update **Studies Showing No Therapeutic Effect**

The Dobelle artificial vision brain implant (see *Update,* Vol. 8 No. 2, October 2004) has not been shown to have any therapeutic effect.

Age Related Macular Degeneration (ARMD)

Risk Factors Associated with Macular Degeneration

What factors seem to lead to the progression of age related macular degeneration?

1. Cigarette smoking
2. Obesity
3. Elevated C-reactive protein
4. Elevated homocysteine levels
5. Family history
6. Dietary fat
7. High cholesterol
8. Low intake of antioxidants
9. Hypertension
10. Caucasian race
11. Female gender

Is there an association between cataract surgery and age related macular degeneration patients developing subretinal neovascular membrane after cataract surgery?

Freeman, E., et al (*American Journal of Ophthalmology 135(6):849-856, 2003*), concluded that further research is necessary to determine this. It is not possible to determine whether or not cataract surgery predisposes ARMD patients to development of subretinal neovascular membrane; this could be possibly related to inflammatory factors or exposure of the eye to certain wavelengths of light and higher intensity of light after cataract surgery. At this point, the evidence is not in as to whether or not cataract surgery contributes to the development of active subretinal neovascular membrane in patients with macular degeneration.

Frederick Ferris III, M.D. AREDS Research Group: "Cataract surgery does not appear to significantly accelerate progression to NV ARMD among persons at low to moderate risk for progression."

What is the probability of macular degeneration affecting the second eye?

There is a 25% chance of affecting the second eye in two years.

Does fruit intake play a protective role in age related macular degeneration?

Cho, E., et al (*Archives of Ophthalmology 122(6):883-892, 2004*) found that higher fruit intake was related to reduced risk of neovascular age related

macular degeneration. Further work is needed to identify the relevant compounds in fruits.

Does age play a role in the development of age related maculopathy?

Mukesh, B.N., et al (*Ophthalmology 111(6):1176-1182, 2004*) described data suggesting that 1 in 3 persons aged 70 years or older will have age related maculopathy lesions over a 5-year period and that the disease will progress to a more severe form after the age of 80 years. The presence of soft indistinct drusen with pigmentary abnormalities significantly increased the risk for development of age related maculopathy.

Is macular degeneration inherited?

Weeks, D.E., et al (*American Journal of Ophthalmology 132(5):682-692, 2001*) indicated that, in summary, three areas of the human genome were found to be shared amongst affected versus unaffected individuals. These are very promising results as two independent research groups have now confirmed these findings. Candidate gene studies have been underway aiming at identifying specific genes. Major challenges in ARM research are determining and defining a continuum of disease and distinguishing alternative causes of macular pathology.

It is important to distinguish between a disease-causing gene and a variation, as well as hereditary versus environmental causes.

The focus on chromosome 1q31 independently confirms a report by Klein, M.L., et al (*Archives of Ophthalmology 116: 1082-1088, 1998*) mapping an age related maculopathy susceptibility gene to this region. There was no evidence that other known macular or retinal dystrophy candidate gene regions are major contributors to the genetics of age related maculopathy.

Genetic susceptibility plays some role and is a complex trait. First degree relatives are three times more likely to develop age related macular degeneration. The incidence by race (in patients 75 to 84 years old) is:

- Black 2.4%
- Chinese 4.6%
- Hispanic 4.2%
- White 54%

What are the genetic findings to date in ARMD?

ARMD is multi-factored with a range of both genetic and non-genetic conditions underlying events in the extended and branching pathways from initiating events to condition to disease endpoints that cause visual loss or blindness. Tools for unraveling the complexity, such as DNA samples from all participants in the major population states, the human genome mapping from the Human Genome Project, and new diagnostic imaging technologies are far more powerful than those available just five years ago.

Several genetic variants are known to, or are likely to, increase susceptibility to environmental factors; several are known to, or are likely to decrease susceptibility. It is very likely that genetic differences among different racial

Supplements

1. Vitamin C (500 mg per day)
2. Vitamin E (400 intl. units per day)
3. Beta-carotene (15 mg per day)
4. Zinc, as zinc oxide (80 mg per day)
5. Copper, as cupric oxide (2 mg per day)

Figure 6: Dietary supplements found to be helpful in preventing age related macular degeneration.

groups include differences in their AMD susceptibility genes. Thus, we can anticipate that there will be different AMD disease subtypes and different responses to therapies, depending on the AMD-relevant genotypes of the patient. They may also require different therapeutic approaches to provide the most effective care.

According to Gorin et al, (Am J Hum Genet 77:389-407, 2005), susceptibility genes for age related maculopathy have been identified on chromosome 10q26. Genetic susceptibility plays some role. First degree relatives are three times more likely to develop disease. Gene therapies for ARMD are still in research phase. The gene encoding complement factor (H)CFH affects the level of inflammation in the outer retina, thereby contributing to AMD.

Update on gene therapy for Leber's congenital amaurosis and dry macular degeneration

Gene therapy is probably five to ten years away for many eye diseases. It won't cure disease, but it will manage disease very well. Replacing a recessive gene is the most promising. Leber's congenital amaurosis (LCA) tested by gene therapy trial treatment showed the following:

- RPE 65 gene defect supports photo transduction cycle— a biochemical process that turns light into an electrical signal
- Single missing gene
- Adeno-associated virus as a vector for gene delivery to carry healthy gene. Phase I human trial used at University of Florida, NIH, University of Pennsylvania and Cornell University, Indiana.
- By supplying a normal copy of the gene, they hope to restore vision

Gene theray for ARMD is in the research phase. Dr. Zhang at University of Utah is using pigment epithelial—derived factor (PEDF) with a proprietary adenovector to deliver the gene in patients with wet ARMD.

What questions will be answered by the AREDS II study?

This study will try to answer the question "do the following decrease the risk of progression of AMD?": Lutein, Zeaxanthin, Omega-3 fatty acids.

Recent studies on PDT, Intravitreal Triamcinclone, Retanne, and Anti-VEGF Therapy for Wet Age Related Macular Degeneration

Lucentis® (Ranibizumab) - The FDA has approved Lucentis® for the treatment of wet ARMD. 0.5 mg is recommended for intravitreal injection once a month. If monthly injections are not feasible, treatments can be reduced to one injection every three months after the first four monthly injections. Compared to continued monthly dosing, dosing every three months will lead to an approximate five-letter (one line) loss of visual acuity benefit, on average, over the following nine months. Patients should be evaluated regularly. "Lucentis provides new hope for patients with wet AMD because it is the first therapy to provide a benefit in vision for a significant number of patients," said Arthur D. Levinson, Ph. D., Genetnech's chairman and CEO. "We are proud that the seminal work in angiogenesis conducted at Genentech, years of clinical study, and the dedication and commitment of thousands of patients and retina

specialists have all contributed to this important approval." Eugene de Juan, M.D., president, American Society of Retina Specialists, said, "In my opinion, the Lucentis® approval stands out as one of the most important medical developments in ophthalmology during my 25 years in the field because it has the potential to reverse vision loss associated with wet AMD."

Avastin® (Bevacizumab) - Avastin® appears to be more efficacious than Macugen® according to R.L. Avery et al (*Opthalmology 2006: 113: 363-372*). Nevertheless, safety and liability concerns are associated with Avastin® use, and patient and physician must consider many factors before selecting such an unapproved, off-label therapy. It can be used to induce an initial therapeutic response as it has been proven to acutely reduce retinal leakage within days of treatment.

Avastin® has been discussed in many anecdotal reports to successfully treat diabetic macular edema, diabetic neovascularization and diabetic rubeosis. Additional uses are cited for branch vein occlusion, central retinal vein occlusion, and treatment for rubeosis in CRVO. There is no evidence that its use in these situations is equal to or better than previously used standard treatments for these conditions at this time. New ongoing studies with Avastin® will clarify this, and we'll keep you updated as they occur.

Retanne® (Anecortave acetate) - Retanne® is a periocular posterior juxtasclera deposit drug that is currently undergoing six additional ongoing clinical safety and efficacy studies. Results to date are that it maintains vision in 80% of patients as compared to 45% in controls.

Macugen® (Pegaptanib) - Macugen® provides a statistically significant and clinically meaningful benefit to a broad spectrum of patients with neovascular ARMD, regardless of angiographic subtype, baseline vision or lesion size according to the VISION trial (VEGF Inhibitor and Study In Ocular Neovascularization). Dr. G. Williams feels that this is his first drug of choice, especially in small lesions.

Intravitreal Triamcinolone—Monotherapy with intravitreal triamcinolone has not been proven efficacious in controlling CNV in patients with ARMD. Effects do not appear to last beyond a few months, and elevated intraocular pressure occurs in 40% of patients.

What is next on the horizon for the anti-angiogenesis puzzle?

Dr. Arup Dos, M.D., Ph. D., associate professor at the University of New Mexico, stated that other approaches to the inhibition of ocular neovascularization besides steroid and anti-VEGF therapies that may bear fruit in the future include anti-insulin-like growth factor 1 (IGF-1), anti-angioprotein, anti-protienase, nonsteroidal anti-inflammatory drugs (NSAIDs), and pigment epithelion derived factor (PEDF). As work evolves in these areas, we will keep you updated.

How does combination therapy compare to monotherapy in the treatment of ARMD?

Combination therapies for the treatment of neovascular ARMD offer the

potential for improved visual outcomes compared with available monotherapies. To date, however, no combination has been proven safe and effective in multicenter, randomized clinical trials. Currently we do not know the proper delivery route, dose, timing, and frequency of combination therapies. At this time the risks of combination therapy appear to be greater than that of monotherapy. Our most successful anecdotal result of combination therapy came with Macugen® followed by PDT, with vision going from Count Fingers to 20/40. We will keep you updated on the results of various combination therapy regimens as they become available.

What are the theories of AMD pathogenesis?

Dr. P. Campochiaro postulated that the major mechanisms are inflammation, abnormalities of the extracellular matrix in Bruch's membrane, and oxidative stress damage.

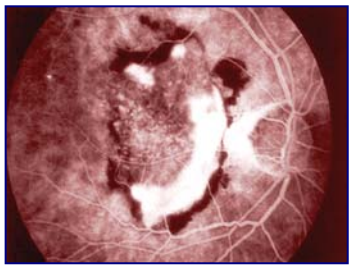

Figure 7: Example of peripapillary CNV membrane that would benefit from subretinal surgical removal.

Does removing subretinal neovascular membranes help in age related macular degeneration?

CNV anterior to RPE with ingrowth sites eccentric to the fovea may undergo surgery with restoration/preservation of excellent function. Surgical removal is probably the best option for large peripapillary CNV.

What is the progression rate of ARMD?

Approximately 1% of people with early dry ARMD and 18% of people with intermediate dry ARMD will progress to advanced ARMD over a five-year period (*AREDS Research Grp. Archives of Ophthalmology 2001:119:1417-1436*).

What information should be shared with patients about Verteporfin PDT treatment?

According to Bressler, Schmidt-Erfurth, and Ergun, the following factors are important to relate to patients:

⚠ Update

Studies Showing No Therapeutic Effect

Rheopheresis Trial Fails to Meet Primary Endpoint

OccuLogix Inc. announced that its MIRA-1 Phase III clinical trial of rheopheresis for the treatment of dry AMD did not meet its primary endpoint. The treatment group demonstrated a positive response, but the trial did not demonstrate a statistically significant difference in mean change between the treated and control groups at 12 months. The company said an anomalous response of the control group is the principal reason that the primary efficacy endpoint was not met.

Meuller, et al found that in 71 patients the following therapies did not help singularly or in combination for central retinal artery occlusion:

- Ocular massage
- Isovolemic hemodilation
- Anterior chamber paracentesis
- Aspirin, Diamox

They also found that there was insufficient evidence to justify intra-arteriole fibrinolysis with urokenase or recombinant thromodoplastin activator.

- Treatment can slow disease progression and reduce the risk of moderate and severe visual acuity loss, but does not often restore visual acuity
- Scotoma size is smaller in treated patients, so loss in reading speed is likely less with treatment
- Follow-up visits, often with fluoroscein angiography and possibly OCT, may be needed every 10 to 14 weeks
- Multiple treatments are often needed, but the number of treatments decreases over time
- Vision rehabilitation is an important part of successful management of AMD

How does PDT compare to anecortave acetate?

Anecortave acetate did not meet the primary non-inferiority endpoint of the clinical study, indicating that the two therapies are not statistically different from each other.

Retinal Vascular Abnormalities

What are the most recent results of radial optic neurotomy on central retinal vein occlusion?

75% get a 4.5 line improvement in some studies. Improved retinal reperfusion, decreased macular edema and visual improvement have been reported. However, numerous unanswered questions in 2006 remain regarding the surgical treatment of eyes with branch vein occlusion.

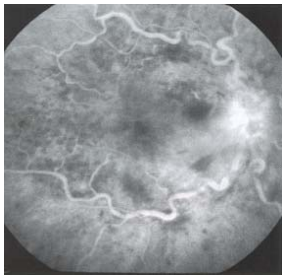

Figure 8: Patient with central retinal vein occlusion pre-operative. Our patient's visual acuity was 20/300.

M. Opremcak, M.D. has stated that in 117 consecutive cases of CRVO, anatomical resolution occurs in 95%, and improved visual function in 71%. The visual improvement averages 2.5 lines with a range of 1 to 12. Radial optic neurotomy and intravitreal Kenalog® equals radial optic neurotomy alone.

The effect of transluminal YAG embolysis (TYE) for retinal artery occlusion

Opremcak, M., et al (*Retina 22(2):213-216, 2002*) have shown that breaking up plaques in arterioles in branch retinal artery occlusion or central retinal artery occlusion for as long as 6 weeks has been successful. They use the term "retinal coma" for the area affected which will revive itself after the blockage is open. The retina is nourished by the choroidal circulation during the blockage of the arterial, hence the term coma.

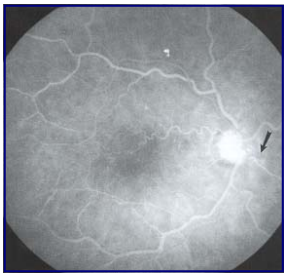

Figure 9: Patient with central retinal vein occlusion status post radial optic neurotomy. Visual acuity 20/30.

Reynard, M. and Hanscom, T. A., (*American Journal of Ophthalmology 137 (1):196-198, 2004*) have shown that neodymium: yttrium-aluminium-garnet laser arteriotomy in a patient with central retina artery occlusion resulted in extrusion of the embolus, reopening of the central retinal artery and return of vision. This technique warrants further study as a primary treatment for this blinding disorder.

Both authors have shown that TYE resulted in disappearances of the emboli

and immediate restoration of blood flow. The YAG laser photo disrupts the embolysis with the lumen of an occluded retinal arteriole without damaging the vessel wall by the rapid deposition of infrared irradiation in the embolus after passing through the vessel wall. Rapid thermal expansion will occur within the embolus until its compression and tensile strengths are excellent. At this point, the embolus will completely or partially shatter, clearing the lumen of the arteriole. The vessel wall is left unharmed because of the greater elasticity and lower absorbing of the infrared irradiate. Energy level of the Nd:YAG was recommended to be below 1 m.

Diabetic Retinopathy

Does removing the submacular hard exudates in patients with diabetic maculopathy improve their vision?

Takaya, K., et al (*Retina 24(1):23-29, 2004*) reported that visual improvement could not be obtained after removing submacular hard exudates in most patients, suggesting that diabetic maculopathy should be treated before massive exudate deposits appear in the macula.

What are some of the established pathophysiological aspects of diabetic retinopathy according to Dr. Robert N. Frank?

- Diabetic retinopathy is clearly a response to chronic hyperglycemia with a dose-response relationship.
- The onset of retinopathy in response to hyperglycemia is slow, and reversal after correction of hyperglycemia is also slow.
- There is probably a genetic disposition to retinopathy as well as to diabetes.
- Regardless of prior duration of diabetic onset, puberty appears necessary for onset of retinopathy.
- Retinal microvessels are affected, but the anatomically similar cerebral microvessels are not affected either in humans or in animal models.

Drugs Affecting Vision

What are the short term effects of Viagra® on young healthy subjects?

Jägle, H., et al (*American Journal of Ophthalmology 137(5):842-849, 2004*) from Tübingen, Germany, investigated the short-term visual effects of a single 100 mg dose of Viagra® (sildenafil citrate) in healthy young men. This led to small but statistically significant transient changes of the outer and inner retinal function as detected by ERG and psychophysical methods. The acute affects were fully reversible within 24 hours.

What are the best tests to evaluate Plaquenil® toxicity?

Traditionally, visual function has been assessed by measuring visual acuity or visual fields. However, many patients with macular disease lack adequate central vision to perform their tasks meaningfully. Standard perimetry relies on

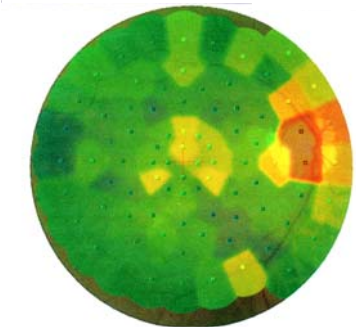

Figure 10: Patient with all other tests normal, including electrophysiology. Visual acuity 20/30. Fluorescein showed pigment epithelial changes.

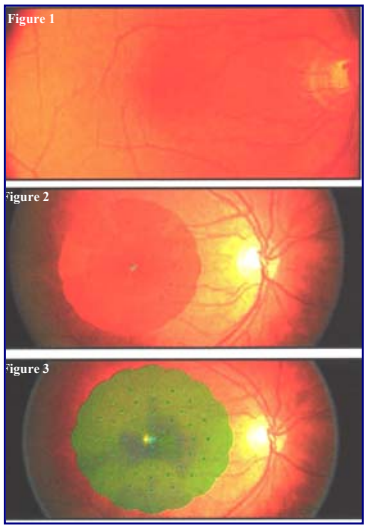

Figure 11: Various tests on a Plaquenil® patient. Picture courtesy Dr. Nelson R. Sabates, M.D.
11.1— Essentially normal
11.2—Abnormal MP1 with other tests normal
11.3—MP1 returns to normal after stopping Plaquenil®

Patient Comments on Music Therapy

- "Really helped a lot. It felt like we were at home ."
- "Very good idea, very soothing. It kept me from thinking about what I was going to face. "
- "Music kept me from being very nervous and I liked the music selections. "
- "Helped keep me very calm. I enjoyed it very much and was very soothed."

Figure 12: Comments from patients who underwent music therapy.

patient fixation to map field defects. Microperimetry has re-emerged as an option for assessing visual function in patients who cannot maintain adequate fixation.

The normal recommended test by the American Academy of Ophthalmology has been to obtain a fluoroscein angiogram, Humphrey 10-2 visual field, and an mFERG if available when patients are taking 6.5 mg/kg per day or more of Plaquenil®. The most reliable test would be the MP1 microperimeter. A recent request showed a patient that had normal standard tests with Humphrey and fluorescein but had significant diffuse decreased sensitivity in the macula which became normal on repeat MP1 testing after stopping Plaquenil® therapy (Figure 10).

Another patient, a 36-year female, had been taking Plaquenil® for rheumatoid arthritis for approximately ten years. She had begun to notice an overall decrease in her vision over the past several months. She had been examined elsewhere on several different occasions and had been told her examination was normal. She presented for another opinion. Her visual acuity was 20/20 in both eyes. Funduscopic examination and fluorescein angiography were essentially normal except for very subtle RPE changes noted in the macula in both eyes (figure 11.1). The standard perimetry (Humphrey visual field) did not show any scotoma or decrease in sensitivity. An MP1 was performed on both eyes and discovered a significant diffuse decrease in macular sensitivities in both eyes (figure 11.2). This was confirmed on repeat testing. The Plaquenil® was discontinued. Three months later repeat MP1 showed a normalization of macular function (figure 11.3) and resolution of the patient's complaints.

Microperimeter effectiveness in evaluating visual complaints in patients with interferon for Hepatitis C and Multiple Sclerosis

Microperimeter testing helps sort out malingers. The test provides objective evidence of complaints not identified by any other tests.

Patient Care

How does music therapy help our surgical patients?

Eighty percent of our patients desire to listen to music when having surgery. To relieve anxiety and relax patients, they are given their choice of several music types. There have been several instances with our patients where a surgery has been cancelled due to a spike in blood pressure in the prep and hold area. On subsequent workup of their high blood pressure, no etiology was found. Our hope is to reduce pain perception and high blood pressure without medications and to relieve the patients' anxiety about surgery preoperatively.

Patients' comments to date are listed (See Figure 12).

update
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