

The Light Pipe

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The newsletter of Georgia Retina, P.C.

Health Food Hype

Often regarded as natural, herbal medications may be a patient's first line treatment for a multitude of conditions. Studies have given credence to some of these claims, but there are potential side effects.

By 1997 more Americans had visited an alternative therapy practitioner than saw a primary care physician. Forty percent of patients are estimated to use alternative therapies. Certainly, this includes many of our patients. Even if many of these remedies do not target eye disease, their side effects may.

Ginkgo biloba is one of the more popular remedies used to improve memory and increase blood circulation to the central nervous system and optic nerve. One of its side effects is reduced platelet function. Users of ginkgo can develop hyphema as well as vitreous hemorrhage. When combined with other anti-platelet agents such as aspirin, the effect is magnified.

Ginseng is taken to boost energy and concentration. Side effects include hypertension and tachycardia which can increase morbidity during anesthesia. It also interferes with insulin while having a hypoglycemic effect of its own. Diabetics risk serious hypoglycemia with this herb.

The ophthalmologic benefits of **bilberry** are touted to include better night vision.

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Intravitreal Steroids- A New Tool in the Retina Toolbox

Steroids have been a mainstay of treatment for ophthalmic disease for decades. Systemic administration carries the greatest risk of side effects, some of which are quite serious. While topical administration of steroid is the easiest and has the least risk of side effects, this route of administration is also probably least effective due to limited drug delivery to the retina. Retrobulbar or sub-Tenon's injections have for many years offered a compromise between better penetration and avoidance of systemic side effects. Intraocular administration of drugs has typically been reserved for vision-threatening conditions such as endophthalmitis. The perceived risk/benefit ratio of intraocular injections for other indications has been influenced by concerns of introducing an infection, detaching the retina, or drug toxicity. However, our experience in managing CMV retinitis with either intraocular antiviral injections or implanted antiviral pellets has shown us that these risks are quite low. In the past two years, the use of *in office* intraocular injections of steroids for a variety of conditions is being explored by investigators. In one study, chronic refractory CME was treated using a 1 mg intraocular injection of KenalogTM. The edema diminished in the majority of patients (89%). Almost one-half the patients had visual improvement of 1 or more lines, and 20% gained 2 or more lines. In a study elsewhere using a 4 mg dose, 75% of patients had improvement, and almost one-half of these had 2 or more lines of improvement. In a prospective study from Germany using a much larger dose of KenalogTM (20 to 25 mg), the eyes demonstrated an average vision improvement of 100%. A number of papers presented last year at the Vitreous Society meeting discussed the use of intraocular steroids to treat exudative ARMD, refractory CME from Irvine-Gass syndrome, or diabetic macular edema. There have even been reports of success using such steroid injections for CME associated with branch retinal vein occlusions. Based on surprisingly excellent results in this otherwise refractory group of patients, intraocular steroids are becoming commonplace. While we were aware of these reports, we chose

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Cystoid Macular Edema (CME)

CME is frequently responsible for central visual loss of variable severity. There are numerous causes; some quite common, others very rare. With CME, the normal foveal depression is lost, the retina is thickened, and may have an increased yellow reflex. Biomicroscopy or fluorescein angiography shows typical cystoid spaces in a radial orientation within the neurosensory retina. Histopathologically, these cysts are mostly in the outer plexiform layer.

One of the most common causes for CME is **Irvine-Gass syndrome** following cataract surgery, particularly if there has been a break in the posterior capsule. In fact, subclinical angiographic CME has been shown to occur in a high percentage of eyes following cataract surgery, but usually does not affect visual acuity. Microvascular damage, particularly common in **diabetic retinopathy**, **retinal vein occlusions**, or **radiation retinopathy**, frequently causes CME. Less commonly **idiopathic juxtafoveal capillary telangiectasia** (IJT) causes CME, usually on the temporal edge of the fovea. Many forms of uveitis cause CME. In **pars planitis**, CME is the major cause of visual loss, and is prime reason of treatment. **Behçet's disease**, **Crohn's**, **rheumatoid arthritis**, and **sarcoidosis** are frequently associated with CME in the presence of ocular inflammation. CME is often seen in the late stages of **exudative ARMD**, due to transudation of subretinal fluid, and represents a poor visual prognosis due to chronic anatomic changes. Any syndrome associated with subretinal neovascularization, such as ocular histoplasmosis or angioid streaks, can have a similar effect. **Peripheral retinal lesions** must be ruled out in any patient with macular edema. **Coat's disease**, **peripheral capillary hemangiomas**, and **malignant melanomas** of the choroid are all capable of inducing CME, when leakage from the periphery accumulates in the fovea. **Hypotony** from any cause is also associated with CME. This can be secondary to glaucoma filtering surgery, wound leaks, choroidal detachment, blunt trauma or ocular ischemia, among others. **Optic nerve swelling** often causes macular edema. True papilledema, pseudotumor, cat-scratch disease, and rarely ischemic optic neuropathy can cause secondary macular edema, particularly when chronic. **Traction** on the macula is also capable of inducing CME. **Epiretinal membranes** often mimic the glistening appearance of CME, but are often associated with true secondary CME. Similarly, traction from proliferation in diabetic retinopathy can cause CME even without directly detaching the central macula. Less common causes include **retinitis pigmentosa**, **X-linked retinoschisis**, and **niacin toxicity**.

One can see then, that CME is a final common pathway for many underlying diseases: ischemic, tractional, inflammatory, toxic, and genetic.

Other diagnoses which mimic CME include macular pucker, early macular hole formation, central serous retinopathy, arterial occlusions with a cherry-red spot, Stargardt's disease, and Best's disease.

Treatment of CME varies with the severity of visual loss, anatomic changes, and underlying cause. Most commonly, topical steroids with or without topical non-steroidals are the first line of treatment in Irvine-Gass syndrome, or any CME associated with inflammation. If not effective enough, these medicines can be combined with periocular steroid injections, oral steroids, or oral NSAIDS.

In CME secondary to vascular disease such as BRVO and NPDR, laser has been the mainstay of treatment, and is proven to be effective, while not necessarily restoring perfect acuity. The laser can be used to directly treat microaneurysms or can be used in a grid fashion in the area of thickened retina. Care must be taken to avoid central and paracentral scotomas.

Peripheral lesions can be treated with laser or cryopexy, often with remarkable resolution of central edema. However, if lipid deposits have formed in the fovea prior to or as a result of treatment, the visual results can be disappointing.

In mechanically induced CME, as in PDR or macular pucker, pars plana vitrectomy and membrane peeling are highly effective at reducing the edema. If vitreous is incarcerated in the anterior segment, vitrectomy is also very helpful.

Some new alternatives are on the near horizon. Direct placement of steroids into the vitreous cavity, either by injection or placement of a long-term delivery device similar to a Vitrasert, seems to be very effective in CME mediated by inflammation. (See "Intravitreal Steroids – A new tool in the Retina Toolbox" on page 1.) Just as interesting is the positive response in diabetic macular edema to steroid treatment in pilot studies, using a long-term delivery system. Pars plana vitrectomy may play a role in treatment of BRVO-induced CME. In this situation, careful dissection of the artery which appears to compress the underlying vein can visibly reduce congestion and edema very rapidly.

In summary, cystoid macular edema is one of the most common causes of visual loss. It is associated with numerous common and uncommon conditions. Several proven treatment modalities are available, and new therapies continue to expand our options.

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St. John's wort is used for depression, anxiety and sleep disturbance. It was recently found to be ineffective for depression. It can interfere with anti-HIV drugs, Coumadin and oral contraceptives among other medicines. It can prolong the effect of narcotics and general anesthetics.

Ephedra is taken for energy boost and weight loss. Being a stimulant, side effects include hypertension, stroke and myocardial infarction. Patients with narrow angles risk angle closure glaucoma. Ephedra interferes with general anesthetics as well as other drugs.

Kava kava is taken for stress, anxiety and sleep disorder. Visual and balance disturbances are part of its side effect profile. Pupil dilation can result from its use, which can lead to angle closure in susceptible patients. This herb can interfere with Coumadin, antidepressants and other drugs. Liver damage has been reported.



Patients undergoing surgery should stop herbal preparations two weeks prior to planned surgery. Emergent surgery in these patients can be associated with more bleeding and problems with the smooth delivery of anesthesia. The most important anesthesia-related herbs are ginkgo and feverfew which reduce platelets. St. John's wort prolongs the effect of general anesthetics and narcotics. Ginseng causes hypertension and tachycardia.

Among less common herbal preparations, **yellow jessamine** can cause diplopia and abnormal eye movements. **Cypress spurge** can lead to pupil dilation, eyelid swelling, conjunctivitis and corneal defects. **Melatonin** has been reported to cause damage to retinal ganglion cells. This effect can be magnified by the use of selective serotonin reuptake inhibitors (SSRI) such as Zoloft. Serotonin is a precursor to melatonin.

Some patients also take large doses of powdered **aspirin preparations**. Patients with rubeosis or retinal neovascularization have presented with hyphema and vitreous hemorrhage. The ETDRS showed that 650 mg of aspirin a day does not increase the risk of vitreous hemorrhage. The effect of higher doses have not been formally studied, but may reduce the platelet function and increase the risk of bleeding.

It is important to specifically ask about the use of herbal supplements in our initial history. Many patients think of these supplements as food and not as biologically active medications. Many physicians also may not consider these supplements as valid pharmacotherapy. Nevertheless, many herbal preparations are biologically active and should be respected as such.

Evaluation of Suspicious Iris Nevi – Is it a freckle or a melanoma?

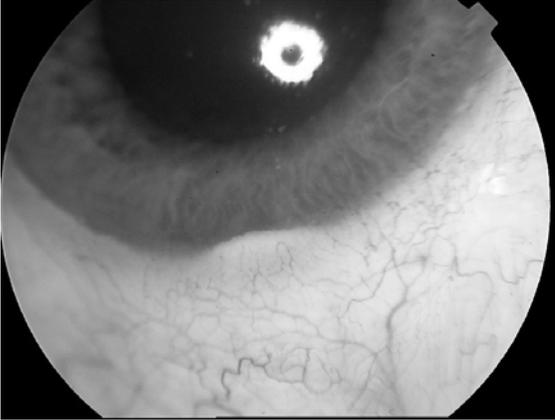
There are a number of benign and malignant tumors that may arise from the pigmented cells of the iris. The iris stroma gives rise to nevi and melanomas, and the iris pigment epithelium gives rise to adenomas and adenocarcinomas. Tumors of the iris stroma are fairly common, while tumors of the pigment epithelium are fairly rare.

In clinical practice we see many iris nevi. The actual incidence of iris nevi is uncertain because they mostly asymptomatic, and many people with an iris nevus do not seek medical attention. There are several types of iris nevi ranging from circumscribed, to the more diffuse iris nevus which may encompass the entire iris, as in the iris nevus syndrome. Flat, superficial pigmented iris lesions that do not alter the iris architecture are referred to as iris freckles. Iris freckles most likely have no malignant potential and should be differentiated from nevi which may occasionally give rise to an iris malignant melanoma. It is helpful to have an organized approach when evaluating a suspicious melanocytic lesion of the iris. There are several characteristics that are readily noticeable at the slit lamp and should raise suspicion that a melanocytic iris lesion may be malignant.

Iris nevi are by definition benign accumulations of abnormal melanocytes in the iris stroma. They are usually defined as stationary melanocytic tumors that replace a portion of the iris stroma. However, they do possess the potential for growth, and while growth should raise suspicion, it does not necessarily indicate malignancy. Furthermore, they may occur anywhere on the iris from the pupil margin to the trabecular meshwork. Traditionally, pigmented masses occupying the trabecular meshwork were considered malignant. However, the presence of melanocytes in the trabecular meshwork make nevi in this area possible, and pigmented lesions are no longer considered malignant based on angle involvement alone.

The diagnosis of an iris nevus should be made clinically and the suspicion of malignancy usually occurs over serial examinations. The lesion should be examined carefully and its size in millimeters, color, thickness and location recorded and documented photographically. An attempt should be made to obtain records of past examinations or photographs that could verify the presence of the lesion in the past. Gonioscopy should be performed comparing the trabecular pigmentation in both eyes to detect pigment liberation from the tumor. The remainder of the anterior segment and sclera should be carefully examined to detect pigment dispersion, cataract formation, angle distortion or scleral involvement. Ancillary tests are of limited use. Fluorescein angiography is probably of limited use be-

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not to utilize these treatments in our practice until the past 6 months, for extra safety. We had concerns that the preservatives or carriers in the intraocular steroid suspension could have toxicities that might not have been evident initially. Fortunately that has not been observed, and we have been pleased with the results thus far. Currently, the most common indications for which we are using intraocular steroids are refractory CME in patients with Irvine-Gass syndrome, diabetic macular edema, or uveitis. We have not been using it for exudative macular degeneration, but may consider this as evidence accumulates.

Conclusion: At this point, many questions still remain unanswered. We have not determined whether simply interrupting an episode of CME will allow *sustained* improvement, or whether the CME will return and necessitate repeated injections. Surprisingly, thus far, the duration of clinical response seems to far exceed what anyone would have ever predicted. Perhaps research in the future

will better clarify these issues. Once we establish which conditions respond to one or a few injections versus those conditions that require multiple repeated injections, we will have defined which group of patients would be better served by having a drug-releasing pellet implanted into the eye. These pellets do not release medicine indefinitely, but if indicated, additional pellets can be placed when the first one runs out of drug. Perhaps some way to replenish an implantable reservoir may be devised.

The most common and serious risks of intraocular injection include endophthalmitis, hemorrhage, retinal detachment, steroid-induced glaucoma acutely or several months later, as well as the acceleration of a cataract in phakic patients. Several cases of migration of Kenalog™ into the anterior chamber have been seen, producing the appearance of a pseudohypopyon.

TECHNOLOGY CORNER

High Speed Vitreous Cutters in Vitrectomy Surgery

A recent innovation in vitreous surgery is the use of high speed cutters. This promises to improve the safety of vitrectomy in cases where the retina is detached and mobile. The reciprocating guillotine action of the vitrectomy handpiece results in significant fluid turbulence of fluid flow and secondary movement of loose, fine membranes and detached retina. Increasing the cutting speed reduces fluid turbulence and movement of tissue. It is now possible to perform vitrectomy with cutting rates up to 2500 cuts per minute, with new software upgrades to the Accurus machine. There is also a handpiece with a 1500 cut per minute maximum. Both of these allow closer shaving of the vitreous base as well as work closer to the retinal surface. Although we do not have much experience with the Storz machine, the Millennium also has a rapid cut rate handpiece. It is still, of course, possible to pull retina into the port and cut it, resulting in a retinal tear, but the risk of this should be reduced. This may be especially of value in complex retinal detachments secondary to diabetic retinopathy and proliferative vitreoretinopathy.



Intraoperative Slit Lamp Illumination for Vitreous Surgery

In the office, the slit lamp allows us to take “optical sections” of tissue; that is, to “cut” them and look at them at an oblique angle, giving us tremendous information about depth, and showing us the relationship of one layer to another. The slit beam is also focused, to reduce scatter and extraneous reflections. These same characteristics are useful in vitrectomy surgery when a slit beam apparatus is mounted on the operating microscope. Instead of illuminating the posterior segment with a fiberoptic endoilluminator (light pipe) (Hence the name of this newsletter!), a slit beam can be used. This permits bimanual surgery using non-illuminated instruments. For example, a conventional intraocular forceps can be used together with a scissor, to lift and cut tissue, since one hand no longer has to hold the light pipe. Zeiss has a slit beam attachment which has been popular in Europe, but has received little attention in the U.S. We will be evaluating and developing this system at Decatur Hospital in the near future.

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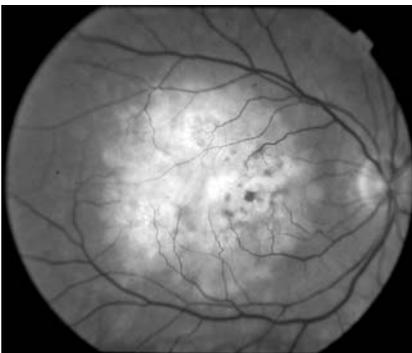
cause both nevi and melanomas that are deeply pigmented are usually hypofluorescent. Transillumination can reveal ciliary body involvement, and high frequency ultrasound is useful when the full extent of the lesion is not apparent.

It is interesting that there are really no clear-cut rules for distinguishing between benign and malignant iris tumors. For example, most iris melanomas are located in the inferior portion of the iris, as are iris nevi. Many iris nevi have sectoral cataract formation. Some nevi demonstrate slow growth and may liberate pigment or undergo spontaneous necrosis. These signs do not prove malignancy, but should raise suspicion. Documented growth is probably the most indicative of malignancy.

A few years ago, Harbour and coworkers reviewed 285 pigmented iris lesions to determine the clinical variables that led to prompt excision and to identify clinical factors associated with enlargement. Five variables were associated with prompt excision: largest basal diameter greater than 3 mm; presence of pigment dispersion; prominent vascularity; elevated IOP; and tumor-related symptoms. The only variable predictive of enlargement was the largest basal diameter. Large lesions over 3 mm in diameter tend to enlarge frequently thus suggesting malignant potential.

Overall, only about 5 percent of suspicious iris lesions will demonstrate clinical growth within 5 years. Iris lesions should be followed by slit lamp examination every 6 to 12 months, and photographs, including gonioscopic photos, should be obtained at baseline if indicated. Photos should be repeated yearly or if growth is suspected. If unequivocal growth is documented, the diagnosis of malignant melanoma should be entertained. Usually, suspicious iris lesions are removed surgically, although radioactive plaque therapy is possible in select cases.

What's your diagnosis? (see page 6 for diagnosis)



35 year old female with history of breast cancer presents with metamorphopsia and decreased vision.

7 year old male found to have reduced vision on school screening exam.



From the Administrator's Desk...



HIPAA Compliance

– Are you prepared?

We're certainly all aware that there are regulations related to privacy rule with which health care providers will have to come into compliance. Our mail is full of offers for courses, magazine articles and insurance company fliers on protecting privacy and improving data security.

Practices have to comply with the HIPAA regulations if they are a "covered entity". This is defined as any health care provider that transmits personally identifiable information electronically. There are three sets of different HIPAA regulations with different compliance dates. The HIPAA Privacy Regulations compliance date is April 14, 2003, Code Set Regulations are October 16, 2002 (unless you have filed for an extension, which will delay your completion until October 16, 2003) and HIPAA Security and Electronic Signature Regulations (Final regulations and deadlines have not yet been published).

HIPAA covers all individually identifiable health information in any form, electronic or non-electronic, that is maintained or transmitted by a covered entity.

Although the regulations are still being debated by government officials, and the date for compliance is still months away, we can't procrastinate! The Secretary of Health and Human Services has the authority to exclude from the Medicare Program those practices that are not HIPAA compliant or that did not file for the extension in time.

START NOW by developing your compliance plan and filing for an extension.! A model compliance plan and the forms to file for an extension are available on the internet at <http://www.cms.gov>.

Barbara Wright
Practice Administrator

We participate in the following insurance plans :

Aetna US Healthcare	Health Market	State Health Benefit Plan
Aetna Select Choice HMO, Elect PPO	Health Network America	Tricare
Aetna Managed Choice POS	Healthstar	Unicare
Aetna Open Choice PPO	Highway to Health	United Healthcare (PPO, EPO, POS, HMO)
Alliance Healthcard	Humana (HMO and POS)	United Payors / United Providers
American Preferred Provider	Managed Care 2000	USA Managed Care Organization
BCBS of Georgia	Managed Care Strategies	
Beech Street / Capp Care	Medicaid - EDS	
Blue Choice PPO, POS and HMO	Medicare - BCBS of Alabama	Other plans are pending,
CCN Managed Care, Inc.	Medicare Railroad	please call to see if we are participating
Cigna (PPO & HMO)	MRN (Medical Resource Network)	(770) 907-9400
Companion Work Place	Multiplan	
Corvel	National Preferred Provider Network	
Coventry Healthcare	Network Atlanta	
Evolutions Healthcare System	One Health Plan	
First Health	Preferred Plan of GA PPO, EPO	
Focus	Principal	
Formost	Phystar dba Starcare	
Galaxy Health Network	Private Healthcare Systems PHCS	
Gaston Loughlin WC	Pronet	
Georgia First	Promina Health Systems	
Georgia Better Healthcare	Prudential	
Healthcare, Inc.	Southcare PPO	

Diagnoses from page 5 fundus photos:

35 year old female with history of breast cancer was diagnosed with **choroidal metastases.**
7 year old male was found to have a **combined hamartoma.**

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