

## Dr. Lander's Eye Manual for Residents

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If you are an internist or family practice doctor, you do need to know a few things about ophthalmology. Fortunately for you, you don't need to know much! But the stuff you do need to know, you need to know well. Your rotation with us ophthalmologists will be extremely brief, and you take pot luck on the pathology you will see while you are with us. This manual is the bare-bones, basic information that I would convey to every resident while he or she is here, if time allowed. It is not a comprehensive discussion of each disease you should know about, and it is not a textbook of ophthalmology. In fact, I really didn't put in a lot of textbook-type information, because there are, of course, textbooks for that. One that is probably (I haven't seen it, but the Academy always puts out good stuff) is Basic Ophthalmology, published by the American Academy of Ophthalmology.

You will get the photos as a separate entity when you are here on your rotation. That way the text is quick and easy to copy or e-mail to any machine. If you want really good pictures, there are many atlases and texts.

If you would like me to cover topics not in this handout, please let me know.

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## What to Expect from Your Ophthalmologist

As for any other specialist, you expect competence and prompt service from an ophthalmologist. Do not assume every ophthalmologist is a good surgeon. Ask around, especially OR staff if you can, about someone before you refer potential surgical cases to a new person in town. There are some people who just don't have, as we say, "good hands." Some training programs don't give residents an optimum number of cases to do. I can tell you that cataract surgery has a very steep learning curve. Watch out for someone who thinks they can do it all—no one is good at everything.

There are certain things that need to be seen promptly. For example, an ophthalmologist to whom you refer should see (or help you arrange to be seen by someone else, if he/she can't) the following:

- Transient visual loss or sudden black-out of vision: drop everything and see patient *now*
- Recent visual field defect, increasing in size: within 24 hours—this is probably a retinal detachment.
- New floaters: within a few days
- Red eye with pain, blurred vision or status-post trabeculectomy: same day
- Corneal foreign body: no later than next day
- Macular degeneration patient with new, recent distorted vision: within 3 days

And just to editorialize a bit, I personally cannot understand an ophthalmologist or any other physician who thinks they are going to turn into a pumpkin if they are in the office past 5:00. Patients can be added on to the end of the schedule for urgent problems.

## Optometrists

Basic training for optometrists is four years of optometry school. This is preceded by 2-4 years of college. The graduates of basic optometry school range from excellent to wretched, with the latter being rather rare these days. On average, the quality of the optometrists who have graduated in the past twenty years is much better than previously. You have to know the optometrists in your community to know their skill level. Don't steer your diabetics from their optometrist to an ophthalmologist, necessarily. Most are capable of doing a perfectly good annual exam. One red flag though is an optometrist who doesn't dilate the pupils as part of the complete exam. You can't do a decent exam without dilating.

Many optometrists have taken a course of study and have qualified to prescribe medications ("therapeutics," as they call it). Those that have bothered to get this designation are usually up on things more than those who didn't. The main thing, though, is that an optometrist knows the extent and limits of his knowledge (true about all of us).

An optometrist who doesn't have therapeutics but knows what he or she knows and doesn't know is better than one who has therapeutics and doesn't.

Some optometrists take extra training in the form of a residency. They handle a good spectrum of pathology in the residencies and produce good clinicians, by and large.

When I use the term “ophthalmologist” in this manual, I mean (unless it’s obvious I don’t) “ophthalmologist or good optometrist.” For neuro-ophthalmology cases, conditions with serious systemic or vision-threatening implications or conditions that could need a procedure, you need an ophthalmologist specifically.

When I use *he*, *him*, *his*, I mean *he* or *she*, etc. We need some gender-neutral pronouns in this language.

### Scribes

Pay attention to the flow of the offices you visit. In fact, pay attention to everything about the offices you visit. You can pick up a lot of ideas for future use. I always find it interesting to be a patient, too.

I highly recommend using a scribe. This person follows you in the room and cuts down on your “chair time” (as we say in the eye biz) dramatically. He or she takes dictation, writes prescriptions and instructions, repeats your instructions or explanations, and helps escort the patient out of the room. These people easily pay for themselves. Another issue for you is that the scribe is a chaperone—a good thing to have in our lawyer-infested society.

### Giving Drops

Don’t forget that lashes are not sterile. If you touch a dropper to the lashes or the eye itself, you will probably have to throw the bottle away—very expensive!

### Giving Ointment

Have the patient keep their eyes closed for one minute after instilling ointment—body temperature melts the ointment and spreads it over the ocular surface.

Patients generally dislike ointments. When you treat a corneal abrasion or conjunctivitis, use drops. They are way more convenient and do not blur the vision.

### Ophthalmoscopy

This intimidates everyone, mostly needlessly. You almost certainly had some brief training on ophthalmoscopy in medical school and never really got good at it. Don’t sweat this too much. Really, as primary care doctors, what you really need to see is the disc, looking for papilledema and at the retinal arteries (arterioles, really), looking for atherosclerosis. You should also know what a cholesterol embolus looks like. Everything else is the purview of the ophthalmologist. You don’t have to look for diabetic retinopathy yourself—you are going to ask your diabetics if

they are up-to-date with their eye exams (You do this, don't you?) and refer them if they are not. Much is made in medical school of looking for diabetic retinopathy, macular degeneration, retinal hemorrhages, cotton-wool spots, etc. It is often very difficult for you to see this stuff. Older patients usually have some degree of cataract, and their pupils are usually small because of sclerosis of the iris sphincter. Fortunately, the optics of the eye are such that if you are going to see anything back there, you are going to see the disc. You will see vessels even if you are totally lost.

The instrument you have available is the direct ophthalmoscope. This gives you a magnified, 2-D view of the fundus. You will see the ophthalmologists using other instruments too. These give us a 3-D view and views that are specialized for certain aspects of the fundus exam, such as viewing the periphery or the disc.

#### Technique, reviewed:

- Darkish room for maximum contrast. Have the patient fixate a distant target.
- Set the lens dial on 0. You will be focusing later.
- Use the small white light. The large one puts too much light into the eye—then you have to deal with the resultant photophobia. The large light is a great flashlight, though, and it is really good for doing the pupil exams.
- Examine the patient's right eye holding the ophthalmoscope in your right hand and view with your right eye. Likewise, left eye, left hand, left eye.
- Find the disc.
- Focus with the lens dial. You are neutralizing the patient's refractive error.
- Examine the fundus in sequence. I look down the arcades and then examine the tissue in between, including the macula. Don't try to see anything outside of this area. The instrument doesn't do beyond the posterior pole very well.
- If you get lost, follow a blood vessel down to the disc, increasing in caliber.
- If you get really lost, have the patient look right at your ophthalmoscope light—they will be pointing their macula right at you.
- Don't carry your scope in the pocket of your white coat—they like to fall out.
- I usually hold my breath while I am using the direct—I don't need their germs, they don't need mine.

#### Visual Acuity

Get an acuity if there is a medical-legal reason to do so. If you are going to treat any type of trauma yourself, you definitely need one. It wouldn't hurt to get an acuity when there is a complaint of visual loss, especially acute, although you will be referring these anyway. Finally, you family docs and pediatrics people will be doing screening visions on children.

#### Visual Acuity Technique:

- If you have a Snellen chart on the wall, be sure that you are having the patient stand the distance from it that the chart says to—either 20 or 10 feet.
- If you don't have a wall chart, use one of the near cards. The Yellow Pages of the phone book is useful too. You can measure the smallest sized print that the patient can read—5mm or whatever.
- Have the patient wear their eyewear appropriate for the testing distance—either distance or reading.
- Be sure that the patient has the non-test eye covered completely. If they use their hand, cover with the palm—you can see through the spaces between your fingers. Be sure you have them seal the space between the cover and the bridge of the nose—it is easy to peek around the cover there.

If they can't see the big "E" on the wall chart, don't put "No vision" on the chart. Poor acuity is measured as...

- CF= count fingers
- HM= hand motion
- LP= light perception
- NLP= no light perception

Don't get fancy with this stuff. In some books you will see for Count Fingers vision to specify the distance at which they can see the fingers (e.g.- "Count Fingers at 10 ft."). You will see to test for "Light Projection", specifying if the patient can tell which direction the light is coming from. Forget about this.

The big white light on the direct ophthalmoscope makes an ideal light for testing light perception. It is even better for...

### Pupils

This is part of a complete physical or neuro exam. You also want to check pupils when there is vision loss, to see if there is a relative afferent pupillary defect (RAPD); we called this a Marcus Gunn pupil in my day.

You are looking for...

size: equal? appropriate for age and lighting conditions?

shape

#### Pupils Technique:

Darkish room, if possible. That way, you are starting out with dilated pupils, so you can see them react better.

Have them focus something across the room. If you let them change their focus as you do the test, the pupils will change size—larger for distance and smaller for near.

Use the big white light on your ophthalmoscope.

Shine the light indirectly at the eyes, and check size and shape.

Shine the light directly into each eye, and check reactivity.



Do the swinging flashlight test to check for an RAPD. Leave the light shining on each eye for about one second, no faster. You will miss RAPD's if you swing back and forth too fast.

I will leave specific findings up to the textbooks to discuss—it is a big topic. A couple of salient items, however:

- An RAPD almost always indicates a malfunctioning optic nerve. This can be glaucoma, or demyelinating or ischemic disease. Other causes are unusual, but we always have to be on the lookout for a lesion compressing the optic nerve.
- About 15% of people have anisocoria (unequal pupils) at any given moment. This can come and go in a given individual and even alternate between eyes. Anisocoria is normal if the pupils react briskly, there is no ptosis and no diplopia.

### Pregnancy

Diabetics need an exam every trimester. The risk of retinopathy is increased.

Pituitary tumor: tumor more likely to grow. Get automated visual field every month.

Some glaucoma meds are contra-indicated. Stop non-selective beta-blockers two weeks before anticipated delivery.

### One-Eyed Patients

Patients with useful vision in only one eye need to wear glasses every waking hour to protect the good eye. The lenses should be of unbreakable material (polycarbonate or TriVex ). The frames should be sturdy—never rimless or wire-mount (a cable holding the bottom of the lens). They also should have an eye exam every year. Please help us remind the patient about these items.

### Prosthetic Eyes

Technically, the prosthesis should be cleaned every day. In practice, no one bothers with this, unless they are symptomatic. If the patient is getting discharge or chronic irritation, they should be evaluated. Some problems, such as extrusion of the orbital implant, should be treated, so bigger problems can be avoided down the road.

### Driving Requirements in Pennsylvania

You need to see 20/40 or better in the better-seeing eye to drive in the nighttime.

You need 20/70 for daytime driving.

If uncorrected acuity is less than 20/40 in either eye, and it can be corrected to 20/40 or better, the patient must wear correction (glasses or contacts) to drive.

If your best-corrected acuity is 20/80 or 20/100, you can get a special license to drive—daytime, local, no highways.

Visual fields must be 120 degrees or more.

We are required to report to Pennsylvania Department of Transportation anyone who doesn't qualify.

## EXTERNAL DISEASE

### I. The Acute Red Eye

As you know, you will see this over and over again. You can treat obvious bacterial conjunctivitis yourself. Also, you can sit tight on presumed viral conjunctivitis to see if it gets better on its own, although the patient will usually appreciate seeing an ophthalmologist. Refer everything else.

Red flags for the acute red eye (referral necessary):

- Pain, photophobia
- Ciliary flush – This is the pattern of redness in which you see bright red adjacent to the limbus. (*Pain and ciliary flush are almost always keratitis or iritis and need to be referred!*)
- Blurred vision
- H/O herpes simplex eye infection (This could be a recurrence.)
- H/O glaucoma surgery (risk of endophthalmitis)
- H/O contact lens wear (risk of corneal ulcer)
- H/O corneal transplant (risk of graft rejection)

The commonest or most important causes of acute red eye are:

Bacterial Conjunctivitis: The keys here are acute, recent onset, purulent discharge and minor discomfort. The discomfort will be in the form of burning, not pain. The redness will not involve a ciliary flush.

Treatment: Antibiotic drops for 5 days. Good choices would be...

- tobramycin
- Polytrim (TMP/polymyxin B)
- Fourth-generation fluoroquinolones: Vigamox, Zymar, Besivance, Quixin

If you don't get a quick response, refer. Don't continue the antibiotic longer than five days—it is not working and is not going to.

Viral Conjunctivitis: You tend to see a lot of lid edema and chemosis (conjunctival edema). It can be pretty dramatic—it can be so severe that you may think that the patient has an orbital cellulitis. The discharge is more watery or mucoid than purulent.

These people are very contagious! Tell them that if they touch their eye, they should wash their hands. They should use the same precautions that you would use to prevent spreading a cold.

If the bug is adenovirus, sometimes Zirgan (ganciclovir) drops will kill it. There is also the option of Betadine instilled at the time of the visit (let the ophthalmologist do this). I have

personally tried this and am not impressed. We tell them that they could (and usually do) take 10 days to clear up. You can manage these yourself, as long as there are not the red flags as above and you are comfortable with the diagnosis. Unless it is mild, the patient will probably want to see an ophthalmologist anyway.

Iritis and Keratitis present with pain and photophobia. The pattern of bulbar injection is often a ciliary flush. Refer these.

In a contact lens wearer suspect a bacterial corneal ulcer. That needs to be seen that day. Ulcers can melt a full-thickness hole in the cornea or cause scarring and permanent vision loss!

In someone with a history of iritis, suspect recurrent iritis. Iritis has a very characteristic feel to it, and the patient will almost always know that they have got it again. Treatment is steroid drops.

A few other acute red eyes you will see often...

Recurrent Corneal Erosions: These are episodes in which a patch of corneal epithelium comes loose. They can be from a corneal dystrophy, especially anterior basement membrane dystrophy, which is very common. They can be a residual effect from a prior corneal abrasion, especially those caused by organic material, like fingernails (those darned 2-year-olds!) or tree branches. What happens is that the hemidesmosomes and other biological connectors that are supposed to be anchoring the epithelium are defective, so the patient wakes up in the morning or the middle of the night, and that patch of loosely-adherent cells sticks to the back of the lid rather than the cornea. These can really hurt. If the patient says they are in severe pain, believe them. The key to the diagnosis is that the onset is upon waking. Refer all of these. We usually have to debride the loose epithelium. Also, there are other treatments we can do to prevent more episodes.

Herpes Simplex Keratitis: The patient has modest irritation, not severe pain. There is a 50% recurrence rate, so you would suspect it if there is a past history of herpetic keratitis. You will know to refer these—there is some pain, and they will usually have a ciliary flush. Usually, a dendritic ulcer on fluorescein staining is visible with the naked eye and a cobalt blue light. Treatment is topical and/or oral antivirals (e.g., acyclovir), with or without debridement.

S/P Trabeculectomy: This is urgent and needs to be seen within a few hours. If the bleb is infected, it can turn into an endophlebitis quickly, destroying the eye. See the glaucoma section for more on this operation.

Contact Lenses: These should always be referred. As a primary care doctor, you really don't have a good way to make a diagnosis. It could be overwear, contact lens-induced keratitis, a corneal ulcer or unrelated to the contact lenses. You need a slit lamp to evaluate.

Acute Angle Closure Glaucoma: Acute red eyes are almost never this, but it is not uncommon to get a referral from a primary care doc who is worried about this possibility. In acute glaucoma, the pupil will be mid-dilated. The patient will have dull pain and blurred vision. If you do suspect this, it needs to be seen right now. We will treat by doing a laser iridotomy—putting a

hole in the iris. This is a quick, simple office procedure. The mechanism of angle closure glaucoma is in the manual's supplement I will give you, when you are here.

Allergic Conjunctivitis/Blepharitis: There are two basic reactions: Type I and Type IV hypersensitivity reactions.

- Type I reactions at their mildest cause itching, and the eyes look normal. At worst, you see a lot of chemosis and lid edema. These are often seasonal. Always ask if the patient is putting any drops in their eyes—this is the other common allergen. Usually, you can't find a specific allergen, but miraculously they almost never get recurrent symptoms after you stop the drops in a couple weeks. Treatment:
  - Mild: OTC anti-histamine drops QID: Naphcon A, Opcon A. Even Visene works.
  - Moderate: anti-histamine/mast cell stabilizer drops: Patanol, Zaditor, Optivar, Elestat. Which is best? I don't know—they all work.
  - Severe: Topical steroids
- Type IV reactions mostly consist of lid edema. It has a very characteristic appearance—kind of a ruddy look. Once you see it, you know. Treat the dermatitis with steroid cream or ointment. I use triamcinolone 0.1% cream TID. Don't let the patient use it longer than about a month, or the skin can break down and get depigmented. If you can't get them off the steroid, send the patient to an allergist.

## II. The Chronic Red Eye

The possible causes are innumerable, and you will end up referring most of these. You can start treatment for blepharitis yourself.

It is common for nursing home patients to have chronic inflammation and discharge, sometimes from an ectropion. Don't put them on long-term antibiotic drops—it will not work. Get them referred, if it is something they can't live with.

Blepharitis: This is seborrheic inflammation of the lids. It is very common in patients with rosacea. The typical picture is burning, chronic redness of the lids and often the globe, and waking with matter on the lashes and lids. You will see thick, red lids, scurf (dandruff) at the base of the lashes and possible rosacea.

This is a condition you can treat. The initial therapy is hot soaks/ lid scrubs/ antibiotic ointment at h.s. Actually, the best choice of antibiotic may be AzaSite, which is a thick azithromycin drop; don't try to use this one yourself, because it requires special instructions for the patient. I am including our instructional handout we give to patients for technique. Be sure to tell the patient that it may take two weeks to notice an improvement. The treatment can be extended indefinitely and often must be—this is a chronic condition. If the patient is not better in a couple weeks, refer. There is more that we can do for it. This includes chronic oral tetracycline. We are using

the tetracycline for its anti-inflammatory effect and salutary effect on the physiology of the sebaceous glands of the lids, rather than for its ability to kill bacteria.

Next page: Blepharitis handout for patients

## **BLEPHARITIS**

Blepharitis is an inflammation of the eyelids. It typically causes burning eyes, thick, red eyelids, and styes. It is sometimes caused by Staph or by other microbes and may be related to some skin conditions, like rosacea. The oil glands in the eyelids are abnormal and become plugged. The surface of the eye becomes acidic and the tear film can break down quickly, causing dryness and burning. It is a chronic, smoldering problem and never really goes away. The symptoms wax and wane, and there is a lot we can do to reduce them, when they get worse. Some patients benefit by doing some form of treatment on an ongoing basis.

These are some of the options:

- Lid Scrubs – Once or twice a day, soak both eyes with a hot washcloth for two minutes. Then close your eyes and scrub horizontally across the margins of the eyelids (at the base of your eyelashes) with a wet Q-Tip. Scrub for about 20-30 seconds, and then rinse the eyes with warm water or the wash cloth.
- You may find that the hot soaks feel really good—go ahead and do them as much as you want. Sometimes, a hot baked potato covered in a wet wash cloth is a good heat source, because it retains heat longer than a washcloth. It is up to you if you want to eat the potato!
- SteriLid or OCuSOFT eyelid cleanser: follow directions on package
- Drops: \_\_\_\_\_
- AzaSite: this is a thick drop. Place a drop in the eye, close your eyes and rub the excess into you eyelid margins. This can be expensive—let us know if your drug plan doesn't cover it.
- Ointment: \_\_\_\_\_ Apply to base of lashes and place a bead in each eye, at bedtime. Close your lids for a minute after applying to let the ointment reach body temperature and spread out.
- Artificial tears: Blepharitis can make dry eyes worse, and vice versa. Putting in artificial tears or lubricating drops (not redness relievers) can help dilute the acids in your tear film that cause burning. Some recommended artificial tears are: Soothe, Soothe XP, Refresh, Optive, Systane, TheraTears, GenTeal.
- Doxycycline pills: 1 pill 2x/day for one month, then 1/day for \_\_\_\_\_

Often, it takes a couple weeks to notice an improvement. If you do not get an adequate response after two weeks, we will change therapies.

Entropion/ Ectropion: (See “Plastics” section) Watch for this especially in older patients. In case you have forgotten, an entropion is an in-turning and an ectropion an out-turning of the lower lids. Both are most commonly from aging changes in the tissues of the lids. They need surgery.

Graves’: Suspect Graves’ as a cause of chronic ocular inflammation, if the patient wakes with lid edema, has visibly injected extraocular muscles, has lid retraction or proptosis or a history of hyperthyroidism. We are limited in doing anything about the inflammation itself, but we can at least educate the patient about the disease and monitor them for other manifestations. Oral selenium supplements could help mitigate the disease. Smoking is a major aggravator. Prednisone helps with the acute inflammatory phase of the disease.

To name but a few other causes of chronic ocular inflammation:

- Infectious: Molluscum, Chlamydia, MRSA, lice
- Inflammatory: Dry eyes, neurotrophic, exposure, toxic (drops, fumes)
- Traumatic: Trichiasis (lashes rubbing on cornea), eye rubbing
- Contact lens issues

### III. Other External Disease Topics

#### Dry Eyes

Dry eyes is very common. Post-menopausal women are the most at risk because of the associated hormonal changes. Often, the patient will tell you that their eyes feel “dry,” but the history will often be “tired” eyes towards the end of the day, or after prolonged use of the eyes with reading or computer. Blepharitis will aggravate dry eyes, because the associated abnormal lipid layer of the tear film causes rapid tear evaporation. Some medications, like anti-histamines, anti-cholinergics, diuretics, and, ironically, hormone replacement therapy for post-menopause (because of the anti-androgen effect) aggravate it too.

If the history suggests dry eyes, you can start therapy. Have the patient use artificial tears frequently. I recommend avoiding cheaper store brands, because they are often preserved with benzalkonium chloride, which can be harsh on the ocular surface if used very often at all. Here are some good brands of tears:

- TheraTears
- GenTeal
- Refresh
- Soothe
- Systane
- anything without preservatives

We commonly use topical cyclosporin A drops (Restasis) on a chronic basis, and this usually works very well. Dry eyes is an autoimmune disease of the lacrimal glands, and the CsA restores



and then maintains normal anatomy and physiology of the glands. The patient must try it for at least three months to see if it will work.

Blocking the tear egress from the eye by blocking the punctae is a treatment option too. You will see patients who have gotten punctal plugs. The silicone ones are prone to falling out or to subluxating and scratching the eye. We can cauterize the puncta and canaliculi too, but that is not a reversible procedure, so we must be very careful on whom we do it, lest we replace dry eye with epiphora.

### Corneal Transplantation

This is done for corneal scars, edema (especially Fuchs dystrophy and bullous keratopathy after cataract surgery) and irregularity (most commonly, keratoconus).

A few things you need to know:

- The wound on a full-thickness transplant is very weak, so patients need to be cautious with their physical activity much longer than they are for cataract surgery. They also should be wearing glasses full-time as protection—this is a permanent admonition.
- It is often a very long time before they are fully rehabilitated and able to see well.
- There is always the chance of rejection. They are kept on maintenance topical steroids permanently to prevent this. Any patient who develops a red eye after a corneal transplant needs to be seen that day! If there is rejection, they need to be on heavy-dosed topical steroids.
- There is a new generation of transplant procedures designed to replace just the corneal endothelium—good for those with corneal edema. If you just replace the endothelium, you can do the surgery through a small incision, there is little astigmatism and less chance of rejection. Also, the wound is much stronger than with a full-thickness graft. DLEK and DSAEK are two of these.
- A triple procedure is corneal transplant/cataract extraction/IOL.
- Fuchs Dystrophy is a common autosomal dominant corneal disorder in which the patient loses so many corneal endothelial cells throughout life that they eventually don't have enough cells to pump water out of the cornea, so it clouds up. The first symptom is cloudy vision upon waking—there is no evaporation of water out of the cornea through closed eyelids at night, and evaporation is the other way water leaves the cornea.
- Keratoconus is a corneal dystrophy in which the central cornea becomes thin, causing extreme irregular astigmatism. Usually, they can see decently with specially-designed hard contact lenses, but sometime you need a transplant.

## OCULO-PLASTICS

By this, we mean lids, lacrimal apparatus and orbit. We have oculo-plastic sub-specialists—they do lid surgery better than most plastic surgeons, and are good to refer to for your blepharoplasties, ptosis surgeries, ectropion and entropion repairs, etc.

### I. Orbital Cellulitis

This is an infection of the orbit, usually spread from ethmoid sinusitis. You see not only lots of lid edema but also chemosis (edema of the conjunctiva). There is usually some degree of proptosis. Do not try to treat these yourself! Have a high index of suspicion when there is severe lid edema. They need prompt attention. The patient will need a CT to confirm the diagnosis, admission to the hospital for IV antibiotics, ID and ENT consults (you often need to have the sinuses addressed +/- drainage of a periorbital abscess), and frequent observation. Potentially, orbital cellulitis can lead to meningitis, brain abscess, loss of vision or death.

I have seen really severe viral conjunctivitis mimic orbital cellulitis clinically. The CT will distinguish. Also in the differential is orbital inflammatory pseudotumor (n.b.).

If you are the one ordering the CT, be sure to get the sinuses and the frontal lobes of the brain (looking for abscess). Lack of sinusitis should make you question the diagnosis.

### II. Bell's Palsy

From the eye standpoint, the important thing is to protect the eye from drying, since the lids don't close fully. In the short term, have the patient lubricate the heck out of the eye. I recommend hourly artificial tears. The best is a brand that is thick and non-preserved, or which has a "disappearing preservative." Examples: Refresh Liquigel, GenTeal Gel, Systane Gel.

You want to have the patient use an ointment at night. Refresh PM is your best bet here. Lacri-Lube is OK, but it is preserved with BAK, which can be harsh to the ocular surface, over the long haul.

If the eye is white and quiet and comfortable, just have the patient lubricate until the lids function normally again. If problems, refer. We may do a tarsorrhaphy or other surgery to decrease corneal exposure. Tarsorrhaphies can be temporary or permanent.

Don't forget to put the patient on a short course of oral steroids to increase the odds of recovery. Also, a systemic anti-viral is indicated, in case the Bell's is from zoster. Finally, a lab or imaging work-up may be in order, if there is an atypical presentation.

### III. Ectropion (out-turned lid)

Senile ectropion is very common. If mild, it will cause tearing. If severe, it will cause a chronically inflamed eye with constant purulent discharge. If really severe, it will allow drying of the ocular surface. Surgical correction is very simple, and they should all be repaired except if very subtle and not causing symptoms.

#### IV. Entropion (in-turned lid)

Senile entropion is very common also. Both senile en- and ectropion are caused by weakening of the tissues that keeps the lower lid at its proper tension. Patients will get rubbing of the lashes on the cornea. This is irritating and unsafe—the chronic rubbing damages the cornea and can lead to corneal ulcers. Get it fixed.

Entropion and ectropion surgery are usually simple outpatient procedures done under local anesthesia. There is minimal systemic risk, so don't be overly restrictive in approving surgery. You could be unnecessarily depriving the patient of a needed procedure. Plastic surgeons can do these, but I usually use an oculoplastics specialist.

#### V. Ptosis

Ptosis is a lowering of the upper lid margin. It is not excess lid skin &/or fat, which is called “dermatochalasis.”

Senile ptosis is from a stretching of the levator aponeurosis. The lid crease is either high or gone. If the patient desires, it can be corrected surgically.

Ptosis can also be neurological (e.g. CN III palsy, Horner's) or muscular (e.g. myasthenia gravis) in etiology. These need a workup when you see or suspect them.

#### VI. Dermatochalasis

This is excess skin &/or fat in the lids. The fat is herniated from the orbit, through dehiscences in the orbital septum. It is an aging phenomenon.

Dermatochalasis can be corrected surgically by doing a blepharoplasty. All the plastic surgeons do these. Insurances will pay for them, if we can document that the excess skin is severe enough to block the superior field of vision. We can do an automated visual field with and without the lids taped (simulating having had the surgery) to see if the patient qualifies.

Dermatochalasis is not lid edema, even though patients think it is “swelling” when there is herniated orbital fat.

#### VII. Lid Edema

There are many causes. Here are some of the commoner ones.

- If chronic, consider Graves' disease. Look for other signs, like thick extraocular muscles or a history of hyperthyroidism. This gets missed a lot. The edema is worst in the morning upon wakening. There may be boggy conjunctiva and/or swollen extra-ocular muscles too. You can see the swollen EOMs best by looking at the lateral rectus.
- Lid edema can be allergic—the patient will have itching too. This can be acute or chronic, and represent acute hypersensitivity, contact allergy, atopic disease or vernal conjunctivitis. There is frequently chemosis and allergic conjunctivitis as well.
- If the edema is acute, in only one lid and accompanied by redness and tenderness and calor, it is probably pre-septal cellulitis from an infected stye. These patients look infected. Note that the globe is not involved.
- Viral conjunctivitis can cause massive lid edema. The patient will be in significant discomfort and light sensitivity and have red eyes with chemosis. There may be palpable preauricular nodes.
- Orbital cellulitis—see above. There will be pain, probably fever and proptosis and possible decreased EOM's or vision. This can be distinguished (most of the time) from inflammatory pseudotumor with the CT and by pseudotumor's lack of response to antibiotics.
- Inflammatory pseudotumor is an inflammatory infiltration of the orbit. It looks a lot like orbital cellulitis, but the patient tends not to be febrile and the sinuses are clear on CT. We treat this with several weeks of oral steroids.
- Some meds: calcium channel blockers, bisphosphonates

If you are confident that you are dealing with preseptal cellulitis or an acute allergic condition, you can treat it yourself. If in doubt, especially if there is involvement of the eye itself, refer.

## VII. Graves' Disease

As you know, Graves' disease is an autoimmune disease that can affect the thyroid and peri-ocular tissues. The thyroid and ocular manifestations occur independently—the ocular disease is not from the hyperthyroidism, and the two can occur years apart. I recently had a case in which the eye findings showed up 12 years after the thyroid disease! The laboratory screen for Graves' is TSH, free T4, and thyroid antibodies. Graves' orbitopathy is characterized on CT by thickening of the extraocular muscles that spares the muscle insertions.

Eye-wise, there is an acute inflammatory phase and a “congestive” phase. In the inflammatory phase, we see lid edema, worse in the morning. If you look at the extraocular muscles, you can see that they are thick and red—this finding is virtually diagnostic. These two findings are classic. If things get really bad in this phase, we can treat it with systemic steroids. Later, we see proptosis and lid retraction. Exposure keratopathy is a concern, and you should ask your patients if they are having much eye discomfort.

Less common is diplopia, from involvement with the extraocular muscles. The real fear is compression of the optic nerve by the enlarged muscles. Patients with Graves' and vision loss

need prompt evaluation! The optic nerve involvement correlates more with diplopia rather than with proptosis.

You will want to refer patients with possible Graves' disease for evaluation and follow-up and for treatment.

### VIII Epiphora

If there is no ocular irritation, tearing is usually from insufficient tear drainage through the lacrimal system.

Look at the lids. If there is an ectropion or lid weakness (e.g., Bell's palsy), that would be it. The tears don't just passively drain into the punctae. They have to be pumped into the punctae by proper functioning of the lids. Sometimes, we can help this with lid surgery.

Look at the punctae. If you can barely see them, punctal stenosis is the etiology. We can open them up in the office, either by dilating or by doing a snip procedure.

Partial or complete blockage of the naso-lacrimal duct or sac is common, especially in the elderly. Refer them in. We can probe and irrigate the lacrimal system here in the office and sometimes improve the tearing, at least temporarily. At least this procedure can help us figure out where the obstruction is—punctum, canaliculus, common canaliculus, lacrimal sac, naso-lacrimal duct or nose. Ultimately, we often need to do a dacryocystorhinostomy (DCR) to relieve the tearing. The DCR is a procedure in which we make an incision medial to the medial canthus and drill a hole through the bone into the nose.

Be sure to check for a palpable lacrimal mass! Carcinoma of the lacrimal sac is rare, but I just saw one last month.

If the patient has edema of the nasal mucosa for whatever reason, the ostium to the naso-lacrimal duct could be obstructed. Obviously, the solution here is to treat the rhinitis.

If the patient has tearing in association with ocular irritation, the tearing is from excess tear production from the eye problem. Send them in for evaluation.

Epiphora in children:

- Pure epiphora could be a number of things, but the really worrisome thing is congenital glaucoma. Refer to ophthalmology.
- Epiphora with yellow discharge is most likely from a congenitally blocked nasolacrimal duct. Babies have a membrane over the end of the duct in utero. Frequently, they are born before this absorbs. It usually will absorb in the first few months of life, but if the membrane doesn't spontaneously resolve by age six months, it is unlikely to do so spontaneously. The membrane should then be probed open under light general anesthesia in the OR, because after age nine months the probing becomes less likely to work.

## IX Chalazions and Styes

These are masses of inflammation around lipid that has escaped from clogged Meibomian or other sebaceous glands in the lids. Patients with rosacea are especially prone. Often patients have several-month periods in which they get chalazion after chalazion-- it probably is related to some sort of hormonal or immune alteration.

These have a few different presentations:

- Typically, you will see a pink mass. This can present to either surface of the lid. Refer these in. We have to follow them to resolution. Rarely, they can be a sebaceous cell carcinoma, which is highly malignant. We either I&D, inject with triamcinolone or do an excisional biopsy, depending on circumstances.
- Sometimes, they will present as an acutely inflamed eyelid, known as preseptal cellulitis (pre-orbital septum, that is). You will want to treat these initially with an oral antibiotic (Gram positive killer of your choice) and have the patient look and feel for a mass after the inflammation is gone. If there is a mass in six weeks, we should treat per above.
- Acute onset of a lid abscess should be incised and drained. The patient will be in misery if we don't.
- Some chalazions respond to injection with Kenalog 40.

## NEURO-OPHTHALMOLOGY

You only think that this is an obscure subject! If you screw this up, you can kill or blind people. You won't see this stuff very often, but watch out! We are only going to cover a few topics that you are likely to encounter or that are particularly dangerous. You will certainly be referring most neuro cases to an ophthalmologist or neurologist.

### I. Temporal arteritis/Giant cell arteritis

If you are only going to remember only one thing from this manual, this would be it. Elderly people with the following need attention ASAP!!!

- Transient visual loss: Amaurosis fugax is a black-out of vision in one eye, lasting 10 to 30 minutes. If it lasts seconds, it's not it.
- Sudden black-out or dimming of vision in one eye (without recovery). This can be from ischemic optic neuropathy or, less commonly, central retinal artery occlusion. Both of these can be from giant cell arteritis.
- Sudden onset of diplopia in an elderly person

In an older patient (over 60), proceed as follows:

Get the patient in to your office or mine now. If yours, give them three or four 20-mg prednisone tablets in a pill envelope and a lab slip to take now to the nearest lab, do not pass "Go." You will be ordering a sedimentation rate, quantitative CRP and complete blood count. The patient must take all the prednisone there at the lab, as soon as the blood is drawn. The patient can go suddenly blind from this disease, if untreated, and the prednisone is usually very effective in preventing this disastrous complication. Then, and only then, do they see the ophthalmologist, if the ophthalmologist wasn't the first person to see them. This is a good test for your ophthalmologist—he should drop everything to take care of this patient immediately, if he is to be the first physician seeing the patient.

If any of the labs comes back abnormal, keep the patient on prednisone, 60mg per day, pending the results of a temporal artery biopsy. You may want to have the patient see a rheumatologist. If the biopsy was positive, their temporal arteritis will need managing. If it is negative, and you have a good clinical case for temporal arteritis (i.e., other symptoms of the disease), you will want the rheumatologist's opinion as to the probability that the patient has the disease. I would also recommend ordering a biopsy of the other side.

Just as reminder, other symptoms of temporal arteritis are headaches (especially, temporal ones), neck pain, jaw claudication, increase in joint or muscle pains, constitutional symptoms.

### II. Amaurosis Fugax

This is transient monocular visual loss, lasting several minutes. Be sure you rule out temporal arteritis, as per above!! However, usually, these are embolic. After you have ruled-out GCA, you need to find the source of the embolus. Work this up and treat as you would for any TIA in the carotid distribution. You need a carotid duplex and an echocardiogram. Also, screen for and address risk factors for atherosclerosis and start daily ASA and a statin.

In a younger patient consider hypercoagulable state, vasculitis, Uhthoff 's syndrome (from MS). Always get an ophthalmologist involved.

### III. Diplopia

First, there are two main things to remember. Our cardiology professor in med school always pointed out the “big print” material, so here it is, in big print, no less.

**A. If it is an elderly patient, you must do the “R/O temporal arteritis” work-up.**

**B. Not everyone who has diplopia needs to be imaged.** In fact, the majority of patients with diplopia, whether or not it is from a cranial nerve palsy, do not need an MRI.

Another thing to be cognizant of is third nerve palsy. They need an angiogram (usually MRA or CTA) and a neurosurgical consult promptly. You need to look for an aneurism of the posterior communicating artery. You can avoid the angiogram, if and only if:

- There is complete motor block, and...
- There is complete sparing of the pupil, and...
- They are not between the ages of 20-50, the prime aneurism ages.

If you do observe a 3<sup>rd</sup>, you must see them the next day. Any pupil involvement and it’s off to the neurosurgeon.

Sudden-onset 4<sup>th</sup> and 6<sup>th</sup> palsies are almost always microvascular. Rule out temporal arteritis promptly in the elderly. Check the “usual suspects” (blood glucose, blood pressure, lipid profile), and watch. They need a work-up if they are not isolated or if they don’t go away after 3 months.

Fluctuating diplopia is often myasthenia gravis. You can have pure ocular myasthenia. Pure ocular “myasthenia” requires an MRI of the brainstem, in case it’s not.

Pure ocular myasthenia needs treatment with prednisone. That decreases the probability of the myasthenia going systemic.

Needless to say, refer anything equivocal.

### IV. Migraine



Flashes and photopsias that last minutes (usually 10-30) are almost always migraine. They are often the classic blind spot evolving into an expanding ring of flashes, but often the episodes are atypical. If there are no other neuro symptoms with the episodes, get a visual field at the ophthalmologist's. A normal field would pretty much rule out other pathology. If there are other neuro symptoms, get an MRI/MRA of the brain with attention to the occipital cortex. The concern here is AVM or aneurism. Keep in mind that flashes that last more than a split second are not from the retina, so don't refer them in on an urgent basis to rule out retinal problems.

## V. Pain

You will hear every variation under the sun if you practice long enough. There are some specific types I want to discuss.

- Quick lancinating pain around the eye(s): This is called the "jabs and jolts syndrome (cryptic, no?)." We don't know what causes it, but it is totally benign. It might be a neuritic pain.
- Vague, dull pain around an eye, normal exam: We almost never find an etiology. Don't scan them, unless the pain is progressively worse or if there are other signs or specific symptoms.
- Episodes of pain around the eye associated with autonomic symptoms could be one of a few syndromes (paroxysmal hemicranias, cluster headache, hemicranias continua, SUNCT). PH and HC can respond to oral NSAIDS. Cluster responds to O2.

## VI. How to Tell if the Optic Nerve is Swollen

Not every nerve with fuzzy margins is swollen. Some discs are just anomalous.

- Swollen nerves are usually hyperemic. The color of a normal disc is orange-pink. A swollen disc is reddish. Compare the two eyes.
- If you see nerve fiber layer hemorrhages near the disc, it's swollen.
- If the capillaries are dilated and visible, it's swollen.
- If the large vessels on the disc are partly obscured by edema, it's swollen.
- If you see spontaneous venous pulsations, the nerve is not swollen.

## VII. Causes of disc edema

Naturally, the list is very long. You should know...

- If both discs are swollen, you may be dealing with papilledema, which is, by definition, disc edema from increased intracranial pressure. Get the patient to a neurologist immediately (or to an ophthalmologist, to confirm the diagnosis, if needed). Of course, it is possible to have any disease of the optic nerve bilaterally, but bilateral disc edema is papilledema until proven otherwise. Needless to say, the commonest cause of papilledema is brain tumor.

- In younger adults, the most likely cause of monocular disc edema is optic neuritis. The patient will also have dim vision and pain on extra-ocular movement. We will have to work them up for demyelinating disease. In patients over 45, it is likely non-arteritic ischemic optic neuropathy. This is a condition with a very complex etiology, which we don't really understand. The etiology is not strictly atherosclerotic, but ION and atherosclerosis share the same epidemiological risk factors. They will have painless vision loss, often an "altitudinal defect" (superior or inferior ½ of the visual field wiped-out).
- Other etiologies include various inflammatory, vascular, infectious, ischemic, neoplastic, hereditary, nutritional and toxic conditions. Don't clog your brain with them—just refer these out.

That's all I will say about etiology. You will be referring every case of disc edema to an ophthalmologist, neurologist or neurosurgeon. If you are the one to order the MRI on a case of suspected papilledema, don't forget to order contrast. If it is monocular disc edema, let the ophthalmologist order the MRI.

### VIII. Optic Atrophy

If you happen to incidentally discover a pale optic nerve, send it to an ophthalmologist for a work-up. It is from a compressive lesion of the optic nerve until proven otherwise.

### IX. Hemianopsias

Homonymous hemianopsias or quadrantanopsias occur from damage to the occipital cortex, or less often optic tract or optic radiation. CVA is the usual cause. These patients need to be evaluated by an ophthalmologist to see if the peripheral vision is adequate for driving. A complete homonymous hemianopsia does not allow enough peripheral vision to drive. There is a type of vision therapy called Vision Recovery Therapy in which the patient does exercises on the computer every day to try to recruit undamaged parts of the brain into performing some side-vision function. The computer is provided by the company, and a vision therapist is the one directing the therapy. Vision therapists are optometrists that specialize in that. I have never seen this to be very effective.

We can put a Peli Lens on one of the patient's glasses' lenses, in cases of dense homonymous hemi. That allows some vision in the obliterated field. Its purpose is to keep the patient from running into things or missing objects. You can't drive with a Peli Lens.

Bi-temporal hemianopsias are almost always from pituitary tumors. Often there is substantial recovery of vision after the tumor is removed. The concern about driving is valid here too.

### X. Pseudotumor Cerebri

Call it “idiopathic intra-cranial hypertension,” if you want to be up-to-date. I call it “glaucoma of the brain.” The ICP goes up, causing the typical headaches of this and all of the other symptoms of high ICP. The MRI will be normal. It occurs almost exclusively in obese females in young adulthood. The following drugs can cause it: tetracyclines, vitamin A, isotretinoin, lithium, Dilantin. The neurologist will manage these. They need an eye exam at baseline, including visual fields. If this is normal, we see them every several months. If they perceive any loss of vision, they need to see an ophthalmologist pronto. Therapy for acute vision loss is emergency LP to get the ICP down and IV steroids, followed by a VP shunt, optic nerve sheath fenestration or microshunt.

### XI. Horner’s Syndrome

This is mild unilateral ptosis with miosis. The miosis is evident in the dark. Leave the patient in the dark for about twenty seconds before checking for the miosis. Horner’s indicates interruption of the sympathetic nerve supply to the eye and lid. Have a neurologist work these up. The pathology could be anywhere along the SNS supply chain: brainstem, cervical chain ganglia, apex of the lung, neck, cavernous sinus. Idiopathic, lung cancer and S/P neck trauma/surgery are the commonest.

### XII. Cataclysmic Neuro-ophthalmology Problems

No need for a lot of detail. You will be referring these stat!

- Sudden blackout of vision in one eye. Described above.
- Sudden onset of diplopia in an older person. Ditto.
- Pituitary Apoplexy: Bleeding into a pituitary tumor is the cause. Severe headache accompanied by cranial nerve III, IV or VI or by acute loss of vision in one or both eyes make you consider the diagnosis. This is a neuro-surgical emergency. The now-gigantic pituitary can compress the brainstem, hypothalamus or chiasm. Hypopituitarism needs to be addressed.
- Painful Horner’s is a dissecting aneurism of the carotid artery, until proven otherwise. They need emergency anticoagulation. Diagnosis is with MRA of the carotid &/or MRI of the base of the skull with T2-weighted imaging—let the neurologist order this.
- Acute CN III Nerve Palsy: This is an aneurism of the posterior communicator artery until proven otherwise. See “Diplopia” section.
- 

### XIII. Drugs That Cause Optic Neuropathy

There are many. Some ones to take note of:

- Amiodarone: onset is usually after several months of use
- Ethambutol: these patients need to be monitored, most say monthly
- Cyclosporin A and tacrolimus
- Methotrexate: preventable by taking folate supplements

- Various chemo agents
- isotretinoin

## GLAUCOMA

Glaucoma should really be called “Pressure-Related Optic Neuropathy” (PRON), in my opinion. But then, people would confuse it with an infestation by large shrimp. Glaucoma is an optic neuropathy that is, at least in part, caused by a pressure that is too high for that individual’s eye to tolerate. Some eyes get glaucoma with a pressure of 17, and others don’t get it with a pressure of 30. The higher the intraocular pressure, the more likely you are to get glaucoma. The only proven way to halt the progression of the disease is to get the pressure down.

### Etiology:

What really causes glaucoma and what is the pathophysiology? We don’t know, exactly. Obviously, pressure has something to do with it—why else would lowering the pressure halt the course of the disease (usually)? It could be that the pressure squeezes the axons of the optic nerve at the disc. There could be a vascular component—abnormal circulation of various types has been demonstrated in and around the optic nerve head in glaucoma. It could also be partly a neuro-degenerative disease. Epidemiologically-speaking, glaucoma is more common in those with a family history, blacks (it is more aggressive and harder to treat in blacks—be especially sure your black patients are getting eye exams), and those with a history of migraine.

### Glaucomatous Cupping:

As primary care doctors, you should be able to look at an optic nerve and recognize obvious glaucomatous disc damage. What happens is that the retina’s ganglion cells die. Their axons form the bulk of the optic nerve, so when they disappear, it leaves a vacant area on the nerve head. That’s the cup. On your rotation here, you will get plenty of chances to see glaucomatous cupping. I will include pictures in this manual. Basically, if someone has an increased cup-to-disc ratio, you should refer them for possible glaucoma. You are looking for the percent of the total area of the optic nerve head that is taken up by the cup, converted into a decimal (e.g.: 40% = 0.4). A cup-to-disc ratio of greater than 0.5 should be referred. There is a lot more to evaluating the optic nerve for glaucoma than that, but that is all *you* have to remember. I should say that you will definitely miss some glaucomatous cupping, no matter how diligent you are. You are looking with a direct ophthalmoscope, which gives you only a 2D view, as opposed to the 3D view the ophthalmologist gets. Also, you are looking through an undilated pupil. Still, you will pick up some cases, if you try.

### Types of Glaucoma

There are a bazillion types of glaucoma, but it kind of boils down to two basics concepts.

1. Open angle glaucoma: There is no physical obstruction of the trabecular meshwork, the eye’s drain. There are many specific sub-types, but the one we see over and over is “primary open angle glaucoma.” The treatment strategy is to get the intra-ocular pressure (IOP) down by either improving drainage of aqueous from the eye or by getting the eye to produce less aqueous.

2. Closed angle glaucoma: Again, there are many sub-types, but the common denominator is that the trabecular meshwork (TM) is closed by the iris. Treatment here depends on the cause of the angle closure. The angle closure is usually from pupillary block (“primary

angle closure glaucoma,” caused by too fat of a lens in too small of an eye). The treatment is to create a hole in the iris with a laser (laser iridotomy). You will get a handout on this on your rotation.

I am sure you have heard about acute angle-closure glaucoma. As you know, there is an acutely painful red eye. They come in with a steamy cornea and a mid-dilated pupil, about to 6-7 mm. Obviously, this requires immediate attention. The IOP is usually sky-high, and the patient could lose vision in a period of a few hours! More common, though, is chronic angle closure glaucoma, in which the pressure is high but it has risen gradually, with slow closure of the angle, so the patient has no symptoms. Both forms are much more common in Asians, hyperopes and in older patients.

Most of the laser iridotomies you will hear about us doing are done as a result of closed angles we found incidentally on exam. If we don't do the PI, the patient will get glaucoma, if they don't have it already. The iris could scar to the trabecular meshwork (“peripheral anterior synechiae”) or they could get an acute closed angle glaucoma attack. Unpleasant prospects, all.

### Strategy in Treating Open Angle Glaucoma

As stated above, the strategy is to get the pressure of the eye down. As initial therapy, the patient can choose laser trabeculoplasty or medications. I usually try to encourage the laser trab, as initial therapy. Why?

- Drops are expensive.
- Drops are work.
- Drops are not effective in many patients. Many studies show that there is a very poor compliance rate in using the drops as prescribed. Also, many patients do not instill the drops in an effective manner, often missing their eye.
- Drops have documentable bad effects on the histology of the eye. Hence, they aggravate dry eye, make glaucoma surgery less likely to work if it is needed, and cause cataracts.
- Drops have frequent side-effects.

If you had glaucoma, would you rather have a 5-minute laser or a lifetime of expensive drops? Thought so. So why do most ophthalmologists use drops first and use laser as a last resort? I dunno.

### Laser Trabeculoplasty

This is an office procedure that is done at the slit lamp. It takes five minutes. Discomfort is minimal to none. There are no activity restrictions afterward. There are two kinds:

- Argon: Laser burns a 50 micron area with each shot. We don't know exactly how it works, but it makes the non-treated part of the trabecular meshwork work better. It was discovered as a glaucoma therapy back in the 1970s, when some researcher was trying to induce glaucoma in monkeys by doing this procedure. Instead of raising the pressure, it lowered it. A new therapy was born.
- Selective: This is a newer procedure. There are a few advantages over argon:

- It causes no anatomical damage to the trabecular meshwork. Therefore, it is safely repeatable, if the effect poops out after a couple years.
- No pain.

Both procedures work about 75% of the time. Sometimes, the effect is temporary, but the SLT can be repeated.

### Glaucoma Meds

There are several classes of glaucoma meds you should know something about. Only the beta blockers and the alpha-2 agonists cause systemic symptoms with much frequency. If you think to do it, ask the patient if they are taking their drops as prescribed—that way we can both harass them into compliance.

#### 1. Prostaglandin analogs:

- Lumigan (bimatoprost)
- Xalatan (latanoprost)
- Travatan (travoprost)
- Zioptan (tafluprost)

These are used only daily and are therefore convenient for the patient. These are used almost universally as first-choice drug therapy for glaucoma. They increase drainage of aqueous from the eye.

Side effects:

- Increased pigmentation of the iris and lids—especially latanoprost
- Red eye
- Atrophy of orbital fat/deep superior sulcus/enophthalmos—especially bimatoprost
- Long lashes (“Lumigan lashes”)—especially Lumigan

#### 2. Beta Blockers

- timolol (Timoptic, Betimol, Istalol)
- metipranolol (Optipranolol)
- carteolol
- levo-bunolol
- and as a component of Combigan

Beta blocker drops can sometimes cause systemic symptoms. Symptoms include shortness of breath, fatigue, depression, impotence and all the other side effects you might see with systemic beta blockers.

We will sometimes call you to see if we can put a patient on one safely.

Absolute Contraindications:

- Reactive lung disease
- Symptomatic heart block or bradycardia
- Decompensated CHF

Relative Contraindications:

Heart block or bradycardia, known cause, no symptoms

- Syncope of unknown etiology

Betaxolol (Betoptic) is a Beta-1 selective beta blocker and can be used safely in all but really unstable and fragile cardiac or pulmonary cases.

Beta blockers work by suppressing aqueous production. They are used qd or BID.

### 3. Carbonic Anhydrase Inhibitors

We almost only use topical now. They are sulfa analogs and so are relatively contraindicated in sulfa allergy. They decrease aqueous production. They are...

- Trusopt (dorzolamide) This tends to burn upon installation, because of the low pH.
- Azopt (brinzolamide) This is a suspension and so sometimes leave a white residue on the lashes, which sometimes bugs people.
- Cosopt is a combination of timolol and dorzolamide.

### 4. Alpha-2 Stimulators

That would be Alphagan (brimonidine). If these cause lightheadedness, they should be discontinued. It is common to get a dry mouth in the first couple weeks of therapy—just have the patient continue the drop, and this will go away. Generic brimonidine has a very high incidence of allergic reactions (red, itchy eyes). They both decrease aqueous production and increase drainage. They might be “neuro-protective” too.

5. There are others that are used so infrequently that they won't be listed here.

## Non-Laser Glaucoma Surgery

The standard glaucoma surgery is the trabeculectomy. We make a new drain, so that aqueous drains through a trap-door we create in the superior sclera. The flowing aqueous lifts the conj up off the sclera, creating a bleb. You will see this bleb if you lift the lid.

**Any red eye in a patient who has had a trab needs a referral to an ophthalmologist that day!** The bleb is thin and does not offer the barrier function of normal conj, and the drain itself is a potential conduit of bacteria into the eye. A conjunctivitis can easily spread to the interior of the eye, causing endophthalmitis and possible loss of the eye. Do not try to treat these yourself!

A combined procedure is a trab combined with cataract surgery.

A seton is an artificial drainage device in which a silicone tube is inserted into the anterior chamber, draining aqueous out on to a plate that is sewn on to the scleral surface. These are gaining popularity, as there are not the bleb problems associated with trabeculectomy.

## Your Glaucoma Questions, Answered



## **1. What is a “glaucoma suspect?”**

That can mean one of two things.

a. Ocular hypertension: If someone has intraocular pressures greater than about 21, we will typically see them once or twice per year.

- We want to be sure the IOP doesn't spike up really high, like 30 or above. If it does, we need to treat prophylactically to keep the pressure out of the 30's, because statistically, there is a very high probability of developing glaucoma with this high of IOP.
- Also, it is important for us to look at the optic nerve every year, so that if glaucoma occurs, we can start treatment.
- Finally, we want to see what the range of pressures is. The more the pressure fluctuates, the greater the risk of glaucoma damage to the nerve.

b. Suspicious cupping: We follow these once or twice per year. We monitor the patient to see what their range of pressures is and to be sure they are not spiking high pressures. Also, we monitor the visual fields, disc appearance and perhaps run one of the newer tests for imaging the optic nerve head or retinal nerve fiber layer (e.g.-OCT).

## **2. Should you be checking IOP's?**

No. If you do a random IOP on someone who has untreated glaucoma, there is a 50% chance that that IOP will be  $<22$ . Therefore, a normal IOP measurement doesn't rule out glaucoma. Also, the great majority of people with  $IOP > 22$  do not have glaucoma. Hence, if you do measure a pressure higher than 22, the probability is that that person doesn't have glaucoma. Pressure screenings are a poor way to screen for glaucoma—that's why you don't see anyone do them at health fairs anymore. Your job is to be sure your patient is up-to-date with their eye exams.

## **3. What is a “normal” pressure?**

Really, a normal pressure is that pressure that doesn't damage the optic nerve. For some, that would be 30. For others, it is 16. If we screen a patient's pressure to be  $>22$ , we follow them once or twice per year, to monitor for disc damage and for really high pressures.

## **4. What about systemic meds and glaucoma?**

- Anti-histamines: These can rarely raise the IOP. If someone is on an anti-histamine for a very long time, like months, we should check their IOP, if they have a history of ocular hypertension or glaucoma.
- Anti-cholinergics: These are of concern only if the patient has narrow angles, because they can cause mydriasis.
- Steroids: These can raise IOP. Patients on significant doses chronically should be monitored for high pressure and for cataract.

## **5. What is rubeosis and what do we do about it?**

Rubeosis is abnormal blood vessels growing on the iris and in the angle. These grow on to the trabecular meshwork, form a membrane which then contracts, closing the angle permanently. The vessels grow in response to ocular ischemia, caused by severe diabetic retinopathy, central retinal vein occlusion, poor carotid flow and other things. It is urgent we treat the ischemia

promptly if the angle is still open, before the angle zips shut. If it does, the patient usually needs a mechanical drain (“seton”) implanted into the eye to control the resultant glaucoma.

And a quick definition of terms you may hear:

- Gonioscopy: A contact lens we place on the eye to view the angle
- Pachymetry: Measurement of the corneal thickness. Thin corneas give falsely low IOP readings and thick ones give falsely high readings. We have to adjust the IOP for the corneal thickness. People with thin corneas are more glaucoma-prone.
- OCT: scan that measures the cup in the optic nerve and thickness of the retinal nerve fiber layer. The RNFL becomes thin in glaucoma. We use the OCT to monitor for disease progression and sometimes to help clinch the diagnosis.
- GDx: scan of the RNFL thickness
- HRT: measures cup

## CATARACT

A cataract is a haze in the crystalline lens. We see this mostly as an aging change, but they can be in patients younger than 60 too. Cataract formation correlates with general health, so the sicker someone is, the more likely they are to have cataracts. Cataracts correlate with other factors too, like (and especially) smoking and prolonged systemic steroids. Even steroid inhalers are a risk factor. In younger healthy patients, they are usually inherited. You hear about other meds causing cataracts, but these tend to be mostly visually insignificant ones. Serious ocular trauma almost always causes cataracts.

There are many types of cataracts. The main types are:

- Nuclear: This is primarily from aging. The patient is looking through an amber haze. Often, they cause a change in refractive error, a so-called “myopic shift.” These tend to be slowly-growing and don’t bother the patient as much as other cataract types.
- Posterior sub-capsular: Visually, this is like having ground glass on the back of the lens. These tend to really bother people with glare and haze. Driving at night is especially a problem. These are the type that a younger person would typically get. The Snellen visual acuity tends to be good in these, even if the cataract is severe.
- Cortical: Cortical cataracts are a white haze in the lens.

### Indications for Surgery

As stated above, the Snellen acuity, as measured on the chart, is a very poor indicator of how much a cataract is bothering a patient. In deciding to do surgery, we go almost entirely by how much the patient’s vision is bothering them. We just have to sound them out and get a feel for it. Some patients are genuinely bothered by relatively mild cataracts. It all depends on their visual requirements and standards. A younger, “with-it” patient will want surgery much sooner than an 80-year-old with dementia. Some types of cataracts tend to bother patients more than others too. A one millimeter posterior subcapsular cataract in the visual axis can be a real problem, even if the acuity is 20/20. The whole concept of the cataract being “ripe” or being “ready to remove” has been obsolete since the advent of modern small incision surgery. It’s not a matter of when the *cataract* is ready—it’s when the *patient* is ready!

Typically, patients describe their impaired vision as being “foggy or hazy.” They almost always say they get glare from headlights at night.

There are times that cataract surgery is medically necessary. If a patient has another pathology, like diabetic retinopathy or glaucoma, that requires that we view the fundus, the cataract must come out if we can’t see the fundus adequately. Also, if a cataract becomes “mature,” it really should come out to avoid acute lens-induced glaucoma; these will cause a white pupil.

One dilemma we encounter often as cataract surgeons is whether to remove a cataract in a patient with macular degeneration. Usually, we have to take an educated guess as to if the surgery will improve the vision enough to warrant the bother and risk.

A cataract severe (“mature”) enough to cause a white pupil should be removed, in most cases, because these can, through various mechanisms, cause acute glaucoma with a red, inflamed eye. If such a patient opts not to have surgery, they at a minimum need to be warned to get immediate attention for a red eye. Also, they need a B-scan ultrasound of the globe to be sure there is not a tumor behind the opaque lens.

### Technique

There are innumerable ways to do cataract surgery. The commonest technique for the standard case is as follows:

- Topical anesthesia: If we avoid a retro-bulbar lidocaine injection, we avoid the risk of puncturing something important with the needle, like the tissues of the orbit, the globe or the optic nerve. Unbelievably, four drops of tetracaine is all the anesthesia you need to do most cases.
- Incision is limbal or clear corneal, self-sealing, about 3.0 mm and temporal. This creates an incision that heals quickly and with no astigmatism. Patients can resume unlimited activity almost right away.
- Phacoemulsification is the ultrasound technique we use to remove the nucleus, the hard part of the cataract. Phaco has revolutionized cataract surgery, because we don’t have to remove the nucleus whole, through a large incision. We pulverize it with ultrasound and suck it up through a small incision. The guy who invented it, back in the 60’s, came up with the idea while having his teeth cleaned at the dentist’s.
- Foldable implants—To keep the incision small, we use foldable intraocular lenses. These are silicone or acrylic.

### Your Role in Cataract Surgery

Sometimes, a patient’s internist or family doc will insist that we postpone surgery because of an active medical problem. You want to be safe, but do keep in mind that cataract surgery is really minimal physiological stress on the body. We usually use topical anesthesia with minimal sedation, and the surgery usually lasts 10 to 15 minutes. We generally don’t have to stop blood thinners. You don’t want to be postponing the surgery unnecessarily.

Pre-op: Except in very unusual circumstances, we don’t need a pre-op check by you. We do a brief physical at the pre-op visit. There is no pre-op lab work either. Patients who need prophylactic antibiotics for surgical and dental procedures do not need them for cataract surgery—it is a sterile procedure that doesn’t put any bacteria into the bloodstream.

You can tell if a patient has an IOL by looking at the pupil with a flashlight. You can see the reflection from the lens’ surfaces.

### “Premium” IOLs

Refers to special IOLs that correct astigmatism (toric IOLs) or allow patients to see both distance and near from the same eye, un-aided by glasses (accommodating or multi-focal IOLs). Almost always, the patient must pay out-of-pocket for these.

# RETINA

## I. Flashes and Floaters

Flashes that come from the retina are always quick, split second and unformed. Flashes that do not fit these criteria are from something else, usually migraine (see neuro section). These quick flashes are from the vitreous pulling on the retina. The usual scenario is a posterior vitreous detachment.

Most people have some degree of floaters. The pathological floaters that concern us are either multiple, small floaters or a large floater. Either type needs to be seen within a couple days, if new. Pending ophthalmologic evaluation, have the patient avoid heavy physical activity, especially jarring (like in running), that would cause the vitreous to flop around. Having the vitreous move around increases the chance that a tear would extend into a retinal detachment.

Multiple, small floaters are often from a tear in the retina. They are from pigment from the outer layer of the retina moving into the vitreous through the tear. Tears need prompt attention, so that they don't evolve into a retinal detachment. We seal them off with either laser or cryo. Either procedure is a fairly simple office procedure involving mild-moderate discomfort. Such floaters can also be from vitreous hemorrhage, which in turn are usually from either a vitreous detachment or neovascularization of the retina from ischemic retinopathy (e.g.-diabetic, CRVO).

Large floaters are mostly from posterior vitreous detachment. This is a physiologic change in the vitreous in which the vitreous shrinks and collapses on itself. These usually occur in late middle age, earlier in myopic males. The vitreous is firmly attached anteriorly so it becomes detached from its connection to the posterior pole of the eye. The large floater is a shadow from the glial tissue that connected the posterior vitreous to the optic nerve. Often, as the vitreous shrinks, it tears the retina. Patients with vitreous detachment need to be seen upon onset of symptoms. Actually, tears can develop later too, so we advise our patients to report new floaters promptly.

Retinal Detachments are generally "rhegmatogenous," meaning that they are caused by tears in the retina. The patient comes to medical attention either because of increased floaters or a visual field defect. If you suspect RD, have the patient be sedate in their activities and get in to see an ophthalmologist within 24 hours. Generally, RDs have a good prognosis, but the prognosis is not as good if the macula is detached or if the detachment is long-standing. Especially prone to RD are middle-aged myopic males, high myopes of any ilk, status-post complicated cataract surgery and status-post severe ocular trauma (e.g., after traumatic hyphema).

## II. Vascular Diseases of the Retina

Here are the main ones.

A. Diabetic Retinopathy: The main thing to remember here is to remind your diabetics to get annual eye exams. Also, you should remind patients that keeping blood sugars, blood pressure and lipids under control minimizes the risk of retinopathy. Smoking dramatically increases the risk of disease. Of course, you already knew all that. It's nice, but not critical, to be able to recognize the retinopathy yourself. Look just temporal to the macula for the first hemorrhages. If you want to understand diabetic retinopathy, there is lots of information out there for primary care folks, and I am not going to re-hash it in detail. Here is some quick information:

The basic pathophysiology is damage to the endothelium of the retinal blood vessels. This causes two problems—ischemia and leaky vessels.

1. Leaky Vessels: There is a blood-retinal barrier, just as there is a blood-brain barrier. Diabetes can cause damage to this aspect of the retinal arterioles, allowing them to leak large molecules. Lipid accumulates in the macula and blocks the transmission of light and the fovea becomes swollen (macular edema). This not only impairs vision while it exists, but if left to stand can damage the retina permanently.

If you see lipid exudate near the fovea, you know the patient is in danger of losing vision per above. We can actually see macular edema with our lenses at the slit lamp, but you would have trouble seeing it with a direct ophthalmoscope. Another finding of incompetent vessels is dot and blot hemorrhages in the posterior pole.

Treatment: If the leaking is focal, we can shut down the leaking vessels with “focal laser.” This sometimes causes scotomas in the patient's vision.

If more diffuse, we have several options. Grid laser is the standard therapy. Several studies are now being done to see if this is still the best first-line treatment. Anti-VEGF drugs, injected intravitreally, are being used more for diabetic macular edema. These include Avastin (bevacizumab), Lucentis (ranibizumab) and Eyelea (aflibercept). If we get desperate, we can do a vitrectomy. We don't quite understand why the vitrectomy works.

2. Ischemia: The retinal capillary network gets areas of dropout. You can't see this directly, but it is visible with a fluorescein angiogram. Bad ischemia (“severe non-proliferative background diabetic retinopathy”) on exam looks like blot hemorrhages, cotton-wool spots, intra-retinal microvascular anomalies (fine, wiggly vessels on the retinal surface) and venous beading. Really bad ischemia looks like neovascularization of the disc or retina (“proliferative diabetic retinopathy”). So that ischemia doesn't induce neo or to treat existing neo, we do a laser treatment, Pan-Retinal Photocoagulation (PRP). The idea is that ischemic retina produces bad chemicals, including VEGF, that induce growth of new vessels. Unfortunately, these vessels do nothing to supply blood to the retina—they just cause trouble. They grow onto the vitreous causing the vitreous to shrink and tear the attached vessels, causing vitreous hemorrhage. Then, they can become fibrosed and shrink, causing traction retinal detachment. New vessels can grow into the anterior chamber angle (rubeosis), causing glaucoma of a very nasty variety (neovascular glaucoma). With PRP you place 1000-3000 laser spots in the mid-peripheral retina. Everywhere the laser hits, ischemic retina becomes infarcted retina. Infarcted retina does not scream for oxygen, and so does not produce the chemicals that cause neo (such as VEGF—vascular

endothelial growth factor). You try to kill off enough retinal cells so that there is adequate blood flow for the remaining untreated retina. The procedure itself does sacrifice some peripheral and night vision, but it usually is very effective.

Anti-VEGF drugs are now being used for neovascularization.

B. Branch Retinal Vein Occlusion: These are thrombotic in etiology. The retinal arteriole and venule are wound up together in a figure-8 connective tissue sheath. When the arteriole becomes sclerotic, it takes up more than its fair share of the space, constricting the vein. We counsel these patients on atherosclerosis prevention and get a blood pressure, lipid profile, etc. You need to remember that patients with this condition have atherosclerosis and to proceed accordingly.

Patients get rapid onset of blurred vision. In the fundus, you see a sector of blot, dot and flame hemorrhages and a dilated venule in the affected area. Most patients with BRVO get anti-VEGF injections to prevent or treat macular edema.

C. Central Retinal Vein Occlusion: These are usually thrombotic, as per above. In younger patients or if repeat episodes, we may refer them to you to rule-out a hypercoagulable state (although, the cause in younger patients is usually an anatomical vascular anomaly).

You see the above findings, involving all quadrants of the retina. These can cause serious retinal ischemia with subsequent neovascular glaucoma. They need to be monitored by the ophthalmologist every month for the first six months. They often get anti-VEGF injections to prevent or treat neo.

D. Branch Retinal Artery Occlusion: These are almost always embolic. We need to get a carotid duplex and an echocardiogram, and check for the factors predisposing to atherosclerosis. You may get involved in the work-up.

The history is sudden onset of a visual field defect—either superior or inferior. The affected retina, acutely, is white. The affected arteriole is threaded and narrow. Sometimes one can see the embolus which helps find the source. The visual deficit is permanent, and there is no treatment.

E. Central Retinal Artery Occlusion: The history is sudden onset of blindness in one eye. With that history, rule-out temporal arteritis immediately!! (Stat ESR, CRP, and platelets, followed by immediate oral prednisone). The other stat issue is that a recent study showed that if the patient is given IV TPA within 6.5 hours of onset there can be some recovery of vision. The stroke team at one of our regional hospitals has a protocol on this. TPA treatment has some big risks, as you know, and it is not considered standard-of-care. We would use TPA, if the CRAO occurred in the patient's only-seeing eye. We need to be sure that we have taken steps to address the underlying embolic source or temporal arteritis.

Exam findings are the same as BRAO, but the entire retina is involved.

### III. Cholesterol Embolus



If symptomatic, these will cause a BRAO or CRAO. Usually, they are asymptomatic and we discover them on exam. They require a work-up to find the source. Cholesterol emboli are yellowish and shiny and wedge themselves in the fork in an arteriole. They often don't block the blood flow, because they tend to be disc-shaped and get wedged in like a valve in the open position. Calcium emboli are white. They usually come from the heart valves. These are plug-shaped and do block the blood-flow distally. There are fibrin-platelet emboli too, but you will rarely actually see these because they break-up very quickly. They can be the cause of an artery occlusion.

#### IV. Macular Degeneration

This is a degenerative condition of the macula. Progression is not inevitable, and there is a huge spectrum of clinical courses, from a dense central scotoma in each eye to no visual impairment at all. In advanced cases the patient cannot drive or read, but peripheral vision is always retained. The etiology is multi-factorial, but there is a definite inherited component. The risk factors for atherosclerosis are risk factors for macular degeneration too. A few issues for you:

Nutritional supplementation may help prevent progression. A big study (Age Related Eye Disease Study, or AREDS) showed that, in patients with existing moderate macular degeneration, a particular regimen of vitamins A,C,E and zinc were somewhat protective against progression. A second go of the study (cryptically called "AREDS 2") is being done to see if other supplements, notably lutein and omega 3 fatty acids, also help. The question is, how much, which supplements and in what patients? These are questions being researched intensively. I am including the hand-out I give to patients on prevention. I am recommending supplements that have the AREDS amounts of C, E and zinc, and also lutein. Don't forget that vitamin A increases the risk of lung cancer in smokers. Lutein and vitamin A compete for the same receptor site, so that's why you usually don't see both in the same supplement.

The same recommendations you would give to someone with atherosclerosis are good for macular degeneration too. Low fat (or really, good fat) diet, omega-3's, etc. Dark green leafy vegetables, daily fruit, and fish could be useful too—all have shown some benefit in small or limited studies.

This is one more reason to quit smoking! Would you rather be blind or dead? Blind is a big motivator.

We have to be vigilant about driving in these folks. Be sure the patient is getting eye exams. Patients often avoid seeing us because they know that their license could be yanked if their acuity drops enough (see section on driving requirements). We can have the patient qualify for a special license when they see 20/80 or 20/100, that enables them to drive only very locally, during the daytime and not on highways. Eye doctors are legally obliged to report patients to the state who don't see at least 20/70.

We have many ways to help macular degeneration patients use the vision they have left. If the patient has trouble reading, a consult with a low vision specialist may be useful. They can work with the patient with various magnifiers, lighting techniques, and other devices. If your patient is having trouble with these things, you may want to suggest a low vision consult. One simple

solution is simply to recommend use of a Kindle or iPad to read on—the print size can be adjusted.

“Wet” macular degeneration occurs when a neovascular membrane grows into the sub-retinal space from the choriocapillaris. Left untreated, these usually lead to large macular scars. We ask macular degeneration patients to watch for distorted or blurred central vision and to report it promptly. We often have the patient monitor for this daily by looking at an Amsler grid. The main thing that you need to remember is that macular degeneration patients need to see the ophthalmologist within a couple days if they notice these changes. (Here is a good test for your ophthalmologist—if he doesn’t get the patient in within 3 days, he flunks). We have new treatments for wet macular degeneration now, so it is more important than ever to get people in promptly.

Treatments for wet macular degeneration include intraocular injections of Lucentis, Avastin, or Eyelea, which are anti-vascular endothelial growth factor meds. These are new but seem to be working. Many injections are required to involute and prevent recurrence of the membrane. Laser has been supplanted by anti-VEGF injections. Surgeries such as macular transposition and removal of the neovascular membrane have been flops.

## Nutrition and Prevention of Macular Degeneration

### Proven to Help

Vitamin C: 500 mg/day Vitamin E: 400 mg/day Zinc: 80 mg/day (must take copper too)( more is probably better)
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We are recommending \_\_\_\_\_

Other doses of these vitamins and zinc may be beneficial. We still don't know the optimum dose on any supplements listed here.

Selenium may also be important.

Don't take Vitamin A/Beta carotene if you are a smoker!! It increases your risk of lung cancer.

If you are on coumadin or warfarin, ask your doctor before starting vitamins. They can affect how the coumadin works.

If you are taking other vitamins, be sure you are not getting too much. Too many vitamins can be toxic.

Also proven to help is a low-fat diet. Ask your doctor if you need details. Generally, the same things that prevent hardening of the arteries and heart attacks prevent macular degeneration. Keep in mind that some fats are probably actually helpful—these are the fats found in tree nuts (except cashews and Brazil nuts) and in fish (especially salmon, albacore tuna, halibut, sardines, herring and cod).

## V. Macular Holes

These are caused by vitreous traction on the fovea. They cause a central scotoma in the patient's vision, although usually the patient is aware of only blurred vision. We can repair these by doing a vitrectomy. The surgery is more effective if done within a few months of onset. The patient must assume a head-down position for several days after surgery, because a gas bubble is left in the eye to put pressure on the fovea to squish it down to the rest of the retina.

## VI. Epiretinal membrane, macular pucker, cellophane maculopathy

These are all the same thing, basically. They are usually idiopathic, but can result from pathology such as retinal tears, laser or trauma. We fix them if the acuity is badly affected. The surgery involves doing a vitrectomy and stripping the membrane.

## VII. Drug Toxicity to the Retina

There are numerous meds that can cause toxicity. The one we fret most often about is hydroxychloroquine. The risk of toxicity is proportional to the lifetime dose. Toxicity is rare, and usually requires about ten years of drug use before it occurs. Monitoring for toxicity is necessary. This involves a baseline exam at onset and then an annual exam every year, starting at year five. Each exam must include a 10-2 Humphrey visual field. Patients should report blurred vision to their ophthalmologist promptly.

Many other drugs can cause retinal toxicity (not a comprehensive list): tamoxifen, digoxin, amiodarone, isotretinoin, various chemo meds, erectile dysfunction drugs, peg interferon, fingolimod, glipizides. Peg interferon patients should have a baseline complete eye exam and then have a dilated exam frequently. Fingolimod patients should have a baseline exam and another exam at three months.

## PEDIATRICS

I don't see very many children, which is why you will be spending time with our pediatric ophthalmologists. Here is some really basic stuff.

Ideally, children should have an eye exam with a cycloplegic refraction at age 3-4. The cycloplegic refraction is good because we can unearth high hyperopia, which can cause reading difficulties when they get into school. Kids are screened in pre-school and by you or the pediatrician for visual acuity and muscle imbalances, and the screenings will pick up a problem most of the time.

When measuring the acuity on a child, be sure the non-tested eye is completely covered, including the space between the cover and the bridge of the nose. Kids like to peek around the cover so that they don't "fail" the test.

### I. Amblyopia

Amblyopia is poor vision occurring when the neurological connection between an eye and the occipital cortex doesn't develop properly. The two usual causes are strabismus and severe anisometropia (asymmetry between the eyes in refractive error). (Usually, the refractive error is hyperopia, rather than myopia, because in myopia the eye is in focus at near, so it gets some clear viewing during the day.) Also, if one eye is occluded for any length of time, it will become amblyopic. That is why cataract surgery has such urgency in babies.

Besides treating the underlying cause, the treatment is to force the patient to use the bad eye. Usually, we do this with patching the good eye. Until a few years ago, we used to institute full-time patching in almost everyone. Turns out, this is excessive, and the pediatrics people are trying to figure out the minimum effective patching regimens for amblyopia of various types, severities and ages of child. Patching is problematic for a few reasons. First, when there is severe amblyopia, the child will initially be very blurred when the good eye is covered, so he will tend to fight the patch. Second, the patch looks funny, which is a problem especially in school. Third, patches are not entirely comfortable, especially in the summer.

Now, a lot of ophthalmologists are going with pharmacologic occlusion. We have the parent give daily atropine drops in the good eye to blur it. This works except in severe amblyopia. Two caveats:

1. The child should wear sunglasses when in the sunlight.
2. The parent should be vigilant about not letting the patient or any other small child have access to the atropine. Atropine is lethal when consumed in not-too-great quantities by a child.

With anisometric amblyopia, we often are not able to get the acuity to a perfect 20/20.

If any amblyopia or strabismus occurs during childhood, the development of the visual system of the brain will usually be compromised permanently. Primarily, the effect is in stereopsis. Those with severe or long-standing strabismus or amblyopia generally are the most affected. Most

people learn to compensate for their deficiencies of this sort, but ability to perform certain occupations could be eliminated (eye surgery comes to mind).

Another hot topic is the age beyond which treatment is ineffective. Until recently, we would not treat anyone for amblyopia over the age of 9, because we thought the visual system was set in stone at that point. We were wrong, and now we don't know what the limits are.

With strabismic amblyopia, you have to correct the amblyopia before you do strabismus surgery. That is because once you do the surgery, there has to be a lock-in mechanism in place to keep the eyes straight and to fine-tune the muscle balance. If you don't have that, the eyes will drift again.

Yes, it's possible to have "bilateral amblyopia." The usual scenario is severe astigmatism or high hyperopia that is roughly the same in each eye. Any disorder that interrupts good vision during childhood can do it. If advanced or chronic, it is usually not reversible to 20/20.

## II. Strabismus

Turned eyes are a concern for many reasons. First, if the child chooses one eye to turn and uses the other eye exclusively, the turned eye will become amblyopic. Second, a turned eye could be an eye with pathology (e.g. cataract, tumor). Third, rarely, a turn can indicate an underlying neurological problem. Fourth, strabismus looks funny, and like it or not, some people react negatively to someone with an obvious abnormality, especially if they happen to be seven years old.

Recent onset of a turn in a child is cause for immediate concern. The child should see an ophthalmologist that week, preferably that day.

There are many different types of strabismus (or, "squint"—where did they ever get that term?). Here are the really common ones:

A. Congenital Esotropia: "Congenital" should be in quotes, because the turn often starts a few weeks after birth. The turn is very large—usually about 45 prism diopters. These should be repaired in the first year of life, usually, so that we can optimize the patient's stereopsis. There is a definite amblyopia risk, especially if the child doesn't alternate between a left and a right eye turn. Differential diagnosis: poor vision in the turned eye.

B. Accommodative Esotropia: This usually starts around age 2, although this is quite variable. The turn is of moderate magnitude and is intermittent, at least at first.

There are two basic scenarios. The first is when there is high hyperopia. As you know, the near reflex involves convergence, along with accommodation and miosis. Hyperopes have to use the near reflex to see even for distance, and they really have to kick it in for near. Some hyperopes' brains learn to adjust the tension on the extraocular muscles so that they can hyper-accommodate but keep the eyes straight. Others do not, and they get an esotropia. The ET is the same at distance as at near. The treatment is full-time glasses, correcting the hyperopia.

The second is when there is a high AC/A ratio. Here, the amplitude of convergence is exaggerated disproportionately to the amplitude of accommodation. The treatment here is bifocals. Bifocals in a child have to be made and fit properly. In a younger child you need a flat-top bifocal. The top of the add must bisect the pupil, so the child is almost forced to look through the add for near. If your patient's bifocals are not fitting this way, refer the child back to the optician for an adjustment.

Children with either type of accommodative ET can develop non-accommodative ET later on. They need to be monitored regularly. Be sure to ask if this is happening.

C. Intermittent Exotropia: This usually starts in toddlerhood. It usually starts out as only occasional, then increases in frequency. If it happens more than about once per day, it could impair the optimal development of binocular vision. Surgery would then be indicated. Always refer these—you need to rule-out monocular blurred vision.

Cover Tests: These are way easier to show than to explain coherently in words. I was considering doing drawings or ripping off pictures from a text, but the pictures are always confusing. My lame attempt is as follows:

Tropia: a constant turn

Phoria: the tendency to turn, but the patient can fuse the eyes straight when using the two eyes together. What keeps the eyes straight in phoria is that when one eye starts to turn in, out or up, the brain receives a different image from each eye. The brain doesn't like that, so it readjusts the efferent impulse to each extra-ocular muscle, straightening them out. So, the eyes are straight when they can work together with both eyes open, but one eye turns, when the eyes aren't viewing at the same time, for example when one eye is covered.

Intermittent tropia: The patient has a phoria. Sometimes they have enough strength in the visual system to pull the eyes in straight, sometimes they don't. When they don't, there is a turn.

Cover-Uncover Test: This will unearth a tropia. Cover OD and see if OS moves. Then, uncover, cover OS and see if OD moves. If you cover OD and OS moves, OS was turned.

Cross-cover Test: This will unearth a tropia or phoria. Have the patient fixate an object. Cover OD then OS then OD then OS. You will see eye movement each time you switch the cover to the other eye. Remember, if there is a phoria, when you cover an eye, it will drift in, out, up or down under the cover. If you want to know if it's a tropia or a phoria, do the cover-uncover test.

### III. Cataract Surgery in Children

Cataracts are pretty uncommon in children. That is fortunate, because it is not a quick, simple fix as it usually is adults. The surgery is urgent, as we mentioned, because of the risk of amblyopia. Often, a unilateral cataract will present as an esotropia, so recent-onset turns need a prompt exam.

These are often complicated surgeries. The child's eye doesn't react to surgery at all like an adult's, so this isn't a job for your friendly neighborhood cataract surgeon. A fellowship-trained pediatric ophthalmologist is de rigueur.

Since we remove the child's lens, we need to replace it with something. Contact lenses are the usual choice, but these are a big problem in babies and toddlers. They like to fall out and are hard to get in and out of a small eye. They are a lot of work. Increasingly, intraocular lenses are being used in pediatric cataract surgeries. The eyes are tolerating them well, but choosing the correct power is a problem, because the eye gets longer with age, changing the refractive error.

We have to monitor frequently for glaucoma, which is common after pediatric cataract surgery.

A systemic work-up is required in congenital cataracts. TORCH titers, a calcium level and a work-up for galactosemia are necessary.

#### IV. Tearing

Most of the time, congenital epiphora is from an anatomic obstruction at the ostium of the nasolacrimal duct, where it enters the nose under the inferior meatus. The obstruction is membranous, so we can cure it by popping the membrane with a lacrimal probe. We do this under general anesthesia, because it would be very painful. If a simple probe doesn't work, we go back in and re-probe, this time leaving the naso-lacrimal system intubated with a silicone tube, that we leave in for several weeks. Also, it sometimes helps to in-fracture the inferior turbinate. Epiphora from lacrimal obstruction is always accompanied by a mucopurulent discharge. Children with purely clear tearing need to be worked-up for glaucoma!



## TRAUMA

Again, I have chosen conditions that are either very common or serious. You will refer the vast majority of trauma, so we don't need to discuss it much.

### 1. Corneal Abrasions

These are pretty common. I recommend that these be referred, unless they are very mild and the traumatic etiology is clearcut. Often, there is a patch of loose epithelium that should be debrided, so that the loose cells don't rub on the bare nerve endings of the cornea every time the patient blinks. Sometimes that "abrasion" is actually an ulcer.

If the patient is only mildly uncomfortable, put them on an antibiotic drop QID, and tell them to stop it 3-4 days later. They should see an ophthalmologist, if they don't experience steady improvement.

Unbelievably, I saw yesterday a patient with an abrasion referred from an emergency department who was given a bottle of proparacaine to use for comfort. No, no, no, no, no! Repeated use of topical anesthetics will first prohibit healing and then melt the cornea.

I usually don't patch any but the most painful of abrasions. Patching is purely for comfort and does not make healing go any faster. If you patch an abrasion, do it the right way—a tight pressure patch that forces the lids closed. Don't forget to put in antibiotic ointment *before* you put the patch on—one of my personal favorite errors. **Never patch a contact lens wearer!** You're begging for an infection.

### 2. Chemical burns

This is common sense, but be sure the patient flushes the eye out immediately and thoroughly with water, getting up under the lids. In the case of strong acids, and especially with strong bases (lye, lime, ammonia), the eye must be flushed for several minutes before heading out to the ophthalmologist or the ED. For chemicals other than strong acids and bases and other really caustic fluids, the urgency in referring depends on the patient's vision and level of discomfort. Your ophthalmologist can help you triage these.

Strong bases can destroy an eye. The cornea becomes opacified and sometimes melts. They almost never can be successfully be transplanted. Usually, admission to the hospital is required.

### 3. Ultraviolet burns

Specifically, welder's flash. You can treat these yourself. The typical history is unprotected exposure to welding earlier in the day. Even a few seconds can do it. Hours later, the patient develops bilateral pain. Treat with analgesics and topical antibiotics. They will get better fast.

### 4. Blunt Trauma

In the office these are typically from sports injuries. Baseballs, tennis, racquet, squash balls, and hockey pucks are all common. The ones from the ED are usually assaults with a fist. These should all be referred. It is common for blunt trauma injuries to result in hyphema. There are many other injuries we need to watch for too.

Hyphema is blood in the anterior chamber. When you see a blunt trauma, look very carefully at the inferior part of the anterior chamber—you will often see a little blood. Sometimes, the hyphema is so small that it doesn't layer and is only visible in the slit lamp as red cells floating in the anterior chamber ("microscopic hyphema").

The issues with hyphemas are re-bleeds and glaucoma. In the first five days after the injury, there is a risk that the vessel that ruptured at injury will re-rupture. When this happens, the recurrent hemorrhage is often much worse than the original one, sometimes filling the entire anterior chamber with blood. This would be considered bad. Big hyphemas usually cause the intraocular pressure to rise to very high levels and so often have to be evacuated surgically (which almost always causes a cataract). Therefore, we want to avoid re-bleeds. We have the patient be a couch potato for the five-day risk period, staying home (except to see the eye doctor) and watching TV, playing video games and reading. We used to admit all hyphemas to the hospital for enforced bedrest, but this has been shown to be unnecessary.

Patients who have had traumatic hyphemas have an increased chance of developing glaucoma sometime during their life. They need an annual eye exam. Please remind them.

There are many other bad effects from blunt trauma, including ruptured globe, orbital fracture, and retinal damage. All cases must see an ophthalmologist.

5. Foreign Bodies Always ask if the patient was wearing glasses or goggles at the time of injury. A high-velocity foreign body in a patient without eye protection must be seen by an ophthalmologist right away to rule-out an intraocular foreign body. If you have a strong index of suspicion, get them seen immediately, tell them not to rub the eye, and make them NPO in case we have to remove it surgically. The commonest scenarios for an intra-ocular foreign body are striking metal on metal and using powered lawn equipment (e.g., weed-whacker).

A. Corneal, non-penetrating: I would refer the majority of these. The only exception would be a very superficial non-ferrous foreign body that you are sure that you removed in its entirety. Iron-containing foreign bodies usually leave a rust deposit in the cornea that needs to be removed with a burr at the slit lamp. If you don't remove the rust, it won't heal and has a strong chance of infection.

B. Penetrating/perforating: There is a difference between these terms, but don't clog up your brain with it. Proceed as above.

## 6. Ruptured Globe

Almost certainly, if the trauma was of the nature to make you wonder if the globe was penetrated or ruptured, you will be referring. Just so you know, here are some signs of a ruptured globe:

- Irregular pupil
- Chemosis
- Very deep or very shallow anterior chamber
- Hypotony

When in doubt, make them NPO put on a shield (you can make one from a paper cup) and get them to an ophthalmologist. Don't put any drops in the eye.

## 7. Lid Lacerations

You usually refer these to a plastic surgeon, but some ophthalmologists repair them too.

## 8. Prevention

The obvious thing here is appropriate eye protection for a given activity.

Racket sports, especially racquetball and really especially squash, require specialized eyewear. Mere glasses are inadequate. I don't think lens-less frames are still being made, but they are bad too.

Hockey requires special eyewear.

The players of many other sports don't traditionally wear protective eyewear, but probably they should at least wear unbreakable lenses in a sturdy frame. Tennis and baseball injuries are definitely not rare.

Every time I am driving down the road and see someone using a weed-whacker without eye protection, I want to yell, *Wear your eye protection, you moron!* Some day, maybe I will.

Mowing the lawn requires glasses.

All carpentry requires eyewear at all times. I have seen nails fly back and penetrate the globe. Everyone knows to wear protection for power saws, etc. but we see a lot of foreign bodies in people who were "just about done but just forgot to do this one last cut and nothing is going to happen because it's just one last little second." Right.

If you are in the same room as carpentry being done, you need eye protection too. I saw a six year-old girl who had a penetrating injury from a nail that had flown across the room from her father's hammering.

Extreme caution is important in handling strong bases, such as lye, lime and ammonia. There are some acids that are as bad as strong bases and require the same handling.

Caution is required in using jumper cables—batteries can explode. 2M sulfuric acid isn't as bad a 9M base, but it's still nasty.

## IN CONCLUSION

If you want a more detailed publication (that is, a real book), I recommend *The Physicians Guide to Eye Care* or *Basic Ophthalmology*, published through the American Academy of Ophthalmology. Those are more comprehensive texts. I wrote this manual knowing that family practice and internal medicine residents really would not spend time reading a 200+ page book, but would probably read a 50-page manual.