

## FAQs Frequently asked Questions

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## AMD

### *Age-related Macular Degeneration*

#### Lucentis v. Avastin

Most authorities find Avastin pharmacologically equivalent to Lucentis, the more highly investigated agent. Both agents have a similar pharmacologic profile and dosing schedule. For practical purposes, Avastin can be considered the parent molecule – Lucentis was developed as a modified subunit. Because a single dose of Lucentis costs \$2000, most retinal surgeons will not keep stock supplies of Lucentis. (Avastin is 1/40<sup>th</sup> the price). The federally funded CATT study, to compare the efficacy and safety of these agents, is underway.

See *Common Retinal Conditions, Age-Related Macular Degeneration*

I have macular degeneration – Will it get worse?

Macular degeneration affects the macula alone – not the paracentral or peripheral retina. Macular degeneration, whether age-related, myopic or related to angioid streaks or Histoplasmosis, can result in complete loss of central vision – a dark central blind-spot – but does not spread to the adjacent retina. Individuals with macular degeneration walk and ambulate without assistance, but may lose the ability to read, discriminate faces and see colors. But the “lights” don’t go out. Anti-angiogenesis agents may stabilize or improve forms of exudative (wet) age-related macular degeneration.

See *Common Retinal Conditions, Age-Related Macular Degeneration*

My doctor says I have macular degeneration but neither laser nor injections will help.

Age-related macular degeneration (AMD) is classified into two categories: wet or dry. Injectable anti-angiogenesis agents or laser photocoagulation are indicated for the wet forms only. To date, there is no interventional treatment for dry AMD. With progression of disease, wet forms culminate into a central fibrovascular scar and dry forms leave an atrophic scar. No form of medical or surgery therapy is indicated for the end-stage condition.

See *Common Retinal Conditions, Age-Related Macular Degeneration*

My mother had macular degeneration. Should I be checked?

The chief predictor of macular degeneration lies in our genetic profile, particularly in the genes controlling inflammation known as the alternate complement pathway. Environmental exposure to ultraviolet light, tobacco and our consumption of anti-oxidants and omega-3s may modify the severity and progression of degeneration.

## CATARACT SURGERY

I had cataract surgery, but I don't see any better?

Good visual acuity requires clarity of the ocular media (cornea, lens, and ocular fluids), good focus, a healthy retina and optic nerve, as well as processing in the central nervous system. A dense cataract may obscure and therefore preclude an adequate ophthalmic evaluation of the macular region. Subtle retinal abnormalities, including macular pucker, macular drusen, macular hole and glaucomatous atrophy might not be recognized before cataract surgery if the presence of a dense cataract.

One to two percent of patients with uneventful cataract surgery experience persistent macular edema – *cystoid macular edema*. Transient minor retinal edema is common and usually resolves in 6-12 weeks. Persistent edema will usually abate within 6 months of the procedure. This post-operative rate is considerably high when there is disruption of the anterior vitreous face and insertion of an anterior chamber lens. Cataract surgery may fail to rehabilitate vision when the new introduced lens is not well situated, if the cornea becomes cloudy, or if the intraocular lens implant irritates the eye.

See, *Eye Surgery and Potential Complications*

## DIABETES

My blood sugar has been well controlled, but my eyes are getting worse.

The general trend and expectation for diabetic retinopathy is slow progression. Laser and pharmacologic therapy may slow and even reverse some alterations, but most patients will experience greater retinopathy over the course of 10-20 years of disease. Laser photocoagulation remains the standard therapy for both background diabetic disease with macular edema and for proliferative diabetic retinopathy.

See, *Common Retinal Conditions, Diabetic Retinopathy*

I have diabetic retinopathy, but my doctor says laser won't help?

Retinal therapy is individualized to disease process and location. Laser is used to treat retinal swelling and the growth of new blood vessels. But other disturbances, such as retinal ischemia, vitreous hemorrhage, and traction on the inner retina may interfere with vision. Laser photocoagulation cannot restore the microcirculation, alleviate traction or break-up vitreous hemorrhage.

See, *Common Retinal Conditions, Diabetic Retinopathy*

How many times can an eye undergo laser for diabetic retinopathy?

Probably six times of laser, but possibly more. In macular edema, too much focal laser to the central areas carries the risk of destroying the remnants of the capillary circulation, which can cause ischemia to the remaining healthy tissue. A good rule of thumb is to treat macular edema no more than three times with laser in the same location. Laser for proliferative retinopathy is often divided into two or three sessions for patient comfort.

See, *Eye Surgery and Potential Complications*

## EYEGLASSES

How often do I need an eye examination?

There is no standard rule. It depends on symptoms, the presence of systemic disorders and family history. Diabetic patients should have a complete examination yearly. Individuals with a family history of glaucoma should be re-evaluated every one-two years after they reach age 40.

Why do I need reading glasses or bifocals?

As the eye ages, the lens within loses its elasticity and deformability and so the mechanism for near focus, accommodation, loses its effectiveness. The process is gradual, but most will experience difficulty in their 40s. Near sighted individuals (myopias) may find it easier to read without spectacles or contact lenses.

Why don't glasses make me see well?

Glasses and contact lenses place the viewed object in focus, but can't aid vision in the presence of media opacity or retinal disease. Media opacity is an obstruction or obscuration of light rays as they travel through the cornea, lens and fluid compartments of the eye. As a camera with damaged film fails to provide a quality photograph, an eye with a damaged retina cannot see clearly.

## FLOATERS

What is a floater?

Floaters are clumps of protein in the vitreous cavity of the eye that cast a shadow on the retina, as they float through the space. Everyone has floaters. Most are spots or squiggles that drift through the field of vision, and are best seen when looking at a monochromatic background like a white sheet of paper or a blue sky.

The sudden onset of floaters may herald the onset of a retinal tear or detachment, especially if accompanied by flashes of light and or a dark shadow or curtain over the field of vision. Floater may be the first symptom of bleeding inside the eye.

See *Common Retinal Conditions, Floaters*

What is a posterior vitreous detachment (PVD)

Separation of the vitreous from the posterior retina occurs spontaneously in seniors, high myopias and operated eyes. Symptoms include new floaters and flashes. Although usually without additional problems or complications, a PVD can cause a retinal tear (and retinal detachment) or lead to macular pucker. Most patients experience floaters for several months before they slowly abate. Rarely, a PVD can cause vitreous hemorrhage without a retinal tear.

See *Common Retinal Conditions,*

## RETINAL DETACHMENT

My doctor says my retinal detachment was repaired, but I still can't see.

The retina's rods and cones (the light sensitive cells) align precisely with their underlying retinal pigment epithelial (RPE) cells. Nowhere is this relationship more important than in the macular region. With separation of the retina from the choroid (macular off detachment) there is a possibility that the retina may not find its way back to the precise orientation had before the detachment. Other causes include retina edema and epiretinal membrane (macular pucker) formation. With macular off detachment, few patients will achieve reading vision (20/40) in the operated eye.

Retinal detachment, See *Common Retinal Conditions*

## TECHNOLOGY

Gene transfer

Results of world's first gene therapy for inherited blindness show sight improvement. 28 April 2008. UK researchers from the UCL Institute of Ophthalmology and Moorfields Eye Hospital NIHR Biomedical Research Centre have announced results from the world's first clinical trial to test a revolutionary gene therapy treatment for a type of inherited blindness. The results, published today in the New England Journal of Medicine, show that the experimental treatment is safe and can improve sight. The findings are a landmark for gene therapy technology and could have a significant impact on future treatments for eye disease. Read [Press Release](#).

See, Gene therapy, Genomics.energy.gov

## Stem cells

Newly published research, by investigators, at the North East England Stem Cell Institute (NESCI) in the journal [STEM CELLS](#) reported the first successful treatment of eight patients with "Limbal Stem Cell Deficiency" (LSCD) using the patients' own stem cells without the need of suppressing their immunity. To date, stem cells have not been used to rehabilitate diseased or damaged anterior segment or retinal tissues.

## Visual prosthesis (Artificial Eye): Can I get a retinal transplant?

There is no useful retinal substitute nor can the retina regenerate. The technical challenge is to develop a substantial artificial sensory array that connects to the optic nerve or sensory substrate of the brain. Research to build a retinal chip or sensor to replace a damaged or diseased retina is underway at several centers testing sensory microchips implanted in the retina or external cameras with electrodes stimulation the optic nerve. The first generation of implants restored rudimentary acuity in patients with retinitis pigmentosa (RP). A federally funded study to evaluate the next generation implant with 60 individual electrode sensors is underway:

Sylmar, Calif., January 9, 2007 – A new implantable technology has the potential to bring light back to blind individuals with Retinitis Pigmentosa (RP). Second Sight<sup>®</sup> Medical Products, Inc., announced today that the U.S. Food and Drug Administration (FDA) has approved an Investigational Device Exemption (IDE) to conduct a clinical study of the Argus<sup>™</sup> II Retinal Prosthesis System at centers of excellence across the United States.

The Argus II is the second generation of an electronic retinal implant designed for the treatment of blindness due to RP, a group of inherited eye diseases that affect the retina. RP causes the degeneration of photoreceptor cells in the retina, which capture and process light helping individuals to see. As these cells degenerate, patients experience progressive vision loss.

The Argus II implant consists of an array of electrodes that are attached to the retina and used in conjunction with an external camera and video processing system to provide a rudimentary form of sight to implanted subjects. An IDE trial of the first generation implant (Argus<sup>™</sup> 16), which has 16 electrodes, is ongoing at the Doheny Eye Institute at the University of Southern California. The Argus 16 was implanted in six RP subjects between 2002 and 2004 and has enabled them to detect when lights are on or off, describe an object's motion, count discrete items, as well as locate and differentiate basic objects in an environment. Five of these subjects are now using their Argus 16 retinal prostheses at home.

The next generation Argus II retinal stimulator is designed with 60 independently controllable electrodes, which should provide implanted subjects with higher resolution images. Second Sight remains the only manufacturer with an actively powered permanently implantable retinal prosthesis under clinical study in the United States, and the technology represents the highest electrode count for such a device anywhere in the world.

The study will be conducted in subjects who:

Have a confirmed history of RP with remaining visual acuity of bare light perception or worse in both eyes with functional ganglion cells.

- Have a history of former useful vision.
- Are fifty years or older.
- Reside within two hours of surface transport from the investigational site.
- Are able to verbally communicate in English.

Subjects with optic nerve disease, glaucoma, diabetic retinopathy, ocular trauma, or a history of retinal detachment are not suitable candidates for this study. Subjects must also be physically able to undergo general anesthesia. If you know of a suitable candidate, or if you are a physician with further questions, please contact [patients@2-sight.com](mailto:patients@2-sight.com) or 818-833-5027.