

A Discussion With
Peter L. Sonkin, M.D.
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Topic: Macular Disease

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Dr. Sonkin is a member of the Tennessee Medical Association, Tennessee Academy of Ophthalmology, Nashville Academy of Medicine, Nashville Academy of Ophthalmology, Society of Heed Fellows, American Society of Cataract and Refractive Surgery, American Academy of Ophthalmology, Association for Research in Vision and Ophthalmology, American Medical Association, and Southern Medical Association.

He has received numerous honors, grants, and fellowships for his work in ophthalmology and has participated as an investigator for three major clinical trials, namely, the National VAM (Verteporfin in AMD) and the current TTT4CNV (transpupillary thermotherapy) and Vitrase studies. He has been published more than twenty times in major journals, and has been a prolific presenter before the Association for Research in Vision and Ophthalmology (ARVO) and other such venues.

Recently married, Dr. and Mrs. Sonkin make their home in Nashville, Tennessee.

(Edited for length and clarity)

DAN: Dr. Sonkin, we greatly appreciate the time you are taking to share your knowledge and experience with macular diseases. Welcome to MDList, and I hope you will enjoy meeting the members of our Internet community. The session is now open for questions.

KAY: I was dxed with MD in both eyes on June 6. I look at the Amsler Grid daily. Everything is clear as can be, no wavy lines or blank spots. The retina specialist took tests of my eyes and many pictures. He looked at the pictures and said "You are lucky, you have dry MD in both eyes". He walked out of the room. End of discussion?? I had cataract surgery on May 7, and ten days later I saw a purple duck that came and went. I went to the doctor and he sent me to the retina

specialist. The image lasted several days, and I haven't seen it since. My visual acuity is very good. No problems reading or driving. What is your opinion of this diagnosis? I am inclined to believe that the RS is wrong.

DR. SONKIN: The initial diagnosis of "dry" AMD is likely accurate. After cataract surgery, many things can occur that can cause temporary symptoms. It is normal to have inflammation inside the eye, and this usually resolves on its own and with the help of normal postoperative eye drops. This inflammation can cause symptoms consistent with what you described. It is also possible that you developed mild "swelling" in the macula, which we call cystoid macular edema or CME. This affects the same part of the retina as AMD, but is a different condition. It often gets better on its own, but sometimes requires special eyedrops or other treatments. This may have caused your symptoms. If your macular degeneration has converted to the wet form, i.e. you have developed abnormal blood vessels under the retina known as CNVM (choroidal neovascularization), then your symptoms would likely be worse and persistent. It is important to monitor your amsler grid daily. With any changes you should see your retina doctor asap. Catching the wet form before it reaches the center of the macula (the fovea) is very important and can often prevent or delay the more severe visual problems. Catching the wet form early rather than late if it develops right in the center will minimize the damage and visual loss. There are other things that might have developed in your eye, but the above are the most likely things. It is difficult to be more specific without actually seeing your eyes. You should follow-up with your retina doctor as scheduled and call him if you develop any new symptoms.

BARBARA: My sister and I have been diagnosed with adult vitillform dystrophy, or Best's disease. Would you please explain the diagnosis further? What is the likelihood that my sons (early 40's) or grandchildren will inherit the same vision problems?

Recently, I have noticed a severe light intolerance, which is reduced with UV protection in sunglasses and a wide brimmed hat. Is there anything else that can be undertaken to reduce the effect of bright light into the macula?

DR. SONKIN: Best's Disease is an autosomal dominant hereditary retinal disease. This means it is inherited, and there is a 50% risk of passage of the disease from an affected individual to an offspring. Each of your sons were at a 50% risk for the disease. Best's disease is usually easy to diagnose by exam and confirm by EOG (electrooculogram) at an early age. If a given son is not affected, the risk for that son's children for Best's disease is essentially zero, unless your daughter- in-law happens to have the disease, which would be very unlikely. If one of your sons is affected, each of his children would then be at a 50% risk.

No retinal disease is good to have, but if you had to pick one, Best's disease is not as bad as many. Usually the visual loss is not as severe and it usually occurs later in life. This is not true in every case, and I am not trying to minimize the effect it has had on you and your sister, but it does have a better prognosis. In contrast to the visual loss, the clinical appearance is very striking and impressive, with an "egg yolk"-like appearance to the macula. Other associated problems can develop late in

the disease but are rare. This includes bleeding and choroidal neovascularization, much like we see with AMD.

You do have an inherited degeneration of your macula, but this is not the same as "age-related macular degeneration". Sensitivity to light is a nonspecific symptom, and is not necessarily related to your macular problem. It could be cataracts or normal light sensitivity.

JUDY: I received two photocoagulation laser treatments about two years ago, within six weeks of each other, due to a severe hemorrhage in my right eye.

As a consequence, I lost all central vision very quickly in that eye with a visual acuity of 20/800. I was told I had to have this surgery to avoid profound vision loss. Apart from "social blindness", how much more profound can you get with no central vision remaining in one eye?

Photodynamic therapy appears to be a better treatment option than photocoagulation, but is only performed on about 20% of the Wet MD eyes examined. Is that statistic correct?

Somehow I am under the impression that these two laser treatments are, in reality, only bandaid solutions, but superior to doing nothing. I am wondering if a saner approach would be to simply try harder to accept our vision loss rather than running after treatments that might or might not work.

My retinal surgeon thinks that macular translocation, once perfected in a few years, might be a better treatment choice for the future. Would you by chance have recent statistics on the validity of these two lasers as opposed to doing nothing, during this time frame, when neo-vascularization occurs?

DR. SONKIN: I am going to try to answer your question in sections.

Thermal laser was shown to be beneficial in a large study done approx 20 years ago called the MPS (macular photocoagulation studies). It showed conclusively that thermal laser was beneficial in preventing visual loss for wet AMD (CNV) not involving the fovea if it is a certain type called "classic" as defined by a fluorescein angiogram (FA). This still holds true today for nonfoveal classic CNV.

Before the approval of PDT, many retinal surgeons also used thermal laser to treat classic CNV that does involve the fovea. This was also shown to be better than observation only by the MPS trials. Thermal laser to the foveal region, however, does not improve vision or necessarily prevent further loss. The MPS study showed that it does reduce the likelihood of SEVERE visual loss (6 lines on the eye chart) versus observation only. The analogy I often used with my patients is that thermal laser to the fovea is similar to amputating a finger to save a whole arm. In other words, the thermal laser to the fovea results in an immediate and permanent blind spot, and often a drop in vision. But this blind spot is hopefully smaller and not as dense (or dark) as the natural blind spot that would have resulted from observation only.

Most retina surgeons now treat fovea-involving CNV with PDT because it minimizes the damage to the overlying retina and hopefully minimizes the visual loss. Neither thermal laser or PDT is a cure. Hopefully research will lead to ways to prevent CNV growth, rather than having to wait to treat after damage is done. Some studies with diode laser have shown promise. Our practice is part of the PTAMD study. There is also a lot of research on antiangiogenic drugs that may work. Our practice also does macular translocations, but this is also not a cure. It is a surgical procedure that moves the macula away from the CNV, but it has many inherent risks and is not done as much since the introduction of PDT.

Although I agree with your analogy that these treatments are somewhat like bandaids, I still feel strongly that they are better than no treatment when they meet proper indications. I know the frustration that I sometimes feel in treating macular degeneration is not at the same level of the patient, but it is definitely present. It is hard to accept that sometimes the best we can do is slow down a process rather than curing it. It is also, however, extremely satisfying when I have a patient improve after PDT. I have several patients that were legally blind that have improved to 20/50 or better after PDT, and I have monocular patients that also have had great responses to treatment. It is also, without a doubt, very satisfying to treat a nonfoveal CNV and prevent its spread to the fovea. This is more satisfying for me than my patients because they often have not lost vision yet and therefore are unable to comprehend what would have happened to the central vision without treatment. This also helps to emphasize the importance of the amsler grid, particularly for patients that have lost vision in one eye from AMD.

DOTTIE: My friend is going to have thermal photocoagulation treatment. Would photodynamic therapy be a better option?

DR. SONKIN: Thermal (hot) laser is still the treatment of choice for classic choroidal neovascularization (abnormal blood vessels under the retina that are well-defined; CNVM; wet type) that does not involve the fovea, or center of the macula. If the CNVM your friend has is mostly "classic" and involves the fovea (very central point of our vision), then most retina surgeons would argue that PDT is better than thermal laser. If the blood vessels do not involve the fovea on fluorescein angiogram, than the thermal laser he received would be the best option. Hopefully the physician that treated your friend is trained in making the proper distinction, which can often be very difficult.

ROB: My father (age 84) only has one eye that's got "useable" vision. The other has long term ARMD and other related problems that render it nearly useless. The remaining eye had it's first leakage a year ago that seems to have spared the fovea. The absorption/clearance of the leakage substances has been very slow and is, perhaps, still ongoing.

He is of the impression that PDT, in the event of another leakage event, could be a substantial risk to the remaining vision. Something about the condition of the rods and cones. I don't know where he got this idea but I didn't want a fatalist attitude as that might lead to ignoring danger signs and assuring loss of remaining vision. I've read that the earlier one can catch and treat the leakage, the better chance there

is of saving central vision.

DR. SONKIN: To fully answer your question I would need more information. In general, though, you are correct that early detection is very important. The location of subretinal fluid is not nearly as important as the location of the abnormal blood vessels (CNV) under the retina that are leaking and resulting in the "wet" AMD. This is determined by exam and fluorescein angiogram (FA). If the CNV is caught before it extends under the fovea and it is "classic" and well- defined, thermal laser can be used to "save" the fovea and the very central vision. If the CNV is "classic" and involving the fovea, PDT is often the best treatment choice. There is some risks with the treatment, but the risks of no treatment is greater. This was proven in large national clinical trials that resulted in FDA approval of the treatment. If the CNV is not mostly "classic", but instead is "occult" in nature (as determined by exam and FA), treatment is completely different. There are several treatments that retina surgeons offer, including TTT and in some cases PDT. Neither is FDA approved for that indication at this time.

With regards to the status of the rods and cones, your dad may have another retinal condition on top of the AMD. With regards to the slow and chronic leakage, this may indicate an "occult" CNV or other variations of AMD, such as serous or fibrovascular pigment epithelial detachments (PEDs). Treatment varies depending on many factors.

The bottom line is to emphasize to your dad the importance of regular exams and home amsler grid evaluation. With any changes, he should see his retinal surgeon ASAP. If he develops a change that necessitates treatment, it is important to let him know that the goal can be to improve the vision. In many cases, however, it is to prevent further loss of vision or to slow down vision loss, both of which are still very valuable.

SHARON: I am now 55 yrs of age and had a sudden onset of eye problems. First diagnosis was central serous retinopathy, then CNV, then the conclusive diagnosis was determined by use of ICG.

I have a variant of idiopathic polypoidal choroidal vasculopathy. For two years, I requested the opportunity to have phimotion angiography and was told it was not suitable. I lost the vision in one eye due to scar tissue from a bleed. I again asked for phimotion angiography when a hemorrhage recently occurred in the other eye. I asked a different RS this time. He was enthusiastic and arranged for the consult and treatment to cut off the feeder vessels.

1. Why are Doctors so reluctant to refer a patient to treatments they are unable to personally provide?
2. If we cut off the feeder vessels of which there are two, does this mean the progress of the disease is halted?
3. Even if the CNV is halted will the macular degeneration progress?

4. Is there an explanation as to why transpupillary thermotherapy (TTT) could be painful in one spot on the eye?

DR. SONKIN: It does sound like you have a complicated retinal history. I will try to answer some of your questions. Central serous retinopathy (CSR) can lead to the development of choroidal neovascularization (CNV). Idiopathic polypoidal choroidal vasculopathy is a complicated disease process, but in simplest terms it is a multifocal growth of CNV abnormal blood vessels under the retina. We do not know the best treatment, but most retina surgeons I know will watch this, unless it is causing problems or progressing. If the CNV is not foveal, thermal laser is often a good choice. If the CNV involves the fovea, some prefer PDT.

Feeder vessel treatment (aka phimotion angiography) has been shown in some studies to be beneficial if a feeder vessel is identifiable, which it often is not. There are risks with feeder vessel treatment, just as there are with any type of laser. If a feeder vessel is successfully treated, there are times when this will result in regression of the entire CNV with stabilization or improvement of vision. It does not cure the underlying problem (AMD or polypoidal or CSR), and persistence or recurrence of the CNV is definitely possible. In answer to your question, halting a CNV membrane does not stop or cure AMD, but it may prevent or delay visual loss.

TTT laser is a large spot diode laser that more frequently causes discomfort because it's wavelength is absorbed deeper to the retina.

With regards to doctors referring patients to other doctors that offer different treatments, that discussion could take weeks. I always refer if I feel someone else is better suited to help a given patient, and I take no offense to my patients obtaining second opinions and I sometimes recommend it. At the same time, I feel very protective of my patients and if I feel a certain type of treatment holds more risk than benefit, that is what I tell them.

I hope your better eye continues to do well. I saw a patient of mine this morning that has polypoidal choroidal vasculopathy. Her left eye has had two thermal lasers and remains 20/20. These lasers were done approximately one year ago, because the extrafoveal CNV were leaking fluid into the fovea and reducing the vision to 20/200. In her right eye, the initial thermal laser was also successful. Approximately four months ago, she developed a CNV in the right eye that involved the fovea, and we are in the process of trying to control it with PDT. Hopefully this will also do well.

DOE DOE: According to a recent report, PDT treatment for "occult only subfoveal choroidal neovascularization" now qualifies for reimbursement under Medicare. Does the qualifying condition include all forms of MD or is "occult only subfoveal choroidal neovascularization" a special type of MD, leaving the others excluded?

DR. SONKIN: When characterizing "wet" AMD on fluorescein angiography, the abnormal blood vessels fall under one of two categories, either "classic" or "occult".

Making this determination requires extensive experience with retinal diseases and treatment, and usually is only done by retina surgeons. In a given eye, the CNVM (wet form) can be completely classic, completely occult, or a combination of the two. Visudyne has been approved for completely classic, predominantly classic (greater than 50%), and now also completely occult. This still leaves out a small group of patients that have a combination of the two that is predominantly occult. Visudyne may also help these patients, but studies have not revealed this yet.

GEOFF: Many thanks for taking the time to answer our questions. I wonder if you have any experience with Central Areolar Choroidal Dystrophy (CACD)? Also, I see there have been studies that indicate antioxidants and zinc may be beneficial. I wonder if this also applies to hereditary types of MD, or is their progress already pre-programmed so that supplements may not have the same effect?

DR. SONKIN: Central areolar choroidal dystrophy (CACD) is an uncommon hereditary degenerative retinal disease. Symptoms of decreasing vision and loss of central vision usually arise in the teenage years through the 20's and 30's, and the rate of subsequent visual loss is very variable. Clinically, the macula develops a "mottled" appearance early on, which is then followed by a very distinct sharply bordered macular atrophy. We believe the underlying problem is the circulation beneath the retina and RPE known as the choriocapillaris. It is unknown whether vitamins will help slow progression of CACD, but given the proposed mechanism, it is less likely to help than in an age related degenerative process such as AMD. It is also important to keep in mind that CACD looks extremely similar to other conditions (too many to list), some of which carry a better prognosis and some worse. Also, there is no definitive test for CACD. Diagnosis is based on clinic appearance, clinic course, and FA. Best of luck, and be sure to follow-up with a retina specialist at least once a year. As your condition evolves, the diagnosis will become more certain. Also, I strongly recommend seeing a good low vision specialist. Although your central vision may become worse, fortunately CACD will not completely blind you and you will retain normal peripheral vision and some central vision. You certainly will want to optimize your ability to use the vision that you do have.

ROSIE: I'm a 62-year old female and have had high myopia since I was 9 years old. I had cataract surgery on both eyes in 2000 and macular hole surgery on my left eye in April 2001. In Aug/Sep 2001, vision in my right eye turned worse. My corrected visual acuity is a fuzzy 20/100 in my left eye and a fuzzy 20/50 in my right eye. [Lists specific results of examinations, not included here.] I have the following questions:

1. Is retina pigment epithelial changes with no drusen dry age-related macular degeneration?
2. Is it the same as RPE - retina pigment epithelial detachment?
3. Can it go from dry to wet without first drusen developing?
4. The new study on zinc supplements mentions that supplements are most successful when taken by persons with somewhat advanced dry macular degeneration. Am I at this stage?
5. Why can my myopia not be correct with glasses?

DR. SONKIN: Given the normal appearance of the right macula, and your history of macular hole surgery on the left, odds are that the RPE changes seen in your left macula are the result of the old macular hole. This is very common and likely is the cause of the reduced vision at 20/100. Although we can close macular holes greater than 90% of the time, this does not always result in vision near 20/20. I tell my patients that the main goal of closing the macular hole is to improve the vision, but the degree of improvement can vary greatly. It depends on the size of the hole, the duration of the hole, the health of the underlying RPE and the surrounding retinal tissue, the overall health of the patient, and last but not least, nature. Not every eye heals the same way. It is possible that the changes in your left macula is AMD, but this seems less likely to me. It is difficult, however, to tell you with any certainty given that I am unable to actually examine your eyes.

Regarding your specific questions:

1. RPE changes without drusen can be a presentation of AMD, but not always. You have to consider the person's age, the appearance of the other eye, the medical history, and the past ocular history (e.g. prior surgery). It is a clinical decision.
2. RPE detachments are a specific subtype of AMD. They are often classified as either serous or fibrovascular, the latter being a form of "wet" occult AMD.
3. Drusen are not always clinically visible, and you can develop wet AMD without first having visible drusen, although this is not common.
4. Vitamins may or may not help your condition. I would recommend a good balanced diet and multivitamin. This is not, however, based on any science.
5. General myopia is correctable with glasses, unless the myopia has resulted in functional damage to the retina or underlying tissues. The analogy I often use when asked why glasses won't always fix visual loss is that changing glasses is like cleaning the windshield of a car that isn't running very well. If the problem is with the engine of the car, it doesn't matter if the windshield is crystal clear and clean, the car won't run any better. The same holds true for the eye. If the problem is with the retina (the "engine" of the eye), new glasses (or cleaning the "windshield") unfortunately won't help.

MAGGIE: About three years ago, I began to find it uncomfortable to open my eyes in the morning. I had the sensation that opening them would cause discomfort - rather like trying to open an eye glued down with matter- although there was no matter in my eyes. Usually, just one eye was affected. Sometimes it was the left and sometimes the right. Within a short time, I experienced several incidents of difficulty in opening one eye, followed by intense discomfort. My eye hurt as if someone had poked it with a sharp stick. This pain would last for about an hour, then disappear.

In the past couple of years, the condition has grown worse. I cannot open my eyes without massaging them, gently, first. If I do this and open my eyes very slowly, I have no problem. Once in a while, I forget and open my eyes rapidly. Usually one eye seems to get stuck to the lid and I am in agony for an hour afterward. The eye

actually puffs and waters for this period. Afterwards, it returns to normal, but my vision in it is blurry for a couple of days or more and my eye is very sore.

I have angioid streaks, but they have never caused any problems, and my vision has not deteriorated. I am curious about what causes the pain I experience when I open my eyes suddenly.

DR. SONKIN: With regards to the angioid streaks, it is important to continue follow-up with your retina doctor. They predispose you to CNV (abnormal blood vessels under the retina), much like those found in wet AMD. Also make sure you have been evaluated for systemic diseases that can cause angioid streaks. The mnemonic we use for causes of these streaks is: PEPSI = pseudoxanthoma elasticum, ehlers danlos, paget's disease, sickle cell anemia, and idiopathic ("bad luck, no cause"). Idiopathic is the most common.

With regards to your other symptoms, I would recommend seeing a corneal/external disease specialist. It is a bit out of my area of expertise. The most likely things I would think of is a corneal dystrophy (which can sometimes be hard to diagnose and there are several different ones), recurrent corneal erosions, abnormal palpebral conjunctiva (under the lids), blepharitis, etc.

SHARON: In your response to Judy you wrote, "There is also a lot of research on antiangiogenic drugs that may work." Would you please be so kind as to list these drugs? Are there known severe contraindications? We would certainly appreciate any insight into drug therapy.

DR. SONKIN: The use of antiangiogenic drugs for AMD is still in the future. There have been a few studies done, including thalidomide and other drugs. That study was stopped due to systemic side effects. Most of the research is still in the basic science stages, although some clinic studies do exist. We can all remain hopeful. Certainly, preventing the problem before it develops is the ultimate solution.

ALAN: About 2 months ago, I suddenly developed what has been diagnosed as posterior vitreous detachment. Is this related to, or caused by, my dry MD? Could it be a precursor to development of wet MD in that eye? Can I expect continuing detachment of the vitreous in that eye?

DR. SONKIN: Our eyes are filled with a jelly-like substance called vitreous behind our natural lens. The vitreous fills the posterior compartment of the eye. As we get older, the vitreous undergoes many changes. One change is that it separates from the surface of the retina. This normally occurs without any major problems except for visible floaters. It can also cause retinal hemorrhages, vitreous hemorrhages, retinal tears, and retinal detachments. I am sure your doctor checked for these things.

Posterior vitreous detachment (or separation), also called a PVD, is not related to macular degeneration. The symptoms will likely persist, but may change and fluctuate. If you develop any sudden changes including a sudden increase in the floaters, flashes, or curtains/veils over your vision, return to your retina doctor for a

recheck.

GIDEON: I was treated only once with PDT on a "classic" leak in January 2000. Since then, despite at least 4-5 additional leaks, but never under the fovea, my RS has decided to let it dry alone, as it really happened. My vision stayed stable since then.

My question is what according to your opinion is the dynamic "life circle" of the sickness? In the beginning, they said one year, then it became one and a half, now we talk about two and more.

The second part of my question is what is supposed to happen after one cycle, should/must we expect others? Or is PDT too new to answer this? How was it before PDT? My first eye, with hot laser (including your finger-hand phenomena) stays still static the 11th year now.

DR. SONKIN: Whether wet AMD goes untreated, or if it is treated with PDT or thermal laser, no two eyes behave the same. I tell all my patients that persistence and recurrence is, unfortunately, always a possibility with any treatment, but that hopefully those are bridges we won't have to cross. It is almost impossible to predict the risk of recurrence in a given eye. I have patients that have been treated only once with both thermal laser and/or PDT, and no recurrences have appeared. There are also patients who have been treated multiple times with no resolution. I have seen patients who were treated with thermal laser 15 years ago with absolutely no recurrences.

NATHALIE: My mother has wet in one eye and dry in the other--now beginning to deteriorate badly (blurring, not distortion). I took her to the Jewish General in Montreal, where, after another angiogram, they said she has occult leakage--not classic--in the dry eye, that may or may not be stable for months or years. After reading your comments, I suddenly realize that perhaps Visudyne may work for this occult leak. What should I do? Should she go and see Dr Harding in Liverpool again? (I took her there two years ago.) Should we risk trying microacupuncture in Scandinavia? I sometimes feel desperate at not being able to grasp all the intricacies of the diagnoses. I know there is no miracle but anything to stop the deterioration would be worth it.

DR. SONKIN: Occult CNV comes in several forms and can vary greatly from patient to patient. There are new treatments that have recently been shown to help occult leakage, but not all affected eyes are candidates. Given that your mother has not seen the retina doctor in two years, I do recommend that she be checked. Even if things are stable and no treatment is needed, two years is much too long between appointments. Certainly, if your mother notices any changes in her vision, she should be seen immediately. With regards to the acupuncture, I really don't have any information on it. I usually only recommend therapies with good scientific evidence of efficacy, but, at the same time, I am aware that there can be alternative treatments that sometimes can provide some help, even if it is only emotional help. Good luck.

NORMA: I have two questions for you:

1. I have wet MD (bad eye 20/400, good eye 20/25 and holding) and was fortunate that leaking in the good eye was spotted and lasered by my retinologist, Dr Charles Weldon. I now have chronic open angle glaucoma and my question to you is will that compromise my existing condition?
2. My husband has developed dry md and will be followed by my retinologist. Are there any precautions we can take for my husband? He has a diet high in fruits and vegetables and avoids direct sunlight.

DR. SONKIN: Glaucoma has not been shown, to my knowledge, to directly affect macular degeneration. The one point I would make is that advanced glaucoma causes loss of peripheral vision, which is not a good combination with advanced macular degeneration and poor central vision. Keep up with your appointments and medicines, and hopefully the glaucoma will never progress.

It sounds like your husband is doing the right things. Make sure he monitors an Amsler grid at home and maintains regular dilated eye exams.

ROSIE: Thank you for your explanations to my previous questions. Could you provide some additional information?

1. To my question #1, you replied that this is a "clinical decision." What does this mean?
2. To my question #5 about correcting myopia, you replied that correction may not be possible if there is functional damage to the retina or underlying tissues. What is the technical term for this medical condition, and what usually is the prognosis?

DR. SONKIN: When I stated that it is a "clinical decision", what I meant was that sometimes the appearance of an abnormality cannot be labeled as a specific disease, rather it "looks most like" a certain diagnosis, and it is, therefore, a clinical decision made by the examining doctor as to what he or she feels is the most likely diagnosis. This is based on the clinical appearance on exam, the symptoms, the history, and ancillary test (such as FA, etc.) Medicine is not always a 100% science, and this is where clinical experience and good training is important.

Regarding your second question, functional damage from myopia can be termed degenerative myopia. I was not stating that this is what I think you have. You asked why myopia can't be corrected with glasses. Degenerative changes in the macula from the myopia is just one possible explanation. Myopia can also cause small hemorrhages in the macula, and this can leave some damage even after resolution of the blood. Again, in your case it might be due to the old macular hole, AMD, etc. Only your examining doctor can provide information specific to your eyes.

TESS: I was diagnosed with Stargardt's disease fourteen years ago. Although there has been the expected deterioration in my eyesight, over the past 4-5 years I have also noticed brightly coloured spots in my central field of vision. They are always

neon purple and neon yellow combinations. However, the patterns, shapes, and sizes differ slightly.

The spots are not there all the time, but they do tend to occur more often when I am tired or stressed. Are they related to Stargardt's in any way or are they more to do with stress? Do these spots mean that my eyesight is deteriorating more rapidly? Is there anything I can do to get rid of them or to avoid getting them?

DR. SONKIN: Your symptoms can be due to many different things, and I would recommend that you discuss it with your retina doctor. Stargardt's and other macular diseases can cause varying symptoms, and may explain what you are noticing, but it is important to rule-out other things. It may be changes in the vitreous or other parts of the eye. Sorry I can't be of more help, but to answer your question reliably really requires an examination.

A note to everyone: I am enjoying all of your questions and discussions. You have a wonderful group, and I am also learning a lot and expanding my perspective on retinal disease. I want to emphasize one important point. Nothing replaces the actual retinal examination. My comments are based solely on your questions and statements. I am not able to examine your eyes, and I would, therefore, definitely defer your treatment decisions to your examining retina doctor. Your questions have been excellent and very detailed, and I hope my answers have not been too vague. Hopefully the information I am providing does help answer some of your questions, and I hope it is also helping to broaden your individual knowledge of macular degeneration and other retinal diseases. I look forward to continued discussions.

BETTE: My major problem now is job hunting with this MD. I have applied for disability, but do not think I will get it. Despite failing my state drivers test, the low-vision doctor gave me a license with no restrictions. I am trying to see about going to school to do medical transcription, as I will need to do something from home. Will I be able to do this? Is this unrealistic?

MAGGIE: I read your question to Dr. Sonkin and identified with your concerns about retraining in order to remain self-supporting. I . . . went through a period where I was terribly worried about how I would continue to make an income. . . After agonizing for a year or more, I came to the conclusion that nobody could really tell me what was going to happen with my sight. I just had to keep the faith and carry on. So far, I am able to do my old job. . . I think the best thing we can do is to move ahead with our plans, instead of letting the "what ifs" paralyze us. Why not retrain and look forward to a new career? Worry about changes in your vision only if they occur. In the meantime, go with the existing situation and make the most of it. I think all of us have to bear in mind that we have sight problems that could force us, eventually, to make some very difficult life decisions. I am trying to cover all the bases, in the event that I have to find a different sort of work. I don't know how things will work out, when and if something really bad happens. Right now, I can cope, and that is what matters.

You remind me of myself two years ago, when I first joined this list. I felt that I had

to know "how long", "how bad," etc., and no one could tell me. Many people on the list told me that things would work out and they were right. They will sort out for you, too, so don't worry. Just be as confident as possible, and retrain in good faith that you will have many productive years at your new job.

DR. SONKIN: I don't think I can add much to Maggie's comments. I agree with everything she said. I do have many patients with very reduced vision that work and go to school, usually with the assist of low vision aids. Reading and using the computer will not make your AMD worse. "Straining" your eyes has never been shown to have any impact on the progression of macular degeneration.

MAGGIE: Recently, I made an inquiry in which I mentioned I have been diagnosed with angioid streaks. In comparison with MD, how common is this condition? My streaks have been confirmed as idiopathic.

DR. SONKIN: Angioid streaks are much less common than AMD. AMD is the leading cause of legal blindness in the USA for patients over 50. I see numerous patients every day with AMD, and probably five to ten patients with angioid streaks per year.

IRVING: Do you have any thoughts on the role of stress in illness, and any suggestions for dealing with stress.

DR. SONKIN: There is no easy way to cope with stress of any form. The best start is realizing that it exists, trying to understand the source of the stress, and going from there. Groups like this site provide incredible support. With regards to the effect of stress on AMD, to my knowledge there are no studies that have supported a relationship. I believe that stress does have an effect on our bodies and all of our systems, but to what degree I don't know. When I am particularly stressed, I seem to get more colds, and I am much more tired. I would think stress would also have an affect on chronic conditions, but to what degree is uncertain.

ALICE: Do you have any information on using a sclera buckle to reinforce an eye that has Myopic MD? I have only read about this reinforcement when the retina has already detached.

DR. SONKIN: A scleral buckle should only be used when the retina is detached. There is no other approved indication for that kind of surgery, to my knowledge. If you are told otherwise, I would be interested in knowing more. We do sometimes place a scleral buckle after severe trauma if we are doing a vitrectomy at the same time.

ALICE: my retina specialist has been doing the sclera buckle procedure for over ten years. He has been to Russia to oversee the procedure there. I think they use synthetic sclera.

I have myopic MD, so the eyes are elongated, but in addition I have these bulges. It is hard to get a correct measurement. I believe normal length is 21. Mine is 27 or so. My RS has been recommending the sclera buckle for me. He told me that the

procedure does not prevent further damage, but slows down the progress of the disease.

DR. SONKIN: In severe myopia, you can sometimes get severe scleral thinning and an outpouching known as a staphyloma. If this is so severe that there is a risk of extrusion of intraocular tissue, sometimes we support the staphyloma (thinned sclera) with fresh scleral graft tissue. This is not the same thing as a scleral buckle, though. Myopia-related degeneration of the macula is also a completely different issue, and I am not familiar with any scientific evidence that a scleral buckle helps with this. I may be wrong, and there may be studies that support this. I personally do not have any experience with this, nor do I know any other retina surgeons that perform scleral buckles for this reason. I would talk with your retina surgeon further.

ERV: I have dry (20/20) in my left eye and wet occult (20/800) in my right, with glaucoma and cataracts in both eyes and a macular hole in the wet eye. I converted to wet in the right eye last year. I understand that PDT is now available for occult MD and would appreciate your thoughts about treatment possibilities in general and related considerations. Is help available in any form for scar tissue?

DR. SONKIN: Your situation is a difficult one. Both the macular hole and the wet occult AMD can result in significant loss of the central vision. It is a clinical decision between your doctor and you as to whether or not the risks and benefits of macular hole surgery and/or PDT treatment weigh in your favor. Surgery for macular holes is very successful (approx 90% closure rate), and laser for CNVM is moderately successful. When both coexist, there is no way to predict either anatomic success or potential for visual improvement. Good luck.

CLAUDIA: My dad (88) went to see his opthm (not RS) today for a checkup. The doctor detects a new macula bleed, but says let's just wait a couple of months and look at it again in January. When he told me this I LOST IT. Now did I overreact? He has minimal vision left. It just seemed very cavalier and sloppy to me to wait and see. Feedback please?!

DR. HENSIL: I agree that the doc didn't explain things well and his bedside manner needs a little help. This doesn't mean he is wrong. A "new bleed" just means there is some blood in the macula, a small hemorrhage. This does not necessarily mean that there is new neovascularization there, and it is only neovascularization (new and leaky blood vessels that are not supposed to be there) that is treatable with laser, and sometimes even that is not treatable. The blood, depending on how much there is, can cover the RS view of what is going on underneath it, and until some of that blood resolves (which can take a couple months) it may be impossible for him to make an assessment of the situation.

With regard to vision, the worst that any person with MD alone will see is about 20/200 (give or take a little) as that is what the peripheral visual acuity is. Every patient I have with MD reads the newspaper, many work or volunteer, all take care of themselves (except those in nursing homes for other reasons), etc. I strongly suggest a low vision evaluation for your dad, as there is no reason that any of his

visual goals can not be met. Of all the eye diseases out there, MD is one of the 'best' to get as you never go completely blind and there is always a lot of vision left to work with (again, unless there is other disease like glaucoma, cataracts, etc. present).

DR. SONKIN: I would like to add one comment. In the past, if a macular hemorrhage was extensive enough to obscure the view of the underlying pathology, then it was true that we had to wait until the blood cleared before considering laser treatment. This was often not very satisfying, because sometimes the blood doesn't clear, and sometimes the underlying pathology progresses and results in permanent damage, while we wait for the blood to clear.

Fortunately, we now have surgical options to move the blood to hopefully improve vision and allow for laser treatment if indicated. One of my partners developed a novel approach to this, in which we perform a vitrectomy, place a blood thinner under the retina to dissolve any clotted blood, and then place a gas bubble in the eye to displace the blood from the macula. I have had several successful patients in which the vision improved after I performed this procedure, and I was able to treat the CNVM (abnormal blood vessels that caused the hemorrhage), hopefully preventing additional bleeding in the future.

DR. HENSIL: Wouldn't this cause a fairly decent rate of retinal detachment? Is it worth risking a retinal detachment in someone who already lost all their central vision and functionally has little to gain from such a procedure?

DR. SONKIN: That is an excellent question. What I describe is a fairly straightforward procedure that is technically not difficult. The subretinal injection of TPA and the gas-fluid exchange does not add much risk over the core vitrectomy. I would estimate the risk of retinal detachment after a procedure such as this to be less than 5 percent. This still must be considered when weighing the risks/benefits, in addition to the risk of infection, bleeding, etc., and I discuss this in detail with the patient and family. We also consider the condition of the fellow eye, the patient's overall health, etc. The procedure is outpatient and done with a local, so there is little systemic risk.

SHARON: What are the contraindications to the health of a patient or their eyes by repeated fluorescein and ICG angiography? I recognize the possibility of allergic reactions, but what about causing damage to the vessels of the eye or the kidney or liver? Are there other concerns?

DR. SONKIN: I do not know of any association between repeated angiograms and damage to the retinal circulation (or other vascular beds). In patients with reduced kidney function, elimination of fluorescein from the body takes longer, and sometimes I will use a reduced dose.

KAY: About three weeks after cataract surgery, I started seeing a purple image in the form of a duck, which would come and go. My doctor sent me to a retina specialist, who told me I had dry MD in both eyes. I was on steroid drops five weeks after the surgery. When I quit taking them, the purple image stopped, and I have not seen it or anything else. I really don't believe the RS and am in the process

of getting copies of my records and pictures. My vision is great. In fact, I was driving without glasses, only needing them for reading before surgery. On the Amsler grid I see no wavy lines or blank spots. It is a puzzle to me.

DAN: Kay, there is substantial evidence that steroids can lead to a form of retinal degeneration called central serous retinopathy (CSR). This is a condition of the macula which can be temporary, and it can be misdiagnosed by doctors who are not familiar with it. You are fortunate that your condition reversed after you stopped taking the drug.

DR. SONKIN: CSR can be slightly difficult to diagnose, but most retina surgeons can easily distinguish it from other conditions by clinical exam and fluorescein angiogram. Steroids can make this condition worse, but usually do not cause CSR. It is an idiopathic condition, which means there is no cause (just bad luck).

DAN: There has been a quantity of recent research which has concluded that corticosteroids (glucocorticoids) administered systemically can actually induce CSR, in addition to increasing the morbidity of the disease. I based my reply to Kay upon these reports, a few of which are listed at the end of this message, and I welcome any more insight that you might have. {Note: The research reports are not listed in this transcript for reasons of space.]

I had the unfortunate experience of being misdiagnosed for 18 months, during which time my ophthalmologist (not an RS) administered a subtenon injection of steroids, after which my blind spots doubled in size within 24 hours. I sought a second opinion from Dr. Lawrence Yannuzzi in NYC, who immediately diagnosed my condition as CSR. Sadly, there are doctors who still do not recognize the disease, and--even worse--are still not aware of the danger of steroid use in connection with it. I only wish I had first met one who was.

DR. SONKIN: Your experience is unfortunately an example of what I feel is the most important thing in medicine, not just ophthalmology. One of the most critical aspects of caring for patients is "knowing what you don't know," and seeking other opinions and help when necessary. Unfortunately, even the best doctors (in many different fields) with the best intentions sometimes fail to refer when needed.

Additionally, I am very familiar with some of the articles you referenced regarding the association between steroids and CSR. My previous comment was meant to reference the clinical application of this knowledge. It is well known that steroids can aggravate an existing and active CSR leak, and I certainly avoid steroid use in patients with CSR. I do not know of any physicians that would avoid steroid use because of the small risk of precipitating CSR, nor would I discuss this as a risk if prescribing steroids. No studies have looked prospectively at this risk, and most of the cases occur in patients with underlying macular pathology and possibly prior CSR episodes. The average CSR patient has 3 to 5 recurrences over their lifetime. I also do not believe any studies have compared the incidence of CSR in the general population to the incidence in patients treated with steroids, but I may be wrong. This would be very difficult to do anyway, because many CSR cases are

asymptomatic if not involving the fovea, and most improve and resolve without treatment.

One last point: literature searches can turn up an association between almost any two entities, particularly medical diagnoses and drug use. How one uses this information clinically can be very difficult, and all things must be considered when weighing the risk/benefit ratio of any surgery or therapy. Thanks for the discussion. I am enjoying the interaction, and I am learning a lot myself. I hope my comments have been helpful so far.

DAN: I agree with everything you said. I hope that Kay has been accurately diagnosed, but if it turns out to be an episode of CSR, it is probably good that she is no longer taking the drug.

You are correct about the paucity of research in this area. It does not generate a lot of interest, due to the relatively small population which is permanently affected by chronic, progressive CSR (my situation), but I hope more study will eventually be initiated.

Thank you for providing your insight into this subject. You are proving to be one of the best guests we have had so far, and I look forward to hearing more from you during these next few days.

CATHIE: When I was 11 (a year after my central vision started to decrease), I was given mega doses of cortisone. It didn't stop or correct the decreasing vision, so that was stopped. I have occasionally wondered what the long term effects were from taking that medication at my young age. Could it have contributed to the vision loss?

DAN: Cortisone has been named in the research as an inducer of central serous retinopathy. It has not, however, been connected with age-related macular degeneration, juvenile macular degeneration, or choroidal neovascularization. For that matter, it is still considered by many as a treatment for those diseases.

ANGELA: I have emphysema and use a steroid inhaler, Pulmacort. Also, at times I have used a steroid medication in a nebulizer. Should I be concerned about using these medications relative to my AMD? I have dry AMD and cataracts in both eyes.

DR. SONKIN: Although steroids have several side effects, regardless of the administration route (topical, oral, sprays, etc.), I know of no definitive evidence that they have any effect (either positive or negative) on macular degeneration. Some studies are actually underway investigating intraocular steroids for the prevention of wet AMD based on initial basic science studies.

MARTHA: 1. My glaucoma specialist tells me that I will probably never lose my peripheral vision to glaucoma as long as I keep watching it and medicating. I see him every three months, and use Timoptic drops each morning. The pressures stay good. May I assume that I probably won't lose my peripheral vision?

2. What is your opinion about cataract operations for those of us with wet MD?

DR. SONKIN: Great questions! With regards to your glaucoma, I would defer discussion of your particular longterm prognosis to your personal doctor who knows your eyes. In general, though, early detection and appropriate therapy is very very effective at preventing peripheral visual loss. In my patients with cataracts and macular degeneration, I try to explain that it is sometimes difficult to determine how much of the visual loss is from the cataract and how much from the macular changes. Cataract surgery is probably just as safe for patients with macular degeneration as it is for those without AMD, but the visual prognosis is more guarded. I do have some patients that have developed wet AMD after cataract surgery, and this has been suggested to be a risk by some reports in the literature, but it has not been proven. My advice would be to discuss it carefully with the cataract doctor and retina doctor, and carefully weigh the risks and benefits. I also always tell my patients it is ok to wait on the cataract surgery, particularly if they are getting along reasonably well.

DOROTHY: I began developing sight problems over two years ago. I have macular pucker in my left eye and dry MD in both eyes, along with some drusen. I eagerly asked my doctor to operate on the macular pucker, and fix the problem in my left eye, (my worst eye) and he said "no," because of dry MD existing!

I went to another RS for a 2nd opinion. She said, "On the angiogram, it looks like it is less likely to be MD predominantly in your left eye and more likely to be an actual cyst. It could be a foveal cyst. There is no treatment for that, but they tend to not progress. We will have to hope for that."

Shall I seek a 3rd opinion? Why does dry MD make it impossible to operate when one has macular pucker?

DR. SONKIN: It is okay to operate on macular pucker in the presence of dry AMD if it is felt that the pucker is the larger problem. Cysts in the macula can be due to a variety of problems. The pucker can cause a cyst. Prior cataract surgery can cause a cyst. Wet AMD can cause cysts. Retinal vascular occlusions can cause cysts. I usually try to treat the cyst with drops or subtenons kenalog injections. If the cyst is due to a pucker, it usually does not improve much without surgery, but surgery for a pucker can be difficult in the presence of a cyst, because of the risk of unroofing the cyst and forming lamellar holes. A typical pucker without cyst is a straightforward surgery.

GIDEON: Regarding PDT, you said, "It usually takes multiple treatment sessions." From one to how many known, during what duration?

You also said, "Wet AMD not treated can smolder for a long time, but the duration varies greatly." What is known statistically (if at all recorded) regarding the duration?

DR. SONKIN: There are specific numbers quoted in the PDT studies, but on average patients require three to four treatments in the first year and approximately

two treatments in the second year. This varies greatly from patient to patient. There is no data, to my knowledge, regarding retreatment rates outside of the first two years.

With regards to the natural duration of a bleed or leak from wet AMD, there is no way to predict how long active leakage and bleeding will persist before scarring sets in. In my experience, fibrosis and scarring usually would set in after six to eight months, but that was what I saw as a resident and fellow before PDT treatment was available and when we sometimes only observed. Hopefully, there will be fewer patients with disciform scars (macular scarring) now that we have PDT.

GREET: I have been treated for allergic rhinitis in recent years with nasal sprays containing cortisone (such as nasacort, et al), and I always felt that the sprays increased my eye problems. Could this be so, or am I just casting around for an explanation?

DR. SONKIN: Neither AMD nor cataract surgery are associated with symptoms such as yours. The nasal spray is also unlikely to be the cause. I am not calling you old, but normal aging of the meibomian glands, eyelid structures, and the cornea, conjunctiva, etc. often result in symptoms such as yours. Seasonal changes can also cause your symptoms, as well as your allergies. I would recommend you see a cornea/external disease specialist. I have not studied these diseases since I was a resident, and my expertise lies elsewhere. Sorry I can't help more.

KAY: Please explain the disease, central serous retinopathy (CSR).

DR. SONKIN: This is an idiopathic retinal condition in which an abnormality of the RPE (supportive layer under the retina) allows fluid to leak under the retina, causing a serous detachment of the retina. When this fluid involves the fovea there is a decrease in central vision. Fortunately, this condition is usually self-limiting and it resolves on its own. Sometimes we help expedite resolution with some laser treatment. This is indicated only in certain situations, e.g. nonclearing fluid, poor vision in fellow eye from CSR, occupational needs, etc. Unfortunately, there are some cases where CSR causes a permanent reduction in vision. Additionally, CSR often leaves RPE atrophy in the area of leakage, and this can, in rare cases, result in growth of CNV (abnormal blood vessels) much like those of wet AMD.

MARY: Is there any way to correctly diagnose cone dystrophy? My RS is pretty sure that is what I have, but another one in another state disagrees. They don't do ERGs here, but I was wondering if that could tell the difference between a cone dystrophy and early onset MD. They say it is either cone dystrophy, or cone/rod dystrophy. Anyway to tell for sure?

DR. SONKIN: The ERG is the gold standard for diagnosing cone dystrophy. Cone dystrophy and cone/rod dystrophy are really the same thing, with minimal difference. Both are photoreceptor degenerations, and are often "lumped" together.

NORMA: My "rotten" eye flashes quite a bit, depending on lighting conditions or stress. I know flashing might be an indication of trouble, but how can we tell which

eye is flashing? How can we determine that it is not the good eye signaling trouble ahead? Also, I wonder if you might explain to me the difference between classic and occult md - sometimes the terms are confusing.

DR. SONKIN: "Flashes" can be a normal symptom of "normal" changes in the vitreous jelly, but is also concerning for retinal tears, retinal traction, and retinal detachment. Fluid under the macula can also cause flashes. It is true that it can be very difficult at times to determine which eye is causing the symptoms. If you are having new symptoms, the best advice is to see your retina doctor, and have it checked.

With regards to your question about classic versus occult wet AMD: try not to worry about this distinction. It is two different forms of wet AMD that are distinguished by the clinical appearance and angiogram findings. It is pertinent for the retina doctor and patient, in that this distinction helps to predict the natural history and is critical in deciding which, if any, treatment would be of benefit.

SHARON: My question today is with regards to drugs we should avoid or limit. It came to me that our personal physician may not fully understand our needs, particularly if they have not been clearly articulated to the doctor. Blood thinners would be a concern, Also, Vitamin E over 400 mg, and apparently steroids. What else should we be questioning?

DR. SONKIN: Systemic medications can affect the eyes. In theory, blood thinners may make active bleeds worse, but this has never been proven. Also, blood thinners have never been shown to increase the risk of macular bleeding from AMD. I tell my patients that if they need to be on aspirin or other "antiplatelet" medications to reduce the risk of stroke, heart attack, etc., that this should take precedence over the small risk to the eye. For patients on coumadin, it is important to make sure the dose is correct and that the PT is at the appropriate level (this is a measure of how effectively the blood is thinned).

SHARON: I also require a definition, if you would be so kind. What does it mean when they say to the right of the disc? Is this the macula? Can you explain disciform scar please. Is there a relationship between the two?

DR. SONKIN: To the "right" of the macula means nothing unless you know which eye. We also do not use the terms right or left. Instead, we describe retinal location in reference to landmarks (e.g. the disk or macula) as being either temporal or nasal. Nasal means towards the nasal side, and temporal away from the nasal side. "Temporal" to the disk refers to the macula. Disciform scarring is the fibrosis and scarring that develops in the later stages of wet AMD.

STEPHANIE: Do you have any thoughts or hopes for stem cell research/progress in the near future? Do you have any ideas about new things on the horizon in that area of research?

DR. SONKIN: This sort of research holds hopes for the future, but is not clinically applicable at this time. It is directed at trying to restore normal RPE cells in the

macula. There is great research work ongoing, and hopefully it will add to our treatment options in the future.

SUE: Of what practical use is an angiogram or series of angiograms when a patient only has drusen, for which nothing can be done?

DR. SONKIN: Fluorescein angiograms are done to help with treatment, treatment decisions, and sometimes to help with diagnosis. We often get angiograms to check for "wet" CNV growth even if not detected on clinical exam, particularly if the patient is having new symptoms or if the patient has high-risk changes, such as multiple soft drusen, extensive atrophy, irregular RPE, etc. We also sometimes get fluorescein angiograms to evaluate the degree of atrophy (dry changes) and to evaluate the retinal circulation. There is a small risk of allergic reaction, GI symptoms including nausea, etc. There is no risk of permanent damage to the retina.

JACK: My report said: "A fluorescein angiogram confirmed the presence of blocked choroidal fluorescence due to the buildup of lipofuscin within the pigment epithelial cells..." Is that typical for Stargardt's? Or is it some additional complication?

DR. SONKIN: Your FA description sounds consistent with Stargardt's. The FA in Stargardt's has a very typical appearance known as a "silent choroid," where the normally visible choroidal circulation is "blocked" as you described. You also get blocked fluorescence from the "flecks" associated with the disease and the RPE pigmentary changes that can develop in the macula. Ask your retina doctor to clarify at your next appointment.

SHERRY: I have glaucoma and had trabeculectomies in both eyes. The most recent one in April developed hypotony right after surgery due to a couple of small bleb leaks. The hypotony resolved in a couple of weeks and my pressures are now excellent, hovering around 8. I was left with some sort of maculopathy. What questions should I be asking the RS when I see him again?

DR. SONKIN: Low pressure (hypotony) after glaucoma surgery is not uncommon. In some cases, it can result in a "hypotony maculopathy", choroidal folds, choroidal hemorrhages, etc. Hypotony-related changes are treated by bringing the pressure up if it is close to zero. It sounds like your pressure is good now. I would wait and see what the retina surgeon finds on exam, and then make sure you have a good understanding of what the problem is, why the vision is reduced, and whether or not there is any treatment that might help.

TESS: I had a bad experience with a retinal surgeon and that has left me wary of them ever since. I know all retinal surgeons are individuals, that they are only human, and should be judged on their own merits. However, when once has been "burned" it is very hard to give that trust back. Probably, in my case, it helps that my optometrist is a low vision specialist who is very familiar with my condition. We have a very good rapport, as he is honest, down to earth, very understanding and, every so often, is quite direct. I know he has my best interests at heart. I do think it is important to have professionals you feel you can trust. After all, it is your vision

they are dealing with!

DR. SONKIN: Retina surgeons are no different than dermatologist, optometrists, hair dressers, salesmen, lawyers, etc., in that there are good ones and bad ones. Although it is hard not to prejudge based on bad experiences, I can assure you there are more good ones than bad ones. If your optometrist finds a new active problem, don't be afraid to see a new retina specialist. After all, you are always in control of what is done, and if you do not agree or understand what is told to you, you can always get a second opinion. This holds true for all that we do, whether it is a recommendation from a retina doctor regarding treatment, or a recommendation from a hardware store salesperson regarding what type of paint to use. Good luck, and remember that the final decision is always the patient's decision.

DOTTIE: I just received an e-mail from my friend who had a torn retina. I know she had it operated on and now I received this. Could you explain just what is going on? She wrote: "I saw the retina man on October 30th. Would you believe I must have further surgery. When the retina tore, little pieces of it flaked off and that is floating around behind the eye. Also, scar tissue has formed and he said he must go inside the eye to extract the jell and that will remove all that crud that is floating around in there."

DR. SONKIN: Your friend's description can mean several things. When we develop a PVD (vitreous separation), it can pull on the retina and cause a tear. If this is caught when it is just a tear, we "seal" the tear down with either laser or cryotherapy. If fluid gets through the tear and under the retina, this is a retinal detachment, and it requires surgery. After a simple PVD, we can frequently develop a "scar tissue" on the macula that causes a wrinkling or puckering. This is known as a macular pucker or epiretinal membrane (erm). What your friend describes sounds most like an erm that has probably caused a reduction or distortion in central vision. The other possibility is that she has developed proliferative vitreoretinopathy (PVR), which is another form of scar tissue that grows diffusely over the retina, including the periphery. This is much more damaging and can sometimes result in a recurrent RD that is difficult to fix.

DR. SONKIN: I have a macular pucker. Could this have been caused by my cataract surgery? Do I need to ask if I should have surgery to have it repaired?

DR. SONKIN: Macular pucker usually occurs after posterior vitreous separation (PVD), but it can be idiopathic or related to such things as retinal vein occlusion, uveitis, etc. Puckers should be removed surgically only if they are visually significant, and this varies from patient to patient. Many surgeons use 20/70 as an approximate cutoff, but in some patients, 20/40 can be bothersome enough to warrant surgery. It is also important to make sure there is not another problem that is causing the majority of the visual disturbance, e.g. cataract, AMD, etc.

JACK: Does the MD get worse faster if you use your eyes for a lot of detail work like reading, computer use, etc?

DR. SONKIN: Although reading and focusing may cause some "straining," this

does not have an effect on the health of the macula. I would recommend continuing with activities that you enjoy, and rest when needed.

JACK: I just read recently, (on the MDForum I believe), that Stargardt's (recessive type) skips a generation. Is that true even if both parents have the gene and pass it on?

DR. SONKIN: Stargardt's is a hereditary disease usually with a recessive inheritance pattern (some dominant forms reported). Recessive diseases skip generations, and do require both parents to either be affected or carriers of the abnormal gene to pass the disease to an offspring.

JACK: What are your thoughts on what is happening with retina implants?

DR. SONKIN: A friend of mine is at the forefront of research into retinal implants. His name is Dr. Mark Humayan, who was formerly at Wilmer Eye Institute (Johns Hopkins), but recently moved to Doheny at USC. Retinal implants are mostly applicable for patients with total or near total blindness, and potentially provides the ability to see light. It is not clinically available and is still being researched. Fortunately, patients with AMD have much better vision already.

KAY After reading about central serous retinopathy, I decided it could be as bad as MD or worse. Couldn't this be found with the extensive exam the RS gave me? I have signed a form ready to mail asking for my records, which I believe another expert should look at. What is your opinion?

DR. SONKIN: CSR overall has a much much better prognosis than AMD, although there is variability with each condition. If you feel unsure of your diagnosis, there is no harm in seeking another opinion.

DR. STROUSE: Recently, there was a discussion on the MDList about the proper amount of lutein a person with macular degeneration should take per day, and there is a lot of variation in the amounts recommended. Robert Abel, Jr., MD recommends 6mg for several months and then 2mg daily after that. Edward Paul, OD, PhD recommends a minimum of 12mg per day. What amount do you recommend? Have you read other articles with varying amounts recommended?

Also, Dr. Abel recommends taking DHA (the omega-3 fatty acid) which is found mainly in oily fish, such as tuna, mackerel, and salmon. He recommends that his macular degeneration patients take an antioxidant, a separate lutein supplement, and 500mg DHA. What have you read about DHA and what would you recommend to the MDListers?

DR. SONKIN: The only good prospective placebo-controlled study that I know of examining the role of vitamins in AMD is the recently released AREDS study. The benefits were limited to a few subgroups and were not overwhelming. The data is for 5 years of follow-up. Some potential side effects were discussed.

I still recommend a good multivitamin and well-balanced diet. I do not believe there

is enough in the literature to warrant a recommendation other than this to my patients. If a patient wants to take higher doses of vitamins, I try to discuss the potential side effects and the potential risks/benefits, and I leave it up to them. Hopefully we will have more information in the future.

DAN: Many of you have written to thank Dr. Sonkin for his time during this past week, and I want to add my words of appreciation. This has been one of our most active sessions, and I'm sure it is due to his obvious expertise and the concern which he shows. This discussion will remain permanently in our Clinic section for the benefit of future visitors to our site and for continued reference by us. Dr. Sonkin, we greatly appreciate the time you have spent with us.

DR. SONKIN: I have really enjoyed the last two weeks of discussion, and I hope my participation has helped to answer your questions and concerns. I have also learned a lot from our conversations, and I am sure this experience will only help me in communicating with my own patients. I look forward to another session in the future, and I wish you all the best.