

FLORIDA DEPT. OF HEALTH

**Moderator: Natalie Gibson
February 17, 2005
11:00 a.m. CT**

Operator: Good day, ladies and gentlemen and welcome to the Diabetic Retinopathy Conference Call.

Today's call is being recorded.

For opening remarks and introductions, I'd like to turn the call over to Ms. Natalie Gibson, Program Epidemiologist and evaluator for Florida Diabetes Prevention and Control Program. Please go ahead, ma'am.

Natalie Gibson: Good afternoon. Continuing with our series of audio teleconferences, the Florida Department of Health, Bureau of Chronic Disease Prevention and Health Promotion, and the Diabetes Prevention and Control Program welcome Dr. Rulx Ganthier Jr., who will discuss the topic of diabetic retinopathy.

Dr. Ganthier is currently at Highland Eye Institute T.A. in Seabring, Florida. He is a board certified ophthalmologist with the American Board of Ophthalmology, member of the American Society of Retinal Specialists, and currently serves as a gubernatorial appointee to the Florida diabetes advisory council, where he serves as a chairman of the healthcare and professional development committee.

He earned his medical degree from the Medical College of Ohio, served his internship at the Henry Ford Hospital in Detroit, and residency at the King Drew Medical Center in Los Angeles.

Following his residency, he was awarded a fellowship on the topic of diseases and surgery of the vitreous and retina at the Henry Ford Hospital.

In addition, he is qualified and approved to participate in Florida's special expert witness program, is involved in several advisory committees, and has delivered numerous lectures covering such topics as age related macular degeneration and diabetic retinopathy.

Dr. Ganthier has a special interest in improving patient and provider diabetes education and communication. We are honored and delighted to have Dr. Ganthier with us today.

As a reminder, all participants listening in should have already completed the pre-test. In addition, each participant needs to sign in on the participant attendance sheet, complete the evaluation forms and pertinent CEU paperwork.

You may refer to the syllabus, continuing education instruction sheet, and/or sign-in sheet for submission details.

I am now going to give you some important CEU information related to today's call at this time. I ask that everyone please refer to the continuing education credit instruction sheet.

Continuing education credits have been approved for the following healthcare professionals: nurses and dietitians. The Big Bend area health education center has approved this program for 1.5 contact hours. Nurse provider number SBN 2654. This is not a national provider. Nurses and health educators in other states, states other than Florida, must request approval from the professional board in their states.

All CE credits will be reported to Florida's CE broker monitoring system. Nurses who want to receive CE credits must complete the appropriate CEU paperwork and register with the correct license number.

The Commission on Dietetic Registration has approved the program for nationwide Dietetic Continuing Education credits. CPEU level 3, 1.5 major session topic code CL0312. Dietitians who want CE credits must complete the appropriate CEU paperwork and register with the correct license number.

Individuals interested in receiving CEU credits must submit the pre-test, post-test and post-tax survey and register for CEU credits via our Web site.

E-mail confirmations have been sent out to everyone who completed the pre-test online prior to today's date. Dietetic practitioners who are under the professional development portfolio will need to record this activity under step 4, learning activity log.

Credit will not be issued to participants who have not signed in, completed the required forms, and registered for CEU credit. All paperwork must be in our office by Thursday, February 24th. Paperwork will not be accepted after that date.

Dr. Ganthier, I will now turn it over to you.

Dr. R. Ganthier: I'd like to say good afternoon to everyone who is participating. And it is my pleasure to discuss this topic.

I will refer to slides as we proceed through this talk. And in addition to what you see before you in terms of the slide material, I will also add some other relevant points that will not necessarily be visible to you.

Let's start with slide number two entitled "The Healthy Eye." Before you, you see a schematic diagram of an eye that has been cut from front to back. And you're looking at the inside of the eye with the front of the eye being to your right, and the rear of the eye to the left.

And if we take each of the bullet points, you will then learn how vision, under ordinary circumstances, is produced. Light rays from the sun, et cetera, will strike the cornea, enter through the cornea, at which point that light will then be bent toward the pupil of the eye, which is the center most portion of the eye that dilates or constricts to less light or more light, followed by a focusing of that light onto the lens, which, to a greater extent, then focuses the light onto the rear of the eye, onto the retina or specifically the macula of the retina.

At which point, the impulses are then followed through into the optic nerve, which enters the rear of the eye. And this optic nerve will then carry these impulses to the brain, specifically the occipital lobe of the brain, which is at the rear of the brain for those of you who are familiar with brain anatomy, where they are recognized as images.

Keep that in mind. And as we proceed through this lecture, I may make reference to this with regard to its relationship to diabetic retinopathy.

Let us proceed to slide number three. What is diabetes? Diabetes Mellitus is the inability of the body to use and store sugar properly, resulting in high blood sugar levels. This results in changes or damage in the veins, arteries, and capillaries in the body. And although my lecture today will be specifically geared toward a diabetic retinopathy, these changes are also happening

elsewhere throughout the body of the diabetic patient. And that is why, for example, patients who are diabetic can also experience damage to the kidneys, the nerves, the heart, et cetera.

Fortunately or unfortunately, but I'm going to say fortunately with regard to patients with diabetes, at least when they develop damage within the retina, it can be observed with the assistance of an ophthalmic exam by a qualified professional. And we can then act.

But unless that, in fact, has been done, one may not necessarily be clued into the fact that damage is occurring elsewhere, but in a much more silent fashion or more difficult to detect fashion.

The total prevalence of diabetes in the United States in 2002, if you take into account all ages, was 18.2 million people; 5.2 million of which were undiagnosed at the time.

More relevant to Florida information, the prevalence of diagnosed diabetes in Florida as of 2003 for individuals 18 years of age or more was approximately 1.1 million or 8.5 percent of Florida's adult population.

What is even scarier, however, is that somewhere between 350,000 to 450,000 others have this diagnosis but are undiagnosed. And we're talking about just in the state of Florida.

Slide four, how does diabetes affect vision? It can affect it in a multitude of ways, some of which we will briefly chat about at this time. The first is that the patient could develop a cataract. And what is a cataract?

Well, a cataract is a clouding of the naturally clear lens in the eye. When we are born, assuming we don't have an abnormality that is going to produce the congenital cataract, and assuming we don't experience some other noxious stimuli, such as radiation exposure, prolonged steroid use,

especially systemic steroids, it will be not until we reach our senior citizen years where one will start to develop a clouding of this naturally clear lens.

But in the diabetic patient, they are at greater risk of developing the cataract earlier in life. And in particular, the diabetic patient may – is more apt to form a particular type of cataract called a posterior subcapsular cataract.

And there are different types of cataracts. But a posterior subcapsular cataract is one where typically the symptoms the patient may report are that although during the day their vision seems OK, they have much greater difficulty at night handling the glare produced by oncoming headlights more so than in the past.

That may be a way in which the patient is unknowingly revealing to you or whomever is listening that they are in fact developing a cataract.

Another way in which that particular type of cataract can affect the vision of a patient is that although they may feel that their distance vision is good with their glasses, of course, that they have a much greater difficulty reading up close. That is another way in which that particular type of cataract can manifest itself.

Secondly, may develop Glaucoma, a disease of the optic nerve. Glaucoma is a condition where – and our understanding of glaucoma is changing and I'll comment on that shortly, but generally speaking, we like to consider glaucoma a disorder where the inter ocular pressure, or the pressure within the eye is at an unhealthy level, typically elevated.

Normal eye pressure runs – is generally thought to run between 10 and 21 millimeters of mercury. And 90 percent of the time, that is an effective marker or index to be aware of as to whether the patient's risk of developing diabetes is heightened or not.

That is, and people whose pressures are in excess of 21, are going to have a greater risk of developing glaucoma than those whose pressures are between 10 and 21.

But I will add a caveat that approximately 10 percent of patients who have glaucoma have what we call normal pressure glaucoma, which means their eye pressure will not be beyond 21, and will in fact be between 10 and 21. And they too also have glaucoma. And so that's why I said 90 percent of the time, because it is not reliable 100 percent of the time.

That is where the expert case of the ophthalmologist will come to play because as I have just stated, 10 percent of the time, it will not be – even though the eye pressure is not elevated, that the patient will have glaucoma.

And in this country, there are approximately 3 million people who have glaucoma. So 10 percent of those people represents 300,000 people that can have glaucoma and may not be aware of it.

And the sneaky thing about glaucoma is that it does not typically produce symptoms whatsoever, referring to chronic open angle glaucoma, which is the most common type of glaucoma.

Thirdly, the risk of developing diabetic retinopathy, this is damage that occurs to fragile blood vessels within the retina, we'll go into much greater detail about this in a moment or throughout the remainder of the lecture.

But just keep in mind that when patients develop diabetic retinopathy, they have damage to their blood vessels, specifically in the retina. And if you recall from the diagram from our very first slide on the healthy eye, the retina was at the rear of the eye.

We will now proceed to slide number five. Epidemiology related to diabetic retinopathy, I always find that giving statistics is a very powerful way of giving a message in terms of the significance and the prevalence of any given disorder.

Bullet number one, each year between 12,000 and 24,000 people lose their sight because of diabetes. Most commonly, they're diabetic retinopathy.

In particular, diabetic macular proliferative diabetic retinopathy, which I will go into greater detail as we proceed through this lecture. Diabetic retinopathy is the most common cause of new cases of blindness amongst adults between the age of 20 and 74 years of age.

The most common cause of new blindness in this country. By the way, the most common cause of blindness in people beyond this age group would be age related macular degeneration, which we will not discuss during this particular lecture.

During the first two decades of disease, nearly all patients with Type I diabetes, and over 60 percent of patients with Type II diabetes, have retinopathy. Type II diabetics are typically referred to as adult onset. Type I diabetes as juvenile.

This Wisconsin epidemiologic study on diabetic retinopathy, which is what WESDR stands for, demonstrated that Type I patients experienced a 25 percent rate of retinopathy after five years disease, and 80 percent at 15 years of disease.

Up to 21 percent of newly diagnosed Type II patients, that is the adult onset, have some degree of retinopathy at the time of diagnosis. The significance of this last bullet point I will make reference to as we proceed later in the lecture.

Slide number six -- diabetic retinopathy. I have here before you listed two types of retinopathy.

The first of which is non-proliferative diabetic retinopathy or NPDR for short and proliferative diabetic retinopathy, or PDR for short.

For this audience that I'm speaking to today, I elected to only break it down into two stages, although as a retina specialist and a purist, there are actually – we like to think of three of them, but the distinction is so subtle and is not a practical point for the scope of today's talk.

The proliferative diabetic retinopathy is the most advanced stage of retinopathy and is a common cause of new blindness.

Slide number 7. Non-proliferative diabetic retinopathy is also called background diabetic retinopathy. It is the earliest stage of diabetic retinopathy, characterized by damaged blood vessels in the retina that leak extra fluid and small amounts of blood into the eye.

At the same time, it is not only blood and fluid that may exit the eye, but also cholesterol or other fat deposits, such as lipids from the blood called hard (\exudates that may leak into the retina.

In the slide within a slide at the top that shows you the appearance of a macula and an optic nerve of the patient, the blood vessels we refer to correspond to what appear to be branching tree limbs emanating from that disk or circular object to the left. And as they – as these blood vessels arch away from the disk, which is the optic nerve, by the way, the tissue that is found inside that – the limits created by the branching blood vessels is referred to as the macula.

And the closer the patient's damage is to the center of the macula, the more greatly affected the patient's vision will become. In the bottom – in the slide just below that one, you will see a slide of a retina in which there has been damage consistent with non-proliferative diabetic retinopathy.

The hard yellowish, yellow whitish, let's put it, deposits that are more easily seen refer to the hard exudates, which if you recall, correspond to cholesterol and other fats.

The presence of the hard exudates from a prognostic standpoint will put this patient at greater risk of avoiding clinically significant loss of vision, as compared to a patient who also has non-proliferative diabetic retinopathy, but does not demonstrate hard exudates.

Slide number 8. With non-proliferative diabetic retinopathy, the central vision is affected by any of the following means. One; hard exudates on the central retina or macula, which you saw an example of on the prior slide. Number two; microaneurysms, which are small blood bulges in blood vessels of the retina, that often leak. Three; retinal hemorrhages, tiny spots of blood that leak into the retina.

Four; macular edema, a swelling or thickening of the macula that is a result of everything above it, at least one component or possibly all three of the features listed above macular edema. And number five, macular ischemia, closing of small blood vessels and/or capillaries.

Now as far as we have come in terms of our understanding of diabetic retinopathy and how to manage the patient with diabetic retinopathy, the first four features are those for which we do have therapeutic approaches that have been proven to be effective.

However, feature number five, macular ischemia, is one in which we do not have a solution for thus far. It is an irreversible component of visual loss, regardless of the patient's stage of retinopathy, that is, whether they have non-proliferative diabetic retinopathy or proliferative diabetic retinopathy, if they have macular ischemia, that will have a rate limiting effect on one's final vision despite therapeutic intervention that is undertaken.

So in a patient that has purely features one through four, their chances of avoiding clinically significant loss of vision or perhaps even improving vision is better from a prognostic standpoint than a patient who has a significant component of macular ischemia.

This – to give you an analogy, the ischemia that leads – that exists in a patient's macula, that results in a – in loss of vision is the same pathology that trans – that takes place in the peripheral circulation of the diabetic patient, which is what would lead to the need for amputation of toes, feet, and sometimes the leg of the diabetic patient.

Fortunately, however, when it comes to the eye, there is no need for a nucleation or removal of the eye for that particular cause. There is a singular indication for that. However, this is not it.

Next slide, slide number 9. With regard to the potential features of non-proliferative diabetic retinopathy, macular edema is a macula that has thickened or has swollen, thereby affecting vision. It is the most common cause of vision loss in diabetics. The vision loss may be mild to severe.

A patient with macula edema can have 20/25 vision and in some cases even 20/20 vision if you catch it very, very early, but can be so severe that the patient can have – cannot even have vision on the eye chart. And the largest letter on the eye chart is equivalent to 20/400.

So 20/20 is perfect. 20/400 is very poor vision. And if you keep in mind the size or the magnitude of that second number is an indication of the patient's level of vision.

So a patient who has 20/20 vision has better vision than the patient with 20/40 vision, is better than the patient whose vision is 20/80, is better than 20/200, is better than 20/400, et cetera.

The peripheral vision remains unaffected. But as you recall, the macula is responsible for this sharp central vision, which as you recall from slide number one of the healthy eye, is where the light rays ultimately are focused onto, as they enter the eye.

Laser treatment may help stabilize vision. If clinical significant macular edema is present, color fundus photography, fluorescein angiography, and laser photocoagulation are indicated. As I mentioned earlier, this macula edema may occur at any stage of retinopathy.

CSME or clinically significant macular edema is terminology used to describe swelling that takes place within the macula as a result of the macular edema, and then a retina specialist is appreciated during the course of our examination typically using the 90 diopter lens or some other contact lens, where we visualize and scrutinize the anatomy of the patient.

Macular edema can be present at this stage and is defined as retinal thickening within 3000 microns of the fovea. Clinically significant macular edema occurs when edema threatens the center of vision or is within 500 micrometers of the fovea.

If laser photo coagulation is not performed, the patient should be carefully monitored and examined approximately every three months for progression. In other words, I may see a patient, and I've identified that they have non-proliferative diabetic retinopathy affecting one or probably both eyes at that time. And they demonstrate micro aneurysms or some small hemorrhages, but the macula has not begun to swell or thicken.

And that patient, I would not yet offer laser surgery and/or other adjuvant approaches. But rather, I would see the patient that closely, perhaps two, three, four months later, rather than a year later and re-evaluate the macula.

There has been no verified, scientifically strong study to indicate that treating the patient prior to the development of the macular edema is helpful with regards to preventing loss of clinically significant loss of vision.

However, at the earliest point that that has developed, the patient will be better off. A way in which I can illustrate this point is the following:

Let's say the next patient I examine had the changes in the macula, producing macular edema or swelling and laser surgery was recommended. And at that point, the patient's vision, let's say, maybe 20/30 as an example.

If for any reason there is a prolonged delay in administering what needs to be done, then – and let's say the patient shows up six to 12 months later, well that patient in all likelihood will have continued to lose some more vision, will have lost more vision, and may perhaps be a 20/40, 20/50, maybe even 20/60 months later.

And if we were to then act, rather than having acted months earlier, instead of trying to salvage 20/60 vision, had we acted earlier, we would have been in a better position to salvage 20/30 vision. That's why it's so important.

Slide number 10. Macular ischemia. If you recall macular ischemia is a condition that I referred to, where there is actually closure of blood vessels. Small blood vessels or capillaries close, blurring the vision.

Macula no longer receives enough blood to work properly. Currently, no effective treatment for macular ischemia exists, which I mentioned here earlier.

Sometimes the macular ischemia is quite evident. And sometimes it is very subtle. And in the subtle cases, subtle to the retina specialist that is, this is where fluorescein angiography, which I will refer to later, is an especially helpful diagnostic modality.

By the way fluorescein angiography is a technology where we inject a patient intravenously with a dye substance called fluorescein. It is a vegetable dye. And this dye passes through the patient's circulation. And within 11 to 12 seconds has reached the circulation of the retina.

And with the use of a special retinal camera, we then take pictures over the course of the next 15 minutes – 15 to 20 minutes of both eyes. And this helps. This can be a very helpful modality in picking up things, such as macular ischemia. Or in cases where we're not certain of whether the patient has other features that I would be concerned with, helps those features to be revealed or ruled out, which will in turn assist with the management of that particular patient.

We will now move to slide 11. PDR or proliferative diabetic retinopathy. If you recall, I mentioned that this is the most advanced stage of retinopathy, whether one is counting retinopathy as having two or three stages. And these, along with a diabetic macular edema, are the most frequent causes of blindness in the diabetic patient.

In this case, in this stage of retinopathy, abnormal blood vessels begin to grow on the surface of the retina, and/or optic nerve. As a result, it cannot provide the retina with normal blood flow.

Proliferative diabetic retinopathy can cause severe visual loss and other serious complications, such as neovascular glaucoma and loss of the eye.

I will comment on bullet points two and three in a moment. But for the time being, I'd like you to look at the slide – at the top upper right hand portion of slide number 11. And that is a slide at a

normal appearing retina or macula in particular. And beneath it is a slide of a retina with proliferative diabetic retinopathy and neovascularization.

Now you might wonder what is neovascularization? Neovascularization means new blood vessels. Neo means new. But because they are abnormal, these blood vessels themselves will start to bleed and leak. And that's opposed to the small blood vessels that leak, that produce diabetic macular edema. The bleeding that can result from these neo vessels can be quite profound.

So profound, in fact, that it can fill up the entire back of the eye with blood, preventing the ophthalmologist or retina specialist from being able to observe the retina, to even know, well, you can have an idea of what led to it, but unfortunately, will not permit things such as fluorescein angiography in terms of informational help. And administration of laser surgery is sometimes prevented because of the presence of this – vitreous hemorrhage that results from this proliferative diabetic retinopathy.

The reason the neo vessels develop is that in the patient with diabetes, in this – since we're discussing diabetes, but the same can hold true for other conditions, such as systemic hypertension, can produce similar consequences.

The retina, due to closure of blood vessels, and loss of blood due to blood leaking from other blood vessels, will say to itself that it's not receiving enough blood, and therefore oxygen, in order to conduct its biological function.

And so what then happens is that abnormal blood vessels are produced, which is abnormal and themselves bleed, and the blood is not really assisting in getting to where it needs to go, which is to the retinal tissue itself. And so, more abnormal blood vessels will form. And it can become a vicious cascade, which can ultimately result in a blind and painful eye.

We'll now move onto slide 12. With PDR, vision is affected when any of the following occur. Number one, vitreous hemorrhage. I mentioned this earlier. When these neo vessels develop, they can bleed quite profoundly. And that bleeding takes place just in front of the retina, or into the vitreous. And the vitreous is the jelly like portion of the eye, that exists immediately anterior or in front of the retina.

And it is within this vitreous, by the way, that sometimes patients complain of seeing floaters, something that's floating. Well, in the non-diabetic patient, that may indeed be a true floater, which is an innocuous aging related change that takes place. However, in the diabetic patient, instead of it representing a floater, it may in fact represent blood from the vitreous hemorrhage.

New abnormal blood vessels bleed into the vitreous gel in the center of the eye, preventing light rays from reaching the retina. Bullet number two. Traction retinal detachment. New abnormal blood vessels begin to shrink and tug on the retina, which may cause the retina to detach.

And thirdly, neovascular glaucoma. Neovascularization occurs in the iris or the colored part of the eye, causing pressure to build up in the eye, damaging the optic nerve.

With regard to vitreous hemorrhage and traction retinal detachment, we have Pars Plana Vitrectomy, which is a type of surgery where we enter through the white part of the eye, after making three small incisions. And with the use of various instruments, including the light source, we remove the blood that's in the vitreous or vitreous hemorrhage in other words. And then with a very fine instrument, we peel away or cut free this scar tissue from the surface of the retina, so it can lay down again and not be pulled off the back of the eye.

And at the same time, we frequently will administer what we call endo laser, which means basically we're giving more laser treatment to the eye now that the blood has been cleared, which we would have done if there was adequate visibility prior to that point.

Bullet point number three. If you recall, I mentioned – I described to you what glaucoma is. But in my previous comments about glaucoma, I did not specify neovascular glaucoma.

Neovascular glaucoma is a particularly bad type or form of glaucoma, which is less amenable to treatment than garden variety, chronic open angle glaucoma.

If you recall, neovascularization, the neo vessels grow. And in this case, they not only grow in the back of the eye, but they're growing in the front of the eye. In this – and when this develops, is when this in itself will lead to elevation of the eye pressure. And then the patient has this neovascular glaucoma.

Although we have medications, typically in the form of eye drops, as well as some systemic medication that can also be taken, to reduce eye pressure, which is the goal in all glaucomas, and is typically sufficient in achieving eye pressure normalization in the patient with garden variety of glaucoma, in the patient with neovascular glaucoma, although we will utilize the medications I just mentioned, for this particular type of glaucoma, the key to success for treatment is to being able to perform laser surgery on that retina in the form of something called PRP for short or pan retina photocoagulation, which I'll go into shortly.

But keep in mind in these very same patients, they may have an eyeful of blood in the vitreous hemorrhage, which will preclude you from being able to do the PRP laser surgery, which will then necessitate doing the Pars Plana Vitrectomy surgery that I referred to, with regard to handling problems one and two, the vitreous hemorrhage and the traction retinal detachment.

Suffice it to say that the patient is in a very, very bad position when they develop neo vascular glaucoma or any of these problems on slide number 12.

Let's go to slide 13. Diagnosing diabetic retinopathy. In that mini slide there to the right, we see a photograph of an ophthalmologist looking through some binocular eye pieces through the slit lamp device, s-l-i-t into the patient's eye on the other side of the slit lamp.

And with use of various lenses and light sources, we're able to get a scrutinizing view and evaluation of the eye from front to back.

Diabetes can cause vision in both eyes to change, even if retinopathy is not present. Rapid changes in blood sugar alter the shape of the eye's lens. And the image on the retina will become out of focus.

One can reduce episodes of blurred vision by maintaining good blood sugar control. For those of you out there who deal with a diabetic patient, this is actually – both points number two and three are comments you have probably heard on many an occasion with your diabetic patients.

For example, the patient may say that my vision was blurred this morning, but this afternoon, it was much, much sharper. If you are able to correlate, and assuming information is available, what the patient's blood sugars were during those times when their vision was reported as being blurred versus when it was reported as being much sharper, there is a strong possibility that correlation will demonstrate high blood sugar with the patient's vision was reported as blurred and a normalization of the blood sugar when the vision was reported as being extremely sharp.

Now I will add that extremely low blood sugars also will result in blurred vision. But of course, if it gets very, very low, the patient may in fact proceed into a diabetic coma, which of course is a much more serious complication of their disease.

Another clinical point I can give you that sometimes is appreciated when I'm examining a patient, it is not uncommon that I will see a patient in whom they do not yet carry the diagnosis of diabetes. And the operative word there was yet.

And the patient may, in fact, not demonstrate diabetic retinopathy at that point. And the patient may not even say to me that they have big swings in their vision being blurred versus sharp, as I referred to earlier.

But the patient gives us a handful of glasses, all of which have been prescribed by one or more other people over the course of the previous one to two years.

Not uncommonly, the basis for that situation is the patient is diabetic and does not realize it. So – and there are other clinical pearls I could give you, but that would extend this talk for beyond the time I have permitted to share with you today.

But this, too, is as far I'm concerned, is a clue that this patient may in fact be diabetic because what happens is – what's happened in that particular situation is that the patient initially had their glasses evaluated and were prescribed and given. And initially their vision was clear or on that day it was clear.

And then a few months later, they discovered that they're not seeing quite as sharply, but of course they didn't know they were diabetic. And now their blood sugars are higher. And therefore, that same prescription will not be as helpful as a new one ended up being and so on and so forth and so on and so forth.

And the reason why it results in multiple refractions and results in multiple glasses. This is another clue that this particular patient might be diabetic.

Let's go on now to slide number 14. Diagnosing diabetic retinopathy. And people with diabetes should see their ophthalmologist immediately if they have visual changes that one, affect only one eye; two, last more than a few days; and three, are not associated with the change in blood sugar.

Bullet point number two, it is important that blood sugar be consistently controlled for several days prior to seeing the ophthalmologist for an exam. Uneven blood sugar causes a change in the eyes focusing power, interfering with the ophthalmologist measurements. This is a reflection of what I had just finished discussing with you.

And by the way, getting back to bullet point number one, certainly if one – the vision in one eye is affected, the patient should be evaluated by the ophthalmologist. But it does not mean that if the patient experiences some change in vision in both eyes that they should not either. I would make that either or both.

Let us now go on to slide number 15. For those diagnosed with diabetes, excuse me, when to schedule an eye exam. For those diagnosed with diabetes, excuse me, when to schedule an eye exam. For those diagnosed with diabetes at 30 years of age or younger, or younger than 30 I should say, the first exam should be within five years after the diagnosis is made. We like to think of this as the juvenile group, but certainly one who's 21 is considered an adult legal in this country.

For those diagnosed with diabetes that are greater than 30 years of age, the first exam should be within the first few months of diagnosis being made. And actually, when I recommend to my primary care colleagues is, you know, while everything is fresh and new, while the diagnosis has just been made, that they should go ahead and make that recommendation at that point, because

one may not necessarily recall to do it down the road, but while it is fresh on their mind that the diagnosis has just been made, that would be the prudent thing to do for the patient's benefit.

Pregnant women should have an exam within the first trimester. That is certainly accurate to say. However, if a patient has a strong family history of diabetes, or in whom they have been labeled, let's say, a borderline diabetic for sometime prior to the goal of that particular lady to become a mother, it would not be a bad idea to have your exam just prior to that for those of you who are planning oriented.

For those who have experienced a high risk condition, such as kidney failure or amputation related to diabetes, schedule an eye exam immediately.

What happens during an eye exam? The ophthalmologist will dilate the – I'm sorry, slide number 16. The ophthalmologist will dilate the pupils and examine the retina with special instruments using bright lights.

Fluorescein angiography. A diagnostic procedure using a special camera to take photographs of the retina after a small amount of yellow dye or fluorescein is injected into the vein of the arm. Fluorescein angiography typically requires the expertise of a retina specialist.

The photographs of fluorescein dye traveling throughout the retinal vessels show which blood vessels are leaking fluid, how much fluid is leaking, how many blood vessels are closed, and whether neo vascularization is beginning, which if you recall I told you the important feature in categorizing a patient's retinopathy.

Slide 17. What happens during an eye exam? Fluorescein angiography helps the doctor determine why the vision is blurred, whether laser treatment should be started, and where to

apply the laser treatment. Or it can act as the map in guiding the laser treatment most commonly for the patient with diabetic macular edema.

Slide 18. Another diagnostic tool that is helpful is ultrasound. Ultrasound is using sound waves to give anatomical information. If the ophthalmologist cannot see the retina because of the vitreous hemorrhage, for example, an ultrasound test may be done in the office. The ultrasound "sees", and that's in quotes because it actually doesn't see, through the blood to determine if the retina has detached.

If there is detachment near the macula, prompt surgery may be necessary. After evaluation, the ophthalmologist/retina specialist will decide whether to treat or re-examine the patient.

Slide 19. Treating diabetic retinopathy. The best treatment is to prevent development of retinopathy as much as possible. An example of this is the information that was derived from the diabetes control and complications trial or DCCT study, which demonstrated that strict control of blood sugar levels will significantly reduce the long term risk of vision loss from diabetic retinopathy.

Laser surgery is often recommended for people with macular edema, proliferative diabetic retinopathy, and neo vascular glaucoma.

I'm going to make an additional comment regarding the DCCT study. As you know, this was a long term study that was originally slated to last 10 years, where they were evaluating young diabetic patients and are basically two categories, one in which there was strict monitoring and control of the blood sugar typically with insulin or the use of insulin pumps. And this was on a, you know, throughout the day, every day, versus what we call conventional control, where they would check the blood sugar once a day and recalculate what they needed to do just on that frequency and not more intensively than that.

And what the results of the study revealed, which by the way was a – the study was performed at 29 centers in the country at that time – the Henry Ford Hospital being one of those 20 – was one of those 29 centers where I happened to be a retina fellow at the time.

And what we found was that the patients that were under the strict control category had a 60 percent lesser chance in terms of severity, and existence of diabetic retinopathy, nephropathy, which is kidney damage, and neuropathy, which is nerve damage from their diabetes, as compared to the group that did not have strict control.

And the results were so profound, that the study was discontinued after seven years, rather than running it throughout its predetermined course of 10 years, because we felt that it was unfair to the patients that were not in the strict category to continue in that vein, knowing the great benefit that existed for the patients that were – had this good tight control.

Now what does tight control mean? Well, for the purposes of the study, the cut-off was 150, meaning in patients that were consistently able to control their – keep their blood sugar below 150, key word being consistently, that those patients fared better, much better than those that were unable to achieve that.

One thing I do need to keep in mind, however, is that during the study, that there was a three times – three fold greater risk of the patients developing a diabetic coma as a result of that strict control. And that's not because their blood sugars were 120 or 110 or even 90, but we're talking about patients who, for example, overestimated the insulin they required, sending them, you know, well, well, well below 50 at times resulting in the coma.

But nonetheless, the message is the same that having consistent control of the blood sugar is better for you – better for the diabetic patient than not having consistent control. You know, what

number you should shoot for is – the diabetic should try to achieve as something they have to be under the guidance of their primary care physician with regards to those specifics.

Slide number 20. Laser surgery for macular edema. Laser is focused – and laser by the way is fine light energy that is focused – laser is focused on the damaged retina near the macula to decrease fluid leakage. Some may see laser spots near the center of their vision following treatment. Usually, however, this will fade with time, may not necessarily disappear completely.

But typically it is not a problematic thing for patients after three to four months have passed. Uncommon for people who have blurred vision for macular edema to recover normal vision, although some may experience partial improvement.

Main goal of treatment is to prevent further loss of vision. I'd like to refer to studies, sound scientific studies when I'm making my recommendations. And I'm going to refer – I'm going to make reference to another study that was also important as the DCCT study was an important study, called the ETDRS or the early treatment diabetic retinopathy study.

This was a study that gave us the scientific knowledge to know how to manage the patients with macular edema, that the best treatment is this laser surgery that I'm referring to here.

In this study, in patients that have this diabetic macular edema, half of the eyes enrolled in the study did not undergo the laser treatment and half did. And what we found was, while following those patients, that eyes that did not receive the laser treatment were at much greater risk of developing clinically significant loss of vision, compared to the patients who did – compared to the eyes that did undergo laser surgery.

The definition of clinically significant loss of vision is equivalent to up to six lines on the eye chart. So in other words, if a patient has let's say 20/40 vision, and they underwent the laser surgery, success is defined as helping the patient keep that 20/40 vision.

And they have a much greater chance of being in that position or having that experience if they undergo the laser surgery. In the patients who, for whatever reason, do not undergo the laser surgery, then they are at much greater risk of that vision falling from 20/40 to 20/400 up to that level in terms of loss of vision.

And as I mentioned to you earlier in this lecture, that once this diabetic macular edema is present, the earlier that the intervention or the therapeutic intervention is performed, the better level of vision really we'll be trying to salvage for the patient.

OK, now we're going to go onto slide 21. And I mentioned several slides ago to you that for proliferative diabetic retinopathy, the treatment is PRP for short or Pan Retinal Photocoagulation.

Laser is focused on all parts of the retina, except the macula. This is, and if you recall, the macula is the portion of the retina that's treated for diabetic macular edema, however, it is – the retina outside of the macula that is treated for this particular diabetic retinopathy problem.

This Pan Retinal Photocoagulation treatment causes abnormal new vessels to shrink, which often prevents them from growing again. Treatment decreases the chance that vitreous bleeding or retinal distortion or detachment will occur. Multiple laser treatments over time are sometimes necessary.

And in the mini slide within the slide, you see arrows pointing to corresponding areas of this – of the retina of this particular patient that have been treated. And the double arrows at the top

demonstrate what appear to be a circular, pretty whitish lesion or lesions because there are many there as you can see.

When they're as white as this, this tells me and the retina specialist that this patient has recently undergone this laser treatment, like within the past few weeks.

Whereas the portion of this line that shows where there's a single arrow, when if you look at these round lesions, these are much darker. And this would correspond to laser treatment in someone who is – had – prior laser treatment much longer – much at a time much more distant in the past, such as few months to perhaps years.

We will now move onto slide 22. Pars Plana Vitrectomy surgery for advanced proliferative diabetic retinopathy occurs when the vitreous or gel like substance in the middle of the eye fills with blood. Performed in the operating room, this microsurgical procedure involves removing the blood filled vitreous and replacing it with a clear solution.

This often presents further bleeding by removing abnormal vessels that cause the bleeding. Multiple laser treatments over time are sometimes necessary. Diabetic retinal detachment requires cutting and peeling of the fiber vascular tissue or scar tissue as mentioned earlier.

Slide 23. There are some take home messages here that we'll go into greater detail in a moment. Bullet number one, one can significantly lower the risk of vision loss by maintaining strict blood sugar control. And this is reflected by the scientific information derived from the landmark diabetes control and complications trial.

Number two, treatment does not cure diabetic retinopathy, but it is effective in preventing further vision loss. When I see patients that, for example, have in stage or proliferative diabetic retinopathy or neo vascular glaucoma, and I'm starting to treat them, the patient at some point

along the way may comment to me that they now have good control of their blood sugar, and it's not all over the place, nor is it high all the time.

And I congratulate them. And I encourage them to continue with the good work in that vein. And that what they're doing now, or what they're achieving now, if they're able to continue that on a consistent basis, it will help slow down the progression of future complications.

But what we are treating now, should that patient be someone with these end stage problems, whether it's retinopathy, neuropathy, or nepropathy, that it will not have an impact on that problem that we're dealing with right now. But I always encourage them to keep up the good work, because you want their confidence to be up, to try to make an effort. And it has been proven that that is scientifically a valid goal to try to achieve.

Most – bullet number three, most people with diabetes retain normal sight. Total blindness is very uncommon if retinopathy is treated in a timely manner. Now I don't see in a timely manner there, nor do you, but I'm mentioning to you that in a timely manner is an important point.

I will give you a real life clinical example of this point. Approximately eight years ago, I had the opportunity to treat a young man who was a Type I diabetic. He was approximately 30-years old. He had diabetes for at least 15 or 16 years at that point. And when I first saw him, his vision was about 20/25 both eyes. But he noticed some floaters that he was concerned about. So he came in to see me. And after examining him, the floaters he had corresponded to a vitreous hemorrhage he had in both eyes. It was not a significant vitreous hemorrhage, but a mild one, mild enough that we didn't have to proceed to the vitrectomy right away. The visualization was adequate enough for me to perform PRP. And I did this PRP session, one session, to the retina of both eyes.

And I – as I am with all my diabetics, as I was with this particular patient, indicated that he would need to have frequent follow-up and re-evaluation because although I've given him this laser surgery at this point in time, that it will take time to determine whether or not his neo vessels will start to shrink or not.

In other words, we give the laser treatment. We typically follow the patient for three or four months, see what happens to the neo vessels. If they start to shrink, we can sit and wait. If instead, there is greater proliferation, there is no sign that the regression is starting, then the patient will require more PRP.

And I guess the patient – I say I guess because I don't know exactly what he was thinking, but he then became lost in follow-up to me for about 2.5 years.

Keep in mind the vision was 20/25 when I first saw him. Well, when he came in – when he made contact with my office and he came in to see me 2.5 years later, he now had light perception vision in both eyes, which means he couldn't see any letters on the eye chart, he didn't even have hand motion vision. He could just tell if I shined a light in his eye or not.

And the reason for that was he went on to develop the traction retinal detachment the vitreous hemorrhage neo vascular glaucoma that he was at risk for because of the proliferative diabetic retinopathy.

I did all that was humanly possible to help him. He unfortunately remained, you know, continued to be blind in one eye. However, I was able to restore his vision in the other eye to 20/60, which under the circumstances was quite a good – an exceptional outcome.

But he then went on to develop kidney failure, requiring dialysis. And then he died shortly thereafter. So that was very unfortunate for such a young person to have those experiences. But

when I spoke with him after he returned to me 2.5 years later, what he stated was that he thought that he was doing OK because his vision was good.

And the reason I bring this up is that just because a patient has good or even decent vision does not mean that they don't have problems. And they will require the expertise of the ophthalmologist or retinal specialist to answer the questions how – what is the status of that patient's eyes?

Regular visits to the ophthalmologist will help prevent vision loss. The human body is very dynamic. And although we have spent the majority of this time talking about one's blood sugar, because this topic is on diabetic retinopathy, I will mention to you that a couple other systemic features are also important.

That is, that patients maintain normal blood cholesterol levels, that patients do not smoke, and that patients maintain good blood pressure control.

For if any of what I just mentioned are present, in other words, if a patient smokes, if their cholesterol levels are elevated, if their blood pressure is not under control, then the severity and the progression of their diabetic retinopathy and its complications will be made worsened, which translates into visual prognosis, et cetera, by the co-existence of these other systemic factors.

It's been my pleasure to discuss diabetic retinopathy with you today. I believe at this point Ms. Gibson will be stepping in. Ms Gibson?

Natalie Gibson: Thank you, Dr. Ganthier. At this point, we can conduct the question and answer session.
Operator, would you please assist?

Operator: Thank you, ma'am. The question and answer session will be conducted electronically. If you would like to ask a question, please indicate so by pressing the star key followed by the digit one on your touch-tone telephone. If you're using your speakerphone, please make sure your mute function is turned off to allow your signal to reach our equipment. We'll proceed in the order you signal us. And we'll take as many questions as time permits. Once again, please press star one from your touch-tone phone to ask a question. If your question has been answered, you can remove yourself by pressing the pound key. We'll pause for just a moment to assemble the roster.

The first question will come from Debbie Klinger of Hartford County Public Health.

Debbie Klinger: I have a question about the use of retinal cameras for screening purposes. Your opinion or any experience with that?

Dr. R. Ganthier: Assuming you're referring to fundus photography, where fluorescein is not utilized?

Debbie Klinger: That's correct. Just a screening kind of picture.

Dr. R. Ganthier: In cases where the abnormality, if there is one, is pretty – is florid, that can be helpful if one does not have other resources available.

However, in cases where it is very subtle, for the non-trained eye, that will not necessarily be a helpful tool. I don't know the availability of a retina specialist in your area, but if you don't have that, then that would be a reasonable thing to entertain. But once again, it will be difficult to detect subtle changes, however, when there are gross changes, some of which, for example, could be seen on – if any of them had the appearance of the slides that you saw during the course of the lecture, then even to the person who's not a retina specialist, he might not know

exactly what's going on, but you can tell that something's not right and then make the appropriate referral.

Debbie Klinger: Thank you very much. I have one more question.

Dr. R. Ganthier: Sure.

Debbie Klinger: And that's in reference to the incidence of nepropathy, which closely coincides with the incidence of retinopathy, so with the Type I patient or maybe even the Type II patient that is developing retinopathy, is there any plan to discuss that correlation of the kidney damage, which often happens along with the eye damage?

Dr. R. Ganthier: What I will tell you is that, and I don't know if the Department of Health has any plans on scheduling such a talk in terms of the correlation between retinopathy and nepropathy and neuropathy, which is I think is what your question is about.

But what I can tell you is that when I see a patient who has retinopathy, there's something that you do automatically. If the patient is under the care of a primary care physician, and doesn't know that the patient is diabetic, which happens more often than you might imagine, I will make contact with the primary care physician and share with the primary care physician my observations and my appreciation of this particular patient's problems. And that they need to be followed up with by their primary care physician.

If it's a mild retinopathy, meaning I've identified the very beginning of retinopathy, or this isn't retinopathy at all, I won't go any further than that.

If, however, the patient has advanced retinopathy, or consequences beyond that, or consequences of that, I will not only make sure that the primary care physician knows, but I will

also make – will give referrals to the patient to see a podiatrist. And I will ask that the primary care physician make arrangements for the patient to be seen by a nephrologist.

Now if the patient, you know, during their evaluation, but with their primary care physician, can appreciate neuropathy, then the primary care physician will sometimes deal with that themselves, or they may choose to send the patient onto a neurologist. But I let them make that call.

Debbie Klinger: Thank you very much. We have one more question, Dr. Lovette.

Dr. Lovette: Hello, sir.

Dr. R. Ganthier: Hello, there.

Dr. Lovette: Thank you for your talk.

Dr. R. Ganthier: No problem. My pleasure.

Dr. Lovette: Question, my question has to do with patients who have established retinopathy. Would you make comments on some of the recent literature where folic acid supplements are added with the promise of possibly reversing the process?

Dr. R. Ganthier: Well, there's a lot of interest in – during the scope of my talk I mentioned laser surgery plus, minus adjuvant therapies. That being one of them, along with a lot of interest in steroids or Kenalog which is injected either sub-conjunctively or intravitreally, as well as a more recent attention for a drug that's been newly released called "Macugen," which in its original indication is for actually wet macular degeneration in that it has an effect on preventing-- it's an anti-VEGF agent-- vascular and nuclear growth factor.

But it's very, very early. And we're still assessing whether this is something that will prove to be helpful or not. But what I can tell you is that we do have clinical experience with steroids to inject sub-conjunctively or intravitreally, which is helpful for the patient with diabetic macular edema and other vascular disorders.

And I will also mention another – I'll mention something else to you. There's only been one pilot study done. And I believe that there is some talk of developing a full fledged study, but there was a pilot study done for patients with diabetic macular edema, in which a set of patients were treated with laser surgery, and also received supplemental oxygen for – I don't remember the specifics – but for many hours in a given day.

And they were followed for three months and compared to patients who received the laser surgery alone. And the information that the investigator is trying to – or tried to communicate was that the patients that have the supplemental oxygen, along with the laser, did better than those that did not have the supplemental oxygen.

Now like I said, it was just a pilot study. There are many pilot studies, some of which going to be proven to be a forerunner of what is generally accepted as accurate in the scientific literature, but only after full fledged study is done. So I can't be definitive in terms of how efficacious it actually is, but there's some people looking at that.

Dr. Lovette: Some of the problems with the studies are sample sizes.

Dr. R. Ganthier: Yes.

Dr. Lovette: And the ability to stratify data to isolate homogenous population. The problem with this population of patients is they have so many other things going on. Their hypertension, other

disease states that are partially controlled. And it makes a real heterogeneous soup out of it. But in any event, I thank you for the response to your question.

Dr. R. Ganthier: Sure. Where are you calling from, by the way?

Dr. Lovette: From Hertford County, North Carolina, in Ahoskie. We are in the great northeastern part of North Carolina.

Dr. R. Ganthier: Northeastern, OK.

Dr. Lovette: East of 95 has the largest incidence of diabetes we think in the country.

Dr. R. Ganthier: OK. That would be interesting because we have quite a bit of that here in Florida as well. But you know what; can you give me your e-mail address?

Dr. Lovette: Yes. It is Kenneth.

Dr. R. Ganthier: Kenneth?

Dr. Lovette: (Kenneth.Lovette@ncmail).

Dr. R. Ganthier: At ncmal?

Dr. Lovette: Ncmal.net.

Dr. R. Ganthier: OK. Thank you very much, Dr. Lovette.

Dr. Lovette: Thank you, sir.

Operator: Moving on, we'll take our next question from Marvis Custer of St. Joseph Medical Center.

Dr. R. Ganthier: Hello.

Marvis Custer: Hi. I was just wondering on your second to last slide, you put that with diabetic retinal detachment that you have to cut and peel away the vascular tissue. And I was just wondering why that is?

Dr. R. Ganthier: Because if you do not relieve the traction, merely removing the blood – removing the blood is assuming that there's a vitreous hemorrhage there as well, is a good thing. Yes.

However, if the macula itself has been pulled off the back of the eye, if you do not relieve the traction that caused the pulling of the macula off of the back of the eye, then the patient's vision is not going to improve.

So you have to peel and cut that scar tissue to permit the macula to relax enough to lay down.

Marvis Custer: OK.

Dr. R. Ganthier: That is the reason for that.

Marvis Custer: OK. And also, was wondering you mentioned about the pregnant women getting eye exams in their first trimester. Is that for everyone or is that just for someone who has diabetes?

Dr. R. Ganthier: I would say that certainly in women who have diabetes for sure, women who have been called borderline, I say that only because how patients have related it to me, but who are not being treated for diabetes, I would also strongly consider an eye exam for those individuals.

Because if they were a borderline before, as you know, once they become pregnant, and those physiologic changes take place – that take place in the pregnant woman, then their risk certainly could – that throws them over the threshold into becoming diabetic.

But it's not something that I would recommend in the general population, no.

Marvis Custer: OK. Thank you.

Dr. R. Ganthier: You're welcome.

Operator: And our next question will come from Virginia Keller of Healthcare District of Palm Beach County.

Dr. R. Ganthier: Hello?

Virginia Keller: Hello, Dr. Ganthier. With the diabetes control complications trial that you mentioned, you indicated that the cut-off of control for blood sugar was 150.

Dr. R. Ganthier: In the study, yes.

Virginia Keller: Right. I just want – that's – 150 is typically use of the correction factor. And I just wondered whether that's where this correction factor came from, where you would subtract the blood value from which you would subtract the blood value?

Dr. R. Ganthier: I cannot tell you for sure. I cannot positively answer your question as to the exact means by which that number came to be published as the number. It's been a while since I've

looked at that particular study, but what I can say is that that was the number. How it became to be that number, I cannot actually tell you.

Virginia Keller: Because typically, that's what the endocrinologist will write.

Dr. R. Ganthier: Yes, yes. And I don't know whether it's as a result of that DCCT study that they used that number or not. But it is the most widely cited study when we're trying to establish diabetic policy.

Virginia Keller: Thank you.

Operator: And as a reminder, it is star one to be placed in the queue. And our last question at this time comes from Helen Curtis of Putnam County Health Department.

Helen Curtis: Good afternoon. I – my question was basically answered regarding pregnant women, but how about women with gestational – who develop gestational diabetes and eye exams?

Dr. R. Ganthier: Yes, women who have a history of having developed that, I would also recommend that. Because as each pregnancy develops or transpires, that risk becomes enhanced.

Helen Curtis: And also if they have no history yet develop during a pregnancy, would you also recommend the eye exam?

Dr. R. Ganthier: If they develop it during the pregnancy?

Helen Curtis: Where they've had no – let's say their first and second pregnancy, there was no gestational diabetes, but on the third pregnancy there is. Would you recommend it?

Dr. R. Ganthier: Yes.

Helen Curtis: OK. Thank you.

Dr. R. Ganthier: You're welcome.

Operator: Now we do have a couple more queuing up. Take a question from Cindy Haynes-Morgan of the North Carolina Diabetes Prevention.

Cindy Haynes-Morgan: Hi.

Dr. R. Ganthier: Hello, there.

Cindy Haynes-Morgan: My question was relating to the WESDR study you spoke of?

Dr. R. Ganthier: Yes.

Cindy Haynes-Morgan: I was trying to take notes and didn't take it quite fast enough.

Dr. R. Ganthier: Sure.

Cindy Haynes-Morgan: If you could tell me what that stands for and briefly talk about that, please?

Dr. R. Ganthier: It stands for – you got your pen?

Cindy Haynes-Morgan: I do.

Dr. R. Ganthier: OK. The Wisconsin Epidemiologic Study for Diabetic Retinopathy.

Cindy Haynes-Morgan: OK.

Dr. R. Ganthier: And what it is, is it's a population based study that took place in Wisconsin, where they looked at that that population of people, and they looked at all kinds of things, but you know, age, gender, diabetes, obviously in this particular case, you know, the percentage of patients that had diabetic retinopathy, what was the severity of their retinopathy? How long did they have retinopathy before the diabetic – the – how long did they have the diabetes before the retinopathy developed, et cetera, et cetera, et cetera? Numerous, numerous factors.

And the important information from that study is that, as I mentioned earlier, that Type I patients experience a 25 percent rate of retinopathy after five years of disease, which – and about 80 percent at 15 years of disease.

And that approximately 21 percent of patients who are newly diagnosed Type II patients have some degree of retinopathy at the time that the diagnosis of diabetes is made.

Cindy Haynes-Morgan: Thank you.

Dr. R. Ganthier: You're welcome.

Operator: And the last question in the queue at this time comes from Carol Bullock of the Medical Nutrition Therapy of Tallahassee.

Carol Bullock: . . . enjoyed your presentation. I was just wondering if we could get your e-mail address.

Dr. R. Ganthier: Sure. I'll tell you what. The – I'm going to give you – it's all in capital letters, OK? G as in George, A-N-T-S-E-B – and those are all in capital letters, @aol.com.

Carol Bullock: F as in Frank or?

Dr. R. Ganthier: No, A as in...

Carol Bullock: N-T.

Dr. R. Ganthier: No, I'll say it again. G as in George, A-N as in Nancy, T as in Tom, S as in Sam, E as in Earl, B as in boy.

Carol Bullock: OK.

Dr. R. Ganthier: @aol.com.

Carol Bullock: OK, great. Thank you.

Dr. R. Ganthier: OK.

Operator: And that concludes the question and answer session today. At this time, Ms. Gibson, I'll turn the conference back over to you for any additional or closing remarks.

Natalie Gibson: Thank you. And I'd like to thank everyone for participating in today's call. Dr. Ganthier, we appreciate you giving us your time and expertise. These insights will be useful for improving diabetes care nationwide.

Participants who have attended our past programs may have noticed changes in our educational audio teleconference series registration and evaluation process. We hope that you find that the

new process reduces the amount of paperwork, streamlines the registration process, and simplifies the continuing education credit application process.

As a reminder, nurses and dieticians who would like CEU credits for this program, need to have completed forms and registration in our office no later than February 24th, which is next week Thursday. The link for our CEU registration form is listed as online CEC for continuing education credits. Now this information is listed on your syllabus and the continuing education instruction sheet.

If you have any questions, please feel free to contact our office at diabetes@doh.state.fl, as in Florida, .us or (850) 245-4330.

We would appreciate any comments you have regarding the quality, registration process, or suggestions for our teleconference series.

Please include remarks in the post task impact survey form. Thank you again. I'd like to also thank everyone for participating on today's call. We hope that you have a wonderful day.

I will now turn the call over to the operator call completion.

Operator: And that concludes today's conference. We thank everyone for your participation. You may now disconnect.

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