IN THE COURT OF COMMON PLEAS

SUMMIT COUNTY, OHIO

VICKIE MIGLORE, et al., Plaintiffs,	)	CASE NO. CV 99 03 0973
versus	)	**DEPOSITION OF**
DR. DAVID COLA, et al., Defendants.	)	**THOMAS M. ZIZIC, M.D.**

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Deposition of **THOMAS M. ZIZIC, M.D.**, a Witness herein, called by the Defendants for Cross-Examination pursuant to the Ohio Rules of Civil Procedure, taken before the undersigned, Christine Leisure, a Registered Professional Reporter and Notary Public in and for the State of Ohio, at the law offices of Becker & Mishkind, Skylight Office Tower, Suite 660, 1660 West Second Street, Cleveland, Ohio, on Wednesday, October 4, 2000, at 10:40 a.m.

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APPEARANCES:

On Behalf of the Plaintiffs:

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On Behalf of the Defendants:

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I N D E X

PLAINTIFF'S EXHIBITS MARKED

None

DEFENDANT'S EXHIBITS MARKED

None

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MR. FRASURE: Let the Record show that we're taking the discovery deposition of Dr. Thomas Zizic.

WHEREUPON,

THOMAS M. ZIZIC, M.D.

after being first duly sworn, as hereinafter certified, testified as follows:

CROSS-EXAMINATION

BY MR. FRASURE:

Q. Doctor, your full name is Thomas M. Zizic?

A. That is correct.

Q. And your office address is in Baltimore?

A. That is correct.

Q. And you're a physician licensed to practice in Maryland?

A. That is correct.

Q. Any other states that you're licensed?

A. No.

Q. What is your position with the Johns Hopkins Hospital?

A. I'm an associate professor of medicine on the part-time faculty, which means I'm pro bono, no salary.

Q. What do you teach?

A. Rheumatology and internal medicine related

problems to rheumatologic patients on our rheumatic disease unit.

Q. How long have you been doing that at Johns Hopkins?

A. Almost 30 years. Full-time for about 17 and part-time for about 13, 12.

Q. Okay. When would the 17 years have roughly been that you were full-time faculty?

A. 1971 to about 1987 or so.

MR. MISHKIND: There's a copy of your C.V. in case you need to use that.

Q. So you have been in private practice since the late '80s then?

A. That is correct.

Q. And with someone else or by yourself?

A. No, I'm part of an approximately 100-physician multispecialty group without walls.

Q. Without walls, I like that. 150-member group?

A. Yes, 100.

Q. I'm sorry, 100. Of different internists and specialists?

A. Primarily internists, general practitioners, four or five OB/GYNs, four or five general surgeons, four or five orthopaedic surgeons, three ENTs, a couple ophthalmology.

Q. Fair enough. Is it an HMO?
A. No, it's just a fully-integrated multispecialty group.
Q. And are you the only rheumatologist in the group?
A. No, there are three rheumatologists, Dr. Holt, Dr. Saba and myself.
Q. And how long have you been with that group, Dr. Zizic?
A. The last three years basically.
Q. Okay. And then what did you do, say, before that, right before that?
A. Dr. Holt and I were in a single-specialty rheumatology practice ourselves.
Q. Is your current group called Physicians Quality Care?
A. Well, it's got a name change now. It's called Clinical Associates.
Q. So you were with another rheumatologist before?
A. Still with that same rheumatologist. We added another rheumatologist this summer, Dr. Saba.
Q. How do you spell that?
A. S-a-b-a. She joined us in August.
Q. I know a Dr. Sabai who is an expert in the HELLP syndrome. It's an unusual name, but I think it's a different spelling.

A. An expert in what?
Q. HELLP, H-E-L-L-P. It's a syndrome involving pregnant women, severe hypertension.
A. No. She has a husband who is a cardiologist is why I asked, a very specialized cardiologist.
Q. You're board certified in what specialties, Doctor?
A. I'm a fellow member of the American College of Rheumatology.
Q. That's the group that certifies?
A. That's the group that certifies.
Q. Are you also board certified in internal medicine?
A. No.
Q. So you don't have to be to be boarded in rheumatology?
A. No. Actually I preceded the board, so I became a founding fellow of the American College of Rheumatology, and have written some of the questions for the boards and chapters in the review textbook for the boards called the Primmer of Rheumatology put out by the American College.
Q. Did you precede the boards in internal medicine?
A. No, I didn't precede. I just joined the faculty immediately on completion of my fellowship

training and had, for my first two years of the faculty, a postdoctoral fellowship from the Arthritis Foundation. And immediately I got into very heavy clinical research activities and just never bothered to take the boards.

Q. I know there's some specialties like nephrology that I think you have to be internal medicine first, don't you?

A. Well, you have to have the qualifications for all the subspecialties of internal medicine, which generally requires two years of internal medicine followed by the accredited postdoctoral fellowship in that subspecialty, whether it's nephrology, endocrinology, gastroenterology, cardiology, rheumatology, neurology, ID.

Q. Infectious disease, right. And you have that?

A. I have the appropriate accredited training at Johns Hopkins, yes.

Q. What percentage of your patients currently do you see as their primary physician, primary care physician?

A. Probably about two-thirds or thereabouts, with the proviso that all of them have rheumatologic problems and I handle their internal medicine complications or diseases because I see them

generally more often than an internist would.

So I don't have any off-the-street primary care internal medicine. They would all have some kind of underlying disease, rheumatic disease, whether it's rheumatoid arthritis or lupus or ankylosing spondylitis or Wegener's or whatever.

Q. And that group, that two-thirds of your practice, you're likely their internal medicine physician, too; is that what you're saying?

A. That is correct. That's how we started the rheumatic disease unit for the Hopkins teaching program. We had senior students who had finished their medicine clerkship, and we admitted them for whatever, congestive heart failure, pneumonia, and we took care of them so we could teach the students internal medicine as well as rheumatology.

Q. Are all of your patients currently -- or at least most of them by referral when they first see you?

A. Yes, I would say -- well, it's hard to say. I mean, you know, my practice is full, so I haven't been accepting new patients for about three years. But if a physician calls and says you've got to see my mother-in-law, I'll see them. And

if it's a patient that I've been following a long time, could you please see my sister or my daughter or my whatever, I will see them. So I don't know --

Q. That's fine. Let's go back. Roughly how many instances of Wegener's granulomatosis have you been involved in the treatment of?

A. Oh, a dozen or so. Probably three or four in our practice right now in terms of patients we're following with Wegener's.

Q. Are you involved, do you think, in all those patients in your group or is some other specialist involved in place of you or --

A. Well, I mean I have two of the patients who are my patients primarily, but we all see all of our patients. I mean it's just the nature of the beast that I'm out of town today, I'm on vacation, whatever, I'm going to a meeting, somebody else sees the patient and vice-versa.

In three weeks I will be in charge of all of the patients for Johns Hopkins, Bayview Hospital, Good Samaritan full-time and part-time rheumatology. Every ten years one full-time and one part-time faculty cover the entire universe of rheumatology while the American College of

Rheumatology meetings are going on for a week with the pre and post meetings.

And so I'll probably see Wegener's during that time or an arteritis because one of them is going to flare and there's nobody. The only game in town is going to be Joe and myself.

Q. Do you speak on Wegener's at medical meetings?

A. I have, yes. One of my topics is a chapter of a textbook I've written on arteritis. And so I have lectured here in Ohio, as well as probably 25 other medical schools, on arteritis, its variants from hypersensitivity vasculitis, to giant cell arteritis, to Takayasu arteritis or aortic arteritis, to Wegener's granulomatosis, to polyarteritis nodosa.

So that whole spectrum of arterities is a topic that I have lectured on a number of occasions. And Wegener's obviously is along with churg-strauss.

Q. How do you spell arteritis?

A. A-r-t-e-r-i-t-i-s. And arterities is a-r-t-e-r-i-t-i-e-s.

Q. What is the best definition of arteritis; would you say?

A. Arteritis is inflammation of blood vessels on the

arterial side of the circulation which sometimes may involve the small vessels from precapillary arterioles, all the way to the large vessels like the aorta.

Q. Do you have any literature in your C.V. that pertains to Wegener's here?

A. Uh-huh.

Q. Would you tell us which ones those would be?

MR. MISHKIND: Let's go off the Record for just one second.

(A discussion was had off the Record.)

A. Okay. I'm just going through starting from the beginning. Now, some of these will have more -- arteritis will be involved in them and Wegener's may be.

Q. What if we focus on those that would deal only with Wegener's. Do you have any of those chapters or articles?

A. Well, not only on those, no.

Q. Okay.

A. Article 20, Acute Abdominal Complications of Systemic Lupus and Polyarteritis Nodosa, and Wegener's is in there. And then chapters --

Q. What page are you at? 13?

A. 15 right now, at least on my copy of the C.V.

Q. Let me ask you this, what is the last page on your C.V.? 19?
A. 19, correct, on the shortened version. It's about 72 pages on the full version. But that's visiting professorships and lectures which I don't usually include in my C.V. unless somebody asks for it. Probably the major one frankly would be the Principles and Practice of Medicine.
Q. No. 2?
A. No. 20 on page 17, the chapter on systemic vasculitis. And there might be a mention of it in the other one, the article I told you about, and there may be a mention of it in Gastrointestinal Manifestations of SLE, because sometimes Wegener's can have that. And in the differential I might have that on No. 9, page 16. But the major one would be --
Q. No. 20?
A. No. 20, correct.
Q. And do you have one or more chapters in that text?
A. Well, I've contributed to a number of editions of that textbook of medicine. I don't remember in that particular edition. No, it looks like I had two chapters in that edition, 1988, Rheumatoid

Arthritis and Systemic Vasculitis, 19 and 20.

Q. Now, rheumatoid arthritis would have nothing to do with this case, right?

A. That is correct.

Q. But the systemic vasculitis would?

A. That is correct.

Q. All right. Let me ask you about your medical-legal experience. How long have you been reviewing medical-legal cases for court cases involving people claiming malpractice on either side now?

A. Okay. I can tell you that in a minute. One year my friends and colleagues called it Zizic meetings in San Antonio, the American College of Rheumatology. I had six papers on avascular necrosis, and as a result of that and writing the Primmer and the Arthritis Foundation handout on osteonecrosis, I got asked to do cases of steroids and osteonecrosis primarily. I'm trying to see where that was. It would have been about 1984, '85.

Q. Has it continued each year, let's say, since in the last 15 years?

A. Well, yes, I would say a few cases early on and then more with time.

Q. And if we were to take the year 1999, last year, Dr. Zizic, how many cases estimated would you say you reviewed that came in to you for review?
A. Oh, I would say probably a dozen cases or so. I mean I probably get called twice as often as that and I --
Q. Cases where you actually received records.
A. About a dozen.
Q. And if we go back, say, three years before that, would that be roughly about the same or more or less?
A. I think it's probably been about the same the last five years or so.
Q. Do you review cases outside the field of rheumatology that would be in internal medicine but not in rheumatology?
A. No. Well, you know, rheumatology is a very interesting subspecialty because it involves every organ system of the body. And so because the lung can be involved so often, you're in the pulmonary arena. Because kidneys are involved by not only our diseases but our drugs, I probably know as much renal disease as most nephrologists, frankly, and have done some studies and looked at biopsies under the microscope more often than I

can tell you.

So I mean it's hard to say. It used to be said many years ago that if you knew syphilis, you knew medicine. Today it's more often said if you know arteritis and lupus, you know medicine because they can involve almost any organ in the body. Therefore, notwithstanding that, mostly I stay in patients who have a rheumatologic illness or corticosteroids within the medical-legal.

Q. For example, you would not review a case that the claim was that a TIA was missed by a primary care physician and it resulted in a stroke to the brain?

A. No. I got involved in a case where there was primary cerebral -- diagnosis of primary cerebrospinal angiitis of the brain which is an arteritis. But it turns out the patient had a migraine and her transient stroke was due to a migraine and she got high-dose steroids for over a month and developed avascular necrosis, which is how I got into the case.

Q. How many cases would you say you've reviewed that involve Wegener's in the medical-legal context?

A. Medical-legal context probably -- well, I know

two, and there may have been a third. I'm trying to think about -- There was a case of Kowalski and I've forgotten the doctor. That was the plaintiff and that was Wegener's granulomatosis in Chicago.

I think there was one other. I just can't remember the name of it. It was either polyarteritis or Wegener's. They both can have renal involvement and I just can't remember the details of that case. It was a while back.

Q. Okay. Over the past five years, you've said approximately twelve cases a year, plus or minus. What is the breakdown roughly for reviewing for the plaintiff or for the defendant when the case comes in?

A. Usually about three-quarters defendant and one-quarter or so plaintiff. Now, I get called about equally as often, and about half the cases that I get called on plaintiffs I, after talking to them for a while, decide it's not a case, that I feel they don't have a valid case. I mean if somebody didn't get 25 milligrams of prednisone a day for more than a month, I don't think the steroids caused avascular necrosis. So I'm going to say, look, don't waste your money and my time.

Q. Don't send the records?
A. Don't send the records. No matter what else is going on in the case, I don't think the steroids caused it.
MR. MISHKIND: But the review is three-quarters defendant when you get the records?
THE WITNESS: Yes, when I get the actual records, it turns out it's about three-quarters defense, one quarter plaintiff.
Q. But this other quarter, let's say, that you tell the attorney I don't think you have anything here, is that typically a plaintiff's attorney calling you more often than not?
A. Well, more often than not, because it's usually steroids and they've got a short course of steroids for poison ivy or they got a couple of injections. And I believe that it takes at least a month of steroids of more than 25 milligrams a day. Our experience, we would say at least two months of continuous steroids.
And so because that's my area of expertise, I don't want to have a plaintiff's attorney send me a case and then just turn around and charge them some money to tell them what I

could have told them over the phone. And so, yes, it's usually plaintiff's attorneys.

Q. Those kinds of cases, just so I'm clear, the question then would be did the use of the steroids affect the immune system? Is that typically the question?

A. No, no. The question that I'm most often asked in those cases is was there a deviation of standard of care in using corticosteroids for this disease in this dosage in this duration under the given circumstances; and as a result of that deviation of standard of care, did the corticosteroids administered cause osteonecrosis or avascular necrosis of bone.

Q. So it's usually a bone disease that is the result?

A. Correct. That's what I've written a lot on, as you can see here. And I comment on it in my report in this case that I think --

MR. MISHKIND: You don't have to go into that. Just wait for his next question.

Q. Fair enough. What is your typical charge in these cases once you actually get records and things to review?

A. I charge $400 an hour, $1,500 for a deposition,

and $3,000 for trial.

Q. And the $3,000 includes the time in and going back, right?

A. No, it's a day of trial. Usually, as you know, most often you come in the night before, meet with the attorney, then have trial and go back the next night.

Q. So that would be $3,000 plus your expenses?

A. Plus my expenses.

Q. Fair enough. What have you reviewed in this case, Dr. Zizic?

A. Well, these two volumes of records that I enumerated in my report, and they're pretty thoroughly enumerated, the two parts of Vickie Miglore's deposition, Dr. Cola's deposition, Dr. Spoljaric's deposition and some typed office notes that were attached to that deposition, and a letter of January 5th, 1998, that is Vickie Miglore's letter to -- I guess it was the insurance company. I don't know. To whom it may concern.

And then I got some of these things -- Well, I read Dr. Hoffman's evaluation, and then recently I got also some reports of various experts. I think I just got this one, Akron

Nephrology Associates, faxed to me yesterday, I believe.

Q. Did you review that?

A. Yes. And then Dr. Spoljaric's office records are here, Dr. Cola's office records are here, the Barberton Citizens Hospital's, Nephrology Associates, Akron City Hospital ER. And I think I just got -- not yesterday, but within the past week or so -- things are a blur here in terms of exactly when I got what, but I got some updated records from Dr. Zarconi. I think it was just the most recent evaluation. Here it is. 7-5 evaluation by Dr. Zarconi.

MR. FRASURE: Off the Record.

(A discussion was had off the Record.)

BY MR. FRASURE:

Q. Have you reviewed records from Dr. Torok, T-o-r-o-k, and Dr. Schirak?

A. I believe I have.

Q. Might those be in the binder?

A. Well, I'll tell you if I reviewed them first. I believe they are. I mean I recall them or I believe I recall them, but I don't know if they were part of Dr. Spoljaric's records, one of them at least.

Q. Maybe part.

MR. FRASURE: Do you know which ones those are in, Howard?

MR. MISHKIND: Just to save some time, I believe that what Dr. Zizic was provided was the records from -- the copies of the notes that were sent by Dr. Schirak's office -- and for that matter, I think Dr. Torok's office -- either to Dr. Cola or to Dr. Spoljaric. I don't believe that he has actual full copies of either of those two offices, the orthopaedist's or the GI's records.

Obviously there's reference in the depositions to them and there's copies that Dr. Spoljaric had, or at least segments of the record that Dr. Spoljaric had. But I think that's the extent of what he's been provided.

MR. FRASURE: Off the Record.

(A discussion was had off the Record.)

BY MR. FRASURE:

Q. Going to your opinions in this case, Dr. Zizic, does your standard of care opinions as to Dr. Cola, my client, chronologically begin with the office visit of August 13, '97?

A. That's fair to say.

Q. Do I read your report correctly to say that as you look back on some previous visits to August 13th --

A. Is it the 13th?

Q. That's right, 13th.

MR. MISHKIND: What did you say it is?

MR. FRASURE: 13th.

Q. Are you saying in your report, looking back at some prior visits, that you see some indication in retrospect of the development of a disease?

A. Yes, there may be some previous indications that there were some symptoms. But I'm not critical of that, because the initial symptoms of Wegener's may smolder for a very long time and be nonspecific enough that it's very difficult for the diagnosis to be made at the various early stages sometimes. And so I'm not critical of him not picking those up. They may be related. Who knows. There's no way to ever tell at this point in time.

Q. So your criticisms against Dr. Cola start on the office visit of the 13th of August?

A. Correct.

Q. And what is that criticism pertaining to that visit?

A. Well, that specific visit, the criticism is that there were a multiplicity of systemic complaints and symptoms, headache, generalized weakness, arms tingling, knees and hands swelling. That's very important. Not just the feet, because that would indicate -- should indicate an arthritis, but difficulty with breathing and felt like a weight on her chest, difficulty in turning her head from side to side, edema, seems to bloat, and pain on the side radiating through to the back. Which she later had pancreatitis, but that would also be a symptom that would be concerning when it radiates through to the back.

And particularly, as I mentioned in my report, the presence of 3 plus blood on the dipstick with negative leukocytes. So that there did not appear to be any infection, and yet there was gross hematuria and she was not having her period. I'm critical of him not saying something to her at that time. The advantage of doing a dipstick is you have the results immediately and you can talk to the patient about it and decide what else you want to do.

So I would have as a criticism that that test was available immediately at the time

he saw her and he should have also spoken to her about the blood in the urine and what he was going to do about it.

Q. What did the standard of care require him to do as of that date when he got the dipstick?

A. Standard of care required him to do at a minimum a complete urinalysis with microscopic, which was in his plan, UA, and a culture and sensitivity on that urine to rule out infection, which can cause blood in the urine as well. Less likely with the leukocytes there, but you should be sure there's not an infection there.

Q. What if the culture had revealed --

A. Less likely with the leukocytes being negative on the dipstick.

Q. If a culture had been done and had revealed no organism, no infection -- Do you follow me?

A. Uh-huh.

Q. Does that tend to point more to something systemic? Maybe that's not the right word. Some inherent kidney disease?

A. Well, it would point you to either a bladder lesion, tumor, cancer, polyp of the collecting system, or an intrinsic renal disease such as glomerulonephritis or vasculitis.

Those possibilities would be evaluated and the microscopic examination may well show you an active sediment which would indicate that there is a glomerulonephritis or vasculitis going on and would lead you to evaluate the kidney with a sed rate and a C-reactive protein and a 24-hour urine protein creatinine clearance, as would a negative culture with the persistence of hematuria with no active sediment.

One would investigate then both the possibilities, both urological more strictly speaking in terms of bladder and collecting system, and nephrological, the kidney itself in terms of vasculitis or glomerulonephritis.

Q. If a full urinalysis had been done, culture and sensitivity also, in hindsight, what do you think it probably would have revealed?

MR. MISHKIND: Let me just object to the form of the question. He can go ahead and answer it.

A. I would suspect that there was no infection, that it would have revealed at a minimum red blood cells, microscopic hematuria, and it may have revealed an active sediment. I mean we won't know and there's no way of knowing that. At a

minimum continuing hematuria.

Q. What do you make of the negative protein on the dipstick?

A. Typical of a glomerulonephritis. Until the late stages of a glomerulonephritis -- unless it is a lupus glomerulonephritis, which it wasn't. This was a necrotizing glomerulonephritis. If it's a lupus glomerulonephritis with immune complex deposition on the glomerular basement membrane, you may see proteinuria as an early manifestation of glomerulonephritis.

But in an inflammatory necrotizing glomerulonephritis, hematuria would be the first thing you would see, and only late when there is scarring and the interstices of that glomerular basement membrane are being widened because of the contraction of the scar opening up the pores, if you will, of that basement membrane, will you get protein leak of a significant amount. Now, we all leak protein. It's a question of magnitude and --

Q. Protein that would show on a dipstick, for example?

A. No, it doesn't usually show. Trace might show. But, no, I mean we all have some -- if you look

at a 24-hour urine, we all have some protein leak because the kidney is not a perfect filter. It can't let all the bad things out, the organic acids and the creatinine and the urea and etc., uric acid, and keep all the good things in. So some leaks out. But it isn't until late-stage glomerulonephritis of the necrotizing variety that you see protein leak.

Q. Okay. Did she have the inflammatory necrotizing glomerulonephritis --

A. Yes.

Q. -- looking back to what she was diagnosed with?

A. Yes.

Q. Okay. And you're saying that type of glomerulonephritis, the protein, if present, it's usually only going to be late in the disease?

A. Generally, yes, it's not initially part of the manifestation.

Q. If we take those types of patients, inflammatory necrotizing glomerulonephritis --

A. That are nonimmune complex, okay.

Q. Is that her?

A. Yes. Lupus glomerulonephritis, for example, where you have antibodies to DNA and DNA or antibodies to nuclear protein and the nuclear

antigen, which is a nucleoside, you may have inflammatory necrotizing glomerulonephritis and membranous changes where you have obliteration of the foot processes on electromicroscopy and protein leak and that can occur more towards the onset.

When you have vasculitis or the glomerulonephritis that you see with Wegener's, you do not have immune complexes, and so the basement membrane does not get damaged until later in the -- much later in the process as scarring is occurring and the scarring contracts the fibrous tissue, which will spread the basement membrane, allowing protein to leak through.

Q. In that situation, why does blood leak out but not protein yet?

A. Well, because the precapillary arterioles and the vessels within the glomerulus. And the glomerulus of the kidney is a very interesting structure because there are post efferent arterioles. So it's got arteries on both sides of the glomerulus. And when you damage that with inflammation, then blood can leak from those damaged arterioles into the urine as its formed

in the glomerulus.
Q. Without protein leaking yet?
A. Without much protein leaking in, that's correct.
Q. So if we looked at all patients who have inflammatory necrotizing glomerulonephritis of the nonlupus type like she has --
A. Of the nonimmune complex type, which lupus is a prime example.
Q. In the early stages, are you saying that most of them would not show protein on dipstick?
A. Most of them would not show protein on dipstick, that's correct.
Q. And most of them would show blood on dipstick?
A. Correct.
Q. Do you have any opinion, Doctor, on whether a repeat of the urinalysis would have then probably led a family practitioner to send the patient to a urologist more than likely for a work-up?
A. I would anticipate if a complete urinalysis and culture and sensitivity had been done, that the more likely referral would have been to a nephrologist.
Q. Why do you say that?
A. Well, because I think you would have microscopic hematuria and I think you would see some

abnormalities in the cast -- I mean in the sediment, active sediment with some cast. As the red blood cells are leaking down, they concentrate in the tubules, and as they go, in the urine there are casts of red blood cells. Because it's not gross blood that's coming lower in the tract, it is blood that is leaking out of the glomerulus.

As it goes down the descending loop to the loop of Henle and the ascending loop and then down through the collecting system, these red cells basically stick together and form casts which are outlines of the inner diameter of the tubules and are seen in the urine. And when you see that, that is typical of glomerulonephritis.

Q. What is it that a primary physician, a nonspecialist would have seen on blood work that would have come back to him or her that would have told him this patient needs to go to a nephrologist?

A. Well, what you would see is -- you generally would not see any abnormality in renal function until way late in the disease. You would see evidence of inflammation more likely than not within the erythrocyte sedimentation that would

be elevated or a C-reactive protein that would be elevated. And those would be -- I mean we're talking about $30 total screening tests for a complete urinalysis with microscopic and a sed rate and C-reactive protein.

Q. And that should have led a primary care physician to send the patient to a nephrologist?

A. Yes, say something is going on here inflammatory that may be in the kidney.

Q. Rather than a urologist?

A. Yes. I'm not saying -- I wouldn't say it was a deviation of the standard of care necessarily to send her to a urologist, because they would have found nothing there and then would have continued the work-up through nephrology with an evaluation of potential glomerulonephritis, which is what she had.

Q. Following up on what you said, if the patient instead had gone to a urologist, the typical work-up there by a urologist would probably have been negative like the IVP and the cystoscope?

A. Well, I would hope the urologist would start out with a complete urinalysis and look at the urine themselves.

Q. Okay.

A. I mean that's what I do as well in my practice. I mean I see blood on a dipstick, I look at the urine myself. I sit down and look at it.

Q. And what would that study likely have led to by the urologist?

A. Well, if you saw blood in the urine and an active sediment, you would have been saying that this is probably intrinsic renal disease and needs an evaluation for potential vasculitis or glomerulonephritis.

Q. And what would a urologist likely have done that would have led to the diagnosis of the Wegener's here?

A. Depending upon the urologist, either worked it up himself, but probably refer to a nephrologist that he generally works side by side with.

Q. What is the ultimate test that would have -- what is the first test that would have revealed Wegener's more than likely done by either a urologist or a nephrologist?

A. Well, I don't think you -- you know, you would be working up the patient for vasculitis or glomerulonephritis of a variety of sorts. So one would not necessarily jump -- although that's one of the possibilities, lupus is another

possibility here.

The first things that would be done would be to get sed rates, C-reactive protein to show that there's an inflammatory process. The second step would be to get a 24-hour urine for creatinine clearance which might show functional changes that are not yet evident on the serum creatinine, and a 24-hour urine to see whether -- the specific sensitive test to see whether there's an increase in protein in the urine.

Then one would have referred the patient to a nephrologist or a rheumatologist, either one, depending upon the locale more than anything else, and who is in the area practicing. Then you would have gotten a connective tissue battery, a lupus package.

Q. A battery of blood --

A. An antibody, an anti-DNA antibody.

Q. Is that blood work?

A. Blood work in the c-ANCA. That would be the next group of tests, c and p-ANCA both, because you may see a p-ANCA in other kinds of vasculitis.

Q. Approximately how far past the initial visit of August 13th would it likely have been when that diagnosis would ultimately have been made that

she had Wegener's and treatment begun?

A. Well, you know, I think it depends. Certainly by August 27th, about two weeks after her initial visit when there's a telephone call in the records that she is complaining of still feels weak, not urinating as much. I'm not reading everything. Still has pains in side. States by Friday, 8-22, she couldn't move, couldn't eat, talk or sleep. Severe neck and jaw pain. Broke out in boils on buttocks and face.

Now, that could be necrotizing vasculitis of her skin and probably was, more likely than not. And after the boils broke, patient started feeling better. Please advise. Patient would like to know what this was. And Dr. Cola writes sounds like infection, would recommend treatment with Augmentin 500 milligrams BID with food. Refer to neuro for a second opinion.

Well, that patient should have been, number one, if it hadn't been done before, seen. You don't take somebody with these kinds of symptoms and not urinating as much with 3 plus blood in the urine and having boils on their buttocks and face and not talk to them on the

phone or not bring them in, but give them a prescription for an antibiotic. That's way below the standard of care.

So I think if she was seen the next day, which is when she should have been seen, that day or the next day -- I don't know when the phone call came in -- that a repeat urinalysis and an evaluation of the patient would have shown that this looked like something that needed to be looked into with ulcerated lesions on her skin of her face and her buttocks. That just as when she came into the hospital within a week or two of this point, she should be diagnosed depending upon --

Q. A week or two from the 27th?

A. Yes, I would say within the week of the 27th.

Q. Early to mid September?

A. Yes, uh-huh.

Q. If diagnosed, say, by mid September, what do you think her kidney function would have been at that point?

A. Well, I think it would be normal or slightly abnormal, but clearly with reversible disease and nonsignificant permanent damage. Any time through the fall, I think, August, September,

October, November, she would have been probably, more likely than not, totally reversible, whatever amount of renal insufficiency she had.

Now, November, December, January, I think probably she still would have had some -- she would have had reversibility to some degree, probably would not have retained totally normal function, but would have had reversibility to some degree. Once you get into February, March, I think she would have enough significant permanent scarring and damage that it would not have been reversible to any great degree.

Q. But December and January reversibility to some degree?

A. Yes, I think there would be some reversibility. How much permanent damage, not as much as there was in March, but --

Q. Would she have needed dialysis if diagnosed in January, say, early to mid January?

A. I don't know if there's any good way to say that, you know. It is possible that she would not have needed dialysis. Whether it's probable or not, I'm having a difficult time saying that. Certainly there was a good chance, at least a 25 to 30 percent chance she wouldn't have needed

dialysis. Whether it's more than that, I don't know.

Q. Can you estimate what kidney function percentagewise she would have been left with had it been diagnosed and treated by early to mid January?

A. My hunch would be since she's stabilized now with a two-thirds loss of her kidney, that she would be less than half her kidney loss if she was diagnosed at that point.

Q. Somewhere between a third and a half?

A. Somewhere in there.

Q. What would have been the required treatment for her, Mrs. Miglore, if she had been diagnosed in mid September, let's say?

A. The preferred treatment would have been corticosteroids, but in lower doses than she ended up needing in March. Probably in the 1 milligram per kilogram range and 60 milligrams a day instead of 500 to 1,000, which she needed for the early part of her treatment and its relapse.

I think it would be split today between people who would go to methotrexate if they didn't have any significant renal damage and pulse cytoxan rather than daily oral cytoxan

because it has less toxicity. But I think most people today without significant renal damage or significant organ damage would use methotrexate.

Q. Rather than cytoxan?

A. Rather than cytoxan, because it's safer and you can keep people on it for years. You know, cytoxan not only sterilizes people -- that wasn't her situation since she already had a hysterectomy, bilateral salpingo-oophorectomy -- but it in increases your cancer risks by about two and a half times and your bladder cancer risks by about 30 times.

Q. Cytoxan does?

A. Cytoxan does. So people without significant renal damage would go to methotrexate and prednisone, and some would even try bactrim and prednisone rather than cytoxan. And if it didn't work or things were progressing despite that, then they would use cytoxan.

Q. How long would you have expected her to be on methotrexate if diagnosed in mid September range?

A. Well, most people today will say that if you are in remission for six to twelve months that you would then -- and it ranges a little bit, but in that range, at least six months. And I don't

know that anybody requires more than twelve months of remission before you taper and discontinue the agent. And given another three months -- I mean another six months to get her under control, probably around a year of methotrexate therapy.

Q. Beginning when it was diagnosed?

A. Beginning when it was diagnosed, yes.

Q. And more likely than not, she wouldn't have needed it any more beyond about a year?

A. I think she would have gone in remission as she has now. She probably would have relapsed later as she will now. I mean the vast majority of patients with Wegener's will relapse and this patient will relapse also. I mean you can bet on it.

Q. What happens when they relapse even with an early diagnosis of Wegener's?

A. They have active disease again. And it may be active glomerulonephritis again. But generally speaking, if they have normal renal function and you're following them closely for that and they start to have active sediment again and are clearly having the disease, you would treat them. And again, if you catch it before you

have much permanent loss, as long as you haven't lost more than half your kidney, you generally -- you know, it's compatible with a relatively normal life unless you end up having further loss.

Now, as you know, beyond the fourth decade, everybody loses about one percent of their kidney function per year. So there's a gradual attrition. Now, for most people it's a moot point because they die of something else before they lose enough kidney function to die of that. On the other hand, the more kidney function you lose from more relapses, the more potential problem you're going to have.

Q. If she had had the diagnosis early, in mid September, let's say, and had the methotrexate for about a year and if she had relapsed, would she need more methotrexate for a while?

A. Yes.

Q. Would she have needed hospitalization either initially or on a relapse more likely than not?

A. Clearly you would probably have an initial hospitalization back in September for a kidney biopsy unless you found the biopsy on the skin, those ulcerated lesions on the skin or the

buttocks. If a punch biopsy of that showed vasculitis or an excisional biopsy and you had a c-ANCA that was positive at that time, you wouldn't need to do a kidney biopsy so you could do it all on an outpatient basis.

But it is possible that she may have needed hospitalization assuming that her c-ANCA was borderline or questionable and the skin lesion was not demonstrative of vasculitis, then one would have gone to a kidney biopsy and it would have required hospitalization. I'm not saying that she would have had the need for no hospitalization. She might have. I just don't know.

Q. Do some patients with Wegener's, even when diagnosed early, require cytoxan rather than methotrexate?

A. They do, particularly -- and one of the problems with this patient's long-term care is methotrexate is a much safer drug to maintain remissions on. But once the creatinine is over 2.5, one can't use methotrexate.

Q. Why is that?

A. Well, because it is excreted by the kidney almost totally and it is just too difficult to not have

side effects, particularly in a woman who has a creatinine that's running 2.8, 2.9 with a 32 cc creatinine clearance. It's just not a safe drug to use in that situation.

Q. Is it your understanding that the only drug that she's been on is the cytoxan?

A. No, she has been on corticosteroids. She's been on cytoxan and she was given bactrim for a period.

Q. Is bactrim a sulfa drug?

A. Yes, trimethoprim sulfa.

Q. What is the effect of her having been on bactrim, b-a-c-t-r-i-m, for a while?

A. That's a correct spelling of it. It sometimes is sufficient in early disease, even without methotrexate or cytoxan to, along with low-dose corticosteroids, control Wegener's.

Q. I understand. But on this particular patient, has the use of bactrim caused any adverse effects to her; do you know?

A. I don't think so. I think the adverse effects of the pancytopenia that she had in the spring of '98 were due to cytoxan, not bactrim. I'm not even sure she was on bactrim at that time.

Q. Is it your understanding she's still on cytoxan

or not since she's in remission?
A. She's been discontinued from her cytoxan and she's on no medication for her Wegener's right now.
Q. Are you able to say what effect on this patient the use of cytoxan had on her that would not have been present with the use of methotrexate if methotrexate had been used instead?
A. Well, the major aspects of it are that at some time in the future she may well develop -- would develop a cancer or a bladder cancer from that use of cytoxan during that period of time.
Q. So it increases her risk of what types of cancer?
A. Well, malignancy in general increases by about two and a half times on people who have had exposure to cytoxan like this, and bladder carcinoma specifically is increased by about 30 times.
Q. With the use of methotrexate instead, does that increase the risk of cancer or malignancy?
A. No.
Q. None whatsoever?
A. None whatsoever.
Q. Is it your opinion the patient could have, if diagnosed earlier, gotten by with methotrexate?

A. Or bactrim or both.
Q. How about corticosteroids, would she have likely needed those?
A. Yes, I would say that she would use them as, again, much lower doses, 60 milligrams a day. And once she was controlled, tapered down to a lower dose ultimately.
Q. Does she need to be on corticosteroids now while in remission?
A. No.
Q. What would you say the net effect of her being on the larger dose of corticosteroids has been versus the smaller dose you said she might have needed had it been diagnosed early?
A. Well, two major effects. One is that the larger doses have contributed in part to her osteopenia or decreased bone mineral density. The larger the dose of corticosteroids and the longer the duration of that dose, the more osteopenia you get or decreased bone density, which is the step before osteoporosis.

And it makes her more vulnerable to renal osteodystrophy, which she clearly is going to get very significantly very soon because she has metabolic acidosis right now of a significant

degree.

Q. What is that term again that you think she's going to get?

A. Renal osteodystrophy.

Q. Is that one word, osteodystrophy?

A. D-y-s-t-r-o-p-h-y.

Q. Thank you. What is that, Doctor?

A. Well, it occurs for three reasons in patients who have chronic renal failure. The first reason is that once you lose more than half your -- actually more than 40 percent of your kidney function, there's not enough kidney mass left to convert vitamin D to dihydro 125, which is the active form of vitamin D which allows you to absorb calcium appropriately from your gut.

Q. Okay.

A. Secondly, you have metabolic acidosis. You have acids that are formed in the normal course of living and eating, particularly proteins, and sulfuric and phosphoric acids are formed, and the body other than the kidney has no way of getting rid of it. And that's why she's with metabolic acidosis right now with the CO2 of 19. That's significantly low.

Q. What is the effect practically on her?

A. Well, what it does is two things. It does further damage to her kidney clearly, directly. Secondly, it further leaches -- and that's what I was getting to on this point, it further leaches calcium out of her bones in order to form the salt. You can't have circulating phosphoric acid and sulfuric acid in the body. You will get severely acidotic and have a cardiac arrythmia and die.

So what you do is leach calcium out of the bones. And the calcium casein binds with the phosphoric adenosine, the phosphate and the sulphate, and it neutralizes it, but at the expense of weakening the bones. So that's the second effect that causes and contributes to renal osteodystrophy.

And then the third thing that happens then is as acid, and particularly phosphate, increases in the blood, which it eventually will, then the calcium goes down concomitantly. The amount of calcium and phosphorous in the blood balance out. Calcium goes down when phosphorous goes up.

Well, the parathormone -- the parathyroid gland, says, oh, we don't have enough

calcium circulating around here, we better put out more parathormone, and so it goes more and you get a secondary hyperparathyroidism, which causes osteoclast with a C, osteoclast, to break down bone, release more calcium, and that further weakens the bone. So those three things cause significant weakening of the bone and that is together what renal osteodystrophy is.

Q. Weakening of all bones?

A. All bones. Now, the second major effect on the bone from the high-dose corticosteroids that she had, 500 milligrams for three days and then 1,000 milligrams on the second course for five days, plus long-term high-dose steroids after that, is osteonecrosis. Osteonecrosis, not porosis, also called avascular necrosis of the bone.

And our studies have shown that when patients get over 80 milligrams average a day for more than a month and get cushingoid, where she got cushingoid during that course, the second course, the 1,000 milligram course --

Q. What is cushingoid?

A. Cushingoid means that you have an altered fat cell distribution. The fat cells in the face and the trunk --

Q. That's the moon?

A. Moon face, uh-huh. Hypertrophy, and just like the fat cells you can see on the face, the trunk enlarging, the fat cells in the bone, in the fatty marrow of the bone, long bone, hips, knees and shoulders, they increase by 69 percent in size as compared to osteoarthritic controls, lipocytes within the bone.

That increase in the lipocyte size in the bone increases the pressure in the bone. That increased pressure increases resistance to bone blood flow and you get a decrease in bone blood flow, you get ischemia or lack of circulation to the cellular components of the marrow. They swell further, increasing the pressure, and you get a vicious circle with its own internal amplification loop that eventuates in the death and collapse of bone that we know as osteonecrosis.

The average time from peak steroids to the time of this is 42 months, so with a range of two months to ten years. So up to ten years from now those steroids received in the spring of 1998 will cause death and collapse of an average of 3.3 of her major bones, hips, knees or shoulders.

Q. 3.3 percent?
A. 3.3 bones.
Q. Okay.
A. 3.3 bones. So that over 50 percent of patients who get this amount of steroids and get cushingoid will have osteonecrosis an average of 3.3 bones per individual patient. So both hips and a knee, one or both hips, a knee, or both knees and another, etc. Average of 3 plus bones per patient --
Q. Break?
A. -- collapse and die and need replacements, which cost about $25,000 a piece, and need to be revised in 10 to 15 years on average.
Q. And these typically are which bones, the hip bones?
A. Hips, knees and shoulders, in that order, and that's been shown by our publication in the American Journal of Medicine 1985. I am first author on that. And it was shown in a metananalysis by Felson and Anderson, almost 3,000 patients, 23 studies. We came to the conclusion average 81 milligrams a day for the highest month of therapy. In their 23-study metananalysis of 3,000 patients, they came to 80

milligrams a day. I'm very happy that our paper was published two years before the metananalysis or else somebody would have said how did you come this close to the average of 3,000 patients.

Q. What is your criticism of Dr. Spoljaric, the standard of care?

A. Just as I put it in my report. I'll be happy to go over that with you.

Q. First of all, that he did not pick up on the plus 3 blood that was on Dr. Cola's records?

A. Well, yes. That's what I was looking for, page 12. Basically I think that -- in part I think it's a little bit difficult at times, because I mean I look at this note that was handwritten, this is a typed one from the 13th and it says urinalysis. And so my assumption as I would be reading that note is that a urinalysis was done to check on that blood and it was okay because nothing further was gotten.

So I criticize Dr. Cola for transferring these records without either mentioning, by the way, in August she had 3 plus blood and we've never done a urinalysis or followed up on that. But nevertheless, he should have also looked at this patient who now has --

Q. He, Spoljaric?
A. Spoljaric, who comes to him and he had the history available. If he read the notes -- now, granted, it's 38 to 40 pages, but it's still -- I mean it was in there for him to see.
Q. The last visit is only one page, right?
A. Well, there are other pages besides, but there's a whole page of phone calls in that, I mean communications to the patient that was clearly a lot of information. And when he got the elevated sedimentation rate, although that could be due to infection and it could be due to something systemic, I think I would have felt that he should at least check the previous records at that point with the elevated sedimentation rate.

So I'm not so critical of his first visit, 12-31-97, but now he's got a sed rate, he was aware the sed rate was elevated because he put it on the request. So at that point, I think his second visit in January, he should have either gone through the records or called.

Q. The second visit wasn't until January 22nd, I believe?
A. Right.
Q. Would it have required him to call her sooner

than just waiting for her to come back in on the 22nd?

A. Well, again, as soon as you appreciate that there were other potentially systemic symptoms going on and that there's 3 plus blood in the urine, yes, I would be critical of him not --

Q. Certainly by the time he gets the sed rate back?

A. I guess my problem is there's some dispute. And what I need you to tell me is when am I to assume that he got these records? Because there's some dispute in the various depositions and testimony that I've read as to when he got these records. So I can't be critical of somebody until I know. What do you want me to assume?

Q. Well, let's assume he got them on January 10th. There's testimony that they were mailed on the 8th of January, and he sees her for the second time on January 22, I believe.

A. All right. You know, I guess clearly by January 22 you should then put it together and look at it and look at these records. Whether in a busy office you get something on the 10th or 11th and the patient is coming back in on the 12th -- I mean the 22nd, 12 days later, and it's 38 pages, you might not have a chance, frankly, to look

through the records before the visit. So I mean that's why I'm saying the second visit, assuming that he didn't have them on the first visit.

Q. Let me ask you this. And I don't know offhand when the blood work was done. I think it was done December 31 by Dr. Spoljaric --

A. I think you're correct.

Q. -- which revealed the increased sedimentation rate.

A. Correct.

Q. Which was 51, right?

A. 52, I think.

Q. Which is pretty high?

A. Right.

Q. I don't know the exact date he got that back, but let's assume whenever he got it back, is that a tip-off that further work needs to be done?

MR. MISHKIND: You mean besides the bone scan that was done?

MR. FRASURE: Right.

A. Without some of the other history and particularly the 3 plus blood in the urine, I mean a sed rate can be an indication of infection as well. I mean it's nonspecific. On the other hand, in the setting of blood in the urine, a sed

rate this elevated would at least want you to think about a differential diagnosis that included an inflammatory process within the kidney causing both the elevated acute phase reactant, be it a sed rate and the C-reactive protein, and want to look into that possibility of glomerulonephritis or vasculitis.

Q. And can we agree that by mid January the patient probably would have had more normal kidney function and much of her problems would have not occurred?

MR. MISHKIND: You mean as opposed to March?

MR. FRASURE: Right, if it was diagnosed in mid January.

A. I think both of the doctors are at fault. Cola much more than Spoljaric because he had been following her for a while. He had the 3 plus blood in the urine, and he was also in a position to have a situation where you were much more likely to have less permanent damage. But I'm not saying that Spoljaric also shouldn't have picked it up.

Q. Just so I'm clear, you also believe that Dr. Spoljaric's deviation from the standard of care

was a proximate cause of her injury, too?

A. Something like 80/20 or 75/25 Cola's responsibility versus Spoljaric's.

Q. I'm not asking you to break it down in terms of negligence. But are you saying that had Dr. Spoljaric acted appropriately that she would have 80 percent of her kidney today, 80 percent total function?

A. No, I don't know that.

Q. Okay. I understand. You say on your last page of your report that to and through January of '98 that her renal function would have been significantly better and, concomitantly, her permanent kidney damage would have been avoided, as well as the various complications she experienced while hospitalized. I take it that those would have probably occurred -- or not occurred had the diagnosis been made before the end of January of '98?

A. Well, I'm not saying that she would not have had some permanent kidney damage. And obviously the earlier you pick it up and treat it, the less you're going to have. I would not be as certain as I am today that she's two or three years away from end-stage renal disease and dialysis and

transplantation, but that's clearly the case today.

Q. Two or three years in your opinion away from end-stage renal disease?

A. End-stage renal disease as defined by the need for chronic dialysis and/or transplantation.

Q. And when you say the need for chronic dialysis and/or transplant or --

A. Ideally what you want to do is transplant her if you could find a suitable match.

Q. When you say a need for chronic dialysis, beginning at that point when the end-stage condition occurs?

A. Right. I mean the definition of end-stage renal disease is when you have a creatinine clearance of 10 cc's or less, or if it's more than that, that you have intractable symptoms of uremia which she's already starting to develop, nausea, vomiting, lethargy, weakness, and she's got metabolic acidosis.

And so a nephrologist has got to -- or whoever is in charge of the dialysis circumstances, but a nephrologist has got to justify dialysis if you have a creatinine clearance more than 10 cc's. You know, nobody in

the government doesn't say we're not going to pay for it unless you're 10 cc's or less. If you have 10 cc's of creatinine clearance or less, you've lost 90 percent of your kidney function, then you don't need to justify it. Creatinine clearance of 9 and they pay for it, no questions. You don't have to justify.

Q. Dialysis?

A. Dialysis or transplant.

Q. What is her creatinine now?

A. Her creatinine clearance is 32 cc's a minute, her creatinine is 2.8.

Q. So if you say that she's two or three years away from end-stage renal disease, what would be her creatinine level that is -- the one that's referable to the 2.7 now, did you say?

A. 2.8. Well, keep in mind that a creatinine is a surrogate measure of renal function and its level in the blood is based on the output of creatinine from creatine from muscle. The bigger your muscle mass, the more creatinine you've got to dispose of.

And so you might have a normal creatinine of 1.2 and our court reporter might have one of .6 and that's her normal creatinine

level. She's smaller, she has less creatinine she's forming. Okay. Now, so the creatinine clearance is a better measure in this situation in that it is 32 cc's, it should be about 100.

And so you're looking at something that is going to go from 32 to whatever, but probably it isn't going to be able -- she's not going to be able to tolerate waiting until it gets to 10 because she's already having symptoms of uremia and she already has significant metabolic acidosis.

Q. What are her current symptoms of uremia, do you believe?

A. I just listed them for you. Nausea, vomiting, lethargy, weakness.

Q. Okay. You've read Dr. Zarconi's report, I think it's August of this year?

A. Yes, I have read it.

Q. Do you feel that it's a little too optimistic about her --

A. Of course he is. He's too optimistic. And he's also in a situation where he --

Q. How do you differ in your prognosis for her versus Dr. Zarconi; would you say?

A. Well, first of all, you need very tight control

of hypertension. That's clearly going to accelerate the renal disease.

Q. Does she have hypertension now?

A. Oh, yes.

Q. Okay. Is it being controlled?

A. Not as well as I would like to see it controlled. You would like to see it under 130 and under 75. The lower the better, because you're going to have a quicker progression to dialysis and transplant without control. She's not on a low-protein renal diet like she was. You should have less than 30 milligrams of protein with essential amino acids added which can extend the period of time before dialysis. And she ought to be on sodium bicarbonate to neutralize her metabolic acidosis. That's standard. I mean we're not talking about rocket science here. We're talking about huge, huge, huge studies, the MDRD study which shows those three factors are very important in terms of maintaining patients not on dialysis as long as possible.

You can sometimes get an extra year or two or three by doing those three things. And so for whatever reason, I would at least from the scanty notes that I've read there -- well,

certainly two of the things, the renal diet is not being prescribed, nor is the sodium bicarb, and I think that's going to accelerate her progression to dialysis.

Q. Is it your opinion that she probably more likely than not will need a transplant?

A. Oh, yes, I mean if she's going to live. Even with it, I mean your average life expectancy is seven years once you go on -- once you're at end-stage renal disease and require dialysis or transplantation. Now, for the most part, you don't increase the life expectancy much more, but the quality of life is a lot better with a transplant.

So if she makes it into her 60s, she's going to be a very lucky lady. And I have to take my hat off to her that here she is with metabolic acidosis and uremic symptoms and she's still working. I mean that's unusual for a patient who has got this degree of chronic permanent renal damage.

Q. You mentioned something and I want to be sure I understood you. It was something about seven years. Could you flush that out for me? You just talked about that a minute ago.

A. Yes, the average life expectancy for individuals who get to end-stage renal disease requiring chronic dialysis -- and if they're lucky enough, transplantation -- is seven years.
Q. From the beginning of the end-stage renal disease?
A. From the time that they require permanent dialysis.
Q. So --
A. I mean I know the figures, but I can provide you the reference if you want.
Q. Earlier I think you said -- and correct me if I'm wrong -- that she's two to three years away from end-stage renal disease requiring at least chronic dialysis?
A. Correct.
Q. So in your opinion probably her life expectancy is no more than ten years from today?
A. Well, she could be lucky. I mean she's a fighter.
Q. I understand. But life expectancy is generally looking at all people with a similar condition, right?
A. Well, and she may be on the upside of that because of the fact that, you know, she did

follow her -- when it was prescribed at the time she was on dialysis in '98 and part of '99, she was following a low-protein diet and she seems to be a very compliant patient, interested in her own health and her body. And my hunch is that she will be on the upside of the average expectancy than on the downside. That's all.

Q. What would be the upside then?

A. Well, I think out of 1,000 people in the Hopkins program who went on chronic dialysis and/or transplant, I think 4 of them reached 20 years. So let's say 10 to 15 years instead of 7 would be optimistic. So somewhere around 15 years total I guess would be very optimistic, even if she tried to do everything right.

Q. From today?

A. From today. So 65, I guess. And her life expectancy would be somewhere between 80 and 85, so she's probably in my opinion going to lose somewhere between 15 years or so of her life expectancy. I mean that's --

Q. I understand.

MR. MISHKIND: That's good.

Q. What is the success rate currently in getting a kidney transplant? I mean I hear all sorts of

things, you read in the papers.

A. Well, you know, we need more organ donors. That's one thing. You get put on a big list and there are a lot of people waiting right now. And what they try to do is, although the allograft kidneys are without a related donor, you try to get as many antigens matching -- you like to get a five-antigen match, but that's not often possible. And part of it is just luck of the draw.

I mean they get a kidney and they've got this big data bank now, so they know exactly who is the closest match to that. And it sort of depends upon who dies that's an organ donor and what kidney.

Q. It's hard to predict?

A. It's hard to say. I couldn't speculate on that.

Q. Okay. Do you think we've covered all of your standard of care comments about Dr. Cola?

MR. MISHKIND: Let me just object, only because he's written a 13-page report and I'm not sure that you have gone through each and every one of the opinions. Obviously if you have specific questions for him that you've not addressed, I'm not going to put the doctor into

guessing whether you've covered everything.

To the extent that he can recall things, otherwise if you want to put specific questions to him -- I mean, Doctor, don't let me dissuade you from answering. It's just that you've written an elaborate report.

A. I just want to add that I know we haven't covered all of the things in my report and I know we haven't covered things that I will enumerate now in addition, because my report is even -- although extensive, it's a summary.

Q. Well, it's mostly history, right?

A. Well, no, I don't think. But the history is relevant to my opinions for the most part.

Q. I understand.

A. Number one, the failure to inform the patient there was blood in the urine. Number two, the failure to do a complete urinalysis with microscopic once you find the dipstick has blood in it. Number three, his failure to return her phone calls.

Q. If we accept her version of the story, right?

A. No, if we accept what is in the medical record. I mean in the medical record she clearly calls on the 27th -- and once before that, but on the 27th

there's clearly not only a call documenting a severe problem with decreased urine that directly relates to the blood in the urine that's at question here, and boils on her face and her buttocks.

Q. Did you see the LMOM at the end of that note?

A. Yes, but you've got to talk to the patient. You either see the patient right then, you arrange for the patient to come in the next day, say call, I want to see you immediately, or you talk to the patient with these kind of symptoms.

You don't leave a message on a machine that I think it's related to infection and I'm going to give you Augmentin. I mean that's absurd. I mean that is just so ludicrous I can't believe anybody would have it in their records. So it's a total deviation of standard of care not to talk to this lady or see her immediately at that time.

Q. Is your assumption that on that message that was left that they said it's an infection and you need Augmentin? Is that your assumption?

A. My assumption is reading his note, which I'm going to go to his original records. And as I look at it -- and you correct me if you think I'm

wrong, but when I read this, it looks like a different handwriting from the rest of the note here, sounds like bad infection, would recommend treatment Augmentin 500 milligrams No. 20 BID with food. LMOM, 8-27, 5:15 p.m. Refer to neuro for second opinion.

Q. Which is another line, another entry?

A. No, it looks to me like it's continuing the same entry. It's before the 9-1.

Q. Okay.

MR. MISHKIND: But I guess what he wants to know is based upon the record or what you saw in Dr. Cola's depo, are you saying that that specific information was left on the machine or was it just left message on machine for patient and you don't know what was left?

A. I don't know what was left. All I'm saying is what is on this chart here with what is above it and then to not talk to the patient personally or not have the -- preferably have the patient come in, is clear-cut deviation of the standard of care.

Q. Well, if the facts are that the patient was told on the machine by a person in Dr. Cola's office that you need to call in and we need to talk to

you, that certainly is -- the patient needs to follow up with that, right?

A. Well, or you send the patient a letter. And the records of the phone call on 9-4, just a week later, and there's a record of Dr. Torok for joint pain referral, needs referral, patient obviously called in.

So if you don't see the patient, you call again, you send the patient a postcard. The patient called in again for another referral. And whether it's his procedure -- and I can't say that it's him or the way his office runs, this is not the way to take proper care of patients. This is a deviation of the standard of care.

Q. All right. Anything else that we haven't covered?

MR. MISHKIND: Same objection, but you can go ahead and answer.

A. Well, I think that the fact that there are multiple phone calls here that are documented in the chart. He's got another one where he documents the fact that before that call, spoke with patient on the phone 8-20. I have a criticism of that.

He's talked to the patient. He knows

there's blood in the urine from his -- if he didn't have it at the time of his visit, he has it then. He spoke with the patient on the phone the day before she went for her tests at the hospital on the 21st. He should have said, look, you've got some blood in your urine, and in addition to the tests, I'm going to have my secretary call over and order a complete urinalysis with microscopic and a culture and sensitivity of your urine.

And frankly, I would have done that because I would have wanted to get an early morning urine. I would want to get a first voiding specimen, so I would have her go to the lab before she voided that morning.

Q. All right.

A. I mean I think Dr. Cola in his deposition recognizes that he should have followed the 3 plus blood up, that this was appropriate to do a repeat and a urinalysis if it was present. So I think in a number of cases in his deposition he feels that it was appropriate to do more than was done for this patient.

MR. FRASURE: All right, Doctor. I don't think I have any other questions. Thank

you for your time.

THE WITNESS: I'll read.

MR. MISHKIND: And I will get you a copy.

(Whereupon, signature was not waived by the witness.)

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(Deposition concluded at 12:30 p.m.)

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WITNESS CERTIFICATE

I, Thomas M. Zizic, M.D., do hereby certify that I have read the foregoing deposition taken on October 4, 2000, in the case of Vickie Miglore, et al. versus Dr. David Cola, et al., consisting of seventy-two pages, and that said deposition constitutes a true and correct transcription of my testimony given at the specified time.

_____
Thomas M. Zizic, M.D.

Dated this _____ day of _____, 20___.

Sworn to and subscribed before me this _____ day of _____, 20___.

_____
Notary Public

My commission expires _____.

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C E R T I F I C A T E

STATE OF OHIO)
) SS
SUMMIT COUNTY)

I, Christine Leisure, a Registered Professional Reporter and Notary Public in and for the State of Ohio, duly commissioned and qualified, do hereby certify that the within named Witness, **Thomas M. Zizic, M.D.**, was by me first duly sworn to testify the truth, the whole truth and nothing but the truth in the cause aforesaid; that the testimony given was by me reduced to Stenotypy and afterwards transcribed upon a computer, and that the foregoing is a true and correct transcription of the testimony so given by him as aforesaid.

I do further certify that this deposition was taken at the time and place in the foregoing caption specified.

I do further certify that I am not a relative, counsel or attorney of either party, or otherwise interested in the event of this action.

IN WITNESS WHEREOF, I have hereunto set my hand and affixed my seal of office at Akron, Ohio, on this 7th day of October, 2000.

Christine Leisure, RPR (Rodriguez)
Christine Leisure, RPR & Notary Public
My commission expires April 1, 2002.