IN THE COURT OF COMMON PLEAS

CUYAHOGA COUNTY, OHIO

Doc. 185

DIANE M. CARRICK,
Executrix of the Estate
of Michael Carrick,

Plaintiff,

-vs-

JUDGE KILCOYNE
CASE NO. 185330

THE CLEVELAND CLINIC FOUNDATION, et al.,

Defendants.

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Deposition of ROBERT J. HEYKA, M.D., taken as if upon cross-examination before Sandra L. Mazzola, a Registered Professional Reporter and Notary Public within and for the State of Ohio, at the offices of Charles Kampinski Co. L.P.A., 1530 Standard Building, Cleveland, Ohio, at 2:35 p.m. on Thursday, September 6, 1990, pursuant to notice and/or stipulations of counsel, on behalf of the Plaintiff in this cause.

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On behalf of Defendant Lakewood Hospital.

ALSO PRESENT:

Patricia Molle.

ROBERT J. HEYKA, M.D., of lawful age, called by the Plaintiff for the purpose of cross-examination as provided by the Rules of Civil Procedure, being by me first duly sworn, as hereinafter certified, deposed and said as follows:

CROSS-EXAMINATION OF ROBERT J. HEYKA, M.D.

BY MR. KAMPINSKI:

Q. Doctor, would you state your full name, please?

A. Robert Joseph Heyka.

Q. And spell your last name.

A. H E Y K A .

Q. Doctor, I'm going to ask you a number of questions this afternoon. If you don't understand, I'll be happy to repeat any question you don't understand or rephrase or whatever I have to do so that you and I communicate. Okay?

When you respond to my questions, please do so verbally. She is going to take down everything we say and she can't take down nods or gestures,

A. Okay.

Q. Doctor, I've been provided with your CV. Is this up to date?

A. There have been some more recent publications. Other than that, it's up to date.
Q. Why don't you tell me what additional publications.
A. Oh, I can't remember them off the top of my head.
Q. How many are we talking about?
A. Oh, three or four.
Q. Could you provide those to your counsel?
A. Sure.
Q. And then Mr. Gore can provide them to me.
MR. KAMPINSKI: Is that okay?
MR. GORE: Will do.
Q. If at any time you need to refer to the record, please do *so,* okay? It's not a hidden ball trick. If you need to look at something, go ahead.
A. Fine.
Q. Doctor, your CV reflects that you commenced at the Cleveland Clinic in a nephrology fellowship in 1985?
A. Yes.
Q. Had you done any work in nephrology prior to that time?
A. Only as part of the training in my residency in

internal medicine.
Q. Why don't you define what nephrology is for me.
A. Nephrology is a subspecialty of internal medicine or adult medicine that deals with diseases relating to the kidneys.
Q. You've been boarded separately in nephrology. When I say separately, you've also been boarded in internal medicine, correct?
A. Yes.
Q. How long has there been a board for nephrology?
A. I don't know.
Q. Did you pass it the first time you took it?
A. Yes.
Q. And that was November of '88?
A. Yes.
Q. And is that what your practice is limited to?
A. Yes. That and we've combined hypertension and nephrology, so in addition to nephrology, say complicated hypertension problems,
Q. Is there an association between hypertension and nephrology?
A. Yes.
Q. What is that association?
A. Well, there's several really. In any individual center the group within that center that would

deal with hypertension is partly determined by historic interest. In other words, we're cardiologists originally interested in it or we're general internal medicine interested in it, or in our case it was initially the interest of a nephrologist, so historically it's been in our department.

In addition, the kidney releases hormones which have a direct effect on blood pressure, and in turn, the kidney can be damaged by high blood pressure,

Q. Is it important then to have somebody who has kidney disease and high blood pressure, to have their blood pressure controlled?

A. Yes.

MR. KAMPINSKI: Just show Mr. Gore's associate entered the room.

Q. Doctor, have you had an opportunity before coming here today to review either Dr. Riley's records as they pertain to Mr. Carrick or his deposition?

A. I've reviewed a summary of his deposition, yes. I've not seen any other records.

Q. Did you at the time that you undertook to treat Mr. Carrick see any of Dr. Riley's records?

A. I don't think so. I'm not certain. I think all the records that we had were transferred from Lakewood Hospital. I don't think I saw any independent prehospitalization records.

Q. All right. There was one letter by you to Dr. Riley. Would those be included in the Clinic records?

A. Yes, they would be under the correspondence section, I'm sure.

Q. All right. There was a letter that you sent, I believe, Dr. Riley dated April 4, 1989. Here. You can take a look at this one. The bottom paragraph of it refers to receipt of background information.

A. Uh-huh.

Q. What is it you're referring to?

A. I don't recall. As I said, it would -- most likely it was the information that was transferred with the hospital transfer from Lakewood. But 1 don't recall if I had seen any earlier information on Mr. Carrick or not.

Q. Did you have any a separate discussions with Dr. Riley either by phone or in person regarding Mr. Carrick?

A. I don't recall.

Q. Do you recall Mr. Carrick?
A. Oh, yes.
Q. Okay. You have separate recollection of him other than what's contained in the records?
A. Yes.
Q. Would you have been his attending at the time of his transfer to the Clinic?
A. Yes, I was.
Q. The discharge summary from the first hospitalization was dictated by someone other than yourself. That was a Dr. Carey?
A. Yes.
Q. And who is he?
A. He was the resident on my service at the time of the hospitalization.
Q. Was he the one that actually took the history from Mr. Carrick?
A. He was one of the people who took the history. I also took a history, and I'm sure several other physicians during his hospitalization as well.
Q. What was Mr. Carrick transferred to the Clinic for, Doctor?
A. The problem was renal failure and possibly needing hemodialysis, as well as the question of

whether there was muscle inflammation or myositis, which had been suggested on a muscle biopsy that was done at Lakewood.

Q. Did you ever describe his primary problem as osteodystrophy?

A. Yes.

Q. And what's that?

A. That is a general term that describes a group of different bone abnormalities related to chronic renal failure or chronic damage to the kidneys.

Q. And is that what Mr. Carrick had?

A. Yes. In my -- yes.

Q. When you say chronic damage, I distinguish that from acute. I assume you do as well.

A. Yes.

Q And in terms of the history, were you able to determine how long the damage to his kidneys had been ongoing?

A. No, not any definite date. The damage that had been done is that as typically seen over years, but 1 couldn't put an exact date on it. In general terms when a person's renal function is about 50 percent of normal or serum creatinine of about 2.0 milligrams per dl., there starts to be changes in the bones that we call renal

osteodystrophy. So I can define it in general terms, but in his particular case I don't know how long.

Q. Do you know how long he had had serum creatinines greater than 2?

A. No.

Q. Is there any particular drug that's a treatment of choice for progressive kidney failure?

A. No.

Q. What is Allopurinol?

A. That's a drug that technically is what's called a xanthene oxidase inhibitor. It's useful to -- xanthene oxidase is a precursor to uric acid which causes gout. Allopurinol is used in decreasing the accumulation of uric acid and the likelihood of gout by interfering with the production of uric acid.

Q. What's gout?

A. Well, I'll answer as an internist. I'm not a rheumatologist. Gout is a disorder of uric acid, which is a waste product in the body, a disorder of uric acid metabolism, either increased production or decreased secretion. And the uric acid crystals settle into joints and cause acute inflammation of joints.

Q. Is there an association with any particular level of uric acid secretion and kidney disease?
A. I don't think I understand.
Q. All right. Are uric acid elevations in the 10 to 14 range associated with increasing kidney disease?
A. They are seen in kidney disease, yeah. They're -- associated doesn't mean one causes the other. But they are seen in chronic --
Q. Are you saying that this is a separate process going on in the body that just happens to be seen at the same time or --
A. Well, uric acid is one of the many waste products in the body that's secreted through the kidney. So any time the kidney is not performing the secretory function, there will be the accumulation of uric acid in the body. Now, that in and of itself does not cause a gout attack and does not mean a person has gout. It's a mark that there is decreased function of the kidneys.
Q. Did Mr. Carrick have gout in your opinion?
A. Yes.
Q. What is the treatment, Doctor, for progressive renal failure?

A. well, again that's a generic term. It means that there has been irreversible, progressive over time, six months or more, deterioration in kidney function. Now, within that there is categories like hypertension, nephritis, there is toxins, there is infections, there's a large list of things that can cause chronic renal failure, Chronic renal failure is just the state where the kidneys have been progressively and irrevocably damaged.

Q. Once again, how do you treat it?

A. Well, if it's caused by hypertension or inflammation, depending what the disease is, you might use, for example, prednisone or you might just control a person's blood pressure.

If you can find an underlying disease causing the kidney problems that you can do something about, you try and treat the primary disorder *so* that it doesn't damage the kidneys any more,

Q. How would you go about finding an underlying process, assuming that you suspected the existence of chronic renal failure?

A. At first you would suspect it, and then either on the basis of a history, physical examination

or kidney biopsy, you would determine what the actual cause was. Not everyone who has renal failure needs a kidney biopsy. If it's unclear from the history and physical examination, we sometimes use a biopsy.

Q. Do you know what the cause of Mr. Carrick's kidney failure was?

A. No. What typically happens at the stage where Mr. Carrick was when I saw him is that you get a generalized picture of small shrunken kidneys with lots of scarring, but multiple processes can lead to that end stage picture.

At that point it's very difficult to try and isolate which of many processes could have gotten you to where you were at that point.

Q. So do I understand you correctly to say that in terms of trying to deal with it then early, you have to do some investigation earlier in the process than when you saw Mr. Carrick?

A. Yes. It was irreversible. Chronic renal failure is irreversible by definition.

Q. Were you able to determine whether or not there had been any investigation to determine what was the cause of --

A. No. I was unable to determine --

Q. Let me finish it -- what was the cause of Mr. Carrick's renal failure?
A. No.
Q. would you agree that the syndrome of progressive renal failure, gouty attacks and uric acid levels of 12 to 14 is a well-described entity?
MR. KELLY: Excuse me, Chuck. Can you repeat that question? I'm sorry. I didn't hear it.
MR. KAIMPINSKI: Sure.
MR. KELLY: Thank you.
Q. Would you agree that the syndrome of progressive renal failure, gouty attacks and uric acid levels of 12 to 14 is a well-described entity?
A. I still don't understand. Do you mean the three of them all being related and causing the kidney damage, or do you mean all three occurring in the same person and not necessarily being related?
Q. Well, I think you told me that the increased uric acid level doesn't necessarily cause kidney damage.
A. Right.
Q. Okay. So I guess my answer to your question of me is no, I don't mean that they all cause

kidney damage.

A. I have seen then -- yes, I've seen patients with chronic renal failure and elevated uric acid who also have gout.

Q. Would the treatment of such a patient involve dietary protein restriction, Allopurinol to reduce uric acid to a normal range, and discontinuing potential toxins, and strict control of blood pressure?

A. Those combinations might not be useful in every patient, but what you've mentioned are useful treatments in some patients, yes.

Q. All right. I apologize if 1 asked this before and you answered, But is Allopurinol the treatment of choice for the reduction of uric acid levels? And I may have asked it differently before.

MR. GORE: You asked a similar question. But go ahead, Doctor.

A. Yes.

Q. Indocin is a nonsteroidal antiinflammatory drug, is it not?

A. Yes.

Q. Could you tell me whether or not that is a cause for acute and chronic kidney failure?

A. Yes, it is. Both.
Q. And is it contraindicated in patients who have creatinines in excess of 2?
A. No.
Q. Where would it be contraindicated?
MR. GORE: In terms of creatinine?
MR. KAMPINSKI: Yes.
A. I don't think it's ever contraindicated. I think if a person needs a nonsteroidal antiinflammatory agent for treatment of arthritis or gout or whatever, and you think that's the best drug to use, then you give that drug, but at the same time you closely monitor the renal function. There are --
Q. When you say closely monitor, what are you talking about? Give me some examples or ideas.
A. I would continue to monitor on a regular basis the person's serum creatinine, look at their urinalysis and monitor their blood pressure.
Q. Is there any time frame that one would not give Indocin to somebody having kidney failure? And when I say time frame, I mean years, months, weeks. Would you not give it for more than a couple weeks, for example?
A It would depend on the circumstances. I can't

make a blanket statement for all situations.
Q. Would you give it for 15 years in a patient with kidney disease?
A. Would I give it for fifteen years?
Q. Yes.
A. No.
Q. Should anybody give it for 15 years?
MR. KELLY: Objection. Go ahead.
Q. Let me ask it differently, Doctor. Do you have an opinion to a reasonable degree of medical certainty based on probabilities as to whether or not the standard of care required of a physician in dealing with a patient having renal disease, whether or not the appropriate standard of care is breached by prescribing Indocin to that patient for a period of 15 years continuously?
A. I think it depends on again if that's the treatment that you think is necessary, and if you think there's no option to that treatment, if that's what the patient needs, then you give them the medication that they need, but at the same time you have to closely monitor the effects of any treatment.
Q. And what effects would cause you to stop that

treatment?

A. If I saw a rise in serum creatinine, protein in the urine, or a rise in the blood pressure, then I would look to alternate treatments besides the nonsteroidal agent.

Q. Is that because that would reflect to you that there was an adverse effect on kidney function?

A. Yes.

Q. And if in fact you saw such an adverse -- well, if in fact such an adverse effect was occurring, would it be below the standard of care under those circumstances to continue to prescribe it?

MR. KELLY: Objection.

A. The only circumstance I can foresee where it would be appropriate to continue is if you had exhausted all other options and in order to treat the patient, that was the only option you had to treat their underlying disease, Then you would make a choice between damage to the kidney and treatment of the other disorder that you wese using the Indocin for.

If in the balance sheet your determination was it was more important to continue to give the Indocin in spite of the changing kidney function, then you would continue to give the

Indocin. That would be the only circumstance.
Q. When you say the underlying disease, now you are talking about the gout?
A. Whatever you're using the Indocin for.
Q. Assuming it was gout?
A. Assuming it was gout.
Q. And what would be the alternative treatments?
A. Well again, I don't claim to be an expert in the treatment of gout. In general, the alternate treatments include, in addition to nonsteroidal inflammatory agents, Allopurinol, prednisone, different cortical steroid preparations, and a group of agents called uricosuric agents that increase the secretion of uric acid in the kidney. Those are the four general classes for treating gout.
Q. Do you know why Dr. Riley stopped using Allopurinol with Mr. Carrick?
A. No.
MR. KELLY: Objection.
Q. Can high blood pressure be caused by narrowing of kidney arteries?
A. Yes.
Q. Did Mr. Carrick have narrowing of kidney arteries?

A. We never did a definitive study, which would have been injecting contrast material into the kidneys with a so-called renal arteriogram to look for it. On the basis of physical examination I was not suspicious of that as a possibility.

Q. Did he have a unilateral small kidney?

A. I'd have to review the chart.

Q. Go ahead.

A. I think in his first hospitalization there was an ultrasound report. The report in the chart says, Findings consisting with medical renal disease. Question enlarged right adrenal gland. There's no mention of a unilateral shrunken kidney compared to the other one.

Do you have the hospitalization one for the first -- there it is, the first hospitalization.

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(Thereupon, a discussion was had off the record.)

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MR. GORE: Here is a copy, do you want this?

A. No. It would have been a copy of the radiology

report on the first hospitalization.
Q. Well, let me ask a question then and see whether it would matter to you anyhow. I mean if he had a unilateral small kidney, would that be an indication or one indication that there was narrowing of one or both kidney arteries causing high blood pressure?
A. That would be one of the possibilities that could give you a unilateral shrunken kidney but that's not the only possibility.
Q. Okay. Would poorly controlled blood pressure and a rise in serum creatinine in conjunction with the unilateral small kidney increase your index of suspicion of that being the cause of high blood pressure?
A. No.
Q. If that were --
MS. HENRY: Pardon? I didn't get his answer.
A. No.
Q. And no test was done to determine that so you just don't know whether that was a cause of his high blood pressure or not?
A. If as it was written in the chart, both kidneys were small and shrunken, which is more

consistent with a systemic process involving both kidneys, not just a single kidney, as one would see with chronic renal failure.

Q. Well, okay. I apologize if there's any confusion in my question, but by the time he got to you, I mean he was in chronic renal failure and was not reversible, and I take it he was in bad shape, correct?

A, That was my assessment, yes,

Q. Sure. Ten years before he got to you, say in 1980, would he have had both kidneys shrunken at that time?

A. It would have -- the kidneys shrink when the kidneys are damaged, so it would have depended on how damaged the kidneys were at that time. I don't know what that -- how damaged they were in 1980.

Q. If hypothetically his high blood pressure were caused by narrowed kidney arteries, is that treatable?

A. Again, there's lots of different reasons that the kidney arteries could be narrowed, Some are treatable and some are not.

Q. Okay. Treatable ones would be treatable how, medication, surgically?

A. Either medication or in some cases surgery.
Q. Is it important if a person has high blood pressure and kidney failure to analyze whether or not the two are related?
A. I don't understand. The two, you mean high blood pressure and kidney failure?
Q. Yes, sir.
A. I think it depends on how badly damaged the kidneys are. There's no doubt that uncontrolled blood pressure is the No. 1 cause of continued worsening of kidney function, so whether one caused the other or not, it's important to control the blood pressure when there is chronic renal failure.
Q. Did you ever determine how Mr. Carrick's blood pressure had been controlled prior to his coming to the Cleveland Clinic?
A. No.
Q. Were you able to do that in looking at the summary of Dr. Riley's deposition? And let me throw in, I mean anything that you derived from that deposition prior to coming here today, go ahead and use that if it forms a predicate for any answer to any one of my questions.
A. I reviewed it a while ago. I don't have much

recollection from the deposition that there was anything in there about the hypertension.

Q. Did you know Dr. Riley prior to Mr. Carrick coming to the Cleveland Clinic?

A. I'd spoken with him over the phone once or twice, I've never met him.

Q. In what context3

A. Patient referrals of his that had come to the Clinic.

Q. Have there been patient referrals since Mr. Carrick by Dr. Riley?

A. I'm not sure.

Q. Was he a nephrologist?

A, I don't know what you mean by a nephrologist.

Q. Somebody who has studied nephrology. Well, all right. Let me withdraw that,

He indicated that there was no nephrology board at the time that he went to school and that he did not do a residency in nephrology, but that he apparently has attended some seminars at the Cleveland Clinic dealing in nephrology. Does that make him a nephrologist?

A. Again, it depends on how you define nephrologist.

Q. Well, why don't you define it. What's a

nephrologist to you?

A. If we define nephrologist either as someone who has taken a nephrology fellowship and then passed the nephrology boards, then he's not a nephrologist.

Q. Well, how else would you define it?

A. That's the way I would define it. That isn't necessarily the way everyone else defines it.

Q. n taking a history did it matter to you whether or not the hypertension was adequately controlled prior to his coming to the Clinic?

A. Yes, it was important from a historical perspective. What I thought was more important was what happened from the day when he came to the Clinic, from that point on, because the damage that had been done would not be reversible. So it was less important what had happened in the past.

Q. Do you have any opinions, Dr. Heyka, based upon anything that you derived either in your treatment of Mr. Carrick or the history taking or in your review of the deposition of Dr. Riley which you believe fell below the standard of care in terms of the treatment by Dr. Riley of Mr. Carrick?

MR. KELLY: Objection.

MR. GORE: Go ahead.

A. I think that in a patient such as Mr. Carrick with chronic renal failure, ,it's important to anticipate bone disease and to follow the measures that are important in preventing the bone disease, and from what I was able to surmise, treatment of his renal bone disease was not -- was not adequate.

Q. All right. When you say anticipate it, how would you anticipate -- I mean what would you do in order to anticipate it?

A. well, as I said earlier, if you take people who come needing dialysis or kidney transplant, close to a hundred percent will have underlying bone disease, and in most studies that bone disease starts when the renal function is about 50 percent of normal. So whenever a person's, creatinine is much above 2 milligrams per dl., or their renal function is 50 percent of normal, it's a good assumption that there is incipient underlying bone disease.

Q. And you said anticipate and follow. How do you follow it then when this occurs?

A. Well, the treatment involves several lines of

treatment. The first thing is to monitor two electrolytes in the bloodstream, one called calcium, the other called phosphorous.

And the first priority is to keep the phosphorus level under control. Once that is done, then you add supplemental calcium to get that to a normal level, and then you may add other medications like vitamin D to protect the bones also.

Q. And should that have been done with Mr. Carrick?

A. In my opinion it should be done with every patient who has chronic renal failure,

Q. Was that done with Mr. Carrick?

A, Not that I could determine when I saw him for the first time at the Clinic.

Q. And the failure to anticipate and follow the bone disease, do you believe that that contributed to cause Mr. Carrick's death?

MR. KELLY: Objection.

A. Yes.

Q. Was he a candidate for transplant?

MR. GORE: Objection. For clarification, when he came to the Clinic?

Q. Well, it's an appropriate clarification and let me ask it two ways. First let me ask it when he

came to the Clinic.

A. In general a person is a candidate for transplant when they reach ESRD or end stage renal disease. Mr. Carrick had not yet reached end stage renal disease, but over the next several weeks he did so. At that point he was a candidate for transplant assuming he was medically able to undergo the surgery.

Q. Which he wasn't?

A. Not in my opinion.

Q. Doctor, assuming he had received appropriate care by Dr. Riley in terms of what you indicated he should have received, that is, anticipating and following his bone disease, would he have ultimately nonetheless needed a transplant at some point in time?

A. I don't know for sure because again that would be dependent on the underlying renal disorder he had.

Q. Which we don't know what it was?

A. which we don't know.

Q. Okay. All right. Let's take it one step further. Assuming that it was one that would have required a transplant, would he have been a candidate for a successful transplant?

A. Once he was medically stable, which he was not when I saw him, yes, he would have been a candidate for a transplant or for any form of dialysis,

Q. Why was he not in your opinion medically stable at the time you saw him?

A. Well, I think there was a long list of reasons. He had diffuse renal bone disease with calcium deposits in his shoulders, along his blood vessels, in his heart, in his soft tissues. His gout was not under control. He was -- his blood pressure was elevated and he had some degree of malnutrition,

Q. He had been transferred from Lakewood Hospital, is that correct?

A. Yes.

Q. Do you know if that was because Dr. Riley wanted him transferred or because the family wanted him transferred? I mean you may not know.

A, I don't know.

Q. There was a diagnosis made of secondary hyperparathyroidism, is that correct?

A. Yes.

Q. And who made that diagnosis?

A. I did.

Q. On what basis?
A. Well, as I mentioned earlier, renal osteodystrophy or renal bone disease involves three or four separate entities. The most common one is what's called secondary hyperparathyroidism That diagnosis was made on the low calcium, the high phosphorus, the extremely high PTH level, and then ultimately on the pathology evaluation of the parathyroid glands that were removed.
Q. So it was confirmed surgically, is what you are saying?
A. Yes. And I should also say on the basis of radiological extra abnormalities were also --
Q. And that is always seen in patients with renal function less than 50 percent?
MR. GORE: Secondary hyperparathyroidism?
MR. KAMPINSKI: Yes.
A. If you look hard enough, studies have shown you will begin to see it at that stage.
Q. What's the treatment for renal osteodystrophy?
A. Well, again, within that category of renal osteodystrophy, you're talking about secondary hyperparathyroidism. The initial treatment is

to control the serum phosphorus.

Q. To lower it?

A. To keep it between 4 to 6 milligrams per dl.

Q. And what was it in Mr. Carrick?

A. I think it ranged between 8 and 10.

Q. So initially would you want to lower it?

A. You would lower it.

Q. Go ahead.

A. That would usually be done with some form of antacid treatment taken with meals that would help lower the extra phosphorus in the diet so that less was absorbed. Once the phosphorus level could be -- and it would also entail changing the amount of protein and dairy products in the diet because they are high in phosphorus, so avoiding high phosphorus foods and using an antacid to decrease absorption of phosphorus.

Once the phosphorus was in the range of 4 to 6 milligrams per dl., the next goal would be to normalize the serum calcium to between, oh, 9 and 11 milligrams per dl.

Q. So that would be calcium supplementation?

A. Yes. And then the third goal, assuming there was evidence of secondary hyperparathyroidism,

would be some form of vitamin D.

Q. How about aluminum?

A. I don't understand the question,

Q. Well, would you want to reduce the aluminum burden if it was present?

A. Yes.

Q. Was there any testing done to determine if there was an aluminum burden present?

A. No.

Q. Why not?

A. The aluminum that a person with renal failure accumulates gets there two ways. Either a person is already on dialysis and the water is contaminated with aluminum. That's no longer a problem because that's monitored in dialysis units. Or the person ingests large amounts of aluminum. That usually occurs by taking aluminum antacids to control the phosphorus.

There was no historic evidence that Mr. Carrick had ever taken any antacids on a regular basis to control his phosphorus. So I had no historic evidence that he had ever been exposed to aluminum to think he had an aluminum overload.

Q. And we know he wasn't on dialysis so you didn't

have to worry about that?
A. Right.
Q. What's Dialume?
A. Dialume is an antacid that would be used to control the serum phosphorus. It's taken with meals. Generically it is aluminum hydroxide.
Q. He was given Dialume on admission, wasn't he?
A. Yes.
Q. Well, then he was receiving aluminum, correct?
A. Yes.
Q. Large doses?
A. No. The buildup that is necessary to cause aluminum overload takes several years.
Q. I see. And all the treatment you've told me so far has been medical as opposed to surgical?
A. Yes.
Q. Well, is dialysis also included in that treatment?
A. No.
Q. Why not?
A. Dialysis in and of itself does not prevent the progression of underlying bone disease. Things in addition, the things that I've mentioned, have to be done in addition to dialysis to protect the bones.

Q. So it would be what you stated in addition to dialysis would be the appropriate treatment?
A. Yes.
Q. Was any of that tried on his admission to the Clinic?
A. Yes. As you mentioned, he was put on Dialume to try and control his serum phosphorus. As I mentioned, that is the first goal. So we would not add calcium supplementation if the phosphorus was under control. And he did require intermittent hemodialysis at different times during his two hospitalizations at the Clinic.
Q. Well, was he dialyzed during his first hospitalization at all? You can look.
A. No. His first dialysis was April 14.
Q. When was the surgery, the parathyroidectomy?
A. The surgery was April 11,
Q. So it was after surgery?
A. Yes.
Q. Well, is there a reason that dialysis was not done before surgery? Or tried, I should say.
A. Well, again, the reason for initiating dialysis is to remove the waste products from chronic renal failure. At the clinical point where a

person had decreasing urine output and side effects from the accumulated waste products, then dialysis would be initiated.

Q. Okay. You lost me.

A. There was no clinical indication to start dialysis before that time.

Q. Well, did he have renal osteodystrophy?

A. Yes.

Q. Okay. Now, I'm real confused. I thought you just told me that the initial way to try to treat it is medically which includes dialysis?

A. No.

Q. Oh, okay. Then you tell me.

A. There's two separate questions. Dialysis is used when a person doesn't make enough urine or when they become sick from waste product accumulation. Because dialysis is not effective in preventing renal bone disease, I said you still have to continue doing everything that you did before independent of whether a person is on dialysis. The dialysis is because their kidneys can no longer function on their own, but will have no independent effect on the underlying bone disease.

Q. Okay. What you are telling me, I think, is that

you have to try dialysis in conjunction with these other modalities, that is, the lowering phosphate intakes, vitamin D supplementation and calcium supplementation, correct?

A. I don't understand.

Q. You try to supplement the calcium, correct? Did you give dietary counseling to lower phosphate intake?

A. Yes.

Q. And was there vitamin D supplementation?

A. No.

Q. Why not?

A. Well, as I mentioned, the first goal in treatment is to control the phosphorus between 4 to 6. There is something called a calcium phosphorus product, If you multiply calcium by phosphorus and the value is greater than 70, it's likely the calcium will not return to bone but will be deposited in spots outside of bone. It's called extra-skeletal calcification,

Mr. Carrick's calcium phosphorus product was above 70, *so* the first goal was to get the phosphorus under control before we did anything else.

Q. Did you do that?

A. We attempted to with the Dialume, yes.
Q. And did it work?
A. No.
Q. Is that why none of the other steps were undertaken then?
A. Yes.
Q. And that's why the parathyroidectomy was undertaken?
A. Yes. There was already evidence that his calcium phosphorus product had been elevated long enough to cause calcification in the heart valves, in the shoulder, along the blood vessels, and that is usually not reversible without taking the parathyroid glands out.
Q. Should in your opinion Mr. Carrick have received a nephrology consult prior to his being admitted to the Cleveland Clinic?
MR. KELLY: Objection.
A. I think he should have been seen by someone who could better treat his bone disease, and likely a nephrologist, yes.
Q. Was Mr. Carrick receiving prednisone when he was admitted to the Clinic?
A. Yes.
Q. And that was for what reason?

A. I don't know.
Q. All right. That had been prescribed when he was at Lakewood, correct?
A. Yes. The record says it had been for two weeks.
Q. Does that have an effect on BUN?
A. Yes.
Q. Raises it, correct?
A. Yes.
Q. Acutely?
A. Yes.
Q. And does it cause steroid myopathy?
A. Yes.
Q. What's that?
A. Well, myopathy refers to damage of the muscles and steroid myopathy means that if you do a biopsy, there's been damage to the muscles caused by steroids a person is taking.
Q. You called in a rheumatology consult, correct?
A. Yes.
Q. And who was that3
A. Who was it?
Q. Yes.
A, I can't make out the signature.
Q. All right. You don't recall who it was?
A. No.

Q. The reason you called in a rheumatology consult was for his gout?
A. Yes.
Q. He discontinued naproxen, continued prednisone ten milligrams a day and obtained a muscle biopsy, correct?
A. No.
Q. What did he do?
A. Aspirated right knee, looked for crystals, inject steroids into the knee, give a PTH, and then start Allopurinol once attack has resolved. The muscle biopsy was done at Lakewood.
Q. I'm sorry. You're right. What's CPK?
A. That's an enzyme in the body. It stands for creatinine phosphorus kinase that comes from three major sources, the skeletal muscle, heart and brain.
Q. And what was his CPK level on admission to the Clinic?
A. The CPK on March 29 was 50% international units per liter.
Q. It had been 1600 at Lakewood. Were you aware of that?
A. Yes.

Q. What do you attribute the reduction to, if anything?
A. Well, I think when he presented to the Clinic, there was a question of myositis or inflammation of the muscles. That's why the biopsy was initially done. Another alternative is that he was receiving injections into his muscle which can cause similar inflammation and similarly raise the CPK.
Q. Injections of prednisone?
A. Of anything.
Q. I see. Sorry. Go ahead.
A. And because it resolved during his hospital stay and if the biopsy did not show any myositis, it is our presumption that the elevated CPK was due to intramuscular injections and not myositis.
Q. Who placed him on Allopurinol?
A. At what point?
Q. During his stay at the Clinic. Was that you or was that the rheumatologist or was that at the Clinic?
A. It was rheumatology.
Q. The same person whose name you can't read?
A. Yes.
Q. Can you see it in the orders as opposed to the

consult?
A. No, I can't make the staff doctor's name out.
Q. Why did he put him on Allopurinol, do you know?
A. Well, because he was using it to treat his gout. He was giving 100 milligrams every other day in conjunction with Colchricine and steroids into the joint.
Q. The prednisone?
A. The oral prednisone -- it was different -- they were putting cortisone into the joints that were inflamed.
Q. What were his BUNs prior to surgery? Were they in excess of 1501
A, You mean the second admission?
Q. Yes. Well, you can look at the first also, I think.
A. Yes. On March 29 his BUN was 150 milligrams per dl.
Q. Okay. And then how about prior to surgery?
MR. GORE: Was the surgery the llth?
MR. KAMPINSKI:: Yes.
MR. GORE: Here it is, on the 10th.
A. On April 10 his BUN was 224 and creatinine of

6.2,

Q. What is the BUN a measure of?

A. Well, the term stands for blood urea nitrogen. It's a measure of urea, which is a breakdown product in protein metabolism.

Q. And what does a BUN in excess of let's say a hundred mean for the risk of somebody who is going to undergo surgery?

A. The BUN can be elevated for several different reasons. One is because the kidneys are not secreting the BUN and it's accumulating in the blood. The other reason is because a person is malnourished and they're metabolizing their protein because they're malnourished, So it depends why the BUN was elevated.

Q. Why was it elevated?

A. Well, my initial assessment was that the serum creatinine, which is another marker of kidney function, was in the range of 5 to 6 milligrams per dl., *so* that the BUN was markedly elevated compared to the creatinine. I thought the BUN was more reflective of muscle protein breakdown from malnutrition and from the steroids than reflecting underlying kidney function.

Q. Because of the serum creatinine level?

A. Yes. That in addition to the urine output.
Q. Which was adequate, inadequate?
A. Which was adequate.
Q. All right. If in fact it's due to malnutrition, are you saying then that there is no increased risk for surgery?
A. No, I'm not saying that. There's definitely an increased risk for surgery with malnutrition, yes.
Q. Well, okay. That may or may not have really been my question. Is the increased BUN a cause for concern for the surgery as opposed to the malnutrition if the BUN is caused by malnutrition?
A. The BUN is only of concern as a marker for something else going on, not being intrinsically toxic.
Q. Is a BUN in excess of 100 reflective of an increase in risk of bleeding during surgery?
A. You're saying an isolated BUN elevation?
Q. Not isolated. I mean this wasn't isolated. I mean he had a BUN, I think you said, of 150 something during his first admission.
A, What I mean --
MR. GORE: I think he means do you

want him to just consider that one factor.
Q. Yes, yes. I'm sorry. Yes.
A. Yes, there could be increased risk of bleeding for surgery.
Q. Who cleared Mr. Carrick for surgery? Would it have been yourself, an anesthesiologist?
A. I can't say for certain. Likely there would have been two or three people that cleared him.
Q. Why don't you take a look and let me know who they were.
A. Looks like he was seen by multiple people. He was seen by Dr. Nakamoto from the renal service, He was seen by rheumatology. He was seen by general surgery.
Q. Did you remain his attending though?
A. The clinic system is divided into physicians in the hospital and physicians in the outpatient.
Q. Okay.
A. So I had seen him on the initial hospitalization and then saw him in the outpatient department. At the time he was readmitted another physician was on the hospital service, so I was the physician who decided to admit him for the parathyroidectomy. There was another physician on the hospital service when he was readmitted.

Q. All right. Does that mean that the attending was someone other than yourself for the second hospitalization?
A. Yes.
Q. And who was that?
A. He was admitted to Dr. Broughan.
Q. Broughan. How do you spell that?
A. B R O U G H A N .
Q. Is he the doctor who did the parathyroidectomy?
A. Yes.
Q. And would he have been the doctor then that would have followed up postoperatively?
A. Yes.
Q. You mentioned Dr. Nakamoto. You and he wrote a paper together, I *see,* is that correct?
A. Yes.
Q. What was his -- a couple of them actually.
A. Pardon?
Q. Couple of them,
MR. GORE: You wrote a couple papers together.
A, Yes.
Q. What was his role in the treatment of Mr. Carrick?
A. Well, Dr. Nakamoto is in charge of the

in-hospital dialysis unit and at the point where it was decided Mr. Carrick needed dialysis for reasons independent of his bone disease, Dr. Nakamoto arranged for his hemodialysis.

Q. That would have been subsequent to the surgery?

A. Yes.

Q. What were the reasons?

MR. GORE: For the dialysis?

MR. KAMPINSKI: Yes, sir.

A. The reasons for dialysis on that day were hyponatremia, or low serum sodium, hypocalcemia secondary to the operation, and metabolic acidosis, or buildup of acid in the bloodstream.

Q. Did you have any role in the postoperative period of Mr. Carrick?

A. No.

Q. Did you even see him?

A. Yes.

Q. But what, just to see how he was doing, not as a treating physician?

A. Yes.

Q. Just so there is no confusion, yes, you saw him just to see how he was doing?

A. Yes.

Q. No, you did not see him as a treating physician?

A. That's right.
Q. So in terms of discussing what occurred postoperatively, I would be better off talking to someone who did see him as a physician postoperatively?
A. Yes.
Q. The decision to have him undergo a parathyroidectomy, whose was that?
A. Mine.
Q. And tell me once again why you made that decision.
A. The main reason for undergoing the surgery was that I did not believe medical treatment would be successful in either controlling or reversing his underlying renal bone disease, It would require surgery to turn his bone situation around.
Q. And his bone situation was in that state in your opinion because he hadn't been treated appropriately prior to his admission to the Cleveland Clinic by Dr. Riley, is that correct?
MR. KELLY: Objection.
A. Yes, that's my opinion.
MR. KAMPINSKI: That's all the questions I have of the doctor. Some of the

other attorneys may.

MR. GORE: Mr. Kelly represents the two doctors and he may question you if he wishes to.

MR. KAMPINSKI: Before I finish off, George, if possible, I would like a complete copy of the Clinic record.

MR. GORE: No problem.

MR. KAMPINSKI: They're not Bates stamped, are they?

MR. GORE: No. What I'm going to do -- I don't care if it's on the record or off. There's so many different things here. I'm going to get the medical records people to put this thing together in proper order and then I'm going to have one of my people number the pages and then I'll give it to you.

MR. KAMPINSKI: Fine. And I assume the other attorneys may want one, too,

MR. KELLY: Yes. In lieu of asking questions, I'm just going to make a simple statement for the record.

I'm covering this for both Mr. Fifner and Mr. Spisak from my office who represent Drs. Zein and Riley respectively. They had both

originally planned to be here. Due to unanticipated late developments, they asked me to cover for them recently and they have also asked me reserve the right to recall this witness on the record, whether that meets with objection or not, should they decide that that's necessary for them to do so.

Other than that, I have no questions,

MR. GORE: Well, for the record, this was scheduled as a discovery deposition for all parties and we can talk about any future recall--

MR. KELLY: I understand.

MR. GORE: -- but I don't think it's going to be appropriate.

MR. KELLY: It may not be necessary.

MR. GORE: Miss Henry represents Lakewood Hospital.

MS. HENRY: I have no questions.

MR. KAMPINSKI: I assume you want him to read it?

MR. GORE: Yes.

ROBERT J. HEYKA, M.D.

C E R T I F I C A T E

The State of Ohio,) SS:
County of Cuyahoga.)

I, Sandra L. Mazzola, a Notary Public within and for the State of Ohio, authorized to administer oaths and to take and certify depositions, do hereby certify that the above-named ROBERT J. HEYKA, M.D., was by me, before the giving of his deposition, first duly sworn to testify the truth, the whole truth, and nothing but the truth; that the deposition as above-set forth was reduced to writing by me by means of stenotypy, and was later transcribed into typewriting under my direction; that this is a true record of the testimony given by the witness, and was subscribed by said witness in my presence; that said deposition was taken at the aforementioned time, date and place, pursuant to notice or stipulations of counsel; that I am not a relative or employee or attorney of any of the parties, or a relative or employee of such attorney or financially interested in this action.

IN WITNESS WHEREOF, I have hereunto set my hand and seal of office, at Cleveland, Ohio, this ____ day of _____, A.D. 19 __.

_____
Sandra L. Mazzola, Notary Public, State of Ohio
1750 Midland Building, Cleveland, Ohio 44115
My commission expires January 6, 1992