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Larry J. Chilton
Chilton Yambert & Porter, LLP
150 S. Wacker Drive, Suite 2400
Chicago, IL 60606

RE: Lebron, P. vs. Shell Oil Company
Oral Deposition of: Ethan A. Natelson, M.D.
Date Taken: November 11, 2008

Dear Mr. Chilton:

Please find enclosed the ***condensed transcript*** *with Changes and Signature page of the above-referenced deposition. Please have your client review the deposition and make any changes to form or substance on the Changes and Signature page. Do not make any corrections or markings inside of the transcript.* ***The witness's signature must be notarized.***

Please return the original transcript within ***30 days*** *from the date of this letter. Your prompt attention to this matter is greatly appreciated. If you have any questions, please do not hesitate to contact us.*

Sincerely,

Client Services Representative
713-524-4600

Job Number: 99582

CC: R. Dean Hartley
Robert E. Gilmartin, III
James Miller

08-E-99582 sh

STATE OF ILLINOIS
IN THE CIRCUIT COURT OF COOK COUNTY, ILLINOIS
COUNTY DEPARTMENT-LAW DIVISION

PAMELA LEBRON, as Personal Representative of the Estate of MICHAEL (MIGUEL) LEBRON,	)	
Plaintiff,	)	Civil Action No. 05 L-9246
vs.	)	
SHELL OIL COMPANY, et al.,	)	
Defendants.	)	

****

TELEPHONIC DEPOSITION OF

ETHAN A. NATELSON, M.D., F.A.C.P.

November 11, 2008

Volume 1

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TELEPHONIC DEPOSITION of ETHAN A. NATELSON, M.D., F.A.C.P., produced as a witness at the instance of the Plaintiff, Shell Oil Company, and duly sworn, was taken in the above-styled and numbered cause on the 11th of November, 2008, from 1:07 p.m. to 3:22 p.m., before Shanon M. Hair, CSR in and for the State of Texas, reported by machine shorthand, at the offices of Schirrmeister Diaz-Arrastia Brem, LLP, Pennzoil Place - North Tower, 700 Milam, 10th Floor, Houston, Texas 77002, pursuant to Rule 201(a), Illinois Supreme Court Rules, and the provisions stated on the record or attached hereto.

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(1:07 p.m.)

ETHAN A. NATELSON, M.D., F.A.C.P.,
having been first duly sworn, testified as follows:

EXAMINATION

BY MR. HARTLEY:

Q. Dr. Natelson, how are you today?
A. Fine. Thank you.
Q. Just for the record, could you tell us your name?
A. Yes. Ethan A. Natelson.
Q. And, Dr. Natelson, I have in front of me a report dated July 14, 2008. Do you have a copy of that report with you?
A. Yes, sir.
Q. Have you updated that report since July 14th?
A. No.
Q. Have you done any other research or going to rely on any other literature since July 14th and what's referenced in that report?
A. Well, yes. We sent you -- at the time of the last attempted deposition, I had some additional papers --
Q. Okay.
A. -- and we -- I believe Mr. Chilton sent those to you and we have you those here also.
Q. Okay. And I have those.
What -- in the first paragraph of your July 14th

letter, you indicate that you've had the opportunity to review the pathology slide material.
A. Yes.
Q. What particular pathology did you review?
A. These were bone marrow slides, and also the kidney biopsy slides.
Q. Did you review any of the liver slides?
A. Yes. The liver slides were there also and I looked at those.
Q. In your practice, Dr. Natelson, do you normally review kidney slides?
A. I would say rarely. If it's a particular case that I have an interest in, I might, but not commonly.
Q. Did you review these kidney slides with any particular pathologist or any other person?
A. Yes, with Dr. Barrios, who submitted a report about them.
Q. Did you review the liver slides with anyone else?
A. Yes. I looked at them with Mary Schwartz, who's the liver pathologist at Methodist Hospital, just having her take a casual look through the slides. I did not ask her to write up an opinion.
Q. And do you normally review liver slides in your practice as a hematologist?
A. Again, I might. It really depends on the individual

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case, but I certainly have looked at liver slides in the past.
Q. Out of patients that you've seen, how many times have you in your practice -- and you can give me a range or a percentage -- reviewed the kidney slides of a particular patient?
A. It would be hard to give you a number on that because I don't -- I'm not a kidney specialist and so I normally don't see patients who have only kidney disease, per se. I'm a hematologist. But if I have a patient who's got a hematologic problem that I have been following and they suddenly develop something else that requires a tissue biopsy, it's not unusual to look at that biopsy, too.
Q. Do you have any training in kidney pathology?
A. No.
Q. Do you have any training in liver pathology?
A. No.
Q. Do you review kidney slides once a month in your normal practice?
A. No.
Q. Once a year?
A. Probably, yes.
Q. And how long have you been practicing?
A. Oh, since -- seeing patients since around 1969.
Q. So, that's about 40 years --
A. Yes.

Q. -- give or take.
A. Yes.
Q. Would it be fair to say then that you, over the course of your 40 years of practice as an oncologist or hematologist, have seen on average 40 -- or have looked at 40 kidney slides of your particular patients in your practice?
A. That's probably so.
Q. How about liver slides? Do you look at liver slides once a month?
A. It depends on the years. In other words, there have been times that I've looked at liver slides more frequently than that and times that I've looked at them less frequently.
Q. Do you think you looked at liver slides as much as you looked at kidney slides?
A. I would say possibly slightly more.
Q. Can you give me -- twice a year, once -- one-and-a-half times a year? Can you give me some type of an idea?
A. I -- it would be really hard to just put any kind of an estimate on there, but -- as a hematologist giving transfusions and seeing patients with hepatitis, I more often would have an opportunity to look at liver biopsies than kidney biopsies.
Q. Do you normally look at those biopsies, liver biopsies, with another -- with a liver pathologist?

A. Yes.
Q. Do you ever review those pathologies of the liver by yourself and rely on your own reading of those slides?
A. No.
Q. Same question for kidney pathology that you might have been looking at. Do you normally look at those biopsies under the microscope with a kidney specialist or kidney pathologist?
A. Yes.
Q. And do you rely solely on your own review of those slides when you're treating your patients in your normal practice or that of the pathologist?
A. I would rely on the pathologist.
Q. You certainly don't hold yourself out as a liver specialist either, do you?
A. No.
Q. Have you had any real training as it relates to the study of liver pathology?
A. No.
Q. Have you written any articles on liver pathology?
A. No.
Q. Have you written any articles on kidney pathology?
A. No.
Q. Do you teach any of the folks who might come through your rounds kidney -- medicine that relates to kidney problems?

A. No.
Q. Same question for liver. Do you treat (sic) any of the folks that may come through on your rounds about liver problems?
A. Well, we commonly discuss liver problems at our morning report, but -- I think if you're suggesting do I teach liver pathology to our interns, the answer would be no.
Q. Would you defer to a pathologist who specializes in kidneys for their opinion as to what the slides actually show?
A. Yes.
Q. Same question for liver pathology. Would you defer to a liver pathologist as to what the slides actually show?
A. Yes.
Q. In the July 14th letter -- why don't we mark that as Exhibit 1.
(Natelson Exhibit No. 1 marked.)
THE REPORTER: Okay. It's marked.
MR. HARTLEY: Thank you.
Q. (BY MR. HARTLEY) Dr. Natelson, let me show you what's been marked as Exhibit No. 1. Can you identify that for me?
A. Yes. This is a letter dated July 14, 2008, that I sent to Mr. Louis Woolf.
Q. And the copy that I have is about nine pages long. Is your copy nine pages? Includes the references.

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A. Let's see. That is true.
Q. Okay. Look in the first full paragraph on Page 1, about the middle of that paragraph. "As you know, it is my opinion that Mr. Lebron suffered several well-described complications of hepatitis B," paren, "HBV," close paren, "virus infection which included" --
A. Yes.
Q. -- then there is a list of items after that.
Is it your opinion that all of the medical problems, situations, syndromes, or anything described after "which included" are solely related to Mr. Lebron's hepatitis B virus?
A. Correct. That is my opinion.
Q. Now, did -- in the first phrase after that "which included," you have "the nephrotic syndrome and renal failure due to" -- is it crescentic? Is that a correct pronunciation?
A. Yes.
Q. -- "crescentic glomerulonephritis."
Did you personally make a diagnosis of crescentic glomerulonephritis?
A. No.
Q. Who did?
A. Well, in the original pathology reports, the word "crescentic" is mentioned, and their description is that of a glomerulonephritis. But when I showed the slides to my

pathologist at Methodist Hospital, he commented, "This is a typical crescentic glomerulonephritis."
Q. And that would be Dr. Barrios?
A. Dr. Barrios.
Q. Barrios.
A. And that didn't surprise me because that was the description in the original pathology.
Q. And what particular pathology report -- do you have those with you?
A. Yes.
Q. What report are you referencing, the April 10th report from the materials obtained April 3rd, 2000?
A. I'm referencing Dr. Barrios' report of May 30, 2007.
Q. Oh, okay. So, you're referencing Dr. Barrios.
MR. CHILTON: Dean, let me interrupt. I think your question though was about the original pathology report.
MR. HARTLEY: It is, it is, and we're going to clean that up now.
MR. CHILTON: Okay. Thank you.
Q. (BY MR. HARTLEY) So, Dr. Barrios diagnosed crescentic glomerulonephritis, correct?
A. Correct.
Q. Did any physician who saw the pathology in the regular course of business, either at Loyola or at La Grange or at any other facility in the Chicagoland area, make a diagnosis

of crescentic glomerulonephritis?
A. Well, the -- I don't have all those original records there, but the kidney doctor that saw him wrote in his notes that he had glomerulonephritis.
Q. Who would that have been?
A. I don't remember the man's name.
Q. Well, it's not -- is it Leonard Potempa?
A. I would just be speculating. I haven't looked at those records for some years.
Q. But it's your opinion that the kidney doctor made the diagnosis of glomerulonephritis as a result of the crescents that were seen on the pathology slides?
A. No. I don't know that he made a diagnosis on the basis of the crescents. He simply says in his consult notes that the patient has glomerulonephritis. That was after the renal biopsy was done.
Q. After the renal -- did the pathologist who reviewed the kidneys come to that conclusion?
A. The pathologist did not use the term "glomerulonephritis" as his final conclusion on that slide.
Q. And as I understand it, you don't have the nephrologist's information to identify for me who the nephrologist was that made the diagnosis of crescentic glomerulonephritis?
A. As I said, I don't recall the nephrologist using the

term "crescentic glomerulonephritis," but he certainly said "glomerulonephritis."
Q. Okay. So, then the only people who have used the term "crescentic glomerulonephritis" would be you and Dr. Barrios?
A. No. That would not be true. I believe Dr. McHugh, another pathologist, has reviewed this and has commented about that.
Q. And did he make a final diagnosis of that?
A. I'd have to look again. In his report, he cited the crescentic changes and said that he had, I believe, membranoproliferative glomerulonephritis.
Q. Secondary to hepatitis B infection. And as I understand what he wrote, he had down there "provisional" because he wanted to see the photomicrographs. Isn't that true?
A. We could pull out his report. I think I may have it here. I don't remember that, but let's see what he says.
He says, "A renal biopsy showed a proliferative glomerulonephritis with partial crescent formation." I take that to believe he is calling it a crescentic glomerulonephritis.
Q. And what's he say at the end?
A. "I would like the opportunity to review the electron microscopy photomicrographs and reserve the right to modify my

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impression of the exact classification of the patient's renal disease."

Q. Does that mean that his diagnosis would then be provisional upon reviewing the photomicrographs?

A. You'd have to ask him that.

Q. Did you review the photomicrographs when you reviewed the material for this case?

A. I didn't have them when I had the slides for Dr. Barrios to look at. And I do have them here, but I'm certainly not an expert on electron microscopy.

Q. So, you're not an expert on electromicroscopy, you're not an expert on the pathology of kidneys, but you've talked to Dr. Barrios who has concluded that it is a crescentic glomerulonephritis and, provisionally, Dr. McHugh has said that; so, in your opinion, he has crescentic glomerulonephritis. Is that true?

A. In my opinion, he has crescentic glomerulonephritis. As to whether or not Dr. McHugh's report is provisional or not, you have to ask him.

Q. And tell me, Doctor, what do you base that diagnosis of crescentic glomerulonephritis on?

A. Well, he had numerous crescents in the kidney biopsy. That's a very specific finding. And I listed a paper in here that discusses what these -- what the significance of crescentic changes are. And that report spells out that when

you see crescentic changes in a glomerulonephritis, it just simply means it's a very inflammatory glomerulonephritis.

Q. Okay. So, would it be fair to say that your opinion and the basis for that opinion that Mr. Lebron has glomerulonephritis -- crescentic glomerulonephritis is the result of your reading the article that tells you what crescent findings on kidney slides indicate?

A. No. That's a piece of information I have to clarify what the term "crescentic" means. There are plenty -- I think I may have other reports on crescentic glomerulonephritis, and I've certainly heard that term used in clinical medicine for many years.

Q. Well, the fact that you've heard it used doesn't mean that the patient has it, does it?

A. No, but I -- as I say, when I reviewed these slides and discussed it with the pathologist, in view of what's there, that diagnosis is apparent.

Q. Okay. Did you happen to look at -- let me strike that for a second.

So, your review of the slides in conjunction with Dr. Barrios and your review of the medical literature on what crescentic glomerulonephritis requires or includes is the basis for your opinion in this particular matter that Mr. Lebron had crescentic glomerulonephritis, correct?

A. My opinion is the fact that all of the pathologists

that have looked at this biopsy have commented on the fact that he has crescentic changes. That means a very specific thing in terms of glomerulonephritis. So, it's -- it just simply means that it has -- that it has an inflammatory character to it.

Q. Do you have the original pathology report?

A. I don't think so.

MR. CHILTON: You mean with him here today?

MR. HARTLEY: Yes.

MR. CHILTON: Okay. Do you -- can you direct us to what report it was?

MR. HARTLEY: It's the Picken report, or Picken. Picken, dash, M-r-o-z-o-w-i-c-z, from Loyola. The session number is S, as in Sam, 00-5459 and the report date is April 10, 2000.

MR. CHILTON: We're taking a look, Dean. We don't have all of the medical records here today.

MR. HARTLEY: Okay.

Q. (BY MR. HARTLEY) Do you know -- do you have Dr. Meehan's deposition with you?

A. Yes, sir.

Q. I think it's an attachment to Dr. Meehan's deposition.

A. That's where I'm looking right at the moment.

Q. In fact, I think it's Exhibit 1.

A. Yes. I'm looking at it right here, I think.

Q. It's -- at the top, it says "Microscopic Description"?

A. Correct.

Q. And at the bottom, it says "Lebron." And if you look on the far right, it says "Page 2 of 3," but it says "S00-5459."

A. Correct.

Q. Okay. Look at the interpretation that you've been talking about that there were crescents shown.

A. I didn't say they were in the interpretation. I said they're in the body of the report. They're in the description under Microscopic Description.

Q. And where are they?

A. Well, I'll read that. It says -- where am I looking at that? And in the -- one down, it says "And three other" -- "there is extensive periglomerular fibrosis around these glomeruli and three other glomeruli show fibrocellular crescents, which in one glomerulus is circumferential."

Q. How many glomeruli are there in the kidney?

A. I would have no idea. Probably millions.

Q. And so out of millions, three had crescents demonstrated on the slide; is that fair?

A. No. That's totally unfair. This is a small --

Q. Why is that?

A. Because this is a tiny biopsy and -- sometimes you'll

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biopsy a kidney and it won't be a good biopsy, you won't see any glomeruli. And sometimes you see two, sometimes you see three, sometimes you see four. And so I would say -- in this particular one, I don't remember how many glomeruli were present, but it wasn't a huge number.

Q. "There are nine glomeruli per level." What does that mean based on your experience as a kidney pathologist or a hematologist reading kidney slides?

A. Well, he's cut a level from the kidney. He's cut a paraffin section. And in that section, he's got nine glomeruli is what he's saying to me; and three of them have crescents. And he doesn't say that all nine are intact so that they can be examined carefully.

Q. If we look down at the interpretation, the final interpretation down at the bottom under the immunofluorescence --

A. Yes.

Q. -- that's a different type of review of the material, is it not?

A. No. He comments there that there are crescents, too.

Q. No. I'm not asking you that. Immunofluorescence is a different type of review of the materials, is it not?

A. Oh, yes. It's a different study, yes.

Q. Okay. And he says, "Out of the six, one shows a small crescent," correct? Shows small crescents. About four

lines down, I believe.

A. Yes, but that has nothing to do with the immunofluorescence aspect of it. In other words, the immunofluorescence is talking about the fact that he's lighting up gamma globulin in the kidney, and he's just telling us that he's got a -- for this particular slide, he's got six glomeruli per level and he's applying immunofluorescent stains to those six glomeruli.

Q. And out of the six that he has, one shows small crescents, correct?

A. That's what he says here, yes.

Q. Okay. And you haven't reviewed the immunofluorescence photomicrographs, have you?

A. No.

Q. So, you wouldn't know whether that's right or not.

A. I don't know the pertinence to that question, but, no, I don't know that it's right or not. Which is the question you're asking, are there six glomeruli, or what?

Q. I guess the question is: You don't dispute what the pathologist at Loyola is reporting since you haven't reviewed that, correct?

A. That's correct.

Q. Have you reviewed the disclosure filed by Hydrosol -- or H Holdings, I believe it's now known as -- wherein they disclose in a legal document what Dr. Nancy Lynn Jones, the

medical examiner for the office of the medical examiner of Cook County, is going to say?

A. I don't think I've seen anything like that.

Q. Would it surprise you to learn that Dr. Jones does not make a diagnosis of crescentic glomerulonephritis?

MR. CHILTON: I'm going to object to the form of the question.

A. I would have no -- I don't know Dr. Jones. I don't know her -- is it a she -- her qualifications. I wouldn't have any comment about what she might say.

Q. (BY MR. HARTLEY) Well, if she's a pathologist, would you agree with me that she would have more experience reading kidney slides than you do as a hematologist/oncologist?

A. That is certainly true.

Q. In your review of the liver slides -- and you said you reviewed those with Dr. Schwartz?

A. Yes.

Q. And who is Dr. Schwartz?

A. She's a liver pathologist at Methodist Hospital.

Q. Did you find any cirrhosis in the 2001 biopsy of Mr. Lebron's liver?

A. I wouldn't be qualified to discuss whether he did or didn't have any cirrhosis in his liver biopsy.

Q. Well, did Dr. Schwartz find any?

A. I don't remember. We were discussing the tumor in

the biopsies. She did look at the original biopsies and commented that they looked like typical hepatitis, as I recall. I don't remember what she -- if she even said anything about the cirrhosis aspect.

Q. Did you look at or determine whether there was any biliary epithelial dysplasia in the 2001 study?

A. I wouldn't personally know how to discuss that if I looked at that myself.

Q. Well, did you talk about that with Dr. Schwartz?

A. What I talked about with her was what was her concept of what that kidney biopsy showed in terms of the malignancy, what were the possibilities of that malignancy.

Q. Did you talk with her about the findings on the 2001 biopsy that there was no evidence of any type of malignancy present in 2001?

A. I don't remember what we discussed on the earlier biopsies. In terms of malignancy, we were talking specifically with the final biopsies.

Q. So, you have no recollection of whether the 2004 liver biopsy demonstrated cirrhosis or not?

A. Well, the 2000 -- the final biopsies is primarily tumor, and so --

Q. 2000 -- the final biopsy's 2005.

A. Okay. Excuse me. The 2004 biopsy, I don't recall anything that I could say sensibly about it. She looked at the

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biopsies and we discussed the tumor.
Q. And there was no comment made on the 2004 biopsy vis-a-vis whether there was cirrhosis present?
A. I don't recall any discussion about that.
Q. Was there any discussion whether there was any biliary epithelial dysplasia present in 2004?
A. I don't recall any discussion about that.
Q. Do you have any appreciation for why biliary epithelial dysplasia would be important in this particular case?
A. No.
Q. Okay. Is it your opinion, Dr. Natelson, that the cancer of Mr. Lebron's liver was a cholangiocarcinoma?
A. I think clinically it was.
Q. I believe that you have suggested that that type of liver cancer is caused by chronic hepatitis B infection. Is that true?
A. It can be.
Q. Are you aware -- bear with me until I find it.
You reference in your paper -- I believe it's Shaib or Shaib.
MR. CHILTON: By "his paper," you mean his report of July 14th, Dean?
MR. HARTLEY: Yes, sir.
MR. CHILTON: Thank you. Do you have the page

number in front of you?
MR. HARTLEY: I'm trying to find it. I've got it marked here on my group of stuff, but I don't see it. It's on Page 6 (sic), your Reference 20, I believe.
MR. CHILTON: Okay. Thanks.
Q. (BY MR. HARTLEY) And it guess it would be Shaib maybe. Is that how you pronounce it?
A. Probably, yeah. That's a good -- that's as close as anyone. Yes, I have that paper in front of me.
Q. Doctor, do you know as a hematologist/oncologist what biliary epithelial are in the liver?
A. Yes. Biliary ducts drain the liver eventually into the common bile duct and into the intestine. The epithelium would be the inner layer of those ducts.
Q. Did you find any chronic inflammatory disease in the biliary epithelium when you reviewed the pathology?
A. I wouldn't be competent to make that distinction.
Q. In your report, you reference the Shaib epidemiology of cholangiocarcinoma in 2004 from Seminars on Liver Disease, correct?
A. Correct.
Q. Did you also come across Yasser Shaib's 2005 article "Risk Factors of Intrahepatic Cholangiocarcinoma in the U.S."?
A. If I didn't reference it in my letter, I didn't have it.

Q. Are you aware that other than the 2005 article by Shaib, there are no other studies conducted in the U.S. which evaluate the role of hepatitis C, hepatitis B, diabetes, or chronic liver diseases with intrahepatic cholangiocarcinoma?
A. Well, it seems to me that I referenced another article that was from the United States. Let me just see what I have in here.
Yes, this one by -- and I won't even guess how to pronounce his last name, Perumal. And this was --
Q. What number is that? Is that a different one that you supplied me with last time?
A. Could be. I don't recall, but it's entitled "Hepatitis C and hepatitis B nucleic acids are present in intrahepatic cholangiocarcinomas from the United States."
Q. Okay. Did they -- did they do any type of statistical analysis in that article?
A. No.
Q. Okay. Would it surprise you that when the same author that you cited earlier, Shaib, Shaib -- however you pronounce it -- in studying 625 cases from the United States found no relationship between hepatitis B and the development of intrahepatic cholangiocarcinoma?
A. I haven't read that article, so I can't really comment about it.
Q. Is it your opinion that -- well, let me take it back.

Do you have an opinion as to the type of cancer Mr. Lebron developed in his liver?
A. He had an adenocarcinoma, which my pathologist in Houston thought was compatible with a cholangiocarcinoma, and Dr. McHugh thought that. And your pathologist in Chicago commented on cholangiocarcinoma, and my interpretation of his report is he thought it was compatible with that but more likely to be metastatic carcinoma. And when I put -- as a clinician, put all of the information about this case together, to me, it's very clear that that was a cholangiocarcinoma.
Q. And as we sit here today, is it your opinion that Mr. Lebron's cancer, the cholangiocarcinoma, was caused by his underlying hepatitis B?
A. Yes. That is a relatively new concept that I discussed with Dr. Schwartz when we were looking at the biopsy. And that's why you see that the newer articles which are coming out which look at this relationship in countries where there are large numbers of cases to study -- hepatitis B is not a common number in the United States these days; so, therefore, you have to study it where the money is -- and those studies now are statistically significant, quite significant, suggest that hepatitis B is a risk factor.
Q. And other than the -- is it P-e-r-u-m-a-l? Is that the article you're trying to reference?
A. That's one that is specific -- you commented -- you

asked about articles from the United States --
Q. Right.
A. -- so that's one that specifically uses the term "United States" in the title.
Q. Okay. Other than that and the article that I brought to your attention, S-h-a-i-b, from Gastroenterology 2005, are you aware of any other studies that involve only persons from the United States?
A. No. I haven't attempted to research that.
Q. Have you tried to determine whether there's a difference in folks living in the United States and their development of hepatitis B and folks who live in other parts of the country and their development of hepatitis B so as to impact the causal relationship between hepatitis B and cholangiocarcinomas?
MR. CHILTON: Dean, I'm going to object to the question. You said "and patients in other parts of the country." I think you wanted to say "other parts of the world."
MR. HARTLEY: Yeah, I did.
A. Well, it's well-known that there are certain risk factors for many diseases in parts of the world that differ depending on where you are. And we don't have a lot of liver flukes in the United States, it's not common, and some of these other countries have. So, there are certain relationships that

are prevalent in other parts of the world that are not prevalent in the United States.
Q. (BY MR. HARTLEY) Would you look with me -- do you have the -- which is your reference 21 with you?
MR. CHILTON: 21 from the original report or from the current report?
MR. HARTLEY: The most current report, which is Perumal, something like that, Vivekanandan Perumal. That's the best --
A. Oh, yes.
MR. CHILTON: Oh yeah.
A. I'm looking right at that.
Q. (BY MR. HARTLEY) Look at Table 4 on Page 1214.
A. Yes.
Q. And they have listed for us in Table 4 a survey of studies on hepatitis B and hepatitis C and intrahepatic cholangiocarcinoma, correct?
A. Yes.
Q. And out of the nine entries on Table 4, only the last two are from the U.S., correct?
A. Well, I don't know what you mean by "only." In other words, he has a series of entries from Japan, China, Italy, and the United States. I -- you know, he only has -- he has three from Japan. He has only one from Italy. He has two from the United States. I mean, I don't know why he only has two or if

there's any significance to the fact that he only has two.
Q. Would it be fair to say that of the nine studies listed on Table 4, there are only two entries for studies done in the United States?
A. Yes.
Q. Would you look up above that table with me in the discussion section on the same page, 1214?
A. Yes. I'm looking in that.
Q. Would you agree that the authors conclude, six lines up, "These results, along with the epidemiology findings of others summarized in Table 4, strongly argue that chronic HBV and chronic HBC are significant risk factors in the United States"?
A. Yes. That's just what I've been saying.
Q. That's the article that you're relying on, correct?
A. Well, I've got several articles here that deal with the fact that hepatitis B is a risk factor for cholangiocarcinoma.
Q. Would you look at Table 4 in the next-to-last entry where they reference a Shaib article from the USA? Do you see that?
A. What page are you on?
Q. 1214. Still on 1214.
A. 1214. And where are we on that page?
Q. The table at the bottom.

A. Yes.
Q. The next-to-last entry in that table referencing the study from the U.S. by Shaib.
A. Yes. Now I'm with you.
Q. Okay. And the result of that indicates only that hepatitis C infection is associated with intrahepatic cholangiocarcinoma, correct?
A. Well, it only discusses hepatitis C from that article. It doesn't say anything about what he thought about hepatitis B.
Q. You haven't reviewed the article, have you?
A. I've read this article and I see the results on the table.
Q. Okay. But you haven't reviewed the underlying data for that table for that particular entry.
A. Oh, now I'm with you. No.
Q. That was the article I was asking about earlier, if you had seen that article.
A. No.
Q. You also reference in your July 14th report on Page 3 that monoclonal gammopathy, with both large and small paraprotein peaks, has been commonly cited as a consequence of or an association with hepatitis as a class of illness and, specifically, with hepatitis B and C and you cite Reference 5 and 6. Do you see that?

A. Yes.
Q. Would you pull out Reference 5, which is an abstract by Blanchard?
A. Yes.
Q. And Blanchard, in the background of the abstract, indicates that chronic inflammation or infection may predispose to development of plasma cell dyscrasias, correct?
A. Yes.
Q. And down at the bottom -- would you agree with me that although this is a 2004 abstract, at least according to your note down at the bottom -- I assume that's your writing --
A. Yes.
Q. -- this study provides a basis for future research efforts focussed on identifying cytokine pathways that may link these disorders and subsequently serve as targets for novel interventions to prevent the development of progression of plasma cell dyscrasia, correct?
A. Yes.
Q. So, this abstract and this study of veterans -- isn't that what it was? Yeah, veterans -- basically provides a basis for future research and may provide a link for the disorders and novel interventions in the future, correct?
A. This article provides the fact that veterans with hepatitis B and C have a high incidence of monoclonal gammopathy.

Q. And the theory is that chronic inflammation or infection may predispose someone to the development of plasma cell dyscrasia; is that right?
A. Well, that's a very, very old theory and I don't know that it's true, but it is certainly true that people with hepatitis B and C get monoclonal gammopathies. Whether that makes them go further into multiple myeloma because of the fact that they were originally caused by hepatitis B or C is not known.
Q. You also cite the Brown study from 2008 -- I think this must be the other U.S. study you were thinking of -- which is your Reference 6, correct?
MR. CHILTON: He's looking for it.
A. I'm trying to find it right at the moment.
Yes.
Q. (BY MR. HARTLEY) Look at Table 3 on Page 3390 for me.
A. Yes.
Q. And it says "Infectious disorders and risk of myeloma in white and black U.S. male veterans."
A. Yes.
Q. Where would Mr. Lebron fall on this particular table?
A. He's got hepatitis.
Q. Is he white or black?
A. Well, he's Puerto Rican by background. That depends

on how you want to classify that.
Q. That's why I'm asking you.
A. I would classify him as Puerto Rican.
Q. So, he falls in neither the white nor the black category, correct?
A. I can't tell you what he would put on his driver's license. I suspect he would have put white, but I don't know that.
Q. In the abstract of the report, Doctor, the authors, in the middle column, indicate about -- I don't know -- four lines down, "Significantly elevated risks of myeloma were associated with broad categories of autoimmune" -- then they give you the relative risk -- "and infectious diseases," I assume, and they give you the relative risk of 1.29.
Do you have an opinion as to whether a relative risk of 1.29 provides a basis for saying that infections are associated with myeloma?
A. I would say it wouldn't confirm it. You'd like to see a doubling of the risk.
Q. Would you rely on that .129 (sic) in making your determination?
A. Well, I didn't determine he had multiple myeloma.
Q. But if we assume that he did.
CHILTON: I'll object --
A. Really, I would say that that would be a negative --

I wouldn't conclude that could confirm the presence of myeloma, that particular number.
Q. (BY MR. HARTLEY) Is it your opinion -- it is your opinion, isn't it, that he does not have myeloma?
A. That's correct.
Q. But if he does have myeloma, it's probably related to his hepatitis B. Is that your opinion?
A. No. That would be false.
Q. Okay. Did you make a determination as to whether your diagnosis of crescentic glomerulonephritis was of the acute nature?
A. I'm not sure what you mean by that. In other words, clinically, he'd been sick for a considerable period of time before he had that biopsy done and he was already developing signs of renal insufficiency. So, he's got a chronic illness.
Q. Well, look on Page 3 of your report. And under Crescentic Glomerulonephritis, you have in bold the quote from Article No. 7, or Reference No. 7, about the accumulation of cells in Bowman's space, etcetera. At the end of that quote, you say, "Thus, in acute severe forms of glomerulonephritis such as that mediated by autoantibodies, crescent formation is prominent."
Are you saying that he has an acute severe form of glomerulonephritis? "He" being Mr. Lebron.
A. No. He has a chronic process, but the crescents

don't go away. In other words, when an illness starts out, it's acute. Crescents form, they continue to form, and then eventually they burn out the kidney. So, an acute glomerulonephritis becomes chronic. The crescents may be there in the acute phase, they're still there in the chronic phase.

Q. Is there any type of lab report or lab test that we can do to determine whether, in fact, he does have chronic glomerulonephritis or crescentic -- I'm sorry -- crescentic glomerulonephritis?

A. No. That's just a -- the crescentic aspect of it is simply a histopathological indication of inflammation. There is no biochemical test that one can do on his serum to point to that finding.

Q. Well, look with me at your Reference No. 7, which is Mathieson, editorial review.

A. Yes.

Q. You remember that?

A. Yes. I've got it right here.

Q. Look at the first column -- left-hand column in the first full paragraph where it talks about, toward the end of that, "Thus, in acute severe forms of GN, such as that mediated by autoantibodies to the Type IV collagen in glomerular basement membrane, GBM -- Goodpasture's disease or anti-GBM disease -- crescent formation is prominent, often affecting all glomeruli seen in a renal biopsy."

Do you see that?

A. Yes.

Q. That's the full quote from the paper that you've quoted in your report, is it not?

A. Yes.

Q. Do you have an opinion as to whether Mr. Lebron had a severe form of GN?

A. Well, he certainly didn't have Goodpasture's disease -- that's a very specific illness -- and he had severe enough glomerulonephritis to lead him to renal failure. So, obviously, I think by definition that would be severe.

Q. Okay. So, if he had severe -- if he had severe kidney disease -- according to the editorial, crescent formation is prominent, often affecting all glomeruli seen. Would you agree with me that in the pathology report all the glomeruli did not have crescents in them, did they?

A. Well, you're asking two different things. Yes, I've already agreed that all of the glomeruli didn't have crescents. And I've also said that he didn't have Goodpasture's disease, which is a particular type of glomerulonephritis that the author is citing. And I think that it would be -- severity is just a judgement in terms of an index of pathology, but obviously his disease was severe enough to cause the kidneys to fail. I would call that severe.

Q. Are you saying that based on your understanding of

kidney pathology, he has severe kidney pathology, but he doesn't have severe enough kidney pathology so that all the glomeruli have crescents in them?

A. In this particular biopsy that we saw, which is a tiny biopsy relative to both kidneys, he has several glomeruli with crescents in them, enough so that all of the observers have commented on that finding. He didn't have them in a hundred percent of them. As to the significance of the fact that he had them in three out of nine, you would be better off questioning Dr. McHugh about the significance of that fact.

Q. Do you have an understanding for -- of what PCNA is?

A. PCNA? You'll have to tell me what those initials stand for and where we're looking at them.

Q. We're on the next page of the Mathieson article, 156.

A. I see it. He calls it proliferating cell nuclear antigen. And what was your --

Q. Do you know what that is?

A. I have no idea.

Q. Do you have with you the La Grange Memorial Hospital report?

A. Is that the one we talked about of the kidney biopsy in Dr. Meehan's deposition?

Q. No. This one's done at La Grange, it looks like, March 30th, 2000.

A. And what's it a biopsy of?

Q. It is a study of antiglomerular basement membrane.

A. If it's -- is it something that I referenced?

Q. No.

A. Well, if it's not something that I referenced, I obviously don't have it here.

Q. Okay. Do you have the medical records with you?

A. No.

Q. Okay. In trying to determine whether Mr. Lebron had crescentic glomerulonephritis, did you look at the lab results from the various hospitalizations?

A. I'm sure I did look at his lab results.

Q. Are you aware of any lab result which may or may not confirm or rule out the existence of glomerulonephritis, crescentic glomerulonephritis?

A. Well, as we said, I don't believe there's any single lab test that rules it in or rules it out, and so I'm not clear --

Q. Are you aware of any lab test that makes it less likely? As --

A. I'm not sure what you're referring to.

Q. Well, as a hematologist/oncologist testifying as someone who's familiar with kidney diseases, are you familiar with any particular lab result of the kidney which makes a diagnosis of crescentic glomerulonephritis less likely?

A. Are we speaking of a blood test on serum or something

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on tissue?
Q. Well, you're the person who's being offered as the expert. Can you think of any?
MR. CHILTON: You're -- I'm going to object. You're asking the question. He's asking you to clarify your question. He's not here to guess or speculate as to what you're asking him. You need to ask a fair and clear question.
MR. HARTLEY: I understand that he's not an expert in kidney diseases.
MR. CHILTON: That's not what I said.
MR. HARTLEY: I understand he's not an expert in kidney pathology, but he's being offered as an expert in kidney diseases and in reading pathology slides of the kidney in this particular case. I am simply asking him is he aware of any laboratory study as a hematologist/oncologist testifying in the area of kidney pathology and kidney diseases which makes a diagnosis of crescentic glomerulonephritis less likely than not? If he doesn't know of any, that's fine, we'll move on.
A. Let's move on. I don't really know what you're talking about lab-wise. This is simply a descriptive term. There is no blood test that proves whether someone does or does not have crescents in their kidney biopsy.
Q. (BY MR. HARTLEY) What is complement C3?
A. Well, complement are a series of proteins that have to do with allergic reactions. They're proteins that are

normally in the blood.
Q. What significance do they have vis-a-vis crescentic glomerulonephritis, if anything?
A. Well, in forms of glomerulonephritis and many other illnesses, complement becomes so-called activated and can be deposited into tissues or cause inflammation.
Q. What does complement C3 particularly look for?
A. I'm not a complement expert.
Q. How about p-ANCA? Do you have any idea what that is?
A. I think you're talking about p-ANCA, and the --
Q. p-ANCA?
A. Yeah. I think that's p-ANCA. That's p-ANCA. That's a -- sort of a test that's positive in certain types of inflammatory kidney disease, such as Wegener's granulomatosis.
Q. And if it's negative, what does that indicate?
A. Well, it's not a test that we use for a 100 percent diagnosis of anything. In other words, it's frequently positive in a rare illness called Wegener's granulomatosis. It may be negative in that illness. It's a test of, you might say, inflammation.
Q. Would that test need to be positive for one to -- if one had crescentic glomerulonephritis?
A. Would not have to be positive.
Q. Okay. So, if the lab that ran this, SmithKline Beecham or Quest -- I'm not sure which one did it -- indicates

that the p-ANCA, if it's present, is seen with crescentic glomerulonephritis, that's not something that we'd be concerned with?
A. It's not a test that proves a diagnosis.
Q. It proves inflammation, doesn't it?
A. Well, it suggests inflammation; but in severe diseases characterized by inflammation, it may be negative.
Q. And would you agree with me that crescentic glomerulonephritis is an inflammatory disease process?
A. Yes.
Q. Okay. What is Bence-Jones proteinuria? I might have butchered "proteinuria."
A. Bence-Jones was a man who originally described a multiple myeloma, or that is to say some of the protein findings in the urine; and it's a protein that comes out in the urine and can be redissolved if you heat up the urine and then it comes back out if you cool down the urine. And --
Q. Why -- I'm sorry.
A. And we generally think of it as synonymous as what we call light chain protein in the urine.
Q. And if you find Bence-Jones protein in the urine, is that consistent with a diagnosis of myeloma?
A. It could be.
Q. Would you agree that it is a -- suggestive of monoclonal gammopathy of undetermined clinical significance?

A. Well, Bence-Jones protein, number one, has to do with urine, and most people we find with MGUS -- or monoclonal gammopathy of unknown significance -- we're talking about serum. So, urine is a different situation than serum. One can have monoclonal gammopathies in the urine and one can have Bence-Jones protein in the urine. And when one finds Bence-Jones protein in the urine, that can be indicative of multiple myeloma.
Q. What other diseases would it be consistent with?
A. Well, one can find monoclonal proteins in the urine of people who -- and it may carry the same significance as monoclonal proteins in the serum; that is to say, one can have monoclonal proteins in the urine of unknown significance.
Q. Are you telling me that Bence-Jones proteins are monoclonal proteins of unknown origin?
A. Well, they can be.
Q. Are you telling me that Bence-Jones proteins are simply -- are indicative of -- strike that.
Other than myeloma, can you think of any other diseases that Bence-Jones protein would be consistent with?
A. Well, I'd have to look that up, but I -- Bence-Jones proteins, it's an old term for proteins that have heat properties; that is to say, you can bring them in or out of the solution of the urine, whether they're heated or cooled. And it's sort of an obsolete term. It's been replaced by

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specifically measuring light chains or monoclonal proteins in the urine.
Q. So, to answer my question, you'd have to look up in some textbook or something what type of disease entities demonstrate with Bence-Jones protein?
A. Bence-Jones proteins are seen in myeloma. I would have to look in the book to see what other obscure illnesses they might be seen in. And there may well be others as we sit here. I don't know that.
Q. Well, how about Fanconi's syndrome?
A. Well, Fanconi's syndrome is a tubular disease of the kidney that can be caused by certain drugs damage the tubules and you get so-called Fanconi's syndrome where you can lose glucose and protein in the urine. And it's possible that they would have Bence-Jones proteins. I don't know that.
Q. Let's assume that they do. Did Mr. Lebron have Fanconi's syndrome?
A. No.
Q. How about a lymphoproliferative disorder? What would that include?
A. Well, lymphoproliferative disorders could be things like chronic lymphocytic leukemia or small cell lymphoma. They also are associated with monoclonal gammopathies and they also may cause glomerulonephritis.
Q. Would you see Bence-Jones proteins in those types of

entities?
A. I don't know the answer to that. You certainly see proteinuria and you see monoclonal proteins in the serum. I don't know the answer to that question.
Q. Let's assume that you do. Does Mr. Lebron have either of those entities, CLL or the other lymphoma that you described?
A. No.
Q. And I'll butcher this. What is a-m-y-l-o-i-d-o-s-i-s?
A. Amyloidosis is a condition -- and there are several types of amyloidosis -- and it's a deposition in the tissue of a waxy protein. And in some instances, it's associated with multiple myeloma. In some instances, it's not.
Q. Do you see Bence-Jones proteinuria in -- whatever it was?
A. Amyloidosis, yes, you may.
Q. Okay. Did Mr. Lebron have amyloidosis?
A. No. They stained his kidneys for amyloid and he didn't have that.
Q. Okay. Oh, let's look at Ronco. I think that's, like, 16 maybe.
A. Yes.
Q. That's not 16. Where is it?
MR. CHILTON: No. 9.

Q. (BY MR. HARTLEY) No. 9.
Now, is it your opinion that Mr. Lebron does not have MIDD?
A. That is correct.
Q. Okay. Would you pull out Dr. Meehan's deposition again --
A. Yes.
Q. -- so that you can see the description of the kidney?
A. I assume you're speaking of the original one that's this Page 2 of 3?
Q. Yeah. I have to find mine. I have too many papers on the table here. Yep, that will do. And let's look at the -- under Pathogenesis.
A. Pathogenesis, yep.
Q. You with me? I'm sorry. We're referring to two different things now. Ronco, 2006 --
A. Yes.
Q. -- on the first page, under Pathogenesis --
A. Yes.
Q. -- "MIDD is characterized by kidney deposition of monoclonal Ig subunits." Do we have that in this case?
A. Well, you have the whole molecule being deposited and the molecule is consisting of two light chains and two heavy chains.
Q. So, does that mean we do or we don't have deposition

of monoclonal Ig subunits?
A. Well, it depends how you're using that term. In other words, I think these people use this term to mean it can be light chain disease where only the light chain is deposited; it can be heavy chain disease where only the heavy chain disease is deposited; and then it can be light and heavy chain disease, which is both chains being deposited. And that whole complex is referred to as monoclonal immunoglobulin deposition disease; in other words, the three types.
Q. Are you an expert on MIDD?
A. No.
Q. Have you ever testified in any case, Doctor, where MIDD was involved?
A. No.
Q. I came across something on the internet, I think -- or maybe you produced it. It is a PowerPoint presentation. Do you remember doing that?
A. A PowerPoint presentation -- well, I did in terms of the photographs.
Q. The photographs of what?
A. Bone marrow and the kidney biopsy, I think.
Q. Okay. That was what that PowerPoint presentation was?
A. Yes.
Q. Think it's called "Monoclonal Gammopathy Associated

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With Liver Disease and With Nephrotic Syndrome."
A. Yes.
Q. And that was prepared for this case?
A. Yes.
Q. Okay. I wondered what that was. I couldn't quite figure out what that was.
But getting back to the Ronco paper, it says "MIDD is characterized by kidney deposition of monoclonal Ig subunits, but at variance with amyloidosis, deposition induces a dramatic accumulation of extracellular matrix that is responsible for glomerular and tubular basement membrane thickening, nodular glomerulosclerosis, and interstitial fibrosis."
Do you see that?
A. Yes.
Q. Look at the pathology report, particularly the immunofluorescent study at the bottom of Page 2 of that report.
A. Okay. I'm looking under Interpretation.
Q. Yes, sir. And it begins "Frozen sections were incubated with the following antibodies," then she gives those to you. Do you see that?
A. Yes.
Q. "There's a diffuse and" -- is it "punctate" --
A. Yes.
Q. -- "stain for IgG and kappa light chain along the

glomerular capillary wall mesangium, as well as tubal basement membrane and focally in the extraglomerular vessels."
Do you see that?
A. Yes.
Q. Isn't that the exact same description that Ronco is giving us in his 2005 diagnosis that we just read together?
A. Well, all this is saying is that there's immunoglobulin stuck in the kidney, and that's not specific for any single entity.
Q. Okay. So, Ronco really didn't need to give us this long description. They could have just said, "There's immunoglobulin stuck in the kidney," and that would have been okay? That's your understanding?
A. No. Ronco gives a very specific discussion of what this entity is. And what the difference between this entity or one of the differences between this entity and the -- and what Mr. Lebron has is this entity is a glomerular path -- a glomerulopathy. In other words, this is sort of a bland destruction of the kidney. The crescentic changes are inflammatory destruction of the kidney.
And in this article, he specifically says in here, "exudative lesions as fibrin caps," which would be the crescents, "and extensive hyalinosis of the efferent arterioles are not observed." So, he's telling us this is bland pathology damage as opposed to inflammatory damage, and he shows us a

nice picture to confirm that.
Q. Okay. That's your understanding of what he's trying to tell us?
A. That's my understanding of what he's telling us in this article.
Q. You've read Dr. Meehan's deposition, have you not?
A. Yes, sir.
Q. And you have put forth in your July 14th report your comments concerning that report and his lack of understanding of the situation, correct?
A. Well, I've commented about certain things he had in his deposition, yes.
Q. Go to Page 2 of your report.
Do you want to take a break, Doctor, or are you okay?
A. No. I'm okay.
Q. Okay. Under bone marrow findings, you have "flow cytometry." Do you see that about -- I don't know -- seven, eight lines down, "Flow cytometry to bone marrow cells can also compute percentages of various cell types"?
A. I know I said that in here, but I'm not seeing -- on Page 2 is that?
Q. Yes, Page 2, under Bone Marrow Findings.
A. Oh, yes. Yes, I see that, uh-huh.
Q. Was flow cytometry done in this case?

A. I don't recall.
Q. Tell me what CD138 is.
A. Well, CD stands for cluster designation, and there are many CD markers on blood cells. And lymphocytes, for example, often have CD20 on them and CD5. This is a particular CD marker that might be on plasma cells.
Q. And if -- you use the CD138 as an antibody. Would that be fair?
A. Well, what happens is that these cells have the CD38 or whatever marker you're using on them and you apply an antibody that's got a fluorescent dye with it and it hooks up with that antigen. The CD marker is the antigen and your fluorescent marker is tied to the antibody, and so what you see is sort of a brownish reaction in the tissue that is called immunoperoxidase reactions and you can tell if a cell is positive for the particular marker you have an interest in.
Q. What impact does that have on determining plasma cells?
A. Well, it's a qualitative assay in this sense because you -- it's hard from a biopsy to pick out individual cells. And what we normally use these things for -- you can use them on individual cells, but typically what will happen is we may have a lymphoma and you're staining the slide and they see if it's positive in a general way for CD5, CD20, CD23, and there are certain patterns of positivity that tell you this is

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chronic lymphocytic leukemia, this is hairy cell leukemia, this is mantle cell lymphoma and so on. There are a series of -- and so most of the time these days on pathology, they're running a series of markers.

And there are markers for plasma cells, but typically we don't use those for quantitation. As I said in my letter, we simply look at the aspirate visually and count the plasma cells.

Q. Well, if the particular sample is stained with antibodies looking for CD138 and you find that it lights up with the antibody that you're -- the sample you're testing, what does that indicate to you?

A. Well, it could indicate it's a plasma cell.

Q. Does it indicate that it's consistent with myeloma?

A. Not necessarily. In other words, any plasma cell would be positive.

Q. So, a -- what other disease process would light up CD138 other than myeloma?

A. Well, if it's a plasma cell marker, anything that gives you increases in plasma cells, that marker would be positive.

Q. Well, let's talk about that then. What gives you increases in plasma cells?

A. Well, any chronic inflammatory state. For example, all forms of hepatitis would have increased plasma cells in the

bone marrow typically, forms of rheumatoid arthritis, forms of lupus, most skin diseases that are very chronic you might see inflammatory plasma cells in the bone marrow, chronic infections, tuberculosis.

You know, as I cited one article, about almost a third of all the bone marrows they looked at had a slight increase in plasma cells if they're done on sick people.

Q. Are you telling me that there are -- there is literature out there that says that if you use -- you can use CD138 to determine whether hepatitis is creating an increase in plasma cells?

A. I have no idea. I --

Q. What do you use CD138 to demonstrate?

A. I don't use it at all. It's a marker that pathologists sometimes use.

Q. You saw that Dr. Durie says that CD138 demonstrates that the myeloma is producing the increase in plasma cells. Do you disagree with that?

A. I would disagree with that.

Q. Why?

A. Because that's -- you can't tell that kind of causation with a marker for plasma cells. A marker is a marker, and you can't tell the fingerprints of a plasma cell if you just apply a standard plasma cell marker to it.

Q. Are you aware of literature that suggests that CD138

should be used or can be used in determining whether an individual has myeloma or not?

A. No.

Q. Are you familiar with the Nig article, N-i-g? Or N-g. I'm sorry. Guess it's "Nig."

A. N-g is usually Nguyen.

Q. Nguyen?

A. Yeah. N -- whether -- if you're Vietnamese, whether your name is N-u-y-e-n or N-g, it's Nguyen.

Q. Okay.

A. Makes it real easy.

Q. That's pretty simple. Are you familiar with Nguyen's article in -- is it Hematologic --

A. If it's an article that I have here on the table that I've referenced, I could look at it. If it's some article you found in the literature, I wouldn't necessarily be familiar with it.

Q. Okay. Look at the third page then of the exhibit attached to Dr. Meehan's deposition, three of three of the pathology report.

A. Three of three?

Q. Under Interpretation of Special Stain, the last big entry.

A. That's interesting. I don't see a three.

Interpretation with special stain?

Q. Do you see that? Is it on there?

A. I don't -- not sure what you're looking at. The interesting thing about this is there are markers at the end of this deposition, and it goes 1, 2, 4, 5, 6, 7, 8, 9, 10.

Q. Okay. Maybe it's not on that exhibit. I'm looking at a different page of my book of -- it's not on there. See if it's -- no, it's not on that one.

Let's approach it from this way: You individually reviewed the aspirate, as well as the bone marrow, correct?

A. The aspirate and the biopsy material that I was provided, yes.

Q. And you looked at the aspirate and you counted plasma cells, did you not?

A. Correct.

Q. And you always got a percentage of plasma cells less than 5 percent, sometimes as low as 3 percent, correct?

A. Yes, sir.

Q. When one uses CD138 staining, will that help you see the plasma cells?

A. Well, yes and no. What happens is that when you look at those immunofluorescent stains, it really damages the cells, and so you see a lot of pigment and you're always hard-pressed sometimes to see what cell that pigment is in. And so I have never tried to use an immunofluorescent marker to count plasma

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cells. That's typically not how it's done in the standard literature.

Q. Are you saying that that is an improper way to count plasma cells?

A. What I'm saying is that the standard way to determine percentage of plasma cells in the bone marrow is by counting by light microscopy by a right stain the numbers of plasma cells in a bone marrow aspirate. That's what hematologists do and have done for generations.

Q. Are you aware of the literature which suggests staining with CD138 increases the sensitivity of the plasma cell so that you can count them better?

A. Well, when you start looking at markers, for example, you --

Q. Answer my question first, then you can explain.

A. Well, why don't you repeat your question because --

Q. Sure. Are you aware of literature which suggests that staining with CD138 increases the sensitivity to view the plasma cells so one can count them?

A. No.

Q. Okay. What does it mean if the plasma cells are clustered?

A. Not much.

Q. Okay. Is that consistent with myeloma?

A. Well, if you're talking about very, very large

clusters or so-called plasmocytomas, that might be suggestive of myeloma; but plasma cells often tend to cluster, even in what we call reactive bone marrows where there's no myeloma present.

Q. What would be the cause of clustering in reactive myeloma -- reactive bone marrow?

A. Well, when cells are reproducing, they often are clustering together; in other words, you can see clusters of red blood cell precursors, clusters of white blood cell precursors, clusters of plasma cells. And when you have conditions in which there are increased plasma cells, sometimes you'll see small clusters of them.

Q. When you said earlier "large clusters" or "small clusters," how do you differentiate by number?

A. I don't know that I would put a number on that, but you're talking about -- generally, when I say "small clusters," I'd say three, four, five cells stuck together; large clusters, maybe a thousand.

Q. Okay.

(Sotto voce discussion.)

MR. CHILTON: Dean, if this is a good time for a break, why don't we take one, unless you're relatively close to being finished.

MR. HARTLEY: Well, I think I'm -- I think I can be done by 4:00 if you -- but if you want to take a break,

that's fine.

MR. CHILTON: Well, yeah, let's definitely take a break now then.

MR. HARTLEY: 4:00 my time, 3:00 your time.

MR. CHILTON: Oh, so you're saying 20 --

MR. HARTLEY: 25, 30 minutes.

MR. CHILTON: Okay.

MR. HARTLEY: You want to take a break? I don't care.

(Discussion off the record at 2:35 p.m.)

MR. CHILTON: You know what, Dean? Let's take just five minutes so people can use the restrooms and you can take a look at your stuff and we'll start right back in.

MR. HARTLEY: Okay.

(Recess from 2:35 p.m. to 2:39 p.m.)

Q. (BY MR. HARTLEY) Doctor, are you aware of whether any anti-GBM antibodies were present?

A. I don't know that as we speak.

Q. Are you aware based on your review of the kidney pathology materials whether there was any systemic vasculitis?

A. No. There was no evidence of systemic vasculitis.

Q. Would you agree with me that one mechanism for renal failure if the patient has myeloma is the result of interstitial fibrosis?

A. Probably so. There are many, many reasons why a

person with multiple myeloma can get kidney failure, and scarring of the kidney and fibrosis would be a possibility.

Q. In your review of the pathology material, did you determine whether the basement membrane in the renal glomerulose was impacted?

A. Well, obviously, the glomeruli were impacted because they were swollen and really, to a certain extent, obliterated by crescent formation, and so certainly the glomeruli were involved.

Q. Well, that's not the same thing. Do you not distinguish between the basement membrane and the glomeruli?

A. Well, the glomeruli are the whole structure. They're a series of specialized capillaries and they have various layers of membranes. And, you know, the significance of each particular layer is something that a pathologist could discuss better than I.

Q. So, that's outside your realm of testimony?

A. Yes.

Q. Pull out Dr. Meehan's deposition, if you would.

A. Yes.

Q. And look with me on Page 41.

A. Yes.

Q. Look at Line 18. Would you read that for me?

A. Line 18 is "The presence of paraprotein IgG, kappa paraprotein, and the serum in urine and the deposit in the

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kidney is evidence of clonal plasma cell proliferation."
Q. Do you agree with that?
A. Yes.
Q. Do you agree that Mr. Lebron was anemic when he presented in March 13th of 2000?
A. Let me look back at my original report. Actually, I don't remember that, and --
Q. Well, let me ask you this: If his red blood count is decreased at 4.3, his hemoglobin is decreased at 13.4, and his hematocrit is decreased at 38.7, is he anemic, at least on March 13th, 2000?
A. Well, I have that figure of 13.4 from December 1999 in my report; and 13.4 for a man, 39 hematocrit, that would be considered very slightly anemic.
Q. Would you agree that Mr. Lebron had mutant immunoglobulin present in his urine?
A. I suppose -- well, he had -- I don't know about that term. That's a very peculiar term to use. He had a monoclonal -- or a clonal immunoglobulin in his urine. And whether it was a mutant or just multiple copies of a normal gamma globulin, I don't know. I don't know -- there were no genetic studies to demonstrate if it was a mutant or not a mutant, and I think the use of that word would be very peculiar.
Q. How about -- did he have as well the immunoglobulin

present in his serum?
A. He had an immunoglobulin -- monoclonal immunoglobulin in the serum and also in the urine.
Q. And would you agree that that same genotype that was found in the urine and the serum was found in the kidney tissue as well?
A. Yes.
Q. Would you agree that the source of that immunoglobulin is a proliferation of plasma cells in bone marrow?
A. Yes. It's generally believed plasma cells would be the general producer of gamma globulin.
Q. What types of crescentic -- how about that, Larry?
MR. CHILTON: Nicely done.
Q. (BY MR. HARTLEY) -- glomerulonephritis have been identified in association with chronic hepatitis B?
A. I'm not certain about the question, "what types." In other words, it can be -- "membranous" or "membranoproliferative glomerulonephritis" are general descriptive terms. "Crescentic" simply means that it's a very inflammatory process.
Q. Well, are there any features of membranous -- is it membranous --
A. Membranous.
Q. -- membranous crescentic glomerulonephritis in the

kidney biopsy?
A. Well, I think I would defer to Dr. McHugh in talking about membranous versus membranoproliferative. They both are seen in inflammatory kidney diseases. Membranous, for example, is more frequent in diseases like lupus. Hepatitis B can be either membranous or membranoproliferative.
Q. Well, can you define for me hepatitis B associated membranoproliferative glomerulonephritis?
A. It's an inflammatory condition of the glomerular tissue in the kidney caused by the presence of hepatitis B and it's a deposition of gamma globulins in the glomerular tissue causing damage. And it can be in the form of what we call immune complexes where the gamma globulin is sticking to a hepatitis antigen and then that complex of antibody and antigen is sticking in the kidney, or it could be simply deposition of the antibody of the gamma globulin in the kidney.
Q. What specific immune complex would you see being deposed in the kidney?
A. You -- the hepatitis B has certain antigens, and they're given letter-numbers. I'm not totally conversant with that, but some of these articles describe that. And he had those studies done on his liver biopsy where they demonstrated hepatitis B antigens in his liver biopsy. Those antigens, same antigens, can be in the kidney, complexed with the gamma globulin.

Q. Do you know if they were?
A. They were never measured in the kidney; the antigens, that is.
Q. What antigens specifically are you referencing?
A. Hepatitis B antigens, proteins produced by the hepatitis B virus.
Q. What about the immune complex deposition?
A. That's it. In other words, "immune complex" simply means gamma globulin attached to an antigen or an antigenic protein. It's a complex of antibody and antigen, and that complex can float into the kidney and damage it; sometimes referred to as immune complex disease.
Q. But you don't know what those immune complex disease antigens would be as we sit here right now?
A. Well, in Mr. Lebron's case, they would be a complex of his gamma globulin with a hepatitis antigen.
Q. Okay. And what particular gamma globulin would we be looking for in the kidney?
A. We'd be looking for the gamma globulin that the hepatitis excited production of.
Q. Well, what would that be?
A. That could be what he had. In other words, when you have an antigen in your system, it stimulates production of gamma globulin. That gamma globulin can be monoclonal or polyclonal, and he had a monoclonal gamma globulin. And --

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Q. What did he have in his kidney?
A. He had gamma globulin stuck in his kidney.
Q. Okay. And what particular -- what else -- is that consistent with a membranoproliferative glomerulonephritis?
A. Yes.
Q. It is?
A. Yes.
Q. Okay. What would you expect the -- let me ask you this: What would -- what -- how would -- what -- does it have a particular letter for it? When you're talking about globulins being in the kidney, is there anything more specific than that?
A. Well, yes. He had an I -- gamma globulins, they're referred to as IgG, IgA, IgM, as general classes of gamma globulins. And people with hepatitis B can have what's called IgA nephropathy with IgA gamma globulins sticking to the kidney or they can have IgG globulins stuck in the kidney. In his case, he had IgG globulins stuck in the kidney.
Q. And is that enough by itself to assert that the immune complex deposition of IgG supports the diagnosis of the -- is it membrano?
A. Membranoproliferative? I think the histology supports the diagnosis of membranoproliferative. The immune complex concept is suspected because he wasn't -- his kidney was not stained for the hepatitis antigens.

Q. Was there any information on whether the hepatitis B antigen was -- hepatitis B antigens serology was positive or not?
A. Well, his hepatitis B --
Q. For the kidney now.
A. We're talking in the kidney. He did not have stains looking for antigens of hepatitis in the kidney tissue.
Q. How would -- if we were looking at this membranoproliferative glomerulonephritis, what are we looking for and what description are we looking for of the slides themselves?
A. We're looking for swelling and inflammation of the glomeruli and -- as they described. In other words, I -- let's go back to look at their description here.
Q. Okay. You're going to review Exhibit 1?
A. Yes.
Q. The attachment to Exhibit 1?
A. Yes.
Q. Okay.
MR. CHILTON: From the Meehan deposition.
MR. HARTLEY: Yes, yes.
MR. CHILTON: Okay.
A. It's the -- the sentence there is "Extensive periglobular glomerular fibrosis around three glomeruli, and three other glomeruli show fibrocellular crescents, which in

one glomerulus is circumferential. There are several foci with tubular separation and lymphocytoplasmic infiltrate in the interstitium." Let's see what else they go on to say.
"Electron microscopic examination demonstrates abundant punctate electron-dense deposits in the mesangial matrix and in the glomerular capillary wall. Those deposits are seen immediately under the endothelium."
These all -- generally, they're talking those deposits are by the electron microscopy. And those deposits, when they're granular, are representing the gamma globulin.
Q. (BY MR. HARTLEY) Okay.
A. And so these -- this report tells me that he's got an inflammatory reaction in the kidney which is consistent with glomerulonephritis.
Q. And can you give me a reference that I might go and review to see if this type of pathology that's described corresponds with the disease process that you have suggested?
A. Well, I -- there are many references about glomerulonephritis and hepatitis B, and --
Q. I'm more concerned with description of the process via pathology.
A. Well, I -- one of the articles I referenced is by DiMarco, and he says here "Renal biopsy showed that" -- excuse me. This is "Therapy with lamivudine and steroids in a patient with acute hepatitis B and rapidly progressive

glomerulonephritis."
Q. What exhibit number is -- what reference number is that?
A. That is -- let's see.
MR. CHILTON: It's No. 14, I believe, Dean.
Q. (BY MR. HARTLEY) "Therapy with" --
A. Yes. That's Epivir -- therapy with lamivudine, which is also called Epivir, which is the drug that he received, that Mr. Lebron received. And in that table, it says "Renal biopsy showed mesangioproliferative glomerulonephritis with crescents." Hepatitis B antigen and hepatitis -- and deposition and glomeruli were shown by immunofluorescence by monoclonal antibodies.
Q. Okay. I understand that. I'm looking more for a definition or an item that we might look at to tell us renal biopsy showed mesangio --
(Discussion off the record at 2:57 p.m.)
Q. (BY MR. HARTLEY) What is it, mesangioproliferative?
A. Yeah, mesangioproliferative.
Q. Mesangioproliferative. Where do I find a definition of what the pathology slide should look like for that? That's what I'm concerned -- that's what I want to know.
A. Well --
Q. A pathology textbook, I guess, would do it, right, for the kidney?

A. You can do that. For example, another article that I referenced by Chuang, C-h-u-a-n-g -- I'm not sure what number that is.
Q. 15 maybe, "Complete remission"?
A. Yeah. He says -- well, let's see. "We used electron microscopy and found viral-like particles appearing in clusters around the ovoid structures within the glomerular capillary basement membrane."
Q. Where are you on that?
A. I'm on Page 872. It actually starts on the prior page. Let's see if I can see where that is.
"The diagnosis of hepatitis B virus associated membranous nephritis is usually established by serologic evidence of persistent HBV infection, the presence of immune complex deposits on kidney biopsy, and the demonstration of glomerular deposits containing one or more HBV-related antigens."
Q. Do we have any of that?
A. Well, you've got the inflammatory reaction of the glomerulonephritis. You've got the antibodies. They weren't stained by the antigen. They weren't stained for the antigen.
Q. Do we have the antibodies in the kidney?
A. You have antibodies in the kidney.
Q. That are HBV?
A. Antibodies, I would assume, against HBV produced by

the presence of the HB -- produced in response to the HBV infection.
Q. Well, I think that's different, isn't it? They're saying that the deposits containing one or more HBV-related antigens.
A. Yes.
Q. HBV-F and hepatitis B core antigens. Is that demonstrated in this kidney? I mean --
A. I told you repeatedly, the kidney was not measured for the antigens, just the antibody. So, when they stained this kidney for the antibody, they see the antibody in one area. Had they stained it for the antigen, they would have seen the antigen superimposed on the antibody because they're stuck together; but they didn't choose to stain the kidney because they didn't know he had hepatitis B.
Q. Who didn't know?
A. The people who looked at the kidney biopsy.
Q. Well, he was diagnosed with H -- with hepatitis B early, wasn't he?
A. Not -- no. The kidney biopsy was long before that diagnosis. The only way they diagnosed him with hepatitis B was after the kidney biopsy. And after they treated him, they caused a recrudescence of his hepatitis B by giving him immunosuppressant drugs and he came in with hepatic coma and that's when they tumbled to the diagnosis of hepatitis B.

Q. So, would it be fair to say that since they didn't know to look for hepatitis B in the kidney, that there were no tests run to confirm whether the antigens or the antibodies are in the kidney?
A. Well, they got the antibodies in the kidney. They didn't test for the antigens.
Q. Where is that?
A. Well, that's the IgG that they're seeing. That's an antibody.
Q. Oh, that's an antibody specifically for hepatitis B. Is that what you're telling me?
A. I'm telling you that hepatitis B is well-known to produce monoclonal antibodies.
Q. Other things produce it, too, though, don't they?
A. Sure.
Q. Okay. So, we don't have specificity as to hepatitis B in this kidney sample, do we? We have the immunoglobulins in there, but they can be caused by any number of different diseases or situations that we've already talked about, correct?
A. Yes. He has immunoglobulins in the setting of an acute glomerulonephritis that has destroyed his kidney in the setting of hepatitis B, and I think one would assume that that's the cause. Now, this -- had they known he had hepatitis B or thought to look for hepatitis B, those -- that kidney

could have been stained for hepatitis B, just like they stained their liver.
Q. When did they do the liver?
A. They did the liver biopsy when he came in in hepatic coma and they couldn't figure out why in the world he would suddenly go into hepatic coma. They thought they were treating somebody with kidney disease and when he came in in hepatic coma, they found out that he had striking titers for the hepatitis B virus and they biopsied his liver for the first time and found that he had chronic hepatitis and that he was positive for the hepatitis B antigens in the liver, as well as the particles of virus in the circulating blood.
Q. That was, I believe, in the fall of 2000. Does that sound about right?
A. It's probably in my original letter. Let's see. In April of 2001, prior to initiation of dialysis, he required hospital admission for an altered mental status and was found to have an elevated blood ammonia level. He was treated for hepatic encephalopathy and that's when they found the viral load. And the liver biopsy was done on 08-22-01.
Q. So, would it be fair to say then that when he was examined for the foamy urine and the kidney studies were done, we don't have any evidence of serological -- we don't have any serological evidence of persistent HBV infection at that time?
A. That would be correct, yes.

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Q. And we don't have any evidence of HBV-related antigens, HBsAg and hepatitis B core antigen, at that time in his kidney, do we?
A. Correct.
Q. The only thing we have is some immune complex deposits on kidney biopsy, correct?
A. No. What we have is a liver biopsy that shows he's got chronic hepatitis.
Q. Not in April of 2000, now, do we?
A. Well, but the liver biopsy was done -- I mean, has to -- when you see a liver biopsy that shows chronic inflammation for hepatitis, you didn't catch the hepatitis yesterday.
Q. In April of 2000, did we have anything other than some immune complex deposit on the kidney biopsy to make a determination that he did or did not have hepatitis B in April of 2000?
A. There was no diagnosis of hepatitis B in April of 2000.
Q. Could hepatitis B have been made in April of 2000 based on the information that you've seen in the medical records?
A. Absolutely.
Q. And what would that have been?
A. Well, this patient came in the hospital because of a

nephrotic syndrome, and the nephrotic syndrome was typically due or frequently due to various forms of glomerulonephritis. And the usual workup of a patient like that is to look for causes of glomerulonephritis that might produce the nephrotic syndrome. Those would be illnesses like lupus, for example; illnesses like HIV, for example; illnesses like hepatitis B and C, for example; diabetes, for example. There are many illnesses that can produce glomerular damage and he simply was not worked up for those illnesses. He didn't have an evaluation for these illnesses.
Q. You mistook my question. There's nothing in the medical records in April of 2000 which would permit you to make that diagnosis based on the records themselves, correct?
A. That is correct.
Q. Okay. Have you reviewed the medical records of Dr. Potempa? Is that how you pronounce it?
A. If they're in the medical records I have, I have reviewed them.
Q. I think he was either a second opinion or he was a nephrologist who originally saw him.
A. That may be so. I can't dispute that.
Q. Okay. You just don't remember?
A. I don't remember, but I do remember that there's a report from a nephrologist that says he has glomerulonephritis.
Q. He's got a report from Dr. -- do you remember this

report from Dr. Potempa in April of 2000 where he said, "Renal biopsy demonstrates a condition from the monoclonal production of light chain and heavy chain. There is a clone in the bone marrow producing these immunoglobulins."
Do you remember that?
A. Vaguely.
Q. And he concluded that this would be associated with myeloma, lymphoma, Waldenstrom's macroglobulinemia -- globulinemia -- you know what it is.
MR. CHILTON: Can you tell us what you're reading from, Dean, what medical record?
MR. HARTLEY: It's Potempa's report or letter to, I believe, one of his other treaters.
MR. CHILTON: Because I could be wrong about this, but I don't think Potempa was a nephrologist. I thought he was just one of his, like, family physicians. That's why I'm curious what you're reading from. You -- because you represented that he was a nephrologist and I don't recall that being correct. It may be.
MR. HARTLEY: In the letter to Reda.
MR. CHILTON: A letter to who?
MR. HARTLEY: R-e-d-a, Joseph Reda.
MR. CHILTON: Okay.
MR. HARTLEY: Dated April 17, 2000.
MR. CHILTON: And it's from Dr. Potempa?

MR. HARTLEY: It is.
MR. CHILTON: And does it list what type of physician he is?
MR. HARTLEY: Well, his letterhead does not say that, but he had requested surgical path, etcetera, so -- and he worked him up.
MR. CHILTON: He was at La Grange, correct?
MR. HARTLEY: Correct.
MR. CHILTON: Okay. Thanks. Just so we have something to reference.
Q. (BY MR. HARTLEY) You don't remember that, Dr. Natelson?
A. I don't remember that.
Q. Okay. Oh, there was one follow-up, I think. I have tried not to go into your deposition from previously so we wouldn't go over the same crap.
MR. HARTLEY: Don't put "crap" down there, Miss Court Reporter.
Q. (BY MR. HARTLEY) Doctor, is chemotherapy of the VAD type myeloma specific?
A. No.
Q. What else is VAD used to treat?
A. Well, VAD has been used to treat situations where you want to suppress immunoglobulins. The treatment was developed originally by Dr. Alexanian at MD Anderson Hospital, I believe,

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and he used it in treatment of myeloma, but it is -- basically would be considered immunosuppressive treatment, and that's how they used it in this case.

Q. Is there any literature that suggests that VAD be used for immunosuppressant purposes?

A. Well, the "D" is dexamethazone. That's a steroid which is used by anybody who does transplant work or -- it's a commonly used immunosuppressive drug, as well as anything else.

Q. It kills lymphocytes, doesn't it?

A. It does. It's been used for a zillion things, but it's a strong immunosuppressive drug.

Q. How about thalidomide?

A. Thalidomide?

Q. Yeah. Is it myeloma specific?

A. No, certainly not.

Q. What other diseases that may be impacted by thalidomide? Or what other diseases may be impacted by thalidomide?

A. Well, it was actually first used in leprosy. It's an antiinflammatory drug and that's how it was actually introduced into practice. It had been used successfully in patients with leprosy. Then it was used to treat inflammation in an unusual illness, and let me just think if I can remember the name of it. It's an illness that's characterized by ulcers in the mouth and vagina, among other places. It's got a peculiar

name.

Q. Not thrush, is it?

A. No, no, no, no, no. It's -- I have a mind blank now, but it's an inflammatory disease that is very distressing and immunosuppressive therapy has been used and thalidomide was an early use and very effective in this condition. And it's also associated -- this particular condition is associated with thrombophlebitis, too. And I'm just blanking on the name. It's an unusual spelling and it will probably come to me in a few minutes.

But, anyway, it's been used as an antiinflammatory drug and it was used in breast cancer, for example. It's used in people with myelodysplasia who have sick bone marrows. It's had a wide variety of uses.

Q. Would glomerulosclerosis be consistent with your crescentic glomerulonephritis opinion?

A. No, because all of these articles that Dr. Meehan cites and those that I cite that talk about this illness, the so-called light and heavy chain deposition disease, you will not find the word "crescent" or "crescentic" mentioned in any of those articles. Therefore, I would take that to believe that that's not a characteristic finding in that disease.

Q. Are you saying that if -- you're saying that because there were crescents seen in a couple of the glomeruli, that -- and they're not reported in any of these articles that you've

looked at or cited by Meehan, therefore, it can't be glomerulosclerosis. Is that what you're saying?

A. What I'm saying is that 30 -- you said three out of nine, I think, in the first one. 30 percent of the glomeruli had crescents in them, and one would think that if you see that kind of a phenomenon in this illness, the doctors writing papers on it, like Dr. Ronco, would discuss that. And not only do they don't say it, but they -- he has a statement in here suggesting that it doesn't occur.

Q. Okay. And would you then say that the patient could not have glomerulosclerosis?

A. I would say he had glomerulonephritis.

Q. That's not the same thing as I'm saying. I'm asking you are you going -- is it your opinion as someone who reviewed the pathology in this particular case of a kidney that Mr. Lebron did not have glomerulosclerosis?

A. I'll defer that to Dr. McHugh. When you think about it, glomerulosclerosis just means scarring or damage to the glomeruli. The process that he had was glomerulonephritis. The end result is sclerosis.

Q. So, you can have glomerulosclerosis and be consistent with your diagnosis of crescentic glomerulonephritis --

A. Well, as an end stage --

Q. -- your opinion as a hematologist reading pathology of the kidney and studying -- and the kidney itself.

A. My position as a hematologist is that Mr. Lebron's illness was consistent with a glomerulonephritis from hepatitis B; and that when you get a kidney that's an end stage kidney and it's burned out, it -- all the glomeruli are fibrotic and sclerotic and you may not be able to know what the inciting situation was.

Q. When you say the kidney is burned out, what do you mean by that?

A. Well, what happens is that initially when someone gets an inflamed kidney, it may be swollen and large and porous to protein, like it was in Mr. Lebron's case, and you're losing a lot of protein in the urine; but at the same time, your waste product levels, the BUN and creatinine levels, are normal. After a while, the inflammation there starts to damage and destroy the glomeruli, so now the waste product levels start to come up.

So, when Mr. Lebron was originally seen in 1999 and had the nephrotic syndrome, his renal function was normal, his BUN and creatinine are normal. When he comes back for the second time around, he's got abnormal renal function with an elevated serum creatinine level. So, the kidneys -- the glomeruli are starting to become shot. And then as time goes by, they shut down entirely, and he has to go on dialysis. And what happens if you looked at those kidneys, they start to get a little small as they scar up and fibrose.

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Q. Do you fault the physicians who treated him when he first appeared with his nephrotic syndrome that you've just described for failing to properly work him up and discover the hepatitis B at that time?

MR. CHILTON: I'll object. And you've already covered this in his prior deposition and we're not going to cover it again, Dean.

MR. HARTLEY: I don't think I did. I talked about treatment. I'm asking if he worked him up.

MR. CHILTON: Well, that wasn't the question you just asked.

MR. HARTLEY: Sure, it was.

MR. CHILTON: I disagree, so I'm objecting to the question.

MR. HARTLEY: Are you instructing him not to answer?

MR. CHILTON: Have I instructed him not to answer?

MR. HARTLEY: I'm just asking if you did.

MR. CHILTON: No.

MR. HARTLEY: Okay. Fine. But you said we're not going there.

MR. CHILTON: Well, we're not going to go back into topics that were covered at the original deposition. Having said that, I did not instruct the witness not to answer.

MR. HARTLEY: Okay.

Q. (BY MR. HARTLEY) Doctor, that means you can answer.

A. Okay. Fine. My opinion is this: You have a patient that comes into the hospital with foamy urine. You find that he's got a strikingly abnormal amount of protein in his urine. He's got nephrotic syndrome. And for one reason or another, he was not worked up. Now, I wasn't there at the time. I don't know if he didn't stay for a workup. I don't know if the doctors didn't advise him to get a workup.

But the bottom line, he was not properly evaluated for that condition at that point in time so that when he came back in 2001, he's far worse than he was. And the -- and had he been diagnosed early on with hepatitis B and appropriately treated, he may never have gone into renal failure. But I don't know why that was the case. In other words, I don't know whether he didn't choose to have a workup, wasn't advised to have a workup, told to come back a week later and never came back. I have no idea. That you'd have to ask his original doctors.

Q. Well, one final question then: Assuming he was hospitalized for this workup, was seen by a couple physicians for this workup, did not refuse to do anything that was asked of him, if the physicians who were treating him did not include hepatitis B in his differential diagnosis and work him up for hepatitis B, did they breach the standard of care?

MR. CHILTON: I'm going to object and I am going to instruct him not to answer. He's not here to testify as to issues related to standard of care and potential actions that were taken by his treating physicians relative to that treatment. I don't think that he's being presented for that purpose in this case and he's not going to testify with respect to opinions on that topic.

MR. HARTLEY: Then that means at trial, he will not be chastising those physicians for not working him up when he should have been worked up in April of 2000. Is that what you're saying?

MR. CHILTON: That's not what I said at all.

MR. HARTLEY: Okay.

MR. CHILTON: You asked a different question.

MR. HARTLEY: We'll take it up with the court then. That's the easiest way to do it. Because if he's going to testify that he wasn't worked up properly so as to impugn the physicians, I think we're entitled to know whether that's a breach of the standard of care so that the jury can actually know where he stands on this issue. But we'll take it up on a later date. It's okay. I don't have anything further.

MR. CHILTON: Anybody else have anything?

MR. HARTLEY: Just a second.

MR. GILMARTIN: Nope.

MR. MILLER: Nope.

MR. CHILTON: We all set?

MR. HARTLEY: Wait a minute. Just a second. I finally got Dr. Barrios' report. Just want to look at this for a second.

Q. (BY MR. HARTLEY) Doctor, did you point out to Dr. Barrios that the lesion that he -- it says here, "As you point out" -- this is in a letter from him to you -- "this lesion can be associated to hepatitis B."

Did you bring it to his attention that whatever was seen on the pathology could be associated with hepatitis B?

A. Well, I gave him the slides. I said, "I want you to look at some kidney slides," and he looked at it.

And about 30 seconds, he said, "This is a crescentic glomerulonephritis and I would be thinking about things like HIV disease and hepatitis C."

And I said, "How about hepatitis B?"

And he said, "Yeah. Hepatitis B certainly can produce this." And then I told him the situation of what the case was all about. And so when he wrote the letter, he had already had a discussion with me about the fact that the patient did have hepatitis B; but spontaneously he brought it up that the first things he would think about when he looked at a biopsy like that would be HIV, hepatitis C, and hepatitis B.

Q. Did you give him the reference to -- it's either Lai or Lai, L-a-i, the citation that he uses, "Crescentic

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glomerulonephritis related to hepatitis B virus"?
A. No, no.
Q. Is Dr. Barrios a renal pathologist?
A. Yes. I went to Mary Schwartz, who's really their head of the department, and I said, "Who would be the best person to look at a kidney biopsy?"
And she said, "Show it to Dr. Barrios."
Q. Okay. That's all I have.
MR. CHILTON: Very good. Anybody else?
UNIDENTIFIED SPEAKER: No.
MR. CHILTON: Okay. We've got the one exhibit. I'll ask the court reporter to attach that to the transcript. Very good. Thanks, everybody.
MR. GILMARTIN: Shanon, I'll take a copy and also a digital file, Bob Gilmartin.
THE REPORTER: Okay. Thanks.
MR. MILLER: And I want a copy, too. This is Jim Miller.
THE REPORTER: Okay.
MR. HARTLEY: Shanon, naturally, I'd like a copy.
THE REPORTER: Okay.
MR. HARTLEY: All right. Thanks a lot.
(Deposition concluded at 3:22 p.m.)

CHANGES AND SIGNATURE

PAGE	LINE	CHANGE	REASON

I, ETHAN A. NATELSON, M.D., F.A.C.P., have read the foregoing deposition and hereby affix my signature that same is

true and correct, except as noted above.

_____
ETHAN A. NATELSON, M.D., F.A.C.P.

THE STATE OF _____)
COUNTY OF _____)
Before me, _____, on this day personally appeared ETHAN A. NATELSON, M.D., F.A.C.P., known to me (or proved to me under oath or through _____) to be the person whose name is subscribed to the foregoing instrument and acknowledged to me that they executed the same for the purposes and consideration therein expressed.
Given under my hand and seal of office this _____ day of _____, _____.

_____
NOTARY PUBLIC IN AND FOR
THE STATE OF _____

STATE OF TEXAS
COUNTY OF HARRIS

I, the undersigned certified shorthand reporter in and for the State of Texas, certify that the facts stated in the foregoing pages are true and correct.

I further certify that I am neither attorney or counsel for, nor related to or employed by, any of the parties to the action in which this deposition is taken and, further, that I am not a relative or employee of any counsel employed by the parties hereto, or financially interested in the action.

SUBSCRIBED AND SWORN TO under my hand and seal of office on this the _____ day of _____, 2008.

_____
SHANON M. HAIR, CSR
Certified Shorthand Reporter
In and for the State of Texas

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STATE OF ILLINOIS
IN THE CIRCUIT COURT OF COOK COUNTY, ILLINOIS
COUNTY DEPARTMENT-LAW DIVISION

PAMELA LEBRON, as Personal Representative of the Estate of MICHAEL (MIGUEL) LEBRON,	)	
Plaintiff,	)	Civil Action No. 05 L-9246
vs.	)	
SHELL OIL COMPANY, et al.,	)	
Defendants.	)	

****

TELEPHONIC DEPOSITION OF

ETHAN A. NATELSON, M.D., F.A.C.P.

November 11, 2008

CERTIFIED COPY

Volume 1

****

TELEPHONIC DEPOSITION of ETHAN A. NATELSON, M.D., F.A.C.P., produced as a witness at the instance of the Plaintiff, Shell Oil Company, and duly sworn, was taken in the above-styled and numbered cause on the 11th of November, 2008, from 1:07 p.m. to 3:22 p.m., before Shanon M. Hair, CSR in and for the State of Texas, reported by machine shorthand, at the offices of Schirrmeister Diaz-Arrastia Brem, LLP, Pennzoil Place - North Tower, 700 Milam, 10th Floor, Houston, Texas 77002, pursuant to Rule 201(a), Illinois Supreme Court Rules, and the provisions stated on the record or attached hereto.

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(1:07 p.m.)

ETHAN A. NATELSON, M.D., F.A.C.P.,

having been first duly sworn, testified as follows:

EXAMINATION

BY MR. HARTLEY:

Q. Dr. Natelson, how are you today?

A. Fine. Thank you.

Q. Just for the record, could you tell us your name?

A. Yes. Ethan A. Natelson.

Q. And, Dr. Natelson, I have in front of me a report dated July 14, 2008. Do you have a copy of that report with you?

A. Yes, sir.

Q. Have you updated that report since July 14th?

A. No.

Q. Have you done any other research or going to rely on any other literature since July 14th and what's referenced in that report?

A. Well, yes. We sent you -- at the time of the last attempted deposition, I had some additional papers --

Q. Okay.

A. -- and we -- I believe Mr. Chilton sent those to you and we have you those here also.

Q. Okay. And I have those.

What -- in the first paragraph of your July 14th

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letter, you indicate that you've had the opportunity to review the pathology slide material.

A. Yes.

Q. What particular pathology did you review?

A. These were bone marrow slides, and also the kidney biopsy slides.

Q. Did you review any of the liver slides?

A. Yes. The liver slides were there also and I looked at those.

Q. In your practice, Dr. Natelson, do you normally review kidney slides?

A. I would say rarely. If it's a particular case that I have an interest in, I might, but not commonly.

Q. Did you review these kidney slides with any particular pathologist or any other person?

A. Yes, with Dr. Barrios, who submitted a report about them.

Q. Did you review the liver slides with anyone else?

A. Yes. I looked at them with Mary Schwartz, who's the liver pathologist at Methodist Hospital, just having her take a casual look through the slides. I did not ask her to write up an opinion.

Q. And do you normally review liver slides in your practice as a hematologist?

A. Again, I might. It really depends on the individual

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case, but I certainly have looked at liver slides in the past.

Q. Out of patients that you've seen, how many times have you in your practice -- and you can give me a range or a percentage -- reviewed the kidney slides of a particular patient?

A. It would be hard to give you a number on that because I don't -- I'm not a kidney specialist and so I normally don't see patients who have only kidney disease, per se. I'm a hematologist. But if I have a patient who's got a hematologic problem that I have been following and they suddenly develop something else that requires a tissue biopsy, it's not unusual to look at that biopsy, too.

Q. Do you have any training in kidney pathology?

A. No.

Q. Do you have any training in liver pathology?

A. No.

Q. Do you review kidney slides once a month in your normal practice?

A. No.

Q. Once a year?

A. Probably, yes.

Q. And how long have you been practicing?

A. Oh, since -- seeing patients since around 1969.

Q. So, that's about 40 years --

A. Yes.

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Q. -- give or take.
A. Yes.
Q. Would it be fair to say then that you, over the course of your 40 years of practice as an oncologist or hematologist, have seen on average 40 -- or have looked at 40 kidney slides of your particular patients in your practice?
A. That's probably so.
Q. How about liver slides? Do you look at liver slides once a month?
A. It depends on the years. In other words, there have been times that I've looked at liver slides more frequently than that and times that I've looked at them less frequently.
Q. Do you think you looked at liver slides as much as you looked at kidney slides?
A. I would say possibly slightly more.
Q. Can you give me -- twice a year, once -- one-and-a-half times a year? Can you give me some type of an idea?
A. I -- it would be really hard to just put any kind of an estimate on there, but -- as a hematologist giving transfusions and seeing patients with hepatitis, I more often would have an opportunity to look at liver biopsies than kidney biopsies.
Q. Do you normally look at those biopsies, liver biopsies, with another -- with a liver pathologist?

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A. Yes.

Q. Do you ever review those pathologies of the liver by yourself and rely on your own reading of those slides?

A. No.

Q. Same question for kidney pathology that you might have been looking at. Do you normally look at those biopsies under the microscope with a kidney specialist or kidney pathologist?

A. Yes.

Q. And do you rely solely on your own review of those slides when you're treating your patients in your normal practice or that of the pathologist?

A. I would rely on the pathologist.

Q. You certainly don't hold yourself out as a liver specialist either, do you?

A. No.

Q. Have you had any real training as it relates to the study of liver pathology?

A. No.

Q. Have you written any articles on liver pathology?

A. No.

Q. Have you written any articles on kidney pathology?

A. No.

Q. Do you teach any of the folks who might come through your rounds kidney -- medicine that relates to kidney problems?

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A. No.

Q. Same question for liver. Do you treat (sic) any of the folks that may come through on your rounds about liver problems?

A. Well, we commonly discuss liver problems at our morning report, but -- I think if you're suggesting do I teach liver pathology to our interns, the answer would be no.

Q. Would you defer to a pathologist who specializes in kidneys for their opinion as to what the slides actually show?

A. Yes.

Q. Same question for liver pathology. Would you defer to a liver pathologist as to what the slides actually show?

A. Yes.

Q. In the July 14th letter -- why don't we mark that as Exhibit 1.

(Natelson Exhibit No. 1 marked.)

THE REPORTER: Okay. It's marked.

MR. HARTLEY: Thank you.

Q. (BY MR. HARTLEY) Dr. Natelson, let me show you what's been marked as Exhibit No. 1. Can you identify that for me?

A. Yes. This is a letter dated July 14, 2008, that I sent to Mr. Louis Woolf.

Q. And the copy that I have is about nine pages long. Is your copy nine pages? Includes the references.

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A. Let's see. That is true.

Q. Okay. Look in the first full paragraph on Page 1, about the middle of that paragraph. "As you know, it is my opinion that Mr. Lebron suffered several well-described complications of hepatitis B," paren, "HBV," close paren, "virus infection which included" --

A. Yes.

Q. -- then there is a list of items after that.

Is it your opinion that all of the medical problems, situations, syndromes, or anything described after "which included" are solely related to Mr. Lebron's hepatitis B virus?

A. Correct. That is my opinion.

Q. Now, did -- in the first phrase after that "which included," you have "the nephrotic syndrome and renal failure due to" -- is it crescentic? Is that a correct pronunciation?

A. Yes.

Q. -- "crescentic glomerulonephritis."

Did you personally make a diagnosis of crescentic glomerulonephritis?

A. No.

Q. Who did?

A. Well, in the original pathology reports, the word "crescentic" is mentioned, and their description is that of a glomerulonephritis. But when I showed the slides to my

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pathologist at Methodist Hospital, he commented, "This is a typical crescentic glomerulonephritis."

Q. And that would be Dr. Barrios?

A. Dr. Barrios.

Q. Barrios.

A. And that didn't surprise me because that was the description in the original pathology.

Q. And what particular pathology report -- do you have those with you?

A. Yes.

Q. What report are you referencing, the April 10th report from the materials obtained April 3rd, 2000?

A. I'm referencing Dr. Barrios' report of May 30, 2007.

Q. Oh, okay. So, you're referencing Dr. Barrios.

MR. CHILTON: Dean, let me interrupt. I think your question though was about the original pathology report.

MR. HARTLEY: It is, it is, and we're going to clean that up now.

MR. CHILTON: Okay. Thank you.

Q. (BY MR. HARTLEY) So, Dr. Barrios diagnosed crescentic glomerulonephritis, correct?

A. Correct.

Q. Did any physician who saw the pathology in the regular course of business, either at Loyola or at La Grange or at any other facility in the Chicagoland area, make a diagnosis

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of crescentic glomerulonephritis?

A. Well, the -- I don't have all those original records there, but the kidney doctor that saw him wrote in his notes that he had glomerulonephritis.

Q. Who would that have been?

A. I don't remember the man's name.

Q. Well, it's not -- is it Leonard Potempa?

A. I would just be speculating. I haven't looked at those records for some years.

Q. But it's your opinion that the kidney doctor made the diagnosis of glomerulonephritis as a result of the crescents that were seen on the pathology slides?

A. No. I don't know that he made a diagnosis on the basis of the crescents. He simply says in his consult notes that the patient has glomerulonephritis. That was after the renal biopsy was done.

Q. After the renal -- did the pathologist who reviewed the kidneys come to that conclusion?

A. The pathologist did not use the term "glomerulonephritis" as his final conclusion on that slide.

Q. And as I understand it, you don't have the nephrologist's information to identify for me who the nephrologist was that made the diagnosis of crescentic glomerulonephritis?

A. As I said, I don't recall the nephrologist using the

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term "crescentic glomerulonephritis," but he certainly said "glomerulonephritis."

Q. Okay. So, then the only people who have used the term "crescentic glomerulonephritis" would be you and Dr. Barrios?

A. No. That would not be true. I believe Dr. McHugh, another pathologist, has reviewed this and has commented about that.

Q. And did he make a final diagnosis of that?

A. I'd have to look again. In his report, he cited the crescentic changes and said that he had, I believe, membranoproliferative glomerulonephritis.

Q. Secondary to hepatitis B infection. And as I understand what he wrote, he had down there "provisional" because he wanted to see the photomicrographs. Isn't that true?

A. We could pull out his report. I think I may have it here. I don't remember that, but let's see what he says.

He says, "A renal biopsy showed a proliferative glomerulonephritis with partial crescent formation." I take that to believe he is calling it a crescentic glomerulonephritis.

Q. And what's he say at the end?

A. "I would like the opportunity to review the electron microscopy photomicrographs and reserve the right to modify my

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impression of the exact classification of the patient's renal disease."

Q. Does that mean that his diagnosis would then be provisional upon reviewing the photomicrographs?

A. You'd have to ask him that.

Q. Did you review the photomicrographs when you reviewed the material for this case?

A. I didn't have them when I had the slides for Dr. Barrios to look at. And I do have them here, but I'm certainly not an expert on electron microscopy.

Q. So, you're not an expert on electromicroscopy, you're not an expert on the pathology of kidneys, but you've talked to Dr. Barrios who has concluded that it is a crescentic glomerulonephritis and, provisionally, Dr. McHugh has said that; so, in your opinion, he has crescentic glomerulonephritis. Is that true?

A. In my opinion, he has crescentic glomerulonephritis. As to whether or not Dr. McHugh's report is provisional or not, you have to ask him.

Q. And tell me, Doctor, what do you base that diagnosis of crescentic glomerulonephritis on?

A. Well, he had numerous crescents in the kidney biopsy. That's a very specific finding. And I listed a paper in here that discusses what these -- what the significance of crescentic changes are. And that report spells out that when

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you see crescentic changes in a glomerulonephritis, it just simply means it's a very inflammatory glomerulonephritis.

Q. Okay. So, would it be fair to say that your opinion and the basis for that opinion that Mr. Lebron has glomerulonephritis -- crescentic glomerulonephritis is the result of your reading the article that tells you what crescent findings on kidney slides indicate?

A. No. That's a piece of information I have to clarify what the term "crescentic" means. There are plenty -- I think I may have other reports on crescentic glomerulonephritis, and I've certainly heard that term used in clinical medicine for many years.

Q. Well, the fact that you've heard it used doesn't mean that the patient has it, does it?

A. No, but I -- as I say, when I reviewed these slides and discussed it with the pathologist, in view of what's there, that diagnosis is apparent.

Q. Okay. Did you happen to look at -- let me strike that for a second.

So, your review of the slides in conjunction with Dr. Barrios and your review of the medical literature on what crescentic glomerulonephritis requires or includes is the basis for your opinion in this particular matter that Mr. Lebron had crescentic glomerulonephritis, correct?

A. My opinion is the fact that all of the pathologists

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that have looked at this biopsy have commented on the fact that he has crescentic changes. That means a very specific thing in terms of glomerulonephritis. So, it's -- it just simply means that it has -- that it has an inflammatory character to it.

Q. Do you have the original pathology report?

A. I don't think so.

MR. CHILTON: You mean with him here today?

MR. HARTLEY: Yes.

MR. CHILTON: Okay. Do you -- can you direct us to what report it was?

MR. HARTLEY: It's the Picken report, or Picken. Picken, dash, M-r-o-z-o-w-i-c-z, from Loyola. The session number is S, as in Sam, 00-5459 and the report date is April 10, 2000.

MR. CHILTON: We're taking a look, Dean. We don't have all of the medical records here today.

MR. HARTLEY: Okay.

Q. (BY MR. HARTLEY) Do you know -- do you have Dr. Meehan's deposition with you?

A. Yes, sir.

Q. I think it's an attachment to Dr. Meehan's deposition.

A. That's where I'm looking right at the moment.

Q. In fact, I think it's Exhibit 1.

A. Yes. I'm looking at it right here, I think.

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Q. It's -- at the top, it says "Microscopic Description"?

A. Correct.

Q. And at the bottom, it says "Lebron." And if you look on the far right, it says "Page 2 of 3," but it says "S00-5459."

A. Correct.

Q. Okay. Look at the interpretation that you've been talking about that there were crescents shown.

A. I didn't say they were in the interpretation. I said they're in the body of the report. They're in the description under Microscopic Description.

Q. And where are they?

A. Well, I'll read that. It says -- where am I looking at that? And in the -- one down, it says "And three other" -- "there is extensive periglomerular fibrosis around these glomeruli and three other glomeruli show fibrocellular crescents, which in one glomerulus is circumferential."

Q. How many glomeruli are there in the kidney?

A. I would have no idea. Probably millions.

Q. And so out of millions, three had crescents demonstrated on the slide; is that fair?

A. No. That's totally unfair. This is a small --

Q. Why is that?

A. Because this is a tiny biopsy and -- sometimes you'll

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biopsy a kidney and it won't be a good biopsy, you won't see any glomeruli. And sometimes you see two, sometimes you see three, sometimes you see four. And so I would say -- in this particular one, I don't remember how many glomeruli were present, but it wasn't a huge number.

Q. "There are nine glomeruli per level." What does that mean based on your experience as a kidney pathologist or a hematologist reading kidney slides?

A. Well, he's cut a level from the kidney. He's cut a paraffin section. And in that section, he's got nine glomeruli is what he's saying to me; and three of them have crescents. And he doesn't say that all nine are intact so that they can be examined carefully.

Q. If we look down at the interpretation, the final interpretation down at the bottom under the immunofluorescence --

A. Yes.

Q. -- that's a different type of review of the material, is it not?

A. No. He comments there that there are crescents, too.

Q. No. I'm not asking you that. Immunofluorescence is a different type of review of the materials, is it not?

A. Oh, yes. It's a different study, yes.

Q. Okay. And he says, "Out of the six, one shows a small crescent," correct? Shows small crescents. About four

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lines down, I believe.

A. Yes, but that has nothing to do with the immunofluorescence aspect of it. In other words, the immunofluorescence is talking about the fact that he's lighting up gamma globulin in the kidney, and he's just telling us that he's got a -- for this particular slide, he's got six glomeruli per level and he's applying immunofluorescent stains to those six glomeruli.

Q. And out of the six that he has, one shows small crescents, correct?

A. That's what he says here, yes.

Q. Okay. And you haven't reviewed the immunofluorescence photomicrographs, have you?

A. No.

Q. So, you wouldn't know whether that's right or not.

A. I don't know the pertinence to that question, but, no, I don't know that it's right or not. Which is the question you're asking, are there six glomeruli, or what?

Q. I guess the question is: You don't dispute what the pathologist at Loyola is reporting since you haven't reviewed that, correct?

A. That's correct.

Q. Have you reviewed the disclosure filed by Hydrosol -- or H Holdings, I believe it's now known as -- wherein they disclose in a legal document what Dr. Nancy Lynn Jones, the

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medical examiner for the office of the medical examiner of Cook County, is going to say?

A. I don't think I've seen anything like that.

Q. Would it surprise you to learn that Dr. Jones does not make a diagnosis of crescentic glomerulonephritis?

MR. CHILTON: I'm going to object to the form of the question.

A. I would have no -- I don't know Dr. Jones. I don't know her -- is it a she -- her qualifications. I wouldn't have any comment about what she might say.

Q. (BY MR. HARTLEY) Well, if she's a pathologist, would you agree with me that she would have more experience reading kidney slides than you do as a hematologist/oncologist?

A. That is certainly true.

Q. In your review of the liver slides -- and you said you reviewed those with Dr. Schwartz?

A. Yes.

Q. And who is Dr. Schwartz?

A. She's a liver pathologist at Methodist Hospital.

Q. Did you find any cirrhosis in the 2001 biopsy of Mr. Lebron's liver?

A. I wouldn't be qualified to discuss whether he did or didn't have any cirrhosis in his liver biopsy.

Q. Well, did Dr. Schwartz find any?

A. I don't remember. We were discussing the tumor in

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the biopsies. She did look at the original biopsies and commented that they looked like typical hepatitis, as I recall. I don't remember what she -- if she even said anything about the cirrhosis aspect.

Q. Did you look at or determine whether there was any biliary epithelial dysplasia in the 2001 study?

A. I wouldn't personally know how to discuss that if I looked at that myself.

Q. Well, did you talk about that with Dr. Schwartz?

A. What I talked about with her was what was her concept of what that kidney biopsy showed in terms of the malignancy, what were the possibilities of that malignancy.

Q. Did you talk with her about the findings on the 2001 biopsy that there was no evidence of any type of malignancy present in 2001?

A. I don't remember what we discussed on the earlier biopsies. In terms of malignancy, we were talking specifically with the final biopsies.

Q. So, you have no recollection of whether the 2004 liver biopsy demonstrated cirrhosis or not?

A. Well, the 2000 -- the final biopsies is primarily tumor, and so --

Q. 2000 -- the final biopsy's 2005.

A. Okay. Excuse me. The 2004 biopsy, I don't recall anything that I could say sensibly about it. She looked at the

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biopsies and we discussed the tumor.

Q. And there was no comment made on the 2004 biopsy vis-a-vis whether there was cirrhosis present?

A. I don't recall any discussion about that.

Q. Was there any discussion whether there was any biliary epithelial dysplasia present in 2004?

A. I don't recall any discussion about that.

Q. Do you have any appreciation for why biliary epithelial dysplasia would be important in this particular case?

A. No.

Q. Okay. Is it your opinion, Dr. Natelson, that the cancer of Mr. Lebron's liver was a cholangiocarcinoma?

A. I think clinically it was.

Q. I believe that you have suggested that that type of liver cancer is caused by chronic hepatitis B infection. Is that true?

A. It can be.

Q. Are you aware -- bear with me until I find it.

You reference in your paper -- I believe it's Shaib or Shaib.

MR. CHILTON: By "his paper," you mean his report of July 14th, Dean?

MR. HARTLEY: Yes, sir.

MR. CHILTON: Thank you. Do you have the page

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number in front of you?

MR. HARTLEY: I'm trying to find it. I've got it marked here on my group of stuff, but I don't see it. It's on Page 6 (sic), your Reference 20, I believe.

MR. CHILTON: Okay. Thanks.

Q. (BY MR. HARTLEY) And it guess it would be Shaib maybe. Is that how you pronounce it?

A. Probably, yeah. That's a good -- that's as close as anyone. Yes, I have that paper in front of me.

Q. Doctor, do you know as a hematologist/oncologist what biliary epithelial are in the liver?

A. Yes. Biliary ducts drain the liver eventually into the common bile duct and into the intestine. The epithelium would be the inner layer of those ducts.

Q. Did you find any chronic inflammatory disease in the biliary epithelium when you reviewed the pathology?

A. I wouldn't be competent to make that distinction.

Q. In your report, you reference the Shaib epidemiology of cholangiocarcinoma in 2004 from Seminars on Liver Disease, correct?

A. Correct.

Q. Did you also come across Yasser Shaib's 2005 article "Risk Factors of Intrahepatic Cholangiocarcinoma in the U.S."?

A. If I didn't reference it in my letter, I didn't have it.

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Q. Are you aware that other than the 2005 article by Shaib, there are no other studies conducted in the U.S. which evaluate the role of hepatitis C, hepatitis B, diabetes, or chronic liver diseases with intrahepatic cholangiocarcinoma?

A. Well, it seems to me that I referenced another article that was from the United States. Let me just see what I have in here.

Yes, this one by -- and I won't even guess how to pronounce his last name, Perumal. And this was --

Q. What number is that? Is that a different one that you supplied me with last time?

A. Could be. I don't recall, but it's entitled "Hepatitis C and hepatitis B nucleic acids are present in intrahepatic cholangiocarcinomas from the United States."

Q. Okay. Did they -- did they do any type of statistical analysis in that article?

A. No.

Q. Okay. Would it surprise you that when the same author that you cited earlier, Shaib, Shaib -- however you pronounce it -- in studying 625 cases from the United States found no relationship between hepatitis B and the development of intrahepatic cholangiocarcinoma?

A. I haven't read that article, so I can't really comment about it.

Q. Is it your opinion that -- well, let me take it back.

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Do you have an opinion as to the type of cancer Mr. Lebron developed in his liver?

A. He had an adenocarcinoma, which my pathologist in Houston thought was compatible with a cholangiocarcinoma, and Dr. McHugh thought that. And your pathologist in Chicago commented on cholangiocarcinoma, and my interpretation of his report is he thought it was compatible with that but more likely to be metastatic carcinoma. And when I put -- as a clinician, put all of the information about this case together, to me, it's very clear that that was a cholangiocarcinoma.

Q. And as we sit here today, is it your opinion that Mr. Lebron's cancer, the cholangiocarcinoma, was caused by his underlying hepatitis B?

A. Yes. That is a relatively new concept that I discussed with Dr. Schwartz when we were looking at the biopsy. And that's why you see that the newer articles which are coming out which look at this relationship in countries where there are large numbers of cases to study -- hepatitis B is not a common number in the United States these days; so, therefore, you have to study it where the money is -- and those studies now are statistically significant, quite significant, suggest that hepatitis B is a risk factor.

Q. And other than the -- is it P-e-r-u-m-a-l? Is that the article you're trying to reference?

A. That's one that is specific -- you commented -- you

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asked about articles from the United States --

Q. Right.

A. -- so that's one that specifically uses the term "United States" in the title.

Q. Okay. Other than that and the article that I brought to your attention, S-h-a-i-b, from Gastroenterology 2005, are you aware of any other studies that involve only persons from the United States?

A. No. I haven't attempted to research that.

Q. Have you tried to determine whether there's a difference in folks living in the United States and their development of hepatitis B and folks who live in other parts of the country and their development of hepatitis B so as to impact the causal relationship between hepatitis B and cholangiocarcinomas?

MR. CHILTON: Dean, I'm going to object to the question. You said "and patients in other parts of the country." I think you wanted to say "other parts of the world."

MR. HARTLEY: Yeah, I did.

A. Well, it's well-known that there are certain risk factors for many diseases in parts of the world that differ depending on where you are. And we don't have a lot of liver flukes in the United States, it's not common, and some of these other countries have. So, there are certain relationships that

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are prevalent in other parts of the world that are not prevalent in the United States.

Q. (BY MR. HARTLEY) Would you look with me -- do you have the -- which is your reference 21 with you?

MR. CHILTON: 21 from the original report or from the current report?

MR. HARTLEY: The most current report, which is Perumal, something like that, Vivekanandan Perumal. That's the best --

A. Oh, yes.

MR. CHILTON: Oh yeah.

A. I'm looking right at that.

Q. (BY MR. HARTLEY) Look at Table 4 on Page 1214.

A. Yes.

Q. And they have listed for us in Table 4 a survey of studies on hepatitis B and hepatitis C and intrahepatic cholangiocarcinoma, correct?

A. Yes.

Q. And out of the nine entries on Table 4, only the last two are from the U.S., correct?

A. Well, I don't know what you mean by "only." In other words, he has a series of entries from Japan, China, Italy, and the United States. I -- you know, he only has -- he has three from Japan. He has only one from Italy. He has two from the United States. I mean, I don't know why he only has two or if

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there's any significance to the fact that he only has two.

Q. Would it be fair to say that of the nine studies listed on Table 4, there are only two entries for studies done in the United States?

A. Yes.

Q. Would you look up above that table with me in the discussion section on the same page, 1214?

A. Yes. I'm looking in that.

Q. Would you agree that the authors conclude, six lines up, "These results, along with the epidemiology findings of others summarized in Table 4, strongly argue that chronic HBV and chronic HBC are significant risk factors in the United States"?

A. Yes. That's just what I've been saying.

Q. That's the article that you're relying on, correct?

A. Well, I've got several articles here that deal with the fact that hepatitis B is a risk factor for cholangiocarcinoma.

Q. Would you look at Table 4 in the next-to-last entry where they reference a Shaib article from the USA? Do you see that?

A. What page are you on?

Q. 1214. Still on 1214.

A. 1214. And where are we on that page?

Q. The table at the bottom.

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A. Yes.

Q. The next-to-last entry in that table referencing the study from the U.S. by Shaib.

A. Yes. Now I'm with you.

Q. Okay. And the result of that indicates only that hepatitis C infection is associated with intrahepatic cholangiocarcinoma, correct?

A. Well, it only discusses hepatitis C from that article. It doesn't say anything about what he thought about hepatitis B.

Q. You haven't reviewed the article, have you?

A. I've read this article and I see the results on the table.

Q. Okay. But you haven't reviewed the underlying data for that table for that particular entry.

A. Oh, now I'm with you. No.

Q. That was the article I was asking about earlier, if you had seen that article.

A. No.

Q. You also reference in your July 14th report on Page 3 that monoclonal gammopathy, with both large and small paraprotein peaks, has been commonly cited as a consequence of or an association with hepatitis as a class of illness and, specifically, with hepatitis B and C and you cite Reference 5 and 6. Do you see that?

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A. Yes.

Q. Would you pull out Reference 5, which is an abstract by Blanchard?

A. Yes.

Q. And Blanchard, in the background of the abstract, indicates that chronic inflammation or infection may predispose to development of plasma cell dyscrasias, correct?

A. Yes.

Q. And down at the bottom -- would you agree with me that although this is a 2004 abstract, at least according to your note down at the bottom -- I assume that's your writing --

A. Yes.

Q. -- this study provides a basis for future research efforts focussed on identifying cytokine pathways that may link these disorders and subsequently serve as targets for novel interventions to prevent the development of progression of plasma cell dyscrasia, correct?

A. Yes.

Q. So, this abstract and this study of veterans -- isn't that what it was? Yeah, veterans -- basically provides a basis for future research and may provide a link for the disorders and novel interventions in the future, correct?

A. This article provides the fact that veterans with hepatitis B and C have a high incidence of monoclonal gammopathy.

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Q. And the theory is that chronic inflammation or infection may predispose someone to the development of plasma cell dyscrasia; is that right?

A. Well, that's a very, very old theory and I don't know that it's true, but it is certainly true that people with hepatitis B and C get monoclonal gammopathies. Whether that makes them go further into multiple myeloma because of the fact that they were originally caused by hepatitis B or C is not known.

Q. You also cite the Brown study from 2008 -- I think this must be the other U.S. study you were thinking of -- which is your Reference 6, correct?

MR. CHILTON: He's looking for it.

A. I'm trying to find it right at the moment.

Yes.

Q. (BY MR. HARTLEY) Look at Table 3 on Page 3390 for me.

A. Yes.

Q. And it says "Infectious disorders and risk of myeloma in white and black U.S. male veterans."

A. Yes.

Q. Where would Mr. Lebron fall on this particular table?

A. He's got hepatitis.

Q. Is he white or black?

A. Well, he's Puerto Rican by background. That depends

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on how you want to classify that.

Q. That's why I'm asking you.

A. I would classify him as Puerto Rican.

Q. So, he falls in neither the white nor the black category, correct?

A. I can't tell you what he would put on his driver's license. I suspect he would have put white, but I don't know that.

Q. In the abstract of the report, Doctor, the authors, in the middle column, indicate about -- I don't know -- four lines down, "Significantly elevated risks of myeloma were associated with broad categories of autoimmune" -- then they give you the relative risk -- "and infectious diseases," I assume, and they give you the relative risk of 1.29.

Do you have an opinion as to whether a relative risk of 1.29 provides a basis for saying that infections are associated with myeloma?

A. I would say it wouldn't confirm it. You'd like to see a doubling of the risk.

Q. Would you rely on that .129 (sic) in making your determination?

A. Well, I didn't determine he had multiple myeloma.

Q. But if we assume that he did.

CHILTON: I'll object --

A. Really, I would say that that would be a negative --

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I wouldn't conclude that could confirm the presence of myeloma, that particular number.

Q. (BY MR. HARTLEY) Is it your opinion -- it is your opinion, isn't it, that he does not have myeloma?

A. That's correct.

Q. But if he does have myeloma, it's probably related to his hepatitis B. Is that your opinion?

A. No. That would be false.

Q. Okay. Did you make a determination as to whether your diagnosis of crescentic glomerulonephritis was of the acute nature?

A. I'm not sure what you mean by that. In other words, clinically, he'd been sick for a considerable period of time before he had that biopsy done and he was already developing signs of renal insufficiency. So, he's got a chronic illness.

Q. Well, look on Page 3 of your report. And under Crescentic Glomerulonephritis, you have in bold the quote from Article No. 7, or Reference No. 7, about the accumulation of cells in Bowman's space, etcetera. At the end of that quote, you say, "Thus, in acute severe forms of glomerulonephritis such as that mediated by autoantibodies, crescent formation is prominent."

Are you saying that he has an acute severe form of glomerulonephritis? "He" being Mr. Lebron.

A. No. He has a chronic process, but the crescents

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don't go away. In other words, when an illness starts out, it's acute. Crescents form, they continue to form, and then eventually they burn out the kidney. So, an acute glomerulonephritis becomes chronic. The crescents may be there in the acute phase, they're still there in the chronic phase.

Q. Is there any type of lab report or lab test that we can do to determine whether, in fact, he does have chronic glomerulonephritis or crescentic -- I'm sorry -- crescentic glomerulonephritis?

A. No. That's just a -- the crescentic aspect of it is simply a histopathological indication of inflammation. There is no biochemical test that one can do on his serum to point to that finding.

Q. Well, look with me at your Reference No. 7, which is Mathieson, editorial review.

A. Yes.

Q. You remember that?

A. Yes. I've got it right here.

Q. Look at the first column -- left-hand column in the first full paragraph where it talks about, toward the end of that, "Thus, in acute severe forms of GN, such as that mediated by autoantibodies to the Type IV collagen in glomerular basement membrane, GBM -- Goodpasture's disease or anti-GBM disease -- crescent formation is prominent, often affecting all glomeruli seen in a renal biopsy."

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Do you see that?

A. Yes.

Q. That's the full quote from the paper that you've quoted in your report, is it not?

A. Yes.

Q. Do you have an opinion as to whether Mr. Lebron had a severe form of GN?

A. Well, he certainly didn't have Goodpasture's disease -- that's a very specific illness -- and he had severe enough glomerulonephritis to lead him to renal failure. So, obviously, I think by definition that would be severe.

Q. Okay. So, if he had severe -- if he had severe kidney disease -- according to the editorial, crescent formation is prominent, often affecting all glomeruli seen. Would you agree with me that in the pathology report all the glomeruli did not have crescents in them, did they?

A. Well, you're asking two different things. Yes, I've already agreed that all of the glomeruli didn't have crescents. And I've also said that he didn't have Goodpasture's disease, which is a particular type of glomerulonephritis that the author is citing. And I think that it would be -- severity is just a judgement in terms of an index of pathology, but obviously his disease was severe enough to cause the kidneys to fail. I would call that severe.

Q. Are you saying that based on your understanding of

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kidney pathology, he has severe kidney pathology, but he doesn't have severe enough kidney pathology so that all the glomeruli have crescents in them?

A. In this particular biopsy that we saw, which is a tiny biopsy relative to both kidneys, he has several glomeruli with crescents in them, enough so that all of the observers have commented on that finding. He didn't have them in a hundred percent of them. As to the significance of the fact that he had them in three out of nine, you would be better off questioning Dr. McHugh about the significance of that fact.

Q. Do you have an understanding for -- of what PCNA is?

A. PCNA? You'll have to tell me what those initials stand for and where we're looking at them.

Q. We're on the next page of the Mathieson article, 156.

A. I see it. He calls it proliferating cell nuclear antigen. And what was your --

Q. Do you know what that is?

A. I have no idea.

Q. Do you have with you the La Grange Memorial Hospital report?

A. Is that the one we talked about of the kidney biopsy in Dr. Meehan's deposition?

Q. No. This one's done at La Grange, it looks like, March 30th, 2000.

A. And what's it a biopsy of?

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Q. It is a study of antiglomerular basement membrane.
A. If it's -- is it something that I referenced?
Q. No.
A. Well, if it's not something that I referenced, I obviously don't have it here.
Q. Okay. Do you have the medical records with you?
A. No.
Q. Okay. In trying to determine whether Mr. Lebron had crescentic glomerulonephritis, did you look at the lab results from the various hospitalizations?
A. I'm sure I did look at his lab results.
Q. Are you aware of any lab result which may or may not confirm or rule out the existence of glomerulonephritis, crescentic glomerulonephritis?
A. Well, as we said, I don't believe there's any single lab test that rules it in or rules it out, and so I'm not clear --
Q. Are you aware of any lab test that makes it less likely? As --
A. I'm not sure what you're referring to.
Q. Well, as a hematologist/oncologist testifying as someone who's familiar with kidney diseases, are you familiar with any particular lab result of the kidney which makes a diagnosis of crescentic glomerulonephritis less likely?
A. Are we speaking of a blood test on serum or something

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on tissue?

Q. Well, you're the person who's being offered as the expert. Can you think of any?

MR. CHILTON: You're -- I'm going to object. You're asking the question. He's asking you to clarify your question. He's not here to guess or speculate as to what you're asking him. You need to ask a fair and clear question.

MR. HARTLEY: I understand that he's not an expert in kidney diseases.

MR. CHILTON: That's not what I said.

MR. HARTLEY: I understand he's not an expert in kidney pathology, but he's being offered as an expert in kidney diseases and in reading pathology slides of the kidney in this particular case. I am simply asking him is he aware of any laboratory study as a hematologist/oncologist testifying in the area of kidney pathology and kidney diseases which makes a diagnosis of crescentic glomerulonephritis less likely than not? If he doesn't know of any, that's fine, we'll move on.

A. Let's move on. I don't really know what you're talking about lab-wise. This is simply a descriptive term. There is no blood test that proves whether someone does or does not have crescents in their kidney biopsy.

Q. (BY MR. HARTLEY) What is complement C3?

A. Well, complement are a series of proteins that have to do with allergic reactions. They're proteins that are

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normally in the blood.

Q. What significance do they have vis-a-vis crescentic glomerulonephritis, if anything?

A. Well, in forms of glomerulonephritis and many other illnesses, complement becomes so-called activated and can be deposited into tissues or cause inflammation.

Q. What does complement C3 particularly look for?

A. I'm not a complement expert.

Q. How about p-ANCA? Do you have any idea what that is?

A. I think you're talking about p-ANCA, and the --

Q. p-ANCA?

A. Yeah. I think that's p-ANCA. That's p-ANCA. That's a -- sort of a test that's positive in certain types of inflammatory kidney disease, such as Wegener's granulomatosis.

Q. And if it's negative, what does that indicate?

A. Well, it's not a test that we use for a 100 percent diagnosis of anything. In other words, it's frequently positive in a rare illness called Wegener's granulomatosis. It may be negative in that illness. It's a test of, you might say, inflammation.

Q. Would that test need to be positive for one to -- if one had crescentic glomerulonephritis?

A. Would not have to be positive.

Q. Okay. So, if the lab that ran this, SmithKline Beecham or Quest -- I'm not sure which one did it -- indicates

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that the p-ANCA, if it's present, is seen with crescentic glomerulonephritis, that's not something that we'd be concerned with?

A. It's not a test that proves a diagnosis.

Q. It proves inflammation, doesn't it?

A. Well, it suggests inflammation; but in severe diseases characterized by inflammation, it may be negative.

Q. And would you agree with me that crescentic glomerulonephritis is an inflammatory disease process?

A. Yes.

Q. Okay. What is Bence-Jones proteinuria? I might have butchered "proteinuria."

A. Bence-Jones was a man who originally described a multiple myeloma, or that is to say some of the protein findings in the urine; and it's a protein that comes out in the urine and can be redissolved if you heat up the urine and then it comes back out if you cool down the urine. And --

Q. Why -- I'm sorry.

A. And we generally think of it as synonymous as what we call light chain protein in the urine.

Q. And if you find Bence-Jones protein in the urine, is that consistent with a diagnosis of myeloma?

A. It could be.

Q. Would you agree that it is a -- suggestive of monoclonal gammopathy of undetermined clinical significance?

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A. Well, Bence-Jones protein, number one, has to do with urine, and most people we find with MGUS -- or monoclonal gammopathy of unknown significance -- we're talking about serum. So, urine is a different situation than serum. One can have monoclonal gammopathies in the urine and one can have Bence-Jones protein in the urine. And when one finds Bence-Jones protein in the urine, that can be indicative of multiple myeloma.

Q. What other diseases would it be consistent with?

A. Well, one can find monoclonal proteins in the urine of people who -- and it may carry the same significance as monoclonal proteins in the serum; that is to say, one can have monoclonal proteins in the urine of unknown significance.

Q. Are you telling me that Bence-Jones proteins are monoclonal proteins of unknown origin?

A. Well, they can be.

Q. Are you telling me that Bence-Jones proteins are simply -- are indicative of -- strike that.

Other than myeloma, can you think of any other diseases that Bence-Jones protein would be consistent with?

A. Well, I'd have to look that up, but I -- Bence-Jones proteins, it's an old term for proteins that have heat properties; that is to say, you can bring them in or out of the solution of the urine, whether they're heated or cooled. And it's sort of an obsolete term. It's been replaced by

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specifically measuring light chains or monoclonal proteins in the urine.

Q. So, to answer my question, you'd have to look up in some textbook or something what type of disease entities demonstrate with Bence-Jones protein?

A. Bence-Jones proteins are seen in myeloma. I would have to look in the book to see what other obscure illnesses they might be seen in. And there may well be others as we sit here. I don't know that.

Q. Well, how about Fanconi's syndrome?

A. Well, Fanconi's syndrome is a tubular disease of the kidney that can be caused by certain drugs damage the tubules and you get so-called Fanconi's syndrome where you can lose glucose and protein in the urine. And it's possible that they would have Bence-Jones proteins. I don't know that.

Q. Let's assume that they do. Did Mr. Lebron have Fanconi's syndrome?

A. No.

Q. How about a lymphoproliferative disorder? What would that include?

A. Well, lymphoproliferative disorders could be things like chronic lymphocytic leukemia or small cell lymphoma. They also are associated with monoclonal gammopathies and they also may cause glomerulonephritis.

Q. Would you see Bence-Jones proteins in those types of

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entities?

A. I don't know the answer to that. You certainly see proteinuria and you see monoclonal proteins in the serum. I don't know the answer to that question.

Q. Let's assume that you do. Does Mr. Lebron have either of those entities, CLL or the other lymphoma that you described?

A. No.

Q. And I'll butcher this. What is a-m-y-l-o-i-d-o-s-i-s?

A. Amyloidosis is a condition -- and there are several types of amyloidosis -- and it's a deposition in the tissue of a waxy protein. And in some instances, it's associated with multiple myeloma. In some instances, it's not.

Q. Do you see Bence-Jones proteinuria in -- whatever it was?

A. Amyloidosis, yes, you may.

Q. Okay. Did Mr. Lebron have amyloidosis?

A. No. They stained his kidneys for amyloid and he didn't have that.

Q. Okay. Oh, let's look at Ronco. I think that's, like, 16 maybe.

A. Yes.

Q. That's not 16. Where is it?

MR. CHILTON: No. 9.

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Q. (BY MR. HARTLEY) No. 9.

Now, is it your opinion that Mr. Lebron does not have MIDD?

A. That is correct.

Q. Okay. Would you pull out Dr. Meehan's deposition again --

A. Yes.

Q. -- so that you can see the description of the kidney?

A. I assume you're speaking of the original one that's this Page 2 of 3?

Q. Yeah. I have to find mine. I have too many papers on the table here. Yep, that will do. And let's look at the -- under Pathogenesis.

A. Pathogenesis, yep.

Q. You with me? I'm sorry. We're referring to two different things now. Ronco, 2006 --

A. Yes.

Q. -- on the first page, under Pathogenesis --

A. Yes.

Q. -- "MIDD is characterized by kidney deposition of monoclonal Ig subunits." Do we have that in this case?

A. Well, you have the whole molecule being deposited and the molecule is consisting of two light chains and two heavy chains.

Q. So, does that mean we do or we don't have deposition

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of monoclonal Ig subunits?

A. Well, it depends how you're using that term. In other words, I think these people use this term to mean it can be light chain disease where only the light chain is deposited; it can be heavy chain disease where only the heavy chain disease is deposited; and then it can be light and heavy chain disease, which is both chains being deposited. And that whole complex is referred to as monoclonal immunoglobulin deposition disease; in other words, the three types.

Q. Are you an expert on MIDD?

A. No.

Q. Have you ever testified in any case, Doctor, where MIDD was involved?

A. No.

Q. I came across something on the internet, I think -- or maybe you produced it. It is a PowerPoint presentation. Do you remember doing that?

A. A PowerPoint presentation -- well, I did in terms of the photographs.

Q. The photographs of what?

A. Bone marrow and the kidney biopsy, I think.

Q. Okay. That was what that PowerPoint presentation was?

A. Yes.

Q. Think it's called "Monoclonal Gammopathy Associated

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With Liver Disease and With Nephrotic Syndrome."

A. Yes.

Q. And that was prepared for this case?

A. Yes.

Q. Okay. I wondered what that was. I couldn't quite figure out what that was.

But getting back to the Ronco paper, it says "MIDD is characterized by kidney deposition of monoclonal Ig subunits, but at variance with amyloidosis, deposition induces a dramatic accumulation of extracellular matrix that is responsible for glomerular and tubular basement membrane thickening, nodular glomerulosclerosis, and interstitial fibrosis."

Do you see that?

A. Yes.

Q. Look at the pathology report, particularly the immunofluorescent study at the bottom of Page 2 of that report.

A. Okay. I'm looking under Interpretation.

Q. Yes, sir. And it begins "Frozen sections were incubated with the following antibodies," then she gives those to you. Do you see that?

A. Yes.

Q. "There's a diffuse and" -- is it "punctate" --

A. Yes.

Q. -- "stain for IgG and kappa light chain along the

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glomerular capillary wall mesangium, as well as tubal basement membrane and focally in the extraglomerular vessels."

Do you see that?

A. Yes.

Q. Isn't that the exact same description that Ronco is giving us in his 2005 diagnosis that we just read together?

A. Well, all this is saying is that there's immunoglobulin stuck in the kidney, and that's not specific for any single entity.

Q. Okay. So, Ronco really didn't need to give us this long description. They could have just said, "There's immunoglobulin stuck in the kidney," and that would have been okay? That's your understanding?

A. No. Ronco gives a very specific discussion of what this entity is. And what the difference between this entity or one of the differences between this entity and the -- and what Mr. Lebron has is this entity is a glomerular path -- a glomerulopathy. In other words, this is sort of a bland destruction of the kidney. The crescentic changes are inflammatory destruction of the kidney.

And in this article, he specifically says in here, "exudative lesions as fibrin caps," which would be the crescents, "and extensive hyalinosis of the efferent arterioles are not observed." So, he's telling us this is bland pathology damage as opposed to inflammatory damage, and he shows us a

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nice picture to confirm that.

Q. Okay. That's your understanding of what he's trying to tell us?

A. That's my understanding of what he's telling us in this article.

Q. You've read Dr. Meehan's deposition, have you not?

A. Yes, sir.

Q. And you have put forth in your July 14th report your comments concerning that report and his lack of understanding of the situation, correct?

A. Well, I've commented about certain things he had in his deposition, yes.

Q. Go to Page 2 of your report.

Do you want to take a break, Doctor, or are you okay?

A. No. I'm okay.

Q. Okay. Under bone marrow findings, you have "flow cytometry." Do you see that about -- I don't know -- seven, eight lines down, "Flow cytometry to bone marrow cells can also compute percentages of various cell types"?

A. I know I said that in here, but I'm not seeing -- on Page 2 is that?

Q. Yes, Page 2, under Bone Marrow Findings.

A. Oh, yes. Yes, I see that, uh-huh.

Q. Was flow cytometry done in this case?

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A. I don't recall.

Q. Tell me what CD138 is.

A. Well, CD stands for cluster designation, and there are many CD markers on blood cells. And lymphocytes, for example, often have CD20 on them and CD5. This is a particular CD marker that might be on plasma cells.

Q. And if -- you use the CD138 as an antibody. Would that be fair?

A. Well, what happens is that these cells have the CD38 or whatever marker you're using on them and you apply an antibody that's got a fluorescent dye with it and it hooks up with that antigen. The CD marker is the antigen and your fluorescent marker is tied to the antibody, and so what you see is sort of a brownish reaction in the tissue that is called immunoperoxidase reactions and you can tell if a cell is positive for the particular marker you have an interest in.

Q. What impact does that have on determining plasma cells?

A. Well, it's a qualitative assay in this sense because you -- it's hard from a biopsy to pick out individual cells. And what we normally use these things for -- you can use them on individual cells, but typically what will happen is we may have a lymphoma and you're staining the slide and they see if it's positive in a general way for CD5, CD20, CD23, and there are certain patterns of positivity that tell you this is

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chronic lymphocytic leukemia, this is hairy cell leukemia, this is mantle cell lymphoma and so on. There are a series of -- and so most of the time these days on pathology, they're running a series of markers.

And there are markers for plasma cells, but typically we don't use those for quantitation. As I said in my letter, we simply look at the aspirate visually and count the plasma cells.

Q. Well, if the particular sample is stained with antibodies looking for CD138 and you find that it lights up with the antibody that you're -- the sample you're testing, what does that indicate to you?

A. Well, it could indicate it's a plasma cell.

Q. Does it indicate that it's consistent with myeloma?

A. Not necessarily. In other words, any plasma cell would be positive.

Q. So, a -- what other disease process would light up CD138 other than myeloma?

A. Well, if it's a plasma cell marker, anything that gives you increases in plasma cells, that marker would be positive.

Q. Well, let's talk about that then. What gives you increases in plasma cells?

A. Well, any chronic inflammatory state. For example, all forms of hepatitis would have increased plasma cells in the

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bone marrow typically, forms of rheumatoid arthritis, forms of lupus, most skin diseases that are very chronic you might see inflammatory plasma cells in the bone marrow, chronic infections, tuberculosis.

You know, as I cited one article, about almost a third of all the bone marrows they looked at had a slight increase in plasma cells if they're done on sick people.

Q. Are you telling me that there are -- there is literature out there that says that if you use -- you can use CD138 to determine whether hepatitis is creating an increase in plasma cells?

A. I have no idea. I --

Q. What do you use CD138 to demonstrate?

A. I don't use it at all. It's a marker that pathologists sometimes use.

Q. You saw that Dr. Durie says that CD138 demonstrates that the myeloma is producing the increase in plasma cells. Do you disagree with that?

A. I would disagree with that.

Q. Why?

A. Because that's -- you can't tell that kind of causation with a marker for plasma cells. A marker is a marker, and you can't tell the fingerprints of a plasma cell if you just apply a standard plasma cell marker to it.

Q. Are you aware of literature that suggests that CD138

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should be used or can be used in determining whether an individual has myeloma or not?

A. No.

Q. Are you familiar with the Nig article, N-i-g? Or N-g. I'm sorry. Guess it's "Nig."

A. N-g is usually Nguyen.

Q. Nguyen?

A. Yeah. N -- whether -- if you're Vietnamese, whether your name is N-u-y-e-n or N-g, it's Nguyen.

Q. Okay.

A. Makes it real easy.

Q. That's pretty simple. Are you familiar with Nguyen's article in -- is it Hematologic --

A. If it's an article that I have here on the table that I've referenced, I could look at it. If it's some article you found in the literature, I wouldn't necessarily be familiar with it.

Q. Okay. Look at the third page then of the exhibit attached to Dr. Meehan's deposition, three of three of the pathology report.

A. Three of three?

Q. Under Interpretation of Special Stain, the last big entry.

A. That's interesting. I don't see a three. Interpretation with special stain?

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Q. Do you see that? Is it on there?

A. I don't -- not sure what you're looking at. The interesting thing about this is there are markers at the end of this deposition, and it goes 1, 2, 4, 5, 6, 7, 8, 9, 10.

Q. Okay. Maybe it's not on that exhibit. I'm looking at a different page of my book of -- it's not on there. See if it's -- no, it's not on that one.

Let's approach it from this way: You individually reviewed the aspirate, as well as the bone marrow, correct?

A. The aspirate and the biopsy material that I was provided, yes.

Q. And you looked at the aspirate and you counted plasma cells, did you not?

A. Correct.

Q. And you always got a percentage of plasma cells less than 5 percent, sometimes as low as 3 percent, correct?

A. Yes, sir.

Q. When one uses CD138 staining, will that help you see the plasma cells?

A. Well, yes and no. What happens is that when you look at those immunofluorescent stains, it really damages the cells, and so you see a lot of pigment and you're always hard-pressed sometimes to see what cell that pigment is in. And so I have never tried to use an immunofluorescent marker to count plasma

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cells. That's typically not how it's done in the standard literature.

Q. Are you saying that that is an improper way to count plasma cells?

A. What I'm saying is that the standard way to determine percentage of plasma cells in the bone marrow is by counting by light microscopy by a right stain the numbers of plasma cells in a bone marrow aspirate. That's what hematologists do and have done for generations.

Q. Are you aware of the literature which suggests staining with CD138 increases the sensitivity of the plasma cell so that you can count them better?

A. Well, when you start looking at markers, for example, you --

Q. Answer my question first, then you can explain.

A. Well, why don't you repeat your question because --

Q. Sure. Are you aware of literature which suggests that staining with CD138 increases the sensitivity to view the plasma cells so one can count them?

A. No.

Q. Okay. What does it mean if the plasma cells are clustered?

A. Not much.

Q. Okay. Is that consistent with myeloma?

A. Well, if you're talking about very, very large

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clusters or so-called plasmocytomas, that might be suggestive of myeloma; but plasma cells often tend to cluster, even in what we call reactive bone marrows where there's no myeloma present.

Q. What would be the cause of clustering in reactive myeloma -- reactive bone marrow?

A. Well, when cells are reproducing, they often are clustering together; in other words, you can see clusters of red blood cell precursors, clusters of white blood cell precursors, clusters of plasma cells. And when you have conditions in which there are increased plasma cells, sometimes you'll see small clusters of them.

Q. When you said earlier "large clusters" or "small clusters," how do you differentiate by number?

A. I don't know that I would put a number on that, but you're talking about -- generally, when I say "small clusters," I'd say three, four, five cells stuck together; large clusters, maybe a thousand.

Q. Okay.

(Sotto voce discussion.)

MR. CHILTON: Dean, if this is a good time for a break, why don't we take one, unless you're relatively close to being finished.

MR. HARTLEY: Well, I think I'm -- I think I can be done by 4:00 if you -- but if you want to take a break,

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that's fine.

MR. CHILTON: Well, yeah, let's definitely take a break now then.

MR. HARTLEY: 4:00 my time, 3:00 your time.

MR. CHILTON: Oh, so you're saying 20 --

MR. HARTLEY: 25, 30 minutes.

MR. CHILTON: Okay.

MR. HARTLEY: You want to take a break? I don't care.

(Discussion off the record at 2:35 p.m.)

MR. CHILTON: You know what, Dean? Let's take just five minutes so people can use the restrooms and you can take a look at your stuff and we'll start right back in.

MR. HARTLEY: Okay.

(Recess from 2:35 p.m. to 2:39 p.m.)

Q. (BY MR. HARTLEY) Doctor, are you aware of whether any anti-GBM antibodies were present?

A. I don't know that as we speak.

Q. Are you aware based on your review of the kidney pathology materials whether there was any systemic vasculitis?

A. No. There was no evidence of systemic vasculitis.

Q. Would you agree with me that one mechanism for renal failure if the patient has myeloma is the result of interstitial fibrosis?

A. Probably so. There are many, many reasons why a

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person with multiple myeloma can get kidney failure, and scarring of the kidney and fibrosis would be a possibility.

Q. In your review of the pathology material, did you determine whether the basement membrane in the renal glomerulose was impacted?

A. Well, obviously, the glomeruli were impacted because they were swollen and really, to a certain extent, obliterated by crescent formation, and so certainly the glomeruli were involved.

Q. Well, that's not the same thing. Do you not distinguish between the basement membrane and the glomeruli?

A. Well, the glomeruli are the whole structure. They're a series of specialized capillaries and they have various layers of membranes. And, you know, the significance of each particular layer is something that a pathologist could discuss better than I.

Q. So, that's outside your realm of testimony?

A. Yes.

Q. Pull out Dr. Meehan's deposition, if you would.

A. Yes.

Q. And look with me on Page 41.

A. Yes.

Q. Look at Line 18. Would you read that for me?

A. Line 18 is "The presence of paraprotein IgG, kappa paraprotein, and the serum in urine and the deposit in the

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kidney is evidence of clonal plasma cell proliferation."

Q. Do you agree with that?

A. Yes.

Q. Do you agree that Mr. Lebron was anemic when he presented in March 13th of 2000?

A. Let me look back at my original report. Actually, I don't remember that, and --

Q. Well, let me ask you this: If his red blood count is decreased at 4.3, his hemoglobin is decreased at 13.4, and his hematocrit is decreased at 38.7, is he anemic, at least on March 13th, 2000?

A. Well, I have that figure of 13.4 from December 1999 in my report; and 13.4 for a man, 39 hematocrit, that would be considered very slightly anemic.

Q. Would you agree that Mr. Lebron had mutant immunoglobulin present in his urine?

A. I suppose -- well, he had -- I don't know about that term. That's a very peculiar term to use. He had a monoclonal -- or a clonal immunoglobulin in his urine. And whether it was a mutant or just multiple copies of a normal gamma globulin, I don't know. I don't know -- there were no genetic studies to demonstrate if it was a mutant or not a mutant, and I think the use of that word would be very peculiar.

Q. How about -- did he have as well the immunoglobulin

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present in his serum?

A. He had an immunoglobulin -- monoclonal immunoglobulin in the serum and also in the urine.

Q. And would you agree that that same genotype that was found in the urine and the serum was found in the kidney tissue as well?

A. Yes.

Q. Would you agree that the source of that immunoglobulin is a proliferation of plasma cells in bone marrow?

A. Yes. It's generally believed plasma cells would be the general producer of gamma globulin.

Q. What types of crescentic -- how about that, Larry?

MR. CHILTON: Nicely done.

Q. (BY MR. HARTLEY) -- glomerulonephritis have been identified in association with chronic hepatitis B?

A. I'm not certain about the question, "what types." In other words, it can be -- "membranous" or "membranoproliferative glomerulonephritis" are general descriptive terms. "Crescentic" simply means that it's a very inflammatory process.

Q. Well, are there any features of membranous -- is it membranous --

A. Membranous.

Q. -- membranous crescentic glomerulonephritis in the

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kidney biopsy?

A. Well, I think I would defer to Dr. McHugh in talking about membranous versus membranoproliferative. They both are seen in inflammatory kidney diseases. Membranous, for example, is more frequent in diseases like lupus. Hepatitis B can be either membranous or membranoproliferative.

Q. Well, can you define for me hepatitis B associated membranoproliferative glomerulonephritis?

A. It's an inflammatory condition of the glomerular tissue in the kidney caused by the presence of hepatitis B and it's a deposition of gamma globulins in the glomerular tissue causing damage. And it can be in the form of what we call immune complexes where the gamma globulin is sticking to a hepatitis antigen and then that complex of antibody and antigen is sticking in the kidney, or it could be simply deposition of the antibody of the gamma globulin in the kidney.

Q. What specific immune complex would you see being deposed in the kidney?

A. You -- the hepatitis B has certain antigens, and they're given letter-numbers. I'm not totally conversant with that, but some of these articles describe that. And he had those studies done on his liver biopsy where they demonstrated hepatitis B antigens in his liver biopsy. Those antigens, same antigens, can be in the kidney, complexed with the gamma globulin.

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Q. Do you know if they were?

A. They were never measured in the kidney; the antigens, that is.

Q. What antigens specifically are you referencing?

A. Hepatitis B antigens, proteins produced by the hepatitis B virus.

Q. What about the immune complex deposition?

A. That's it. In other words, "immune complex" simply means gamma globulin attached to an antigen or an antigenic protein. It's a complex of antibody and antigen, and that complex can float into the kidney and damage it; sometimes referred to as immune complex disease.

Q. But you don't know what those immune complex disease antigens would be as we sit here right now?

A. Well, in Mr. Lebron's case, they would be a complex of his gamma globulin with a hepatitis antigen.

Q. Okay. And what particular gamma globulin would we be looking for in the kidney?

A. We'd be looking for the gamma globulin that the hepatitis excited production of.

Q. Well, what would that be?

A. That could be what he had. In other words, when you have an antigen in your system, it stimulates production of gamma globulin. That gamma globulin can be monoclonal or polyclonal, and he had a monoclonal gamma globulin. And --

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Q. What did he have in his kidney?

A. He had gamma globulin stuck in his kidney.

Q. Okay. And what particular -- what else -- is that consistent with a membranoproliferative glomerulonephritis?

A. Yes.

Q. It is?

A. Yes.

Q. Okay. What would you expect the -- let me ask you this: What would -- what -- how would -- what -- does it have a particular letter for it? When you're talking about globulins being in the kidney, is there anything more specific than that?

A. Well, yes. He had an I -- gamma globulins, they're referred to as IgG, IgA, IgM, as general classes of gamma globulins. And people with hepatitis B can have what's called IgA nephropathy with IgA gamma globulins sticking to the kidney or they can have IgG globulins stuck in the kidney. In his case, he had IgG globulins stuck in the kidney.

Q. And is that enough by itself to assert that the immune complex deposition of IgG supports the diagnosis of the -- is it membrano?

A. Membranoproliferative? I think the histology supports the diagnosis of membranoproliferative. The immune complex concept is suspected because he wasn't -- his kidney was not stained for the hepatitis antigens.

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Q. Was there any information on whether the hepatitis B antigen was -- hepatitis B antigens serology was positive or not?

A. Well, his hepatitis B --

Q. For the kidney now.

A. We're talking in the kidney. He did not have stains looking for antigens of hepatitis in the kidney tissue.

Q. How would -- if we were looking at this membranoproliferative glomerulonephritis, what are we looking for and what description are we looking for of the slides themselves?

A. We're looking for swelling and inflammation of the glomeruli and -- as they described. In other words, I -- let's go back to look at their description here.

Q. Okay. You're going to review Exhibit 1?

A. Yes.

Q. The attachment to Exhibit 1?

A. Yes.

Q. Okay.

MR. CHILTON: From the Meehan deposition.

MR. HARTLEY: Yes, yes.

MR. CHILTON: Okay.

A. It's the -- the sentence there is "Extensive periglobular glomerular fibrosis around three glomeruli, and three other glomeruli show fibrocellular crescents, which in

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one glomerulus is circumferential. There are several foci with tubular separation and lymphocytoplasmic infiltrate in the interstitium." Let's see what else they go on to say.

"Electron microscopic examination demonstrates abundant punctate electron-dense deposits in the mesangial matrix and in the glomerular capillary wall. Those deposits are seen immediately under the endothelium."

These all -- generally, they're talking those deposits are by the electron microscopy. And those deposits, when they're granular, are representing the gamma globulin.

Q. (BY MR. HARTLEY) Okay.

A. And so these -- this report tells me that he's got an inflammatory reaction in the kidney which is consistent with glomerulonephritis.

Q. And can you give me a reference that I might go and review to see if this type of pathology that's described corresponds with the disease process that you have suggested?

A. Well, I -- there are many references about glomerulonephritis and hepatitis B, and --

Q. I'm more concerned with description of the process via pathology.

A. Well, I -- one of the articles I referenced is by DiMarco, and he says here "Renal biopsy showed that" -- excuse me. This is "Therapy with lamivudine and steroids in a patient with acute hepatitis B and rapidly progressive

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glomerulonephritis."

Q. What exhibit number is -- what reference number is that?

A. That is -- let's see.

MR. CHILTON: It's No. 14, I believe, Dean.

Q. (BY MR. HARTLEY) "Therapy with" --

A. Yes. That's Epivir -- therapy with lamivudine, which is also called Epivir, which is the drug that he received, that Mr. Lebron received. And in that table, it says "Renal biopsy showed mesangioproliferative glomerulonephritis with crescents." Hepatitis B antigen and hepatitis -- and deposition and glomeruli were shown by immunofluorescence by monoclonal antibodies.

Q. Okay. I understand that. I'm looking more for a definition or an item that we might look at to tell us renal biopsy showed mesangio --

(Discussion off the record at 2:57 p.m.)

Q. (BY MR. HARTLEY) What is it, mesangioproliferative?

A. Yeah, mesangioproliferative.

Q. Mesangioproliferative. Where do I find a definition of what the pathology slide should look like for that? That's what I'm concerned -- that's what I want to know.

A. Well --

Q. A pathology textbook, I guess, would do it, right, for the kidney?

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A. You can do that. For example, another article that I referenced by Chuang, C-h-u-a-n-g -- I'm not sure what number that is.

Q. 15 maybe, "Complete remission"?

A. Yeah. He says -- well, let's see. "We used electron microscopy and found viral-like particles appearing in clusters around the ovoid structures within the glomerular capillary basement membrane."

Q. Where are you on that?

A. I'm on Page 872. It actually starts on the prior page. Let's see if I can see where that is.

"The diagnosis of hepatitis B virus associated membranous nephritis is usually established by serologic evidence of persistent HBV infection, the presence of immune complex deposits on kidney biopsy, and the demonstration of glomerular deposits containing one or more HBV-related antigens."

Q. Do we have any of that?

A. Well, you've got the inflammatory reaction of the glomerulonephritis. You've got the antibodies. They weren't stained by the antigen. They weren't stained for the antigen.

Q. Do we have the antibodies in the kidney?

A. You have antibodies in the kidney.

Q. That are HBV?

A. Antibodies, I would assume, against HBV produced by

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the presence of the HB -- produced in response to the HBV infection.

Q. Well, I think that's different, isn't it? They're saying that the deposits containing one or more HBV-related antigens.

A. Yes.

Q. HBV-F and hepatitis B core antigens. Is that demonstrated in this kidney? I mean --

A. I told you repeatedly, the kidney was not measured for the antigens, just the antibody. So, when they stained this kidney for the antibody, they see the antibody in one area. Had they stained it for the antigen, they would have seen the antigen superimposed on the antibody because they're stuck together; but they didn't choose to stain the kidney because they didn't know he had hepatitis B.

Q. Who didn't know?

A. The people who looked at the kidney biopsy.

Q. Well, he was diagnosed with H -- with hepatitis B early, wasn't he?

A. Not -- no. The kidney biopsy was long before that diagnosis. The only way they diagnosed him with hepatitis B was after the kidney biopsy. And after they treated him, they caused a recrudescence of his hepatitis B by giving him immunosuppressant drugs and he came in with hepatic coma and that's when they tumbled to the diagnosis of hepatitis B.

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Q. So, would it be fair to say that since they didn't know to look for hepatitis B in the kidney, that there were no tests run to confirm whether the antigens or the antibodies are in the kidney?

A. Well, they got the antibodies in the kidney. They didn't test for the antigens.

Q. Where is that?

A. Well, that's the IgG that they're seeing. That's an antibody.

Q. Oh, that's an antibody specifically for hepatitis B. Is that what you're telling me?

A. I'm telling you that hepatitis B is well-known to produce monoclonal antibodies.

Q. Other things produce it, too, though, don't they?

A. Sure.

Q. Okay. So, we don't have specificity as to hepatitis B in this kidney sample, do we? We have the immunoglobulins in there, but they can be caused by any number of different diseases or situations that we've already talked about, correct?

A. Yes. He has immunoglobulins in the setting of an acute glomerulonephritis that has destroyed his kidney in the setting of hepatitis B, and I think one would assume that that's the cause. Now, this -- had they known he had hepatitis B or thought to look for hepatitis B, those -- that kidney

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could have been stained for hepatitis B, just like they stained their liver.

Q. When did they do the liver?

A. They did the liver biopsy when he came in in hepatic coma and they couldn't figure out why in the world he would suddenly go into hepatic coma. They thought they were treating somebody with kidney disease and when he came in in hepatic coma, they found out that he had striking titers for the hepatitis B virus and they biopsied his liver for the first time and found that he had chronic hepatitis and that he was positive for the hepatitis B antigens in the liver, as well as the particles of virus in the circulating blood.

Q. That was, I believe, in the fall of 2000. Does that sound about right?

A. It's probably in my original letter. Let's see. In April of 2001, prior to initiation of dialysis, he required hospital admission for an altered mental status and was found to have an elevated blood ammonia level. He was treated for hepatic encephalopathy and that's when they found the viral load. And the liver biopsy was done on 08-22-01.

Q. So, would it be fair to say then that when he was examined for the foamy urine and the kidney studies were done, we don't have any evidence of serological -- we don't have any serological evidence of persistent HBV infection at that time?

A. That would be correct, yes.

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Q. And we don't have any evidence of HBV-related antigens, HBsAg and hepatitis B core antigen, at that time in his kidney, do we?

A. Correct.

Q. The only thing we have is some immune complex deposits on kidney biopsy, correct?

A. No. What we have is a liver biopsy that shows he's got chronic hepatitis.

Q. Not in April of 2000, now, do we?

A. Well, but the liver biopsy was done -- I mean, has to -- when you see a liver biopsy that shows chronic inflammation for hepatitis, you didn't catch the hepatitis yesterday.

Q. In April of 2000, did we have anything other than some immune complex deposit on the kidney biopsy to make a determination that he did or did not have hepatitis B in April of 2000?

A. There was no diagnosis of hepatitis B in April of 2000.

Q. Could hepatitis B have been made in April of 2000 based on the information that you've seen in the medical records?

A. Absolutely.

Q. And what would that have been?

A. Well, this patient came in the hospital because of a

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nephrotic syndrome, and the nephrotic syndrome was typically due or frequently due to various forms of glomerulonephritis. And the usual workup of a patient like that is to look for causes of glomerulonephritis that might produce the nephrotic syndrome. Those would be illnesses like lupus, for example; illnesses like HIV, for example; illnesses like hepatitis B and C, for example; diabetes, for example. There are many illnesses that can produce glomerular damage and he simply was not worked up for those illnesses. He didn't have an evaluation for these illnesses.

Q. You mistook my question. There's nothing in the medical records in April of 2000 which would permit you to make that diagnosis based on the records themselves, correct?

A. That is correct.

Q. Okay. Have you reviewed the medical records of Dr. Potempa? Is that how you pronounce it?

A. If they're in the medical records I have, I have reviewed them.

Q. I think he was either a second opinion or he was a nephrologist who originally saw him.

A. That may be so. I can't dispute that.

Q. Okay. You just don't remember?

A. I don't remember, but I do remember that there's a report from a nephrologist that says he has glomerulonephritis.

Q. He's got a report from Dr. -- do you remember this

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report from Dr. Potempa in April of 2000 where he said, "Renal biopsy demonstrates a condition from the monoclonal production of light chain and heavy chain. There is a clone in the bone marrow producing these immunoglobulins."

Do you remember that?

A. Vaguely.

Q. And he concluded that this would be associated with myeloma, lymphoma, Waldenstrom's macroglobulinemia -- globulinemia -- you know what it is.

MR. CHILTON: Can you tell us what you're reading from, Dean, what medical record?

MR. HARTLEY: It's Potempa's report or letter to, I believe, one of his other treaters.

MR. CHILTON: Because I could be wrong about this, but I don't think Potempa was a nephrologist. I thought he was just one of his, like, family physicians. That's why I'm curious what you're reading from. You -- because you represented that he was a nephrologist and I don't recall that being correct. It may be.

MR. HARTLEY: In the letter to Reda.

MR. CHILTON: A letter to who?

MR. HARTLEY: R-e-d-a, Joseph Reda.

MR. CHILTON: Okay.

MR. HARTLEY: Dated April 17, 2000.

MR. CHILTON: And it's from Dr. Potempa?

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MR. HARTLEY: It is.

MR. CHILTON: And does it list what type of physician he is?

MR. HARTLEY: Well, his letterhead does not say that, but he had requested surgical path, etcetera, so -- and he worked him up.

MR. CHILTON: He was at La Grange, correct?

MR. HARTLEY: Correct.

MR. CHILTON: Okay. Thanks. Just so we have something to reference.

Q. (BY MR. HARTLEY) You don't remember that, Dr. Natelson?

A. I don't remember that.

Q. Okay. Oh, there was one follow-up, I think. I have tried not to go into your deposition from previously so we wouldn't go over the same crap.

MR. HARTLEY: Don't put "crap" down there, Miss Court Reporter.

Q. (BY MR. HARTLEY) Doctor, is chemotherapy of the VAD type myeloma specific?

A. No.

Q. What else is VAD used to treat?

A. Well, VAD has been used to treat situations where you want to suppress immunoglobulins. The treatment was developed originally by Dr. Alexanian at MD Anderson Hospital, I believe,

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and he used it in treatment of myeloma, but it is -- basically would be considered immunosuppressive treatment, and that's how they used it in this case.

Q. Is there any literature that suggests that VAD be used for immunosuppressant purposes?

A. Well, the "D" is dexamethazone. That's a steroid which is used by anybody who does transplant work or -- it's a commonly used immunosuppressive drug, as well as anything else.

Q. It kills lymphocytes, doesn't it?

A. It does. It's been used for a zillion things, but it's a strong immunosuppressive drug.

Q. How about thalidomide?

A. Thalidomide?

Q. Yeah. Is it myeloma specific?

A. No, certainly not.

Q. What other diseases that may be impacted by thalidomide? Or what other diseases may be impacted by thalidomide?

A. Well, it was actually first used in leprosy. It's an antiinflammatory drug and that's how it was actually introduced into practice. It had been used successfully in patients with leprosy. Then it was used to treat inflammation in an unusual illness, and let me just think if I can remember the name of it. It's an illness that's characterized by ulcers in the mouth and vagina, among other places. It's got a peculiar

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name.

Q. Not thrush, is it?

A. No, no, no, no, no. It's -- I have a mind blank now, but it's an inflammatory disease that is very distressing and immunosuppressive therapy has been used and thalidomide was an early use and very effective in this condition. And it's also associated -- this particular condition is associated with thrombophlebitis, too. And I'm just blanking on the name. It's an unusual spelling and it will probably come to me in a few minutes.

But, anyway, it's been used as an antiinflammatory drug and it was used in breast cancer, for example. It's used in people with myelodysplasia who have sick bone marrows. It's had a wide variety of uses.

Q. Would glomerulosclerosis be consistent with your crescentic glomerulonephritis opinion?

A. No, because all of these articles that Dr. Meehan cites and those that I cite that talk about this illness, the so-called light and heavy chain deposition disease, you will not find the word "crescent" or "crescentic" mentioned in any of those articles. Therefore, I would take that to believe that that's not a characteristic finding in that disease.

Q. Are you saying that if -- you're saying that because there were crescents seen in a couple of the glomeruli, that -- and they're not reported in any of these articles that you've

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looked at or cited by Meehan, therefore, it can't be glomerulosclerosis. Is that what you're saying?

A. What I'm saying is that 30 -- you said three out of nine, I think, in the first one. 30 percent of the glomeruli had crescents in them, and one would think that if you see that kind of a phenomenon in this illness, the doctors writing papers on it, like Dr. Ronco, would discuss that. And not only do they don't say it, but they -- he has a statement in here suggesting that it doesn't occur.

Q. Okay. And would you then say that the patient could not have glomerulosclerosis?

A. I would say he had glomerulonephritis.

Q. That's not the same thing as I'm saying. I'm asking you are you going -- is it your opinion as someone who reviewed the pathology in this particular case of a kidney that Mr. Lebron did not have glomerulosclerosis?

A. I'll defer that to Dr. McHugh. When you think about it, glomerulosclerosis just means scarring or damage to the glomeruli. The process that he had was glomerulonephritis. The end result is sclerosis.

Q. So, you can have glomerulosclerosis and be consistent with your diagnosis of crescentic glomerulonephritis --

A. Well, as an end stage --

Q. -- your opinion as a hematologist reading pathology of the kidney and studying -- and the kidney itself.

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A. My position as a hematologist is that Mr. Lebron's illness was consistent with a glomerulonephritis from hepatitis B; and that when you get a kidney that's an end stage kidney and it's burned out, it -- all the glomeruli are fibrotic and sclerotic and you may not be able to know what the inciting situation was.

Q. When you say the kidney is burned out, what do you mean by that?

A. Well, what happens is that initially when someone gets an inflamed kidney, it may be swollen and large and porous to protein, like it was in Mr. Lebron's case, and you're losing a lot of protein in the urine; but at the same time, your waste product levels, the BUN and creatinine levels, are normal. After a while, the inflammation there starts to damage and destroy the glomeruli, so now the waste product levels start to come up.

So, when Mr. Lebron was originally seen in 1999 and had the nephrotic syndrome, his renal function was normal, his BUN and creatinine are normal. When he comes back for the second time around, he's got abnormal renal function with an elevated serum creatinine level. So, the kidneys -- the glomeruli are starting to become shot. And then as time goes by, they shut down entirely, and he has to go on dialysis. And what happens if you looked at those kidneys, they start to get a little small as they scar up and fibrose.

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Q. Do you fault the physicians who treated him when he first appeared with his nephrotic syndrome that you've just described for failing to properly work him up and discover the hepatitis B at that time?

MR. CHILTON: I'll object. And you've already covered this in his prior deposition and we're not going to cover it again, Dean.

MR. HARTLEY: I don't think I did. I talked about treatment. I'm asking if he worked him up.

MR. CHILTON: Well, that wasn't the question you just asked.

MR. HARTLEY: Sure, it was.

MR. CHILTON: I disagree, so I'm objecting to the question.

MR. HARTLEY: Are you instructing him not to answer?

MR. CHILTON: Have I instructed him not to answer?

MR. HARTLEY: I'm just asking if you did.

MR. CHILTON: No.

MR. HARTLEY: Okay. Fine. But you said we're not going there.

MR. CHILTON: Well, we're not going to go back into topics that were covered at the original deposition. Having said that, I did not instruct the witness not to answer.

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MR. HARTLEY: Okay.

Q. (BY MR. HARTLEY) Doctor, that means you can answer.

A. Okay. Fine. My opinion is this: You have a patient that comes into the hospital with foamy urine. You find that he's got a strikingly abnormal amount of protein in his urine. He's got nephrotic syndrome. And for one reason or another, he was not worked up. Now, I wasn't there at the time. I don't know if he didn't stay for a workup. I don't know if the doctors didn't advise him to get a workup.

But the bottom line, he was not properly evaluated for that condition at that point in time so that when he came back in 2001, he's far worse than he was. And the -- and had he been diagnosed early on with hepatitis B and appropriately treated, he may never have gone into renal failure. But I don't know why that was the case. In other words, I don't know whether he didn't choose to have a workup, wasn't advised to have a workup, told to come back a week later and never came back. I have no idea. That you'd have to ask his original doctors.

Q. Well, one final question then: Assuming he was hospitalized for this workup, was seen by a couple physicians for this workup, did not refuse to do anything that was asked of him, if the physicians who were treating him did not include hepatitis B in his differential diagnosis and work him up for hepatitis B, did they breach the standard of care?

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MR. CHILTON: I'm going to object and I am going to instruct him not to answer. He's not here to testify as to issues related to standard of care and potential actions that were taken by his treating physicians relative to that treatment. I don't think that he's being presented for that purpose in this case and he's not going to testify with respect to opinions on that topic.

MR. HARTLEY: Then that means at trial, he will not be chastising those physicians for not working him up when he should have been worked up in April of 2000. Is that what you're saying?

MR. CHILTON: That's not what I said at all.

MR. HARTLEY: Okay.

MR. CHILTON: You asked a different question.

MR. HARTLEY: We'll take it up with the court then. That's the easiest way to do it. Because if he's going to testify that he wasn't worked up properly so as to impugn the physicians, I think we're entitled to know whether that's a breach of the standard of care so that the jury can actually know where he stands on this issue. But we'll take it up on a later date. It's okay. I don't have anything further.

MR. CHILTON: Anybody else have anything?

MR. HARTLEY: Just a second.

MR. GILMARTIN: Nope.

MR. MILLER: Nope.

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MR. CHILTON: We all set?

MR. HARTLEY: Wait a minute. Just a second. I finally got Dr. Barrios' report. Just want to look at this for a second.

Q. (BY MR. HARTLEY) Doctor, did you point out to Dr. Barrios that the lesion that he -- it says here, "As you point out" -- this is in a letter from him to you -- "this lesion can be associated to hepatitis B."

Did you bring it to his attention that whatever was seen on the pathology could be associated with hepatitis B?

A. Well, I gave him the slides. I said, "I want you to look at some kidney slides," and he looked at it.

And about 30 seconds, he said, "This is a crescentic glomerulonephritis and I would be thinking about things like HIV disease and hepatitis C."

And I said, "How about hepatitis B?"

And he said, "Yeah. Hepatitis B certainly can produce this." And then I told him the situation of what the case was all about. And so when he wrote the letter, he had already had a discussion with me about the fact that the patient did have hepatitis B; but spontaneously he brought it up that the first things he would think about when he looked at a biopsy like that would be HIV, hepatitis C, and hepatitis B.

Q. Did you give him the reference to -- it's either Lai or Lai, L-a-i, the citation that he uses, "Crescentic

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glomerulonephritis related to hepatitis B virus"?

A. No, no.

Q. Is Dr. Barrios a renal pathologist?

A. Yes. I went to Mary Schwartz, who's really their head of the department, and I said, "Who would be the best person to look at a kidney biopsy?"

And she said, "Show it to Dr. Barrios."

Q. Okay. That's all I have.

MR. CHILTON: Very good. Anybody else?

UNIDENTIFIED SPEAKER: No.

MR. CHILTON: Okay. We've got the one exhibit. I'll ask the court reporter to attach that to the transcript. Very good. Thanks, everybody.

MR. GILMARTIN: Shanon, I'll take a copy and also a digital file, Bob Gilmartin.

THE REPORTER: Okay. Thanks.

MR. MILLER: And I want a copy, too. This is Jim Miller.

THE REPORTER: Okay.

MR. HARTLEY: Shanon, naturally, I'd like a copy.

THE REPORTER: Okay.

MR. HARTLEY: All right. Thanks a lot.

(Deposition concluded at 3:22 p.m.)

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CHANGES AND SIGNATURE

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I, ETHAN A. NATELSON, M.D., F.A.C.P., have read the foregoing deposition and hereby affix my signature that same is

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true and correct, except as noted above.

_____

ETHAN A. NATELSON, M.D., F.A.C.P.

THE STATE OF _____)

COUNTY OF _____)

Before me, _____, on this day personally appeared ETHAN A. NATELSON, M.D., F.A.C.P., known to me (or proved to me under oath or through _____) to be the person whose name is subscribed to the foregoing instrument and acknowledged to me that they executed the same for the purposes and consideration therein expressed.

Given under my hand and seal of office this _____ day of _____, _____.

_____

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I, the undersigned certified shorthand reporter in and for the State of Texas, certify that the facts stated in the foregoing pages are true and correct.

I further certify that I am neither attorney or counsel for, nor related to or employed by, any of the parties to the action in which this deposition is taken and, further, that I am not a relative or employee of any counsel employed by the parties hereto, or financially interested in the action.

SUBSCRIBED AND SWORN TO under my hand and seal of office on this the 20th day of November, 2008.

SHANON M. HAIR, CSR
Certified Shorthand Reporter
In and for the State of Texas

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July 14, 2008

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RE: Cause No. 05 L 9246; Pamela Lebron, as Personal Representative of the Estate of Michael (Miguel) A. Lebron, vs. Shell Oil Company, a Delaware Corporation, et al., In the Circuit Court of Cook County, Illinois County Department, Law Division.

Dear Mr. Woolf,

At your request, this letter is an update of my opinions concerning the above referenced action. Considerable time has elapsed since I drafted my initial report concerning this action in February 2004, and gave my deposition which was taken in October 2004. Since that time, Mr. Lebron has died, and I and others have had the opportunity to review his pathology slide material, further depositions have been taken, and additional information has appeared in the peer-reviewed medical literature which is pertinent to the issues under discussion. As you know, it is my opinion that Mr. Lebron suffered several well-described complications of hepatitis B (HBV) virus infection which included the nephrotic syndrome and renal failure due to crescentic glomerulonephritis, a mild reactive bone marrow plasmacytosis, monoclonal (oligoclonal) gammopathy, chronic hepatitis with early cirrhosis, re-activation of acute hepatitis B following administration of immunosuppressive drugs, and, after several years of undergoing chronic hemodialysis, death from hepatic replacement with adenocarcinoma of unproven primary, but clinically most likely to represent cholangiocarcinoma. As you are aware, no autopsy was performed.

The most recent plaintiff opinions I have received are put forward in the deposition of Dr. Shane Meehan and which supplement those opinions originally

EXHIBIT
NATELSON 1

expressed in the much earlier deposition of Dr. Brian Durie, and in Dr. Durie's letter dated September 7, 2005. I will contrast my opinions with theirs, not in any particular order of importance, and reference the pertinent medical literature as it applies to each particular issue.

1. **The bone marrow findings**: Mr. Lebron had a reactive plasmacytosis (*a non-neoplastic increase in the numbers of mature plasma cells*) in his single bone marrow study, with the original report listing the presence of plasma cells in the aspirate as amounting to 5 % of the total cellularity (normal percentage, 1-3 %). Plasma cells are typically quantitated in a bone marrow aspirate specimen because the cellular elements are dispersed and can be accurately and individually identified and counted. In a bone marrow biopsy, thousands of cells are tightly compressed together and cell lineage and percentages are impossible to accurately assess by routine light microscopy. Flow cytometry of bone marrow cells also can compute percentages of the various cell types, but plasma cells are fragile and the flow studies typically underestimate their numbers. I have quantitated the plasma cells in the bone marrow aspirate slides from Mr. Lebron that were provided to me by counting 300 total cells, several times, and always get a percentage of plasma cells less than 5 %, and sometimes as low as 3 %. I would not dispute a count of 4 %, or a very trivial increase. Virtually all of these plasma cells are small, mononuclear, and not unusual or immature in appearance. Most people, including physicians, do not realize how commonly a non-neoplastic plasmacytosis (*a mild increase in plasma cells*) is identified among routine bone marrow examinations. From an era when bone marrow aspirations were applied more widely in diverse illnesses now diagnosed by other techniques, we find, "**Reactive bone marrow plasmacytosis of varying degrees is quite common, occurring with a frequency of 28.6 % in one recent series of 1,000 bone marrow biopsies, excluding patients with documented plasma cell disease such as plasma cell myeloma**" (1). Many inflammatory conditions, particularly including chronic infections, collagen vascular disorders, and hepatic cirrhosis, regardless of cause, are associated with an increase in reactive plasma cells, and, as I referenced in my original report, hepatitis B is a typical example of this category (2-3). Thus, the mild, reactive plasmacytosis present in Mr. Lebron's marrow has no particular diagnostic significance, would be expected with his inflammatory conditions of hepatitis and glomerulonephritis, and certainly does not suggest multiple myeloma.

2. **Monoclonal Gammopathy**: Mr. Lebron had a tiny monoclonal gammopathy demonstrated in both his serum and urine. In his deposition,

Dr. Meehan did not seem aware of how small these protein peaks were in comparison to what we usually encounter in patients with multiple myeloma. In Mr. Lebron's serum and urine they required high resolution electrophoresis to be demonstrated, not showing up on his original studies, and typically measured only 0.2 to 0.3 gm/dl in serum during his entire period of medical observation. These concentrations would be identical to the findings of Tsianos, who found mean values of 0.29 gm/dl present in serum oligoclonal bands among 25 samples from patients with hepatitis B (4). As I mentioned in my initial report, monoclonal gammopathy, with both large and small paraprotein peaks, has been commonly cited as a consequence of or an association with hepatitis as a class of illness and, specifically, with hepatitis B and C (5-6). Studies of large numbers of United States military veterans with monoclonal gammopathies of undetermined significance have demonstrated this phenomenon in association with number of chronic inflammatory states including both hepatitis B and C (5). Dr. Meehan did not appear to appreciate or accept the frequency of this association, which is well-described in the peer-reviewed medical literature, both domestic and from international sources.

3. **Crescentic Glomerulonephritis**: The original pathology review and subsequent descriptions by all who have examined the kidney biopsy, including Dr. Meehan, have cited the obvious finding of glomerular crescents. Crescents are a *sine qua non* of inflammatory glomerulonephritis, and well-described as a consequence of hepatitis B infection. In understanding the significance of glomerular crescents in a kidney biopsy, we read, "**Accumulation of cells in Bowman's space, surrounding the glomerulus and compressing it, leads to an appearance in histological sections which is known as a 'crescent'...the extent of crescent formation has long been recognized as an indicator of the severity of the inflammatory process...thus in acute severe forms of GN (*glomerulonephritis)* such as that mediated by autoantibodies...crescent formation is prominent...**". Further, by this review, "...**activation of the coagulation system leading to fibrin deposition is often prominent...**" (7). Crescents are seen in a number of types of glomerulonephritis, including animal models, and are composed of several types of cells (8). Now, Dr. Meehan alleges that Mr. Lebron did not have immunoglobulin deposition in the kidney from glomerulonephritis, but rather had immunoglobulin deposition from a form of glomerulosclerosis or a nodular sclerosing glomerulopathy, which may be associated with multiple myeloma. He also alleges that glomerular crescents appear in this pathological entity. However, none of the references he cites, nor other

articles that I originally cited, mention the words crescent or crescentic in the description of the histology of the kidney in that type of immunoglobulin deposition disease (9-11). In the article by Ronco on this subject that Dr. Meehan references, there is a nice photograph showing an affected glomerulus, representative of this type of immunoglobulin deposition disease, surrounded by a pristine Bowman's space with no hint of any crescent formation. This particular report also teaches us, in reference to the histological appearance of this more bland immunoglobulin deposition disease, and in contrast to a crescentic glomerulonephritis, "...**exudative lesions as 'fibrin caps'...are not observed**" (9).

4. **Nephrotic Syndrome and Chronic Renal Failure**: In Dr. Brian Durie's deposition he seemed unaware that hepatitis B could lead to the nephrotic syndrome and renal failure. Of course, if one is unaware of the natural history of a disease process, he or she is then rather unlikely to consider that diagnosis when evaluating a patient with the nephrotic syndrome and renal failure. Dr. Meehan was aware of this association. In fact, the sequence of glomerulonephritis, progressive proteinuria and renal failure as a consequence of hepatitis B is a rather frequent occurrence and well-described around the world as I indicated in my initial report.

5. **Hepatitis B virus reactivation**: Following Mr. Lebron's treatment with corticosteroids and chemotherapy drugs which were given as form of immunosuppressive treatment, hoping to reduce his immunoglobulin levels and benefit his renal disease, he became acutely ill with deteriorating liver function tests and hepatic encephalopathy. At that point the diagnosis of an acute flare of relatively quiescent chronic hepatitis B infection became evident when his viral load studies demonstrated virus particle levels in excess of one million copies per ml of serum and his liver biopsy confirmed chronic disease changes. In the years that have elapsed since his initial therapy, much has been written about this viral reactivation syndrome in hepatitis B, and its therapy and prevention (12-13). We now know that lamivudine (*Epivir*), or similar drugs must be given to the carriers of hepatitis B before initiation of powerful immunosuppressive drugs to prevent this complication. We also now know that early treatment with lamivudine or similar drugs may reverse the nephrotic syndrome and prevent renal failure (14-16). Thus, it is unfortunate that Mr. Lebron's physicians did not elicit the history that his wife had been rejected years earlier as a blood donor, when he was first seen for the nephrotic syndrome, and discharged with no attempt at treatment. Hepatitis B is an extremely infectious illness, and had his physicians had this history, he would, in all

likelihood, have been tested for hepatitis B early on.

6. **Multiple Myeloma and its Diagnostic Criteria**: Dr. Meehan tells us that Dr. Durie's original 1970s criteria which must be met for the diagnosis of multiple myeloma are now "**obsolete**" and, instead, the suggested criteria Dr. Durie cited in his deposition and from his 2003, report are now employed (17). And, he believes that Mr. Lebron had multiple myeloma by employing these criteria. How is it then, that when we read an article from 2008, in which Dr. Durie is the second author, along with 22 other contributors from around the world, and which describes the findings in 1,689 patients with multiple myeloma, we learn, "**Standard criteria were applied for diagnosis of multiple myeloma**" (18). Yet, the citation for these standard criteria in this 2008 article is Dr. Durie's report from 1977. As Dr. Meehan agrees, Mr. Lebron does not come even close to meeting these 1977 criteria. In fact, the 2003 proposed criteria described in that article's Table V require no particular level of paraprotein, marrow plasmacytosis or specific type of tissue impairment, and, thus, are so non-specific and non-discriminatory that many patients with HIV infection, and diseases such as lupus, and hepatitis B and C could easily meet these criteria and claim to have multiple myeloma, too (19). Perhaps this is why Dr. Durie and others are not using them in 2008.

7. **Death from extensive hepatic adenocarcinoma:** When Mr. Lebron was found to be terminally ill, his liver was entirely replaced by adenocarcinoma. Dr. Meehan and others who have reviewed this tumor tissue opine that it could represent metatastic adenocarcinoma or a primary cholangiocarcinoma (*bile duct*) tumor. While Mr. Lebron did not undergo autopsy, he did have colonoscopy and CT and total body PET scanning prior to his death. This tumor in the liver was extremely "hot" by PET scan yet there were no hot spots or masses in his pancreas, kidneys, colon or intestine suggestive of a primary site of origin. He did not have breast cancer. Clinically, it would be very unusual for metatastic adenocarcinoma from the typical sources (breast, stomach, colon, prostate, kidney, pancreas, or lung) to completely replace the liver with tumor and reveal no primary site by the studies that he had accomplished. However, this clinical pattern of extensive intrahepatic tumor, alone, is commonly encountered in cholangiocarcinoma. Thus, cholangiocarcinoma is the most likely diagnosis, particularly in this setting. It has long been known that primary liver cell adenocarcinoma is a quite common complication in patients with chronic hepatitis B infection and in cirrhosis, itself. In recent years, it has become more widely appreciated that hepatitis B can also predispose to hepatic

cholangiocarcinoma. Because hepatitis B and C infections are endemic in Asian countries, some of these statistically significant observations on large series of patients have been described among those populations, as well as by nucleic acid testing in the United States (20-23). Such reports demonstrate that, in contrast to Dr. Meehan's opinion, it is now generally accepted that hepatitis B is a significant risk factor for cholangiocarcinoma.

8. **The relationship between multiple myeloma and a second primary malignancy, such as adenocarcinoma:** Dr. Durie, in his letter, claims multiple myeloma predisposed Mr. Lebron to metastatic adenocarcinoma. Certainly, patients with multiple myeloma who are treated with large doses of alkylating agents such as alkeran and cyclophosphamide and/or radiation therapy have a higher incidence of a secondary acute myeloid leukemia (AML) than the general population. Because of occasional anecdotal reports which describe a second solid tumor, the question of a specific predisposition to a second malignancy in multiple myeloma was studied by Dr. Alexanian, in 628 multiple myeloma patients at The MD Anderson Cancer Center, and by others (24). There has been no consistent epidemiological evidence that multiple myeloma predisposes to a specific second malignancy such as adenocarcinoma.

9. **What is missing for a diagnosis of multiple myeloma:** Multiple myeloma is a progressive and typically incurable disease with a mean 3-4 year survival following diagnosis. Its course is characterized by progressive anemia, osteolytic bone disease, an increasing paraprotein (*monoclonal gammopathy*) in the serum or urine, and, often, hypercalcemia. Over several years of observation, Mr. Lebron manifested none of these findings. No bone marrow study was ever repeated. Thus, his clinical course would be inconsistent with multiple myeloma.

10. **Etiology of multiple myeloma:** At the present time, multiple myeloma continues to be an illness of unknown cause, with the exception of rare familial appearance, and in a recent exhaustive epidemiologic review, has shown no consistent and statistically significant association with chemical causation (25). As commented upon in a recent article by the editor of a major hematology text, "**Only a small percentage of the blood cancers have a cause related to exogenous or environmental factors. The only blood cancer for which there is convincing scientific evidence for an avoidable environmental cause is AML (and the closely related disease myelodysplasia), and only in a minority of cases**" (26).

In summary, when one reviews and becomes familiar with the natural history of hepatitis B infection and its complications, all seemingly diverse aspects of Mr. Lebron's illness fall into place easily and there should be no confusion with the syndrome of multiple myeloma, which he did not have, and which is not chemical-related in the first place. Please let me know if you require any additional information. As you are aware, I have taken photographs of Mr. Lebron's pathology slides which include those of his liver, bone marrow and kidney.

Cordially yours,

Ethan A. Natelson, M.D.

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