

ORIGINAL

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

MICHAEL A. LEBRON and :
PAMELA LEBRON, his wife :
Plaintiffs :

VS. : Case No. 00L10021

SHELL OIL COMPANY, et al. :
Defendants :

ORAL DEPOSITION OF

ETHAN A. NATELSON, M.D.

OCTOBER 27, 2004

Allied
ADVANCED
REPORTING
INCORPORATED

1647 COLQUITT HOUSTON, TEXAS 77006-5203

PH: 713.524.6777 FAX: 713.524.6888 TOLL FREE: 800.223.9409

EMAIL: AARI@ALLIEDADVANCEDREPORTING.COM

ORIGINAL

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

MICHAEL A. LEBRON and :
PAMELA LEBRON, his wife :
Plaintiffs :
VS. : Case No. 00L10021
SHELL OIL COMPANY, et al. :
Defendants :

ORAL DEPOSITION OF

ETHAN A. NATELSON, M.D.

OCTOBER 27, 2004

Called as a witness by the Plaintiffs, taken before
Debbie Leonard, CSR, RMR, CRR, a Certified Shorthand
Reporter in and for the State of Texas, reported by
machine shorthand, on the 27th day of October, 2004,
from 9:09 a.m. to 10:40 a.m., at the offices of Stehlin
de Ipolyi Oncology Clinic, 1315 St. Joseph Parkway,
Suite 1800, Houston, Texas, pursuant to the Texas Rules
of Civil Procedure and the provisions stated on the
record or attached hereto.

A P P E A R A N C E S**COUNSEL FOR PLAINTIFFS:**

Mr. R. Dean Hartley
(via telephone)
HARTLEY & O'BRIEN
2001 Main Street, Suite 600
Wheeling, West Virginia 26003
Phone: (304) 233-0777

COUNSEL FOR DEFENDANT, SHELL OIL COMPANY:

Mr. Larry J. Chilton
CHILTON YAMBERT PORTER & YOUNG, LLP
150 South Wacker Drive, Suite 2400
Chicago, Illinois 60606
Phone: (312) 460-8000

COUNSEL FOR DEFENDANT, E.I. DUPONT DE NEMOURS & COMPANY:

Mr. Jim C. Ezer
SCHIRMEISTER DIAZ-ARRASTIA
Pennzoil Place - South Tower
711 Louisiana, Suite 1750
Houston, Texas 77002
Phone: (713) 228-3545

COUNSEL FOR DEFENDANT, HYDROSOL, INC.:

Mr. Robert E. Gilmartin
(via telephone)
CLAUSEN MILLER
10 South LaSalle Street
Chicago, Illinois 60603
Phone: (312) 855-1010

COUNSEL FOR DEFENDANT, PPG INDUSTRIES, INC.:

Mr. James R. Miller
(via telephone)
DICKIE, McCAMEY & CHILCOTE
Two PPG Place, Suite 400
Pittsburgh, Pennsylvania 15222
Phone: (412) 392-5238

1 **ALSO PRESENT:**

2 Joseph Ayoub, M.D.

3 **COURT REPORTER:**

4 Ms. Debbie Leonard, CSR, RMR, CRR
5 Allied Advanced Reporting, Inc.
6 1647 Colquitt Street
Houston, Texas 77006
7 Phone: (713) 524-6777
8 Fax: (713) 524-6888
9
10
11
12
13
14
15
16
17
18
19
20
21
22
23
24
25

I N D E X

PAGE

Appearances 2

WITNESS: ETHAN A. NATELSON, M.D.

Examination By Mr. Hartley 6

Examination By Mr. Gilmartin..... 68

Changes and Signature 70

Reporter's Certificate 72

E X H I B I T S I N D E X

ORAL DEPOSITION OF
 ETHAN A. NATELSON, M.D.
 OCTOBER 27, 2004
 NOS. 1 - 8

NUMBER	DESCRIPTION	PAGE
1	Supplemental Report	62
2	Handwritten notes	61
3	Billing records	62
4	Article Lamivudine for Hepatitis B in Clinical Practice Journal of Medical Virology	65
5	Article Lamivudine for Patients with Chronic Hepatitis B and Advance Liver Disease The New England Journal of Medicine	65
6	Article Lymphohaematopoietic cancer risk among chemical workers exposed to benzene Occup Environ Med Bloemen, Youk, Bradley, et al.	65
7	Article Advances in biology of multiple myeloma: Clinical applications Blood Hideshima, Bergsagel, et al.	66
8	Article Pathology and Genetics of Tumors of Haematopoietic and Lymphoid Tissues World Health Organization Jaffe, Harris, Stein & Vardiman	68

1 ETHAN A. NATELSON, M.D.,
2 having been first duly sworn, testified as follows:

3 EXAMINATION

4 BY MR. HARTLEY:

09:09 5 Q. Dr. Natelson, good morning.

6 A. Good morning.

7 Q. Could you tell us your full name?

8 A. Ethan A. Natelson.

9 Q. Do you have a copy of the report that you
09:09 10 prepared dated February 15, 2004, with you today?

11 A. Yes.

12 Q. Would you pull that out for me?

13 A. Yes, sir.

14 Q. Have you prepared any additional reports
09:09 15 since the February 15th report?

16 A. Yes, there was a brief report that had to do
17 with quotes from an article and comparison quotes from
18 a kidney biopsy.

19 Q. Okay. Let me put you on hold and see if I
09:10 20 can find that, because I remember that now. I don't
21 know where it went. Bear with me just a second.

22 MR. CHILTON: Dean?

23 (Pause)

24 Q. (By Mr. Hartley) Okay, Doctor. Let's talk
09:10 25 for a moment about the second page of your report and

1 particularly the pathology that you're referencing --

2 A. Yes.

3 Q. -- in the second full paragraph. Do you have
4 the actual pathology report from Loyola with you?

09:10 5 A. Yes.

6 Q. Let's pull that out.

7 A. All right. Okay. Yes, I've got that.

8 Q. And it appears that that pathology, at least
9 on my copy, has at the bottom of it, C16, C17, and C18
09:11 10 in handwriting. Does your copy have that?

11 A. Well, I've got -- this report has been copied
12 several times. I've got one that says L30, L31, L32,
13 L33, L34.

14 Q. Okay.

09:11 15 A. It appears several times in these records
16 that I have.

17 Q. All right. Is your surgical pathology report
18 from Loyola University with a specimen number S00-5459?

19 A. Yes.

09:11 20 Q. And at the bottom of the -- under "Gross
21 Description" it has, "EM specimen is composed of four
22 pieces measuring .2 by .2 by .2 CM"?

23 A. Yes.

24 Q. On the second page it looks like it has at
09:12 25 the bottom "4/10/00 Maria M. Picken, MD, Ph.D."?

1 A. Correct.

2 Q. And the third page would have, "physician
3 notification, Dr. L. Potempa, P-O-T-E-M-P-A, notified"?

4 A. Yes, sir.

09:12 5 Q. Okay. So we've got the same report then?

6 A. Yes.

7 Q. In your narrative report, you indicate for
8 me -- or indicate for Mr. Woolf, rather, that you're
9 unsure or uncertain whether the pathologist
09:12 10 interpreting the biopsy was even aware of the fact that
11 Mr. Lebron had chronic hepatitis. And look with me on
12 the clinical information of that first surgical path
13 report.

14 A. Clinical? "Proteinuria, nephrosis, normal
09:13 15 serum albumin."

16 Q. Yeah. It says here, "Proteinuria, nephrosis,
17 normal serum albumin."

18 A. Yes.

19 Q. Do you see that?

09:13 20 A. Yes.

21 Q. Does that in any way indicate to the
22 pathologist reviewing this what's going on at least in
23 the patient's clinical history?

24 A. Well, the clinical history is -- that tells
09:13 25 him -- or one should suspect a nephrotic syndrome, in

1 other words, heavy proteinuria.

2 Q. Okay. And to be fair, you're not a
3 nephrologist, correct?

4 A. That's correct.

09:13 5 Q. Okay. Would the clinical information and
6 history that is presented on the surgical path report
7 have given indication one way or the other for the
8 pathologist here, Dr. Picken, that Mr. Lebron did or
9 did not have any type of -- or any suspected type of
09:14 10 kidney problems from the clinician? Do you understand
11 the question?

12 A. Not exactly.

13 Q. It was a poor question after I said it.

14 What does the pathologist realize the
09:14 15 clinician is including as potential underlying causes
16 of what they think is the kidney problem in this
17 particular case?

18 A. Well, generally the pathologist may either
19 have nothing at all, and they constantly complain about
09:14 20 that, or they will have some general reason for the
21 biopsy. In other words, hematuria, blood in the urine,
22 proteinuria, protein in the urine, kidney failure, some
23 general comment as to why the biopsy was done.

24 And then typically what happens is, the
09:15 25 attending physician may call on the phone or come and

1 appear in person and go over the biopsy with the
2 pathologist and provide additional information.

3 Q. What does the term "nephrosis" mean in the
4 clinical information?

09:15 5 A. Well, it's an old term. It just means that
6 there's proteinuria.

7 Q. Okay. So it's a duplication?

8 A. Yes.

9 Q. All right. And what is the significance of
09:15 10 having normal serum albumin when you have proteinuria?

11 A. Well, it may be no significance. In general,
12 as one starts losing protein, because albumin is a
13 small molecule, the more protein you lose in the urine,
14 the lower the serum albumin usually goes. At the onset
09:15 15 of an illness, it may not be very low. If, let's say,
16 you're putting out a gram or two or three grams a day,
17 when you start putting out 10 or 15 grams of protein a
18 day, it's typically quite low.

19 Q. Does the fact that you have a normal finding
09:16 20 in light of the proteinuria for albumin give the
21 pathologist any indication of what might be the cause
22 of the underlying disease?

23 A. No.

24 (Mr. Ezer entered the deposition room.)

09:16 25 MR. CHILTON: For the record, Jim Ezer

1 just arrived. He didn't bring any food for any of us
2 this time.

3 MR. HARTLEY: The kind of guy he is, you
4 know.

09:16 5 MR. EZER: I was going to participate
6 telephonically, but they said you were so --

7 Q. (By Mr. Hartley) Let's look then at the
8 second page of the surgical path report.

9 A. Okay. I have that.

09:16 10 Q. All right. And this particular instance in
11 the Lebron case, you have suggested that the underlying
12 problem may be the result of a hepatitis, correct?

13 A. Well, on this report I suggested that the
14 underlying problem is a type of glomerulonephritis.
09:17 15 There are many causes of glomerulonephritis, hepatitis
16 being one of them.

17 Q. Well, I guess my question to you then is,
18 when we go to trial in this case, are you going to
19 assert to the jury that his hepatitis B is the
09:17 20 underlying cause of his nephritis?

21 A. I would say it's consistent with that.

22 Q. Well, there's a lot of things consistent with
23 it as well though, correct?

24 A. He has a glomerulonephritis, and he has
09:17 25 hepatitis B, and it's known that hepatitis B can cause

1 a glomerulonephritis. He didn't have the definitive
2 staining for the hepatitis B particles in the kidney
3 that can be done, but his illness is quite consistent
4 with hepatitis B induced glomerulonephritis and
09:17 5 ultimately renal failure.

6 Q. So what you're saying is that, well, it could
7 be the result of his hepatitis B, but it could be
8 consistent with other things as well; is that fair?

9 A. Yes. Any glomerulonephritis -- there could
09:18 10 be many potential causes for glomerulonephritis.

11 Q. Now, you reviewed this case and prepared a
12 report in February of 2004. Did you at any time obtain
13 the slides to review?

14 A. No.

09:18 15 Q. Did you have anyone contact -- but you had
16 suggested that you would like to see those, correct?

17 A. Correct.

18 Q. Okay. And you never were able to get those?

19 A. Well, I never received them. I mean, I don't
09:18 20 know if efforts were made or not made or if they were
21 secured by someone. All I know is, I never received
22 those slides.

23 Q. Did you call Mr. Woolf and say -- or
24 Mr. Chilton and say, "Where are the slides? I would
09:18 25 like to take a peek at them"?

1 A. I'm sure I asked Dr. -- Mr. Woolf that, if I
2 could review the slides. I'm quite confident I did.

3 Q. Okay. And that was never presented to you?

4 A. That's correct.

09:18 5 Q. Now, would the staining that you have
6 suggested a moment ago determine whether the underlying
7 kidney disease is or is not the result of his hepatitis
8 B?

9 A. Well, they would be strongly suggestive.

09:19 10 Q. Okay. We could be -- if we had done
11 staining, we could move closer to a "strongly
12 suggestive" as opposed to a "consistent with." Would
13 that be reasonable?

14 A. Yes.

09:19 15 Q. Assuming the staining comes back positive?

16 A. Yes.

17 Q. Okay. Now, in light of that, did you suggest
18 that maybe someone ought to go and contact the
19 pathology department, get the blocks, cut some new
09:19 20 slides and do some staining for hepatitis B?

21 A. I suggested to Mr. Woolf that that could be
22 possible, yes.

23 Q. And what was the response to that?

24 A. "We'll look into it."

09:19 25 Q. Okay. Now, that would certainly be a much

1 more definitive way of determining whether the
2 hepatitis B that Mr. Lebron experiences is at least a
3 partial contributing factor to the underlying kidney
4 problems, correct?

09:20 5 A. Well, it would be strongly suggestive that it
6 was the etiology because that's been noted in many
7 articles on the subject.

8 Q. And you would agree with me, would you not,
9 that staining, giving the slides some special stains,
09:20 10 would be able to one way or the other rule out whether
11 the hepatitis B is, in fact, the cause of the
12 underlying -- or the cause of the kidney problems?

13 A. I don't know that it would 100 percent prove
14 it or disprove it, depending on what you saw. It's
09:20 15 just that it's commonly present. One, of course, would
16 know that at the time of that kidney biopsy there was
17 active hepatitis B in the kidney, but I don't know that
18 that would 100 percent prove that the hepatitis B was
19 the cause of the glomerulonephritis. It would simply
09:21 20 be strongly suggestive.

21 Q. In the articles that I reviewed that you
22 cited, it appears to me that for the changes that we're
23 seeing in Mr. Lebron, particularly the immunoglobulin
24 deposition, IgG and IGA, that the disease process had
09:21 25 to be fairly far advanced, does it not, for hepatitis B

1 to be the contributing factor?

2 A. I don't know that that's necessarily so. You
3 know, hepatitis B has a variable progression in people.
4 One can develop cirrhosis within four or five years or
09:21 5 take 16 or 20 years to get cirrhosis from it. It's a
6 very variable condition. And I -- as we speak here
7 today, I don't know if that would be a correct
8 statement, that one would have had to have had
9 hepatitis B for many years in order to get renal
09:22 10 failure.

11 Q. Would you agree with me that one would need
12 to have -- does hepatitis B cause cirrhosis?

13 A. Yes.

14 Q. Would you agree with me that the articles
09:22 15 that you cite indicate that one needs to have a
16 cirrhotic liver before the hepatitis is, in fact,
17 causing the kidney problems?

18 A. No, that's totally incorrect.

19 Q. Okay. So the article -- one of the articles
09:22 20 that you cite that suggests that is wrong?

21 A. Well, I don't know if the article is wrong.
22 We would have to look at what was said. But one can
23 certainly have kidney failure and not have cirrhosis
24 from hepatitis B.

09:22 25 Q. I'm saying for the -- for the kidney failure

1 to be related to the hepatitis B, the liver needs to be
2 cirrhotic?

3 A. No, that's not correct.

4 Q. Okay. Would you agree with me that hepatitis
09:22 5 B does not usually cause major renal problems?

6 A. Yes, that is correct.

7 Q. As we sit here today, can you explain to me
8 your theory of a causative mechanism that hepatitis B
9 would be the potential cause of Mr. Lebron's renal
09:23 10 problems?

11 A. Well, the chart indicates that the patient
12 has chronic hepatitis B as evidenced by persistent
13 viremia and an abnormal liver biopsy. And we know that
14 in adults with chronic hepatitis B, renal failure can
09:23 15 certainly occur. And so he's in a setting where it
16 wouldn't be unusual to get renal failure and
17 progressive nephrotic syndrome.

18 Q. All right. How does the hepatitis B cause
19 the renal failure, if you know?

09:23 20 A. Well, there are several postulated
21 mechanisms. Some believe that what happens is, the
22 particles -- the hepatitis virus antigens get into the
23 kidney, they stimulate antibody production. And then
24 you get antibody and sometimes antigen together, what's
09:24 25 referred to as an immune complex, landing in the

1 kidney, and you can get glomerulonephritis from that.
2 And then as one gets glomerulonephritis, it's often a
3 progressive disease, and the claim is that in adults,
4 once you get glomerulonephritis from the hepatitis B, I
09:24 5 think one of the articles claims a third of the
6 patients end up in renal failure.

7 Q. How does one determine whether the individual
8 has glomerulonephritis resulting from hepatitis B?

9 A. It's as in any form of glomerulonephritis,
09:24 10 it's a clinical -- it's a clinical judgment.

11 Q. What does one utilize in making that clinical
12 judgment to make such a determination?

13 A. One looks for evidence that, A, the patient
14 has chronic hepatitis B. As we discussed, one can look
09:25 15 for the particles of the virus in the kidney itself.
16 And one attempts to exclude other causes for
17 glomerulonephritis, things such as lupus or the many
18 causes of glomerulonephritis.

19 Q. And does light chain disease cause
09:25 20 glomerulonephritis?

21 A. Well, it causes renal failure. It can cause
22 a form of glomerulonephritis. It sort of more tends to
23 plug up the tubules of the kidney. But heavy light
24 chain proteinuria can certainly cause
09:25 25 glomerulonephritis and renal failure.

1 Q. What about myeloma?

2 A. Multiple myeloma?

3 Q. Yes, sir.

4 A. Yes, multiple myeloma is frequently
09:25 5 associated with renal failure.

6 Q. And were the particles of hepatitis B looked
7 for in the kidney in this particular case?

8 A. No. As we discussed, they were not stained
9 for specifically.

09:26 10 Q. Okay. Is it simply your opinion that folks
11 with hepatitis B can get renal failure, and therefore
12 it is possible that Mr. Lebron's renal failure is the
13 result of his hepatitis B?

14 A. His clinical course is quite consistent with
09:26 15 what's described in patients with hepatitis B that
16 develop renal failure.

17 Q. Tell me what his clinical -- what
18 specifically in his clinical course do you find to be
19 consistent with those folks who have hepatitis B that
09:26 20 go on to develop renal failure.

21 A. Well, he has chronic hepatitis B, number one.
22 And I don't see evidence that he has a -- well, he has
23 chronic hepatitis B. He develops the nephrotic
24 syndrome. Both of those well described in hepatitis B,
09:27 25 the nephrotic syndrome that is. And he's developed

1 renal failure. And I don't find that he has any
2 particular underlying disease that can explain that
3 renal failure, other than what I see from the hepatitis
4 B and from what's described his course is not usually
09:27 5 for a person with hepatitis B who develops that
6 problem.

7 Q. So if there were another underlying disease
8 that could be associated with the nephrotic syndrome
9 and the ultimate renal failure, your conclusion that
09:27 10 hepatitis B is the causative factor in this development
11 would be undermined, would you agree?

12 A. Yes, there are many diseases that can cause
13 glomerulonephritis and renal failure, from lupus to
14 Goodpasture's syndrome to many things, and post
09:27 15 streptococcal disease and so on. But I didn't see any
16 particular etiology in this case that seems reasonable,
17 other than the -- other than what I saw for
18 hepatitis B.

19 Q. And you've omitted the disease multiple
09:28 20 myeloma. And would you agree that if multiple myeloma
21 is present, that that would undermine your conclusion
22 that hepatitis B is causing the nephrotic syndrome and
23 the ultimate renal failure?

24 A. Well, yes and no. In other words, we know
09:28 25 that multiple myeloma can certainly cause renal

1 failure. Typically we see that in far advanced
2 myeloma, or we see it in people that have so-called
3 light chain myeloma and have huge amounts of light
4 chains in the urine.

09:28 5 Q. In light of that, what would be a huge amount
6 of light chains in the urine?

7 A. For example, 3 grams, in other words. I've
8 seen people with much higher than that level. But
9 let's say a substantial amount of light chain in the
09:29 10 urine and evidence -- other evidence of myeloma, such
11 as large numbers of atypical plasma cells in the bone
12 marrow, anemia, bone lesions, many other features of
13 myeloma.

14 Q. Would 3 grams of light chain be sufficient to
09:29 15 say that that may be causing the nephrotic syndrome and
16 the renal failure?

17 A. Yes. If I saw someone with myeloma and -- in
18 the first place, myeloma presenting as renal failure is
19 very uncommon. It's usually a late onset of illness in
09:29 20 people with myeloma. But if I had someone with
21 extremely heavy proteinuria with light chains, I would
22 certainly consider that as a possible cause of the
23 renal disease.

24 Q. Did you -- in reviewing the materials from
09:29 25 the various lab results, what amount of kappa light

1 chains were discovered by either the pathology or by
2 the blood work?

3 A. I don't believe that he had free light
4 chains. He had an IgG kappa molecule that was present
09:30 5 in the blood in small amounts. I think the largest
6 measurement was about 0.4 grams per DL, and he had the
7 same protein measured in the urine in very small
8 amounts. I don't believe he had any light chains
9 demonstrated.

09:30 10 Q. Look at the third page of the surgical path
11 report.

12 A. Yes.

13 Q. And in the next-to-last sentence which
14 begins, "There is a diffuse and punctuous [sic] stain
09:30 15 for IgG, paren, 3-plus, close paren, and kappa light
16 chain, paren, 3-plus, close paren, along the glomerular
17 capillary wall." Do you see that?

18 A. Yes.

19 Q. What's the significance of that finding?

09:30 20 A. Well, the significance of that is, when you
21 have a whole gammaglobulin molecule, you've got heavy
22 chains and light chains in it. So if you stain it with
23 an IgG stain, it's going to light up because you're
24 marking the heavy chain part of the molecule. When you
09:31 25 stain it with light chains, it's going to light up

1 because you're staining the light chain part of the
2 molecule. But it's one molecule.

3 Q. Where on the compendium of small, medium, or
4 large does "3-plus" on the pathology for the light
09:31 5 chain indicate to you? What does that indicate to you?

6 A. It indicates that there's strong staining for
7 the IgG kappa in this man's kidney.

8 Q. Does that indicate to you the amount of IgG
9 kappa that was found in light of the 3-plus finding as
09:31 10 far as quantification of small, medium, or large
11 amounts of light chain kappa?

12 A. It means that there's a substantial amount
13 that they see in the kidney. It doesn't tell you
14 anything about how much is actually in the urine or in
09:32 15 the blood. The kidney has a significant amount. And
16 if you assume that the most it could have would be
17 4-plus, 3-plus would be substantial.

18 Q. And how does that impact a determination of
19 whether -- strike that.

09:32 20 Does hepatitis B cause kappa light chain
21 deposition in the kidney itself?

22 A. It causes the whole molecule as we see here
23 to be deposited in the kidney. So if you stained it
24 with a light chain antisera, it would be positive just
09:32 25 like this is.

1 Q. So are you saying that the interpretation by
2 Dr. Picken of both heavy and light chain findings are
3 diagnostic of hepatitis B causing this problem?

4 A. I would say that they're consistent with it.

09:32 5 Q. Are they also consistent with the myeloma
6 causing it?

7 A. Yes. They could be consistent, yes,
8 depending on the clinical circumstances, if you knew
9 the patient had myeloma.

09:33 10 Q. Now, you seem to be critical of the
11 description by Dr. Picken of light and heavy chain
12 deposition disease in the second page of your report.

13 A. Yes.

14 Q. Might be confusing, I believe you call it,
09:33 15 and suggests separate deposits of both light and heavy
16 chains in the kidney?

17 A. Correct.

18 Q. Now, we've just talked about the third page
19 of Dr. Picken's report. And I believe it does -- her
09:33 20 staining at least shows both heavy and light chain
21 deposition, does it not?

22 A. It shows the whole molecule being deposited.

23 Q. Well, does that mean it's showing a light and
24 heavy chain?

09:33 25 A. Well, if you stain it for light chains, it

1 will show the light chain. If you stain it for heavy
2 chains, it will show the heavy chain. You've got one
3 molecule being stained.

4 Q. So how would you describe, with whatever
09:34 5 degree of specificity you would like, the findings that
6 are demonstrated by or described by Dr. Picken as
7 opposed to light and heavy chain deposition disease?

8 A. Well, I would simply say the patient has
9 glomerulonephritis with IgG kappa deposition, because
09:34 10 that's what she demonstrated.

11 Q. And is IgG kappa light or heavy?

12 A. It's neither. It's the whole molecule.

13 Q. All right.

14 A. When we talk about light or heavy, we're
09:34 15 talking about arms or legs.

16 Q. Okay. Explain that for me.

17 A. Well, it's very simple. Let's assume I come
18 to my office this morning, and I find a body lying on
19 the floor, and I call the police. If I said to the
09:34 20 police, "I have a pair of arms and legs on my floor,"
21 they would assume I had a dismembered body. And they
22 came there, and they found an intact body on the floor,
23 and they say, "Why did you say you have a pair of arms
24 and legs? You have a body." That's what we have here.

09:35 25 In the old speak -- and actually, I've

1 got some articles in here that show both old and new
2 use of definitions -- the way this was defined is, if
3 you had the whole molecule, it was called
4 immunoglobulin deposition disease, the whole
09:35 5 immunoglobulin. And then we had light chain disease if
6 you only had the light chain part. You had the light
7 chains amputated, if you will. Or you had heavy chain
8 disease. You had three possibilities: The whole
9 molecule or the light chain part or the heavy chain
09:35 10 part.

11 Well, this newer definition in some of
12 these articles that people use, what they've done is
13 they've taken -- instead of using the term
14 immunoglobulin deposition disease to represent the
09:35 15 whole molecule, they've used that to reference the
16 entire syndrome.

17 So in other words, they've got the same
18 three elements in there, but instead of calling it
19 immunoglobulin deposition disease, light chain disease,
09:36 20 and heavy chain disease, they call it light chain
21 disease, heavy chain disease, and light and heavy chain
22 disease; in other words, the whole molecule, because
23 it's got both light and heavy chains disease in it,
24 they use that very cumbersome definition of light and
09:36 25 heavy chain disease.

1 And if you look at the medical record,
2 it's caused intense confusion because some of the
3 doctors thought, well, there's some light chains in one
4 part, some heavy chains somewhere else. We got a
09:36 5 mixture of this and a mixture of that. And some of the
6 writings it says he's got light chain myeloma, which
7 has never been demonstrated.

8 So it causes tremendous confusion. And
9 in some of the articles that I have here, you can see,
09:36 10 even with the same authors on the article, they use the
11 same -- they use the old speak, and then in another
12 article they use the new speak in terms of this
13 definition. I don't like the new speak because I think
14 it's very cumbersome and confusing. And this chart is
09:37 15 a classic example of that.

16 Q. With all of that said, how does that impact
17 our attempt to determine what is causing the renal
18 failure?

19 A. Well, it simply tells us that there is a
09:37 20 gammaglobulin molecule stuck in the kidney. Now,
21 that's not uncommon at all. In many forms of
22 glomerulonephritis there frequently are gammaglobulin
23 molecules stuck in the kidney containing both the light
24 and heavy chain.

09:37 25 What's unusual in this case is that the

1 antibody is monoclonal. Typically, in most forms of
2 glomerulonephritis, the antibodies are what we call
3 polyclonal. There are many kinds of IgGs. Here it's
4 monoclonal, which is a little less common.

09:37 5 Nevertheless, as I pointed out in the
6 literature I referenced, monoclonal antibodies are seen
7 with some frequency in hepatitis B. They are seen in
8 some frequency with people who get renal failure who do
9 not have myeloma and do not have hepatitis B. But they
09:38 10 still have a monoclonal deposition of material in their
11 glomerular line and get renal failure.

12 So it's simply a type of classification
13 of renal disease, the fact that you have a monoclonal
14 protein.

09:38 15 Q. Let's compare -- I just want to understand
16 why this is in here. On page 2 of your report --

17 A. Yes.

18 Q. -- in the first paragraph, you have a
19 sentence that reads, about midway in that paragraph,
09:38 20 "Serum and urine proteins electrophoretic studies
21 failed to identify a monoclonal protein." Do you see
22 that?

23 A. I'm on page 2. Yes, because the original
24 studies of serum in urine that were done did not find a
09:39 25 monoclonal protein. They were subsequently found on

1 later studies. But the original reports didn't
2 identify them because they were in such small
3 concentrations.

4 Q. All right. Then look down at your next
09:39 5 paragraph, third sentence. "A small monoclonal
6 gammopathy of IgG kappa specificity was identified in
7 the serum."

8 A. Yes.

9 Q. That seems to be inconsistent. Are they
09:39 10 different tests?

11 A. They're different dates. I probably should
12 have put the date in. But the original testing that
13 was done on this man didn't show a monoclonal protein
14 in either the serum or the urine. But subsequent
09:39 15 testing, when he had more proteinuria and it was later
16 in the course of the illness, that's when they were
17 identified.

18 Q. Now, the reference that you're making for the
19 IgG kappa in the serum --

09:40 20 A. Yes.

21 Q. -- was that the same time the pathology was
22 done, or do you know?

23 A. I think that would be true, yes. I believe
24 that when the kidney biopsy was taken, the -- there had
09:40 25 been demonstration that there was IgG kappa. I may

1 have some of the lab values here. Let's see if I can
2 find that.

3 Q. I have a lab report dated -- it looks like
4 April 1, 2000, from SmithKline Beecham clinical labs.

09:40 5 A. Okay. That may be so. I'm just looking
6 back. I had pulled out a couple of them, but -- let me
7 just see what the earliest one I have is.

8 I have -- I guess I don't have that --
9 those early ones pulled out. But I believe that's
09:41 10 correct. At the time of the biopsy, the monoclonal
11 gammopathy was known in the serum and the urine.

12 Q. If hepatitis B were, in fact, causing the
13 renal failure, how would treatment be different, if it
14 would?

09:41 15 A. Well, this is not known with certainty, but
16 there -- the notion is that if you have hepatitis B and
17 you're starting to get renal insufficiency from it,
18 early treatment with Interferon or with Epivir might
19 prevent the progression of disease, because most people
09:42 20 with hepatitis B get a pretty good response in terms of
21 clearing of the virus with Epivir and Interferon.

22 And so the thought is that if you could
23 make that diagnosis early and treat the person early,
24 you might prevent the -- you might interrupt the
09:42 25 process and prevent the patient from going into

1 complete renal failure.

2 Q. Does hepatitis B progress?

3 A. Well, if it's chronic, it's quite variable.

4 There are some people who are chronic carriers, and
09:42 5 they keep having a viremia, but you just don't see much
6 tissue damage from it. And other people progress on to
7 cirrhosis.

8 Q. Other than the renal problems, is there any
9 way to determine whether the hepatitis B in Mr. Lebron
09:43 10 progressed?

11 A. Well, I would have to say yes in that at one
12 point he develops what could be called hepatic
13 encephalopathy. He develops very high ammonia levels
14 in the blood and confusion, and that's telling you that
09:43 15 the liver is not working very well, and that could
16 certainly be construed that he's got significant liver
17 disease.

18 Q. And does -- in his particular case, does the
19 development of hepatic encephalopathy indicate to you
09:43 20 that the underlying hepatitis B is causing that liver
21 problem?

22 A. No.

23 Q. Okay. What else could be causing the liver
24 problem?

09:43 25 A. Well, people get --

1 Q. What else could be causing his liver problem
2 so as to demonstrate hepatic encephalopathy?

3 A. Well, many things. If I were to, let's say,
4 look at our hospital population and look at the
09:44 5 diagnosis of the next hundred people that came in with
6 hepatic encephalopathy, a number would be alcoholics
7 with alcoholic cirrhosis. That would probably be the
8 number one category. It could be something like a
9 Tylenol overdose that caused liver damage.

09:44 10 There would be a lot of possibilities as
11 to why someone's liver wasn't functioning. It could be
12 chronic hepatitis C, HIV disease. Many illnesses can
13 affect the liver and cause liver dysfunction and you
14 could lead to encephalopathy.

09:44 15 Q. Does myeloma cause liver dysfunction?

16 A. Very, very rarely.

17 Q. Okay. But it can, correct?

18 A. I would have to say probably yes, but it
19 would be very, very unusual.

09:45 20 Q. How would myeloma cause liver problems -- let
21 me ask you this question. Let's assume that his
22 hepatic encephalopathy is the result of a
23 myeloma-induced liver problem. Can you postulate for
24 me how that would have occurred in Mr. Lebron?

09:45 25 A. Certainly, yes. Basically two ways. One,

1 you can get plasma cell infiltrates in the liver like
2 you could with any tumor. So livers can be infiltrated
3 with multiple myeloma cells and cause damage.

4 In addition, in certain patients with
09:45 5 myeloma, amyloid can develop, which is kind of a waxy
6 material that deposits itself in the liver and other
7 organs and can certainly cause major liver disease in
8 people with myeloma. Amyloid deposition is not
9 unusual.

09:46 10 You can see major liver disease in people
11 with myeloma from iron overload. If a patient is
12 chronically anemic and requires lots of transfusions,
13 the iron level can get very high in the liver, and that
14 actually occurs in people with many forms of hepatitis,
09:46 15 hepatitis C in particular.

16 Q. And if we assume that the hepatic
17 encephalopathy is the result of a hepatitis B-induced
18 liver problem, how would one look for that?

19 A. Well, it's a diagnosis that would be made by
09:46 20 finding evidence on a biopsy of chronic -- suggestive
21 of chronic viral hepatitis or viral-induced cirrhosis,
22 finding of circulating hepatitis B activity, and
23 evidence of abnormal liver function. Again, it's a
24 clinical diagnosis, as we frequently do in medicine.

09:47 25 Q. How far advanced does the hepatitis B need to

1 have gone so as to cause damage to the liver to the
2 extent that one experiences hepatic encephalopathy?

3 A. Well, I would think, in general, that you
4 would have to say -- you wouldn't have to have
09:47 5 cirrhosis, but you would have to have significant
6 damage to the liver.

7 Q. And in this particular case, has that been
8 demonstrated?

9 A. He has a single liver biopsy. And generally
09:47 10 speaking, the liver biopsies in terms of cirrhosis are
11 classified as to how much fibrosis that's in there.
12 And I think the scale that they're using goes up to 6.
13 And he's a 2 on fibrosis. So he does not have
14 cirrhosis, based on this liver biopsy.

09:47 15 Q. So he is in the lower third of the scale for
16 fibrosis of his liver, at least for this particular
17 laboratory, correct?

18 A. Well, keeping in mind that when you're doing
19 a liver biopsy, you're taking a tiny fragment out of
09:48 20 that liver, and what you hope is that it's
21 representative, and sometimes it's not representative.

22 You can put the needle into a
23 regenerating nodule of the liver, and it looks like
24 pure cirrhosis. You can stick that needle a few
09:48 25 centimeters adjacent to that, and it doesn't look too

1 bad. So there's always the potential for a sampling
2 problem when you're doing a liver biopsy. But this
3 biopsy, as it stands, does not confirm cirrhosis.

4 Q. Okay. Would that undermine your opinion that
09:48 5 the hepatic encephalopathy is the result of hepatitis B
6 causing liver damage?

7 A. Not at all, because the report says the
8 patient has chronic liver disease.

9 Q. Now, what report says that?

09:48 10 A. The liver biopsy says chronic -- I believe it
11 uses the term "chronic" in there. Let's see.

12 Q. Let's pull that out, if we could.

13 A. All right.

14 Q. Do you know where that's from?

09:49 15 A. I'm sure I have it here. Hang on. I've got
16 it right in front of me. It says, "Chronic hepatitis
17 B, grade 2, stage 2."

18 Q. What does that indicate to you, grade 2,
19 stage 2?

09:49 20 A. It just means that he's got significant liver
21 disease. In other words, he's got enough liver disease
22 where they can see it on the biopsy and quantitate it
23 in terms of, he's got a 3-plus lobular inflammation,
24 and he's got 2-plus fibrosis. I'm not a liver
09:49 25 pathologist, and I would defer to a liver pathologist

1 in terms of how bad this is, but it's obviously
2 recognizable that he's got significant liver disease.

3 Q. So are you saying that you really don't know
4 how severe the disease is, at least from reviewing the
09:49 5 liver biopsy?

6 A. Looking at this single biopsy, which, as I
7 said, may or may not be representative, all I can say
8 is, the biopsy supports chronic hepatitis B infection.

9 Q. Okay. So really what that says is that he
09:50 10 has -- had hepatitis B for a long time, doesn't it?

11 A. Well, we don't really know how long, but you
12 would have to guess probably at least a couple of years
13 to get this fibrosis in there. But again, I would
14 defer to a hepatic pathologist on that because when
09:50 15 you're looking -- usually in these people you get
16 serial biopsies, and you can look for progression, and
17 you go from point A to point B. Here we just have a
18 single biopsy, and I don't know that we could say with
19 any certainty exactly how long this disease has been
09:50 20 present.

21 Q. Well, if -- the nomenclature is "chronic," is
22 it not?

23 A. Correct.

24 Q. "Chronic," as I understand that term, means
09:50 25 long-standing?

1 A. Well, chronic probably means different things
2 to different people. In the case of hepatitis B,
3 generally, the term "chronic" refers to the fact that
4 if you can demonstrate that a persistent -- that you
09:51 5 have a persistent viremia for more than six months,
6 that's considered chronic hepatitis B.

7 But in this case, I would have to suspect
8 from the liver biopsy that it's been there a lot longer
9 than six months. But -- so "chronic" is depending on
09:51 10 how you would like to use the term.

11 Q. Can hepatitis B be secondary to the
12 development of myeloma?

13 A. Well, only if, for example, you got it
14 through blood transfusions, which would certainly be
09:51 15 unusual these days. But I've had patients who have
16 gotten a transfusion-related hepatitis who were
17 transfused because of anemias. I don't believe
18 Mr. Lebron had been transfused.

19 Q. Prior to the development of hepatitis B?

09:51 20 A. Correct.

21 Q. When, in your opinion, was hepatitis B first
22 diagnosed, not present, but first diagnosed?

23 A. I believe it was -- there was a positive --
24 10/4/2000 there's a positive test in here.

09:52 25 Q. So it was not until -- when did he

1 demonstrate the changes in his serum or blood which
2 demonstrate elevated proteinuria?

3 A. Late 1999 he had -- was seeing his
4 physician -- or complaining of foamy urine and had
09:52 5 proteinuria demonstrated.

6 Q. So it would be a year later he was diagnosed
7 with hepatitis B?

8 A. Yes, that's correct.

9 Q. Is it likely that his hepatitis B was present
09:52 10 in 1999?

11 A. I think, on the basis of this liver biopsy,
12 yes.

13 Q. And what's the date of your liver biopsy?

14 A. It's in 2001. Let's see if I can find that
09:53 15 again. It's 8/22/2001.

16 Q. That's a year after the hepatitis B antibody
17 is found, correct?

18 A. Correct.

19 Q. And it's two years after he starts spilling
09:53 20 protein into his urine?

21 A. Yes.

22 Q. Would you fault Dr. Barton for not treating
23 him with Interferon in 2000 once the hepatitis B was
24 diagnosed?

09:54 25 MR. CHILTON: Objection.

1 You can answer.

2 A. Well, I think the treatment with the Epivir
3 may have been started in 2000. I don't remember the
4 exact date it was started, but it -- it was toward the
09:54 5 end of the year in 2000, I believe, is when that
6 treatment was started.

7 Q. (By Mr. Hartley) And is Epivir the same as
8 Interferon?

9 A. No. Epivir is the lamivudine. I don't
09:54 10 remember exactly when he got the Interferon, but I
11 think he was getting the Epivir, which is an antiviral
12 drug, in the year 2000.

13 Q. Well, is Epivir more effective than
14 Interferon for the treatment of hepatitis B?

09:54 15 A. Well, Interferon has been the classic drug
16 that's been used. Epivir is the more recent drug. But
17 many people think it's a better drug. It's certainly
18 much more convenient to give because it's an oral drug.

19 Q. Would one over the other permit the
09:55 20 hepatitis B to progress to demonstrate grade 2
21 hepatitis in 2001?

22 A. Well, typically, Epivir works fairly
23 promptly, is what the literature suggests. And, in
24 fact, his -- the number of copies of the virus in the
09:55 25 blood went down fairly promptly once they started the

1 treatment. So the disease, at least in terms of viral
2 proliferation as you see in the blood, was controlled.

3 Q. Do you find it reasonable that he was treated
4 with chemotherapy -- VAD therapy that he received in
09:55 5 light of his disease process being the result, in your
6 opinion, of hepatitis B?

7 A. Well, I think if it was known earlier that he
8 had hepatitis B and that was a consideration, I
9 certainly would have treated him with Epivir first or
09:55 10 Interferon to see if that would slow or modify the
11 progression of the renal disease.

12 We certainly use chemotherapy in people
13 who have progressive renal disease to try to slow it
14 down, whether they have an underlying neoplasma or not.
09:56 15 We use it in a sense as an immunosuppressant technique.
16 But I would have given the first shot with antiviral
17 drugs.

18 Q. Was that done in this case?

19 A. Well, he had already had fairly advanced
09:56 20 disease by the time I think the Epivir was started.

21 Q. That's not my question. Was that done in
22 this case once the diagnosis was made?

23 A. No. Diagnosis of which?

24 Q. Hepatitis B.

09:56 25 A. Oh, once he had the diagnosis of hepatitis B

1 established, yes, I think the treatment was fairly
2 prompt.

3 Q. And what led to the need then for the VAD
4 treatment?

09:56 5 A. Well, the idea of the VAD treatment was to
6 prevent progression of renal disease.

7 Q. Did the treatment that Dr. Barton instituted
8 for the underlying hepatitis B put the hepatitis B in
9 remission?

09:57 10 A. Well, it reduced the viral load. I believe
11 it eventually went down to zero. It was something -- I
12 recall one lab report was about 28,000 copies. Then it
13 went to 14,000 copies. And then I think eventually it
14 went to zero. So you would have to say yes, the
09:57 15 therapy was effective at reducing the viral load.

16 Q. Did you try and chart the development of the
17 viral load throughout the course of his disease?

18 A. No.

19 Q. Have you looked at anything other than -- let
09:57 20 me ask you this. Strike that.

21 When did the viral load go to zero?

22 A. I can't tell you that as we sit here. I
23 would have to look back through the records.

24 Q. Take a peek, if you can. Do you think you
09:58 25 can find that for me?

1 A. Not very easily, because I've got a box full
2 of all of these -- a box full of all of these medical
3 records, and they've been looked at, and they're out of
4 order, and it might take a week. A lot of paperwork
09:58 5 here.

6 But what I do have are a couple of -- I
7 think I pulled out a couple of the readings that I
8 might have in front of me. Let me just see if I find
9 the dates on here that might get into that.

09:58 10 Well, here is one, for example, 7/31/01,
11 and the hepatitis particle level is down to 3,800.

12 Q. Is that -- how would you quantify that or
13 qualify that?

14 A. Well, I would say the earlier ones were
09:59 15 28,000 and 14,000. So obviously it's on the way down,
16 3,000.

17 Q. What does that mean as far as Mr. Lebron is
18 concerned, that we've now dropped the viral load from
19 28,000 down to 3,800?

09:59 20 A. Well, the theory is that chronic viremia is
21 essentially a marker for the liver being constantly
22 chewed on by the hepatitis B. And we reduced the
23 viremia. One would expect that the liver disease would
24 improve and wouldn't progress.

09:59 25 Q. So you've arrested the destruction of the

1 liver by lowering the viral load?

2 A. That's the theory. For example, January 30th
3 of '01 he had the 28,000 level. And so the theory with
4 both hepatitis B and C is that you -- you attempt to
10:00 5 lower the viral load with drugs. And in some instances
6 what happens in those illnesses is, the viral load will
7 go down. Almost always goes down. But in many cases
8 when you stop the drugs, it goes right back up. And
9 the objective is to try to get it down and keep it
10:00 10 down.

11 Q. Was that done in this case? Were they
12 successful in attempting to keep it down?

13 A. Well, it came down. I mean, I don't know
14 what his current viral load is. I think the last
10:00 15 records I have were in 2003. I don't know what his
16 current viral load is or even if he's still on the
17 Epivir or what his current situation is with regard to
18 the hepatitis B.

19 Q. Have you ever told Dr. Barton or have you
10:00 20 called Dr. Barton to discuss your theory that the
21 changes that have been demonstrated in this renal
22 failure, et cetera, may be the result of his hepatitis
23 B and not some light/heavy chain deposition disease?

24 A. No.

10:01 25 Q. Do you think that would help Dr. Barton in

1 understanding how to better treat this patient?

2 A. Well, not necessarily, because the cow is out
3 of the barn. In other words, he's got renal failure.
4 It's not going to get better. And so whatever one
10:01 5 thought might be the cause of that, it's too late to
6 fix it.

7 Q. Let's talk for a moment about the diagnosis
8 of myeloma in this particular case. As I understand
9 your report, the information that you have would not
10:01 10 lead you to make a diagnosis of multiple myeloma in
11 this particular case; is that fair?

12 A. That is fair.

13 Q. Now, in your report or attached to your
14 report would be the new criteria for the diagnosis of
10:02 15 plasma cell myeloma, correct?

16 A. I have the World -- if you're referring to
17 the World Health Organization article, I have that in
18 there.

19 Q. Is that table 106-2, the next-to-last page of
10:02 20 your -- or is it table 6.07? It's the last one.

21 A. Just a minute here. Yes. Table 6.07 is from
22 the World Health Organization.

23 Q. And how did the World Health Organization
24 come up with this diagnostic criteria? Do you know?

10:02 25 A. No.

1 Q. Okay. Were you consulted about it?

2 A. No.

3 Q. Were you a member of the criteria team?

4 A. No.

10:03 5 Q. Have you written any articles on multiple
6 myeloma?

7 A. Actually, I think I have. Case reports about
8 myeloma but not about criteria.

9 Q. What articles have you written about myeloma?

10:03 10 A. Well, I would have to look back at my CV and
11 let you know.

12 Q. Would you be kind enough to do that for me?

13 A. Certainly. Well, let's see if I can find
14 that.

10:04 15 Well, amyloidosis is a common -- follows
16 myeloma. And article 5 in my CV has to do
17 with amyloidosis of the eyebrows, and I believe -- it's
18 been a long time ago. We're talking about that paper.
19 I believe one of the patients in that paper had
10:04 20 multiple myeloma as a cause of their amyloidosis.

21 Q. Okay.

22 A. Then number 18 was a patient that also
23 had amyloidosis and had bilateral enlargement of the
24 lymph nodes in the chest. And --

10:04 25 Q. Is that myeloma bilateral enlarged lymph

1 nodes in the chest?

2 A. No. Amyloidosis -- in other words, this
3 patient had amyloidosis, I believe, as a component of
4 myeloma and developed very large lymph nodes in the
10:04 5 chest, and those lymph nodes -- again, it's a long time
6 ago. I think they had amyloid in the lymph nodes. So
7 it was sort of part of the myeloma complex, you might
8 say.

9 Let's see. Number 49 was extrahepatic
10:05 10 biliary obstruction in multiple myeloma. That was a
11 case where the multiple myeloma was blocking the bile
12 drainage from the liver. So that had to do with
13 myeloma.

14 Number 54, cardiac tamponade is the first
10:05 15 clinical feature of multiple myeloma. That had to do
16 with myeloma involving the pericardium, the sac
17 surrounding the heart.

18 That's probably -- well, yes, I think
19 that's probably the limit of my writings about myeloma.

10:06 20 Q. Consistent with what you testified in the
21 Cave case, would you agree with me then that the real
22 work was done on those articles by Dr. Sher in number
23 49 and Dr. Lee in number 54 as the last authors?

24 A. 49 and 54?

10:06 25 Q. Those are the two articles that you talked

1 about myeloma.

2 A. No. These are -- you see 49, for example,
3 Mike Seider was one of my interns, just like Dr. Ayoub
4 is here today. So this was my case, and I got him --
10:06 5 gave him the data, got him to do some work and write it
6 up. But I was the driving force behind writing that
7 paper, but I gave him the experience as an intern of
8 learning how to write a paper.

9 And the other one you mentioned --

10:06 10 Q. 54.

11 A. Yeah. That paper also was my writing. Larry
12 Stern, who's the first author, is a cardiologist here
13 at the hospital. It was his case, so I put his name
14 first, but I wrote the paper.

10:07 15 Chok Lee is a general internist in the
16 hospital. And you couldn't possibly read anything he
17 ever wrote. So I can tell you that he didn't write
18 anything to do with this paper.

19 Q. So that was -- so the fact that -- in the
10:07 20 Cave case, when you testified that the last author
21 usually carries the heavy load to do the writing of the
22 article, that doesn't apply at least to those two
23 particular citations that we just discussed, correct?

24 A. I don't remember exactly what I testified to
10:07 25 or what the question was. But sometimes the last

1 author is the prime mover. Sometimes it's the first
2 author. It really depends on the clinical
3 circumstances, or I should say the political
4 circumstances probably more than the clinical.

10:07 5 Q. Have you made any presentations on myeloma to
6 any professional organization?

7 A. I don't recall such as we sit here today.

8 Q. Have you ever been awarded a grant to study
9 myeloma?

10:08 10 A. No.

11 Q. Have you ever been asked to work for any
12 international foundation that has been studying
13 myeloma?

14 A. No.

10:08 15 Q. Can you give me a feel for what percentage of
16 your -- I guess the question is, do you treat myeloma
17 patients?

18 A. Yes.

19 Q. Can you give me a feel for what percentage of
10:08 20 your patient load involves patients with myeloma?

21 A. Well, I have a number of patients with
22 myeloma, and I've been seeing patients for 35 years, so
23 I've seen a large number of people with myeloma. In
24 terms of actual numbers, I would say that probably at
10:08 25 least once a week I see a patient with myeloma.

1 Now, that's not necessarily a new
2 patient, because the ones we treat frequently are here
3 in the office to get an infusion of Aredia or various
4 drugs that we use or to monitor their blood counts, so
10:09 5 I may be seeing the same one over and over again.

6 But I would say at least once a week I'm
7 seeing someone with multiple myeloma in the office.

8 Q. How many patients do you think you treat
9 presently, total, for myeloma in your practice?

10:09 10 A. Oh, it could be around 20 to 30.

11 Q. And how many patients do you actually -- do
12 you have in your total practice?

13 A. I can't give you a number on that because
14 many patients I might see once or twice a year. In
10:09 15 other words, if I treat someone for Hodgkin's disease
16 and they're in remission, I don't see them very
17 frequently, but they still periodically come back.

18 I see roughly about 40 patients a week in
19 the office. Some are new and some are old.

10:09 20 Q. So out of 40 patients, 1/40th of the patients
21 would be a myeloma patient, correct?

22 A. No, I didn't say that. I said at least once
23 a week I see a patient with myeloma. I might see two
24 or three. It depends on the phase of the moon, who's
10:10 25 on the office schedule. But I have a number of people

1 with myeloma that I see with some frequency.

2 Q. Out of the 40 patients you see a week, how
3 many on average are myeloma patients?

4 A. Probably two to three.

10:10 5 Q. Okay.

6 A. We're talking about soft data.

7 Q. I understand.

8 A. I would have to go look through the numbers.

9 Q. I'm just trying to get a feel for it. Do you
10:10 10 know of Dr. Brian Durie?

11 A. Only by his name. I recognize the name.

12 Q. Have you ever had any diagnostic criteria
13 named after you?

14 A. No.

10:10 15 Q. Would you agree that the individual who
16 creates the diagnostic criteria for a particular
17 disease probably understands best how he meant that
18 diagnostic criteria to apply?

19 A. I can't disagree with that. Presumably, the
10:10 20 person who set out criteria had an idea of why he set
21 them out.

22 Q. Would you agree with me that the IgG -- I
23 want to make sure I use your term. How did we come --
24 what did we come to a conclusion as to the actual
10:11 25 terminology that you wanted to use for Mr. Lebron's

1 disease?

2 A. I would say he has -- if I were writing that
3 pathology report, I would say he had a
4 glomerulonephritis with monoclonal immunoglobulin
10:11 5 deposition disease.

6 Q. And can that be a manifestation of multiple
7 myeloma?

8 A. Yes.

9 Q. All right. Did Mr. Lebron's bone marrow
10:11 10 demonstrate excess plasma cells?

11 A. He had 5 percent plasma cells in the bone
12 marrow. And generally we say about 1 to 3 percent,
13 rare articles will say up to 4.5, but 5 percent would
14 be considered to be a slight excess in plasma cells,
10:12 15 yes.

16 Q. Did his serum or urine contain monoclonal
17 protein?

18 A. Both his serum and urine contained the
19 monoclonal IgG kappa protein.

10:12 20 Q. And would you agree with me that the damage
21 to the kidney is consistent with a myeloma-related
22 organ or tissue impairment?

23 A. Well, it's more consistent with a
24 glomerulonephritis. And it's very hard when you see
10:12 25 deposition in a kidney like this to know which is the

1 chicken and which is the egg, in other words, whether
2 or not the immunoglobulin caused the damage or that the
3 damage was, let's say, set up, for example, by the
4 hepatitis causing -- calling forth an antibody response
10:13 5 and then the antibody sticks in the kidney.

6 In other words, it's very hard to know
7 when you're looking at a kidney biopsy like this the
8 actual mechanism, whether or not it was that
9 gammaglobulin that really did the deed or was that just
10:13 10 part of the whole issue.

11 Q. Well, I guess my question is, is the kidney
12 disease that's present in Mr. Lebron at least
13 consistent with gammaglobulin damage?

14 A. It's consistent with the immunoglobulin
10:13 15 deposition disease, yes, which may cause renal disease.

16 Q. Dr. Natelson, how many times have you
17 testified for Mr. Woolf?

18 A. Let me think about that one. I testified for
19 him in a case that had to do with acute leukemia in
10:14 20 Washington, D.C. Then the case you were involved in.
21 Then many years ago I testified in a case in Houston
22 that had to do with acute lymphoblastic leukemia, and
23 he was involved in that case, but he wasn't the one who
24 originally hired me. McCofski [phonetic] or something
10:14 25 like that was the name of that case. We're probably

1 talking 10 or 12 years ago. Those three come to mind.

2 Q. In any case where the plaintiff is alleging
3 that benzene has caused a blood disease, is it still
4 true that you've never testified for a plaintiff?

10:14 5 A. That would be correct, yes.

6 Q. Have you ever reviewed a case and found that
7 the blood disease was, in fact, caused by his alleged
8 exposure to benzene or any other hydrocarbon?

9 A. Yes. You're talking about a case for a
10:15 10 legal -- a case that I've seen or a case for a legal --

11 Q. A legal case, yeah.

12 A. Not a legal case.

13 Q. Okay. So any time a defense firm has come to
14 you with a hydrocarbon-exposed individual and have
10:15 15 asked you to review it, you've never found that the
16 hydrocarbon induced the blood disease; is that fair?

17 A. Well, I don't know if that's exactly fair
18 because I reviewed cases where I've given an opinion
19 and not been asked to write a report or testify.

10:15 20 Q. Well, have you -- you just told me that
21 you've never found in a legal case that the blood
22 disease was caused by the exposure to benzene or to
23 hydrocarbons, correct?

24 MR. CHILTON: Object. He testified --
10:16 25 he just testified that he never testified for -- to

1 that in a legal case. You're asking a different
2 question now. You're mischaracterizing his testimony,
3 Dean.

4 MR. HARTLEY: The record will speak for
10:16 5 itself, but I'll even change the question.

6 Q. (By Mr. Hartley) Have you ever found, when
7 asked by a defense firm, that the exposed individual
8 who had exposure to hydrocarbons, benzene, whatever it
9 might have been, developed his blood disease as a
10:16 10 result of that exposure?

11 A. No.

12 Q. Dr. Natelson, do you have what I would call
13 an emphasis in your practice for any particular
14 disease?

10:17 15 A. I would say no. I would consider myself a
16 general hematologist. Earlier in my career I was very
17 interested in blood coagulation disorders, and I
18 occasionally still see such patients, but I would say I
19 essentially do general hematology.

10:17 20 Q. Do you have a feel for what the majority of
21 your patients present to you with?

22 A. Just in rough terms, I would say probably
23 60 percent of the patients I see have some form of
24 malignancy, and lymphoma would be high on that list.
10:17 25 About 40 percent would have non-malignant disorders,

1 various bleeding problems, anemias, even
2 hepatitis-related issues if that affects the blood.
3 Quite a variety of things.

4 Q. How would you quantify from a percentage
10:18 5 basis the myeloma patients that you see?

6 A. Well, myeloma isn't a common disease. I
7 certainly see a lot more lymphoma patients than myeloma
8 patients. I would say, if looking at the
9 malignancies -- and at one point we were doing studies
10:18 10 on pancreatic cancer, and I saw a huge number of people
11 with pancreatic cancer, but we're not doing that at the
12 moment.

13 I would say today, if looking at the
14 malignancies I would see, I would have to say lymphoma
10:18 15 would be number one. Probably five times as many
16 lymphoma patients as I see myeloma patients, at least.

17 Q. How about AML?

18 A. I see patients with AML and I -- I don't have
19 huge numbers. Actively treating two at the moment, but
10:19 20 that's not a huge amount of my practice.

21 Q. What other malignancies do you treat other
22 than lymphoma -- and when you say lymphoma, are you
23 including NHL?

24 A. Yes. Hodgkin's disease, non-Hodgkin's
10:19 25 lymphoma, hairy cell leukemia. When you talked about

1 leukemia, I assume you meant acute myeloid leukemia. I
2 have a number of patients with hairy cell leukemia,
3 chronic lymphocytic leukemia. And people with chronic
4 lymphocytic leukemia live for many years, so you
10:19 5 gradually accumulate huge numbers of them. So I have a
6 large number of people with CLL I see.

7 Patients with myeloma we discussed. I
8 have some patients with myelofibrosis and myeloid
9 metaplasia, patients with polycythemia vera. What
10:20 10 else? Those would be some that come to mind as we
11 speak.

12 Q. I noticed on page 4 of your report in the
13 paragraph that begins, "I suspect."

14 A. Yes.

10:20 15 Q. In the middle -- about the middle of that
16 paragraph, you indicate that it, referencing back to
17 myeloma, had its peak instance around age 64 to 66.
18 What significance does it play for an individual who
19 has developed myeloma at a much earlier age, if
10:20 20 anything, in your causation analysis?

21 A. Not a lot. In other words, people who have
22 myeloma at an earlier age perform -- other than, let's
23 say, age-related complications, the pace of the disease
24 is not a great deal different in the young person as
10:21 25 opposed to the older person with myeloma.

1 Q. Does it give you any indication as to the
2 cause of the myeloma if it's at an earlier age?

3 A. No.

4 Q. And the last -- next-to-last line of that
10:21 5 paragraph you talk about the fact that median survival
6 after diagnosis of myeloma remains about three to four
7 years, although some patients have an indolent course
8 and live longer than ten years. Is that still true
9 today?

10:21 10 A. That's true today, yes.

11 Q. What does it indicate if the individual is
12 able to survive the disease process longer than ten
13 years to you, if anything?

14 A. Well, I guess good luck. In other words, I
10:21 15 have -- I've had people live 15 years with myeloma.
16 Now, at that time they were usually in renal failure,
17 had numerous fractures, chronic anemia, looked
18 miserable, but they did manage to live 15 years. Most
19 people that have had myeloma for, let's say, ten years
10:22 20 have had considerable disability.

21 Q. The article number 12 that you reference in
22 your report by Perez --

23 A. Let me find number 12. Yes.

24 Q. Do you have that with you?

10:22 25 A. Yes.

1 Q. Pull it out for me.

2 A. I've got it.

3 Q. Is this article -- it looks like it's a
4 letter to the editor?

10:22 5 A. Yes.

6 Q. Is this in Spanish?

7 A. Yes.

8 Q. Do you read Spanish?

9 A. Scientific Spanish, yes. It's basically like
10:22 10 reading English. The -- what I generally do is, I look
11 at it, and I have -- we have several Spanish-speaking
12 people in our office, and I get them to translate for
13 me if there's a sentence or two I don't understand.

14 Q. Second paragraph in the letter to the editor,
10:23 15 there's a Thomas M. Pesiente [phonetic]. I assume that
16 that is a case report of one patient, correct?

17 A. Yes. We present a patient with hepatitis
18 through virus B, hepatitis B, intense
19 hypergammaglobulinemia and the appearance of what are
10:23 20 called oligoclonal. It's another term for small
21 monoclonal bands.

22 Q. Look at number 14.

23 A. Yes.

24 Q. Do you speak Russian?

10:23 25 A. No.

1 Q. Do you have someone translate Russian for
2 you?

3 A. Yes.

4 Q. Have you translated number 14?

10:23 5 A. What I did with this article, to be specific,
6 is it has an abstract that's in English.

7 Q. Correct.

8 A. And then I gave this article to a Dr. Luda
9 Caminisky, who's Russian who's an internist in this
10:24 10 building. She, of course, speaks fluent Russian, and I
11 asked her particular questions about this article, and
12 I've outlined those, in other words, in what I sent
13 you. I had four questions, and then she gave me the
14 answers to what I wanted to know. And she looked
10:24 15 through the article in detail.

16 Q. And that's the material that you sent to
17 Mr. Woolf. I think it was via e-mail, wasn't it?
18 Wasn't it via e-mail, or was it hard copy?

19 A. No, no. I'm confident I wouldn't have sent
10:24 20 this by e-mail because it's my handwritten notes and
21 her handwritten notes. If I sent this to him, I would
22 have probably mailed it.

23 Q. Okay. What did you bring with you today?

24 A. Well, everything you asked for. I've got my
10:24 25 CV. I've got billing records. I've got the list of

1 cases I've previously testified in. I've got all the
2 articles I referenced in the letter and a couple of
3 other ones that I haven't referenced, then the medical
4 records that are here. That's generally the bulk of
10:25 5 what I've got here.

6 Q. Did you say you brought some handwritten
7 notes --

8 A. Well, that's in this -- talking specifically
9 about this article.

10:25 10 Q. When we started out, you indicated that there
11 was something you sent to Mr. Woolf after February 15th
12 of 2004.

13 A. Oh, yes, that's correct. And you said you
14 had that. That's the supplemental report on the --
10:25 15 comparing the biopsy language with the textbook
16 language about glomerulonephritis. And yes, you're
17 correct, that was e-mailed to him. That was
18 typewritten. That wasn't handwritten.

19 Q. Okay. That's what I was remembering to be
10:25 20 typewritten.

21 A. Yes.

22 Q. Did you bring that with you?

23 A. Yes, that's here.

24 Q. All right. Let's make that an attachment,
10:25 25 because I can't find mine.

1 A. Okay.

2 (Exhibit 1 marked for identification.)

3 Q. (By Mr. Hartley) Doctor, let me show you
4 what's been marked as Exhibit 1. Can you identify that
10:26 5 for me?

6 A. Yes.

7 Q. What is it?

8 A. Well, it's entitled, "Glomerulonephritis
9 Descriptions." And what it amounts to is that I've
10:26 10 abstracted a statement from a well-known nephrology
11 text on glomerulonephritis, showing the language that
12 the textbook uses and then below it the language that
13 the Lebron pathology report uses. And my reason for
14 doing this is to demonstrate that the report tells us
10:27 15 that this patient had glomerulonephritis.

16 Q. And what significance does that play in your
17 evaluation of this case?

18 A. Well, I think that's the etiology of the
19 nephrotic syndrome in the patient; is a form of
10:27 20 glomerulonephritis in this patient.

21 Q. Now, we talked about some handwritten notes a
22 little earlier. Do you have those?

23 A. Yes.

24 MR. HARTLEY: Can we mark those, Debbie,
10:27 25 as Number 2?

1 THE REPORTER: Sure.

2 (Exhibit 2 marked for identification.)

3 MR. CHILTON: Dean, I'm sorry. On
4 Exhibit 1, did you want just the Glomerulonephritis
10:27 5 Descriptions marked, or did you want the cover e-mail
6 marked also that Lou forwarded on to you with that?

7 MR. HARTLEY: Let's do them both. Let's
8 call them Number 1 both. We'll just staple them
9 together.

10:28 10 MR. CHILTON: That's fine.

11 THE WITNESS: The other thing he's
12 asking for are these handwritten notes. They're part
13 of this article, are stapled to this article.

14 Q. (By Mr. Hartley) How many pages is that?

10:28 15 A. It's two pages.

16 MR. CHILTON: Dean, I'm just having her
17 re-mark Exhibit 1 so it's the full cover sheet and
18 references, et cetera, that Lou forwarded to you as a
19 supplemental report. Okay?

10:28 20 MR. HARTLEY: That's fine.

21 MR. GILMARTIN: So then Exhibit 1 is a
22 three-page document made up of an e-mail, the
23 Glomerulonephritis Descriptions he talked about, and
24 the supplemental references to report page, correct?

10:29 25 MR. CHILTON: It's actually a five-page

1 group exhibit that also has the fax cover sheet and
2 cover letter from Lou Woolf to Dean.

3 (Exhibit 1 re-marked for identification.)

4 Q. (By Mr. Hartley) Let me show you,
10:30 5 Dr. Natelson, what's been marked as Exhibit Number 2.
6 Can you identify that for me?

7 A. Yes. I've got them over here.

8 Q. What is it?

9 A. Well, it's four questions I asked
10:30 10 Dr. Caminsky to specifically pay attention to when she
11 read this article in Russian and to give me a response
12 back. And so the first page is my questions to her,
13 and the second page is in her handwriting. It's her
14 response to my questions.

10:31 15 Q. All right. Fine. How many pages of billing
16 records do you have?

17 A. Let me see. There are five pages of billing
18 records in here.

19 MR. HARTLEY: Would you mark that,
10:31 20 Debbie, as Number 3?

21 THE REPORTER: Sure.

22 (Exhibit 3 marked for identification.)

23 Q. (By Mr. Hartley) Can you identify for me,
24 Doctor, what is Exhibit Number 3?

10:31 25 A. Exhibit Number 3 are the billing records from

1 this case.

2 Q. How many hours do you have in this case?

3 A. I would have to sum that up. Let me just --
4 my wife has done that for me, I see. That's great.

10:32 5 22-and-a-quarter hours she's got written here.

6 Q. What do you bill an hour now?

7 A. I believe it's something like \$250 an hour
8 for review of records and \$450 an hour for testimony.

9 Q. Has your CV been updated since April 15th,
10:32 10 2002?

11 A. Not substantially in any particular way. I'm
12 trying to think if there's anything --

13 Q. I have 72 articles written as of 2002. Have
14 you written anything since April of 2002 that's been
10:33 15 published?

16 A. No.

17 Q. Have you published any abstracts since April
18 2002?

19 A. Not medical abstracts.

10:33 20 Q. All right. Any chapters in any books?

21 A. No.

22 Q. Any teaching video cassettes?

23 A. No.

24 Q. Any patents that you've obtained since 2002?

10:33 25 A. No.

1 Q. Are you still teaching at Baylor?

2 A. Very rarely. I'm still on clinical faculty,
3 but I don't teach there to any degree.

4 Q. Do you give any lectures at any medical
10:33 5 school presently?

6 A. Yes. I gave a lecture about a week ago at
7 the University of Texas Medical School.

8 Q. What was that on?

9 A. Myelodysplasia.

10:33 10 Q. Have you ever given any lecture at a medical
11 school on myeloma?

12 A. Probably years ago when I was at Baylor, but
13 certainly not in recent years.

14 MR. HARTLEY: Just to clear up the
10:34 15 Exhibit Number 1, Mr. Chilton, how many pages does
16 Exhibit Number 1 now have?

17 MR. CHILTON: Five, Dean. What he
18 originally marked as Exhibit 1 was one page. That's
19 page 3 of the group Exhibit 1 now.

10:34 20 Q. (By Mr. Hartley) All right. You said you
21 brought with you a couple articles that were not listed
22 in your report?

23 A. Yes.

24 Q. What would they be?

10:34 25 A. There are two articles that have to do with

1 the use of Epivir or lamivudine in patients with
2 chronic hepatitis B and liver disease.

3 MR. HARTLEY: Can we mark those then as
4 4 and 5?

5 (Exhibit 4 and Exhibit 5 marked for
6 identification.)

7 A. Then I have an article on -- it's called
8 "Lymphohaematopoietic cancer risk among chemical
9 workers exposed to benzene." It's a 2004 article.

10:35 10 Q. (By Mr. Hartley) Who is the first author?

11 A. Bloeman, B-L-O-E-M-A-N.

12 MR. HARTLEY: Let's make that as
13 Number 6.

14 (Exhibit 6 marked for identification.)

10:35 15 A. Then I have a recent article from Otto Wong
16 entitled, "Is there a causal relationship between
17 exposure to diesel exhaust and multiple myeloma?"

18 Q. (By Mr. Hartley) What impact does that have
19 in Mr. Lebron's case?

10:36 20 A. Possibly none. Just in terms of the question
21 of causation of multiple myeloma.

22 Q. Okay. Anything else?

23 A. An article on -- entitled, "Advances in
24 biology of multiple myeloma: Clinical applications."
10:36 25 It's from 2004 in the journal Blood.

1 Q. Who is the author of that?

2 A. H-I-D-E-S-H-I-M-A is the first named author.

3 MR. HARTLEY: Let's attach that as
4 Exhibit Number 7.

10:37 5 (Exhibit 7 marked for identification.)

6 A. Then another Wong article entitled, "An
7 updated mortality study of workers at a petroleum
8 refinery in Beaumont, Texas, 1945 to 1996."

9 Q. (By Mr. Hartley) I don't need that one.

10:37 10 A. Okay. Then I wasn't sure that you had -- I
11 think you have this World Health Authority article. Do
12 they -- that wasn't with my original letter.

13 Q. I do have it. Let's mark that, because I
14 always have a hard time finding my copy.

10:37 15 A. Okay. Now I'm having trouble finding mine.
16 Let me just see where it is.

17 MR. CHILTON: That's the one that was
18 produced by Durie at his deposition.

19 MR. HARTLEY: I think it is.

10:37 20 Q. (By Mr. Hartley) It's from where the table
21 that is attached to his report comes from, isn't it,
22 Doctor? It's either from IARC or from Blood. Maybe
23 I'm wrong.

24 A. We just had it out here about five minutes
10:38 25 ago, and now I can't find it.

1 THE WITNESS: Did you personally pick it
2 up, along with the article?

3 MR. CHILTON: I don't believe so.

4 THE WITNESS: Do you want to look back
10:38 5 on the desk in the cafeteria?

6 I just had it out, I think, before we
7 started. At the moment, I can't put my finger on it.

8 Q. (By Mr. Hartley) That article seems to
9 disappear for me too. I don't know where it goes. I
10:39 10 never can put my finger on it.

11 A. It's becoming a real chronic problem for me.
12 Hopefully it's in this other room here, because we were
13 just looking at it.

14 Q. Let's say that's going to be Number 8. If
10:39 15 you can't find it today, we can always supplement the
16 record and call it Number 8.

17 MR. HARTLEY: Is that all right, Larry?

18 MR. CHILTON: That's fine. I think what
19 we're referring to -- are we talking about the one
10:39 20 that's edited by Jaffe?

21 MR. HARTLEY: I think so.

22 THE WITNESS: Yes.

23 MR. CHILTON: Entitled, "Pathology and
24 Genetics" and was put out by the World Health
10:39 25 Organization.

1 MR. HARTLEY: Right.

2 MR. CHILTON: That was produced by Durie
3 at his deposition.

4 MR. HARTLEY: Okay.

10:39 5 MR. CHILTON: That's where it comes
6 from.

7 (Exhibit 8 marked for identification.)

8 Q. (By Mr. Hartley) Is there anything else you
9 have, Dr. Natelson?

10:39 10 A. I think that's about it.

11 MR. CHILTON: Dean, just so the record
12 is clear, that prior report, the supplemental report
13 that Lou sent to you, one of the pages of that, I
14 believe, was an additional four articles as an
10:40 15 attachment.

16 MR. HARTLEY: Right.

17 MR. CHILTON: So you would have had
18 that, but just so it's clear.

19 MR. HARTLEY: Okay. That's all I have,
10:40 20 Dr. Natelson. I don't know if anybody else has any
21 questions or not.

22 **EXAMINATION**

23 **BY MR. GILMARTIN:**

24 Q. This is Bob Gilmartin. I've got one
10:40 25 question. Can you spell Epivir for me?

1 A. E-P-I-V-I-R, I believe. The actual chemical
2 name of the drug is something close to lamivudine. I
3 always have trouble pronouncing it. It's
4 L-A-M-I-V-U-D-I-N-E, but it's also known as Epivir.

10:40 5 Q. E-P-I-V-I-R?

6 A. Yes, I believe that's the spelling of it.

7 MR. GILMARTIN: Thank you. That's all I
8 have.

9 MR. CHILTON: Anybody else?

10:40 10 MR. HARTLEY: I guess that's it.

11 (Off the record at 10:40 a.m.)
12
13
14
15
16
17
18
19
20
21
22
23
24
25

ORAL DEPOSITION OF
ETHAN A. NATELSON, M.D.
OCTOBER 27, 2004

CHANGES AND SIGNATURE

	PAGE	LINE	CHANGE	REASON
1				
2				
3				
4				
5				
6				
7				
8				
9				
10				
11				
12				
13				
14				
15				
16				
17				
18				
19				
20				
21				
22				
23				
24				
25				

CHANGES AND SIGNATURE
ETHAN A. NATELSON, M.D.
 Page 2

PAGE	LINE	CHANGE	REASON
-----	-----	-----	-----
-----	-----	-----	-----
-----	-----	-----	-----
-----	-----	-----	-----
-----	-----	-----	-----
-----	-----	-----	-----
-----	-----	-----	-----
-----	-----	-----	-----

I, ETHAN A. NATELSON, M.D., 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.

THE STATE OF _____:
 COUNTY OF _____:

BEFORE ME, _____, on this day personally appeared **ETHAN A. NATELSON, M.D.**, known to me or proved to me on the oath of _____ or through _____ [description of identity card or other document] to be the person whose name is subscribed to the foregoing instrument and acknowledged to me that he/she executed the same for the purposes and consideration therein expressed.

Given under my hand and seal of office this _____ day of _____, 2004.

 Notary Public in and for the
 State of _____
 My commission expires: _____

1 THE STATE OF TEXAS :

2 COUNTY OF HARRIS :

3

4 I, Debbie Leonard, CSR, RMR, CRR, a Certified
5 Shorthand Reporter in and for the State of Texas, do
6 hereby certify that the facts as stated by me in the
7 caption hereto are true; that the above and foregoing
8 answers of the witness, **ETHAN A. NATELSON, M.D.**, to the
9 interrogatories as indicated were made before me by the
said witness after being first duly sworn to testify
the truth, and same were reduced to typewriting under
my direction; that the above and foregoing deposition
as set forth in typewriting is a full, true, and
correct transcript of the proceedings had at the time
of taking of said deposition.

10 I further certify that I am not, in any
11 capacity, a regular employee of the party in whose
12 behalf this deposition is taken, nor in the regular
employ of his attorney; and I certify that I am not
interested in the cause, nor of kin or counsel to
either of the parties.

13

Certified by me on this 4th day of November, 2004.

14

15

16



ORIGINAL

17

Debbie Leonard, CSR, RMR, CRR

Texas CSR No.: 8190

Expiration Date: 12/31/05

18

Allied Advanced Reporting, Inc.

19

Firm Registration No. 252

1647 Colquitt Street

20

Houston, Texas 77002

Phone: (713) 524-6777

21

Fax: (713) 524-6888

22

23

24

25

[illegible]