CAUSE NO. 21916*JG02

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| DANIEL T. MINNEHAN, Individually and as Next Friend for Joshua Stephen Minnehan and Kyle Marshal Minnehan<br><br>Plaintiff, | ) | IN THE DISTRICT COURT OF |
| vs. | ) | BRAZORIA COUNTY, TEXAS |
| AMERICAN OIL COMPANY, Individually and f/k/a Amoco Oil Company and Amoco, Inc.; et al.,<br><br>Defendants. | ) | 239th JUDICIAL DISTRICT |

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ORAL DEPOSITION OF
ETHAN NATELSON, MD
OCTOBER 15, 2004

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ORAL AND DEPOSITION of ETHAN NATELSON, MD, produced as a witness at the instance of the Plaintiff, and duly sworn, was taken in the above-styled and numbered cause on October 15th, 2004, from 11:10 a.m. to 12:40 p.m., before Susan A. Love, Certified Shorthand Reporter in and for the State of Texas, reported by computerized stenotype machine at the Stehlin de Ipolyi Oncology Clinic, P.A., 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.

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# I N D E X



# E X H I B I T S

| EXHIBIT NO. | DESCRIPTION | PAGE |
|---|---|---|
| 1 | Black Binder of Articles Produced by Dr. Natelson | 58 |
| 2 | Handwritten Notes by Dr. Natelson and Pages from Daniel Minnehan's Medical Records | 58 |

ETHAN NATELSON, MD,

having been first duly sworn, testified as follows:

EXAMINATION

BY MR. KAESKE:

Q. Hi, Doctor.

A. Good morning.

Q. What kind of doctor are you?

A. I'm a hematologist.

Q. All right. Have you ever diagnosed anyone with benzene-related acute myelogenous leukemia?

A. I've diagnosed a person with chemical related. I don't know that it was benzene. He was exposed to many different chemicals. But specifically benzene related, no.

Q. All right. So just so I can get a clear question and answer: As far as you can recall, you've never diagnosed anyone with benzene-related acute myelogenous leukemia, correct?

A. That would be correct.

Q. Have you ever treated anyone with benzene-related acute myelogenous leukemia?

A. Again, I've treated one patient that I can think of who had exposure to benzene, in addition to many other chemicals. I don't know that his was benzene related. It was chemical related. But I haven't treated anybody that

I thought had only benzene and only exposure to benzene-induced leukemia.

Q. So as far as you can recall as you sit here today, you've never treated anyone with benzene-related acute myelogenous leukemia, correct?

A. Yes, that would be correct.

Q. All right. How long have you been practicing in the Houston area?

A. I started doing hematology about 35 years ago. But in terms of practice, I began to practice when I got out of the Air Force, which would be around 1972.

Q. How many acute myelogenous leukemias do you think you've diagnosed since 1972?

A. I would have to speculate. In other words, I don't count them, but I would say probably in the range of -- and this would be real speculation. I would say perhaps four to six a year for all of those years. We'd have to do some calculations.

Q. And do you think that the first year that you saw an acute myelogenous leukemia patient in practice was in 1972?

A. Yes, because that's when I went into practice.

Q. When you diagnose someone with -- I presume you're intending to give opinions about the causation of Mr. Minnehan's leukemia; is that correct?

A. Yes.
Q. When you -- and I also presume that you're going to opine that it's not related to benzene exposure, correct?
A. I'm going to opine that I don't know the cause for it.
Q. Well, are you going to say that it's not related to benzene exposure?
A. In all medical probability, yes, that it's not related to benzene exposure.
Q. I guess you're going to give the jury the opinion that, in your opinion, Mr. Minnehan's acute myelogenous leukemia is not related to any exposure that he had to benzene, correct?
A. I'm going to give what I would say in all medical probability, in any particular case of acute leukemia, it's very difficult to, with certainty, assign a cause. So all one can say is, looking at the case, the milieu it was diagnosed in, the type of leukemia, the circumstances, you can give your best estimate if it was or wasn't. And in my estimate, it would be in all medical probability, it is not caused by benzene.
MR. KAESKE: Object as nonresponsive.
Q. (BY MR. KAESKE) And that's just for her.
A. I understand.

Q. It's not for you.

But I'm just trying to find out if one of the things that you're going to say to the jury is you think his acute myelogenous leukemia was not caused by exposure to benzene.

A. I don't believe that it was.

Q. And you don't know what caused his acute myelogenous leukemia, correct?

A. That's correct.

Q. You are not going to give the opinion that smoking caused Mr. Minnehan's acute myelogenous leukemia, correct?

A. That's correct.

Q. List for me, if you would, all the reasons you believe -- well, let's start with this: You do a differential diagnosis with your own patients, correct --

A. Yes.

Q. -- outside of litigation? When you see a patient, you'll do a differential diagnosis, correct?

A. Yes.

Q. And part of the purpose in doing a differential diagnosis is to not only diagnose what the disease is but also to figure out if there's a cause, correct?

A. Yes.

Q. And what you will do to make a differential

diagnosis in one of your patients is you'll first ask yourself, what is the disease that the patient has, correct?

A. Correct.

Q. And then the next thing that you will do is you will ask whether or not there are any confounding factors or anything in the background of the individual that might explain the illness, correct?

A. Correct.

Q. And then the third thing you do is you'll ask yourself, in a case like this one, what's the allegation and is it reasonable, correct?

A. Correct.

Q. All right. Now, as far as the first question is concerned, what is the disease, you don't have any dispute that Mr. Minnehan has acute myelogenous leukemia, correct?

A. Correct. He has acute myeloid leukemia, or what we call AML.

Q. And in addition to that, you would agree that there is ample epidemiologic evidence, as well as other scientific evidence, in the form of case reports and scientific studies of the mechanistic methods of benzene-inducing acute myelogenous leukemia that show that benzene does, indeed, cause acute myelogenous leukemia,

right?

A. It is generally accepted that, under certain conditions, benzene may cause AML.

MR. KAESKE: Object as nonresponsive.

Q. (BY MR. KAESKE) Sorry. My question was really bad. Let me try again.

You will agree with me, won't you, that there is ample scientific evidence showing that benzene causes acute myelogenous leukemia, correct?

A. Benzene has the capacity to cause acute myelogenous leukemia.

Q. All right. Now, as far as any confounding factors are concerned with respect to Mr. Minnehan's disease, and in particular, Mr. Minnehan's case, there are no factors that you would say -- that you can look to as history and say, "These are the things that I think caused his acute myelogenous leukemia," outside of benzene, correct?

A. Well, as I said, I don't know the cause of his acute myeloid leukemia.

Q. Well, there are no confounding -- if you're doing this differential diagnosis that you would do with one of your own patients, you wouldn't come upon any confounding factors for the cause of his acute myelogenous leukemia, correct?

A. Well, just as we've said, we recognize that benzene can cause acute myeloid leukemia under certain conditions. Cigarette smoking is a risk factor for acute myeloid leukemia; and he had smoked, at least, at one time in his life.

Q. Well, look, you said cigarette smoking is a risk factor for acute myeloid leukemia, right?

A. Correct.

Q. Now, as far as risk factors are concerned, you have previously testified that in order for you to be comfortable with an epidemiologic study or with causation between a substance and a disease, you need to see a three-times risk, correct?

A. That's correct.

Q. And you will agree with me that there are no studies published in the peer-reviewed scientific literature that show a three-times risk for smoking and acute myelogenous leukemia, right?

A. No, I would disagree with that. In other words, we generally believe that -- or the Surgeon General's report tells us there's a doubling of the risk. But there are studies that show a three-times increase in risk.

MR. KAESKE: Object as nonresponsive.

Q. (BY MR. KAESKE) Doctor, do you have the Surgeon General's report with you?

A. Yes, or part of it.
Q. And you're convinced that the Surgeon General's report says that there's a doubling of the risk for smoking and acute myelogenous leukemia?
A. Well, I have the summary pages of that. We can look at it and see just what they say. He cites a lot of studies, and the conclusion is that cigarette smoking is a risk factor. I don't recall if it says doubling. But that conclusion is that cigarettes are a risk factor.
MR. KAESKE: Object as nonresponsive.
Q. (BY MR. KAESKE) And I don't mean to niggle with you here. But, look, you just told me that the Surgeon General's report says that there's a doubling of the risk; and we were talking about whether or not there are any studies that show a tripling of the risk, or three-times risk.
A. Yes.
Q. And so when I said the three-times risk, you said yes, there was a study. And then you pointed me to the Surgeon General's report, which you said was a doubling. So let's deal with that first. Do you believe -- because apparently the Surgeon General's report forms a portion of the basis of your opinion that smoking can increase someone's risk for acute myelogenous leukemia, correct?
MR. DILLARD: Object to form.

A. Yes. The Surgeon General's report, I suppose you could say, validated what we know from the general opinions in the medical literature.

MR. KAESKE: Object as nonresponsive.

Q. (BY MR. KAESKE) The question was: Does the Surgeon General's report form part of the basis of your opinion that smoking is a risk factor for acute myelogenous leukemia?

A. Yes.

Q. All right. Now, that report doesn't say anywhere in it that there's a doubling of the risk of AML from smoking, correct?

A. We'd have to look at it. I don't recall. It's a long report. I just have the conclusions. We could look at them -- they're in this book -- and see exactly what they are. But it indicates the risk has increased.

Q. Fair enough. And we should look at it.

A. Yes.

Q. But a moment ago when you told us that it showed that there was a doubling of the risk, upon further reflection, you're not sure without looking at it whether or not there's a doubling of the risk. Is that fair?

A. That would be fair.

Q. Okay. So let's go ahead and pull it out, if you've got it with you, and take a look at it and see what

it says.

A. The final pages cite a number of reports that show varying risk benefits. And he says here: "Among those who smoked more than a pack of cigarettes per day, the risk increased twofold. In 2002 IARC concluded that there's now sufficient evidence for a causal association between cigarette smoking and myeloid leukemia."

And then under "Final Conclusions: "The evidence is sufficient to infer a causal relationship between the smoking and acute myeloid leukemia. And, too, the risk for acute myeloid leukemia increases with the number of cigarettes smoked and with duration of smoking."

MR. KAESKE: Object to the nonresponsive.

Q. (BY MR. KAESKE) Doctor, my question specifically is: Does the Surgeon General's report say that there is a doubling of the risk for acute myelogenous leukemia, not all leukemias, not all myeloid leukemias, but for acute myelogenous leukemia with smoking?

A. Well, as I said, among those who smoked more than a pack of cigarettes per day, the risk increased twofold. That's doubling, isn't it?

Q. The risk of what, Doctor?

A. The risk of acute leukemia is what they're talking about.

Q. Can I take a look at that for just a second?

Maybe I'm --

A. (Passes document.)

Q. Can you cite me to an epidemiologic study that shows a doubling of the risk for acute myelogenous leukemia and smoking?

A. This one. This is an article by Pascoletti from 1997. And according to the table in this for acute leukemia, he says that for all smokers the O.R. is 2.3; for light smokers, 2.25; and for heavy smokers, 2.30.

Q. All right. Can I take a look at that?

A. (Passes document.)

Q. And this is the article from the British Journal of Hematology for Pascoletti?

A. Yes.

Q. Now, can you cite me any articles that show a three-times increased risk for smoking and acute myelogenous leukemia?

A. Isn't that what I just showed you? The risk is more than three.

Q. I'm asking you a different question now.

A. I'm sorry.

Q. Give me whatever studies you think answer the question, including that one if you think that's one of them.

A. I've given you that study. I have many, many

articles in my files, but I -- and the Surgeon General cites a few, I believe, that increased the risk above three; but I don't have them as we talk here today.

Q. What I'd like for you to do for me, if you could, please, sir, is name for me the articles that you can today that show a tripling of the risk for smoking and acute myelogenous leukemia.

A. Let's just see here (perusing documents). Well, I can't as we sit here today. I don't have the entirety of the Surgeon General's report, and I don't recall any specific article that would say that.

Q. All right. The binder that's on the table in front of you, those are articles that you have pulled specifically for this case; or is that just your benzene file, in general?

A. No. That is specific articles for this case.

Q. All right. Fair enough.

And where did you get those articles?

A. Well, these are all articles that I would accumulate, as I accumulate many references in my career. In other words, these references come from various sources. They may be journals that I subscribe to, like Blood or The New England Journal of Medicine. They may be articles that I've ordered through our library here in the hospital. They may come from various sources.

Q. I guess what I'm getting at is: How did you come upon this particular set of articles?

A. Well, the particular set that I selected -- obviously I can't anticipate what you're going to ask me, so I can't have articles that answer everything. But I picked this set of articles because they address particular points that I think have some potential significance about this case.

Q. Yeah. But how did you go find those articles? Or did you go find those articles?

A. Oh, I did. In other words, I have several thousand articles in my other office over in the hospital on various subjects, diverse subjects, not necessarily just including benzene literature. But I look through my files, and I pick out the articles that I think might be appropriate for a particular case.

Q. And so how did you come about this set?

A. Well, for example, one of the features of this particular case is the particular chromosome abnormality present, inversion 16. Well, it wouldn't make sense for me to pick an article that talked about the Philadelphia chromosome. That's a different chromosome. So I picked articles that had to do, at least from the chromosome standpoint, with chromosome inversion 16.

Q. And did you already have those articles that are

in there about the cytogenetics and chromosome 16? Did you already have all those articles in your files before the Daniel Minnehan case came to you?

A. I would say probably 95 percent of them I did.

Q. And where did you get the other 5 percent of them?

A. The other 5 percent would be gotten, for example, if I had a particular article -- or a particular aspect in inversion 16 that I was looking for, I would go into some type of med search file, put in what topic I was after; and if I came up with a particular reference I was interested in on that particular subject and if it wasn't a journal we had in our hospital library, I would get my secretary to order it for me.

Q. Did Mr. Dillard first contact you about this case?

A. I don't think so. Actually it might have been Mr. Perry. It's been some time ago. I don't remember who the first person was that contacted me about this case.

Q. Did any of the lawyers make reference to any articles for you to locate for this case?

A. No.

Q. Did any of the lawyers provide you with any articles for this case?

A. No.

Q. You've certainly discussed with the lawyers this inversion 16 thing, right?

A. Yes.

Q. And you know that it's their feeling that that means that it can't be benzene related, right?

A. I don't think that that would be a correct statement.

Q. What do you think would be a correct statement about inversion 16?

A. I think that what would be a correct statement is that when one sees a case of inversion 16, it is far more likely to be de novo leukemia than a chemical-induced leukemia.

Q. Are you someone who has personally studied -- have you conducted any epidemiologic studies on cytogenetics and acute myelogenous leukemia?

A. No. I've ordered them occasionally on patients, but I don't do them myself.

Q. No. Sorry.

Have you conducted any epidemiologic studies on cytogenetics and benzene-related leukemia?

A. No.

Q. What you know, to the extent that you know something about cytogenetic markers and acute myelogenous leukemia, is what you have read in the literature,

correct?

MR. GALBRAITH: Object to form.

A. That would be correct.

Q. (BY MR. KAESKE) Have you ever treated a patient with acute myelogenous leukemia M4 inversion 16?

A. I don't know. In other words, I have treated a lot of leukemia patients over the years; and I don't recall which particular defect they had. And so I can't tell you that I have or I haven't.

Q. You would certainly agree with me that people have been relating acute myelogenous leukemia and benzene exposure since long before we were able to conduct cytogenetic testing on acute myelogenous leukemia patients, correct?

A. That is correct.

Q. And you would also agree with me that there is no epidemiologic study that shows a statistically significant doubling of the risk for acute myelogenous leukemia with any given cytogenetic marker, correct?

A. I'm not sure I understand that question at all.

Q. One of the depositions that you've got in your stack of depositions is the deposition of Richard Irons, right?

A. Yes.

Q. Did you read that deposition?

A. Yes.

Q. You saw where Dr. Irons and I had a discussion about inversion 16, correct?

A. I read it. I remember more of the questions you asked about 5 and 7 than inversion 16, but we could look at it. I'm not sure which question you're referring to.

Q. Well, here's the point: There isn't any -- first of all, let me see if I can understand what your opinions are about cytogenetic markers.

Do you think that there is some cytogenetic marker that is diagnosed of a benzene-induced leukemia?

A. No.

Q. Do you think that there is some set of cytogenetic markers that you would require to say that within a reasonable degree of medical probability, someone's acute myelogenous leukemia is related to benzene exposure?

A. Not as a sole aspect of the case. In other words, certain markers are more common in chemical-induced leukemias than others. But you're looking at the whole of the picture. That's one thing that you look at in terms of opining an opinion.

MR. KAESKE: Object as nonresponsive.

Q. (BY MR. KAESKE) Maybe you didn't understand my question.

Are there a set of -- are there any cytogenetic markers or chromosome aberrations that you, in your opinion, need to see, in addition to whatever other things, but that you need to see in order to give the opinion that within a reasonable degree of medical probability, someone's benzene exposure is related to their acute myelogenous leukemia?

A. Well, I think, as I said, you can't take an individual case and prove causation, by any means. In other words, we only infer causation by certain things, chromosome abnormalities being one of them. There are certain chromosome abnormalities that are more common than others in a chemical-induced leukemia, but there is no single chromosome abnormality that proves a chemical-induced leukemia.

MR. KAESKE: Object as nonresponsive.

Q. (BY MR. KAESKE) So I infer from your answer that you believe that cytogenetic markers are something that you use to prove causation, correct?

A. They are one aspect of what we look at to try to come to a conclusion on causation.

Q. All right. And can you point me to any study published in the peer-reviewed scientific literature that says that -- well, first let me ask you this: Do you think that there is -- that it is generally accepted in

the scientific community that there are certain chromosome abnormalities that can be used to prove or disprove causation between benzene exposure and an acute myelogenous leukemia?

A. Not prove. I don't believe there are any chromosome abnormalities that can prove "yes" or prove "no," as best I'm understanding your question.

Q. Well, I think you said in your answer just a moment ago that chromosomal abnormalities are part of your analysis for causation; is that correct?

A. Correct.

Q. Is there any article in the medical or scientific peer-reviewed literature that says chromosome abnormalities are to be used as a device or piece of information in determining causation for a benzene-related leukemia?

A. I don't know if they would state it in that particular language, but there are plenty of articles that say that particular chromosome abnormalities are seen more commonly in benzene-associated leukemia.

MR. KAESKE: Object as nonresponsive.

Q. (BY MR. KAESKE) And that's certainly not what I asked you.

You will agree with me that there is a difference between looking at a bunch of cases and seeing

what kind of chromosomal abnormalities that they have, and inferring from chromosomal abnormalities that someone has, what the cause of their disease might be, correct? Those are two different things?

A. Well, a chromosomal abnormality is one thing; and the causation is another thing. Yes, I agree with that.

Q. All right. What I'm asking you for is: Are there any articles published in the peer-reviewed scientific literature that say, in determining the causation of an acute myelogenous leukemia, whether or not it's related to benzene exposure, that what you should do is you should consider the chromosomal abnormalities?

A. Well, yes. I think that the articles suggest that that's true.

Q. Name one.

A. Mauritzson's article, for example, where he looks at chromosome abnormalities in a large number of people and says that certain abnormalities are more suggestive of de novo and other abnormalities are more suggestive of chemical induced.

Q. That article that you just said, that's whose article?

A. I believe it's Mauritzson.

Q. And that's a chemotherapy article?

A. Not exactly. In other words, it's sort of a

statistical analysis of chromosomal abnormalities in myelodysplasia and acute leukemia, with particular attention to what we call therapy related or chemical-induced leukemia or secondary leukemia.

MR. KAESKE: Object as nonresponsive.

Q. (BY MR. KAESKE) It's not a benzene related -- it doesn't look at benzene-related leukemias, correct?

A. Not specifically, no.

Q. Right. So my question was about benzene-related leukemias, not about chemotherapy or chemical-induced leukemias.

So now that we're clear on that, can you name me an article published in the scientific peer-reviewed literature that says that when you're determining causation for benzene-induced acute myelogenous leukemia, that you look at particular chromosome changes?

A. Well, there are numerous articles that cite the fact that chromosome abnormalities of 5 and 7 appear frequently in benzene-related hematologic diseases. That's not a proof, but that's just a statement of frequency.

MR. KAESKE: Object as nonresponsive.

Q. (BY MR. KAESKE) Sir, I'm talking about methodology. Do you understand that?

A. I'm totally misunderstanding, obviously, what you're asking me.

Q. Let me start with this. And I'm sorry for the confusion. Have you ever seen any articles -- well, I don't want to know if you've ever seen.

Can you name for me an article in the peer-reviewed scientific literature discussing the methodology for diagnosing causation in acute myelogenous leukemia, specifically with respect to benzene exposure, that states that chromosome abnormalities are to be used in determining or in helping to determine the causation for an acute myelogenous leukemia with respect to benzene exposure?

MR. GALBRAITH: Object to the form.

A. The answer would be "no" to that.

Q. (BY MR. KAESKE) All right. Thank you.

Do you believe that acute myelogenous leukemias that are caused by benzene exposure have a worse prognosis than are acute myelogenous leukemias that are not known what the cause is?

A. Yes, that would be true.

Q. And when that is the case, that they have a worse prognosis, what does that involve? What does that worse prognosis for benzene-induced leukemia involve?

A. Well, generally a benzene-induced leukemia --

and in the hematologic literature, we lump benzene-induced leukemia in with chemical-induced leukemia, therapy-induced leukemia, secondary leukemia. We consider all those synonymous.

MR. KAESKE: Object as nonresponsive.

Q. (BY MR. KAESKE) Sir, I'm just asking you about benzene-related leukemia.

A. I understand. And I'm just explaining that when I give my answer, benzene is considered to be the same as these other compounds that we have a great deal of information about.

Q. Well, sir, you, at trial, may or may not be able to give that opinion.

A. Correct.

Q. So right now I'm just asking about benzene, okay? Now, I understand that you haven't specifically treated a benzene-related leukemia; but you seem to have an opinion about -- I mean, I just asked you and you gave me an opinion about benzene-related leukemias and their prognosis; and so I want to focus on that. So if you could, just give me an answer that relates specifically to benzene-related leukemia.

MR. DILLARD: Object to form.

A. Well, the answer is that in articles on benzene-related leukemia, there is a frequency of

abnormalities in chromosome 5 and 7. Whenever we see abnormalities in chromosome 5 and 7, they perform badly with treatment. So, therefore, by inference, if I saw a benzene-induced leukemia that had a chromosome 5 and 7 abnormality, it would perform badly with treatment.

MR. KAESKE: Object as nonresponsive.

Q. (BY MR. KAESKE) I guess I didn't ask a good question again.

It isn't something about the 5 or 7 abnormality itself that makes the person have a bad prognosis, right? It's just a relationship that you're seeing between people that have a particular chromosome abnormality and their prognosis, right?

A. Yes, there is a relationship between the type of chromosome abnormality and prognosis, that's correct.

Q. Sorry. I gave you two choices, and you said yes.

A. Okay.

Q. This is what I'm saying: There isn't something about the fact that it's chromosome 5 or that it's chromosome 7 where the damage is that specifically relates to the prognosis, correct?

A. Now, say that one again, that question again. I'm not sure I understood that one. I think you said it's not where the specific damage is? I'm not sure exactly how you said that.

Q. Look, if you've got a chromosome 5 abnormality, it isn't because your chromosome 5 is messed up that you have a bad prognosis, correct?

A. I wouldn't know how to answer that. In other words, as I've said, we recognize that a 5 or a 7 chromosome abnormality, meaning your 5 or 7 chromosomes are messed up, when you have that, your prognosis is poor.

Q. Yeah, but it isn't because you've got that particular chromosome abnormality that your prognosis is poor. It's because of something else that has to do with the disease that your prognosis is poor, right?

A. I can't tell you the difference. In other words, the chromosome abnormality relates to the disease directly. And when you see that chromosome abnormality, you imply that whatever mechanism is offered of theirs is making the prognosis worse.

Q. Well, it isn't just chromosome 5 and 7 abnormalities that have a poor prognosis, is it?

A. That's correct. There are others that have a poor prognosis.

Q. Yeah, and they are ...

A. Well, generally, they're divided up into three general categories of, let's say -- I mean, obviously no leukemia is good. But certain what we call balanced translocations have a better prognosis than, for example,

multiple chromosome abnormalities, when there are a number of chromosomes abnormal, or chromosomes 5 and 7. And there are some that are so-called good risks; some that are, let's say, intermediate risks; and some that are bad risks.

Q. So you can't name me any of the other chromosome abnormalities that are high risk?

A. That are high risk? As I just said, multiple --

Q. That have a bad prognosis.

A. Multiple chromosome abnormalities.

Q. Among which chromosomes?

A. It can be any of them. If I saw four or five different abnormalities in an acute leukemia, that would be a very bad prognosis.

Q. All right. What else? What other leukemias have a bad -- what other acute myelogenous leukemias have a bad prognosis?

MR. GALBRAITH: Object to form.

A. I would have to look at the articles. There may be several. But we could look at those and see which ones are in which category. Generally speaking, the multiple abnormalities in the 5 and 7 are considered to be bad; the balanced translocations, considered to be good; and then there are some that are in the middle that are translocations, let's say, 9 to 11, that are worse than

some of the translocations but better than others.

Q. (BY MR. KAESKE) All right. Now, when we say -- when you say that the prognosis is poor, what do you mean by that?

A. Well, I can give you the exact statement from one of these articles as to what that means.

Q. No, I don't want it from an article. You're a clinician, right?

A. Yes.

Q. So just give me your clinical analysis or give me -- tell me in layman's terms when you say it's a poor prognosis, what can that person expect?

A. What that person can expect is to stay in remission -- if you're lucky to get into remission, stay in remission three or four months and probably have about a 10 percent survival at the end of three years.

Q. And when you say that they stay in remission for three or four months, then what happens?

A. They relapse.

Q. Then they relapse?

And then what would, if that person -- what would the rest of the clinical course for that person be like?

A. Well, generally, when one puts a leukemia into remission with chemotherapy, oftentimes -- not always, but

oftentimes the first remission is the longest. And as you keep getting into second and third remissions, the interval of time between each remission gets shorter and shorter; and finally the drugs don't seem to work at all.

So in a person who is having constant relapses, one might try to proceed with a marrow transplant as an alternate form of treatment.

Q. So when you say that someone has -- when we talk about this poor prognosis, what you mean by that is if they achieve relapse [sic], the relapse [sic] will likely not last; is that correct?

A. No, the relapse will last.

Q. Sorry.

A. They won't go back into remission easily.

Q. Sorry. I used the wrong word. You saw that.

A. Okay.

Q. So when you say that someone has a poor prognosis, what you mean by that is if they achieve remission, the remission will likely not last?

A. That's correct.

Q. And then if someone goes into remission but relapses and their cancer comes back, a choice at that point is transplant, is what you just said, correct?

A. Well, one can try alternate drugs; and sometimes you're successful with that. But frequently that tends to

make you want to try something a little more dramatic, like a transplant.

Q. So then if you try a transplant and you have one of these patients and they don't have a related donor, then what does the prognosis look like?

A. Well, the best results of a transplant would be with a related donor. Then you go to the so-called national donor match, and you find people who look in the test tube like you but they're not exactly like you. And that's the next best choice to try to do a transplant.

Q. Okay. But -- and my question kind of started where you ended.

A. Yes.

Q. If you're one of those people where you don't have a related donor and so you've got to go into the national matched donor pool, then what's the prognosis like for that kind of transplant?

A. Well, it's not as good. In other words, the prognosis for a related transplant is better than an unrelated donor transplant. But it's still better than nothing.

Q. But can you not give me any details, any risk analysis, any, "This is the percentage of likelihood," or what's going to happen, what are the possible risk factors for an unrelated donor, that kind of thing?

A. Well, the situation for acute leukemia, in general -- adult acute leukemia is not great. We typically say that our ability to achieve a cure with chemotherapy alone is probably no better than 20 percent. If we add a transplant to that -- let's assume we got the person in remission and then did a transplant -- they don't always live happily ever after. We might improve that cure rate possibly up to, let's say, 40 percent. But many people that you transplant, and even if the transplant appears successful, ultimately relapses or will die of some other transplant-related complications. So in the end analysis, adult acute leukemia is often fatal.

Q. Do you feel like you're familiar with Mr. Minnehan's course up until now?

A. Yes.

Q. That big huge stack over there (indicating), have you read it all?

A. I can't swear that I've studied every page carefully, but I've looked at each one of them, some in more detail and some in a very cursory fashion. And I have an idea of what happened with him.

Q. Okay. You'll agree with me, won't you, that the course of his leukemia has been a complicated one, correct?

A. Yes.

Q. And you will agree with me that the course of his leukemia looks like one of those folks that had a poor prognosis, correct?

A. No, not exactly. I wouldn't agree with that statement.

Q. Why not?

A. Because he went into remission fairly promptly and he stayed in remission for an extended period of time, considerably more than three months. And the fact that he ultimately relapsed, people with a great prognosis relapse. People with normal chromosomes relapse. And so the fact that he relapsed is not surprising, whether he had a good risk or a bad risk leukemia. He actually stayed in remission longer than most people with a bad risk leukemia.

Q. And can you cite me to an article in the peer-reviewed scientific literature that talks about how long people with a poor risk of acute myelogenous leukemia stay in remission before they relapse?

A. We could look out there. There may be some articles that comment about that.

Q. Well, why don't you see.

A. (Peruses documents) Well, this article which -- well, let me identify this. It's an article on secondary leukemia, and it's from -- each year the American Society

of Hematology meets; and a textbook is published, which is the educational booklet; and an expert in that area gives a dissertation. And this is from one of those. It's by Mr. Applebaum, as the lead author.

And in talking about people with secondary AML with cytogenetic abnormalities associated with a poor outcome from chemotherapy, he says: "Accordingly, it's not surprising that the overall long-term survival of patients with secondary AML is low. Among 155 patients reported by the MD Anderson group, the median survival was only 3.5 months and fewer than 10 percent were alive in three years."

Now, if their median survival was only 3.5 months, that meant a lot of them relapsed before 3.5 months; so they didn't stay in remission very long.

MR. KAESKE: Objection, nonresponsive.

Q. (BY MR. KAESKE) That actually doesn't say anything about even achieving remission, does it? That which you just read to us doesn't even talk about achieving remission?

A. Well, actually it does, because it goes on to say: "No single form of post-remission chemotherapy has been demonstrated to be superior." In other words, they're talking about the fact that, yes, you can get these people into remission; but you can't keep them

there. They want to relapse very promptly.

Q. So that we're clear, the question that I asked you that prompted you to look for these medical articles was: I'm looking for an article that says what you said, which is that 20 months of remission versus three months of remission is not indicative of a poor prognosis acute myelogenous leukemia. And so we're specifically now looking now about relapse rates.

A. Well, let's see if I can find something. These papers are not specifically designed with that in mind, but I may have another that addresses that. Let's see (peruses documents).

Okay. This is from Dr. DeVita's textbook, the Sixth Edition, on Cancer: Principles, published in 2001. And he says: "AML that occurs as a consequence of prior cytotoxic therapy or that has developed from an antecedent hematologic disorder, for example, myelodyplastic syndrome, has a particularly poor outcome, with a lower incidence of achieving a complete remission and shorter duration of survival than for patients with de novo AML. Multiple studies have demonstrated the prognostic importance of cytogenetic abnormalities in AML, making this the single most important predictor of outcome." And he goes on to say, as you asked me before, which chromosomes have a good prognosis and which ones

have a bad one.

So I believe he's answering your question, that people who have a bad prognosis acute leukemia don't go into remission as easily and they don't stay in remission as long. And the ultimate result is a high incidence of death early on.

Q. But what you just read, that doesn't say that they don't stay in remission as long, does it?

A. Well, I think -- if it doesn't say it, I think it implies it. I will have to look to see if he says -- he's talking about the ability to go into remission.

Q. That's right. And that's why it particularly doesn't answer my question. My question was only, Doctor, in response to one of the things that you said. I'm looking for an article that says, as you have said, that people with these poor prognoses don't stay in remission for as long as 20 months.

A. Let me look and see. As I say, I always can't anticipate what I might be asked. And so when I pick articles, I have to pick something general; and so they don't necessarily answer a specific point. But I may have one in here that will get to that. Let's see. I know the one I'm looking for here (perusing documents).

Well, I don't think that I can -- most of these articles deal with ability to achieve remission and

survival. I would have to tell you that I don't have here today articles that show that the length of remission is shorter. Although, I can tell you from 35 years of clinical experience that it is and there are many articles that say that, but I don't have them here today.

MR. KAESKE: Objection, nonresponsive.

Q. (BY MR. KAESKE) Just as we sit here today, you can't name for me an article in the scientific peer-reviewed literature that shows that length of remission for a poor prognosis for acute myelogenous leukemia is shorter than 20 months?

A. That would be true.

Q. List for me, if you will, all the reasons you believe that Mr. Minnehan's leukemia is not related to benzene exposure.

A. Well, first is if we look at the chromosome abnormality itself. We know that inversion 16 is a rare chromosome abnormality under any circumstances. But in some of these articles, they will look at large numbers of patients with what we call de novo leukemia and what we call therapy-induced or secondary or chemical-induced leukemia, of which I put benzene in that category.

The articles suggest that in de novo leukemias, this abnormality appears about some -- around 4, let's say 4.3, somewhere around that percent of the

time.

Q. I don't mean to cut you off. I just want an enumeration of the list of reasons.

A. Okay. Well, No. 1 is the particular chromosome defect that he has. No. 2 is the fact that it is an isolated chromosome defect, that it is not seen with other chromosome defects, because typically in chemical-induced chromosome defects of inversion 16, they don't appear as an isolated event. You have an inversion 16 plus three or four other things. So when you have an inversion 16 as a sole abnormality, it is much more likely to be caused by de novo as opposed to chemicals. That's No. 1.

No. 2 would be the fact that whereas we have extensive literature for 20-plus years on inversion 16 and many, many cases of de novo leukemia with it, there's not a single case in the world's literature of benzene-induced inversion 16. So we have no real basis to think that benzene could do that. Although I would admit that on a theoretical basis it might, we have no actual evidence that it can.

So we've got the chromosome defect. We've got the fact that it's never been in an isolated form. We've got the fact that it's never been described after benzene. And then we've got the fact of the occupation and the allegation.

As best I can determine it, Mr. Minnehan was a refinery worker, a pipefitter, a boilermaker, just like many of his peers in the areas that he worked and just like many of his peers around the country in other refineries. And when you look at epidemiologic studies of refinery workers from around the country, there isn't any increased incidence of acute leukemia and hasn't been for many, many years. So I would have to think that whatever he got exposed to, his fellow workers got exposed to it, too, in the same doses in his refineries and everybody else's refineries. And if that exposure was enough to cause acute leukemia, we would see a high incidence of it in other like-refinery workers; and we don't. So what that tells me is he's not any different than anybody else in his refinery, and there would be no reason for me to think that he had enough exposure to cause acute leukemia. Those would be generally the most important reasons I could cite.

Q. So I've got three reasons: One, you've never seen chromosome 16 associated with a secondary leukemia?

A. Well, I haven't seen it in the literature -- excuse me. I've seen chromosome 16 with secondary leukemia, yes; not from benzene. And where we've seen it from secondary leukemia --

Q. Hold on. So the first thing is you've never seen

a report of a benzene-related secondary leukemia -- sorry. You've never seen a report of a benzene-related chromosome 16 inversion, correct?

A. Correct.

Q. And so that's the first reason it's not benzene-related?

A. You could say one. I don't know which one you'd put the most weight on, but that's a reason.

Q. Okay. Then the second reason is because he's only got 16 inversion by itself, not with others -- not with other aberrations, correct?

A. Yes.

Q. Then the third one is, you don't see there to be an increased incidence of leukemia -- of acute myelogenous leukemia among refinery workers; is that right?

A. Of the type that he has, yes, that's correct.

Those three, I think, are all important things. And they enabled me to say -- and as I've said before -- in any one case of leukemia, one can never be certain about causation. But one can easily say that more likely than not, it is not caused by chemical exposure because of those reasons.

MR. KAESKE: Object as nonresponsive.

Q. (BY MR. KAESKE) I just want to make sure that I've got the entire list.

A. You've got the list.
Q. Well, but I need to have the list with the answers to the questions, and then we can proceed.
A. Okay.
Q. First is that you've never seen a report in the scientific literature of chromosome 16 and benzene exposure, correct?
A. Of inversion 16 in benzene exposure, correct.
Q. All right. Second is some problem you have with the fact that he's got inversion 16 all by itself, as opposed to with other problems --
A. Correct.
Q. -- with chromosomes, correct?
MR. DILLARD: Object to the form.
A. Yes.
Q. (BY MR. KAESKE) And the third thing is that you haven't seen any increased incidence of acute myelogenous leukemia among refinery workers, correct?
A. Well, the bulk of the studies of refinery workers do not suggest an increase.
Q. All right. Do you have any particular opinions about Mr. Minnehan's particular exposure?
A. You mean how many parts per million or whatever he got exposed to?
Q. No, not necessarily. Although if you have an

opinion about that, then I want to know about it.

A. Okay.

Q. I presume you do not.

A. I do not. I don't know what he was exposed to.

Q. Fair enough.

What I meant was -- and, look, if I can just kind of speak freely for a second, what I understood your third thing to be was, basically: When you look at like-associated workers, you don't see -- when you look at like occupations, you don't see an increase in acute myelogenous leukemia. That's not an exposure-based statement. It is just a statement about epidemiologic studies in this occupation.

And so now my question is: Is there something specific that you're going to say about exposure, or his particular exposure; or are you just going to rely on your understanding of the refinery worker studies?

MR. DILLARD: Object to form.

A. Just my understanding of the refinery worker studies as they've been published.

Q. (BY MR. KAESKE) Okay. Fair enough.

And in part, that's because you don't know what Mr. Minnehan's exposure to benzene was, correct?

MR. DILLARD: Object to form.

A. I don't know what his exposure to benzene was.

Q. (BY MR. KAESKE) Are there any other reasons that you have for your opinion that Mr. Minnehan's exposure was not benzene -- sorry -- that Mr. Minnehan's acute myelogenous leukemia was not benzene related?

A. No. Those would be the important things that I said.

Q. You will agree with me, won't you, that all of Mr. Minnehan's treatment since October 24th of the year 2000 has been related to his acute myelogenous leukemia, correct, or its effects?

MR. DILLARD: Object to form.

A. Well, generally speaking, that's true. I mean, you could look at certain specifics. For example, he had to have his spleen removed because he had what we call ITP. That may or may not have been related to his acute leukemia, but he did undergo a splenectomy.

Q. (BY MR. KAESKE) Well, he underwent a splenectomy because he had ITP specifically because of the disease process that he's been withstanding since the year 2000, right?

A. Not necessarily. I do lots of splenectomies for ITP patients who don't have a acute leukemia. That can occur in many different settings. So I am not a hundred percent certain that it was related to the acute leukemia,

but it was certainly part of his treatment program.

Q. Are you going to give an opinion that ITP and his subsequent splenectomy was not related to his disease process in general, whether it be his acute myelogenous leukemia or his graft-versus-host, or any of the rest of that?

A. I can't give an opinion on that because I don't have enough detail. I only have the notes that are in the chart, that they were taking out his spleen because he had a low platelet count and they thought that it would improve.

Q. As an answer to my specific question: Are you going to give an opinion in this case that his splenectomy or his ITP were not related to his disease process or his graft-versus-host disease?

A. No.

Q. Are you going to give an opinion in this case that any of the medical treatments that he has underwent since October 24th, 2004 [sic], are not related to his acute myelogenous leukemia or his graft-versus-host disease?

A. No.

Q. Have you spoken with any of his treating physicians in this case?

A. No.

Q. Have you spoken with anyone, other than the lawyers, about this case?

A. No.

Q. All right. Have you seen -- the treatments that you have seen that are in these medical records -- and by the way, have you been given, as far as you know, all of the medical records from Mr. Minnehan from the year 2000 until today?

A. Well, the records speak for themselves. I'm not sure what the most recent date was on those records. I'm sure that since he's continuing to get care, there would be some records that I don't have there.

MR. KAESKE: Do y'all know how up-to-date the medical records are you've given him?

MR. PERRY: I don't.

MR. KAESKE: When was the last time you sent him medical records?

MR. PERRY: I'm not sure.

Q. (BY MR. KAESKE) Doctor, when was the last time you received medical records?

A. It's been some time ago, and I -- for example, obviously I have records. I made some notes here and I have records up through 4-29-04, but that's six months ago, I guess. And I don't know that I had any notes since that particular time.

Q. You would agree that all of the treatment that Mr. Minnehan has received over at MD Anderson since October of 2000 has been reasonable and necessary for the treatment of his acute myelogenous leukemia --

A. Yes.

Q. -- and its complications, right?

A. Yes.

Q. Have you been provided any bills?

A. I don't believe there are any bills in that material.

Q. Okay. Whatever the bills are that have been associated with the treatments that he had, those costs would also be reasonable and necessary costs associated with the treatments that he's received at MD Anderson for his acute myelogenous leukemia or its complications, correct?

MR. PERRY: Object to form.

A. Yes.

Q. (BY MR. KAESKE) Are you going to give any opinions in this case about -- I just want to ask this one or two last questions about this: Are you going to give any opinions in this case about what exposure levels of benzene are required to cause acute myelogenous leukemia?

A. If asked, yes. I can only cite what the literature tells me.

Q. But you don't have an opinion in this case that Mr. Minnehan's exposure was not enough to have caused his acute myelogenous leukemia, correct?

A. I don't know what his exposure was.

Q. Okay. So let's see if we can get a "yes" answer to my question, then.

A. Okay.

Q. You do not have an opinion that Mr. Minnehan's exposure to benzene, whatever it was, was not enough to cause his acute myelogenous leukemia, correct?

A. Well, indirectly I think I've said that because I said that were he to have had enough exposure to cause acute leukemia, so would many of his coworkers in his refineries and many other refineries. And because I don't see that, that tells me that whatever exposure he had, it was below those limits.

MR. KAESKE: Object as nonresponsive.

Q. (BY MR. KAESKE) Are you going to give the opinion that Mr. Minnehan's exposure to benzene, whatever it was, was not enough to cause his acute myelogenous leukemia?

A. Yes.

Q. Will you agree with me that benzene-induced acute myelogenous leukemia is not a disease that is caused by achieving some amount of cumulative dose to benzene?

MR. DILLARD: Object to form.

A. Well, I'm not certain. Are you asking that one has to achieve a particular dose? Is that what you're asking?

Q. (BY MR. KAESKE) Not exactly.

A. What --

Q. Let's take lead, for example.

A. All right.

Q. You get sick when you get a certain amount of lead in your body, right?

A. Yes.

Q. Benzene is not that kind of disease where you get sick when you get a -- well, acute myelogenous leukemia and benzene is not that kind of disease where you get a -- you hit a certain threshold and then you get acute myelogenous leukemia, correct?

MR. DILLARD: Object to form.

A. Well, it's certainly not akin to lead poisoning. In general, the studies suggest that one can relate the cumulative dose to benzene along with the risk of catching leukemia.

MR. KAESKE: Object as nonresponsive.

Q. (BY MR. KAESKE) Sir, I'm not talking to you now about -- just so that you're clear, I'm not at all talking to you about risk. I don't even want to know

about risk. I don't even want to consider risk.

A. Okay.

Q. I want to talk about biologically how the disease is caused, okay? When we talk about biologically how acute myelogenous leukemia is caused by benzene exposure, it is not a disease that is caused by achieving a certain amount of benzene toxicity in the blood or in the body before you can achieve acute myelogenous leukemia from benzene exposure, correct?

MR. DILLARD: Object to form.

A. I think that would be true, yes.

Q. (BY MR. KAESKE) All right. I want to take a break for a second; and then I want to see whether or not I have any more questions to ask you. Would that be all right?

A. Certainly.

Q. Thanks.

(Ten-minute recess taken)

Q. (BY MR. KAESKE) Would you read those notes into the record, please?

A. Sure. It starts out: "David Minnehan, 5/30/58, 051-54-4729. Oil field worker-pipefitter-boilermaker supervisor. 10/2000 in Alaska, chest pain, pancytopenia."

"10/27/2000, bone marrow, AML-M-4. Bone marrow, 64 percent blasts, inversion 16 defect.

Peroxidase, positive; platelet count, 110,000; white cell count, 3000; hemoglobin, 13.9."

"Initial treatment, Fludarabine and Cytosar" -- and that's F-l-u-d-a-r-a-b-i-n-e and Cytosar, C-y-t-o-s-a-r -- "14 courses of chemotherapy. Relapsed summer of 8/2002. Re-induced with Fludarabine, Cytosar."

"Deep vein thrombosis of arms, left axillary vein thrombosis. On Lovenox," L-o-v-e-n-o-x.

"Normal bone marrow chromosomes post-therapy."

"Splenectomy for ITP, 8/7/03."

"Note says still smoking in 10/27/03, 40-pack history."

"2/6/04, normal bone marrow chromosomes."

"2/3/04, receiving Tacrolimus," T-a-c-r-o-l-i-m-u-s, "Prednisone, and Rapamycin," R-a-p-a-m-y-c-i-n."

"4/29/04, positive Coombs' test." That's C-o-o-m-b-s apostrophe. "LDH, 701 (normal range 313 to 618)."

Q. Are those all the notes that you've got on that page?

A. Yes.

Q. Do you have an opinion about Mr. Minnehan's prognosis?

A. Well, I don't -- I think we would have to say his prognosis is uncertain at this point. That would be the best I could do in terms of the likelihood of his relapse.

Q. What about in the terms of the likelihood of his survival from the graft-versus-host disease?

A. Well, I think that it's probably unlikely that he will die of graft-versus-host disease. I think his threat would be on a relapse of the leukemia.

Q. Do you know what the status of his graft-versus-host disease is now?

A. No.

Q. You have previously testified, and it's certainly your opinion, that graft-versus-host disease can be a fatal condition, right?

A. Yes, it can.

Q. Or that it can lead to other fatal conditions?

A. Yes.

Q. Have you read Dr. Gardner's deposition in this case?

A. Yes.

Q. Do you have any criticisms of Dr. Gardner's testimony in this case?

A. You would have to pick a specific thing. Dr. Gardner said many things in the deposition, and it might well be that there are some things that I don't

agree with.

Q. Did you take any notes when you read his deposition?

A. No. I may have scribbled something on the front cover. I believe that was his deposition that I said something because he made a comment about smoking.

Q. What does that say?

A. It says: "See 43." And let's see what --

Q. And then what are the words it says after that?

A. "Acute phase reactant."

And let's see what I was dealing with on page 43. Oh, yes. What that had to do with, his serum ferritin. That's f-e-r-r-i-t-i-n. And the serum ferritin is generally a reflection of how much iron is in the body in most people. But when you have an inflammatory state like graft-versus-host disease, it often goes sky-high and you lose that relationship. And so what he was saying is that his ferritin is 4400 and that that means he's got a lot of iron overload.

I don't believe that to be the case because in this setting, it's totally inaccurate for that reason. Rheumatoid arthritis patients may have levels of 15,000 and not have high iron levels. So I think that one can't look at his ferritin level and accurately tell his degree of iron overload. You would have to do it by getting a

CAT scan or MRI and looking at the liver.

Q. Do you know whether or not Mr. Minnehan has been treated because of his high iron levels?

A. I have no idea.

Q. So you don't know what his treating physicians believe about that point that you just made?

A. Correct.

Q. Any other criticisms of Dr. Gardner?

A. He let's see. It says "pages 38 through 39." I think it was the same thing. In other words, he had a liver biopsy and there was no evidence that he had iron overload from that and that led into the discussion on the ferritin levels. So that was part and parcel of that same comment.

"Pages 71 through 72, smoking." I don't know why I cited that. I mean, it had to do with smoking; but there's nothing that he said that I notice I would take any issue with on that page.

Q. All right.

A. And page 80 is the next page. Well, yes, here on page 80, what he is talking about is the fact that in certain leukemias, you don't find any chromosome changes. He states over here: "Now with the FISH you can find abnormalities in almost 90 percent."

So it's a moving database. And the

implication is that, well, if we did FISH studies on people who had normal chromosomes in leukemia, we would find all their abnormalities. And, actually, there's extensive literature on that, some of which I have here, that says that's not true. And so I would disagree with the statement that when we have a person with normal bone marrow chromosomes, we can simply do a FISH analysis and find the defects.

Q. Anything else that you disagree with him about?

MR. DILLARD: Well, he said you would have to ask him specific questions.

A. Yeah. I mean, it's conceivable that there's something else. But, I mean, those -- I made some notes about those things. But I don't know what else -- you know, it's been some time since I read his deposition; and I don't know what else he said in there that I might disagree with him on.

Q. (BY MR. KAESKE) Do you agree or disagree with him that Mr. Minnehan is likely to develop or ultimately have osteoporosis?

A. I don't know. I wouldn't have any comment about that. I don't know if he will or will not.

Q. Do you have an opinion as to whether or not he will ultimately be diagnosed with diabetes?

A. I don't have an opinion in the sense that it

depends on how long he has to remain on these drugs for graft-versus-host disease. And that's an uncertainty because his graft-versus-host disease could get better. It could get worse. It could stay the same. And those kinds of complications -- the osteoporosis, the diabetes, and so on -- all relate to the duration that one has to be on the steroids and the immunosuppressor drugs for graft-versus-host disease, and we don't -- so I can't give an answer to that.

Q. Well, has he been on them long enough now to lead to some future problem like we've just discussed?

A. I don't think so.

Q. What about potential future renal failure?

A. Again, that relates to how long you have to stay on these drugs. We know, for example -- and what he's probably referring to is when we use drugs like Cyclosporine, which is often used in graft-versus-host disease. Depending on how much you give and how long you give it, it can be toxic to the kidneys. And so if you monitor the blood levels and the kidney function starts to be impaired, you try to reduce the levels. Again, all of that depends on how long will he need to be on these drugs. So I don't know if he will or won't get any problems with his kidneys.

Q. Well, how much longer do you think he needs to be

on these drugs before he's going to develop these problems that we've talked about?

A. I don't -- well, I'm not certain I know the question. I think you're referring to how long does it take to get kidney abnormalities from Cyclosporine or these kinds of drugs?

Q. Yeah, or any of these things. You said you don't know whether the duration is long enough. How long of a duration are you looking for?

A. Well, I would think that you have to be on it for several years. Of course, you can acutely get damage if you shoot your levels up very high; renal failure can occur very quickly. But in most people, if you're watching the levels and monitoring the kidney function, it would be many years. I have one patient I'm seeing right now who has got a kidney transplant, and she's been on those drugs for about ten years and hasn't had any major complications. But that's one person. In other words, it's different for different people. It certainly has the potential to damage the kidneys.

MR. KAESKE: Object as nonresponsive.

Q. (BY MR. KAESKE) I don't have any other questions. Thanks.

MR. DILLARD: Thank you, sir.

MR. PERRY: Reserve all questions.

(Exhibits 1 and 2 marked)

MR. KAESKE: Before we leave, I'd just like to read into the record the depositions that we've got here. This is the deposition of Dr. Raabe, R-a-a-b-e; Dr. Egilman, E-g-i-l-m-a-n; Dr. Mehlman, M-e-h-l-m-a-n; Dr. Irons; Mr. Minnehan; John Spencer.

And actually, Doctor, before we leave, I do need to ask you one more question.

THE WITNESS: Okay.

MR. KAESKE: Dr. Beran, B-e-r-a-n; and Dr. Nicas. And that appears to be it.

Q. (BY MR. KAESKE) You don't have any notes in this case other than that one page of handwritten notes that you've got right there?

A. That's correct.

Q. Is that right?

A. And these, of course, copies of particular pages from the medical records.

Q. All right. And you haven't taken any notes when you read any of the depositions?

A. No. That's correct.

And this deposition, do you want it as part of this or back to that? It's Dr. Gardner's.

Q. Well, I think you read all the notes on there into the record, didn't you?

A. Yes.

Q. Then that will be fine. Thanks.

(Proceedings concluded at 12:40 p.m.)

CHANGES AND SIGNATURE

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I, ETHAN NATELSON, MD, have read the foregoing deposition and hereby affix my signature that same is true and correct, except as noted above.

_____

ETHAN NATELSON, MD

THE STATE OF _____ )
COUNTY OF _____ )

Before me,_____, on this day personally appeared ETHAN NATELSON, MD, 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 purpose and consideration therein expressed.

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

_____

NOTARY PUBLIC IN AND FOR
THE STATE OF _____

My Commission Expires: _____

| | | |
|---|---|---|
| CAUSE NO. 21916*JG02 | | |
| DANIEL T. MINNEHAN, Individually and as Next Friend for Joshua Stephen Minnehan and Kyle Marshal Minnehan | ) | IN THE DISTRICT COURT OF |
| Plaintiff, | ) | |
| vs. | ) | BRAZORIA COUNTY, TEXAS |
| AMERICAN OIL COMPANY, Individually and f/k/a Amoco Oil Company and Amoco, Inc.; et al., | ) | |
| Defendants. | ) | 239th JUDICIAL DISTRICT |

REPORTER'S CERTIFICATE
ORAL DEPOSITION OF ETHAN NATELSON, MD
OCTOBER 15, 2004

I, Susan A. Love, Certified Shorthand Reporter in and for the State of Texas, hereby certify to the following:

That the witness, ETHAN NATELSON, MD, was duly sworn and that the transcript of the deposition is a true record of the testimony given by the witness;

That the deposition transcript was duly submitted on _____ to the witness or to the attorney for the witness for examination, signature, and return to me by _____;

That pursuant to information given to the deposition officer at the time said testimony was taken, the following includes the amount of time used by each party at the deposition and all parties of record:

Mr. Michael L. Kaeske, Jr., Attorney for Plaintiff, (1:20);

Mr. Stephen C. Dillard, Attorney for Defendant Phillips Petroleum Company, Individually and a/k/a Phillips Oil Company, Phillips Chemical Company and Phillips 66 Company, (0:00);

Mr. Stan Perry, Attorney for Defendant Shell Oil Company, (0:00);

Mr. James B. Galbraith, Attorney for Defendants American Oil Company, Amoco Chemical Company, Amoco Corporation, Amoco Oil Company, BP Amoco Corporation, BP Chemicals, Inc., (0:00);

That a copy of this certificate was served on all parties shown herein on _____ and filed with the Clerk.

I further certify that I am neither counsel for, related to, nor employed by any of the parties in the action in which this proceeding was taken, and further that I am not financially or otherwise interested in the outcome of this action.

Further certification requirements pursuant to Rule 203 of the Texas Code of Civil Procedure will be complied with after they have occurred.

*****

Certified to by me on this _____ day of _____, _____.

_____

Susan A. Love, CSR
Texas CSR No. 5183
Expiration: 12/31/2004
Henjum Goucher Reporting Services
12621 Featherwood Drive, Suite 140
Houston, Texas 77034
Phone: (281) 464-6000
Fax: (281) 464-7733

FURTHER CERTIFICATION UNDER TRCP RULE 203

The original deposition was/was not returned to the deposition officer on _____.

If returned, the attached Changes and Signature page(s) contain(s) any changes and the reasons therefor.

If returned, the original deposition was delivered to Mr. Michael L. Kaeske, Jr., Custodial Attorney.

That $_____ is the deposition officer's charges to the Plaintiff for preparing the original deposition and any copies of exhibits;

The deposition was delivered in accordance with Rule 203.3, and a copy of this certificate, served on all parties shown herein, was filed with the Clerk.

Certified to by me on this _____ day of _____, _____.

_____

Susan A. Love, CSR
Texas CSR No. 5183
Expiration: 12/31/2004
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Houston, Texas 77034
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