NO. E-149519
IN THE DISTRICT COURT OF
JEFFERSON COUNTY, TEXAS
172ND JUDICIAL DISTRICT

CHARLES DOUGLAS YORK
VS.
TEXACO, INC. ET AL

DEPOSITION OF ETHAN A. NATELSON, M.D

January 26, 1998
Houston, Texas

RXAMINATION INDEX

EXHIBIT INDEX

A P P E A R A N C E S

FOR PLAINTIFF:
Mr. Glenn W. Morgan
Mr. Larry W. Thorpe
Reaud, Morgan & Quinn
801 Laurel Street
Beaumont, Texas 77701

FOR DEFENDANTS, MOBIL CHEMICAL COMPANY, INC., TEXACO, INC., TEXACO CHEMICAL COMPANY, TEXACO REFINING AND MARKETING, INC., CHEVRON, U.S.A., INC. (including Gulf Oil Corporation), CHEVRON CHEMICAL COMPANY AND UNOCAL CORPORATION:
Mr. Stephen C. Dillard
Fulbright & Jaworski, L.L.P.
1301 McKinney, Suite 5151
Houston, Texas 77010-3095

FOR DEFENDANT VELSICOL CHEMICAL CORP.,:
Mr. Robert P. Scott
Abrams, Scott & Bickley L.L.P.
600 Travis Street; Suite 6601
Houston, Texas 77002-3007
-and
Ms. Bonnie J. Semilof
Spriggs & Hollingsworth
1350 I Street, N.W.
Washington, D.C. 2005-3305

FOR DEFENDANT DuPONT:
Mr. M. C. Carrington
Mehaffy Weber, Keith & Gonsoulin,
A P.C.
2615 Calder Avenue, Suite 800
Beaumont, Texas 77704

FOR DEFENDANT FINA OIL & CHEMICAL COMPANY:
Mr. Kirk E. Martin
Jenkins, Grove & Martin, L.L.P.
2615 Calder, Suite 500
Beaumont, Texas 77704

ALSO PRESENT:
Mr. Charles York

THE VIDEOGRAPHER:
Mr. Devon Rochelle

5
On the 26th day of January 1998, commencing at 1:19 p.m., at the offices of Dr. Ethan A. Natelson, 1315 Calhoun, Suite 1800, Houston, Harris County, Texas, ETHAN A. NATELSON, M.D., F.A.C.P., appeared before me, Jack B. Moorhead, a Certified Shorthand Reporter in and for the State of Texas, and being by me first duly sworn, testified by his videotaped deposition as hereinafter set out, pursuant to Notice and the Texas Rules of Civil Procedure with the following stipulation by counsel:

It was stipulated that if the original deposition has not been signed by the time of trial or any hearing, an unfiled/unsigned copy may be used in lieu thereof.

THE VIDEOGRAPHER: Today is January 26, 1998. The time is 1:19 p.m. We are now on the record

(Witness sworn.)

EXAMINATION
QUESTIONS BY MR. DILLARD:
Q. State your name, please, sir.
A. Ethan Allen Natelson.
Q. And what is your occupation?
A. I am a physician, a hematologist.
Q. You are a medical doctor?
A. Yes.
Q. And how long have you been a medical doctor?
A. Since 1966.
Q. Have you brought with you a current resume or curriculum vitae that identifies your background and your training and education
A. Yes, I have.
Q. And is this a copy of it here today?
A. Yes.
(Natelson Exhibit 1 marked for identification.)
Q. All right. Your resume has been marked as Exhibit Number 1, Doctor, and I am going to ask you a few questions about your background. I am not going to go through the entire resume in any detail.
But can you tell us, first of all, where you attended medical school?

A. Baylor College of Medicine.
Q. And when did you graduate?
A. In 1966.
Q. Did you have any additional formal training after you received your M.D. degree in 1966?
A. Yes, I did.
Q. And would you describe that for us, please, sir, in a summary fashion?
A. Well, I did a residency training in the Baylor affiliated hospitals, which consists of the Ben Taub Hospital, Methodist Hospital, the Veterans Administration Hospital, and then I did a fellowship in hematology at the Methodist Hospital.
Q. Just some formal questions. Is your license to practice medicine on file with the appropriate authorities in Houston, Harris County?
A. Yes.
Q. You have said, now, a couple of times that you specialize in a particular field of medicine called hematology?
A. Yes.
Q. Can you describe for us briefly what the field of hematology consists of?

A. Well, it's a broad field that covers certain types of malignancies, for example, leukemias and lymphomas. In the area of malignancy, it covers certain inflammatory conditions, such as autoimmune diseases. It also covers bleeding disorders, such as hemophilia and problems with bleeding from low platelet counts, and it also involves the evaluation of various causes of anemia. So it's a rather broad-based field.
Q. When you say anemia, would one of the anemias be called aplastic anemia?
A. Yes.
Q. Are you certified as -a specialist in any particular field?
A. In hematology.
Q. And when were you certified as a specialist in the field of hematology?
A. I will have to look to be sure. I think 1974. Let me see if that's right. Yes, 1974.
THE WITNESS: Can you hang on one second while I get this telephone call?
MR. MORGAN: Yes.
THE VIDEOGRAPHER: Off the record.
(Off the record.)

THE VIDEOGRAPHER: Back on the record at 1:24 p.m.
Q. Doctor, how does one become board certified in a particular specialty in medicine?
A. Well, each specialty sets up particular requirements for their board certification. And those requirements may involve a period of training time, it may involve a period of patient experience. And they will vary from specialty to specialty. And at the time I was certified in hematology the requirement was one to two years of training specifically in hematology.
Q. And are you what is called a board certified physician?
A. Yes.
Q. All right. Are you affiliated with any hospitals, and if so which ones?
A. I am affiliated with St. Joseph Hospital.
Q. And that's where we are today, in the professional building next to St. Joseph Hospital?
A. Yes.
Q. Do you have any teaching responsibilities?
A. Yes. I am what is called the director of

medical education in St. Joseph Hospital, and I'm specifically in charge of our transitional internship here at the hospital.
Q. And can you just briefly describe for us what that means?
A. Well, in terms of the internship I recruit -- interview and recruit 12 transitional interns that spend a year here at St. Joseph Hospital and then go off into various types of subspecialty training. I'm also in charge of all of our teaching programs, each one, such as surgery or obstetrics, as a subdirector, who is intimately involved with the program. But I am the person in charge of all the programs.
Q. In your capacity as a hematologist have you had occasion to examine and treat a patient, by the name of Charles Douglas York?
A. Yes.
(Natelson Exhibit 2 21 marked for identification.)
Q. And did you, at our request and pursuant to this deposition notice, bring with you today your records concerning Mr. York?
A. Yes, I did.

Q. When did you first see Mr. York?
A. If I can look in the record to tell you that.
It would be 7-20-83.
Q. And what were the circumstances at that time?
A. Well, Mr. York came to see me for a second
opinion concerning his blood condition.
Q. All right, sir. And do you know who referred
him to you?
A. Yes. His doctor, Dr. Esslinger.
Q. Did you take a patient history from Mr. York?
A. Yes, I did.
Q. And do you have your notes concerning that
patient history?
A. Yes.
Q. Will you relate that patient. history to us
that you took from Mr. York?
A. Yes. Mr. York had been seen by. Dr. Esslinger,
basically because of weakness, and was found
to be anemic. He had undergone a bone marrow
examination about a month or so before I saw
him, and on the basis of that marrow.
examination it was thought that he might have
what's called a megaloblastic anemia, which is
due to a deficiency of vitamins. He had
received vitamins but hadn't improved.

He was then sent to Baylor College of Medicine for evaluation because that's where Dr. Esslinger was in the habit of sending his hematologic patients at that time. Mr. York had been evaluated at the Methodist Hospital, and I spoke with the physician who evaluated him there. And what Mr. York related to me was that the conclusion at Baylor was that he had a so-called pre leukemic syndrome -and that he had a marrow disorder for which there was no specific treatment and, that they would simply like -to follow that illness along to see if it, in fact, did eventually enter into acute leukemia. And I confirmed that by speaking to one of the physicians that saw him at the Methodist Hospital.

Mr. York indicated that he was employed as a pipe fitter at that time, and I specifically questioned him about drug exposures, chemical exposures that might or might not have had a role to play in his anemia; and there were no specific things that stood out in his history that were helpful.

Q. So you took an occupational history from him?

A. Yes.
Q. Was that something that you routinely did?
A. Yes.
Q. And what type of questions would you have asked him in that occupational history?
A. Well, I would have asked him what, if any, medications he had taken in the preceding three or four months. I asked him had he had any exposure to any unusual chemicals or drugs, and had he had any exposure to radiation, had he had. hepatitis, was there any family history of illnesses of the blood.
Q. And what did he tell you in response. to that?
A. Well, as I best recall, there were no unusual exposures that had relevance to this illness.
Q. Did Mr. York report to you that he had had an occupational exposure to benzene?
A. No.
Q. Did the history that you took from him, was it such that would have brought out that kind of information?
A. I think so, yes.
Q. And if he had reported to you an occupational exposure to benzene .or hydrocarbons, would you have noted that in your records?

A. Yes.
Q. And are there any such notations in your records?
A. Let me look and see what I said. There are no such notations.
Q. Did you run some tests?
A. Well, we reviewed the slides of the bone marrow that he had had done elsewhere, and also we repeated his bone marrow study to see if we could further clarify why he was anemic.
Q. Just in general terms what is a bone marrow study and what's the purpose of it?
A. Well, all the circulating cells that we have are produced in the bone' marrow, and the cells that are present in the bone marrow may look somewhat different than the ones that circulate, but we can recognize them as the precursors of those cells. And when a person has a lack of circulating blood cells, it's sometimes very helpful to look inside the bone marrow and to look at the way the cells are developing in the bone marrow. And many times that will shed light on the cause for the anemia.
Q. Where is the bone marrow located, and how do

you obtain access to the bone marrow for purposes of such studies?
A. Well, the bone marrow is located throughout many of the bones, typically centrally, such that it's highly concentrated in the sternum, in the ribs, and in the pelvic bones. As you get further out the arms and legs, the bone marrow gives out. So that when we want to study the bone marrow, we typically. put a needle into the sternum, which is probably the most cellular, area of the body in terms of bone marrow, or into what's called the posterior iliac crest, which is the back part of the hip. Now, there are certain advantages from doing it in the hip area, because you can also do a biopsy with it.
Q. And what are you looking for, the number of cells?
A. Well, we look for a number of things. We look for the numbers of cells, the total cellularity in the bone marrow. We look for the presence of cells that shouldn't be there, tumor cells. We look for the presence of other things that shouldn't be there, such as fibrosis or scar tissue. We look for the

presence of organisms that -- such as tuberculosis, for example, or footprints of organisms, such as what we call granulomas, which is a reaction of the bone marrow. We look at bone structure. There are really many things we look at, including the morphology of the cell; that is, what is the character of the cell under the microscope.
Q. What are the basic or main cells that are manufactured in the bone marrow?
A. Well, most of what is in the bone marrow are what we call myeloid cells, or white cell producing elements. We then have a large number of red cell producing elements, and a smaller number of what are called megakaryocytes, which are the parent cells for platelets. And out in the circulating blood what we see are large numbers of white cells, red cells and platelets. Those are the three general cell types
Q. And what are the roles or functions of those different blood cells?
A. Well, the red cells carry the oxygen, such that if a person doesn't have enough red cells we say they are anemic. And they may have

some symptoms of weakness, depending on how anemic they are.
The platelets can control bleeding.
And a person who lacks platelets tends to bleed easily.
The white cells -- and there are varying number of types of white cells -- in general, they fight bacterial and viral infections, depending on what cell type you are talking about.
Q. When did you do your examination of Mr. York's bone marrow?
A. I would have to look that up to see what day: that was done. Bear with me. He has had more than one bone marrow. I did one on 7-20-8:3.
Q. And what did your bone marrow by examination reveal?
A. Well --
MR. MORGAN: Excuse me. Excuse me. Are we -- I thought you were referring to the bone marrow review he did of Methodist Hospital? Are you asking for one that he performed?
MR. DILLARD: I think that was my question.

MR. MORGAN: That he performed?
MR. DILLARD: Correct.
MR. MORGAN: Okay.
A. Yes, I performed one on 7-20-83. And at that time his hemoglobin was 7.3, with the normal being about 14; his platelet count was 12,000, with the normal being around 200,000; and his white cell count was about 4,100, with a reduced number of what we call neutrophils, which are the cells that fight infection. And essentially what the marrow showed were a number of abnormalities: One. is that there wasn't enough in the way of cellularity in, the marrow, the marrow was what we call hypoplastic or hypocellular: There was a reduction in the total number of cells. In addition, when we stained the cell for iron, we saw a few of what were. called ringed sideroblasts, which are an abnormal accumulation of iron in the. red cells themselves. And we also saw the fact that the red cells look slightly abnormal in morphology, the red cell precursors. Those were the striking things that we saw.
Q. On the basis of that and your examination, did

you ultimately arrive at a diagnosis of Mr. York's condition?
A. Well, we thought that he had aplastic anemia. This was especially true when we reviewed the bone marrow biopsy.
You see, when we do a bone marrow, what happens is we remove a small amount of bone marrow and put it on a slide. And that we can look at right away. We also take a. little biopsy, or a snip of the bone marrow, that then goes to pathology and is sectioned the following day. And sometimes we see more accurate estimation of the total cellularity from the biopsy than we do from what we call., the aspirate, which is the type -- part of what we look at right away.
And when we looked at the total picture of this bone marrow, I felt that the problem was more aplasia, that is lack of cells, than dysplasia, which is abnormal cell production.
Q. What is aplastic anemia?
A. Well, aplastic anemia is a relatively rare illness in which the bone marrow becomes empty. And generally there are no malignant

cells there. We just see a lack of cell production of all of the cell lines. And when that occurs, because of the fact that blood cells live such a short time, the blood count in the peripheral blood, the circulating blood, drops very abruptly.
Q. Is it a cancer?
A. Well, it is not a cancer, no. We don't think of aplastic anemia as a cancer.
Q. Or is it a leukemia?
A. Well, it is not exactly a leukemia. It is a lack of production of cells.
Q. You mentioned it was a rare condition?
A. Yes.
Q. Can you give us some frame of reference, how rare?
A. Well, the general dogma in hematology is that in the United States there are about two cases or so, two to three cases per million per year.
So, for example, if you say Houston has, let's say, 3 million people, we might expect to see six patients a year in the city of Houston with aplastic anemia.
Q. Does it afflict both sexes?

A. Yes.
Q. Does it afflict essentially all age groups?
A. Yes.
Q. I mean, children, do they get it on -
A. It can affect children, yes.
Q. And young adults and adults?
A. Yes. It would affect all ages and both sexes.
Q. Is it a disease or is -- is it a disease or a condition? Which is the proper term?
A. I would say a condition would be the better.
Q. Is it a condition that crosses different socioeconomic lines?
A. Yes.
Q. How about countries? Are there certain countries in the world where it just doesn't exist for some reason?
A. Well, the incidence will vary. For example, it's found in much higher incidence in the oriental countries, such as Japan and china The incidence varies from country to country. But in the United States it's around two or three per million per year.
Q. And does it afflict all races or ethnic origins?
A. Yes, it can affect all races.

Q. And would it afflict all populations, all occupations, for example?
A. Well, I'm not sure I understand that question exactly.
Q. Do you know of any occupation that would, for some reason, be immune from contracting this condition?
A. No.
Q. From July the 20th, when you did the bone marrow examination, what's the next step you took in the care of Mr. York?
A. Well, generally,. when one has a patient with aplastic anemia, there are two primary avenues that you go down. And that was true in 1983 when I saw him -- it really is still true today in 1998. And we either try to go toward bone marrow transplantation, or we try to use drugs to remedy the situation.
-So we did what's called HLA typing, which is a match, to see if there was a family match for the possibility of a bone marrow ., transplant. Marrow transplants can be done outside-of family matches; but in 1983, and even today, that was a very hazardous undertaking. And, in fact, he did not have a

match. so it did not look like there was a good family match to do a marrow-transplant. The second alternative at that time, and still today, was the use of a drug called ATG, or anti-thymocyte globulin, which was made by the Upjohn Company, or ATGAM, A-T-G-A-M, was its trade name. And that particular drug had been used successfully in reversing the course of aplastic anemia.
Q. And is that the course of treatment that you followed in his case?
A. And that is the course we followed. We gave him a course of ATGAM, which is given intravenously in the hospital over several days. And there are various protocols for the treatment with that drug, various-dose levels to give; but we gave him a particular dose over about six days in the hospital.
Q. How does the ATG therapy work? What are- the mechanics of how it works in the body?
A. Well, to describe what this drug is, lymphocytes, or more specifically lymphocytes from the thymus which sits up in the chest, are injected into a horse, and the horse makes antibodies or immunity against these

lymphocytes, and then you harvest the plasma from the horse. And it can also be made from rabbits and other animals, but this material was from a horse. You harvest those antibodies, and they will destroy T-lymphocytes when injected into the body. They hone in on these lymphocytes and damage them.

And it's thought that in aplastic anemia, one of the reasons that that illness is sustained is that shifts occur in the population of T-lymphocytes and in the substances they produce that tend to suppress the bone marrow. That is to .say,. there is nothing the matter with the environment of the bone marrow, but somehow the lymphocytes are preventing growth. And if you whack them with a hammer by giving them a large amount of anti-thymocyte globulin, sometimes you cause a reversal of the process and you allow the bone marrow to recover. And at that time, depending on the dose you used and the duration, people were claiming about a 50 percent response rate in patients with aplastic anemia.

And so, in fact, that's what we did,
we gave him a course of this material.
Q. Was that done in the hospital or as an
outpatient?
A. It was done in the hospital, because there can
be some fierce side effects of that drug. It
produces a serum sickness-like reaction, and
you can get sudden anaphylactic shock and
death; you can get a lot of edema around the
body; you can get high fevers; drops in blood
pressure. And so you want to be very cautious
about giving that drug. And you like to drip
it in slowly over several hours, and you have
the precaution of certain medicines, such as
epinephrin and steroids, in case the person
has an acute severe reaction. We also
pretreat the patients with corticosteroids to
try to reduce the severity of the reactions.
And so that all entailed coming in the
hospital for several days.
Q. Do you have your discharge summary from that
hospitalization?
A. I'm sure I do. Let's see.
Q. I do, if you don't. I was just going to
A. I think I might have. Let me just see here.

Yes, I am looking at it right now.
Q. All right. And what's the date on that?
A. The date is, date of admission, 8-2-83; date of discharge, 8-9-83.
Q. I noted under the history there on this hospital discharge that you, again, make reference to the employment history of Mr. York. Is that correct?
A. I said history indicated patient is employed as a pipe fitter.
Q. okay.
A. And I also said that the patient had not been taking any medications on a regular. basis and that the past medical history was. unremarkable.
Q. I notice that it says, no etiologic factors for the aplastic anemia were established. Now, what does that mean?
A. I'm looking to see where that was that I. said that. Ah, I see. Yes, no etiologic factors for the aplastic anemia were established. Well, generally, when we look at patients who have aplastic anemia, about 7:5 percent of the time we don't discover any etiology; and we call that idiopathic, meaning

of itself. About 10 percent of the time we find a drug or a chemical that might conceivably be causative, an antibiotic or an anti-inflammatory drug, for example. And probably 8 or 10 percent of the time we find a history of hepatitis. Certain kinds of hepatitis may precede aplastic anemia. So what we are looking for are a history of chemical or drug exposures that might provide a clue as to why the aplastic anemia developed, or looking for a history of hepatitis. Those are the things that we - some of the things we are after. And, of course, a history of radiation exposure, which would be unusual.
Q. Now., did Mr. York continue under your care after he was discharged in August of 1983?
A. Yes. Because what happens when you give a person a course of anti-thymocyte globulin is there typically is no immediate benefit. That is to say, you don't give the. course of treatment and the next week everything is wonderful. It usually takes weeks or months. to get the full effect of that drug. And so what we did is- continued to follow him. We

placed him on male hormones, or androgens, which can also stimulate the bone marrow and sometimes will help to get a faster improvement after ATG. And we initially had treated him with corticosteroids, another type of hormone. And we proceeded to follow him over several months.
Q. Did you do another bone marrow on him?
A. Yes.
Q. And when was that?
A. We did one on 9-30-83..
Q. Was he admitted to the hospital for that?
A. No.
Q. Done on an outpatient basis?
A. That would have been done on an outpatient basis.
Q. Do you have your history and physical examination records from that procedure, or from that trip to the hospital?
A. Well, that-s. done in the office, in other words; and so .I. would just have my report from that.
Q. The reason I asked is I am looking at, in the records of your office that we have subpoenaed, a history and physical examination

form that's dated 9-30-83.
A. Yes.
Q. Okay.' Do you have that in your records here?
A. Probably so. Let me see if I can backtrack to find that.
I have an admission 9-29-83, and a discharge 9-30-83.
Q. All right. And, again, in the -- under the category of present illness, if you would just read the first couple of sentences in the second paragraph.
A. I said, the history indicates that a diagnosis of aplastic anemia was established about four months ago. The patient had no apparent etiology for this illness and was taking no drugs at the time of the diagnosis. Attempts were made to do HLA typing between the patient and his sister in preparation for a marrow transplant, but there was no compatibility seen. The patient was then treated with a combination of anti-lymphocyte globulin. Following treatment with the anti-lymphocyte globulin, there was, again, no significant improvement, and he was placed on long-term therapy with androgens, including

Deca-Durabolin and Halotestin.
Q. All right. And you did another bone marrow examination at that time. What did that reveal?
A. Well, that showed much more in the way of cellularity. It was more cellular. In other words, it looked like the cells were coming back into the bone marrow.
Q. In response to the ATG therapy?
A. Yes. Generally you expect to see a ,significant response in about three, three to four months afterwards. The complete response to this drug may not occur for a long period of time, for many months.
Q. When did you next see him after September 30 of 1983?
A. Well, we -- after September 30? Let me see my office notes. .
We saw him on-10-20-83.
Q. And what was the purpose of that visit?
A. Just for a continued follow-up. And what we saw at that time was his platelet count had started to improve a bit.
And then we saw him the following month on 11-16-83, and at that point the

platelets were continuing to improve, but so was his hemoglobin, the red blood cell count was improving.
And we continued to see him up until 10-25-1984. And at that time his hemoglobin had recovered to normal. It was 15.3. And his platelet count at that time was 55,000, which was not normal, but was a very satisfactory improvement over the initial values.
Q. Between November of '83, I think you said November 16, 1983 --
A. Yes.
Q. -- and October of 1984, did you have occasion to see him?
A. Yes. Let's see. I have my office notes here.
Tell me again those dates you are talking about.
Q. November 16, '83 and October of '84.
A. Well, I -saw him in January of '84, in April of '84, and then for the last time on 10-25-84.
Q. So you saw him three times, then, in 1984?
A. Yes.
Q. And did his condition improve or progress

throughout those visits?
A. Well, he showed steady improvement. In other words, what happened first was he lost his transfusion requirements. He had such a severe anemia, he needed blood transfusions. And first the anemia stabilized to the point where he didn't need transfusions; and then the hemoglobin, which is a reflection of the red blood cell count, returned into the normal range. And at that point that was the last time we saw him.
Q. And what was that date, again?
A. That was 10-24-84 -- yes, 10-24-84.10-25-84.
Q. Was he discharged from your care at that time?
A: Well, I suggested at that time that he see us again in about six months.
Q. And did he return?
A. No, I didn't see him.
Q. Was he taking any medication that you had prescribed as of the time you last saw him?
A. No. We suggested that he stop all of his medications at that point. And, parenthetically, I had relayed this material to Dr. Esslinger, who was continuing to follow the patient.

Q. But the ATG therapy that he received was only in the summer of '83?
A. Yes. Generally, that drug, it's a one-time shot. You can try it a second time, but it becomes very life threatening to give it a second occasion. You can also go to different species. This was horse anti-thymocyte globulin; and if a person doesn't respond, you can try rabbit anti-thymocyte globulin, which some people feel is a little more potent. But it's very, very hazardous to come back and try a second course of this drug again. You can get very fierce reactions.
Q. How would you characterize his response to the ATG therapy?
A. Well, he had a superb response.
Q. Is it your understanding that he returned 'to his duties as a pipe fitter?
A. I don't know what he did after I saw him. In other words, I saw him at the last time in 1984, and what happened subsequently I don't know.
Q. When you last saw him in '84, do you know if he was back a-t work at that time?
A. I don't recall.

Q. Are you familiar with the term "secondary aplastic anemia" and "idiopathic aplastic anemia"?
A. Yes.
Q. What do those terms mean?
A. Well, as I mentioned earlier, most patient's with aplastic anemia are classified as idiopathic, meaning we don't identify a specific etiology. Obviously, there may be one; but we can't identify it. And so we classify those patients as having so-called idiopathic aplastic anemia.
In other circumstances we think we know the cause. That is to say, a person may have had an episode of hepatitis immediately preceding the aplastic anemia, and we know that there is a good relationship there and we may suspect that hepatitis was the causative agent. We also may obtain a history of certain drugs that have been strongly associated with aplastic anemia that were taken, let's say, in the preceding- two, three or four months prior to the illness. And, again, by inferring the fact that we know large number of people may have gotten

aplastic anemia from that drug, we say that that's quite possible that that was the etiology in the particular case you are looking at. So that we use our clinical judgment to assign a cause.
Q. What type of drugs have been associated with aplastic anemia?
A. Well, for many years the most frequently reported association was with an antibiotic called chloromycetin or chloramphenicol. That antibiotic is still available, but not used as frequently today. And I have seen several patients with aplastic anemia from chloramphenicol, I think.
Also, the phenylbutazone, or Butazolidin was reported as causing aplastic anemia, and that drug was taken off the market.
Also Gold injections, which have been used for rheumatoid arthritis, have been thought to cause aplastic anemia. And as I say, I have seen patients who have had aplastic anemia in which we thought Gold was the etiology and also in which I thought phenylbutazone was the etiology.

Sulfonamides is another drug of sulfa antibiotics that occasionally has caused aplastic anemia. And there have been isolated reports on other drugs, such as Allopurinol or Colchicine, which are used for gout.
So, actually, a large number of drugs have been reported to cause aplastic anemia. But over the years the ones we think of immediately are chloramphenicol and phenylbutazone because the bulk of the early case reports dealt with those two drugs.
Q. And has the chemical benzene been associated with aplastic anemia in some individuals?
A. Yes.
Q. Are you generally familiar with the literature on benzene induced aplastic anemia?
A. Yes.
Q. In an individual who has been exposed to benzene and has aplastic anemia secondary to that exposure, what are the levels of benzene exposure that are thought to be required in order to do that?
A. Well, very high levels. In other words, we sometimes talk about part per million years. Like you talk about smokers have pack per year

smoking histories. And in the case of hematologic diseases consequent to benzene, many of the epidemiologicstudies suggest that you need very high total concentrations, greater than a hundred part per million years of exposure, usually in the range of 200 part per million years.
Q. What is a part per million year?
A. Well, that means that you've been exposed to that many -- you have a year's worth of exposure. In other words, it's like you were exposed every day to 200 part per million for 365 days. That gave you a part per million year. In other words, that gave you that many part per million years. So it's a cumulative exposure.
Q. In an individual who has very high exposure to benzene and who develops aplastic anemia, do you have an opinion as to the time lag or latency period between that exposure and the onset of the aplastic anemia?
A: Well, generally, we think of the hematologic effects of benzene as quite similar to those that we see with chemotherapy. In fact, benzene was originally used as a chemotherapy

drug; and it's toxicities to the marrow are very similar to what he have even today with our chemotherapy drugs. And we generally believe that there -- after a major exposure or, let's say, the last major exposure, we think the bulk of the leukemias or aplastic anemias occur within a few years. And generally by 10 or 11 years after the exposure, you are back to baseline in terms of incidents.
Q. Are these drugs; these medications that you referred to a few moments ago, thought to be significant factors in aplastic anemia today?
A. Yes. I must say, even though chloramphenicol is still used around the world, it rarely is used in the United States. And I have not seen a chloramphenicol induced aplastic anemia for many years. I have seen an Allopurinol induced aplasia. That's probably the most recent one that I saw that I thought a drug was responsible. But chloramphenicol is not very widely used anymore, and phenylbutazone has been taken off the market, so that one wouldn't expect to see that problem here in this country.

Q. Have you had occasion to treat a patient with benzene induced or benzene caused aplastic anemia?
A. No, not to my knowledge.
Q. In the literature is benzene considered to be a significant factor today in terms of causing aplastic anemia?
A. No. It would be considered to be indistinguishable from baseline. In other words, most studies of aplastic anemia, looking at large numbers of patients with this illness, don't list any cases from benzene.
Q. We were taking a look at your records as we were about to start the deposition a few moments ago.
Have you had occasion to see Mr. York since October 25th of 1984?
A. Yes.
Q. And when was that?
A. That was on January the 15th of this year.
Q. And what was the purpose of that?
A. Well, Mr. York called and said that he would like me to see him as a patient and reevaluate his situation.
Q. Did you examine him at that time?

A.
Q. And what did that examination consist of?
A. Well, I took a history of what had happened to him since the last time I saw him in 1984 and I examined him briefly on physical examination and we did some laboratory work.
Q. And what did the physical examination consist of?
A. I examined his abdomen to see if I could feel if his spleen or liver were enlarged or if he had any swollen lymph nodes.
Q. What did that reveal?
A. The physical exam were normal.
Q. And what lab work did you run?
A. We ran a complete blood count and a routine chemistry panel.
Q. And do you have those results today?
A. Yes.
Q. Okay. We are going to want to get a copy of those before we leave. But, if you would, summarize for us the results from the laboratory analysis.
A. Well, in terms of his blood counts, his blood counts were perfectly normal. He had a hemoglobin of 16.1; a platelet count of

215,000; and a white cell count of 5,500; and the differential cell count, meaning the distribution of white cells that he had, was normal. So his -- and his blood looked normal under the microscope. So essentially he had a normal blood count.
In terms of the chemistries that we obtained, these consist of a variety of things, including blood sugar and calcium levels and protein levels and so on. And the only notable abnormality -- abnormalities detected were that his cholesterol level was a little bit elevated at 225, and the recommended normal is up to 200; and a liver function test, the SGPT, was elevated at 9.5, and the upper limits of normal in this laboratory are 50.
Q. By history, did you obtain any additional information from him that you had not gathered before when you had seen him 13, 14 years earlier?
A. Yes, there were several pieces of history. He indicated that in the ensuing years -- and I've forgotten which year this was -- he had some trouble with coronary artery disease and

required an angioplasty procedure on his heart. He also indicated that he had chronic bursitis with his right shoulder and had required steroid injections on several occasions for the shoulder. He also indicated that he had chronic hepatitis, and that he had had a liver biopsy -- and I believe he said the most recent biopsy was about two years ago -- and that he had seen a gastroenterologist for the liver situation. Those were the things that stand out -- oh, and that he had changed occupations, and that he was working in an attorney's office now.
Q. As far as his blood counts were concerned, you said the platelet count was what, 215,000?
A. 215,000, yes.
Q. And that that is a normal platelet count?
A. Yes. Generally, the normal platelet count, the lower limit of normal is usually given as 150,000, and the usual normal range is, say, 150,000 to about 350,000.
Q. Did you evaluate the appearance of the platelets?
A. Yes. We stained a slide and I looked at it under the microscope and I thought it was

within the range of normal.
Q. How about the size of his platelets, was there anything abnormal about that?
A. Nothing striking on examination of the slide.
Q. Was there any evidence that he had aplastic anemia?
A. Not now, no.
Q. To your knowledge, between the time that you had last seen him in 1984 and you saw him again here recently in 1998, had he had any relapse or recurrence of aplastic anemia?
A. No. He indicated that his blood counts had been fine during that period.
Q. I think the record will pick up what you said, but there was a page right about that time.
A. All right. Okay. I asked him about his blood counts, and he indicated that his blood counts had been essentially normal. in the ensuing years since I saw him.
Q. Are there other patients that you have had occasion to follow for this long a period of time that have had aplastic anemia and received-the ATG therapy?
A. Yes.
Q. What kind of a track record do you have with

that?
A. Well, you know, it's interesting. If you look at the literature on the subject, it says that perhaps the response rate is 50 to 60 percent. I guess I may have a selected population. I would say, in my experience, it has been closer to about 70 or 80 percent have had good responses to it. So that, obviously, some people don't respond, and I suppose if I treated 5,000 patients I might arrive at the 60 percent figure. But for the numbers that I have treated with it, I have had better than a 50 to 60 percent response rate to it.
Q. And Mr. York was one who did respond?
A. Yes.
Q. For those that do respond to it -
A. Yes.
Q. -- like Mr. York, have you had any of them relapse
A. No, I have had no relapses.
Q. Based upon what you know about Mr. York's case and the opportunity that you have had to follow him over this period of time, I want to ask you if you have any opinions about his prognosis from the standpoint of his aplastic

anemia.
Is there any reason that you have,
based upon what you know and what you have:
seen, to believe that he will have a relapse
in the future?
A. Well, I don't see anything about his blood.
counts now that would suggest that he would
have a relapse.
Q. And in terms of reasonable medical
probability, what would your opinion be?
A. Well, if you look at the large populations of
people that have been treated for aplastic
anemia, and treated successfully, there is an
increased incidence of hematologic abnormality
over the ensuing years.
And, for example, I can think of in
my files I have one study of 500 and some odd
patients followed longitudinally with aplastic
anemia, and I believe about -- and don't hold
me to the exact numbers -- but about, say, 19
developed a peculiar condition we call PNH,
which is where the red cells develop abnormal
membranes, and they undergo breakdown very
easily.
Also, in that same group about 11 of

those patients later developed myelodysplasia;
and I believe in about five of those patients
they went on to develop acute leukemia.
And we generally think that in
people who have had aplastic anemia there is a
slight increased incidence over the general
population of further hematologic diseases,
such as leukemia or PNH. In other words,
these numbers would come to just a few
percent; but a few percent is much higher than
what we see in the general population.
Q. Given that it's a small percent, in terms of
reasonable medical probability, what's the
future outlook for Mr. York?
A. Well, I think in reasonable medical
probability his blood counts will remain
normal.
is Q. You mentioned that he reported having
bursitis -
A. Yes.
Q. -- in his right shoulder?
A. Yes.
Q. For which he was receiving injections?
A. Yes.
Q. Do you know what they were -- it was a

steroid?
A. He said he had had steroid -- several steroid injections in his shoulder.
Q. And did he indicate to you when the bursitis problem started?
A. He may have, and I don't recall exactly what he said about that in terms of what he's told me.
Q. Had he reported a bursitis problem to you at any time back in '83, '84 time frame?
A. Well, one of the expected side effects of anti-thymocyte globulin is a diffuse inflammation of all of the joints. And after we gave him -- gave it to him, he had that. In other words, he had swelling of his knees, swelling of his elbows, and he had generalized inflammation of his joints back in 1983. And that subsided. Generally it subsides about two or three weeks after you have given the drug. It seems to peak at about a week or two after they leave the hospital, and then starts to gradually subside.
Q. And did it subside in his case?
A. Yes.
Q. Do you have an opinion, again in terms of

reasonable medical probability, as to whether there is any link between that ATG therapy back in 1983 and the bursitis that he is reporting in his right shoulder now?
A. I would doubt it, because this is a single joint problem. And, of course, bursitis in the shoulders is a very common problem. And I would think that if it was, let's say, drug related, I wouldn't know why it would simply be in one joint and not multiple joints. I think that would probably be very unusual. So I would say, in my opinion, it would not be related to the ATG.
Q. And would the passage of time be of significance to you, between the week or two that you said where the peak occurs, if it has such effect?
A. Well, generally, the inflamed joints that one sees after ATG are an acute phenomenon. I really have not seen anybody that I have followed that I have given the drug to who has a chronic illness in their joints from it., So I don't think of that as a toxicity from ATG.
Q. Now, this one -- was it one liver test that was elevated?

A. Yes, of this group. Several of the tests -:hat he had could be considered liver tests. We measure the bilirubin and a test that's called the alkaline phosphatase and the total proteins and so on. And one particular one was elevated, the SGPT.
Q. He said he last had a liver biopsy two years ago?
A. I believe that's what he told me.
Q. Do you have any knowledge of what that showed?
A. Only what he told me about it. I don't have the report.
Q. Is it your understanding that his hepatitis is in an active phase, or is it a chronic phase, or how would you describe it?
A. Well, he indicated to me that-he was told that he has what's called chronic persistent hepatitis as opposed to chronic active hepatitis. In recent years, those two terms, the distinction between them is somewhat blurred. But based on what he told me, the abnormalities in his liver were mild, and his physicians did not recommend specific treatment for the hepatitis.
Q. Did he report any history of having been

diagnosed with cirrhosis?
A. No.
Q. And are you familiar with the medical literature, as far as the percentage of patients who have chronic hepatitis C who develop cirrhosis?
A. Yes.
Q. And what does that demonstrate?
A. Well, in general terms, the dogma that we use today is that after an acute, episode -- or after an acute episode of hepatitis with hepatitis C, about 80 percent of patients develop some form of a chronic phase of the illness. And in probably around 20 percent of those patients, cirrhosis ultimately develops at an average of about 15 to 20 years out from the date of the episode of hepatitis.
Q. Again, looking at Mr. York's situation and knowing what you know about him and the opportunities that you have had to see him, do you have an opinion, in terms of reasonably: medical probability, as to whether or not he would fall in that 20 percent?
A. Well, as I say, I haven't seen the liver biopsy. I don't really have any reports from

his gastroenterologist. I guess I would have the defer to the gastroenterologist on that in terms of an opinion. Although, I must say from what he is telling me the liver biopsy findings are very minimal at this point, and that would certainly be in his favor.
Q. In this disease, hepatitis C, if a patient does not develop cirrhosis, does the issue of liver transplant arise?
A. No. We typically consider transplant in circumstances where the patient develops cirrhosis or develops liver cancer. And some centers still try to transplant. livers for liver cancer development, if it's a very localized lesion. But, primarily, liver transplants are done for symptoms of liver. failure.
Q. All right. We talked earlier about idiopathic versus secondary aplastic anemia. Do you :have an opinion, in terms of reasonable medical probability, as to the cause of Mr. York's aplastic anemia?
A. Yes.
Q. And what is that?
A. I think that in looking at the entire picture,

I would feel comfortable with the diagnosis of idiopathic aplastic anemia.
Q. And why is that?
A. Well, in looking at the common etiologies. In other words, we have no evidence that he had hepatitis preceding this illness, we didn't uncover any particular drugs that were at fault, we know that he had an outstanding response to the anti-thymocyte globulin and has persisted in remission. And that suggests to me that this is an -- that his disease was basically an immunologic problem.
In the case of chemicals, what we are talking about is basic stem cell damage. In other words, the aplastic anemia that you see from benzene is really a form of myelodysplasia. And I would not think that someone who had that from benzene would get better and have a sustained improvement in their blood count with anti-thymocyte globulin.
Q. Which Mr. York did?
A. Which Mr. York did.
MR. DILLARD: Thank you, sir.
That's all I have got right now.

While they are conferring, could I look at your records?
THE WITNESS: Sure.
MR. SCOTT: Steve, are you still going?
MR. DILLARD: Oh, no. I said I pass the witness.

EXAMINATION
QUESTIONS BY MR. SCOTT:
Q. Dr. Natelson, my name is Bob Scott, and I have just a couple of questions.
You have used a term "idiopathic," and can you tell us what that means in lay terms?
A. In lay terms, it means of itself. We use that term a number of times in medicine. In other words, we talk of idiopathic thrombocytopenic purpura. That's a situation where the platelet count goes down. And we don't know specifically what set the illness off. It's of itself, so to speak, and we don't have a specific etiology.
In the case of aplastic anemia, we use that same term "idiopathic," meaning that

we cannot assign a specific cause to it based on our level of knowledge.
Q. You also discussed with Mr. Dillard the platelet size, I think, which you have seen from the blood counts and so -- in examination of Mr. York's marrow or blood. Have you seen any indication that Mr. York has either large or small platelets?
A. Well
Q. Smaller than normal?
A. -- there is a range of normal. In other words, platelets are typically about, oh, they are smaller than what we -- what we see in the blood as small lymphocytes. We usually use the lymphocyte as a reference point. We look at the diameter of the small lymphocyte and then look at the diameter of the platelets and see how close they correlate. And the platelet is about the fifth of the size of a lymphocyte. And there is a normal variation. And in people, for example, who have a high platelet turnover, the platelets are coming out of the bone marrow too fast, they may be large. And there are certain hereditary syndromes in which they are too small.

And I would say from my looking at his peripheral blood film, I was not impressed that there was anything out of the ordinary in the distribution of his platelet size.
Q. You also indicated that you don't think you have seen a case of benzene induced or caused aplastic anemia in your career; is that true?
A. That is correct.
Q. Do you have an explanation for that, given that, at least as you have indicated, there are reports in the literature of benzene induced aplastic anemia?
A. Well, there are certainly reports; but many of these date back in what I would call older literature. Let's say before I was a hematologist. And they date back to the times that we had very little control on benzene exposure in our factories. And with time, control of benzene exposure has improved and the hazards have been recognized and the companies have taken steps to modify that. So in many areas, we haven't seen hematologic problems from benzene for a long, long time.
Q. All right. And you have been a hematologist since 1966?

A. Well, I became a physician in 1966. I would say along around 1968 I began to really become exclusively interested in hematology.
MR. SCOTT: I think that's all I have right now.

EXAMINATION
QUESTIONS BY MR. CARRINGTON:
Q. Dr. Natelson, my name is M.C. Carrington. I have a couple of additional questions for :you, as well.
Earlier I believe it was Mr. Dillard asked you about Mr. York and when he returned. to work. I was looking at your records. 0n his January 17th, '84 visit to you, did he indicate to you that he was back at work full-time?
A. Let me just look at my note. '84. Yes, I did make that note. I said, Mr. York continues to do well since his recovery from severe aplastic anemia and is now working on a full-time basis.
Q. And I assume you obtained that history or information from Mr. York?
A. Yes.

Q. Certainly, I would assume, based on that, that as far as you were concerned Mr. York was fit to work from your medical standpoint if he was able to do the work from his standpoint?
A. Yes.
Q. And at any other time after that did you ever suggest to Mr. York that he not return to work?
A. No.
Q. Did you ever suggest to Mr. York he not return to work as a pipe fitter?
A. No.
Q. Let me back up. You described a benzene induced aplastic anemia as causing damage Ito the stem cells.
A. Yes.
Q. Is that an acute type reaction, an acute situation, something that occurs with some degree of a rapid onset?
A. Well, exposure to benzene, high levels of benzene, can produce an abrupt aplasia which is temporary. And the patient may have a :Fall in their blood count and may recover from it. But then later on, a few years later, may (jet another fall on their counts as their bone

marrow becomes further abnormal. So that it may be an acute event, or it may be sort of a subacute event, you might say, from the benzene exposure.
Q. Does that vary depending on the amount of exposure?
A. Oh, I think in part it depends on the amount of exposure. In other words, the highest exposures would give you the abrupt drop in white blood cell count. And they may produce damage that isn't fully evident for some few years after that exposure.
MR. CARRINGTON: That's all the questions I have. Thank you, Doctor.
MR. MARTIN: I have no questions.
MR. MORGAN: Steve, do you have anything else?
MR. DILLARD: No, not until after you question him.
MR. MORGAN: Why don't we take a break for a little bit.
THE VIDEOGRAPHER: Off the record at 2:27 p.m.
(Brief recess.)
THE VIDEOGRAPHER: Back on the

record at 2:37 p.m.

EXAMINATION
QUESTIONS BY MR. MORGAN:
Q. Dr. Natelson, my name is Glen Morgan, and I represent Mr. York and I have a few questions for you. Some I thought of myself and others were prompted after I got here by some of the questions these other lawyers asked you. will try to be as brief as I can; and if you don't understand any questions I ask you, let me know that so I can try to rephrase it, perhaps repeat a different question so that we can be on the same wavelength. Okay?
A. All right.
Q. Just to get a little more background information on you. Since we-are here talking about a case where Mr. York alleges exposure to benzene as being a cause of the aplastic: anemia, I need to ask you about your experience in terms of writing articles. Have you ever written an article about the relationship between benzene and aplastic anemia?
A. Not specifically. I think I did write an

article on a review of aplastic anemia on one occasion for our St. Joseph Hospital Journal, but not specifically in relation to benzene.
Q. All right. What about have you presented any type of lecture or other type of presentation to either a peer group or others concerning benzene and the cause or relationship between that chemical and aplastic anemia?
A. Not specifically benzene. I have talked about aplastic anemia, but not specifically in relation to benzene as an isolated event.
Q. Have you ever conducted any studies concerning the exposure to benzene in resulting aplastic anemia?
A. No.
Q. The relationship between benzene and aplastic anemia-has -been known for many years, hasn't it?
A. Yes.
Q. And benzene, I think you would agree, is clearly established as a causative factor in . aplastic anemia?
A. Yes.
Q. The literature -- and you mentioned earlier something about the older literature -- dates

back to the late 1800s and early 1900s, describing this relationship, doesn't it?
A. Correct.
Q. You were in medical school, I believe you said, in the '60s?
A. Yes, 1962 to 166.
Q. And, of course, 60 or 70 years after this older literature we're talking about, I assume that they were still recording that relationship in the medical books that you were studying from?
A. Yes.
Q. Your textbooks at the time had that information in it?
A. Yes.
Q. And I'm sure, again, that you learned it there, and I guess you continued throughout your internship, et cetera,. to .be aware of that relationship?
A. Yes.
Q. Now, you mentioned something about being a hematologist. Are you an immunologist?
A. No.
Q. Are you an occupational medicine specialist?
A. No.

Q. What is the difference between immunology and hematology?
A. Well, immunology has to do with the study of the functions of the immune system specifically. And it may have to do with the cytokines and materials that lymphocytes make. It may have to do with the defense system the body has against foreign antigens. It basically has to do with your immune system.
Q. Do you find immunologists involved in the treatment of aplastic anemia?
A. Not commonly, no.
Q. And then occupational medicine specialist, how does that differ from hematology?
A. Well, an occupational medicine specialist might be seeing a variety of occupational injuries, from fractures and sore backs to skin rashes to blood related problems. A hematologist is more specific in the area of blood related problems.
Q. All right, sir. And I take it you are not an industrial hygienist?
A. No.
Q. You don't have any training in that area?

A. No.
Q. Also, I assume that you are not trained in the treatment of liver disorders. You mentioned earlier that would be -- and you would refer to or defer to a gastroenterologist for that?
A. Well, it depends on what you mean by trained. I'm a board certified internist, general internist; and in our training, liver disease is a common medical problem. I personally, in my training, did about 60 or 70 liver biopsies. And so I consider myself somewhat knowledgeable in terms of liver disease, but I don't specialize in their treatment.
Q. Well, I guess perhaps my phraseology was a little wrong. While you may have received training in a lot of areas, you basically specialize your practice to the practice of hematology?
A. Yes.
Q. And you have done that for how many years now?
A. Well, since about 1969.
Q. All right. So for the last almost 30 years you have been treating people who have blood disorders, among those of which would be aplastic anemia?

A. Correct.
Q. Now, do you know what a pipe fitter does for a living?
A. Not entirely.
Q. Do you know -- have you ever been to the Mobil facility in Beaumont?
A. No.
Q. Have you ever been to the Mobil chemical facility in Beaumont?
A. No.
Q. Were you ever at the -- well, just let me list some of these plant sites that are involved in this case and see if you have ever been there. I will just list them all, and if I come to one you have actually been to the premises, well, then stop me.
Either Gulf or Gulf Chemical, Texaco or Texaco Chemical, or Jefferson Chemical, Fina, or might be American PetroFina, Velsicol, or Union 76. Any of those facilities?
A. No.
Q. Have you ever requested documents from any of those facilities I have just mentioned concerning the benzene or the presence of

benzene or the aromatic hydrocarbons on their premises?
A. No.
Q. In Mr. York's case, from the occupational history standpoint, you realized he was a pipe fitter. Did you request any work records from him or from any of his employers to show what he may have been doing for a living?
A. No.
Q. You mentioned that this was a referral from Dr. Esslinger?
A. Yes.
Q. What type of relationship did you. have with Dr.' Esslinger back at that time, that was in the early '80s?
A. Well, the relationship was like this, Dr. Esslinger was in the habit of referring his hematology patients to Dr. Robert Heddig, who died a number of years ago, who was the chairman of hematology at Baylor. And as a fellow in hematology, I would often see these patients with Dr. Heddig. And I assume that's how Dr. Esslinger knew my name, and he suggested that I see this patient. But I have never personally met him or had any specific

dealings with him.
Q. Are you aware that Dr. Esslinger was, at one time, the company doctor for Mobil?
A. No.
Q. You mentioned -- you mentioned that you have been practicing hematology for almost 30 years; and as I understand, you've been testifying in cases involving benzene exposure for almost 20 years; is that right?
A. No. I would think that that's probably not true. I don't know how many years I have offered testimony in -- I have given testimony on a variety of subjects In the case of benzene, I can't tell you when I first gave testimony in benzene; but it certainly wasn't 20 years ago.
Q. Do you recall testifying that your testimony in the different types of trials could date as long as 20 years?
A. Correct.
Q. All right.
A. But those trials may be anything from subdivision suits to malpractice suits. And most of those from 20 years ago had nothing whatsoever to do with benzene.

Q. Well, you are typically retained as an expert witness approximately six to eight times per year; is that correct?
A. That would be probably true, yes.
Q. And as I understand it, the majority of the cases in which you are consulted, you consult on behalf of the defense?
A. The majority, yes.
Q. For instance, in this case, who first contacted you about giving your testimony in this case?
A. It would be Mr. Dillard.
Q. Is this the first time that you have had occasion to work with Mr. Dillard on a case?
A. No.
Q. On how many other occasions have you dealt with Mr. Dillard?
A. I can't tell you exactly. I recall a case that, I believe, the patient's name was Morin, M-O-R-I-N; and that would be what comes to mind in terms of dealing with Mr. Dillard.
Q. Have you ever met Mr. Dillard outside the office setting?
A. Outside my office or his office?
Q. Yes.

A. Yes, I have been to his office, I believe, on one occasion.
Q. Have you ever met him outside his office or your office?
A. No.
Q. Have you ever testified in trial for Mr. Dillard?
A. I don't believe so.
Q. Has Mr. Dillard referred cases to you in the past to review where you have not given a deposition?
A. Possibly.
Q. Well -
A. I don't recall. In other words, I have seen cases for various attorneys in which I'm never asked -- I'm asked to give an opinion, but never deposed and never testified at trial. And I don't keep any particular record of those.
Q. Do you know what firm Mr. Dillard is with?
A. Fulbright & Jaworski.
Q. Do you do work for other attorneys in that law firm?
A. I don't know. I don't pay much attention 'to what firm the material comes to -- comes from.

Q. Comes from. Well, you knew Mr. Dillard.
A. Yes.
Q. On how many occasions would you .s ay that you have dealt with him, then?
A. Certainly no more than three occasions; maybe not even that.
Q. Over what time period?
A. Forever. In other words, I have perhaps discussed a case on the phone with him. I recall this Morin case that had to do with - in part with hepatitis C. Certainly no more than a very handful of cases.
Q. What about Mr. Scott, do you know Mr. Scott:?
A. I met Mr. Scott.
Q. Have you worked with Mr. Scott or anyone from his law firm on other cases before this one?
A. Tell me what his law firm is, and I -
MR. SCOTT: Abrams, Scott & Bickley.
A. I may have. I don't recall a specific case.
Q. All right. What about Mr. Carrington, who is also here? He's with the law firm of Mehaffy, Weber. They have both a Beaumont and a Houston office.
A. I'm certain I have not worked for his law firm.

Q. And what about Mr. Martin, who's here for Jenkins, Grove & Martin?
MR. MARTIN: Close enough.
A. I'm not familiar with that law firm, either.
Q. All right. At one time you were charging $200 an hour to testify. Has that rate changed?
A. No.
Q. Is it the same?
A. It could be the same, yes.
Q. Is it the same in this case?
A. Yes.
Q. And you charge that amount for just reviewing the records -- I mean, I'm sorry, for just testifying or also for reviewing the records?
A. For reviewing the records.
Q. How much time do you think you have had in this case so for, up to now?
MR. DILLARD: You are talking about his treatment of Mr. York?
MR. MORGAN: No. I'm talking about his expert testifying time that you've asked him to do.
MR. DILLARD: We're hereto take his deposition.
A. I really haven't discussed the case. In other

words, I mean, I have seen Mr. York again in the office for an hour or so. The deposition has been set a couple of times, and so I have gotten the records ready that I had. But not a large amount of time.
Q. Well, are you talking 10 hours, up to the deposition?
A. Certainly less than that.
Q. Have you ever talked to any type of oncologist or hematologist in the Beaumont area concerning what knowledge they may have concerning work sites in the area?
A. No.
Q. Have you done any research? .And I don't mean formal research, but any inquiries, casual conversation, or anything like that, with medical provider, be it a doctor, hospital, nurse, concerning the chemical situation as it would exist in the petrochemical area of Jefferson County?
A. No.
MR. DILLARD: Objection, vague.
Q. The money that you charge, I take it, goes to you directly, and in your pocket, as opposed to some research facility?

A. Correct.
Q. You are not employed by St. Joseph's Hospital?
A. Yes, I am.
Q. Your salary is paid by them?
A. Well, yes and no. Part of my salary is. I'm what's called a contract employee, and they pay me a salary for being the director of medical education.
Q. All right. But in terms of your work responsibilities throughout the day and the patients you see, those are your patients, not the hospital patients?
A. Correct.
Q. Have you requested from Mr. Dillard any type of exposure information that he may have concerning Mr. York and his work years?
A. No.
Q. Did Mr. Dillard offer to send you any information along those lines?
A. No.
Q. Have you seen Mr. York's deposition testimony?
A. I don't believe so.
Q. Would you find it beneficial to have all of the information that exists concerning Mr. York's work life, his exposure history to

benzene, et cetera, in order to make an opinion about the cause of his condition?
MR. SCOTT: Objection to the form of the question. It assumes such exists.
A. Well, I think that -- in his particular case, I think we have the long follow-up now. And I think I'm satisfied with the original information I got. And seeing what the years have allowed to happen, I feel confident that this problem is not one that's consequent to benzene.
Q. So it's your testimony, then, that you do not need any exposure to benzene evidence that may exist as, in your opinion, is it not relevant?
A. To this particular case, yes.
Q. And that's because of the response to the treatment that he received, the -- you call it the ATGAM?
A. ATGAM or ATG, anti-thymocyte globulin.
Q. And had it not been for that response, or had his response been due to something else, then we would look back to the benzene, then, as the potential cause, wouldn't we?
A. No, not entirely. Because his bone marrow findings were not typical of benzene aplastic

anemia.
Q. Okay. I will get into all that, but in terms of the causes that you mentioned earlier, you mentioned hepatitis, you mentioned drugs, ,and you mentioned chemicals, correct?
A. Yes.
Q. Now, if it's -- we know of no history of hepatitis that preexisted the diagnosis of aplastic anemia, correct?
A. Correct.
Q. And we know of no drugs that were used that preexisted the aplastic anemia diagnosis that would have caused aplastic anemia, correct'?
A: Correct.
Q. And, by the way, the drugs, if -- you know that's an immediate response, isn't it?
A. Well, within a few months. In other words,, weeks to months.
Q. Okay. As opposed to a chemical exposure, which you could have exposure over time that,, just as you described, a latency period could exist of many years in some cases?.
A. Yes.
Q. Okay. So what I'm saying is, is if the response -- if Mr. York's condition changed,

but it wasn't as a result of the ATG, then, wouldn't that necessarily, in light of the history of these other conditions not existing, indite some chemical type of exposure?
A. You lost me on that question. You will have to repeat that one.
Q. Okay. So basically you think it's non-benzene related because of the response to the AT GAM treatment?
A. And the character of the bone marrow diagnosis.
Q. Okay. Now, my question deals with -- for instance, are you aware -- let me go back. What's the difference between ATGAM and ALG instead of ATG?
A. ATG -- well, the initial preparations were called anti-lymphocyte globulin, meaning that lymphocytes were used to inoculate the animal to make the antibodies. Well, thymocytes are lymphocytes. They are a specific type of lymphocyte. There are more T-cells in then, so that anti-thymocyte globulin is anti-lymphocyte globulin, but anti-lymphocyte globulin is not always anti-thymocyte

globulin. Thymocytes are a particular type of lymphocyte that tend to excite a response in the horse that produces a high titer anti T-cell immune response. And so the preparation is referred to as ATGAM, or anti-thymocyte globulin.
Q. All right. Now, which treatment was Mr. York given?
A. He got ATGAM, which is Upjohn's product. And I guess you would have to question Upjohn as to exactly how they made that product, but my understanding is that that was an equine - was a horse anti-thymocyte globulin.
Q. Could you look at your records where you describe the treatment for me?
A. Which particular record
Q. Earlier you read something about the treatment. Perhaps it was on the discharge summary.
A. Well, I said here, he has been treated for this with blood transfusions, anti-lymphocyte globulin, and androgens. Is that what you're talking about?
Q. Well, that certainly would be one reference. Does that tell us which type of treatment he

received?
A. No. Because, as I say, anti-thymocyte globulin is anti-lymphocyte globulin.
Q. All right.
A. And anti-lymphocyte globulin is the more general term.
Q. So it just -- it's the same thing, you are saying, then?
A. Yeah.
Q. In essence?
A. Yes.
Q. Do you know who Dr. Neil Young is?
A. Yes.
Q. Who is he?
A. I believe he's with the NIH. He has written a number of articles on aplastic anemia.
Q. Would you consider him to be an expert or an authority in the field of benzene and aplastic anemia?
A. Well, I don't know that anybody is a particular authority in hematology. He has written some articles, and I have read some of his material.
Q. You don't consider him to be an authority in the area?

A. I wouldn't consider anybody to be an authority in the area. In other words, every doctor learns by experience. And one doctor's experience may be different from another doctor's experience. And what you try to do is you may gain a piece of information from one article and a piece of information from another and there may be errors in one article or errors in another and you try to amalgamate what fits from the literature with what fits from your clinical practice and your knowledge and your attendance at lectures and so on. And that's the way you conduct your practice. You don't set yourself up to follow one particular knight on a horse as this is the only way to do something or this is the only cause of an illness. We try to look from all areas of the medical literature.
Q. So I take it that you do not think that Dr. Huang is an authority in the field, either?
A. He's written many articles in the area.
Q. He's not an authority?
A. He's an expert, but he may not be the ultimate authority in that area.

Q. And you do not consider him to be an authority, do you?
A. He has written many articles on the subject. Tell me what you believe an authority means, and I will tell you if that's what that means.
MR. MORGAN: Well, first let me object to the responsiveness of my question.
THE WITNESS: Okay.
MR. MORGAN: Your answer to my question.
Q. Doctor, what is an authority in the field?
A. I don't know. You tell me. My -- I don't like to use the word "authority," because it gives the impression that that is the dogma, that that is the only way. To me, I like to use the term "expert." A person has wide experience in the area, but it doesn't always mean that what he or she says is correct.
Q. All right. So you are saying, then, that the word "authority" is just a word that you do not use?
A. I don't like the -- yes, I don't use that word.
Q. The word you use as --
(Brief interruption.)

Q. So as opposed to authority, you use the word "expert"?
A. I use the word "expert."
Q. All right. Would you consider Dr. Young to be an expert in the field?
A. Yes.
Q. He is the director of hematology for the National Institute of Health, I believe; is that correct?
A. I don't know what his titles are.
Q. And what about do you know Dr. Sinsenbruner?
A. No.
Q. Or of him?
A. No, I don't recognize the name.
Q. Would you agree that a chemically induced aplasia can respond to ATG treatment?
A. Well, certain kinds of aplasia may respond to ATG, at least according to Dr. Young.
Q. So you are aware that Dr. Young is of the opinion that virus associated aplasias can respond to the ATG treatments?
A. He is of the -- he has published some abstracts I have seen in which he has treated a number of patients with ATG that he -- that have various forms of myelodysplasia. And he

has short-term results indicating that they have an improvement in their blood counts. I believe the last abstract I saw, some of these patients, the maximum was about six or 700 days post therapy.
Q. When a doctor uses the term "rule out," what does that mean?
A. To exclude.
Q: All right. Did you have any type of ruling out to do in this case when you saw Mr. York, when you first saw him?
A. Well, in the case of aplastic anemia you are looking for common things: That is to say,, if Mr. York had come in and said; I just finished my bottle of chloramphenicol a few weeks ago, I would say, gee, it's quite possible chloramphenicol might have caused that aplastic anemia. That might be a close association, but he gave me no such history. So ruling out in the terms of aplastic anemia is difficult because there isn't any one chemical test that we use to assign aplastic anemia to one category or another.
Q. Well, I understand. But what you do is by the

process, I guess, of elimination you start narrowing down the potential causes, don't you?
A. Yes. We go on the basis that common things occur commonly. And we try to see if the illness fits into the common pattern that we identify.
Q. All right. Well, let's go through what you would have gone through. And you have told us the causes of aplastic anemia.
A. Yes.
Q. So I assume we start there. You knew when Mr. York presented himself in your office that he had aplastic anemia?
A. Correct.
Q. And I believe you used the term "severe aplastic anemia"?
A. Yes.
Q. Now, that disease, untreated, results in death, doesn't it?
A. Correct.
Q. Because the factory of our cells, the bone marrow, is not producing the cells. And once our cells in our body start dying off, as they normally do, there is nothing to replace them

and we die, correct?
A. Well, we die, not specifically from -- not directly from the loss of cells but the diseases that that causes. In other words, if you don't make any white cells, you get infected and may die of infection; if you don't make any platelets, you may bleed to death.
Q. For instance --
A. So that that's what the cause of death usually is in aplastic anemia.
Q. For instance, a common cold could result in someone who has aplastic anemia in death?
A. It might.
Q. Same thing, shaving, cut yourself -- I mean, I know it has to be a severe cut -- but basically bleeding to death would be --
A. Bleeding to death usually is intracerebral bleeding, such as like a massive stroke or hemorrhage that might cause death.
Q. Okay. So we are dealing in 1983 with a potentially fatal condition.. I assume you advised Mr. York of that?
A. I don't recall the conversation, but I probably did.

Q. All right. And in trying to find the cause -- well, first of all, does the treatment depend upon the cause?
A. Well, to a certain extent. In other words, if we feel like that a person has aplastic anemia from Gold, for example. That Gold is in the system. We can remove it by chelating agents. And some believe that chelating therapy helps the recovery of Gold induced aplastic anemia. And I have done that years ago on a patient with that problem.
In the case of a drug, you try to remove that drug from further use in the patient's case in the hopes that the marrow may spontaneously recover.
In the case of certain of these other illnesses, such as hepatitis, we don't have a specific treatment that might help.
Q. Okay. Let's go through now, look at the first time you saw him, your notes from that first visit.
A. All right.
Q. And let's be. in your office, Mr. York presents himself with aplastic anemia, severe, it's potentially life-threatening, and we want to

try to find the cause. I assume you asked him if he had taken any of the type of drugs that we mentioned?
A. Correct.
Q. And what did he tell you?
A. He said no.
Q. All right. Now, benzene is not a drug, is it?
A. No.
Q. Okay. So drugs, we are talking about aspirin, prescribed medication from a doctor, things like that?
A. Yes.
Q. Okay. So your inquiry as to the drugs, basically, was negative?
A. Correct.
Q. The responses. were negative.
The other thing that was hepatitis, I believe you mentioned.
A. Yes.
Q. Did Mr. York have any evidence of preexisting hepatitis at that time?
A. He had no history suggesting the hepatitis.
Q. Okay. Now we are left with chemical exposures. Can you tell me what the history. was from the chemical exposure standpoint?

MR. DILLARD: I'm going to object to the characterization "we are left with," his testimony about the cause of aplastic anemia being unknown in most cases, object to that.
MR. MORGAN: Excuse me. I thought we were reserving everything except to the form of the question.
MR. DILLARD: Well, I think it misstates his testimony.
MR. MORGAN: Well, that's supposed to be reserved.
MR. DILLARD: Okay.
Q. Go ahead.
A. So we are talking about his chemical history. Sure, I asked him if he was exposed to any chemicals in his work, and he said no.
Q. Well, show me in your records where it says that.
A. I can't show you that.
Q. Why can't you show me that?
A. Well, I didn't -- I don't -- well, maybe I have it written somewhere, but -- and let's see. In my letters to Dr. Esslinger, let's see what I said.
I said he had -- patient had no

apparent etiology for this illness and was taking no drugs at the time of the diagnosis.
Q. And show me -- I think there's a reference in your records somewhere about his chemical exposure.
A. Well, I don't see that right offhand. Well, I don't see that. I don't see that specific statement.
I said -- talked about no apparent etiology. If I said that, I'm not finding that right off the bat.
Q. Well, in your office practice, do you have a questionnaire that the patient fills out?
A. We do now, up here in this office. I was in a different office in 1983, and we did not have any such questionnaire.
Q. All right. Do you make notes when you interview a patient?
A. Not generally during the interview.
Q. Afterwards do you make notes?
A. I dictate a note of some sort, yes.
Q. And you just dictate it from memory as opposed to some handwritten notes or something that you might make?
A. I usually dictate it from memory, because it's

typically -- well, usually almost exclusively is on the day that I see that patient, a few hours afterwards. And I rely on my memory.
Q. Are there some handwritten notes in your file?
A. There are none, that I know of.
Q. Okay. So, in looking through your file, do you see any mention of any type of inquiry or any information at all concerning his occupation and exposure to chemicals?
A. I think I said he was employed as a pipe fitter. As we have seen, I don't specifically state anything about his chemical exposure.
Q. I know. This is 1983, we have discussed the fact that you knew the relationship. I take it that you did not discuss the relationship between benzene and aplastic anemia with Mr. York?
A. Well, I discussed the -- I asked him if he was exposed to any chemicals, and he said no.
Q. Again, that's based upon your memory today, some 16 -- 15 years later, correct?
A. Yes.
Q. You don't have anything in your notes, again, that specifically says patient denies any exposure to chemicals such as benzene,

toluene, et cetera, do you?
A. I don't specifically say that in my notes, that's correct.
Q. And would you expect a 30 year old -- how old was he, by the way, when he came in, 29 or 30?
A. Yes, 30.
Q. Would you expect a 29 or 30 year old pipe fitter to know the relationship between benzene and aplastic anemia?
A. No.
Q. Do you know whether or not a 29 or 30 year old pipe fitter would know what chemicals he worked around?
A. I couldn't say that with certainty.
Q. Okay. Now, I'm looking at a pathology report from St. Joseph's Hospital that is dated, it looks like, July 20th, 1983. Could you pull that out for me?
A. I may or may not have that because that -- if it's from the hospital, that doesn't always come back to my records. I have one from when?
Q. July 20th, 1983.
A. I don't know if I have a copy of that in my records. All of the material from the

hospital doesn't always get back in the file.
I don't think I have that.
Q. Can I see your records for a minute?
A. Yeah.
Q. Doctor, first of all, these handwritten notes.
A. Uh-huh.
Q. I asked you earlier if you had any handwritten note in your file, and you said you did not. There are obviously -- there is a page of handwritten notes in here. When were these prepared?
A. Within the last several days. Those are not patient's notes. They're a time line just for my convenience.
Q. Let me show you the pathology report we had from St. Luke's Hospital and ask you to read the first sentence under comments.
A. I think you said St. Luke's Hospital. I think this is from St. -- this says St. Joseph Hospital.
Q. I meant St. Joseph, if I said St. Luke's.
A. It says, clinical information indicates that this patient has been exposed to chemicals.
Q. All right. What does clinical -- what does that mean?

A. It's means at the bedside or talking with the patient.
Q. Okay. Can I see that, please?
And what doctor is this?
A. That's Dr. Helen Schwartz, who is one of the pathologists here.
Q. All right. Now, can you show me in your records where you followed up on that after July? Do you have something in your records where you went in after receiving this information from the doctor, Dr. Schwartz, over there and investigated his comments?
A. It's a she, and she has never seen the patient.
Q. I'm sorry. She. Is the answer, then, to my question that you did not do anything to follow up on that comment?
A. Yes, that's correct.
Q. Okay. And I guess in terms of why she was able to tell clinically that this was a chemically induced aplastic anemia, and you were not of that opinion, I guess, is something we should ask her?
A. You would have to ask her. To my knowledge, she has never seen Mr. York or spoken with

him.
Q. Okay. So you disagree with Dr. Schwartz and her opinion, or at least her comment, about the cause of this aplastic anemia, correct?
A. Well, I don't know how she can make the comment without ever having spoken to the patient or seen any of his clinical material.
Q. Did you talk to her?
A. I doubt it.
Q. Okay.
A. Normally when we do a bone marrow -- might explain how come she is signing that report - normally when we do a bone marrow, we prepare slides and we also take a biopsy. And the biopsy is sent to pathology, and they process the biopsy and issue us a duplicate report, primarily on the biopsy. But all they see is a test tube with formaldehyde in it and the piece of bone material. They don't see the patient.
MR. MORGAN: I object to that as being nonresponsive.
Q. Doctor, often times there's a hospital chart that accompanies the patient; isn't that true?
A. Correct.

Q. And that the pathologist or hematologist, whomever is involved in the treatment, has access to that chart, don't they?
A. Yes.
Q. So you don't know if she looked at the chart -- so you don't know if she looked at the chart and then looked at the specimen, based upon everything she saw, felt like that this was someone who had been exposed to chemicals or not?
A. I don't know why she made that comment.
Q. Okay. Back to my question in terms of trying to rule out potential causes.
A. Yes.
Q. We've got notes in your file that say no drugs?
A. Correct.
Q. We've got notes in your file that say no hepatitis?
A. Yes.
Q. But we don't have anything that says no benzene exposure?
A. Correct.
Q. Can you explain why you write the other things down that did not exist but you. didn't write

the benzene down that you say did not exist?
A. Well, there are many other causes of aplastic anemia that are known, certain hereditary abnormalities that can result in aplastic anemia. I don't -- did I even say that he didn't have hepatitis in the past? I think I simply said there were none of the known etiologies, and to me that includes all the ones I knew about. So I felt no particular need to enumerate each one.
Q. Now, you have been testifying on behalf of defendants, I think, by 1983, haven't you?
A. By 1983? I don't know that. I really can't tell you that with certainty. I know I have. testified in some malpractice trials as long ago as that. But I -- probably not in terms of benzene related problems.
Q. You knew that in 19 -- in 1983 you knew that there were claims being made that benzene was causing aplastic anemia, among other conditions, didn't you?
A. Correct.
THE VIDEOGRAPHER: Excuse me. We've got about five minutes left on the tape.
MR. MORGAN: Why don't you go ahead

and change it.
THE VIDEOGRAPHER: Off the record at 3:22 p.m.
(Off the record.)
THE VIDEOGRAPHER: Back on the record at 3:24 p.m.
Q. Dr. Natelson, I had asked you earlier about Dr. Sinsenbruner. His full name, I just found it, is Dr. Lyle Sinsenbruner. He is a professor of medicine and the director of bone marrow transplantation at the University of Maryland School of Medicine. Do you not know him?
A. I don't know him.
Q. Or know of him?
A. I don't know of him.
Q. Have you ever read any articles that he has authored on the subject of benzene or aplastic anemia?
A. I may have but -- because many times when I will read an article I don't always check to see who the author is, unless there is some particular need to. So I may well have read material he has written in the past.
Q. Have you ever attended an Aplastic Anemia

Foundation of America conference?
A. No.
Q. Were you even aware that there was such an organization?
A. I was not aware that there was such an organization.
Q. And you are certainly not -- I guess since you didn't even know, you are not a member and do not receive any type of newsletter from that organization; is that correct?
A. No.
Q. Do you receive literature from other areas, other -- concerning other diseases or conditions that you treat?
A. I'm not sure how you mean. I subscribe to a number of medical journals, if that's what you have in mind.
Q. But nothing that seems to be specifically related to something like aplastic anemia?
A. Well, many times we -- drug people come through the office here trying to sell a particular medicine for a particular illness, and they will bring a variety of information on that particular medication. But, generally, what we use are the articles that

come from our medical literature.
Q. Look at your note from September 30th, 1983.
Do you have that?
A. Yes. 8-30-83?
Q. I wrote 9-30-83. Is that the wrong date?
A. I have one from 8-30-83. I don't have one for
9-30.
Q. Read the history that you had. I thought --
A. Mr. York's skin rash has faded now completely,
and he seems to be over most of the adverse
effects from the- anti-lymphocyte globulin.
That's 8-3 -- 8-30-83.
Q. This was one when you did the second bone
marrow?
By the way, you mentioned that was
on an outpatient basis.
A. I don't remember. It could have been
inpatient. We usually do them on an
outpatient basis. He may have been in the
hospital for some other reason, and we did it
there.
Q. Because as you noticed earlier when you
testified, when you said it was outpatient,
when they asked you to look at the records,
you said it shows he was admitted 9-29 and

discharged 9-30, which would that indicate an inpatient procedure, or inpatient, at least, stay?
A. You know, it doesn't always mean that. In other words, many times what will happen is we will do the bone marrow in the office and then decide to admit the patient to the hospital. It's a lot more convenient to do it in the office. Sometimes we admit the patient -- we don't admit a patient to the hospital specifically to do a bone marrow test, that doesn't happen. But we sometimes may do it during the course of the hospitalization. I'm sorry. You are asking me to look at something. I have a discharge summary from 9 -- well, this is 9-30-83.
Q. Yes, sir. Earlier you mentioned that -- as you were reading through the history, I thought you indicated that the history showed aplastic anemia had been diagnosed approximately four months earlier with no etiology and there was no drugs to point to. And in looking back at the record, I didn't see that in the records -- I'm sorry, I'm looking at your discharge summary as opposed

to the admission -- okay.
Look at your history and physical
from your report, not the discharge summary.
A. Let's see if I can find it. Yes, I think I
have that.
Q. All right. Now, at the very top, just so I
have the record straight, under present
illness it says, this is one of several
St. Joseph Hospital admissions for this 30
year old male who had aplastic anemia. He was
admitted on this occasion because of weakness
and nausea?
A. Yes.
Q. Okay. Now, here is where we are talking about
the history again. It says, the history
indicates that a diagnosis of aplastic anemia
was established about four months ago. The
patient had no apparent etiology for this
illness and was taking no drugs at the time of
the diagnosis.
Now, I assume that in light of what
you told us earlier that you're pointing to
the cause there, or trying to rule out things?
A. Yes. I said the patient had no apparent
etiology for this illness.

Q. All right. And, again, we discussed -- I don't want to go back into it, but we have discussed the potential causes of aplastic anemia and drugs being one of them. And, again, I ask you why, if your comment earlier that no etiology covers everything, why you say no apparent etiology and then also say was taking no drugs?
A. I can't tell you that. It was not necessary to say that. I could have simply stopped and said the patient had no apparent etiology for this illness. Perhaps for some emphasis, I added that last part of the sentence, emphasizing the fact that there was no potential drug relationship.
Q. And, again, the information you have -- you were operating on from a chemical exposure standpoint is, as we sit here today, you recall-asking Mr. York if he was exposed to any chemicals; and he told you no?
A. Correct.
Q. But that's based on memory, not based on your record review?
A. That's based on my memory of the conversations, yes.

Q. And I guess just to sort of help make my point, how many people do you think you see a day?
A. On days I see patients in the office, sometimes I would say anywhere from 10 to 20.
Q. And has your practice been steady throughout the years?
A. More or less. Probably see a few more patients nowadays than I did in 1983.
Q. You see patients how many days a week?
A. In this office two days a week, Tuesdays and Thursdays.
Q. And then you see patients at other places?
A. In the hospital. If I have consultations in the hospital, or patients I have admitted to the hospital, I see those.
Q. And do you see people in the hospital, how many days a week?
A. Well, it may be every day of the week. If they are my patient and I have admitted them there, I make daily rounds. And depending on if I'm asked to see a patient for some specific reason from another physician, I might see a patient. So there is no day, let's say, that's protected where I don't see

patients. Let's put it that way.
Q. And do you have a day off during the week?
A. No. But we have -- I have weekends off on a rotational basis with some other physicians.
Q. There was some talk about hepatitis C after the diagnosis of aplastic anemia.
A. Yes.
Q. And in looking through your records, I think that we see that there was a blood transfusion given to Mr. York.
A. Yes.
Q. Is it thought that the transfusions were. somehow contaminated and as a result of those he contracted hepatitis C?
A. That's the most likely explanation, yes.
Q. And are you of the opinion, based upon reasonable medical probabilities, that that is the cause of his hepatitis C?
A. Yes, that's the most likely situation.
Q. And that treatment was something that was reasonable and necessary as a result of his aplastic anemia?
A. Yes.
Q. Why do you do blood transfusions in treating aplastic anemia?

A. Well, he received both blood and platelets. He had an extremely low platelet count, and I don't recall the exact circumstances, whether he was actively bleeding or whether we thought he was going to actively bleed, but we typically transfuse platelets when we think there is a potential for a life-threatening bleeding.
Q. All right. You also mention something about the type of levels needed from an exposure standpoint to benzene in order to cause aplastic anemia. Do you recall the levels --
A. Yes, yes.
Q. Now, I didn't think that you were someone who felt qualified to talk about statistics and different studies or different levels of exposure. Do you feel like that you are competent to get into a discussion about the different levels of exposure to benzene necessary to cause aplastic anemia?
A. Well, I have read the literature on the subject; but, obviously, I am not out in the field measuring benzene levels on people.
Q. Yes, sir. But my question is, do you feel competent and qualified to testify concerning

the levels necessary to cause aplastic anemia -- levels of exposure to benzene necessary to cause aplastic anemia?
A. I feel confident in the sense that I know what the literature says about the subject.
Q. All right. Tell me what it says about the subject, what kind of levels are required. And I want to know, since you are, you just told me, aware of the literature, tell me what the literature says about levels of exposure necessary to benzene to cause aplastic anemia?
A. Well, Dr. Huang, whose name you have brought up, has published a number of papers looking at studies of petrochemical workers and trying to identify what sort of exposure might result in a statistical increase of hematologic related illnesses. And his general conclusion has been, and the numbers may vary a little bit over the years, that you need to have at least about 100 to 200 part per million year exposures.
Now, you may give a person an abrupt, sudden exposure of high levels of benzene one time and the blood counts may go down. At what that level is, I don't know

with certainty, but I would guess it would have to be extremely high.
And I have seen patients who have had an acute exposure to chemicals and dropped their blood counts, but seldom do we really have accurate measurements as to what that exposure was. We know they got sprayed with a chemical and presumably had very high levels, but we don't have those actual measurements.
So that acutely a person can develop a big drop in their blood counts. We are talking - these kinds of levels primarily relate to looking at populations for leukemias, lymphomas, and aplastic anemia.
Q. All right. Is that the only literature that you are familiar with?
A. Well, there are various government publications on the subject of benzene exposures. And I can't sit here and quote what levels they say. I have them in my files. But they are high exposures.
Q. Any other literature that exists on the subject you are familiar with?
A. Again, I can't quote you. I have a large file on hematologic illnesses in which there are

many papers that deal with benzene exposure.
Q. Well, you were able to quote off the top of your head Dr. Huang's numbers.
A. Yes.
Q. And you told me that the other publications would be governmental publications, but you're not familiar what their numbers are. And then you told me there are some other reports in the literature, but you are not familiar with those numbers either, are you?
A. I can't tell you, as we sit here, whether one says 75 or one says 50. I don't keep that kind of information handy in my head.
Q. How is it you know so quickly Dr. Huang's numbers?
A. I may have recently read an article from him. I know quickly Dr. Young's numbers, too, because I have recently seen his articles. Certain things that may have recently come across my desk, I would remember more frequently than something I've seen a long time ago.
Q. Well, what does Dr. Young, the hematological director of the National Institute of Health, say the numbers are?

A. Oh, in terms -- I was talking about the numbers of the ATG. We were talking about treatments that you brought up.
Q. No, sir. Dr. Huang is often times used by defendants in lawsuits. Are you aware of that?
A. Correct. Yes, I am.
Q. And so you and he are much aligned in cases, aren't you?
A. We may be in certain cases.
Q. Have you worked on a case where Dr. Huang has also appeared on behalf of a defendant in the case?
A. I'm sure I have, yes.
Q. Have you talked to Dr. Huang before -
A. No.
Q. -- personally?
A. No.
Q. Does Dr. Huang say anything about lower levels of exposure than these numbers you have given us?
A. I don't recall specifically his comment about low level exposure. I recall his comments about higher exposures.
Q. Of course, if we only focus on the higher

exposures, that means, if I am right, that
it's harder to get a disease, isn't it?
Wouldn't that be correct
A. Harder to get what?
Q. Well, aplastic anemia from a benzene
standpoint, let's say.
MR. SCOTT: Object to the form of
the question.
A. I don't understand what you are saying.
Q. Okay. Just so I will be straight and the
jury, and perhaps they are not confused, but
the higher the number it takes to -- the
higher number of parts per million of exposure
to benzene, the harder it is for an individual
who's exposed to benzene to get the disease of
aplastic anemia. Is that a true statement?
A. The higher they get exposed to, the less
likely they would have the illness?
Q. Yes, sir. In other words, if I'm exposed to
benzene every day, to 50 parts per million,
then under the numbers you gave us from
Dr. Huang, I'm not going to get aplastic
anemia from that, correct?
A. You will if you are exposed long enough. In
other words, it's cumulative. He's talking

about part per million years.
Q. All right. Explain that to me, then, how that's broken down.
A. Well, it's the amount of exposure. In other words, a part per million year is the amount you are exposed to over the course of -- if you were working over the course of a year.
Q. All right. So if I go to work one day and I'm working a shutdown and working 12 hours that day and I get exposed to 12 hours at 50 parts per million --
A. Okay.
Q. -- for that day.
A. For that day.
Q. Do I put 50 down on the column?
A. And if that's the only day you ever got exposed during the course of .a year, it would be -- you would have 50 divided by 365 days in the year.
Q. Okay. And your interpretation of the math that's involved here, is you add up your exposures on a daily basis over the 365 days, you divide that number by 365, and if it comes up to between 100 and 200, then that's something you would feel at that point you

point to the exposure of the benzene being the cause of the aplastic anemia?
A. If you are exposed to one part per million of benzene for a year, you have a one part per million year exposure. And depending on what you are exposed to, you may be exposed to a massive level in a short period of time and accumulate the same degree of risk.
Q. And as we sit here today you do not know what Mr. York's exposure history was in terms of benzene, do you?
A. I do not know.
Q. If I told you that it was in excess of 100 parts per million years.
A. Yes.
Q. Then would that change your opinion about the cause?
A. No. Because we now have the results of. the long-term therapy. And, again, as I said, the character of the bone marrow is really unlike what we would expect to see in benzene.
Q. If I told you that it was more than 200 parts per million years, would you change your opinion?
A. No.

Q. How many cases of aplastic anemia that --
well, first of all, have you ever testified,
of all the cases you have testified in, that a
worker's aplastic anemia or leukemia was
caused by benzene exposure?
A. No.
Q. And in those cases, have other doctors been of
the opinion that, just like in this case, that
the individual's condition was, in fact,
caused by benzene exposure?
A. Yes.
Q. So it's just a difference of opinion, in your
mind?
A. Yes, I would have a difference of opinion.
Depends on the case and the circumstance.
Q. In terms of counting these cells, you
mentioned that Mr. York's numbers went up.
And you said, I think, that they were
somewhere around the 200,000 range?
A. The recent, most recent platelet count was
215,000.
Q. 215,000. Is there something scientific that
counts those for us? Do we have a computer -
in today's world that we have some many
advances technologically, is there something

you put the blood in and it spits out the number of 215,000?
A. Yes.
Q. What's that called?
A. Well, it's a cell counter of one type. Ours happens to be made by Colter. There are many different kinds of cell counters.
Q. And the cell counter by Colter that you used, I take it you put the blood in and it does its business and then spits out a number?
A. Correct.
Q. And it said 215,000?
A. Yes.
Q. Okay. And then what about the size of the cells and the shape of the cells, is there again some advanced machine or computer that tells us if the cells are normal shape and size?
A. Yes. One can use certain equipment for that, or you can simply look at them under the microscope.
Q. Well, what piece of equipment did you use to determine that they were normal in size and shape?
A. I used my microscope.

Q. Okay. So that is your subjective interpretation of the way they looked?
A. Yes. When you look at blood under an oil lens in a microscope, the -- you can actually do a platelet count fairly accurately. For every platelet that you see in an oil field on a well made slide, that's worth 20,000. So if I looked in an oil field, and I looked through 10 or 15, 20 different oil fields and I only saw one platelet in each field, that person has a platelet count around 20,000. If I saw two platelets on average, that person would have a platelet count around 40,000. And someone who has looked at blood films for many years can look at a slide and come awfully close to what the machine counts in terms of a platelet count. And you also have a general . idea of what the range of normal might be and whether something is distinctly outside the normal range or within the normal range. Now, obviously, the human eye is not going to be as accurate as a computer in terms of sizing cells. In other words, when I look at a slide, I'm not going to be able to tell you to the micron what the average diameter is

and a machine can do that much more accurately.
Q. A11 right. And so in this case you just looked at it yourself and did not use the machine?
A. We -- well, we ran his counts through a machine; but in terms of the platelet sizing, I looked at it.
Q. Okay. You mentioned that Mr. York went back to work. Did you advise him .at that time in light of his condition about possibly being exposed to benzene?
A. I don't recall. I would doubt it, but I don't recall that.
Q. If someone has aplastic anemia, would it be something -- would you recommend that they stay away from chemicals such as benzene?
A. In high doses, certainly.
Q. Would they be more susceptible to having some problems with exposure to benzene than, say, someone who does not have aplastic anemia?
A. I don't know that we know that with certainty, but you might surmise that.
Q. Would you -- if you had known that a pipe fitter works in the petrochemical area and is

exposed to these aromatic hydrocarbons, would you advise them to go back to work in that type of environment?
A. Probably not.
Q. You've had cases referred to you by defense attorneys or that you saw on your own where you have requested work records of the individual, haven't you?
A. Well, I have been sent work records by various attorneys on patients. I don't know that I have ever specifically requested a work record for chemical exposures. I have requested work records to look at blood counts and chemistries on patients over the years. But looking for drug exposures, I can't recall such an instance. I may have, but I don't recall it.
Q: How many aplastic anemia cases have you seen?
A. Oh, personally, probably in the range of 20. Probably in the range of 20, I would say, over the years.
Q. All right. You mentioned earlier that you had about a 70 to 80 percent response rate to the treatment that we mentioned, the ATGAM treatment.

A. Yes.
Q. Which, of course, means that 20 to 30 percent did not respond?
A. Correct.
Q. What happened to them?
A. Well, one I can think of, the most recent one died of severe infection. That's one that comes to mind. I can't think off the top of my head what has happened to some of the others. Some have had very slight responses. Some have had striking responses. So when I say improvement at 70 or 80 percent, that includes the partial responses, as well. as the complete responses.
That's -- the most recent case I can think of died.
Q. And you can't remember the other ones that did not respond to treatment?
A. I just can't recall at this point.
Q. Wouldn't you feel like that the doctors who study this type of disease who perform these different groups, these cohort studies, since they see more people, they may see thousands of people over their life, that those people would be in a better position to tell us about

this disease and causative factors, et cetera, than you and me, only having seen 20?
MR. SCOTT: Object to the form of the question to the extent it misdefines cohort studies.
A. I mean, I can't comment about other investigators that I don't know. In other words, all of this work gets published. And I'm familiar with what's in the medical literature about aplastic anemia and the causation. And most of the recent reviews of aplastic anemia provide charts as to what those authors thought were the predominant etiologic factors. Whether, someone who collects all of that data would be in a better position than someone like myself who reads the data, I don't know.
Q. You mentioned that you would not expect -a relapse, but you would not expect a relapse of aplastic anemia, would you?
A. In Mr. York's case, at this point in time, I would think it would be very unlikely-he would relapse with aplastic anemia.
Q. Right. If he has a problem, I think you mentioned, it's either going to be

myelodysplasia or some type of leukemia?
A. Correct.
Q. Again, from the same original incidence of aplastic anemia?
A. That's simply based on what others have noted, looking at large cohorts of aplastic anemia.
Q. How can we tell today, as we sit here, that Mr. York will or will not come down with some form of leukemia in the future as a result of this aplastic anemia?
A. Well, I think if you were to have chromosome studies done now on his bone marrow and they were perfectly normal, I would think that obviously it would be far less likely that he would have later development. of leukemia than if we did chromosome studies on his marrow and a number of abnormalities were seen.
Q. I guess what you are saying is absent a chromosomal study of the bone marrow, we are unable to tell right now what the future holds for him?
A. With certainty that's correct.
Q. And even, I guess, with the chromosomal. study you still wouldn't be sure that he would not come down with some form of leukemia?

A. I think it would be pretty unlikely at this late date, after treatment. And, of course, if he had a chromosomal defect, certain ones are more hazardous than others, particularly multiple defects. So that I think that kind of a study would have some predictive activity.
Q. Well, let's combine what we know his condition is today with the hepatitis C that he also has.
A. Yes.
Q. What is the difference between chronic persistent and chronic active hepatitis?
A. Well, I think that that distinction used to be very critical a number of years ago. And many GI doctors today don't like to make that distinction. What we thought was that chronic active hepatitis meant there was tremendous inflammation in the liver, there was an active going process going on where the liver was being chewed up as opposed to chronic persistent hepatitis .in which there was still viral activity but the liver wasn't actually undergoing necrosis or cell death on a vigorous basis. So the thinking was that

chronic persistent hepatitis was less vigorous than chronic active hepatitis.
Today, most people are looking at what's called viral loads. They actually measure the number of viral particles in the blood. And you can get some idea as to how vigorous the infection is by the actual amount of virus that's being synthesized. And sometimes the distinction of persistent versus active is not as nice as we used to think it was.
Q. Well, then, let's just put it in terms of having some problems and really needing treatment for it.
A. Yes.
Q. What would the -- what type of treatment would you normally give if you began to have liver problems to try to stop that?
A. Well, in the course of hepatitis C, if a person has a lot of viremia, they have a lot of virus in the bloodstream, and they have a bad looking liver biopsy, if there is active inflammation ongoing, you can treat them with interferon, or some people combine interferon with another antiviral drug called Ribavirin.

And what you find is about 20 to 25 percent of the time the viral levels will drop dramatically in the blood, and the liver may heal to a certain extent.
Now, unfortunately, in a number of those patients, once you stop the therapy the virus comes right back; but in some people the virus seems to stay dormant and, obviously, those people you've benefited a lot by the therapy.
Q. What effect does interferon have on the bone marrow?
A. Well, it depends on the dose you use. In high doses it certainly can suppress the bone marrow. The dose that's generally used for hepatitis is typically a three day a week dose. It's not what I would consider a high dose. And it's not very suppressive on the bone marrow. It may be a little suppressive, but not much.
Q. You wouldn't advise any suppression of bone marrow in an individual who has been diagnosed with aplastic anemia, would you?
A. Well, I think it depends on the circumstance. For example, let's take this case. If I knew,

having done a bone marrow examination on Mr. York, that the bone marrow, not just the peripheral blood that I have looked at, but the bone marrow really looked normal and it had normal chromosomes. Now, the GI doctor told me, look, his blood is really full of virus, do you think -- I really feel like he ought to get benefit from interferon treatment, I would say go treat him: Because the risk of one is less than the risk of the other on the progression of the liver disease.
Q. What you're saying there, I believe, is that if you don't treat the liver, he will die from that?
A. That's correct.
Q. And it's better to go ahead and try the immediate cause of what may be the death, even if it affects the bone marrow, and ultimately he dies from that, because he's going to die from one of the two anyway?
A. Well, I think in a person who has a normal cellularity bone marrow, it's not likely that the usual doses of interferon would suppress that very much. And so I think it would be probably safe to use it, particularly if

you've got aggressive liver disease that needs treatment.
Q. Almost the squeakiest wheel has to get the oil?
A. Correct.
Q. In talking about the future, there was a 1991 article from the New England Journal of Medicine that talks about immunosuppression in aplastic anemia, postponing the inevitable, question mark. And it goes -- I take it you read the New England Journal of Medicine on occasion?
A. Yes.
Q. Do you subscribe to that?
A. No. Well, I take that back. I don't personally, but the office does up here.
Q. And you are welcome to look at this, but in looking at some of the parts that I have read that I can understand, it talks about long-term survival and the effects of the treatment of the immunoglobulin treatment, either the ATC or -
A. Yes.
Q. And it says that in one study of 103 patients with severe aplastic anemia who were treated

with anti-lymphocyte globulin, the risk of these hematologic complications increased over time, reaching 57 percent after eight years, without attaining a plateau. And then it says, quoting further, in a further study of 223 patients who survive more than two years, clonal disease developed in 31, with a median follow-up of four years with no plateau in the risk of clonal disease was noted. Thus, immunosuppressive therapy can lead to improvement in hematologic status and survival in the majority of patients with severe aplastic anemia, but the patients are probably not cured and remain at high risk for clonal malignant disease.
Would you agree with that?
A. Well, they remained at much higher risk than the general population. But as I -- the study I quoted earlier, some 500 patients, a little bigger than this study, the numbers were certainly increased over the general population, but they were not overwhelming numbers.
But, yes, someone who has aplastic anemia is at risk of a clonal disease, and

what they're specifically referring to is myelodysplasia, acute leukemia, and PNH.
Q. What is the treatment for myelodysplasia?
A. Well, that's a very complex area. Because myelodysplasia is sort of a general term. And within that general term there is some distinct syndromes of myelodysplasia.
Q. Can you tell by the type or severity of aplastic anemia what type of myelodysplasia that you may have?
A. You mean what type of myelodysplasia you may eventually get.
Q. Yes. In other words, Mr. York, as we sit here today, you've talked about he's at increased risk for developing this disease. Can you tell what type of myelodysplasia he may develop?
A. No.
Q. All right. And you are about to tell me, I believe, that the treatment for the different types of myelodysplasia is also different?
A. Yes. That varies considerably, because in some cases of myelodysplasia we see almost normal looking bone marrow, but there are things such as ring sideroblasts or distinct

abnormalities that we can identify in the marrow, but no increase in what we call blasts or leukemic cells. And' we put those people in one category.
Then there are people who have refractory anemia with an increase in their blast count in the bone marrow, but not enough blast to make us want to call it acute leukemia.
Then there are people who have a tremendous proliferation in white cells, and we call that chronic myelomonocytic leukemia, or something of that nature. And depending on what -- in a way, the more blast forms you have in the bone marrow, the worst the long-term prognosis is. And those that have a great increase in blast forms, you are more liable to want to jump in with some form of therapy than someone who simply has what we call refractory anemia with ring sideroblasts.
Q. I guess what I am asking is, is the treatment for the myelodysplasia conditions that you discussed extensive, or is it something you can just give prescription medication to and have it go away?

A. Again, it depends, but the most severe myelodysplastic syndromes essentially need to be treated like acute leukemia. They may be treated with high doses of chemotherapy, with bone marrow transplants. Dr. Young has some evidence in the short-term, he has used anti-thymocyte globulin in some of these patients with benefit. I've used it in a few and have not seen any responses. But the more vigorous the myelodysplasia, the more vigorous the treatment.
Q. Again, are any of the treatments -- give me some idea, I'm not familiar with treatment for leukemia, for instance.
A. Okay.
Q. So is it -- again, is it very minor treatment or is it --
A. No, this would be-a major treatment. Basically, in the case of leukemia, we think of this as a good cell/bad cell situation. And there are bad cells in the bone marrow, and they need to be hit with a hammer. So you give high doses of chemotherapy and you hope that you can have enough surviving good cells to re-populate the bone marrow.

In cases of myelodysplasia, often times there really aren't enough good cells lying around, and so, therefore, you have to ablate the bone marrow with chemotherapy and put in somebody else's bone marrow cells, actually do a transplant. And in some instances that will be curative, or cause remission. Sometimes just treatment with the chemicals as we treat acute leukemia can cause a remission. In some few instances, you can give what are called maturation agents; that is, drugs that, for obscure reasons, tend to want to make the leukemic cells turn normal. And you can improve some bone marrows by low doses of continuous chemotherapy by trying to drive the abnormal cells into a growth mode where they turn more normal.
In some of the lesser forms of myelodysplastic syndrome, you can use male hormones to stimulate the marrow, erythropoietin injections to stimulate the bone marrow, certain vitamins to stimulate the bone marrow, and the prognosis may be much more benign.
Q. From a cost standpoint, what's the range of

cost for the treatment in its least aggressive form versus the most aggressive? Are we talking about tens of thousands versus hundreds of thousands?
A. I would say that's fair, because even least aggressive treatments, simple injections of erythropoietin are extremely expensive. And so these days in medicine, there are very few inexpensive treatments.
Q. Now, in terms of the other treatment modalities, because you mentioned bone marrow transplant, Mr. York does not have that available to him, does he?
A. Well, he doesn't have a related bone marrow transplant. These days we have a national registry, and one can put your numbers into the registry and expect to find a match of somebody who sort of looks like you, but they are really not your relative, and you can get a transplant from a so-called unrelated donor. But the risks of that are vast because our current typing is not perfect, and they may look identical but they aren't identical. And so transplantation has improved since 1983 in a major way, and we do do more unrelated

donor transplants, but there are a lot of hazards with that.
Q. Are you saying that one of the hazards that you say is vast would be that it just would reject?
A. Well, what tends to happen is that the bone marrow will usually take, but then you reject. You get what's called graft-versus-host disease, and the bone marrow tries to reject the body that it's in. Also, overwhelming infections occur. And using mismatched or, let's say, matched but unrelated donor marrow has a high track record of graft-versus-host disease. Let's put it that way.
Q. And if you have that condition result, does it usually result in death?
A. Not always, but it's often a chronic disabling illness. In the very acute forms it may result in death.
Q. So if Mr. York were to come down with some form of myelodysplasia or leukemia and it required a bone marrow transplant, not only does he have to worry about the surgery, of course, the condition, but also in his situation, whether or not he really can find a

match?
A. That's correct.
Q. Would the type of treatments be the same for the acute leukemias and the PNHs that you mentioned earlier?
A. Well, acute leukemia would require very vigorous treatment. PNH is often a very chronic illness that may not do you in, but just may cause some chronic disability. People can have PNH for many years. They usually can't have acute leukemia for many years.
Q. And, again, the cost of treating PNH or a --
A. Well --
Q. -- range --
A. It's different in different patients, but I would say in most patients it would not be vast. It's a variable illness in terms of some patients have problems with clotting problems, thromboses. Others have problems with poor blood production and may require blood transfusions. So it's a little different in different people. But as a general rule, I would say that the treatment of PNH is not enormously expensive, as opposed

to acute leukemia or myelodysplasia.
Q. So it might be fifty to a hundred thousand, the course of treatment, versus the hundreds of thousands we talked about, potentially, on the others?
A. Well, it would certainly be hundreds of thousands if you got into the bone marrow transplant or high doses of chemotherapy arena. How much it would be to treat someone for PNH, it's hard to put a number on that. I guess it depends how many transfusions they might need, how well they respond to therapy, which is often with corticosteroids and androgens. There are a lot of. variables, but it might not be very expensive to treat PNH.
Q. Again, relatively speaking?
A. Relatively speaking.
Q. You mentioned earlier that Mr. York told you that he had, at least your recollection what he told you was he had a shoulder that bothered him with bursitis?
A. Correct.
Q. And when did you meet with Mr. York?
A. Oh, this would just be a few weeks ago, or whatever the 15th, whenever it was. That

would be on January 15th.
Q. Okay. And today is the 26th?
A. Uh-huh.
Q. So 11 days ago. And your recollection was --
do you recall which shoulder it was that
bothered him?
A. He said it was his right shoulder, and that he
had had repeated injections of steroids to the
point where the orthopedist felt like that he
had some atrophy of the tissue from the
steroids and that it wouldn't be wise to give
him an additional set of injections.
Q. Okay. You don't recall Mr. York telling you
that that has occurred in both shoulders?
A. I believe he just told me the right shoulder.
Q. I think the records reflect that he has had
injections of cortisone in both shoulders,
that the problems exist in the right and the
left shoulder.
A. That may be so, but I simply recall him
telling me about his right shoulder.
Q. Do you have any notes from that visit?
A. I'm sure I dictated a note, but it hasn't made
it back to the chart. And that's probably
because this is an old chart from my other

office, and they are probably trying to figure out what chart to put that note on.
Q. Where is it typed?
A. We send it out to a typing service.
Q. And if it were back -- where would it be if it were back in your office but not filed?
A. It would be -- we could probably retrieve it. It's probably here somewhere.
Q. Okay. You mentioned something about not seeing as many cases because of the control of benzene. Could you tell me when the control of benzene started?
A. I can't tell you the exact years. I know the levels have gradually diminished over time; that is to say, the government regulatory levels that were permissible. I can't cite the years that the changes occurred.
Q. Would you recommend any exposure to benzene, regardless of the level, you, yourself?
A. Well, we get exposed every time we pump gas. I mean, it's hard to avoid benzene. I mean, it's a ubiquitous compound. But, obviously, if you can avoid a high exposure you are better off doing that.
Q. Do you know what, for instance, Mobil did?

Well, let me ask you this.
You knew it -- when you were in medical school you knew the causation of diseases from exposure to benzene, we have been through that?
A. Yes.
Q. And that was general accepted knowledge in the medical community at that time?
A. Yes.
Q. Have you ever advised people who worked on some type of either board or some capacity where you were called upon to, perhaps, advise some entity medically?
A. About benzene?
Q. About anything.
A. Well, I sit on numerous committees at the hospital. Some of that has to do with a variety of things, from chemotherapy order forms to a number of things. So, yeah, I'm sure I have given advice on a lot of things.
Q. Well, let me ask you this way, then. If you were employed by Mobil, for instance, and you knew that because of the process involved with taking crude oil from that state and making different products, that part of the process

would involve benzene, and you were employed by them, say, in the '60s and '70s, would you make any recommendations to them concerning precautions that should be taken in order to allow people to work around the products that may have benzene in them?
A. Well, I wouldn't be in a position to know about the particular equipment that they have or what potential vapors would be coming out of that equipment. So with specific reference to a particular factory, I could only advise in general terms, that high doses, that exposure to benzene is not good, and if they could do something to avoid that, that would be desirable.
Q. Would you advise allowing workers to wash tools with benzene?
A. No, certainly would not.
Q. Would you advise allowing workers to wash their hands with benzene?
A. No.
Q. If someone were going to work around either benzene or a product containing benzene, what type of protective devices should be used; or should they just simply not be exposed to it

at all?
MR. SCOTT: Objection.
A. No.
MR. SCOTT: I'm sorry. Object to the form of the question. You haven't defined the product containing benzene.
A. I think that's the situation in terms of the fact that if you are talking about contact that might be on the hands, you are talking about protective gloves or covering that you might have. If it's exposure to benzene in the vapors, then it's however you are going to prevent that by mask devices.
Q. Would a simple paper respirator, paper mask, stop the inhalation of benzene fumes?
A. No.
Q. Would we have to-go to a fresh air mask before we would be able to prevent the inhalation of those benzene fumes?
A. I'm not an expert on masks, so I can't tell you what you'd have to have.
Q. From a medical standpoint, if they had -- if Mobil had come to you and asked -- and I just use Mobil because it's one of the plants. If Mobil had come to you and said, Dr. Natelson,

we have got some products out here that contain benzene, we've got drums marked benzene on them, should we warn people about benzene and the effects of it, what would you tell them?
A. You've got sealed drums of benzene?
Q. Well, yes, sir, they are sealed; but they are not unable to be opened.
A. Well, there should be signs on them indicating that they are a toxic chemical and that access should be restricted to them.
Q. Okay. And in what type of -- should you -- in your opinion, should a person know what effects may be caused by exposure to that substance?
A. Well, I think if somebody. is working regularly with a particular substance, it would be helpful to let them know what they might expect if they got exposed to it. In other words, is it an irritant, will it make your eyes run, will it make you choke, or does it damage the skin; and then you could, of your own, better evaluate what problems you might be having.
Q. For instance, you may see a chemical that says

it may be caustic and cause irritation or rash to the skin, that's one thing. You may decide 1 will go ahead and work around it, it can't be that bad, as opposed to seeing something that says may cause leukemia or even death?
A. Yeah, that would get your attention more.
Q. And benzene, would it -- would benzene deserve a warning that involved cancer or death being associated with the word "benzene"?
A. I think if you are talking about a vat of benzene that's sealed, there should be signs on it indicating that it's a major health hazard.
Q. Doctor, you've seen other people with diseases that are known to be caused by benzene from the petrochemical. plants in the Beaumont, Port Arthur area, haven't you?
A. Yes.
Q. And you've never asked for records from the Mobil facility, or any other petrochemical facility in that area, have you?
A. No. Sometimes they have come with records, but I haven't specifically asked for records.
Q. You've never been provided with depositions from any of the employees of the different

entities that I mentioned earlier, have you?
A. On certain cases I have seen depositions that I have reviewed.
Q. Well, do you recall any depositions from the Mobil facility in Beaumont concerning the warnings that they give to the employees, both of Mobil and the contract employees, concerning working with benzene?
A. No.
Q. Have, you ever been provided with any information about the level of benzene exposure that the pipe fitters may receive at any of the facilities that I read to you earlier?
A. No.
Q. Have you been provided with any of the levels of benzene exposure, regardless of the occupation that may be working in the area, but from any of the plants that I mentioned earlier?
A. No.
Q. In terms of Mr. York himself, this was a pretty stressful situation, wasn't it?
A. I'm sure it was, yes.
Q. And when you treated him, he had severe

aplastic anemia, the bone marrow transplant was not something that was available to him, and I believe the ATGAM treatment at that time was experimental, wasn't it?
A. Yes, more or less I would say, yes.
Q. And so he was just telling me how he had to sign all these forms because it was such an experimental drug. And, of course, that would add to the anxiety, wouldn't it, that you are taking some experimental drug that we weren't really sure what all would happen?
A. Yes. Actually, I think I probably had more anxiety than he did, because I knew better what the end results might be if he didn't respond and so on. In terms of his situation, I mean, he was a good patient, he accepted what we offered, and of course then everything went well.
Q. I hope you were being facetious that you had more anxiety than he did.
A. I had a lot of anxiety.
Q. More than someone who's facing a potential fatal disease?
A. Well, because of the fact many times -- that's true all right. But the knowledge of how

fatal it is, many times a patient, they only know about themselves, they haven't seen many other people die of aplastic anemia. And they don't really appreciate how bad an illness it can be. And so I'm sure he had a lot of anxiety; but I'm telling you, I did, too.
Q. Well -- and as we sit here today, Mr. York may not appreciate someone going from aplastic anemia after several years of remission into some form of leukemia; but you have seen that, haven't you?
A. Correct.
Q. And I guess at this point your concern for Mr. York, being his physician, that that might happen, might be even greater from an anxiety standpoint than Mr. York may have?
A. Well, as I say, at this point in time, I think that going into leukemia, he's certainly got a higher risk than the general population; but I wouldn't think it's vast at this point.
Q. And, of course, you have seen the effect of this type of disease on spouses and family members?
A. Yes.
Q. You knew Mr. York had some small children at

the time?
A. Yes.
Q. And you mentioned -- and I just used the word remission -- and you mentioned that Mr. York had a response to the ATG and persisted in remission. Now, that, of course, is different than cured?
A. Yes.
Q. Remission means there is still something there but it is not -- he's not 100 percent over the incident?
A. Well, remission is a peculiar word. In other words, it means that the -- you may not be able to see or identify the something else that may still be .there, but there is a statistical risk that something may happen in the future. So remission is a -peculiar word. Not really a very satisfactory word, but we use, that particularly in cases of leukemia and cancers and also in aplastic anemia.
Q. You know Dr. Gardner, do you not?
A. Yes, I do.
Q. He's a hematologist?
A. Yes.
Q. And as a hematologist, I believe you have

respect for him; is that right?
A. Yes.
Q. Is there a safe level of exposure to benzene that has no risk associated with it?
A. Well, I think low levels of benzene we don't statistically associate a risk with. In other words, you are talking about a 1, 2, 3, 4 percent level of exposure, I don't think there's significant risk for that. We can't demonstrate that statistically, let's put it that way.
Q. Well, as we sit here today, do you know specifically of a safe level of exposure to benzene with no risk associated to it?
A. No. But it's my belief that low levels are not with risk.
Are you trying to tell me -- I can't tell you whether 6.5 is worse than 5.
Q. Well, you are telling me on the one hand that there is no safe level of exposure that has no risk with it, but then you say but there are some levels that are safe. That seems to be inconsistent.
A. No. In other words, we know the high levels that have been statistically shown to cause

leukemia. Now, there may be people who are slightly below that high level who may still be in that ballpark. In other words, we know in very general terms. And the reason for that is that usually the exposure data is not perfect in the case of benzene. And so, you know, we say, okay, I think I have said a hundred part per million years, we know statistically you've got to have at least that to catch leukemia. Well, maybe in an individual guy it's only 95, maybe only 90. But certainly not two or three. So I can't tell you what that number is, but low level exposure we don't think causes leukemia. I don't.
Q. Well, let me show you testimony you gave three months ago --
A. All right.
Q. -- on October 22nd, 1997 on page 145.
A. I think I'm saying the same thing today.
Q. Well, let me, just so the record will be clear then, perhaps I was misunderstanding.
The question asked of you in this deposition, which you gave under oath, was, as we sit here today do you know specifically of

a safe exposure level to benzene with no risk? And you ask, with no risk associated with it? And the questioner said, yes, sir. And your answer was, I would have to say no. Is that still your sworn testimony today?
A. Yeah. I don't think I said anything different.
MR. MORGAN: Thank you. That's all I have right now. I think some of these other attorneys will ask you some other questions and perhaps I will have some after they ask; but right now that's all I have.
MR. DILLARD: You all want to stretch for a minute.
THE VIDEOGRAPHER: Off the record at 4:27 p.m.
(Brief recess.)
THE VIDEOGRAPHER: Back on the record at 4:36 p.m.

EXAMINATION
QUESTIONS BY MR. DILLARD:
Q. Dr. Natelson, I'm going to do my best to wrap this up.

I wanted to ask you, looking at your resume, you're, of course, board certified in two different fields, both internal medicine and in hematology; is that correct?
A. Yes.
Q. And as a result of your board certification in internal medicine are you able to address issues that involve the liver?
A. Yes.
Q. You are comfortable with that and feel that you are an expert concerning liver matters?
A. Yes, within reason. In other words, I would defer to a gastroenterologist who specializes, highly specializes in liver disease; but in the general sense of things, yes, I'm experienced in the area of liver disease.
Q. All right. Now, I want to focus on probabilities.
A. Yes.
Q. Reasonable medical probabilities, as opposed to on the one extreme, certainty, and the other extreme, possibilities. Are you with me so far?
A. Yes.
Q. Okay. Focusing on reasonable medical

probability, is there any evidence that this gentleman has leukemia or myelodysplasia or PNH or that he's likely and reasonably medically probably, probability to get those diseases in the future? Is there any evidence of that?
A. Well, you have asked two questions. One, does he have them at this. time; and the answer is no, there is no evidence that he has those diseases at this time.
What is the probability that he will get them? I would say if -
Q. I'm asking is it probable?
A. Is it probable? It certainly is not probable that he will get them.
Q. All right. Now, he came to you in 1983 at the referral of a physician in the Beaumont area; is that correct?
A. Yes.
Q. And he was a pretty sick individual at that time; is that correct?
A. Yes.
Q. And you were entrusted with his care and treatment, correct?
A. Yes.

Q. And, in fact, I think you said he had been to another hematologist before he came to see you?
A. Yes, that's correct.
Q. All right. So you had a patient who had been to another hematologist, and for some reason had left that physician's care, and he come to see you, correct?
A. Yes.
Q. All right. And he entrusted his care and treatment to you, correct?
A. Yes.
Q. We, either me or any of these lawyers in this room, didn't send this gentleman to see you, did we?
A. No.
Q. All right. None of the clients, the companies that we represent, sent this gentleman to see you, did they?
A. No.
Q. All right. In fact, none of us in this room had ever met back at that time, had we?
A. Not to my knowledge.
Q. All right. And you did treat him?
A. Yes.

Q. And, frankly, you treated him with success, did you not? I'm not asking you to brag and I don't mean to be immodest about it; but you did treat him successfully, did you not?
A. No. He was fortunate, and he responded well to the treatment.
Q. All right. And then 14 years went by and he came back once again 11 days ago and sought out your care and treatment and opinions, correct?
A. Yes.
Q.. All right. And, again, we did not send him to you at that time, did we?
A. No.
Q. We didn't recommend that he come to see you, did we?
A. No.
Q. And we haven't paid you for him to come over and see you, correct?
A. No.
Q. All right. In fact, we have never paid you for anything in connection with Mr. York, have we?
A. That's correct.
Q. All right. And we are certainly willing to

pay you for the time that you have spent here today, but we certainly have not had any -- made any payments of any sort to you prior to today, have we?
A. That's correct.
Q. All right. Now, looking at the patient history that he gave you, I think you said that there are a number of causes that are known for aplastic anemia, but that far and away most cases of aplastic anemia are of unknown cause; is that correct?
A. That's correct.
MR. MORGAN: Objection, leading, asked and answered.
A. Most of the causes are said to be idiopathic. Most of the cases are said to be --
MR. MORGAN: Object to the responsiveness of the answer, in light of my objections to the question.
Q. Approximately what percentage in the published literature are felt to be of unknown cause?
A. Well, I, a few years ago, perhaps. two years ago, looked at a series of papers that totaled something over 700 patients from many different areas of many different studies.

And in that group, I believe that the numbers that were assigned to the idiopathic group was either 75 or 77 percent, something in that nature.
Q. All right. And of the other 22 to 25 percent where the cause is known, there are a number of possible causes, I take it?
MR. MORGAN: Objection, leading.
Q. Is that correct?
A. There are a number of other causes that are known of aplastic anemia.
Q. All right. My question to you is, did you list each and every possible cause of aplastic anemia, and expressly in your own writing, rule each and every one of those out in your records, in your patient history records?
A. Not in the patient records, no.
Q. Ail right. As a routine examination and a routine practice in the history and fiscal that you took, did you ask Mr. York questions that would have brought out information as to possible causes?
A. Yes.
Q. All right. And that was your custom and practice back at that time?

A. Yes.
Q. And is so today?
A. Yes.
Q. So that when you wrote in your records in any number of places, no etiology factors were identified, what did that mean?
A. That meant to me that this case was going to fall into the idiopathic group, because there was nothing that stood out, to my mind, from the history and the physical and the data that I had that suggested a specific cause.
Q'. Now, you were asked a question about this pathology report of a Dr. Schwartz. And I think Mr. Morgan -- that's the attorney representing Mr. York here today -- I think Mr. Morgan's question was something to the effect of her opinion as to the cause of his aplastic anemia. Is there any reference whatsoever in that passage to an opinion of that doctor as far as the cause of aplastic anemia?
A. No.
Q. All right. Who was the physician who saw Mr. York, the hematologist who saw Mr. York before you had seen him?

A. That would be Dr. Larry Rice at Baylor.
Q. All right. I want to show you -- because they have been subpoenaed in this case, I want to show you the records of Dr. Larry Rice, specifically his notation concerning the history that he obtained from Mr. York, and ask you if you would read that into the records -- into the record from the report of Dr. Rice?
A. He says pipe fitter, dash, frequently works in chemical plant. Never sick from exposure. No specific exposures. Some lead, no benzene.
Q. No benzene?
A. No benzene.
Q. All right. And that was before he came to see you, correct?
A. Yes.
Q. Now, as far as the bursitis that Mr. York has reported,. whether it's left shoulder or right shoulder or both shoulders, in reasonable medical probability would bursitis that developed some years after the ATG therapy, in your opinion, be related to that therapy or not?
A. No, in my opinion it would not be related.

Q. Is that because any reaction of that sort would be an acute reaction, as you said, following shortly on the heels of the ATG therapy?
MR. MORGAN: Objection, leading.
Q. Let me ask it again.
Why is that?
A. Well, the reason is is that we expect an acute arthritic problem, or acute bursitis when we give ATG. It doesn't happen in everybody, but the bulk of the patients get that. It's temporary, may last for a few weeks, and then it's gone. And to cause a bursitis in one or even two joints many years later, I'm not aware that the literature suggests, and certainly not in my experience has it been, that that might be caused by the ATG.
Q. All right, sir. You were asked a number of questions, again by Mr. Morgan; the attorney representing Mr. York, about whether some of these companies had supplied records to you. I would ask you this. Mr. York was in your office 11 days ago. Did he bring any such records to you?
A. No.

Q. Incidentally, was he by himself; or did he have someone with him?
A. No. He had an attorney with him.
Q. All right. Did they bring any records?
A. No.
Q. All right. And who was the attorney?
A. Attorneys here at the table.
Q. Mr. Morgan?
A. No, not Mr. Morgan. This gentleman.
Q. Mr. Thorpe?
A. Mr. Thorpe, yes.
Q. Okay. As Mr. York's treating hematologist, and having seen him again recently and going back to the time you saw him some 13 years ago for the first time, has Mr. York ever asked of you if you felt that his aplastic anemia was caused by benzene exposure?
A. Well, not until this last visit on the 15th. We discussed to a certain extent etiology of aplastic anemia.
Q. What did he ask you?
A. Well, I don't remember the specific -- well, I may have been specifically asked whether or not I thought it was caused by benzene. But I do remember discussing the reasons why I

didn't think it was caused by benzene.
Q. And what did you tell him?
A. Well, I .said there are two things. One, the initial morphology of the bone marrow. Many of the cases of benzene were reported in what I would call the older literature, let's say literature before 1960, and it really wasn't into the 1960s when bone marrow biopsy was popularized by Dr. Domaschk, who was a well-known hematologist. And in the older literature many times we saw a patient have a pancytopenia; that is to say, the blood counts were very low in the peripheral blood. And it was assumed, based on the marrow study, that this was so-called aplastic anemia. But in the case of benzene it was always thought to be very peculiar. People used the term proliferative aplasia. And-that almost goes against each other. Proliferating means growing very fast. Aplasia means absent. And the reason for that was because they noticed there were a bunch of cells in the bone marrow. It really wasn't aplastic. And I think not until we got into the era of doing bone marrow biopsies did- we find out that many

of those patients really had myelodysplastic syndromes that caused the pancytopenia, all right, but were, in fact, aplastic -- were not really aplastic anemia.
And that actually occurred in this particular case. If you will look, initially the bone marrow done was a sternal bone marrow. And many years ago all the bone marrows were done from the sternum. But the problem with doing sternal bone marrows is you can't do a good bone marrow biopsy. The bone is too thin. And so on the basis of that bone marrow from the sternum, they thought megaloblastic anemia. In other words, they didn't think aplastic anemia.
It really wasn't until we saw a -bone marrow biopsy and we could actually see the total cellularity in the bone marrow that it became clear that the problem was really. an aplastic one, not a dysplastic one as Dr. Rice originally thought, that this might be a dysplastic bone marrow, but --~so I think that the -- in benzene induced aplasia, most of what we are talking about is really myelodysplastic syndromes. And those are stem

cell defects, like acute leukemia, and often eventuate into acute leukemia. Those illnesses, I don't believe, are caused -- that you can cause that kind of an illness to go into remission with anti-thymocyte globulin, although apparently you can improve the blood count somewhat.
But I don't believe there is any literature that suggests that you can take a person with myelodysplasia, such as caused by benzene, and make that person have a normal blood count with anti-thymocyte globulin.
So when I look at the whole case here, the initial marrow morphology showing really aphasia rather than dysplasia, and then I look at the response to the anti-thymocyte globulin, I'd have to say that this bone marrow was likely suppressed on an immune basis. And we interrupted that with the anti-thymocyte globulin; such that he was allowed to go back in remission. And that is what we think of as idiopathic aplastic anemia, and I think, unlike what we think of in terms of the type of dysplasia that we would see from benzene or our chemotherapy

drugs.
MR. MORGAN: I object to the responsiveness of the answer.
Q. Along that line, we talked to Dr. Gardner, whose name was brought up a moment ago. Did he ask for and did you send him the slides?
A. Yes, I did. He did write and ask if I would send him -- and this -- I can't tell you exactly when it was. Probably three or four, maybe even five years ago. He wrote and asked for the slides on Mr. York, which I did send them -to him, and he wrote me back a short note saying he agreed with the diagnosis of aplastic anemia and sent me the slides back.
MR. DILLARD: Thank You. That's all I have got right now.

EXAMINATION
QUESTIONS BY MR. SCOTT:
Q. Dr. Natelson, I don't want-to beat a dead horse, but in 1983, it was your opinion that Mr. York had an idiopathic aplastic anemia; is that true?
A. Yes.
MR. MORGAN: Objection, asked and

answered.
Q. And, of course, at that point you had no idea there was any lawsuit or any of these defendants were involved, true?
A. That's correct.
Q. You were a treating physician at that time?
A. That's correct.
Q. Your opinion 11 days ago in 1998 was exactly the same as it was in 1983, true?
A. Yes.
MR. SCOTT: Thank you, sir. That's all I have.

EXAMINATION
QUESTIONS BY MR. CARRINGTON:
Q. Mr. Natelson, when you have an office visit with a patient for any particular type of disease, whether it's leukemia or aplastic anemia, whatever it may be, do you inquire as to medications they are presently taking or have recently taken?
A. Yes.
Q. And that's not always for diagnostic purposes, is it?
A. No, because sometimes we are looking at

possible drug interactions. In other words, I might prescribe a medicine that might be contraindicated or modified by some medication they are taking.
Q. So, generally, you will ask questions about prescription medications they're taking for purposes of future treatments that you may be anticipating rendering?
A. Yes.
Q. And you generally make those notations in your records?
A. Yes, many times. Today we have a patient form that the patients fill out, and they will list their current medications in there.
Q. Well, in 1983 you didn't have that form so-you would make the notations in the record?
A. Generally. If I felt they were important, yes.
Q. And I would assume-that, for instance, in this case, you noted in the record, initial visit, that Mr. York was not taking any prescription medication. Do you make those notes so that months down the road if you are thinking about prescribing medication you can refer to any medication they may be taking?

MR. MORGAN: Objection, leading.
A. Well, when you say he wasn't taking prescription medicines, actually he was. He had be given injections of vitamin B-12 by Dr. Esslinger. He had been taking some medicines for the blood condition. But in general, the -- if I think it's an important medication, I would make a notation. Someone came in there on digitalis for a heart problem, I would make a notation of that.
MR. CARRINGTON: That's all I have.

EXAMINATION
QUESTIONS BY MR. MORGAN:
Q. Dr. Natelson, then I take it from that last exchange with Mr. Carrington that you are saying that your notation of the drugs, the fact he was not taking any drugs,- was really for drug interaction purposes and not for diagnostic purposes; is that right?
A. No, that would be false.
Q. So it was just the opposite, it was for diagnosing information as opposed to just treatment information?
A. Well, I asked -- I mean, you might use it for

two things. One doesn't prohibit the other.
Q. Yes, sir.
A. In his particular case, I 'was looking at drugs that he might have been taking that could have played a role in aplastic anemia.
Q. Right. You mention that there was studies that showed 75 percent of unknown -- of aplastic anemias were of unknown causes. What paper was that?
A. I can get you -- I'm not -- that's not a single paper. That's an amalgamation of about 10 or 12 different studies on aplastic anemia where the causation was listed, and I collected all that data and collated it.
Q. Did you do that for litigation purposes?
A. Yes. That was in a case called Frias case.
Q. All right. And you have that somewhere -
A. Yes.
Q. -- here in the office?
A. Not up here, but in my other office, yes.
Q. Could you get a copy. of that to the court reporter and have him attach that as Natelson 2 if you will, the entire packet?
A. Yes.
Q. I'm sorry, Natelson 3.

A. Okay.
Q. And can -- well, let me ask it this way.
Could the bursitis that Mr. York has in his
shoulders be related to viral immune complexes
from the hepatitis C?
A. From hepatitis C? Possibly, yes.
Q. Well, do reasonable medical probabilities say
that one of the side effects of the-viral
immune complexes that are associated with.
hepatitis C are known to be bursitis?
A. Well, it may be so; but in general, when you
have someone who has, let's say, something
floating around in the blood, like a complex
that's causing an arthritis or a bursitis, you
would say, well, why would it only attack one
joint, or perhaps two, as opposed to all the
other joints in the body. So, generally,
people that have a systemic cause for their
arthritis have it in multiple joints.
Q. In all joints?
A. No. In multiple joints.
Q. Well, more than one is multiple, is it not?
A. More than one is multiple, yes.
Q. Okay. You also mention that you had given a
presentation or perhaps prepared a paper at

St. Joseph's concerning aplastic amenia?
A. Yes.
Q. Is that listed on your curriculum?
A. I'm sure it is, yes.
Q. Could you -- which one is that?
A. Well, there is one, Number 52, current therapy of aplastic anemia. That's in the Houston Medical Journal in 1985.
And then there is another patient - another article, number 38, treatment of iron overload in patients with chronic anemias. And that had to do -- I believe one of the patients in that article had aplastic anemia and had iron overload from the frequent transfusions. So both those papers would have dealt, to a certain extent, on aplastic anemia.
And then number 55 was an article about bone marrow depression from allopurinol. And that paper discusses aplastic anemia. That looks to be it.
Q. You mentioned you published an article in the St. Joseph's Hospital Journal?
A. That's correct.
Q. That's not listed?

A. Well, that's the same thing. In other words, this journal has gone by several names. It used to be the St Joseph Hospital Journal, then the name was changed to Houston Medical Journal. And so that's really one and the same.
Q. Okay.
A. So some of these articles I have in here will say St Joseph Hospital Medical Journal and other ones will say Houston Medicine, but that's -- Houston Medical Journal, rather, but that's basically the same journal. They changed its name.
Q. All right. Could you get a copy of numbers 38, 52 and 55 for me?
A. Sure.
Q. And attach that as Natelson 4?
A. Yes.
Q. Also, are those peer reviewed articles?
A. In the Houston Medical -- in the St. Joe Medical Journal, no. Some of them are. In other words, this journal underwent an evolution. And when it was the Southern Medical Journal, or the -- rather, the St. Joseph Hospital Medical Journal it was not

peer reviewed. When it became the Houston Medical Journal, it did become peer reviewed. And so if it says Houston Medical Journal, it was peer reviewed. If it says St. Joseph Medical Journal, it was not peer reviewed. They are the same journal.
Q. Mr. Dillard was talking to you about the fact that you hadn't been paid. You remember those questions?
A. Yes.
Q. You certainly expect to get paid; don't you?
A. I would like to, yes.
Q. And you expect Mr. Dillard, or at least the people on that side of the table, to pay you?
A. Yes.
Q. And they have paid you on other cases?
A. Yes.
Q. When Mr. York came in 11 days ago, did you tell him that you had been contacted by the defense in this case or that you. had testified on many occasions and seen people at the request of Mr. Dillard in the past?
A. No.
Q. This case is set for trial in May of this year, and we've sort of asked the court to

allow us to go toward the latter part of the month. Is there any particular thing going on in May that would prevent you from being in trial, if you were given enough notice?
A. Not that I'm aware of, no.
Q. Okay. How much notice would you need of the trial date in order to be physically present in trial?
A. Probably a few weeks.
Q. A couple of weeks, three weeks?
A. Yeah, I think so. Because there are certain meetings that my presence is required at that I have on my calendar that my secretary in my other office keeps. So if you were to say today a particular date, I wouldn't be able to tell you if that's a good day or not a good day for me, or if I will be in the city.
Q. Well, but at least if I give you two months notice, which May is four months?
A. Yeah, but there are a lot of days in May; and I would have to look and see if I have some meeting out of the city, depending on what the day might be and if I could modify that.
Q. Okay. Do you have a calendar where that would be kept?

A. My secretary in my other office, Carol Reese, keeps that.
Q. Okay. If you would, I would appreciate a copy attached to the deposition as Natelson 5 of the month of May -
A. Okay.
Q. -- as it currently exists,.
And then would you require a subpoena to be present or would just someone letting you know the day and time to be there?
A. No, I don't think it would be necessary to subpoena me. I would be happy to come.
Q. Okay. So if either side were to call you and ask you to come over at a certain date and time, you would accommodate them?
A. I would certainly do that, yes.
MR. MORGAN: Thank you. That's all I have.
MR. DILLARD: Doctor, you understand that we scheduled your deposition here today, and that we expect to pay you for the time that you have spent here today, but that you are Mr. York's treating doctor and we have not retained you as an expert witness in this case. You understand that, sir?

THE WITNESS: That's fine. I will certainly understand that.
MR. DILLARD: Okay. Thank you, sir.
MR. SCOTT: Can we attach as Exhibit 6 to Dr. Natelson's deposition his typewritten notes? Do you have them in your file, Doctor, from this recent meeting?
THE WITNESS: Well, this copy is the only existing one. Then I gave a copy to Mr. Morgan.
MR. SCOTT: Glen, will you agree that we can attach a copy of that as Exhibit 6 to this deposition?
MR. DILLARD: Well, let's include in that the records from the 15th of January. There were two pages.
MR. SCOTT: Oh, yeah. We don't have the whole file. Those are more recent.
MR. DILLARD: Could we make those today and -
THE WITNESS: Oh, you are talking about the -
MR. SCOTT: Those two pages of blood counts and so forth.
THE WITNESS: Before you leave I can

give you these.
(Natelson Exhibit 6
marked for identification.)
MR. SCOTT: And we'll attach those collectively as Exhibit 6. And is it correct that those are the only written records you have got which evidence in any way your meeting 11 days ago with Mr. York and his lawyer, Mr. Thorpe?
THE WITNESS: Correct.
MR. SCOTT: Thank you.
THE VIDEOGRAPHER: Off the record at 5:04 p.m.
(Deposition concluded)

THE STATE OF TEXAS

I have read the foregoing testimony given by me on January 26, 1998 in the case of CHARLES DOUGLAS YORK VS. TEXACO, INC., ET AL and have made all corrections deemed necessary.

ETHAN A. NATELSON, M.D. 10

SUBSCRIBED AND SWORN to before me, the undersigned authority, on the day of 1998.

NOTARY PUBLIC IN AND FOR THE STATE OF TEXAS