In The Matter Of:

April Awkward, et al. v.
Jerome I. Snyder, M.D., et al.

Ronald Alan Sacher, B.Sc.
Vol. 1, June 8, 2001
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APRIL AWKWARD, et al.,) IN THE
Plaintiffs) CIRCUIT COURT
vs.) FOR
) BALTIMORE CITY
JEROME I. SNYDER, M.D., et al.,) CASE NO.:
Defendants.) 24-C-00-004585

The Deposition of RONALD ALAN SACHER, B.Sc. was taken on Friday, June 8, 2001, commencing at 3:19 p.m., at the offices of Epstein, Becker & Green, P.C., Suite 700, 1227 25th Street, N.W., Washington, D.C., before Diane Gomez, Registered Professional Reporter and Notary Public in and for the District of Columbia.

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PROCEEDINGS

Whereupon —

RONALD ALAN SACHER, M.D.

a Witness, called for examination, having been first duly sworn, was examined and testified as follows:

MS. JONES: Sharon, just let me tell you we're going to limit him to causation, damages, and life expectancy.

MS. CHRISTIE: Okay. Well that takes out a whole bunch of questions.

EXAMINATION

BY MS. CHRISTIE:

Q: Dr. Sacher, I apologize for mispronouncing your name when we first met. My name is Sharon Christie. I represent the plaintiffs in this case. I'll be asking most of the questions today.

If at any time you don't understand my question, please tell me. I will be happy to

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rephrase. Please make sure that all of your answers are verbal so that the court reporter may take them down. And if at any time you feel that you need a break, please tell me and I'll be happy to do that. All right?

A: Yes.

Q: Great. Would you state your full name and business address, please.

A: My name is the same as I stated earlier, Ronald Alan Sacher, and business address is University of Cincinnati Medical Center, Hoxworth Blood Center, H-o-x-w-o-r-t-h, 3130 Highland Avenue, Cincinnati, Ohio 45267.

(Sacher Deposition Exhibit 1 was marked for identification and was attached to the transcript.)

Q: Dr. Sacher, let me show you what we've marked as Exhibit 1 which is a copy of curriculum vitae which Ms. Jones has provided to me. I ask you to take a look at that and let me if this is the most up-to-date version.

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A: It is, yes.
Q: Okay. Let me — if you want to hold on to that, I'll ask you a few questions, and if you need to refer to it, please do. Let me start first with the first couple of lines. There are a lot of different letters following your name. And I would ask you, if you would, to please go through each of those designations one by one and tell me what it stands for and what training was required in order to obtain that designation.
A: Certainly. The B.Sc. is a Bachelor of Science which is equivalent to the premed degree in United States. I did that in physiology and biochemistry in South Africa.
The M.B.B.Ch. is Bachelor of Medicine Bachelor of Surgery, which is the equivalent of an M.D. Again, my education was in Johannesburg, South Africa, and this is would be the medical degree that is given there.
F.C.A.P. is the Fellow of the College of American Pathologists. My training initially

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in South Africa was in pathology, although my focus was hematology, and in that capacity I have been and am a member of the College of American Pathologists, and for at least seven years at Georgetown was chairman of the department of laboratory medicine as the head of clinical pathology. I also have pathology boards.
F.A.S.C.P. is Fellow of American Society for Clinical Pathology, which is a similar organization. It is an elected fellowship to the College of Clinical Pathology, as was the F.C.A.P. to the College of American Pathology.
D.T.M.&H. Is Diploma in Tropical Medicine and Hygiene. It is the equivalent of tropical medicine board exams.
And F.R.C.P.C. is Fellow of the Royal College of Physicians of Canada. I completed my training in Canada in hematology and hematopathology, and this is equivalent to those boards.
Q: All right. Now, your medical

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training or what we would call medical school in the United States as I understand it was in South Africa. Is that correct?
A: My medical school was in South Africa, that is correct.
Q: And then did you complete a residency along the lines of the training in the United States varying from a few years up to I suppose seven or eight years, depending on the specialty?
A: Actually, I never did a residency in the United States. I completed some of my residency in Canada, although I was already certified when I reached Canada, and did Canadian certification. I was then attracted to Georgetown University Medical Center where I completed part of a fellowship, and subsequently my credentials were recognized and I became a member of the faculty in 1978.
Q: Okay. And the fellowship that you worked on at Georgetown, what was the focus of the concentration?

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A: That was a fellowship in hematology practicing as an internist, and subsequently all my clinical activities at Georgetown were in the department of medicine or internal medicine in the division of hematology and subsequently in the division of hematology oncology.
Q: You also mentioned some work in the area of hematopathology. Is that right?
A: Yes.
Q: Could you define that for me?
A: I am a certified hematopathologist and as a hematopathologist, that is the pathology discipline that studies the tissues and diseases of the tissues and organs that are involved in the manufacturing of blood cells as well as diseases that affect all the blood cell and blood fluid organs.
Q: All right. Now, you've listed your current appointment from November of 2000 to the present as professor of internal medicine at the university — internal medicine and pathology at

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the University of Cincinnati. Can you estimate for me what percent of your professional time is spent in activities related to your work as a tenured professor at that medical center?

A: Well, let me preface my answer by saying, of course, that I just recently joined the University of Cincinnati. I was recruited there to run the Hoxworth Blood Center, which is a nationally regarded blood center. And I am the head of this program, and in that program it involves many aspects of laboratory testing, the procurement and storage of blood and blood components, the laboratory of cellular immunology and transplant immunology, the laboratory of cell therapy.

So everything related to blood and its processing and its manipulation is included in that domain. Now, to the extent that I am the administrative head of this program, I also am involved in teaching internal medicine and do morning report and ward rounds and lectures to the

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residents and fellows in hem/onc and the residents in medicine.

I have not restarted my practice in Cincinnati that I had in Washington because I've been getting familiar with the processes and procedures there, but that is due to start in July, and my practice involves the management and treatment and diagnosis and management, if you will, of patients who have all manner of blood diseases ranging from leukemias, lymphomas to clotting and bleeding problems. I have a special interest in that area as well as hematologic problems of pregnancy. But my practice would be the same, I would hope, as it was at Georgetown which involved treating patients with all blood diseases.

Q: Okay.

A: So I think that covers my clinical teaching and administrative responsibility. I also have a research program and actually just recently we applied for a very large NIH grant to

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study the natural history of perinatal Hepatitis C. When I was at Georgetown and even currently I'm involved with transfusion transmitted viruses and the outcomes of these sorts of problems.

Q: Okay. How long were you at Georgetown?

A: I was at Georgetown actually from 1977 in end of July, beginning of August as a fellow and then 1978, first of July, as an assistant — excuse me, assistant professor of medicine and pathology. I then got promoted from 1978 to I believe it was 1989, but that's in my CV, to full professor. I was tenured in 1983 I believe as an associate professor, and I became department chair of laboratory medicine in 1993, and left Georgetown in November of 2000.

Q: Okay. In your practice that you referred to before that you hoped to restart in Cincinnati in July, I believe you said, and you hoped that it was similar to that in Georgetown, did that practice involve care of patients on an

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inpatient basis as well as an outpatient or no outpatient, all inpatient? What kind of mix did you have?

A: My practice from 1978 to 1993 roughly involved in patient and outpatient. The inpatient activities were essentially rotating on service, and in the earlier years it was up to three to five months a year actually being the inpatient attending of record, seeing all the inpatients.

Also during that period we had an outpatient clinic which I was also involved with. So I developed quite a large outpatient base that I was following, and in 1993 when I took over the department of lab medicine, I mainly concentrated on the outpatient practice, but as a teaching attending I did inpatient teaching attending one month a year.

Q: Okay. Was your work with the patients — let me focus on the last five years say, just to narrow down the time frame a little bit. Your last five years at Georgetown was your

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work with the patients as the sole treating physician, or were you working in conjunction with other physicians in the treatments of these patients?

A: If the patient was a hematology patient, in other words, had a blood problem as broadly defined, then — and the patient was admitted under our service, I was the primary attending of record. However, clearly we also had a consult service where we would be asked to review patients who had a variety of blood problems that were identified as ancillary to their other diagnosis for what they were admitted.

Q: Okay. For those patients for whom you would be the primary — the attending physician, you mentioned broadly blood problems. What types of diagnoses did that include?

A: Oh, the gamut of hematologic problems. In fact, for the most part a lot of my practice were esoteric hematology complex cases, thrombosis and thrombotic predisposition, bleeding

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problems, the management of patients with lymphomas and lymphoproliferative disorders and the management of patients with myeloproliferative disorders and leukemias.

Q: Okay. Was there any particular area in which you concentrated or specialized within the broad category of blood problems?

A: I would say that although it encompassed every aspect of hematology, the majority of my patients had, in the latter part of my time at Georgetown, clotting and bleeding problems and problems in pregnancy. However, I had a very large patient population, as does any hematologist with lymphomas and lymphoproliferative disorders as well as some patients with myeloid leukemias. That tended to be more inpatient intensive, so towards the latter part of my time at Georgetown I had less in the way of the acute leukemia management, but I still did manage my patients as well who had that.

Q: Okay. You've listed in your CV a

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very large number of publications, chapters in books, etc. And please feel free to look through when I ask this question, but do any of those publications, whether they be for journals or for any other purpose, either listed in your CV or perhaps some that haven't made it to your CV, that you have authored, do any of those deal with any of the issues that you believe are pertinent to the opinions that you're giving in this case?

A: Yes, in a general sense I think there are some that actually deal with the diagnosis of lymphoproliferative diseases, and I think that's the focus obviously of the case in Mr. Awkward.

Q: Okay. Could you point those out to me, please.

A: Just before I do that, I will make a general comment about a virus that we've been studying that causes lymphoproliferative diseases, and that is called the human T lymphotrophic virus, and we have been following this — when I say we, colleagues of mine from around the

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country, we have an NIH grant that in fact follows a cohort of people who are infected with this virus that can produce a lymphoproliferative disorder. But I'm not going to specifically refer to those publications.

Q: All right.

A: Page 15, citation — fifth bullet from the bottom starting off with Ratko, R-a-t-k-o, that is an article that we published on the use of immunoglobulin preparations that includes individuals with chronic lymphocytic leukemia and lymphoproliferative disorder who are notoriously deficient in these natural antibodies. And there are a couple of publications that I have in this compound called IVIG, and I've given talks as well on the subject of replacement therapy in chronic lymphocytic leukemia and lymphoproliferative disorders.

Q: Okay.

A: Page 16, bullet five from the bottom, first author Evers, E-v-e-r-s. This is a case of

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an individual with a T-cell lymphoproliferative disorder that we reported. And actually on that page second bullet from the bottom, that is where I allude to the IVIG role in bone marrow translate malignancy in immune hematologic disorders including lymphocytic leukemias and lymphocytic lymphomas.

Q: Okay.

A: The last citation under Articles on page 20, before books, is a publication on a disorder called macroglobulinemia. This is a lymphoproliferative disorder of a type. It's analogous to a lymphoma, and in these individuals they make an abnormal antibody.

Q: Okay.

A: Then under Books, the book that I authored, citation one under Books, Clinical Interpretation of Laboratory Tests as well as its previous edition, the tenth edition, I wrote a fair amount on lymphoproliferative disorders in that with regards to the diagnosis and its more

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diagnosis than management.

Q: Okay.

A: Also citation two under Books I was the co-author of this little manual called The White Cell Manual which really evaluates diseases of white blood cells which clearly includes lymphoproliferative disorders.

Under Chapters, page 21, citation one, this is Immunohemolytic Anemias in Hematologic Diseases that includes chronic lymphocytic leukemia and lymphoma.

Page 25, citation 10, this is an individual who had an acute lymphoproliferative disorder, acute lymphocytic leukemia or lymphoblastic leukemia that we published on because it had an unusual chromosomal marker, but it is included in that category.

Q: Okay.

A: And then page 25 under Journal Supplements, this is a supplement that I edited that includes lymphoproliferative disorders as one

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of the categories as well.

Q: Is that true for all of those that are listed, or were you referring to one in particular?

A: No. Citation four.

Q: Four. I'm sorry.

A: And I don't believe the attracts that I've got are really any more additive than what I've gone through and I think in general that covers the broad field of lymphoproliferative diseases that I've published in.

Q: Okay. Thank you. Dr. Sacher, have you ever been sued for medical malpractice?

A: No.

Q: When did you first become involved in reviewing medical/legal cases as an expert?

A: Actually, the first time I was ever asked to review it as an expert was probably in 1979. I was doing some research on platelets and the immune abnormalities that platelets have. And this was a case of an individual who had a heart

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attack after getting the swine flu vaccine. I was approach by a plaintiff's attorney and rendered an opinion against the U.S. Government in terms of the vaccine and its probability of causing a clotting event. That was the first time.

Q: Okay. In the last five years can you estimate for me on a yearly basis the number of cases that you review?

A: It's increased in the last year or two, and I would say up to about two a month.

Q: Okay. It's increased up to two a month now?

A: Yes. On an average. Some months three; some months one.

Q: Sure.

A: But in the last five years, I would say probably in the order of 10 to 15 a year.

Q: Okay. And over the last five years can you estimate the percent of cases you review on behalf of plaintiffs versus defendants?

A: Probably in the order of 70 to 75

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percent defense.
Q: Okay.
A: So it's 25 to 30 percent plaintiff.
Q: Okay. Other than the case that you mentioned, it sounded like your first case involving swine flu, are there other cases that you reviewed on behalf of plaintiffs that you have given opinions favorable to the plaintiffs?
A: Yes.
Q: Are there cases that you have reviewed and given testimony in, either by way of deposition or at trial, involving issues that are similar to the issues in this case involving lymphoproliferative disorders?
A: Yes.
Q: How many, other than this case?
A: I can recall one definitely, and there may have been others that I can't recall, but I would certainly say one where I gave a deposition.
Q: Okay. Do you recall approximately

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how long ago that case was?
A: I would think about five or six years ago.
Q: Do you know where the case was pending, what state?
A: It was in Virginia.
Q: A Virginia case, okay. Do you remember the name of either the plaintiff or the defendant in that case?
A: No.
Q: Were you testifying on behalf of the plaintiff or the defendant?
A: Plaintiff.
Q: Plaintiff. And do you recall who the plaintiff's lawyer was?
A: It won't come to me. I can't remember.
Q: Okay. If during the course of the questions this afternoon, sometimes answers will come to you. If it comes to you, please let me know.

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A: I will.
Q: Do you remember the name of the defense lawyer? That would have been who was probably asking you most of the questions.
A: No, I don't recall that either.
Q: Okay. How about the name of the plaintiff or the defendant?
A: That I don't recall.
Q: That case that you were just referring to, do you recall what the nature of the allegations were?
A: Failure to diagnose a lymphoma. Or should I say, to be more accurate, delay in the diagnosis of lymphoma.
Q: All right. And do you recall how old the plaintiff was at the time of the alleged delay in diagnosis?
A: No, I don't. This was an individual who had a growing mass that was never biopsied.
Q: Okay. Can you estimate for me — you've given me your estimate in terms of numbers

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of cases that you've reviewed. Can you estimate for me what percent of your professional time is spent either reviewing cases or giving testimony in deposition or at trial?
A: Oh, it's less than 20 percent I would say.
Q: Okay. On a monthly basis can you estimate the number of depositions you give per month? Again, focus on the last five years.
A: This year I believe I've given about six or seven, but prior to that maybe at the most six a year.
Q: Okay. How about trial testimony, how often does that happen?
A: I think that of the cases I've reviewed, it's very likely that 20 to 30 percent went to trial.
Q: Okay. And you testified in those cases?
A: Yes.
Q: Okay. What are your fees for review

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of records?
A: $400 an hour.
Q: How about deposition?
A: $500 an hour portal to portal with a minimum of a thousand.
Q: And trial?
A: Same rate for trial.
Q: As deposition?
A: Correct.
Q: Have you ever reviewed a case where there was an underlying allegation of a failure to diagnosis or treat a septic arthritis?
A: No.
Q: Have you ever reviewed a case prior to this one for either Ms. Jones or anyone in her law firm?
A: Yes.
Q: On how many occasions?
A: I would say probably two or three.
Q: And how about the other attorneys involved in this case, there's a firm in Baltimore

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by the name of Goodell, DeVries, Leech, now, & Dann — it used to be Leech & Gray — have you ever reviewed cases on behalf of anyone at that firm to your knowledge?
A: Yes, I have.
Q: Okay. And on how many occasions?
A: Quite a number of occasions, because I was also retained as an expert in transfusion-transmitted disease litigation and factor concentration for hemophiliacs, and they represented Bayer Corporation.
Q: Okay. Outside of that class of cases, do you have any idea of how many times you worked with the Goodell firm?
A: Probably about five or six.
Q: All right. And within that classification of cases involving the hemophiliacs, was it, and clotting factors?
A: Yes.
Q: Do you have any idea how many cases that involved?

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A: Oh, there was a very large list and I got records on a good number of them. Many of them were settled.
Q: Okay.
A: I really couldn't even estimate. More than ten.
Q: Okay. When were you first contacted regarding the Awkward case?
A: Probably just shortly before January the 5th of this year.
Q: Okay. Do you remember who made the contact with you?
A: Ms. Jones.
Q: What were you told about the case?
A: I don't recall specifically other than there was an issue of septic arthritis and there was a diagnosis of a lymphoproliferative disorder made on autopsy.
Q: At some point were you provided some material records or any other material related to the case?

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A: Yes.
Q: And what was it that you were provided?
A: I was initially provided with material records, both relating to hospital admissions, Saint Agnes Hospital and subsequently Union Memorial, and subsequently I was also provided with records from Mr. Awkward's primary care physician, Richard Hunt, M.D. Thereafter I was given some depositions. I also did receive the complaint.
Q: Okay. How about pathology slides, have you seen any slides?
A: Yes, I have.
Q: Okay.
MS. CHRISTIE: Jean, did you provide the doctor with a full set of pathology slides.
MS. JONES: Yeah, at least what was represented as a full set.
MS. CHRISTIE: Right. That's the best we can do. Okay.

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A: Excuse me.
Q: Yes, sir.
A: I also did receive some records from Church Hospital, I neglected to mention.
Q: All right. And you mentioned depositions. Can you tell me what depositions have been provided to you?
A: The deposition of David K. Sarver; deposition of Philip M. Buttaravoli, and also some attachments to his deposition, including articles.
Q: All right.
A: The deposition of Michael M. Kaufman, Jerome Ira Snyder. April C. Awkward, Desiree P. Awkward, Jerome Awkward, LaTracia Awkward. And I believe that covers what I was given, as well as, of course, the slides that we noted.
Q: Right. All right.
A: And I did see the x-ray of the joint, of the knee.
I should also mention there was a small amount or very limited records from the

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University of Maryland Hospital.
Q: Okay. And the records that, the papers that you have in front of you, does this constitute your file on this matter, or are there other things that you have related to this case?
A: No. This constitutes my file.
Q: All right. And the only other thing that I see here is the curriculum vitae of Dr. Snyder. Is that correct?
A: Correct. This was attached to his deposition, and it somehow got out of place.
Q: Okay. That's fine. After reviewing the material that you had been provided, Dr. Sacher, did you feel you had sufficient material in order to form opinions in this case?
A: Yes, I did.
Q: Okay. You didn't feel like you needed to see anything else?
A: No.
Q: Okay. And I'm going to get to your opinions in a minute, but let me go through the

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do-you-know questions first. Do you know Dr. Snyder?
A: No.
Q: Do you know any of the experts? And let me just review the names and see if any of these ring a bell. Dr. Sarver?
A: No.
Q: Dr. Kaufman?
A: No.
Q: Dr. Buttaravoli?
A: No.
Q: Whose name I always mispronounce.
A: No.
Q: Dr. Berg?
A: No.
Q: Dr. Remsen?
A: No.
Q: Dr. Crane, Leo Crane?
A: No.
Q: Dr. Dr. Weisburger?
A: No.

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Q: Joel Morse, who is an economist I believe?
A: No.
Q: Do you know any of the plaintiffs in this case? That would be April Awkward, Desiree, LaTracia Awkward, or Jerome Awkward?
A: No.
Q: And did you know the decedent who was also named Jerome Awkward?
A: No, I did not.
Q: Doctor, let me ask you this: When you were provided the records and the material to review, were you asked to review this material to develop an opinion with regard to any particular area?
A: Yes.
Q: And what was that?
A: I was asked to evaluate the degree of lymphoproliferative disorder that he had, what type it was. I was also asked to assess the effect that this had on the disease outcome as

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well as his potential likelihood of longevity.
And, in general, I was also asked to review the mechanism and the cause of death.
Q: All right. Let me take these one by one then. Did you come to an opinion as to the degree of lymphoproliferative — that's a tongue twister — disorder that Mr. Awkward had?
A: Yes.
Q: And what's your opinion in that regard?
A: My opinion is, and I concur with the original pathologist's diagnosis, which was called a well-differentiated lymphocytic lymphoma, that he had what we now call a small-cell lymphocytic lymphoma, and it is exactly the same disease entity. That more likely than not he had substantial involvement of his bone marrow as well as several organ systems and that this played a role in the rapid progression of the toxic shock syndrome, if you will, from the beta hemolytic streptococcal infection that he had, leading to

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the progression of the beta hemolytic step infection in his thigh.
Q: How do you define well-differentiated lymphocytic lymphoma?
A: There are many ways to classify lymphomas, but perhaps I could just start off by simply defining what a lymphoma is.
Q: That's fine.
A: It is an unregulated growth and proliferation of cells of the immune system termed lymphocytes.
Q: And what does the term well-differentiated — well, what does the term "lymphocytic" add to that description of lymphoma?
A: Lymphocytic refers to the cell appearance or morphology and the well-differentiated refers to the maturation of the cell.
Q: Okay. You also mentioned small cell. What significance does that have?
A: The mature lymphocytes are generally

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small cells.
Q: Okay.
A: And the well-differentiated implies that they are small and they have regular round nuclei.
Q: Okay. There have been descriptions, I'm sure you've seen it in the testimony in this case, opinions given that this was in essence an indolent disease, not — and I'm going to paraphrase, but one of the experts has testified that in his opinion this was something that Mr. Awkward and many persons die with, do not die from. What's your opinion in that regard?
A: That is true in a good number of patients with this type of lymphoma, and I might also mention at the start that this type of lymphoma can spill over into the bloodstream at which time it's called a chronic lymphocytic leukemia but it's the same disease entity and same cells as the swelling in the lymph nodes which is refers to as the lymphoma, "oma" meaning tumor.

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Q: Okay. What about in Mr. Awkward's case, do you believe that it was true that it was indolent in his particular case?
A: Well, the disease itself in the way that it progresses for the most part implies a slow growth, and in that context is indolent. But there are several clinical stages that we refer to, or degrees of disease, that have prognostic implications.
Q: What are those?
A: There actually are two different classifications, but I will refer to the one term, the Rai, R-a-i, classification. And by my estimation and review of the records as well as autopsy findings and Dr. Hunt's records, Mr. Awkward had at least a stage three.
Now, that staging is generally referred to in the context of chronic lymphocytic leukemia, but I'm using those two terms synonymously because he had extensive involvement of his bone marrow. And many patients, many that

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I've treated, have we refer to as aleukemic lymphocytic leukemia, which is the same as having the lymphoma predominantly involving the marrow but also involving the tissues without necessarily extensive involvement of the bloodstream.

Q: Okay. In your opinion did he have the lymphoma or did he have the leukemia or does it not make much difference?

A: In my opinion he had the lymphoma from the point of view that he had a large lymph node in his abdomen.

Q: Yes.

A: Many lymph nodes, as a matter of fact, according to the autopsy. And also an enlarged spleen. He also had involvement of other organs such as the liver. But he had involvement of his bone marrow as shown by the autopsy findings of lymphoid nodules in a very unusual site, the patella. The kneecap.

So I believe that he had extensive involvement of his marrow that didn't spill out

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into his bloodstream. Or if it did, it was attenuated by the treatment that he had been getting, the Prednisone.

Q: Let me back up first to ask you some questions about the staging. You said I believe you said you're using the Rai classification?

A: Yes.

Q: Okay. And in your opinion Mr. Awkward is at least a stage three.

A: Correct.

Q: Under that classification how many stages are there?

A: Four.

Q: All right. How do you determine, how would you determine what stage a given patient was in?

A: It is based on the laboratory findings as well as the clinical findings. And, again, I should also preface my answer by saying that there are a lot of classifications, and this is a complex disorder because it's based not only

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on the nature of the cells and their involvement of the organ systems and lymph nodes, their pattern of involvement, but also the effect that they have on the bone marrow.

And in this case why I'm categorizing him as at least stage three is that the records from Dr. Hunt indicate that he was anemic for a good many years while he was being followed by Dr. Hunt, and indeed at least three years previously he had evidence of splenic malfunction.

Q: Could you point out to me specifically what it is you're relying on in Dr. Hunt's records?

A: First of all, if one looks at the laboratory results dated, and I believe this is May the 28th, 1998, and I might say that this is the most recent laboratory test that I saw prior to his admission to Union Memorial, at that time he had a hemoglobin of 11.1 with a hematocrit of 35.7. That is anemic. So by definition anemia puts him into stage three.

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Q: Simply by virtue of having anemia on blood, as you pointed out, on his blood work?

A: Correct.

Q: That's sufficient to put him into stage three?

A: That is correct.

Q: All right.

A: Parenthetically, this test result also shows platelets that are somewhat large, suggesting increased platelet turnover and a test called a sedimentation rate which is very high, indicating that there was some active inflammation going on, presumably the same disorder that was causing Dr. Hunt to treat him with steroids, i.e., what was called vasculitis.

Q: Okay.

A: This anemia was noted also in December of '97 on Dr. Hunt's records. And indeed this anemia was also noted in April of '96, but more specifically there was evidence of splenic malfunction noted here by the demonstration of

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what's called Howell-Jolly bodies.
Q: And what are those?
A: Those are remnants of the nuclear material in red blood cells that are normally removed by the spleen, and finding them implies either having no spleen or a spleen that's not functioning very well.
Q: Was that finding noted on any of the subsequent blood studies?
A: I did not see any mention made of that.
Q: Okay.
A: On the subsequent studies, but they do indicate moderate Howell-Jolly bodies, and that certainly is of significance.
Q: Would you expect to see that as a continuing finding?
A: Yes.
Q: If the spleen was malfunctioning in 90 — I believe you said '96?
A: I would expect that, yes. But of

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course we do know that his spleen was enlarged, and enlargement of the spline actually in and of itself implies grade two. Subsequently with, of course, the anemia, this implies grade — well, stage three.
Q: Okay. Are there other causes of splenic malfunction other than a lymphoproliferative disorder?
A: Yes.
Q: Can you tell me what some of those are?
A: Well, certainly certain autoimmune disorders might cause that. If the spleen is overworking that it can't perform its cleaning function of removal of these particles from the red blood cell, then it will not do so, and it may be overworking by clearing out other bits of immune debris.
Q: Okay. Is there any indication in your opinion of any reason other than a lymphoproliferative disorder that would cause

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evidence of a splenic malfunction in Mr. Awkward?
A: No.
Q: Let me just take a look at this. In April of '96 I believe you've indicated in your opinion he was also anemic at that time. Is that correct?
A: Yes.
Q: Okay. And at that time he had a hemoglobin of 13.1 and a hematocrit of 39.2. Is that right?
A: Correct.
Q: Okay. Other than the anemia, are there any other findings that in your opinion are findings that would put Mr. Awkward in a stage three classification as opposed to some other level?
A: No. That's the definition of it.
Q: Okay.
A: I did find that there was a point where his platelet count was low, but this was preterminally and it could be explained by other

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reasons. Platelets counts being low puts one into a stage four.
Q: Okay. That was my next question, what would put someone into a stage four. Is there anything other than a low platelet count?
A: No, that's the definition, the phenomenon of thrombocytopenia, which is a low platelet count.
Q: Okay.
A: I will again note that he did have an increase in his mean platelet volume, suggesting increasing platelet turnover, which goes along with a lymphoproliferative disorder or an autoimmune disorder.
Q: Okay. Back to the classification for a moment, what puts someone in a stage two, what findings puts him in a stage two?
A: Enlarged lymph nodes and spleen.
Q: All right. And how about a stage one?
A: Stage one is an enlarged lymph nodes

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alone, and stage zero is just simply an elevation of the white cell counts.
Q: Okay. The staging that you referred to, what is that used for in terms of treatment of patients?
A: Well, it's used primarily for prognostication and clearly treatment, since this is an indolent disorder, for the most part it's not a curable disorder, treatment is based on clinical symptoms and rate of progression of either white blood cell counts or tissue enlargement.
Q: Okay. In terms of prognostication, are you referring to life expectancy or other prognostications?
A: Life expectancy.
Q: Okay. What in your opinion is the life expectancy of someone in a stage three as Mr. Awkward was?
A: Between three and five years.
Q: And is that from the point of

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diagnosis or from the point that they first go into a stage three?
A: That's actually an interesting question to answer, because most of the time it is on the basis of the diagnosis. But, of course, we don't know how long they've that that disease before. But I will, to a reasonable degree of medical certainty, say that he had this disorder, this lymphoproliferative disorder dating back at least to 1996.
Q: What are you relying on for your determination that his life expectancy was three to five years? And by that I mean in terms of any studies or medical literature. Is there anything of that nature that you rely on?
A: Yes. The medical literature speaks to the issue of the Rai stages, and Rai stage factor is the prognostic category based on survival codes.
Q: Okay. And is there some book, text or journal articles that I could go to that

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discusses the Rai classification and in terms of life expectancy?
A: Yes.
Q: And what would that be?
A: In fact, any general hematology textbook that's current and up to date would refer to this. There are other ones termed the Binet classification, B-i-n-e-t, but I think a lot of North Americans use Rai, since he's a New Yorker.
Q: Okay. Can you give me any examples? You said any good hematology text, which I'm sure to you has a lot of meaning but to me, not being a hematologist, has no meaning at all. Are there any in particular that you could point me to?
A: Well, certainly my book is good.
Q: Okay. All right.
A: I'd be happy to refer you to that one. However, there are many general texts of hematology. There are texts that — Williams Hematology or Wintrobe that are useful resources. They're not authoritative but they're useful

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resources.
Q: Wintrobe?
A: W-i-n-t-r-o-b-e. There's Hoffman is another one. There are a good number of them.
Q: All right. Good. Now, you mentioned in terms, I believe in terms of classification that you saw involvement of the spleen, the liver and the bone marrow, I believe in Mr. Awkward's autopsy. Could you point out to me what it is you're referring to when you say that involvement of the spleen, what shows the involvement of the spleen and the liver and the — you mentioned a little bit about the bone marrow.
A: Well, actually I reviewed the slides myself.
Q: Oh, that's right. I had forgotten about that. Then tell me what it is you're relying on in the slides or in any of the other material that you've reviewed to base your opinion that he had involvement of those organs.

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A: Well, my answer is a combination of the gross findings on the autopsy as performed by the pathologist at the time who described enlarged lymph nodes in the abdomen and also an enlarged spleen.

Q: All right.

A: There was no description of any microscopic findings, although I'm sure that they were done since slide seem to have been taken. However, on my review of the slides there was quite evident accumulations of these small lymphocytes with mature features in a number of organs. And including, as I said, in the bone marrow, and the bone marrow site was the patella, which is very unusual. This is normally a very acellular or a hypocellular, meaning having very few cells, and yet there was a lymphoid aggregate in that bone marrow.

So I have no doubt that in fact extensively there was involvement of the rest of the marrow, and I think I'm almost sure that the

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anemia was a consequence of that.

Q: Okay. I'm going to have to ask you, if you would, to explain in layman's terms what you mean when you talk about the patella and lymphoid aggregates in the patella and what meaning that has.

A: The patella is the kneecap.

Q: Yes.

A: And normally in an adult the patella would be this kneecap. The marrow component of it really is devoid of cells. Since the major blood producing sites are in the vertebral column and the sternum and the ribs.

Q: Right.

A: Now, lymphoid aggregates implies that there are nodules, there are accumulations, circular accumulations of collections of these mature lymphoid cells or mature lymphocytes. So that is what I was referring to. There are collections of these lymphocytes in the acellular kneecap.

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Q: Where did they come from?

A: They come from — originally, of course, they are produced in the bone marrow and then they get programmed in different sites in the bone marrow or in the thymus gland, and then they circulate in the bloodstream and then they go to the organs that are termed lymphatic organs. That includes lymph nodes and the liver and the spleen.

Q: Okay. Then how in Mr. Awkward's case did they end up in the kneecap?

A: I think this emphasizes the degree of involvement that he had, and, as I've pointed out, he already had evidence of it extensively in the lymph nodes of his abdomen at a number of sites. Also had involvement of his liver, had an enlargement of the spleen with extensive involvement there. In fact, he even had some in his lung. And why it ended up in the kneecap I think is proportional to the degree of involvement of the marrow. It's a very unusual site, and I think it implies extensive disease.

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Q: Is it — but these lymphoid aggregates, they're not produced in the kneecap, are they?

A: No, but they in fact cede there from the bloodstream.

Q: Could their presence be explained in any way by the type of infection that he had?

A: No.

Q: Could it be explained in your opinion in any way other than the lymphoproliferative disorder?

A: No, not these types of cells.

Q: And you told me about the spleen, you found evidence in the spleen?

A: Yes.

Q: As well as the liver?

A: Yes.

Q: And that was on the autopsy slides from the spleen and the liver I assume?

A: Yes, in conjunction with the gross description of an enlarged spline.

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Q: Yes. Okay. All right. Was there evidence in your opinion of involvement of any other organs other than the spleen and the liver?
A: Well, I believe I indicated that I did see a tissue section from the lung, but for the most part those were the major organs.
Q: Okay. And what specifically did you see in the lung?
A: A small aggregate of lymphoid cells. In a site where they should normally not be.
Q: What significance does that have in terms of your opinion as to the progression of Mr. Awkward's disease?
A: That particular location, really very little significance. Other than the fact that again it's implying, but I think there's much more compelling evidence, that it's quite widespread and he had fairly extensive accumulation of these abnormal lymphocytes.
Q: Okay. Can you describe for me generally the progression of a well-differentiated

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lymphocytic lymphoma of the type that Mr. Awkward had from the beginning to end stage?
A: Unfortunately it's not an arithmetic or natural progression as it goes from stage one to the next to the next to the next. It may actually start out quite extensive and suddenly involve a number of different sites and produce its effects. But, in general, as I implied by the definition, this is unregulated growth of cells that are normally part of the immune system, and as being part of the immune system they circulate in the circulatory system that involves the immune circulation. We refer to this as the lymphatic system.

So any lymph node that's part of the lymphatics can undergo enlargement and any organ system where the cells normally congregate can undergo involvement, liver and spleen being part of that. So, therefore, in summary, it starts off in the marrow but it could also start off at anywhere along that circulation where these

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abnormal cells grow out of control.
Q: Okay.
A: And then they may either grow and expand that area where they originated or they may spill out into the rest of the circulation and spread and cede to other areas.
Q: And is there any treatment for this disorder?
A: There is treatment. One can control the rate of growth and accumulation of these cells, for example, in the blood and even in the marrow. One can control the rate of expansion of the lymph nodes themselves or even enlargement of the spleen by either chemotherapy or irradiation therapy. But — and indeed there are some individuals who have actually been "cured" by bone marrow transplantation. But my own personal experience in an individual who got a bone marrow translate from his identical twin, it relapsed. So the implication always is that these indolent diseases are not readily curable. They may be

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controllable to some degree, but ultimately they get out of control.
Q: Okay. To what degree in your opinion is a well-differentiated lymphocytic lymphoma, stage three, as you said Mr. Awkward was in, to what extent is that controllable?
A: Again, in the aggregate — now clearly there are individuals, and I personally had experience with people who have a difficult time with any treatment that you give to them, because if the marrow is extensively involved and treatment is given, all their blood counts decline, and sometimes they don't respond very well to that. These cells are very complex and they're very idiosyncratic. They are part of that individual's immune makeup. So depending upon which cells of this immune makeup are abnormal and accumulate, so you can get a variety of features. Vasculitis could be one. Immune destruction of any tissue could be another. Or immune destruction of blood cells. Or it could be growth

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and proliferation of any site within that circulation.

So it's very difficult to say, one, how they can naturally progress. We do talk in terms of aggregates and groups of people. And also, although using the Rai stage, and, again, this is part of the reason why there's so many different classifications and stages, is because not only their appearance but their biological behavior can be very varied. There is a type of lymphoma called a mantle cell lymphoma, which is also part of the spectrum, that does very much worse.

Q: Okay. But how about in Mr. Awkward's case. Do you have an opinion as to whether his disease at the stage in which you've testified that he was in at the time of his death, the extent to which that was controllable?

A: I think that the lymphoproliferative disorder per se might have been controllable but its biological behavior was such that, as

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demonstrated by this galloping infection that he had, that at any time he could succumb to a devastating infection.

Q: Okay.

A: Whether or not that was treated.

Q: Let's take away the infection for the purposes of my question. Okay. And let's assume for the purposes of this question that he did not develop the streptococcal, Strep A infection that he had. If he had been — if he had been diagnosed and treatment was begun, do you have an opinion as to the degree to which his disease, his lymphoproliferative disease could have been controlled?

MS. JONES: Objection and a hypothetical. Go ahead.

A: Without further data, I could not speculate. Namely, the degree to which his natural antibody level was decreased, and I believe it was. Again, this is based on my review of the pathology diagnosis; for example, the spleen

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showing quite extensive diminution of normal natural lymphocytes. And also the adrenal gland showing really quite extensive atrophy from the steroids that were given to control the vasculitis presumably. So it is difficult to dissect those two features out, because I believe that they are related.

Now, conceivably, if he was given treatment for the lymphoproliferative disorder, had that been diagnosed, he may well have lived three years. Again, my figure that I'm throwing out is based on best estimates of the staging system as well as his own idiosyncratic disease features.

Q: Okay. Based on your review of the records did you see any clinical signs of the lymphoproliferative disorder outside of the blood work that you've referred to, and the findings on autopsy. Prior to — I'm talking about prior to the time he went to Saint Agnes Hospital. Did you see any clinical signs other than the blood work

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that we've talk about?

MS. JONES: Objection to the form. Go ahead.

A: Clinical meaning from external examination?

Q: Yes.

A: No, I didn't see anything on external examination, but clearly, as I pointed out, laboratory-wise as well as subsequent on autopsy, it was there.

Q: Okay. What about the vasculitis, do you think the vasculitis was in any way connected to the lymphoma?

A: I do believe that it was, and I guess in answering your previous question, could that have been a clinical sign, I believe that more probably than not it was. Because we had a phenomenon that was presenting itself with evidence in the laboratory of ongoing inflammation, yet it seemed to be controlled by steroids. And, although there was no tissue

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diagnosis of vasculitis, the clinical features were suggested, as Dr. Hunt had indicated; and I do believe that without an explanation for the vasculitis that — and knowing that he had a lymphoproliferative disorder, that they were related. More probable than not.

Q: You mentioned earlier, when I asked you sometime ago about your opinions, that you had opinions involving — regarding the involvement of the bone marrow and the several organ systems. I believe we've covered that. I just want to make sure. We talked about the liver, the spleen, the patella. Was there anything else you were referring to?

A: No, I think we covered that.

Q: Okay. You also said that you believed that lymphoma played a role in the rapid progression of the toxic shock syndrome. Is that right?

A: Yes.

Q: Okay. Explain to me what that

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opinion is and what you're basing it on.

A: Well, toxic shock syndrome is really an aberrant immune response to the organism. And it involves a concept termed superantigen, antigen meaning a foreign material that comes into the body that is normally handled and processed by the immune system.

Now, clearly the immune system and immune surveillance involves the cells of the immune system as well as mediators. His immune system we know was abnormal. It was abnormal for several reasons. One, he had the lymphoproliferative disorder, and, two, he was also clearly immunosuppressed from even that low dose of steroids as determined by his adrenal gland. However, the uniqueness of this organism — because not everybody who get a streptococcal infection develops toxic shock syndrome — the uniqueness of this organism in producing the toxin in an individual who is already susceptible and compromised by a disturbed immune function I

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believe was all additive and allowed this process to gallop. And it clearly was. And, in fact, even the histology and the appearance of the tissue, that there was no inflammatory response to the toxin implies a, what we call a paralysis of the immune system or paresis as well as the toxin itself causing tissue destruction.

Q: Do you have an opinion as to when the infection began?

A: I know exactly when it began, but I believe that it probably was evident, was certainly in his thigh on the 18th, but I don't know when it began.

Q: But you do believe it was present at the time he was seen at Saint Agnes?

A: Probably.

Q: I take it you believe that the site of infection was the thigh?

A: Yes.

Q: And what are you basing that opinion on?

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A: On my review of the records, material as well as my background, education, experience, and knowledge of the immune system.

Q: Okay. Tell me, in terms of your knowledge of the immune system, what is it that leads you to conclude that it was the thigh where the infection started?

A: Well, clearly that is the template on which I'm analyzing the medical records, and the records show extensive involvement and tissue destruction of the thigh and relatively little destruction or damage of the knee joint itself, or even the cartilage of the knee joint. So I firmly believe that this occurred in the thigh, and I think that any fluid accumulation and involvement of the knee was secondary to the evolving toxin release from the thigh.

Q: If the infection began in the thigh as you've testified and there was fluid collection in the knee as a result of the, if I've understood you correctly, as a result of the spreading

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toxins, do you have an opinion as to whether an examination of the fluid in the synovial joint would have shown the presence of the streptococcal A infection?

MS. JONES: I'm going to object only on the basis of timing. Are you talking about —

MS. CHRISTIE: At Saint Agnes.

MS. JONES: I don't know what Dr. Sacher was talking about was accumulation of flood by the time he got to Union or —

MS. CHRISTIE: I'm talking about at Saint Agnes.

A: My answer actually was considering the evolution toward the time from Saint Agnes to Union, when clearly a tap was done.

Q: Right.

A: I believe and I have an opinion that if a tap was done at Saint Agnes it would not have been diagnostic.

Q: Why not?

A: Because I think that this was a

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predominantly toxin-related damage produced by a local infection in the thigh and, with the absence of the inflammatory response, I don't know how easy it would be for anybody to interpret the results at Union when it was already manifest, just looking at those results, compared to 36 hours previously at Saint Agnes where I really am convinced that it would have been nondiagnostic.

Q: Can you explain to me how you believe the infection spread once it started in the thigh?

A: Well, I think it's evident that these sorts of phenomena are related to toxin-induced tissue damage. Of course the organisms are there producing the toxin, but the toxin causes severe damage to the tissue, and once that tissue barrier is compromised, then, of course, it spreads beyond that site of original injury.

Q: And how does it spread? Is it from adjacent tissue to adjacent tissue, or does it spread in some other fashion?

A: In those circumstances, from adjacent

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tissue to adjacent tissue. Of course, one can get blood spread subsequently. On even as part of that.

Q: How about in Mr. Awkward's case, do you have an opinion as to how the organism spread from the thigh?

A: I think it was by contiguity, from adjacent tissue to adjacent tissue.

Q: You've said that you don't believe a tap at Union Memorial would have been diagnostic. Is that correct?

A: That is correct.

Q: Do you have any opinion as to what the cause of the knee pain was that he complained of on admission to Saint Agnes?

A: I believe that was referred pain.

Q: And what do you mean by that?

A: Specifically, that there was infection in the thigh and that with the presumably evolution of the toxin, that although this — at what point did the toxin totally

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overwhelmed the system I don't know. It was in that 36-hour period. But this produces a clinical reaction, and obviously the bodies senses that as a need for a response, and pain is part of that response.

Q: In terms of the knee fluid, when Mr. Awkward got to Union Memorial they did tap the knee, correct?

A: Yes.

Q: And that did yield some positive results in terms of the presence of an infection. Is that correct?

A: Yes.

Q: Do you have an opinion as to at what point between the time Mr. Awkward got to Saint Agnes and the time that he was admitted to Union Memorial, at what point along that time line the knee — a knee tap would have been positive?

A: No. It would be total speculation. I really have no opinion at what point it would be other than evolving.

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Q: But it was not diagnostic in your opinion at Saint Agnes. Is that correct?
A: That is correct.
Q: Would not have been. Okay.
A: In fact, I would have expected much more of an inflammatory response under normal circumstances in sepsis of a knee joint than he had. Again, I think it was really quite attenuated by his disease processes, both the steroids as well as the lymphoproliferative disorder.
Q: What do you mean by that? I'm not sure I understand that.
A: Specifically, one tends to find higher levels of certain types of cells in an aspirate from the knee in a septic arthritis, and he didn't have nearly that degree. And also there were I think occasional organisms identified. I don't know how one would necessarily interpret that in the setting of the lower counts and the fact that he really had little, if any, elevation

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of his white cell count, overall blood count.
Q: Okay. At which point are you talking about?
A: At Union.
Q: At Union?
A: Yes.
MS. JONES: Could I just take a short break?
MS. CHRISTIE: Oh, sure. Absolutely.
(There is a recess from the record.)
Q: The higher levels of certain types of cells that you mentioned, what types of cells are you referring to?
A: Cells that are called polymorphonuclear leukocytes, neutrophils.
Q: Okay. And I want to make sure I understand what you said. You would have expected to see higher levels of those cells at Union?
A: Yes.
Q: And then you mentioned occasional organisms?

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A: I looked at what I thought were two tissue reports, reports of fluid. One was really mostly unremarkable other than the occurrence of occasional bacteria.
Q: Okay.
A: In fact, if we go back to Union, perhaps I should look at the record to be quite accurate.
Q: Okay. That's fine.
A: I was referring specifically to the report entitled Sterile Body Fluid collected on 1/20/99 at 1317 hours.
Q: Okay.
A: Indicating few gram positive cocci in pairs, no polymorphonuclear leukocytes seen.
Q: Okay. What is the significance of the few gram positive cocci in pairs?
A: Well, in this context one could argue that this may well be organisms that have colonized the outer portion of the skin. One can still get organisms, even though one might

Page 73

sterilize the skin, that actually may grow. Certainly one would expect that if bacteria were in the joint, there would be an inflammatory response in response to those bacteria.
Q: Can you say to a reasonable degree of medical probability that the report of the few gram positive cocci in pairs is as a result of contamination from the skin as opposed to what was actually in the knee aspirate itself?
A: No, I can't. Again, I'm not sure, because there was another report of surgical wound left knee with moderate polymorphonuclear leukocytes and many gram positive cocci in chains, where these are coming from. But, again, I think that with all of this and the relatively low white cell count in the bloodstream, it would be difficult to totally interpret and make the diagnosis of a septic arthritis, primary in the knee.
Q: Okay.
A: I think this is most consistent with

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spread from the thigh.
Q: Very early on when I asked you what you were asked to do with regard to this case and what you had opinions that you're prepared to render, you said you were asked to evaluate the degree of the lymphoproliferative disorder and the type, which we discussed, correct?
A: Correct.
Q: Okay. You also were asked to evaluate the outcome of the disease? And I wasn't quite sure what you meant by that. Or I may have misunderstood what you said?
A: Well, perhaps I doesn't articulate it as well as I should, but the implication was the relationship between the disease, the lymphoproliferative disease, the steroids that he had been receiving, and the rapid deterioration following the infection with the organism and then subsequent development of the toxic shock syndrome.
Q: All right. In my understanding,

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based on discussion we've just been having is that your opinion is that the lymphoproliferative disorder, as well as the steroid use, added to the rapidity with which this infection spread. Is that right?
A: Yes. Although, again, I think I also implied in a previous answer that the superantigen toxic shock syndrome phenomenon could occur in anybody with a degree of rapidity, but I believe that since this is all immune related that it probably compounded the situation.
Q: Okay.
A: And I think we discussed that.
Q: Indeed. You've told me that you believe that Mr. Awkward had a life expectancy of three to five years, correct?
A: Correct. I think we already discussed my statement that I thought more probable than not it would be closer to three for the reasons that I articulated.
Q: Yes. And then you did explain that.

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And then you also said that you were asked to evaluate or discuss the mechanism and cause of death. Is that right?
A: Well, only in the context that this was a galloping toxic shock syndrome from a streptococcal organism and this produced a fulminant picture with death as the outcome.
Q: Okay. You've answered my question. You don't think the cause of death was anything other than the infection, do you?
A: No.
Q: Have you been asked to evaluate any other aspects of this case?
A: Not as far as I know. I think we've covered pretty much everything. Obviously, I'll respond to any questions that are asked of me.
Q: And I do understand that. We have talked about your opinions regarding life expectancy, and I believe we've covered your opinions regarding the cause of Mr. Awkward's demise and the degree to which the underlying

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lymphoma and steroid use contributed to that. I just want to make sure we've covered all the opinions that you hold in regard to those topics.
A: I believe so.
Q: Okay. If you give me a second, I think we're all going to be off the hook here.
Doctor, you mentioned early in the deposition that one of the things that you also reviewed was the x-ray of the knee.
A: Yes.
Q: Did you find anything that was significant in that x-ray with regard to the opinions that you've rendered today?
A: No, I wouldn't say that it changed or added to my opinions.
Q: Okay. If you review any additional material or you develop any additional opinions in this case that we've not yet discussed, and there may be other depositions that you see because there are a few more depositions that remain to be taken, would you please let Ms. Jones know that

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you have additional opinions so that she can let me know.

A: I'd be happy to do that.

Q: Thank you. And then finally, this case is scheduled for trial November the — beginning I believe November the 26th of this year in the circuit court for Baltimore City. Do you intend to testify at trial?

A: I do, yes.

MR. MANOOGIAN: Objection. He will testify or not at the request of counsel. So I think that's a matter you would have to take up with Ms. Jones. However, to be on the safe side, plan on him being there.

MS. CHRISTIE: I assumed he would be but that's why I asked.

Q: Occasionally I have had experts say no, I'm going to be out of the country on that day, then I know they're not going to be there.

That's all the questions I have.

EXAMINATION

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BY MS. LACEY:

Q: Doctor, this is Lauren Lacey. I'm representing Saint Agnes in this matter. I just have I think one or two questions for you, and they're pretty much follow-up questions.

I listened to your deposition testimony today, and I take it that you have no criticisms of the care rendered by Saint Agnes' staff or nurses. Is that correct?

A: That is correct.

Q: And I heard the exchange before. In the event that you testify at trial, I am assuming that you will not testify to any breaches in the standard of care by Saint Agnes as well?

A: That is correct.

MS. LACEY: Okay. Thank you very much. No further questions.

MR. MANOOGIAN: My questions will take about two hours. Do you want to do them now?

We have no questions. We do not waive reading and signing.

(Thereupon, at 4:51 p.m., the deposition was adjourned.)

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District of Columbia
to wit:

I, Diane Gomez, a Notary Public of the District of Columbia, do hereby certify that the within-named witness personally appeared before me at the time and place herein set out, and after having been duly sworn by me, according to law, was examined by counsel.

I further certify that the examination was recorded stenographically by me and that this transcript is a true record of the proceedings.

I further certify that I am not of counsel to any of the parties, nor related to any of the parties, nor in any way interested in the outcome of this action.

As witness my hand and notarial seal this_____ day of _____, 2001.

Diane Gomez
Notary Public
My commission expires: 6/14/2005

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Deposition of Ronald Alan Sacher, B.Sc.

June 8, 2001

CERTIFICATE OF DEPONENT

I hereby certify that I have read and examined the foregoing transcript, and the same is a true and accurate record of the testimony given by me.

Any additions or corrections that I feel are necessary, I will attach on a separate sheet of paper to the original transcript.

RONALD ALAN SACHER, B.Sc.

WITNESS: Ronald Alan Sacher, B.Sc.
CASE: April Awkward, et al. vs. Jerome I. Snyder, M.D. et al.
DATE TAKEN: June 8, 2001

Please note any errors and the corrections thereof on this errata sheet. The rules require a reason for any change or correction. It may be general, such as "To correct stenographic error," or "To clarify the record," or "To conform with the facts."

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Lawyer's Notes