SARAH FOGEL, by her Parents and Next Friends, STUART M. FOGEL and MICHELLE L. FOGEL, etc., et al.,	) CIRCUIT COURT
	) ANNE ARUNDEL COUNTY
	) MARYLAND
Plaintiffs,	) CASE NO. C-96029703 OT
- vs -	)
WALTER LOCKHART, M.D., et al.,	)
Defendants.	)

TRANSCRIPT OF DEPOSITION OF ROBERT CLANCY, M.D., taken by and before CARMEN T. SANTIAGO, Professional Reporter and Notary Public, at The Wood Center, 324 S. 34th Street, Philadelphia, Pennsylvania, on Wednesday, July 22, 1998, commencing at 2:15 p.m.

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22nd Floor - Lewis Tower Building
Philadelphia, PA 19102
(215) 735-2332

A P P E A R A N C E S:

STEIN, MITCHELL & MEZINES, L.L.P.
BY: GERARD E. MITCHELL, ESQUIRE
1100 Connecticut Avenue, Northwest
Washington, D.C. 20036
Counsel for Plaintiff

WHITEFORD, TAYLOR & PRESTON, L.L.P.
BY: JAMES R. CHASON, ESQUIRE
210 West Pennsylvania Avenue
Towson, Maryland 21204-4515
Counsel for Defendant,
Walter Lockhart, M.D.

GOODELL, DeVRIES, LEECH & GRAY, L.L.P.
BY: THOMAS V. MONAHAN, JR., ESQUIRE
Commerce Place
One South Street
20th Floor
Baltimore, Maryland 21202
Counsel for Defendant,
Patuxent Medical Group

I N D E X

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E X H I B I T S

NUMBER	DESCRIPTION	PAGE MARKED	PAGE ATTACHED
1	Documents	7	n/a
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(By agreement of counsel, the signing, sealing, certification, and filing are waived; and all objections, except as to the form of the question, are reserved until the time of trial.)

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R O B E R T C L A N C Y, M.D., Sworn.

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EXAMINATION BY MR. MONAHAN:

Q Good afternoon, Dr. Clancy. This is a second deposition in this Fogel case. The first time occurred on May 30, 1997.

Could you tell me what, if anything, you have reviewed since then, that you had not seen when I last took your testimony?

A Okay. The basic materials were mostly in the form of hospital discharge summaries. Pretty much exclusively Johns Hopkins for Sarah in the time of her life, following the newborn period and largely related to her illness and the business about the vasculitis. Secondly --

Q Can you recall, before you finish,

how many such hospital admissions you saw discharge summaries for?

A I couldn't give you a number off the top of my head, but I could just share with you that every year there were multiple admissions.

Q Are they all there in front of you?

A Yes, they are.

Q Let me take a look at what you've been provided. Go ahead and tell me what else you've been provided since the initial deposition.

A Well, the other was the opportunity and the request to reexamine the child, and that was several months ago. I prepared a report describing my physical findings at that time. The report was dated June 12th, 1998.

Q What was the date of the exam?

A Probably the same date. I've seen some summaries of opinions from medical experts from the defense side of the case, stating opinions about causation and life expectancy. For example, Dr. Mark Scher who is a child neurologist and there was a neonatologist. I don't remember the names. I have those in here

someplace as well.

This is a package of information I received on April 8th, 1998 from Mr. Malone. And these are sort of summaries of opinions from Stanley Graven who is a pediatrician and neonatologist, and Mark Scher, Herbert Grossman, pediatric neurologist, Michael Rothman, a neuroradiologist. And there's references to other opinions, but the opinions weren't supplied.

Q Are there any other additional materials you've reviewed since May 30th, 1997 that relate to this case?

A The other issue was that the child had a scan done, an MRI scan done in, I believe in March.

Q Of this year?

A Of this year. And the particular purpose of the scan was to examine her cervical spine to see if there was any physical abnormalities of the cervical spine, and there were not.

Q Did you look at the films or just the report?

A Just the report.
Q Where was that done?
A I believe it was done -- well, what I used to call the D.C. Children's Hospital, now it's got another name. Children's National Medical Center in Washington.
Q Have you reviewed everything that's in this notebook that I'm looking at?
A Yes. Those are pretty much summaries.
Q I'm going to ask that this be marked as Exhibit Number 1.

(At this time, a document was marked for identification as Exhibit 1.)

BY MR. MONAHAN:
Q I gather Exhibit-1 was sent to you by Mr. Mitchell or someone in his office, correct?
A Correct.
Q And when did you get that?
A Well, I believe it was around January, this January. And I may be able to give you a more exact -- as I mentioned to you, I actually have a lot of medical records now

that are sort of the flesh part of these. These are just summaries that I did not bring with me. I don't have a cover letter here with this so, that's what I recall.

Q Do you know what medical records have been provided to you before you prepared your report of June 12th, 1998?

A Let me just look at the report for a second, see if I mention it.

MR. MITCHELL: When you say before, do you mean since the last depo, or do you mean inclusive of what he had already received.

MR. MONAHAN: Either one, however he sees fit to describe it.

THE WITNESS: I actually think I had the medical records when I saw her the second time in June, because part of the request was for me to make sense of the vasculitis story. I think I had the medical records then.

BY MR. MONAHAN:

Q Had you reviewed the records of her

treatment at Johns Hopkins in 1996 and 1997, at the time you prepared your June 12th, 1998 report?

A I believe I had. Yes.

MR. MITCHELL: For the record, I think he had those same records when he ordered the first report.

THE WITNESS: I did not.

MR. MONAHAN: He did not, according to his deposition testimony.

MR. MITCHELL: Okay.

THE WITNESS: I was not even aware of the vasculitis problem at the time of the first deposition. I think I learned about it during the deposition.

BY MR. MONAHAN:

Q Since then, since that first deposition, however, I gather you've had a chance to see Johns Hopkins' records regarding the multiple admissions for treatment of vasculitis and its sequela, is that right?

A Yes.
Q And you've undoubtedly seen records where it's described as vasculitis of unknown etiology by the Johns Hopkins physicians?
A Yes.
Q Do you agree that it's still a vasculitis of unknown etiology?
A That's sounds fine to me. I think the diagnosis is really one that is offered by the pathologist having looked at the tissue. In her particular case, it was a lung biopsy, and the course that was described would be consistent with the vasculitis. I, by no means, am an expert in this, but what I know about vasculitis, it certainly made sense.
They did look for some of the usual suspects when vasculitis shows itself, and none of those were found. I don't personally relate the vasculitis to the events of birth or anything that happened in the neonatal period. So in the sense that I don't think anyone knows why she has it, it is idiopathic. Yes.
Q Idiopathic is a fancy way of saying we don't know the cause of it?

A Well, the joke is that the doctors are the idiots and the pathology is someplace they haven't looked, idiopathic.

MR. CHASON: I like that.

BY MR. MONAHAN:

Q My wife says I don't have a sense of humor, so let me see if I can cut through the jokes and see if I can understand it. Is it true that idiopathic, as you've just used it, means that you don't have an explanation for the cause of the vasculitis?

A Right. And just, again, this is really terminology. There's always a reason for anything that happens in one sense, and if I could use an example. There are many children who end up developing a seizure disorder. And they come to the doctor and you do the scans, and you do the blood tests, and you do all the usual testing for why a child might have a seizure disorder.

And you end up with saying, well, I know she's got a seizure disorder. They witnessed the seizures, but I'll be darned if I know why this particular child has it at this

point in her life.

With time, some of those cases that were considered, quote, idiopathic, the cause is revealed in time. It turns out that there is a genetic background. They finally realized, well, the grandfather had the same thing as a genetic basis, or something shows up that wasn't clear.

It's not like it was a dumb luck sort of thing. There's always something behind these diseases, but there's just a lot of things where they don't really know what the fundamental mover is of the disease.

Q Is it fair to conclude from what you've told me that there are disease processes that current state of medicine simply is not able to identify the cause, even though there is a specific cause?

A Yes.

Q Do you think that's the case with her vasculitis, that there must be some cause, but the current state of medical expertise just hasn't advanced far enough for us to be able to say what it is?

A I think that's basically what it is.
Q I notice from your report that you indicated that you believe it first came on in March of 1996. What was the clinical picture then that led you to that conclusion?
A Well, really, this was all hindsight from my point of view. At the time, I don't think anyone understood what was really going on. So it's not meant to be critical in any way, but the basic issue is that she was having what are called paroxysms of coughing. And a paroxysm is just an explosion of coughing. She would be sitting there and just start hacking and hacking and hacking.

And of course, with all the trouble she had had neurologically and knowing that she had trouble swallowing, the first ten choices on the list is that she's aspirating, she's aspirating, she's aspirating.

And they're figuring that when she's swallowing her saliva or refluxing food into her throat, or just trying to swallow food, it's all going down the wrong pipe and she's just coughing because of aspiration. And by

far, like I said, the top ten players on that list would all be choking and aspiration.

Q Since she's not a smoker?

A Since she's not a smoker. Right. But then, as they say, the plot thickens insofar that she was also getting fevers that were unexplained, what are called FUOs, fevers of undetermined origin. And any one fever you can say, well, they must have had a virus and there was no rash, so it was just a virus.

But then when there's a pattern of having repeated fevers when there was no clear infection and so forth, and these paroxysms of coughing, they started to wonder if there was actually inflammation in her lung tissue. The inflammation triggered the fever. The inflammation triggering these bouts of coughing.

And it wasn't until she was so critically sick, and actually went so far as to get a lung biopsy that they were able to bring it into a single diagnosis, when you could look under the microscope and say, Oh, my God, the blood vessels are all inflamed, vasculitis,

even though they didn't know why they were inflamed. The cause was, quote, idiopathic.

Q And that diagnosis was arrived at in the latter part of 1996?

A Correct.

Q Have you reached a conclusion as to whether separate and apart from the vasculitis she does have a swallowing disorder?

A I believe she does, which is on a neurologic basis. And it's sort of part of the cerebral palsy picture that swallowing is a motor act and requires skill and coordination. Just like her arms and legs lack skill and coordination, the swallowing mechanism is defective also.

Q Are there hospitalizations that she has had before the diagnosis of vasculitis, which now with the benefit of hindsight, were probably due to the vasculitis and not due to aspirations from the swallowing disorder?

A They may very well be. It's actually hard to know. When they actually did some of these studies, swallowing studies, this is why they stayed on that path so long. They say,

Well, what do you know? The swallowing is abnormal and she was refluxing some, but it all seemed to be out of proportion to the intensity of these paroxysms. They had a little bit of swallowing disorder and a lot of paroxysms.

And like I say, it wasn't until the fever showed up that they took a step back and saw the bigger picture that this was more than just a typical case. So it's hard to say that any one hospitalization was actually just the vasculitis. I mean, for all I know it could have been some of both.

For example, if she did aspirate, that would inflame the lungs. Her lungs are already affected by the vasculitis, so they share the same tissue. Aspirations would affect the lungs and so did the vasculitis. So there could be some commingling of affects with that.

Q Do you think it's likely that, with the benefit of hindsight, some of the symptoms that in early, mid and into the fall of 1996 were attributed to gastroesophageal reflux, were, in fact, symptoms of vasculitis?

A Again, it could well be. If you could sort of replay that whole tape knowing then what you knew before that they probably were coexisting at that point.

Q Have you reached any conclusions to a reasonable degree of medical probability, that any of the hospitalizations in 1996 were solely due to aspirations or solely due to gastroesophageal reflux or solely due to vasculitis?

A I really didn't -- when I read through the records, I was just looking for understanding. I wasn't really reading the records to assign for each hospitalization what was the culprit here. So I can't really sit here and say I have an opinion about that.

Q You do comment in your report of June 12th, 1998 that her coughing spells culminated in a two-month hospitalization at the end of 1996. That would have been when the biopsy was obtained and the diagnosis made, correct?

A That's correct.

Q And she, in fact, was so ill that she had to be on intensive life support at some

point during the hospitalization, correct?
A That's correct.
Q Do you agree that the symptoms that brought her to that extreme condition requiring intensive life support were due to vasculitis?
A Yes, I do.
Q Why do you say you're, by no means, an expert in vasculitis?
A Well, there's actually an entire field of medicine of so-called collagen vascular diseases, which falls under the rubric of so-called rheumatology. So there are physicians who spend their whole careers treating children with conditions like lupus, juvenile rheumatoid arthritis, keratoconjunctivitis, SICCA, things that are recognized as immune disorders.
I certainly have patients with those disorders because many times, you know, there will be neurologic questions raised, but I, myself, am not the immunologist or rheumatologist that's the expert in that.
Q You're probably no older than me, so I don't say this to be insulting, but were

there subspecialties of pediatrics in immunology and rheumatology when you did your pediatric residency?

A Yes.

Q Did you have any rotation through those subspecialties?

A Yes, I did.

Q Is it fair to say, however, that since you started your pediatric neurology training, you have specialized in pediatric neurology?

A Yes.

Q Have you ever been an attending principally responsible for treating a patient with vasculitis, as opposed to having a consultant in rheumatology or immunology who you're relying upon to treat vasculitis?

A Occasionally. For example, one of these disorders goes by the name of pulseless disease, and it's a vasculitis of the aorta and the carotid arteries. And because of the vasculitis, these blood vessels thickened, and then the blood can't go through them, and these children have strokes and so forth.

So when the presentation of that disorder is stroke, they will end up on the neurology service, and you are their primary caregiver. Now, obviously we would be very dependent on the opinions of the rheumatologist to suggest diagnostic testing or how much steroids to give and so forth. But if it's a primary vasculitis, then I would not be taking care of their first line of treatment.

Q Have you ever had any patients like Sarah Fogel with pulmonary vasculitis?

A No.

Q Have you ever had any patients like Sarah who have vasculitis in her extremities to the point that it led to amputation of toes or fingers?

A As a consultant, yes. And the scene would be basically children who have heart or lung transplants. There's a large focus in this hospital for transplantation of organs, including heart and lungs. And there are other reasons for pulmonary vessels to be defective, and some of those are part of a systemic arteritis. One of my jobs is to take care of

the neurologic needs of those children, so I've had patients who have lost arms, for example, from vasculitis.

Q I gather from your report that you agree that her vasculitis has manifested itself in her lungs and in her peripheral circulation of her feet.

Do you have an opinion as to whether it has manifested itself anywhere else?

A I don't think it's actually manifested itself anywhere else. The point of that actually was that this was not a disease that was confined to the lungs. It actually had systemic manifestations, for example, the toes. So, it wouldn't surprise me, for example, if some time in the future there could be other manifestations. If it was in the heart and her toes, it might show up someplace else.

Q Are you able to say today that more likely than not it has not had any effect upon the vasculature of the brain?

A I don't think that's been specifically examined, really, to answer the

question one way or the other. In terms of, for example, doing an angiogram, that would be the gold standard test. And it's not that it's not done, but it's a painful test and there's a certain morbidity with it.

So unless there was really an issue that, Oh, this happened and we've got to get the answer to this question, just to do it because you want to know for academic reasons probably wouldn't be in the child's best interest.

Q Do you consider yourself to be current as to the appropriate ways to treat vasculitis of this magnitude? I'm talking about your personal knowledge of the literature and so forth.

A Well, in general, steroids are one of the mainstays of treatment. It's one of the first lines of treatments. And to that extent, I certainly agree that the steroids are the usual drug that the physician turns to for any kind of inflammation.

If it's asthma or, you know, even widespread poison ivy, you take Prednisone for

that. So it's certainly still one of the drugs of choice if it's tolerated. So I think that's a perfectly reasonable drug to have this child on.

Q Do you intend to offer any opinions concerning the prognosis for her vasculitis? And you probably understand I'm asking you if you have opinions that you can express to a reasonable degree of medical probability?

A No. I can't say that I have enough personal knowledge of the pulmonary vasculitis to talk about natural history and long-term responses.

Q And similarly, do you have opinions that you can express to a reasonable degree of medical probability as to how, if at all, it affects her life expectancy?

A Well, the only thing I can say at this point is that she clearly has responded to the steroids. That I think it would be one issue if they tried them and they pushed it to a high level, and they had no response. But they've actually had a good response in her particular case.

And, you know, part of the time when you give a drug and the person gets better, you want to pat yourself on the back and say, Well, my prescription of the drug is making her better. And the cynic always comes around and says, Well, a lot of these drugs are episodic. The illness waxes, and then it's going to wane. So you don't know whether it's the steroids making her better. It could have just been intended for this to resolve.

So one of the ways to answer that question is to take the person off the drug, and if it's really just the disease that's waning, then you take them off the drug and they continue to get better. On the other hand, if the disease flares up again, you're on stronger grounds to say, No, it really was the medications helping ameliorate this, and it wasn't just intended to take a turn for the better.

This actually happened in her insofar that they gave her a high dose. She seemed to respond, and they tapered quickly and she had a clear relapse. Put her back on the drugs and

her symptoms came back under control. So again, the real cynical person can say, That's all coincidence, but knowing that this is the drug for vasculitis and that this is sort of the nature of relapses with things like asthma and hyperimmune disorders, I think it's clear that the steroids are benefiting her.

So part of, you know, your question was what this does in the long run. And part of the answer is, Well, is she responding to the treatment? If there's no response to the treatment, that's bad for the patient or whatever the circumstances. She, actually, is doing pretty well with this treatment.

Q The relapse you're describing is what you referred to in your report as having occurred in May of 1997?

A That's right.

Q To your knowledge, has she been on daily steroids ever since?

A Let me see. I think it's alternating something or another. Let's see what it is. Yes, she's on daily steroids and what's alternating there is the dose. That's six

milligrams one day, and then a smaller dose. So the answer to the question is she's on daily steroids.

Q And is it true that even with that daily dosage of steroids, she still requires continuous supplemental oxygen?

A Yes. Continuous in the sense that it's on a daily basis. I believe that they are in the process of trying to wean that away. I recall that perhaps for one hour in a day she's not on oxygen, and they're trying to gradually extend that time when she's off of oxygen. But she's on oxygen through the night and on a daily basis.

Q Have you seen any records suggesting she's ever been maintained on anything less than 23 hours per day of oxygen, since the vasculitis was diagnosed?

A No.

Q Would you agree the need for that oxygen is due to the effects of the vasculitis on her lungs?

A I think so. Yes.

Q Is it likely she will always require

the daily supplemental oxygen?

A I don't really know that to be the case. Really, it's going to depend on, in a way, what course the disease takes. And if it's put in remission and stays in remission, I can certainly conceive that they could wean her off the oxygen.

You know, frankly, for some of these things, the situation is on an average good day she's off the oxygen, but she catches a cold and her lungs are congested, and she's back on oxygen during times of illness or stress, and things like that.

Q Are you describing a scenario that might happen?

A Yes.

Q You're not suggesting that you know, to a reasonable degree of medical probability, that that's going to happen?

A No. I think I could certainly envision a day where she's off of oxygen, knowing there will be days when she has an illness or cold when she might have to go back on it, but right now she's on it.

Q Well, I'm here, as you know, to try and hear now what opinions you may express at trial. Is it fair to say that you don't hold yourself out as an expert in the treatment of vasculitis, and predicting its course to the point where you intend to come to court and say, I believe to a reasonable degree of medical probability, that she will have the following course at age five or six or seven?

A I will not be doing that.

Q As it relates to vasculitis?

A As it relates to vasculitis.

Q You've told us several times that this disease can wax and wane. Is it fair to say also that you're not going to come to court and say, I know it's not going to get worse?

A I don't think anyone can sit there and say that they know for a fact that it's not going to get worse.

Q How about to a reasonable degree of medical probability, is it also fair you're not going to come to court and say, I know to a reasonable degree of medical probability that it's unlikely to get worse?

A I'm not going to be saying that.
Q What is the path of physiology by which the vasculitis has injured her lungs to the point that she requires this supplemental oxygen?
A I'm not sure I can give you -- again, that's a little out of my field. Just to remind you, though, what the lung does, it's really a big sponge with blood vessels in it. So this is the space where oxygen meets the blood to exchange the carbon monoxide and the oxygen.

So blood supply is crucial to the lung. That's the whole reason to be there is to be a surface membrane to exchange gases. So here we have the vessels that inflamed. And the vessels are not able to regulate themselves and flow properly and so forth. So it's a critical finding for that tissue.
Q I note your report says that fortunately, she's not having any unusual or opportunistic infections. Now, you stated that after talking about the vasculitis and that she's on steroids.

Is she at a more compromised position in terms of fighting off infections by virtue of both having the vasculitis and being on steroids?

A Mostly, in my opinion, the steroids. To a secondary extent, any person that has a lung disease, it's like the old smoker with emphysema. If he happens to get a plain old chest cold, it may be harder on them than if they were otherwise healthy.

So from a general point of view, a chest cold in her would be harder on her than a chest cold in an otherwise healthy youngster. But most of it has to do with the prednisone itself.

Q And what is it about the steroids and prednisone in particular that make it harder for her to fight off infection and a cold?

A The idea of prednisone is that it's suppressing the immune system. I mean, vasculitis in a very general sense is an immune attack on the body. It's like an arthritis. Your immune cells are attacking the lining of your joints. If you have a skin rash from

lupus, the immune system is attacking the tissues of your skin.

The steroids suppress the immune system to alleviate that attack. But in so doing, that same immune system is suppressed and fighting unwanted things like infections.

Q If she gets an infection, is discontinuing steroids an option? And if so, what happens to the vasculitis?

A Generally, they would not be discontinued. They might be lowered. But the key here would be more along the lines of early intervention with antibiotics, things like that. So another child, they might have a cold for a week before the mother bats an eyelash. This mother is going to be in the doctor's office looking for a penicillin prescription on the first day of an illness.

Q Have you spoken to any of the physicians at Johns Hopkins who are involved in her care?

A No.

Q Have you been provided with any information concerning their opinions, other

than what's revealed in the medical records from Johns Hopkins?

A No.

Q Have you been advised that any of them will testify in this case?

A The only thing I have is the business about Mike Johnson who is at the Kennedy Kreeger Center.

Q So in my mind the question was more, has Mr. Mitchell or anybody for the Fogels, representing the Fogels, indicated that they expect to call any physicians from Johns Hopkins to testify?

A I know nothing about that.

Q Are there any opinions about Sarah's vasculitis that you expect to render at the trial in this case that we haven't talked about?

A I don't think so. I mean, my opinions have been basically the causation issue. Secondly, that the vasculitis is not connected to this. It's just, you know, unrelated, if you would, bad luck, another bad thing happened to this child. And we talked

about life expectancy in the first deposition.

MR. MITCHELL: Tom, are you going to move on to another topic?

MR. MONAHAN: After, probably, one more question.

MR. MITCHELL: Let me know then whether you want me to ask more questions about vasculitis or do it at the end. I have one or two questions on it, no matter how you do it.

MR. MONAHAN: Let me ask this question and I'll let you jump in.

BY MR. MONAHAN:

Q Does the fact that she has contracted this vasculitis in all likelihood shorten her life expectancy beyond the opinions that you expressed in the deposition of May 1997, when you were unaware of that diagnosis?

A No. If I've understood your question, my original opinion was that, pretty much based on her neurology, the degree of her cerebral palsy, the fact that she had the

feeding tube and those things, that realistically I thought a ten-year life span would be a reasonable estimate for her.

Because of the fact that she has really done well in the steroids and that her needs seem to be met, I didn't see where that was going to be shortened any further because of the vasculitis.

Q That's all I have on vasculitis. If you want to ask a few questions. Go ahead.

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BY MR. MITCHELL:

Q If it's okay, yes. As I understand what you just told Mr. Monahan, you don't know of any causal connection between a traumatic neurological injury, which you opined about before at birth, and the inception of her vasculitis; is that right?

A Correct.

Q Did the neurological injury at birth, however, affect her respiratory centers so as to result in hypercapnia, excessive CO2, and difficulty breathing, hypotension and so forth?

MR. MONAHAN: At what time?

BY MR. MITCHELL:

Q Subsequent to the initial injury and intermittent thereafter.

A At the time of birth, in the days following birth, yes.

Q If she was born with a vasculitis potential, would these events, say the hypotension and the hypercapnia and so forth, tend to aggravate the vasculitis?

A I don't think so.

Q Now, as I understand it, the vasculitis would be affected, am I right, by subsequent periods of difficulty breathing?

MR. MONAHAN: I object to the form.

THE WITNESS: I'm not sure of the question he's asking.

BY MR. MITCHELL:

Q Well, if someone has vasculitis, is the vasculitis aggravated by any neurologically based or respiratory difficulties?

MR. CHASON: Objection.

MR. MONAHAN: You're talking about pulmonary vasculitis?

MR. MITCHELL: Yes.

MR. MONAHAN: I object to the form.

MR. CHASON: Objection.

THE WITNESS: If I have a patient right in front of me who, at that moment, might develop vasculitis three years down the road, but actually has it, then if something else changes, carbon dioxide and oxygen, then that will affect blood flow to the lungs because it affects all of us. Those are physiological changes.

So for any of us, if I dialed up the carbon dioxide concentration in the room, it will have an effect on your pulmonary vessels, but that has to be as the vasculitis is actually in there. Not if it's, sort of, destined to happen two years from now. I wouldn't make any connection between them.

BY MR. MITCHELL:

Q And you don't have an opinion when the vasculitis actually began?

MR. MONAHAN: I object to the form.

MR. CHASON: Objection.

THE WITNESS: Well, I think the symptoms of it appeared in early 1996 with the fevers and the coughing. Now, how much earlier before there would there have been changes that you could see with a microscope that didn't affect the health of the child, I don't think anyone knows that.

MR. MITCHELL: Okay fine. That's it, Tom. Thanks.

MR. MONAHAN: Jim?

MR. CHASON: I'll just wait until you're finished, though I may have some questions on vasculitis.

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BY MR. MONAHAN:

Q I'm going to ask you a couple things more on vasculitis, or at least one. Have you

done any literature search specifically relating to the vasculitis in connection with this case?

A No.

Q Are you aware, one way or the other, whether there's literature that describes a causal connection between birth trauma and systemic vasculitis?

A Well, let me -- I hope this answers your question. It was my impression from reading the Hopkins records that, first of all, they were fairly shocked with the diagnosis. They know pulmonary vasculitis, but who has ever seen it in such a young child.

And that, really, few people could sit there and say, Well, my vast experience with pulmonary vasculitis and the children of this age, you know, X, Y and Z. I asked myself the question, might there be a connection between it. And I didn't go to the literature for this, but it would be beyond my personal belief to think that there would be.

I think of them as so separate and so distinct in their character, that it would

be -- I mean, if someone told me, I have a medical paper that says if you have birth trauma, you're going to get vasculitis, I probably wouldn't believe it.

Q You've specialized in pediatric neurology for how many years?

A Twenty.

Q And I think you told me you've never seen a child, an infant, with pulmonary vasculitis like this, correct?

A Correct.

Q Yet, I imagine you've seen an awful lot of children with problems that were attributed to birth trauma?

A Yes, I have.

Q Can you quantify, at all, how many such children you've seen in your twenty years?

A Hard to say. I think, actually, in my original deposition, I had prepared a review of birth trauma and how it expresses itself in newborns. I think we had thirty cases at that time, and that was for a fairly finite time interval. And I don't remember what it is now, but if you allow me for five years, five years

at CHOP, you looked at all the cases we considered to be traumatic and there were thirty of them.

Probably every couple of months there's admitted, to this hospital, a child that is traumatized to the point where they had seizures or they had a stroke or something disastrous had happened. So over twenty years, I would imagine seeing 120 such cases.

Q Four times thirty?

A Yes.

Q Did that paper ever get published?

A No.

Q Have you called Dr. Axel and asked what's going on?

A Well, actually, parenthetically, it turns out that his wife had cancer and died about three or four months ago. And he has got four little girls, and he was doing his job and taking care of his wife and his girls, so he was in no shape to do anything right now.

So I actually was calling him to find out where the heck is the manuscript, and it was the day his wife had died, my usual

excellent timing.

Q How horrible.

A So it's no further along than two years ago.

Q Let me ask the same question about -- are you able to quantify a number of children you've seen with sequela of brain injury from birth asphyxia or hypoxia or asphyxia presumed to have occurred in the delivery process?

A I'll have to, in a sense, make an estimate on this because I don't keep particular records, but probably two or three asphyxiated kids for every physically traumatized kid. Far and away, asphyxia, however it came to pass, is much more common than physically traumatized brains.

Q And obviously in none of those have you seen pulmonary vasculitis like this?

A Correct.

Q All right. Let's move on. When you did your evaluation in June of this year, did you make any notes?

A Let me look. These were from my first examination, and you have copies of

these. These are notes from the medical records. I don't remember seeing any notes when I went through this this morning, but as I mentioned to you, I do have things at home. I don't really think I retained any notes on this exam.

Q Would you be willing to check?

MR. MONAHAN: And Gerry, would you agree to let us know if there are and provide them if there are any notes?

MR. MITCHELL: Yes.

THE WITNESS: I don't think I have any. Here they are.

BY MR. MONAHAN:

Q Can I just compare what you assumed I had with what I do have, to make sure that I'm as far as along as I should be.

A Yes.

Q Thank you. Can we mark then as Exhibit-2 the three pages of handwritten notes you gave me, which appear to be dated June 3rd, 1998. Is that the actual date of the exam?

A If you bear with me, I can look in my

calendar because that's where I actually keep all this stuff, if it's important to you.

Q It's not that important.

A It probably is. It looks like the report is dated differently.

Q The report is dated June 12th.

A June 12th. So that's probably when it was typed or dictated.

(At this time, a document was marked for identification as Exhibit-2.)

BY MR. MITCHELL:

Q You have some other notes there, Doctor. What are they?

A Yes. This was from the review of the medical records, and I believe these were copied last time also.

MR. MITCHELL: Apparently, there weren't any exhibits marked in the last deposition.

BY MR. MONAHAN:

Q Correct. And there was another set of handwritten notes. What were they?

A Okay. One is the medical records.

That was one. Two is the notes from the physical examination. And then the third set is what you just marked.

Q Since May 30, 1997, have you consulted with any other physicians concerning this case?

A No.

Q In your June 12 report, you noted that Sarah is cushingoid. What does that mean?

A Cushingoid refers to the appearance of a person who is taking steroids. And basically, it's the fat pads on the cheek. The steroids amplify fat cells, and so babies have fat cheeks. And so hers are unusually fat and their faces become more rounded. So the cushingoid appearance is that, sort of the Campbells' soup face, the big, round, puffy cheeks.

Q I gather that's a consequence of her taking the steroids required by the vasculitis?

A Yes.

Q Your report also says that you may further characterize her seizure disorder based upon results of a recent ambulatory EEG

monitoring. Have you done that?
A I've never received that.
Q Do you know why not?
A Not really. The issue was that she has these events. And actually, what the mother said is that the doctor calls them seizure-like episodes. And they are confused about whether this is really an epileptic seizure or if it's some other kind of abnormal brain attack, because they haven't really responded to the seizure medications.
And one way of deciding is to let the EEG run all day and all night and, you know, read the EEG when the event's actually happening, rather than try to read between the lines which is what's usually done. So that was from Anne Burgen, I believe, at Hopkins.
Q At the time of your deposition of May 30, you told Mr. Farley -- I'm reading from page seventeen -- that this encephalopathy, which means injury to the brain, correct?
A Right.
Q Is not a progressive condition. It's a fixed injury to the brain. Is that still

your opinion?
A Yes.
Q Is there anything in the records from Johns Hopkins which has suggested to you that there has been progression of her brain injury?
A No.
Q If a patient has a birth-related injury from trauma, in your experience, that would lead to a fixed injury of the brain, would it not?
A Yes.
Q And in that sense, what that means is that within a week or several weeks or several months, you would see the injury that's occurred to the brain. And from that point on, you would expect it to be static; is that right?
A The injury is static, but the manifestation of the injury can change over time. So, let me give you a very simple example of this, and let's put it out of the context of this case so we don't get confused.
Let's imagine that it's just a birth asphyxia case, and the brain has been injured.

And in the first day of life, the baby is totally limp, low tone, floppy. So here's a brain injury and what's done is done, and it's set in stone, and whatever, it's not going to change.

The way it looks in the first couple of days of life is that it's low tone, hypotonic. As time passes, the natural progression of the, again, clinical look is actually they may go through a phase where the tone looks pretty good because they're ultimately destined to have high tone, so you start off with low tone.

The tone is increasing, and the high tone and low tone balance to the point where they go through a phase where everything looks pretty good. And then later, the child shows a little bit of high tone, and then more high tone. And by the time they're two years of age, they're stiff as a pretzel.

And then with a lot of physical therapy, the therapists are actually able to get them to take a few steps, but they're still very stiff. And by the time they're a teenager

and they've heavier and not as motivated, they're back in a wheelchair.

So here's one injury that has happened that has produced one damage scene to the brain. But the way it looks as a newborn, and six months, and at two years, and at twelve years may evolve sort of in that changing, the brain is still changing. I mean, the injury is not changing, but the child's brain is changing because they're supposed to change. It's a developing child. It's an evolving nervous system.

So the injury is fixed, but the manifestation of the disease may change. So seizures, for example, newborn seizures have one look. Later on as the child matures there's another kind of seizure, like an infantile spasm, which has happened in Sarah.

And then with time, another kind of seizure may show up, and it's not that the disease is changing. It's the way it's manifesting itself in that particular age and brain maturity.

Q So you're talking about

manifestations in terms of the clinical manifestations when one does a physical examination on the child?

A Yes. The clinical consequences of the injury. And again, the big picture is trying to distinguish conditions that are truly progressive like a tumor is progressive, or hydrencephalus gets worse with time, the water builds up more and more, and the child's brain is actually getting worse and worse.

So in the day before CAT scanning, for example, where if you wanted to really look at the brain, it was going to be pretty nasty. you would have to do an angiogram or something that would be painful and dangerous and invasive.

Now, we do scans left and right, but in the old days, the big decision was, Well, was this just something that happened. It's done, it's done, or is this is a tumor, hydrencephalus, metabolic disease, things that fundamentally change over time.

Q Now that we are in the era of CAT scans and MRIs, it would be unexpected, would

it not, to see progressive changes in the brain in a patient, if the only cause of injury to the brain is a birth-related trauma?

A Well, I guess it depends on the character of the changes. In other words, if it was wholly new changes and had nothing to do with that, the answer would be yes. Even in the case of, again, a simple case like birth asphyxia where there's no medical/legal issues.

The baby is born at home, whatever happens happens, you do the scan the first day it has a certain look. Looks different a week later. Looks different a year later because again, the radiological expression of that damage does change over time.

So the question isn't whether there is any change. There's always some change, but whether they are consistent with the prime mover, the injury, the hemorrhage, the asphyxia, whatever.

Q Do you consider yourself an expert in interpreting MRIs of the brain?

A Well, I'm certainly experienced at it in the sense that it's part of the things that

I do. I obviously am not a neuroradiologist. I routinely confer with the neuroradiologist here to look at outside films or the films that are done here. So it's a little bit yes and a little bit no of an answer.

Q In your practice, do you ever order MRIs and CAT scans, and treat patients on the basis of your interpretation of those studies, without the benefit of a consult with a neuroradiologist?

A No.

Q Do you ever author reports?

A No.

Q Interpret MRI's or CAT scans of the brain?

A No.

Q Do you have any training in interpretation of fetal monitor traces?

A Rudimentary. Again, the usual. You have some exposure during medical school. I'm aware of the issues with fetal heart rate monitoring. I can look at them and know that the fetal heart rate is 150. I can recognize variability, but I'm not the guy standing in

the labor room calling it in real time. It's always retrospectively. It's always after OBs have looked at them and made their statements about them.

Q Again, I'm not trying to insult you. This just speaks to my ignorance, but were fetal monitor traces used during your medical school years?

A Yes. I was in medical school in 1975. They were there. I must look particularly old today, I guess.

Q No. For some reason I was thinking that they weren't commonplace in 1975?

A They weren't commonplace, but I trained at Hopkins, so they had a high risk population.

Q Since you have specialized in neurology, have you pursued any further training and interpretation in field monitor traces?

A You know, the occasional lecture on fetal heart monitoring at a Grand Rollins, but that's hardly training. That's just attending a lecture.

Q How occasional is that? That's probably less than annually.
A Yes. Less than annually. A handful, five times maybe, national seminars, that kind of thing.
Q Five times in your career as a neurologist?
A In my professional life.
Q Have you ever had any training in attempting to reconstruct what a fetal monitor tracing would show after it was turned off, either based upon what came before or what was advent later in terms of the clinical picture of the job?
A No.
Q Are you aware of any literature that talks about how one can do that?
A No.
Q Have you ever heard any lecture on that subject?
A No.
Q Do you intend to offer opinions in this case about what the fetal monitor tracing likely would have showed if it had been left on

for the additional time between when it was shut off, and when this child was delivered?

A No.

Q Have you ever been asked to express any opinions or speculate on that subject?

A In this particular case?

Q In this case, what the fetal monitor strip would have shown between the time it was shut off and when the child was born?

A No.

Q Do you intend to offer any opinions in this case that the fetal monitor strip that actually exists, demonstrates evidence of neurologic injury to the child from a vacuum extraction? Does that question make sense to you?

Essentially, can you look at any of these panels and say, From this tracing, I can tell that there's neurologic insult going on to this child as a result of the vacuum extraction?

A I wouldn't know how to do that.

Q Have you been provided with any additional laboratory work, laboratory results

since your initial deposition, that in any way support in your opinion your conclusions that Sarah's neurologic problems are due to birth trauma?

A By laboratory, you mean, like, blood tests?

Q Correct, electrolight studies, anything of that nature?

A No, but in the same breath, you would not look to that answer from blood tests either.

Q How about any genetic testing? Have you been provided with any results of genetic testing since your initial deposition, which has either changed or added additional support to your conclusions?

A It's hard for me to remember now what I remember before and after that. What I remember is that there were some concerns for dysmorphism. In fact, they went to the geneticist at Hopkins, and the concerns were that her fingers were flat and that her skull didn't look right.

And not that you sweep those under

the rug, but aside from that, there's really no other genetic issues going on with her that they considered, like, a classic genetic syndrome, if you would. Really didn't come up with anything that could explain anything really.

The business about the head to some extent is mechanical in the sense that they were concerned that the sutures were overriding and her head was small. So if you look at any person with a small head, that's abnormal morphology. It's dysmorphic. The question is is it genetically driven dysmorphic.

So, I don't have any problem with people saying, Oh, your head is small, that's dysmorphic. Of course it is. It's abnormally formed. But in her particular case, it's because the brain's been damaged, and the whole head is small, and it's not small like in Down syndrome where all the Down syndrome children also have genetically determined small head.

Q Did you assess whether or not she has an arched or high palate?

A I examined her. I was not struck

that her palate was high-arched.
Q Is that something that's seen with genetic abnormalities?
A Yes, but also children with swallowing problems. I mean, just as a secondary effect of not having normal, you know, mouth action, swallowing, tongue movements, things like that.
Q If one is born with that, would that be too early a time for a swelling disorder to manifest itself in a high or arched palate?
A It could be. In other words, babies actually practice swallowing and breathing from the eleventh week.
Q So if one is born with that and it's due to a swallowing problem, it would lead one to conclude that the swallowing problem had existed early on in utero, wouldn't have just occurred at the time of delivery?
A Correct.
Q Is the current state of genetics research such that, in all likelihood, there are many genetic abnormalities for which we don't yet have the medical capability of making

the diagnosis?

A Yes.

Q Would you agree that the current state of genetic science is such that in all likelihood, the majority of genetic abnormalities are those for which we do not yet have the current medical knowledge to identify the specific cause?

MR. MITCHELL: I'm going to object to that. There's no foundation for that and I think it calls for speculation. How can you estimate a percentage about something you don't know anything about?

BY MR. MONAHAN:

Q Let me make it clear. If you take a whole population of kids who have dysmorphic features and birth anomalies for which there is no other explanation beside a genetic cause that's currently known, would you agree that the majority of those children, the current state of genetic testing would likely not identify a cause in the majority of them?

A Well, majority means I know there's

more than 51 percent. I don't really know what the number is, but part of the issue though is when you say dysmorphic, what exactly you mean. So, for example, if I brought our geneticist in and examined you, okay --

Q You wouldn't have enough time.

A Again, the issues is that if you really start looking they would say, well, you didn't know it, but you've got an extra rib on that side, and this one ear is pinned down and the other one is free. They're picky, picky, picky people.

So they find little things and they are dysmorphic, and the question is sometimes where do you draw the line between just variability versus -- because we all know when you see what looks like a Down syndrome, we all know that's bad. It confers bad prognosis. Whatever happens in Down syndrome we know about that.

So most people have variations, anomalies, flat fingers, this, that and the other. And, yeah, they're dysmorphic, and of course they're genetic. Somehow your genes

told your fingers to be flat or whatever, but the question is is there really a disease behind that, or is that part of -- we're different people. No one's perfect. We all have little flaws, things like that.

So, when you have somebody, again, brain-wise who is so impaired as Sarah, and you're looking for, you know, a genetic explanation for brain disease, then it's got to be more than just, Oh, your fingers are a little flat. There are usually very unusual looking children and/or when you do the brain scan, they say, Well, holy cow, there's a whole part missing here, or you can really see the dysmorphic character of the brain.

So I don't see how you can take Sarah and say, Well, here she is so impaired, so severely and neurologically abnormal, and then all we've got dysmorphic is that her fingers are flat. You know, yeah, they are flat, but that's not why she's this way and the brain scan doesn't show that.

So I don't have any trouble with people raising that as a possibility. But, you

know, in the whole scheme of life, I would be completely unsatisfied to say that, Well, yeah, this is just another genetic syndrome. It would not satisfy me as a believable explanation.

Q Out of curiosity, was Down syndrome named and known well before anybody was able to identify the precise chromosomal explanation for it?

A Yeah. Hippocrates, I mean, you know -- and of course, it was the easiest one to identify because when you finally looked at the chromosomes, there's actually an entire extra chromosome. There's an extra chromosome 21. The things they look for now is this little running base is on the wrong position. They're in the microscopic level with most of this stuff.

Q But that's an example where one can identify the child with a genetic defect, and for centuries that was possible before medical science caught up with the ability on a microscopic level to pinpoint the exact cause.

A That's exactly right. But then

again, Down syndrome is overtly dysmorphic, and they stand head and shoulders above the crowd or head and shoulders below the crowd, in their particular --

MR. MONAHAN: Off the record.

(At this time, a discussion was held off the record.)

BY MR. MONAHAN:

Q Based upon your review of additional records from anywhere, but in particular from Hopkins, do you intend to offer any opinions that any CT, MRI or other imaging of Sarah's brain, since the period immediately after birth has formed the basis for your conclusion that her injuries are due to birth trauma?

A In other words, things, like, well down the road?

Q I think we talked in May of 1997 about what you believe is indicated by the CTs and MRIs done at Anne Arundel Medical Center in her first ten days or so of life. Do you intend to offer any opinions about any subsequent CT, MRI, or other brain imaging?

A No.
Q I gather you haven't been advised of any opinions that Dr. Goldberg may have about CTs or MRIs from 1998?
A Correct.
Q And you, personally, haven't examined those?
A I've received the report faxed and that was it.
Q But that report, as I understand it, gave you information relating to whether she has a cervical spine injury?
A Correct.
Q But you're not relying upon it for any support for your opinions regarding the cause of her brain damage?
A That's correct.
Q In your experience, when children are born with CT or MRI evidence of bleeding in the subdural space, but not in any deeper aspect within the skull, would you agree that it would be unlikely that such a child would have brain damage?
A No. I wouldn't agree with that. I

mean, first, they don't have to have brain damage. There are children that do have subdurals, and it's not obliged for them to go on to have handicap, PCP, and so forth.

The subdurals to me are a sign that, well, trauma is what's behind this as opposed to, like, with your typical case of birth asphyxia or meningitis. You're not going to see a subdural hemorrhage. There's no reason for it to be there.

Part of it also has to come down with what you can't see with a CAT scan. So, for example, if you also see bruising or swelling or bleeding in the substance, sure.

Q In the substance, meaning what?

A In the substance of the brain, in the actual tissues of the brain as opposed to the space called the subdural space.

Q And we can agree the subdural space is outside of the brain tissue?

A Correct. It is not brain tissue, per se. And it is for that reason then, that you ask questions of a functional nature there. You say, Well, okay I can see with my eyes on

the scan there is some bleeding there.

Well, how is that brain behaving today? And you look at the child and the child's having seizures and is comatose. Well, the brain's not working real well today. And then you look at an electrical study, the EEG, and they say, Oh, it's a very low voltage or very weak signal. That brain's not working well today.

So really, to get to the whole story, it's never just a radiologist looking at a scan, but it's looking at the function of the child, the clinical picture and the imaging.

Q Is the Faulk cerebri also outside the brain?

A It's not brain substance. It's inside the head obviously, but it's not brain tissue. You don't think with the Faulk cerebri.

Q How about the transverse sinus? Is that also outside the brain tissue per se?

A Yes.

Q In your first report of April 30, 1996, you made mention of Sarah's abnormal

liver function tests after birth, that a liver ultrasound had showed a calcified portal vein that was felt to be secondary to portal vein thrombosis.

Do you recall which records you're referring to? Is this Hopkins?

A Hopkins, I believe.

Q Your report goes on to say, The most likely candidate for this was the prolonged presence of her umbilical artery catheter. It's not clear to me whether you are attempting to record someone else's opinion, or was this your opinion in April of 1996?

A I think I was parroting the medical records as much as anything there. You know, the whole issue is that in a newborn baby, the biggest blood vessel that is available goes through the umbilicus and up the aorta -- not the aorta, the vena cava. And this is where the portal vein connects. So if there's a blood clot there, it just propagates into the liver, basically. That's what I recall in the best sense of what was going on.

Q Do you agree with that opinion that

the most likely candidate for the calcified portal vein was the prolonged presence of her umbilical artery catheter?

A I think it's probably the biggest single factor. And of course, the question is what's the whole condition of the child. So for example, if she was shocky, low blood pressure, things like that, when the blood pressure is low, blood doesn't flow as briskly. So it was more stagnant and more prone to clotting.

So, yes. There is a catheter in there, and any kind of foreign body will trigger a blood clot. But at the same time, things like slow, sluggish circulation is another factor, and it's hard for me now to know what's going on with that. But I think probably her medical condition, meaning her shockiness and the catheter together probably were the two main players in that.

Q At what point in time are you talking about?

A Right after birth. I mean, the catheter was in place right after birth, so

they could do the blood sampling and so forth. And she was critically ill, obviously, right after birth, so those were the times when the blood clot could form.

Q Do you intend to offer any other opinions to a reasonable degree of medical probability as to an explanation for the abnormal liver function studies or the findings on the ultrasound?

A No.

Q I'm not sure I understand the next statement you made where you say, quote, However, she underwent a coagulation workup which showed marginal decreases in both protein S and protein C, closed quote.

What is the significance of those proteins?

A Okay. Blood clotting is a balance of two forces. It's like a seesaw. And on the one hand, there are chemicals that create the blood clot. And on the other side, there are chemicals that dissolve the blood.

So as we sit here now, my blood is forming little clots here and there, but the

body also dissolves them. So it's a seesaw balance, sort of thing. You've probably heard of a condition called hemophilia, and these are the folks that they cut themselves shaving, and they can't stop bleeding because they lack that chemical necessary for bleeding.

Q Factor something?

A Factor 8 deficiency, von Willebrand's disease, there's a whole laundry list of conditions that lead to bleeders, hemophiliacs. And on the other side of the world from them are the people who are overly prone to blood clot. Their problem is just the opposite. They are too likely to form clots as opposed to too likely to bleed.

In the chemistry of this, there are these two substances that are known. Actually, there's a bunch of them, protein S and protein C. If you're deficient in these chemicals, then the result is that you're prone to blood clotting.

Now, the trick in this is the following. If you've already formed the blood clot, like, in this child. She's clotted the

blood in her portal vein, the clotted blood consumes protein S and protein C. It's stuck in that blood clot.

So then when you do a blood test and take a sample from the arm, they say, How much protein S and protein C do you have? Well, it's usually a little low because it's all stuck in the portal vein.

So at the time, they said, She's got calcium in the portal vein. Might this be one of these coagulation defects, knowing that nothing else has ever clotted on her. So they measured the protein S and protein C, and it was a little low. But then again, it's sitting there in the portal vein.

So that was basically ascribed to the fact that she had the clot, but was not the cause of the clot. Did you buy that?

Let me rephrase it this way. The question is which comes first? Is this one of these genetically determined, Oh, my God, like, hemophiliac, I'm prone to bleeding because I'm lacking this. Or is it, I'm genetically lacking in protein S and protein C, and

therefore, I'm prone to bleeding.

If you're genetically programmed for this, you tend to have the kidneys, the liver, other things clot. So when they measured it, it was marginal. And it was attributed to the fact that those things were removed from her bloodstream and deposited into blood clot. So they never came out and said this is a genetically driven protein S or protein C deficiency.

Q Your initial report of April 30th, 1996 makes mention of a variety of metabolic testing performed, with the knowledge of the presence of metabolic acidosis. Are you referring again to Hopkins?

A Yes.

Q And your report goes on to say, This was certainly not conclusive, however. Do you intend to offer opinions to a reasonable degree of medical probability as to the cause of Sarah's metabolic acidosis?

A Okay. So you're looking for clarification of that part, basically.

Q Right.

A Okay. This is the way the numbers worked out in this. She was metabolically acidotic. And that's one of these things where you do the test, and the numbers come out a certain way. You have metabolic acidosis.

So there was no getting around that. She had it. The question is why is she acidotic. It was not a respiratory problem because that's easy to check too. If it's a respiratory cause for acidosis, then you should have too much carbon monoxide in your bloodstream. The CO2 is fine.

If anything, they had overventilated her deliberately, and the CO2 level was actually low. So the acidosis was not from the carbon monoxide.

The next question is, well, maybe there's one of these chemical disorders. You know, there's, again, dozens of these different, you know, biochemical disturbances that can show up in newborn babies, propionic acidemia, methylmalonic acidemia, all these kind of biochemical things. Because as part of those, you also have metabolic acidosis.

And when they did the testing, they came up with nothing conclusive on that. And, in fact, I think probably what was the basis of the metabolic acidosis was the portal vein thrombosis, because the liver is your big chemical warehouse in your body, and it's what's managing all this acid based business.

So the main vein for the liver is clotted and she has metabolic acidosis. And as that resolved, the metabolic acidosis resolved. So I really think the metabolic acidosis was a result of the clotting of the vein in the liver.

And when they did the testing for the specific biochemical disorders, whatever they were, they didn't come up with anything definitive on that.

Q Now, I gather the body at some point broke down the clot in the portal vein?

A I mean, we'd have to gather that. I don't know that it was specifically imaged to show that it happened. It essentially always happens.

Q If that didn't happen, wouldn't she

have gone on to some liver failure?
A Exactly. We have to assume that that happened.
Q If the metabolic acidosis is a consequence of that clot, would it follow that once the body dissolves that clot in the vein, it becomes patent and the metabolic acidosis would resolve?
A That's what happens. yes.
Q Are there any other opinions you have reached from your review of all the additional records from Hopkins, that you've been provided since your original deposition, as well as from seeing Sarah Fogel that we haven't discussed?
A I guess the only thought I had, I don't know if it's an opinion. It's just an observation about it, is that, you know, before this business about the vasculitis was known, obviously they were focused on reflux and aspiration because those are the things that can happen to children that so damage neurologically.
You know, so people say, Well, everyone knows that when you're brain damaged

you have trouble swallowing. You can aspirate and that's one of the things, they die early, things like that. So all the early hospitalizations were focused on that, she is aspirating, she is refluxing. This is going to do her in, basically.

And it really wasn't until time had passed that they realized that the real culprit was the vasculitis. Again, bad luck for Sarah. Not related etiologically to this. But I also felt though, that now that the vasculitis is under control with the steroids, she really hasn't had these crises.

So, in other words, she really isn't aspirating that bad or having so much bad reflux that I'm as worried as I was initially from that point of view. I have something else to worry about. I've got the vasculitis to worry about for her sake.

But at least her track record with respect to aspiration, which is probably the most dangerous thing for a brain damaged baby, is less. And I can't quantify how less, but it, again, just thinking about what her

circumstances are now.

Q I think I've asked you this already. On balance, do your conclusions about her life expectancy essentially stay the same as what you've told me previously in May of 1997?

A Yes, I do.

Q Have you testified recently on any medical malpractice cases?

A Yes.

Q When did you last do that?

A Let me think of what this was. It was just about two weeks ago in Wilmington, Delaware, and it was Eric Miller versus, I guess it's Kanaga, K-A-N-A-G-A.

Q Is that an individual or hospital?

A That was Dr. Kanaga, and I testified on behalf of the plaintiff for causation. And it was a baby who had 34 minutes of no heart beat and is severely brain damaged right now.

Q Why did they need you?

A They didn't. No. There was nothing difficult about it.

Q Who were the attorneys you were involved with?

A Brant and Dalton.
Q Is that the plaintiff's attorney?
A Plaintiff's attorneys.
Q Who took your deposition?
A Her name, believe it or not, is Nasczi, N-A-S-C-Z-I, something like that. It's probably the longest deposition I've ever been involved in my life.
Q Do you know her first name, just in case there's another Nasczi in Delaware?
A I can't recall. I never got past the Nasczi part.
Q Have you testified at all recently for any defendant medical malpractice cases?
A Yes. It's a case called Tipjew, T-I-P-J-E-W, versus Miller, and I just can't think of the firm.
Q Where is that case?
A New Jersey.
Q Post and Schell, by any chance?
A No.
Q You don't recall the attorney?
A That's what I'm trying to think.
Q What was the nature of the --

A That actually was a chorioamnionitis case. And then the last one was for the defense in a Virginia case. It was an arbitration panel thing, and the attorney's name was Frank Hilton.

Q I know Frank. He's out in Harrisonburg?

A Harrisonburg, Virginia.

Q Nice guy.

A And I cannot recall the name.

Q Did you ever work with Ron Hodges?

A I don't believe so.

Q What was the nature of that allegation in Mr. Hilton's case?

A This was an ancient case. The patient is now 25 years old, and the allegations were birth asphyxia. And it actually turned out that the youngster, the neonate, had a blood disorder called DIC, disseminating intravascular coagulation, and he had a stroke.

So his CAT scan had a stroke and he was bleeding. And they did the blood studies, and this and that. And the claim was that it

was just another case of birth asphyxia, so it went to the arbitration panel.

Q What was the outcome?

A For the plaintiff.

Q Who was being sued?

A The doctor, mostly, but the hospital because the chain of command business about the nurse didn't recognize. Oh, they only had one fetal monitor at that time. It was, like, a 1975 case, something like that, and there was one used on another patient, and the hospital should have had more machines to monitor this particular patient as well.

Q Was it UVA?

A No. The child was sent to UVA where the DIC was diagnosed.

Q Rockingham?

A Rockingham Memorial Hospital.

Q Can you think of any other cases which you've recently testified for defense?

A Not testifying, but a case for Fulbright and Jaworski, and it's a case in Texas. The child probably has a condition called pyruvate dehydrogenase deficiency, but

when it shows up in the newborn period, they have seizures and they're depressed and it mimics birth asphyxia. And I reviewed that and made a report on that. It has not gotten to the point of the deposition.

Q What do you charge for your time?

A $300 an hour.

Q When you saw Sarah, she was still being fed through a gastrostomy tube?

A Yes.

Q Do you have an opinion as to whether that's going to be required permanently?

A I expect it to be. Yes.

Q Is it still your opinion that it's unlikely she will ever be ambulatory?

A Yes, it is.

Q Is it still your opinion that it's unlikely she will ever be continent of bowel and bladder?

A Yes. I expect her to be incontinent of bowel and bladder.

Q Those are all the questions I have. Thank you very much.

MR. CHASON: Let's go off

the record.

(At this time, a discussion was held off the record.)

- - -

BY MR. CHASON:

Q Doctor, we've talked a fair amount about vasculitis, and I'm only going to have a few questions. What are the known causes of vasculitis?

A The known causes would be, number one, infection. So if a germ, if you would, irritated the blood vessel, that would inflame it.

Q That would be a limited condition, once the infection resolved, the vasculitis would resolve?

A Actually, not necessarily. For example, the CMV virus is a -- not CMV -- HIV virus causes -- the AIDS virus causes a vasculitis that as long as it's alive, then the cell stays in there.

Q Does CMV cause vasculitis?

A Not that I'm aware of. It causes brain damage, but not vasculitis. Second thing

would be in the broad area of allergic vasculitis so that your system is allergic to something. And there's an antigen that triggers the response.

And then the third general area would be what is called autoimmune. And this is where somehow the body is instructing the immune attack against its own tissues. It's not a foreign tissue. It's not a germ or the pollen in the air, but a normal substance on the cell.

And probably there are some tumors that can produce, probably through immune mechanisms again. And that's my sum total knowledge of what would cause it.

Q In Sarah Fogel's case, is this a vasculitis of unknown origin?

A Yes.

Q And I take it from something you said quite a while ago that there can be genetic causes. There can be genetic predisposition, if you will, to the occurrence of vasculitis?

A Yes. I think that's true. And just, for example, quickly, have you ever heard of

this thing called lupus?
Q Yes.
A So, in lupus, you know, why does it attack this person and not that person. There are actually some blood types that if you happen to be this specific type of blood type, you're much more likely to get lupus than someone else.
So it's like your gene system has set you up as a sitting duck, if you would. The trigger still has to show up.
Q Was this patient's amputation of toes secondary to her vasculitis?
A I think so. Yes.
Q Do I understand correctly your earlier testimony that vasculitis is a condition which would probably exist throughout the body, but may manifest itself in particular areas of the body?
A I believe so. Yes.
Q Do you believe that chorioamnionitis existed at the time of birth in this patient?
A I'm not sure I remember that part real well. The baby was described as being

foul smelling for sure, and that's supposed be one of the clues to look for that.

Q To look for chorioamnionitis?

A To look for chorioamnionitis, the infection that's making it smell bad. I don't believe there was ever fever, belly pain, white cells, a germ that was cultured that would have confirmed that.

Q Those are the kinds of things one would look for as signs and symptoms of the existence of an infection of the chorioamnion?

A So I suspect there could be this because there's foul-smelling there. Let's look into it. Is there fever? Is there belly pain? Do we culture a germ by sending the specimen to the lab, and the answer is no.

Q In mom or the baby?

A In the baby.

Q Would you look for those signs and symptoms in the mother?

A Yes.

Q And there's lab values?

A Yes.

Q And it's your testimony that those

were all normal?
A That's what I recall as I sit here now.
Q To what then do you attribute the foul smell which was described in which you've observed?
A I don't understand why it was foul smelling.
Q In a patient with vasculitis who is on steroids and responsive to steroids, does it occur that that patient can later fail to respond to steroids?
A It would have to -- I'd have to say yes, it's possible.
Q You've talked about your testimony from your prior deposition with regard to this patient's life expectancy. And as you probably don't know, I've gotten into this case late, so I'd like to ask you just a couple follow-up questions. When you talk about a ten-year life expectancy, is that from time of birth?
A No. It's, quote, from the present, meaning that the issue is that we'll just take a hypothetical patient. They are three years

old, and using these tables again from Grossman and whatever, you're saying from today, just like an insurance adjustor, well, from today, what are your chances of dying in the next five years, ten years, whatever.

So that statement of ten years would have been from the first time I saw her back in 1996. What's your life expectancy today, ten years from today. So that would be, when I offered my opinion of ten years, it was from the time I had seen her.

Q And by virtue of having lived until at least July 22, 1998. Does that, in your opinion, based on the studies that you referred to, mean that Sarah, as of today, continues to have a ten-year life expectancy?

A Well, that's a good question.

Q Do you have a good answer?

A I was actually wondering if someone was ever going to ask me that because, first of all, I'd have to look and see where she is on the table. I'm assuming you know the table we're referring to, you know. And so they say, you're severely handicapped, no mobility, tube

feed, all that kind of stuff. So as of today, where are you?

I believe she's still in that same group, and we can look, but she's still in that age group. So I'd have to say still that it would have been as of 1996.

If I saw her for the first time today and never saw her before and I pulled the table out, I would say from today, from July 22nd on. But I'm not going do that to you because my statement was I expected to see her live ten years from the time I first saw her next to the table. In fact, so far, she's done that.

Q So you hark back to the point which you expressed that opinion, and not revise that to be ten years from today?

A I would not do that.

Q I'm interested in your opinion with respect to the fact that this patient has what I think you've identified as an additional complicating factor. That is the vasculitis. As a result of the vasculitis, she's taking steroids which enhance the likelihood, as I understand your testimony, that she will

contract an infection?
A Correct.
Q Why is it that that does not, in your opinion, affect her life expectancy?
A You know, the first question is would vasculitis reduce life expectancy, and I would surely think it would. I have in mind that if there was no other factors involved, she just had vasculitis, she would probably live 30, 40 years with vasculitis. So, the vasculitis isn't what's going to affect her life expectancy now, which is far more, in my mind, determined by her cerebral palsy.
So what's way down the line for her, vasculitis causing her death, I don't see that as a player for much longer. So that I would still think that what would take her life sooner would be the complications of her cerebral palsy.
Q Don't both her pulmonary vasculitis and the chronic use of steroids predispose her to, among other things, lung infections and pneumonia?
A Yes.

Q And it predisposes her to a greater degree than a similar patient who didn't have vasculitis, and was using chronic steroids, correct?

A Well, again, it might on paper, but I guess the point is that to examine the child, I don't know if you've ever seen her, but she looks amazingly robust, number one. Secondly, they have all this medical care that they're getting with the nurses.

And it's certainly the mother's -- well, I can't speak for the mother's intention, but I know if this kid sniffles, she's going to be treated. So things that are, like, infections are treatable.

It's not like they're going to throw their hands up in despair. They've invested a lot in this child. So I don't really see the vasculitis as being life-threatening to her for a much longer period of time, than what is a very short period of time for her life. In other words, the next ten years or ten years from when I first saw her.

Q I understand what you've just told

me. It does not, I think, really address what my question was. Let me try to rearticulate it.

All other things being equal, isn't it true that her vasculitis and her required use of chronic steroids, predisposes Sarah Fogel or increases her risk rather, would be a better term, of pneumonia over that which she would have if she didn't have those conditions and treatment?

A I think you'd have to say yes to that.

Q Isn't it true that one of the big risks for patients who have the underlying neurologic condition that Sarah has, isn't one of the big risks pneumonia, one of the major reasons for their early death?

A Well, aspiration pneumonia as opposed to, you know, pneumococcal pneumonia, for example.

Q So she's at increased risk by virtue of her neurologic condition for aspiration pneumonia, and she's at a separate increased risk by virtue of her vasculitis and chronic

steroid use for bacteriological and viral based forms of pneumonia?

A Correct.

Q Let me tell you where I'm going. You're talking about imaging of the brain and the static nature of her injury, which you've opined about. Does there come a time in the development of the brain when, if injury to the brain is due to a hypoxic event at birth, you expect at some point in time that the imaging, the appearance on imaging of the brain stays the same from thence forward? Is there such a point in time?

A Probably, yes.

Q And at what ages?

A Maybe around age five.

Q What changes do you expect to be occurring if it is due to a hypoxic event at or around birth from ages two to five?

A Probably sort of the biggest biological factor that's changing is the white matter. The white matter is the insulation around the nerves. So, for example, a newborn infant has very little white matter. The eyes

have some, the feeding mechanism has some.

A lot of that comes into play between age two to five where you can see the changes in the white matter because it just wasn't there before. It wasn't visible on the scan before. And, of course, that assumes that there's no other medical factor going on.

So just to give you a little curve in this story because at one time, for example, Sarah took ACTH when she had these infantile spasms. And if I took ACTH and if you took ACTH, that actually produces visible changes in the appearance of the skin, and it's part of its character to do that.

She's taking steroids now. Steroids are like an oral form of the ACTH. It's a tricky situation because we have the lesion, the injury in a changing nervous system. And then to boot, there was a course of ACTH, there's the steroids.

And again, if you just go to medical literature and say, What does ACTH do to the scan, it will tell you there's changes. So it's not a super-simple situation for her.

Q If I understand you correctly, in the normal child, one would expect on brain imaging from between the ages of two and five to see the presence of increasing amounts of white matter?

A Right.

Q Does one expect to see that in a patient which has suffered an anoxic or hypoxic event at birth?

A Yes. In other words, the timetable is still embedded that between the biological ages of two and five, that's when the white matter is supposed to show up and do its change. It's still trying to do what it was programmed to do.

Q Other than the increase in white matter which you have indicated would be apparent on imaging, between the ages of two and five, are there other changes in the brain which one would expect to see on imaging? Again, between the ages of two and five in the patient with a birth hypoxia.

A You might notice the so-called atrophy a little more, especially if the

patient has been having seizures. Again, I've complicated your question because you're saying everything else is unchanged, just plain birth asphyxia, nothing else.

I think you may appreciate some atrophy some more, if there's been a lot of seizures, which in and of themselves will produce some atrophy that can amplify that and make it more conspicuous.

Q After the seizures, would one expect to see atrophy?

A You would still expect to see atrophy between age two and five.

Q All right. I would like to get from you, and I don't necessarily have to have it before you leave, but I would like to get your current CV, if you have it available?

A I can print it up in a minute.

Q Do you expect -- again shifting gears to the fetal monitor strip. Will you be expressing any opinions with respect to whether or not the fetal monitor strip, at any time, demonstrates signs of fetal distress?

A It was my understanding that there

was never any sign of fetal distress on it, and not so much through experts making statements, but just reading the medical records. I don't think anyone -- it was never part of my, you know -- it was never a key ingredient to my analysis in the case.

Q Do you consider yourself an expert in the interpretation of fetal monitor strips?

A No.

Q I'm going to try to articulate this question. Well, I doubt that I'll do it, but work with me, if you will.

Is it correct to say that in the universe of children with cerebral palsy, that some of them may be due to birth trauma, that another portion of them may be due to a determinable genetic cause, and that another portion of them will be due to an undeterminable genetic cause?

A Sure. I mean, that's sensible.

Q Okay. Do you hold the opinion that those three categories cover all of the universe of causes of CP?

A Well, the other category is

undetermined acquired causes. So as an example, in the case of trauma, you pretend, if you would, that the baby is perfectly fine, destined to be 100 percent normal, and for whatever reason has severe trauma.

And so he's been -- that's an acquired disease. It's something from without coming into the child. Same thing could have been asphyxia or meningitis or a toxin, or something from without affecting the child.

And we know about a lot of those. I mean, if somebody has a cord prolapse for an hour, well, sure, that's enough to do it. I think there probably are acquired things that we're not very savvy about in that formula too.

So there's the genetic things we know about. Genetic things we don't know about. The acquired things we know about, and there's probably some acquired things we don't really know about.

Q Are you familiar with any study or literature which identifies what percentage applies to each of those four categories of the universe of children with CP?

A I think it's been looked at from a number of different points of view, but my rule of thumb is that -- let's just say perinatal events, in other words, bad things that happen to kids right around time of birth probably accounts for 15 percent or 20 percent of cerebral palsy.

The rest of the world of cerebral palsy you look at the labor and delivery, and say that I can't see anything here that would have done this to the child. So whatever happened that wasn't a birth deal, it was either before birth or whatever is going on.

Q Or genetic?

A Or genetic. Only 15 or 20 percent of the world of CP is attributable specifically to perinatal acquired disorders.

Q And when you use that term, perinatal acquired disorders, are those the identifiable ones or does that include the unidentifiable causes?

A Identifiable.

Q So it's 15 to 20 percent are identifiable events that are not genetic. What

percentage are unidentifiable, not perinatal events that are not genetic? In other words, I want to get all four subcategories, if I can.

A Well, part of it is that since, for example, we talked a little about chorioamnionitis. This whole story about infection, people are just learning what the rule is. So a couple years ago, nobody would have mentioned that because it wasn't considered to be a player in this. So that's something that's not genetic. It is acquired.

And I think we're still learning what role, if any, this infection story plays in perinatal disease and CP and so forth. So part of it is that I don't really know what the answer is. I certainly, firmly believe that the vast majority of these kids have nothing to do with perinatal asphyxia or trauma or things like that.

Q Okay. I'd like to try to, if I can, get what percentage or what the percentages are between genetic versus non-genetic, just that one major division. What percentage of cases of CP involve genetic disorders, whether they

are known identifiable genetic syndromes, or not?

A Okay. And again, I'm not citing any special literature, so I'm sure you could pull a book out and show me something different, but I think probably about a third of them are clearly genetic.

Q So something approaching 50 percent of CP cases in your opinion, if I'm understanding you correctly, those fall under the category of non-genetic, but unknown etiology; is that right, if a third of them are genetic?

A Identifiable genetic. In other words, you look at the kid and say, Oh, that's sector syndrome. That's genetic. That's why he has CP. A third are overtly genetic.

Q A third are identifiable. Let me go back because that was not my question. My question was of all CP cases of the universe of CP cases, what percentage of them are genetic, whether they are known etiology or unknown etiology?

So, what portion of the 100 percent

is comprised of genetic causes, even if we don't know now what they are?

A Okay. Again, probably not answerable in the same light that if you say, This kid's dysmorphic, Down syndrome, whatever, he's genetic. Then there's other children where you say, It might be genetic. It's possible. I suspect it, but unless you have something more than that, it's just a speculation.

So in terms of things that are clearly, I'm comfortable it's genetic or acquired disease affirmed, or perinatal asphyxia trauma, that gets us into about half of them where you're pretty comfortable. And probably half where they might be genetic, but you just don't know. Truly don't know.

Q So 50 percent might be either unidentifiable genetic or unidentifiable acquired. Okay. But if I understand your earlier testimony, the vast majority of patients are not due to identifiable causes from birth trauma?

A Yes. That's true.

Q Are there incidents of metabolic

acidosis of unknown etiology?
A Yes, there are.
Q To your observation where Sarah's -- on either of your examinations, were Sarah's fingers, to any degree, flattened?
A When I saw her, no. Now, especially the last exam because she was chubby from the steroid business. That would sort of blur your vision, but I didn't really appreciate anything dysmorphic about her when I saw her.
Q What about shape of her head?
A Well, again, she is microcephalic. And from a strict point of view that's formation that's not right. It's dysmorphic, but in terms of, well, does that tell me something genetic about her, no, it doesn't, because it's from acquired damage.
Q Thank you very much.

(Witness excused.)

(Deposition concluded at 4:35 p.m.)

C E R T I F I C A T I O N

I, Carmen T. Santiago, Professional Reporter and Notary Public, do hereby certify that the foregoing is a true and accurate transcript of the stenographic notes taken by me in the aforementioned matter.

DATE: 7/28/98

CARMEN T. SANTIAGO