SUPERIOR COURT OF COMMON PLEAS

CUYAHOGA COUNTY, OHIO

GLORIA MASLANKA, Individually and as Parent and Natural Guardian of Shane Maslanka,

Plaintiff,

vs.

METROHEALTH MEDICAL CENTER,

Defendant.
_____/

CERTIFIED COPY

CASE NO. CV-05-552424

EXHIBITS ATTACHED

DEPOSITION OF PATRICK D. BARNES, M.D.

Thursday, March 8, 2007

Pages 1 - 85

REPORTED BY: DARCIE L. MOORE, CSR NO. 3143

GROSSMAN & COTTER
CERTIFIED COURT REPORTERS
Comp-U-Scripts Weber & Volzing

Mailing Address:

117 S. California Avenue, #D-201 • Palo Alto, CA 94306
Phone 650.324.1181 Fax 650.324.4609

465 California Street • San Francisco, CA 94104
Phone 415.395.9330 Fax 415.395.9254

A P P E A R A N C E S

FOR THE PLAINTIFF:

BECKER & MISHKIND CO., L.P.A.
BY: MICHAEL F. BECKER, ESQ.
Becker Haynes Building
134 Middle Avenue
Elyria, Ohio 44035
(440) 323-7070 Telephone
(440) 323-1879 Facsimile
mbecker@beckermishkind.com

FOR THE DEFENDANT:

REMINGER & REMINGER
BY: CHRISTINE S. REID, ESQ.
1400 Midland Building
101 Prospect Avenue, West
Cleveland, Ohio 44115-1093
(216) 687-1311 Telephone
(216) 687-1841 Facsimile
creid@reminger.com

INDEX

EXHIBITS

BE IT REMEMBERED that, pursuant to notice, and on Thursday, March 8, 2007, commencing at the hour of 1:22 p.m., thereof, at the Offices of Grossman & Cotter, Comp-U-Scripts, Weber & Volzing, 117 South California Avenue, Suite D-201, Palo Alto, California, before me, Darcie L. Moore, a Certified Shorthand Reporter, there personally appeared

PATRICK D. BARNES, M.D.,

called as a witness by the Defendant, and who, having been administered the oath by me, was examined and testified as hereinafter set forth.

EXAMINATION BY MS. REID

(Whereupon, Defendant's Exhibits 1 and 2 were marked for identification.)

MS. REID: Mike, before I forget, just one thing for the record. As you know, we had filed a motion a long time ago about the late production of Dr. Barnes as an expert witness in this case, a motion to exclude. You replied. It has not been ruled upon. But can we agree that taking the deposition today in no way waives our position that we put in that motion? I just thought about it.

MR. BECKER: Correct. I won't use the fact that you've taken the deposition as a waiver. I will argue to the trial court there has been no prejudice. Fair enough.

Go ahead.

BY MS. REID:

Q. Dr. Barnes, can you state your name for the record, please?

A. Patrick David Barnes. That's B-a-r-n-e-s.

Q. Dr. Barnes, as I introduced myself, my name is Christine Reid. And I represent Metrohealth Medical Center in this case. And we're here for your deposition today.

I know you've been over this process before, so I won't go over the rules in too much detail. But one thing that's important, make sure you understand the question I've asked you before you answer it. I'm positive I will butcher a term or phrase or something today. So if you don't understand what I'm asking or I misuse a medical term, please correct me, because, obviously, on the flip side, when you answer a question, we're going to rely on that answer and assume you understood it.

A. Okay.

Q. All right. You were kind enough to provide me a copy of your file. I just want to ask you a couple questions about the file materials. Your initial contact, it looks like, from Mr. Becker's office at least in writing via letter was on August 18th, 2006. Is that correct?

A. Yes, ma'am.

Q. Did you have a conversation or an e-mail communication with Mr. Becker's office prior to that time?

A. It's possible. That's commonly the way I'm contacted.

Will you review a case? Yes, no, whatever.

Q. In this initial August 18th, 2006 letter, which we'll have marked at the end of the deposition, it looks like you got a copy of all the ultrasound films and the discharge summary; is that correct?

A. Yes, ma'am.

Q. Do you review the discharge summary around the same time you're reviewing the films?

A. I usually don't -- I usually review the films first. I usually get the reports, too. So the reports -- they're not listed in there. I usually get the reports, too.

Q. In fairness, it says, "I'm enclosing, under seal, the reports from the aforementioned films." They did come but --

A. Oh. Yeah. They're under seal.

Q. Do you remember them coming in a sealed envelope?

A. Yes. Usually that's what Mr. Becker and a lot of other attorneys do, because they want pretty much a blind read. I do request the discharge summary. I usually

don't look at it until usually afterwards. The reason I require the discharge summary is I want to -- it's not because of Mr. Becker or anyone else, but I want to make sure the case is being properly represented to me for review.

Q. What do you mean by this?

A. Well, for instance, when I first reviewed this case, I wrote up here, "36 weeks," which is marked out, WGA and then put "27." I thought it was a 36 weeker when I read the discharge summary and found out that it's actually -- wherever that is on there.

Q. Okay. Oh, you're looking at GA, by day, 36 weeks?

A. And then, by exam, 27. So I didn't quite understand what was going on there. Of course, when I reviewed this thing, I'm going this is not what I would ever expect for a 36 weeker. Then I looked at that and crossed out "36" weeks and put the "27" when I realized, you know, there's something wrong or discrepant here.

Q. Okay. And you're referring, just for the record, to your handwritten notes on the back of the August 18th letter; correct?

A. Yes, ma'am.

Q. All right. Now the fact that you have at the top of your notes date of birth and the gestational age

corrected to 27 weeks, does that indicate that you did look at the discharge summary before you reviewed the films or before you authored the report at least?

A. Commonly I will look at it to get the gestational age.

Q. All right.

A. Yeah. That's usually it. And the rest of it, you know, I don't rely on the rest of it anyway. But I really do need the gestational age to interpret imaging, like anyone else.

Q. Okay. Now as far as the reports, the reports from Metrohealth Medical Center, the interpretations of the ultrasounds at issue, did you ultimately review those?

A. Absolutely.

Q. Okay. When do you do that in the process of your review?

A. Usually after I go through all the imaging, I'll review those and compare them and see where I disagree or agree.

Q. All right. Do you make any notes on these interpretations as to whether you agree or disagree with the interpretations done at Metro?

A. Sometimes I do. But I didn't on any of those, I don't think.

Q. Okay. As we sit here -- and we can go through

this in more detail, do you know if there are points on these interpretations that you have areas of disagreement with?

A. There may be some.

Q. Okay.

A. I don't remember offhand because I don't rely on those. There may be some.

Q. All right. Well, we'll go through that as we go through the films then in a more organized fashion, or at least attempt to.

On the back of the August 18th letter are your handwritten notes. I'm assuming what you've written here are the interpretations of the films as you reviewed them?

A. Yes, ma'am.

Q. Okay. And then ultimately what's in your notes was put together into your report in this case?

A. Yes.

Q. Which was dated -- the report was October 3rd of 2006.

A. I think that's correct. You may have my copy. There you are.

Yes, that's correct.

Q. Did you have any discussion with Mr. Becker between August 18th of 2006 and October 3rd of 2006?

A. Yes.

Q. What do you recall about that discussion?

A. Okay. If you look on the front of the correspondence of August 18th, 2006, there is --

Q. A phone conference.

A. -- a phone conference of 9/20/06 from 1:00 to 1:25. Then I have written down there LOP 9/26/06. Then you see -- the other note is about the statement that I sent.

Q. What does "LOP" mean?

A. That's probably when I did my letter of opinion draft, first draft, I would imagine.

Q. Okay. Do you send a draft to Mr. Becker then?

A. Yes. Different attorneys are different. They all want to take -- some want to take their notes and send me what they think is what I told them over the phone, and then I correct it.

Others say, "Send me a draft. I want to look at it. Then I'll call you or I'll e-mail you and then put it on letterhead and send it to us." I'm not quite sure which way this went, but it was one of those two ways.

Q. So you can't recall whether Mr. Becker drafted something for you and sent it to you or whether you drafted something and sent it to him?

A. I don't recall.

Q. Could have been either way?

A. Correct.

Q. The report that's in your file that's not on letterhead, this is what you e-mailed to Mr. Becker ultimately?

A. Yes, ma'am.

Q. And that's your copy of the final report?

A. Yes. And then I think probably I had -- I changed the date when he wanted it to put it on letterhead, probably.

Q. Okay. And it's the same date on the letterhead, so...

Do you know if there were any changes made to your report prior to this October 3rd of 2006?

A. If there were any changes. That's possible.

Q. Okay.

A. It's possible. I don't recall what they were. I don't think anything substantive.

Q. All right.

A. Commonly the attorneys will say, well, could you use this language or could you be a little stronger here, a little stronger there.

I don't recall that about this particular case.

Q. Can't remember one way or another about that either?

A. (Shaking head.)

Q. In your file materials here, do you have the report of Marilyn Siegel. It's the March 24th, 2006 report. Do you know when you received this report?

A. Sometime after -- pretty sure sometime after I reviewed this and had at least a phone conference. I don't know whether I received it before or after my letter is what I don't -- I don't think I referred to it in my letter.

Q. No. It's not referred to in any of the enclosure letters you've produced here, nor is it referred to in your letter.

A. Might have been faxed to me. Doesn't look like a fax sheet.

Q. Do you have any other correspondence at your office or at home that would indicate when you received Dr. Siegel's report?

A. No.

Q. Is it reasonable to assume you received it before you did your opinion letter?

A. Yeah. It's possible. I just remember when I -- with Mr. Becker and many attorneys, then they'll say, well, now that you've told me what you've told me or you've provided the letter, I want you to review, you know, the plaintiff or the defense neuroradiologist or whatever, who I may or may not know. And they send it to

me. And then they ask me, let's have a phone conference, talk about it, or whether to address the issues or not in my letter. So sometimes I address that in the letter, and sometimes I don't. They'll commonly say please address that in your letter or don't. So I have no idea when I got that.

MS. REID: Do you know if you would have correspondence, Mike, that would indicate when this was sent to Dr. Barnes?

MR. BECKER: No, I don't. It wouldn't be in here, in this file that I have here. I don't have cover letters in this file.

MS. REID: Maybe when we get back to the office, I'll follow up with you, Mike, and see if we can answer that question.

MR. BECKER: Okay.

BY MS. REID:

Q. Do you know Dr. Siegel, Dr. Barnes?

A. I do. It must have been ten years since I've seen her. I used to see her at the SPR meetings.

Q. And what's that?

A. Society -- doesn't everybody know? Society For Pediatric Radiology meetings is where I would see her.

Q. What is your understanding of her practice?

A. Well, I don't know now; but her past practice is

a pediatric radiologist at the Children's Hospital in St. Louis was primarily body imaging, as I recall, rather than neuroradiology except for ultrasound.

Q. Is it your understanding that her practice focuses primarily on ultrasonography?

A. Yes. And also CT and/or body imaging techniques. I think she's written articles and textbooks on CT and ultrasound.

Q. Have you ever, in your practice, referred to any of her texts on ultrasonography in particular?

A. In past years, yeah.

Q. Is she respected in the field?

A. Yes.

Q. Can you tell me a little bit about your practice so I can understand what you do on a day-to-day basis.

A. It is havoc. Oh, from about 7:00 in the morning till 6 o'clock at night I'm doing -- I'm supervising and conducting and interpreting and teaching CT and MRI, brain and spine and head and neck in children all the way from the fetus to the, oh, teenage years. So I run the CT and MRI. I'm kind of the medical director of the Packard Hospital, Children's Hospital, MRI/CT center and the chief of pediatric neuroradiology. So I'm teaching the residents, fellows, medical students. And we work side by side during the day. So we go pretty much all day. I

also -- I do not offer -- usually do not offer primary reading of the ultrasound exams. The pediatric radiologists do that. But I consult with them and the neonatologists a lot, just about every day, on neonatal ultrasound, that there may be a problem with interpretation. Do we need a CT? Do we need an MR? So we usually have access to ultrasounds on just about everybody we image, particularly preterms. So that's pretty much what I do every day. Yeah.

Q. You mentioned that the Lucile Packard Hospital has a CT/MRI center.

A. Yes, ma'am.

Q. Is there a separate ultrasound center, or how does that work?

A. Well, we're downstairs in the basement, CT/MR. Ultrasound is upstairs in the general department. And we're all interconnected electronically.

Q. Is there a director, separate director, of the ultrasound center?

A. No. We have a chief of pediatric radiology, Dr. Barth. And then everybody else rotates and does ultrasound with the rest of the imaging studies they do, CT, body CT, plain film, reading, so on and so forth.

Q. The only ultrasound you review, I assume, is of the brain?

A. Yes. Occasionally of the spine when there is a question about: Is there a tethered cord or not? Do we need an MRI?

Q. Can you break down what percentage of your day or your week relates to interpreting CT/MRI versus what percentage relates to ultrasounds?

A. It's probably about 85, 90 percent CT/MRI. And, of course, on the neonates and the young infants, which makes up about 20 percent of what I do, maybe half of them will have ultrasounds that we get asked about, or an ultrasound comes with them from one of the satellite hospitals to interpret.

Q. Okay. Your daily practice is overseeing residents and their interpretation. Is that --

A. Well, yeah, pediatrics is a little different than the adult hospital. We're there working.

Q. Okay.

A. So this has always been my problem with pediatrics versus adult.

On the adult side, residents and fellows commonly run the program. The faculty drop by once or twice a day.

Here, we work side by side with the residents and fellows because the pediatric cases -- we're not doing a very good job in medical school in this country teaching pediatrics. We have residents and fellows coming who had

very little pediatrics. It's astonishing. Plus the fact is the children can get sicker faster, plus most of them need sedation or anesthesia. So we need to be there. We check all their cases before we say we're done, and now you can recover them from anesthesia or whatever.

Q. Right.

A. Plus it's a much more -- it's more of a clinic. And the people we consult with are pretty rarely, you know, medical students from other services or residents from other services. It's highly faculty-run but with very large training programs there. So I spend a lot of time with the neonatologists, perinatologists on these particular cases and everything, and the residents and fellows, too.

So did that answer your question?

Q. I think so.

Do you do any other teaching, didactic lecturing teaching as well at Stanford?

A. Yes. At least twice a month teaching residents and fellows, medical students, didactic lectures on pediatric neuroradiology, all aspects of it. And I particularly give a series of neonatal brain imaging lectures and also fetal, which does include ultrasound; but they're comparing ultrasound CT and MR and how to utilize which and when. And also with regard to lectures

involving residents and fellows and other programs, neonatology, pediatrics, pediatric neurology, pediatric surgery, teach them some pediatric neuroradiology. Basics.

Q. Gotcha.

In your packet of materials, you also were provided Dr. Siegel's discovery deposition as well as the films that she reviewed at the time of her deposition?

A. Yes, ma'am.

Q. In reviewing Dr. Siegel's deposition, did you pay attention to what her practice is and what it involves?

A. Yes.

Q. What's your sense of, you know, her role in interpreting neonatal head ultrasounds?

A. It's a major role for her. Yeah.

Q. Bigger role than your practice?

A. For me personally?

Q. Yeah.

A. Well, I would say so. She's probably reading more ultrasounds every day than I read. Yeah. Sure.

Q. You know, why don't we --

MR. BECKER: Excuse me. Did you mean head ultrasound or just any ultrasound?

MS. REID: I said, "head ultrasound."

THE WITNESS: Depending on who she shares it

with. You know, like at our place, there's seven or eight pediatric radiologists rotate. I don't know how many at her place rotate. So for any one pediatric radiologist, I may be reading as many as they are, but certainly not as many as all of them are.

BY MS. REID:

Q. Gotcha. Before we start, I just want to make sure I understand exactly what your opinions are going to be today. I think if we focus it down into what area you're going to testify on, then we can cut to the chase.

A. Okay.

Q. It's my understanding you are going to provide opinion testimony as to the timing of the intraventricular hemorrhage and the PVL in this case; is that correct?

A. Well, primarily the PVL.

Q. Okay.

A. But both, but primarily the PVL.

Q. Primarily the PVL. Are you going to provide any opinions as to what caused the PVL or the IVH in this case?

A. Not specifically, no. I mean there are general mechanisms that we know that are associated with this and the so-called pathophysiology; but to point to this event versus that event or who did what or who didn't, I'm not going to be offering any opinions on that.

Q. Nothing that would go into the standard-of-care issues presumably is what you're saying?

A. That's correct.

Q. But we can talk in general terms as to what the pathophysiology of PVL is, for example?

A. Uh-huh.

Q. Okay. Why don't we -- let's get started with this -- the IVH issue because it sounds like we're going to spend more time on the PVL. Is that fair?

A. Okay.

Q. You, in your initial -- your review of the initial ultrasound and this film, which is 8/3 of 2001, you did find evidence of intraventricular hemorrhage; is that correct?

A. Yes. Grade 1 and grade 2.

Q. All right. Do you have an opinion, as we sit here today, as to when that -- that grade 1 and grade 2 hemorrhaging occurred?

A. You can't really time it that well --

Q. Okay.

A. -- from ultrasound. I mean, you look at this, and you have areas that are what we call hyperechoic, meaning increased echoes. And you have some areas that are the term hypoechoic, meaning decreased echoes. So trying to figure out: Are we dealing with acute; that is,

increased echoes, because it's clotted? And then the hypoechoic, that has decreased echoes, is it not clotted? So when is it not clotted? It can either be hyperacute, meaning hours --

Q. Right.

A. -- to probably less than a day, hasn't clotted yet. In those cases, it may not be increased echoes. And then it clots and becomes more echoic. And then also obviously it evolves. And over the next days to a few weeks, it can start liquifying again and become hypoechoic. So you don't know where you are in that for sure. So that's why you do serial imaging.

To give you an idea is: Were you hyperacute, or were you more subacute?

Q. What stage are you in?

A. Correct.

Q. And looking at the serial ultrasounds, does that help you make a determination as to where you are?

A. It does and it doesn't in this case.

Q. Why is that?

A. Because this one particular area and you know we had a little right and left problem.

Q. Exactly. If it will help you use the film --

A. No. It's okay.

Q. Okay.

A. So on the left side, which is mismarked as right to begin with --

Q. Right.

A. -- and then we catch up with that later, there are the classic grade 1, grade 2, germinal matrix subependymal hemorrhages. They're right where they should be.

On the right side, more on the right side than the left side, are these little dark dots in them. You look at that and you try to decide: Are those preclotted or post clotted? On the follow-up, I'm not sure that it helps. Except for one area on the first ultrasound that's just above where the left clot is that you try to decide if it really is part of it, or is in the immediate periventricular region that is hypoechoic center -- and I think this is what Dr. Siegel is pointing at -- hypoechoic center, dark, relatively dark, very, very small, just a few millimeters, and around it all these echoes. So the question becomes: Is that part of the hemorrhage? Is that cystic PVL? Or -- and I was a little surprised she didn't mention this -- is this periventricular hemorrhagic venous infarction? And you can't distinguish among those; but as this evolves, you see it on the coronal and sagittal images on the first ultrasound. Except at the end where they decide to flip the images and do it right,

you don't see it right. Then on the next ultrasound -- that first ultrasound was August 3rd.

Then the second ultrasound of August 7th you see it maybe only on one of the sagittal images where she has it circled. Then it's missing for two or three scans.

Then beyond that, we begin to start seeing a macrocystic area come up in that that's quite complex.

So that in and of itself tells me that it's more likely an early periventricular hemorrhagic venous infarction rather than cystic PVL. And you'll find that in Dr. Siegel's book. And I think she's even published on it before.

When it's associated with the higher grade intraventricular hemorrhages, what they call grade 4 --

Q. Right.

A. -- and years ago we mis-, kind of, classified it and mischaracterized it. We thought it was increasing hemorrhage in the ventricles that then burst through the ventricular wall out into the brain.

When, in fact, what we found out is it wasn't that at all. It could be very early on and that subependymal hemorrhage, like we see here, there is a vein that goes right through it. It is a subependymal vein, meaning it's just below the lining of the ventricles. It's also known as the thalamostriate vein or branches of

it. When you get that germinal matrix hemorrhage, it squeezes it. It blocks the blood flow that's coming toward the ventricle. And then right next to it, you'll get a hemorrhagic infarction, which is a stroke that has hemorrhage into it. It will start out, if you catch it very early, as a center that's hypoechoic surrounded by increased echoes. That center presumably is when the hemorrhage is either still liquid before it clots or it's the area where there is necrosis and loss of tissue, liquefaction associated with this infarction. And there is a classic picture of it in Dr. Barkovich's book in that stage early-on ultrasound. And then as opposed to most of those, this one seems to be quite small. Some of them are big and associated with increasing intraventricular hemorrhage. And that's the worst injury from germinal matrix hemorrhage, okay, that a preemie can have that doesn't have PVL.

Q. Okay.

A. So I think because of the way this started out originally, then it disappeared for two or three scans and then it cavitated, I wonder if that's not the evolution of an early stroke like that, which you'll find in Dr. Barkovich's book, and others can be quite early. Can be within the first day after a baby is born. Can be prenatal. Can be prenatal intrapartum or even prepartum.

I mean that's possible. They're very hard to time. So the rest of the germinal matrix hemorrhages looked to me to evolve from that point on except for that one. And then later on, when we talk about the PVL, all of the rest of that seems to evolve postnatal.

Q. Okay. Let me clarify one thing. My sense of what Dr. Siegel is saying is that there were cystic changes within the hemorrhage; correct?

A. Yes, that's what she's saying.

Q. And then a small cyst which she associates with PVL; correct?

A. Yeah. She separated the two.

Q. Right.

A. As you know, one in the germinal matrix and then one in the periventricular cyst.

Q. Right. Do you see either of those cyst formations either in the germinal matrix or in the periventricular region?

A. Yes. That's what I'm talking about.

Q. Right. Okay. That's where I'm losing you.

A. Yeah.

Q. So you agree with Dr. Siegel's interpretation that there is cyst formation in both of those areas?

A. Yes, ma'am, I do.

Q. Okay. So when you look at the ultrasound, you

see the same cyst she sees?

A. Well, for that particular point, yes.

Q. The particular point meaning August 3rd of 2001?

A. Yes.

Q. Okay. Where the dispute then is, is what's the significance of those cysts?

A. Correct.

Q. All right. And that's what you were just explaining to me?

A. Yes.

Q. I mean, feel --

A. No. That's right. That's what I was explaining, because you see that. You know immediately what the issues might be here when you see that.

Q. Okay.

A. Particularly when you think you're looking at the 36 weeker and you're going, "What the hell is going on here? I mean, why are we having this at 36 weeks? Is this prenatal?" You go -- you start looking at it for a 36 weeker, and you go, "My gosh, you know, we don't expect to see this kind of hemorrhage at 36 weeks gestation. We see this kind of hemorrhage 34 weeks or before."

Q. Right.

A. Then you start looking at it evolve. Then you go, oh, "My gosh, this evolves over the next couple months

towards term." Then I go, "This baby is not term. This baby is not 36 weeks. This baby is 27 weeks." So now when you adjust it for the correct gestational age, it begins to make sense.

Q. Because these complications we're dealing with are complications of prematurity?

A. Yes, ma'am.

Q. Okay. Let's go ahead and put the August 3rd film up. I think that will help me, at least, keep my head straight.

A. I have to put this here because I'm right-handed.

(Discussion off the record.)

BY MS. REID:

Q. We're going to -- Dr. Barnes, what you've put on the view box is -- was it Exhibit 1A?

A. Yes. It's Exhibit 1A from Dr. Siegel's deposition. What was the date? It says here. Is that right? August 30th?

Q. No. That's the date of the film. We put the film dates on. It's Exhibit 1A from Dr. Siegel's deposition. All right.

What she has circled there are both the cystic changes that the germinal -- that are close to the germinal matrix and the cystic changes within the periventricular space; correct?

A. Correct. For instance, on the first row of the images -- and these are what we call coronal images, kind of like you're looking at the front of the baby. And of course the transducer is put on the -- which is going to emanate the sound waves into the baby's head and how they get reflected back, it will record that electronically and by way of computer. So what we're seeing at the top of the baby's head -- and this is the fourth image over on the top row, as I said. And you can usually get it exactly by the time. This image was taken at -- I'm going to use military time, if that's okay.

Q. That's fine.

A. 16:05:59. On August 3rd, 2001. And so as you're looking at the front of the baby here. And, conventionally, this is usually left; this is usually right. But we found out later that's reversed. Where she has an arrow, that yellow arrow there is pointing to this area right here that has increased echoes. It's bright white; and it has a few dark dots in it, kind of gray dark. And it's right adjacent to this dark triangular area in the middle and this dark area just above it that's part of the frontal horns of the lateral ventricles, which is toward the front of the brain.

So when I look at her deposition -- and I agree -- there are these mixed echo hemorrhage, larger on

the left than on the right. And it's actually pushing -- it's actually pushing on the ventricular system, causing some mass effect, which is another indicator that it's acute or recent. Some would probably say less than three days when it shows that type of mass effect.

Okay. So that's one thing she's pointing to.

On the second row, second image, again, it's a coronal image or like we're looking at the front of the baby's face even though the right and left, by convention, may be reversed. Where a little further back and what she's pointing to here, what she's circling right here inside of the yellow circle are these increased echoes here and that tiny little dark dot there. I'm pretty sure that's what she's talking about.

Q. Right.

A. And then you see where that is. And it's directly adjacent to this part of the lateral ventricle, the frontal horn, that has an echo in it that I'm sure is probably hemorrhage. And below that is the large hemorrhage that you see here. So that's a pretty classic relationship where you see germinal matrix hemorrhage that's large is causing mass effect. It's usually on the ventricle, plus it's squeezing the vein that will run around the ventricle and down like this. Then when you do that, that vein can burst and hemorrhage; or it can

thrombose. The blood clot inside of it blocks it and backs up into the brain, blocks the arterial inflow with back pressure that's bringing oxygenated blood. And that's how you get your little stroke.

Q. So that's the process of infarct you described?

A. Right. So what you have to differentiate it from is: Are we dealing with periventricular hemorrhagic venous infarction versus hemorrhagic PVL, periventricular leukomalacia? And it can be difficult to tell the difference between those two. This type of infarction that I'm describing classically occurs towards the front. And the PVL classically occurs towards the back, but also can involve the front.

Q. But isn't that image you were just pointing to more towards the back than towards the front?

A. Actually, it's about right in the middle. So it's more toward the front than the back, and it's right in the middle. And that's where you get these periventricular hemorrhagic venous infarctions, is towards the anterior part of the ventricular system rather than the back.

Q. Under that scenario of the infarction, what causes the cyst formation?

A. Well, early on, as I said, is when it's darker than the echoes. The echoes is edema and can be some

clotted hemorrhage. The dark area can be the hemorrhage that's not clotted; or it's necrosing, dying brain in the infarction that doesn't show increased echoes. And then as it evolves and that blood is cleared out of that stroke and as the liquefaction that's liquid becomes a cyst, then you will see it later as really dark. And that's what we kind of see later. This turns really dark here. It's this tiny little thing. And it's not just this. Okay. See that? It's not just that, that I think she's circling. I think it's that part also right there.

Q. So as of August 3rd, you don't believe that that process of liquefication and cavitation has occurred yet?

A. I do not from what we see because we clearly go on later and see a big cyst there.

Q. So that's where you differ from Dr. Siegel significantly?

A. Yeah. I can't rule out that there was some small hemorrhagic PVL there or something else. I can't rule that out. Now you're implying, when you do that, that there has been two different things happening at two different times, which is certainly possible in a preemie. But what I'm saying is that that one little area disappears, like they will tend to do in an intermediate phase there, that looks pretty normal. And then here comes the big cysts that are classic acystic

encephalomalacia or the cyst of this type of stroke that we call porencephaly. That's p-o-r-e-n-c-e-p-h-a-l-y. That's just a hole in the brain that's probably going to stay there as opposed to the cyst and PVL that may increase to a point and then disappear.

Q. Okay.

A. Yeah.

Q. So you don't think that this -- we're talking about the second image on the second row, that that -- the process that's identified there has anything to do with PVL?

A. I can't rule out that that's a component maybe of early PVL. Whether it's happened -- whether this is the result of what's happened in the previous two or three days or even before that. If it happened before it, you try to figure out the timing of where you still have something very echogenic, increased echoes --

Q. Right.

A. -- and then a cyst that's forming in it already; wheres, the classic PVL evolution, you may know, is increased echoes from edema. And then those disappear. Then the cysts, tiny cysts, come up. They may increase inside. That's the classic evolution. Here we have increased echoes with a little dot in the middle of it. So if that's evolving PVL from way before, and if you read

and know the literature, you know, you start seeing the cystic phase between two and six weeks before it starts.

Q. Right.

A. But there is that evolution of where: Edema. Edema goes away. You do an ultrasound. You don't see anything, like in this case for the other injury. And then later on, here come these cysts. And they get bigger and bigger. If you're going to reason that that is cystic PVL, which is what we're talking about here, it's hard to reconcile that from something much earlier. Then it disappears here. The cyst and the edema disappear for two or three scans and then come back.

Q. Isn't that what Dr. Siegel is saying, though, Dr. Barnes, is that it is from something much earlier?

A. Well, I don't know. I can't tell from her deposition just how much earlier she's talking about.

Q. Because that could be -- that's a reasonable evolution, isn't it, if the process started several weeks before?

A. Several weeks? Well, the edema, depending upon the severity and classification that's listed for the edema and the flares that are talked about in the literature, those flares that last longer that they report past seven days or even two weeks are usually due to repeated injury, repeated lack of blood flow, lack of

oxygen, repeated injury.

And then there is a phase of that within the edema that you can see these little cysts form. But this completely disappears unlike the third scan -- let me get that date here. On the third scan of 8/15. I think that completely disappears. And it's not on the scan of 8/24. I think that's what she said, too. And then starting about 8/31 is where we start seeing this cystic change there. So if that's true, then we may have two processes going on. But then what I would suggest, if we have two processes going on, we started out with this hemorrhagic infarction I'm talking about. And then we evolved toward cystic PVL after that, that to my way of thinking fits postnatal better than prenatal. Because I don't know how much prenatal she's talking about here, in terms of if she thinks that is prenatal and prepartum, you know, what timing range is she talking about? Is she talking about a day, two days, three days, seven days?

Q. Well, is what you're saying that there is a time frame that is consistent with this progression that could date it in utero; and there is a time frame that isn't consistent?

A. Well, I don't know. I was hoping, by the time that she gave her opinion, that she would give us or me or whoever some idea about the timing of that lesion and how

to explain its evolution in terms of what we see from this first ultrasound on, where it disappears.

Q. Okay.

A. So that's my only question about it. When I first saw it, I struggled a little bit to figure out what was going on here. And then -- because as you follow these scans along, I don't see cystic changes anywhere else in this area until we get to August 31st for sure. In fact, you will see there under No. 4, I say no cysts are apparent on that until we get to August 31st. The first study is on August 3rd. This one is on the 31st. It's what? 27, if my math is correct. 27 minus 3, what is that? Or 31 minus 3. It's not quite a month --

Q. Okay.

A. -- when you start seeing the cysts. Now you go, bang, cystic PVL.

And if you look at the literature and particularly the article that I co-wrote in 2000 with Dr. Ment, et al., the practice parameter that we wrote as an evidence-based medicine exercise evaluating quality of evidence of the literature for writing practice parameters for imaging in the newborn, at-risk neonate, 36 weeks and under versus the term, the quality of evidence did not support using the noncystic phase of PVL with regard to timing and, particularly, prognosis. And that's been the

problem with ultrasound.

Q. I'm not following what you're saying.

A. Well, what I'm saying is, No. 1, to make a diagnosis of PVL of this type, the cystic phase, you've got to have the cystic phase. Okay. So whether you consider the increased or decreased echoes relevant with regard to edema or what's going on, it's the cystic phase to define it.

When you do the timing, the cystic phase is used, that two- to six-week phase. Plus the vast majority, if you go to the evidence-based medicine literature, you start seeing at about three weeks or less the cystic phase.

Q. Changes.

A. Yeah.

Q. So you're saying that you need the cystic changes to make a conclusion as to the timing?

A. That's what the literature says. That's not what I'm saying. And we have to live by the evidence-based medicine literature these days. So that's why we wrote that parameter.

Then we did two subsequent research studies that I also coauthored comparing ultrasound with MR, actually two others with MR on preterm brain injury, which has kind of been my area of research since I arrived here at

Stanford. And probably about five or ten years before that, when I worked with Dr. Volpe and others at Boston Children's, is comparing MR with ultrasound. So we did a big study here at Stanford comparing ultrasound in preemies of varying gestational ages, particularly under 30 weeks was our group with MR and tracking the timing of cystic PVL.

Q. Now, where I'm getting confused a little bit is in the first paragraph of your report, you state in the final sentence that this 8/3/01 ultrasound does show the periventricular echoes and evidence of the earliest phase of PVL.

A. Yes. That's true. Elsewhere. Oh, yeah, outside this area. Yes, I think there are increased echoes. That's the next part we're going to get to.

Q. Okay. So you're referring to just the echogenicity of the white matter?

A. Yes. And including this area.

Q. Okay.

A. So I wasn't sure -- you know, I read these in sequence. I put them in line. I put them in sequence. I usually report the findings as I go. So that did include this area.

Q. Okay.

A. Yeah. Plus the hypodensities that I describe

with regard to the subependymal germinal matrix hemorrhage grade 1 and grade 2 that I describe here, plus this echogenic area out here. And I put that into the periventricular echoes, plus some basal ganglia and thalamic echoes, not knowing at that time what we're dealing with. But then it becomes clear as it evolves.

Q. But as you look at this 8/3/01 film, it's fair to say there is evidence of early stages of PVL?

A. Yes. With edema.

Q. With edema?

A. Yes, ma'am. I agree with that.

Q. And does it take some time from the initial insult to develop the edema which we're seeing on these films?

A. Yes. The answer is yes.

Q. All right. How much time does it take from the initial insult to develop edema which -- I mean on ultrasound we're seeing it as increased echogenicity; right?

A. Yes, ma'am.

Q. What's the time frame between that initial insult to edema showing itself as increased echogenicity?

A. That's the big question about ultrasound. That's why the evidence-based medicine literature has great difficulty in supporting timing with regards to the

noncystic type.

Q. Got you.

A. Most of the literature you will read will say you can see it earliest around 24 hours. 48 hours after the insult you start seeing it. Then it will increase to a point, maybe up to seven days, and then start coming down. Then you will see from Dr. Danaman's in his articles, he described several different patterns. You will find this in Barkovich's book about the flare pattern, extended flare, and all this stuff and how that can persist. That particular article that was written had good quality of evidence to support what he was saying. The problem is because there was so much controversy among all the other articles writing about the flares and that type of thing with regard to timing, it could not be evaluated with regard to evidence-based medicine standards.

Q. Obviously you have to hit me over the head because this is just what you were explaining to me before.

A. No.

Q. I see. And I apologize.

A. If you had deposed me five years ago, six years ago, I probably wouldn't even be talking about evidence-based medicine. And that's been the problem with medicine is because we have so many different standards

across the country, you know, you guys and the government and the payers have stepped in and said this makes no sense. If you're going to devise standards of practice, you have to put out the standards and guidelines that have scientific basis, meaning good scientific methodology and biostatistical significance. And, man, that is work. And so that's required for the peer-review literature now. That doesn't mean you can't have an article that's accepted, okay, for the literature. But on those articles, there is going to be a label or indication that this is not level 1 or level 2.

Q. Right.

A. This is level 4. We're allowing it to be published, but the reader is warned. Well, that's a lot of the past literature on PVL, on germinal matrix hemorrhage. And it's reflected in the current literature. And it's reflected -- and I'm happy to see it -- in Dr. Barkovich's newest addition. Recognizing what we published ten years ago, five years ago, because of the advancing technology we have, we were wrong about.

Yeah. Sorry about that lecture.

Q. No, no. What you're telling me -- let me just summarize -- is that you can't time periventricular leukomalacia by looking at the edema or the increased echogenicity. You have to wait until there is cyst

formation and then use this two- to six-week window that we have in evidence-based medicine; correct?

A. Yes, ma'am. That's what I'm saying.

Now, that doesn't mean some people can't offer that opinion at work every day and say the literature says this and that. It might be about that -- it might be about that timing. But the whole problem is particularly with the rough postnatal courses of these babies, the preemies, the respiratory problems, the profusion problems, the problems with sepsis and the other problems they get, they're getting repeated insults to the brain. You know, that's not a criticism of anyone. That's the reality.

Q. Well, that's the reality of premature babies --

A. Yeah.

Q. -- overall.

A. Yeah. They do much better in the mother. They do much better in the mother than they do outside the mother.

Q. Okay.

A. That's why we try to prevent it. And we just don't do a very good job of it in this country of preventing it.

Q. Preventing what?

A. It's one of my -- preterm birth.

Q. Preterm birth.

A. It's one of my major campaigns, and everybody else, regarding cerebral palsy is -- the problem is we just can't seem to prevent preterm birth in this country. And I mean extreme low birth weight. I mean prior to 30 weeks. We're getting down to 24, 25 weeks now that we just can't prevent.

Q. What do you believe to be the major cause of preterm labor in this country?

A. I think it has a lot to do with prenatal care. We have probably the most mixed culture of any country in the world. And that's why our rates of morbidity and mortality and our rates of cerebral palsy in this country, particularly regarding preterm, are higher than other parts of the country. There's no one in the world who has better neonatal care here.

Q. Right.

A. So, yes, they're surviving. But, boy, as they survive, morbidity, cerebral palsy, other types of neurodevelopmental delay that we see as attention deficit, hyperactivity disorders, cognitive, is increasing in this country. It is because they do survive, including -- including improvements in obstetrical care. They are surviving.

Q. We talked about this a little bit before, but IVH

and PVL in this picture we're seeing here, they are common central nervous system complications of prematurity?

A. Yes. Well, common. Certainly for the very low birth weight. I mean, we've done so well in this country. This is the experience at Boston with Dr. Volpe and here now at Lucile Packard Children's Hospital where we have very good neonatology here, including with the primary emphasis on the preterm baby with -- what? -- three or four satellites feeding into this hospital. We don't expect to see IVH like this anymore. It's been declining. And that's because we're much more successful in trying to get the mother closer to term. We are seeing quite a few babies -- and they all come to Packard -- that are under 30 weeks. We don't expect to see PVL like we used to. And what we're seeing is the -- now, is not -- we rarely see this, what's called cystic PVL anymore, which is classic for postnatal preemie. What we see is the more subtle diffuse light matter injury. And Dr. Barkovich addresses it in his book reviewing the literature over the last ten years, for which we've contributed to also. It's this more subtle white matter injury which you don't see the cysts on ultrasound. So that's why we came up with MRI near term. It didn't clear quality of evidence for that practice parameter in 2000, but subsequently it has cleared it. That's why now all the at-risk preterms,

30 weeks and under, get an MR just before discharge because the ultrasound misses that subtle injury and will pick it up on MR. Now we let the parents know that this baby is going to need some early intervention because it has some features on its MRI that is predictive that there may be cerebral palsy, or there may be visual. There may be hearing. There may be cognitive problems. And we try to get those babies into early intervention for that.

So this cystic PVL, when we see this, it is getting -- it is unusual to see. It is unusual to see. We don't like to see it.

Q. All right. Why do you think you're seeing less of it?

A. Oh, improved perinatal care, particularly obstetrical and post obstetrical neonatal intensive care. We're seeing less of it. They're better -- we can take care of their lung disease better. We can take care of their circulatory problems better, prevent infection. And there is new types of treatments for these babies.

I'm part of two national multicenter projects, one on preterms with new types of treatment to see if we can prevent this type of injury. We're doing a good job with regard to germinal matrix hemorrhage. Still, there are babies who get it. And some get it very, very badly. We're doing a good job in preventing in many ways this

gross cystic PVL like we have in this case that we know causes terrible brain injury and terrible neurologic outcome. But we've still got this other problem. So we keep the heat on with regard to obstetrical standards and with regard to NICU standards to hold down the major germinal matrix hemorrhages with the posthemorrhagic hydrocephalus to hold down the cystic PVL. And we're working on the subtle white matter injury to where what we have is we may not have cerebral palsy. What we have are children who can't communicate well. They may be autistic. They're hyperactive. It's become a problem in this country. They go to school. They can't. They have to be pushed back. So we're kind of attacking that problem. We're getting a handle on this one. But that's the problem that we're attacking.

So that's the evolution.

Q. Do you have an opinion, Dr. Barnes, as to whether or not, let's say Shane had been born at 29 weeks versus 27 weeks, these complications would have occurred?

A. 29 versus 27?

Q. Yes.

A. It's -- the lower the birth weight, particularly under 1,000 grams, the lower the gestational age, particularly under 30, when you even go further than that, yes, the outcome is worse, just by gestational age and

birth weight alone.

Q. Okay.

A. And that's not even considering obstetrical care, neonatal intensive care. It's just the truth. The older you get --

Q. The less likely --

A. Yeah, the less likely. The less likely, the better the outcomes, and so forth. Yeah.

Q. Well, what is it about the gestational age and the birthweight that causes PVL?

A. It has to do with this immaturity of the blood vessels of the brain to be able to dilate and contract relative to rises and falls in cardiac output to protect the baby's brain. They just can't react to do that. For instance, if there is decreased oxygen in the blood to the baby's brain, okay, what's supposed to happen in the mature brain, blood vessels dilate up so whatever cardiac output is there can deliver more. Well, these vessels just sit there. Or if the blood pressure goes up and you get a surge like in what's the reperfusion phase, they just sit there, dilate. And now they get too much hemorrhage, and you bleed. So it's a rock and a hard place. You either get ischemic injury, or you get hemorrhagic, or you get both. And that's the tough part with the preemies. They cannot control that blood flow in

the brain.

Q. And that causes both hemorrhage and PVL? That's the type of geneses of PVL as well?

A. Yes. Now the -- and we know that from our experience postnatally. Okay. Particularly in the very low birth weight with the worst pulmonary disease where you can't get oxygen into the babies. So what do you do? You have to turn the pressure up on the ventilators. What happens with that? Well, as you turn the pressure up on the ventilators, the blood that drains from the baby's brain back to the heart to recirculate again gets blocked. It gets a back pressure on the baby's brain. And those veins are back up and what do they do? Where the really fragile blood vessels are in the subependymal region are, they burst. So here's the rock and the hard place. We're trying to get oxygen in this baby's lungs, and we can't do it. We measure the blood oxygen. It will not go up. So we put more pressure on those bad lungs to try to get it up there. And there's varying and pretty fancy techniques they do to do that, plus they have to maintain this baby's cardiac output. It's a fine line. Are we not going to get enough oxygen and blood flow up there, so we get PVL? Are we going to get too much? And we're going to get hemorrhage.

Q. So it's a challenge for --

A. Oh, yes. And the younger they are, the tougher the challenge.

MR. BECKER: Particularly those that don't get the right medicine.

MS. REID: Thanks for adding that in, Mike.

Q. On that issue -- never mind. We won't go down that road. Strike that.

I want to show you or hand you the interpretation from Metro of this 8/3/01 ultrasound that we have up on the view box right now.

A. Okay.

Q. If you would take a look at that, I'm curious whether you have any differences of opinion whether what was interpreted at Metro at the time?

A. Oh, just the reverse right and left.

Q. Okay.

A. I think there already is some intraventricular extension. They said they can't rule it out. And I agree.

Areas of hypogenicity in the blood suggest some duration of a few days old. That could be true.

And then there is echogenicity of the white matter slightly increased. I agree with that.

They don't mention specifically this area right there, the tiny little area right there; but I don't find

fault with that in any way.

Q. Well, if there are areas of hypogenicity within the bleed, suggesting that these may be of some duration of at least a few days old, doesn't that place it into the prenatal period or in utero?

A. I mean, somebody will have to count back. How old was this baby at the time?

Q. This was done at 58 hours.

A. Okay. If you can see this variation in the hemorrhages over two to three days, that's your range. So, yeah, it could extend back. Yeah.

Q. So it is a reasonable conclusion that there is hypogenicity or cystic formation that was -- occurred in utero?

A. Possible, yeah. It would be included in that range. Yeah.

What I would say is it's not the germinal matrix hemorrhage that's really damaging this brain. It's really the PVL.

Q. Okay.

A. I mean, I'm not -- don't get me wrong. This is not good. Clearly, the literature shows that if you have higher than grade 2 germinal matrix hemorrhage and you have posthemorrhagic hydrocephalus, that puts into the increased probability of cerebral palsy, depending on how

you define it.

Q. That's what we have here?

A. Well, you have a grade 2 hemorrhage. So you're right at that cusp. You eventually have some hemorrhage that gets you to a grade 3.

Q. Grade 3. Right.

A. So you're in that. That's true.

The problem here is the extensive cystic periventricular leukomalacia that we do get later.

And the question is, you know, there is this argument, debate that we still have with the neonatologists. They, many neonatologists, classify the degree and severity of germinal matrix hemorrhage based on the first ultrasound only. Whatever happens after that, they do not upgrade it. And the reason that they don't is because there's so many factors involved in what happens later that can cause the increased hemorrhage other than just prematurity and the complications of it.

There are others who insist that we need to upgrade it later, one to two weeks down the line when this -- there is more intraventricular hemorrhage, and people upgrade it. What they're saying is, is it really messes up the prognostic indicators. It's confounding, because in many of those cases what we find is cystic PVL. And we know that in those cases that do not have germinal

matrix hemorrhage above 1 and 2, the No. 1 indicator of outcome is the white matter injury, PVL, in the kids that do not have grade 1 or 2 hemorrhage.

Q. Okay.

A. So that's what confounds it. So most of the neonatologists in this country say: That's a grade 1 on one side. That's a grade 2 on the other.

Q. But in this case, I mean, it ultimately progressed to a grade 3.

A. Yes. And my neonatology friends would say it's not a true grade 3. You have increased hemorrhage, that's true; but it's not a grade 3 for prognostic purposes in the presence of PVL.

Q. But certainly this intraventricular hemorrhage, which there may be evidence that it occurred -- began in utero, was significant in Shane Maslanka's course?

A. Good question.

It depends on the obstetrical and neonatal care. Because most people do not expect -- most people do not expect for the initial hemorrhage that's grade 1, grade 2, grade 3, whatever it is, to progress unless there is some problem.

Q. Okay.

A. And that common problem that makes it worse is --

Q. It's --

A. Well, hypoxia-ischemia, hypoperfusion, okay, what's manifested as PVL. And then you're dealing with this baby who you can't get oxygen to. And then you try to get more blood flow to it. And what do they do? They hemorrhage some more. I understand why they think of it that way; and maybe you can, too. I know it's tough. They would call this -- later on, this was a grade 1 on one side, a grade 2 on the other side, complicated by recurrent intraventricular hemorrhage and cystic PVL, is what they would call it.

Q. It was never called that in this case.

A. Yeah. I don't know what they called it on the discharge summary finally.

Q. All right. I want you to assume for a minute that Dr. Siegel's opinion in this case is correct, that this cyst that we see on this 8/3/01 film is the result of PVL.

A. Okay.

Q. If that is correct, that hypothesis or opinion she has, then we can date that back two to six weeks and date it back to in utero?

A. Yeah. It's possible. I don't understand -- you know, maybe two weeks.

Q. Okay.

A. But six weeks? I mean, if it goes through the

classic evolution that everyone talks about, okay, why in the world, if we're going to make this six weeks back, do we still have echoes --

Q. Gotcha.

A. -- with a dot in it? Okay.

Q. I'm going from what you just told me on the evidence base, that cavitation, cyst formation occurs two to six weeks after the initial injury. Right?

A. That's right. That's what the literature shows. Correct.

Q. Okay. So if this cyst formation or cavitation is a result of PVL, it's got to date back at least two weeks?

A. That's what some would say. That's what it represents.

Q. That's what it represents. And that's where you disagree that it doesn't represent that?

A. Correct.

Q. All right. Let's talk about the progression of PVL cysts just in general.

A. Okay.

Q. First of all, is there a typical size that you see of these cysts?

A. Depends on duration and severity of the insult.

Q. Okay.

A. If it's one or it's several. So you can have

tiny cysts that appear earlier than others. Then they coalesce and make big cysts, or they stay small, depending on severity and duration of the insults and if there's no more insults. And they may resolve. And you may not see much. For instance, on the state-of-the-art imaging now and MRI, you look at it and go, "Wow. I don't see anything on that."

Q. Okay.

A. Or you can have the more severe hypoxic-ischemic insults because that's what it mostly correlates with.

Q. Right.

A. The more severe or longer duration, more rapid evolution and progression of the brain injury, big cysts, okay, that become coalescent. Then after a period of time, they start to collapse, they disappear. Sometimes they persist. And they get incorporated into the ventricles, so called. Then you get these large ventricles that enlarge because the periventricular white matter was destroyed. So it depends on severity and duration.

Q. Okay. So it's variable, depending on severity and duration.

A. Correct.

Q. The progression. So the fact that this cyst that Dr. Siegel points out is 2 millimeters doesn't say to you

that can't be a periventricular cyst -- it can't be a cyst because of periventricular leukomalacia, the size itself?

A. That's correct. It invites other things to consider. No. 1, it's anterior and it's small. We know those have a better prognosis than the large, more posterior cysts.

Q. In this scenario where we have Shane Maslanka, 27 weeker in the NICU, ultrasound is the appropriate diagnostic study?

A. Absolutely. That's what the practice parameter says.

Q. We wouldn't be shipping him down for CT or MRI during the first 30 days of life?

A. That's probably true. We would be probably -- in this day and age, and, of course, this is 2001 before the practice parameter was written. But the old practice parameter, yes, this is what you do.

Q. Okay.

A. And this baby has all the predictors, from an imaging point of view, just using ultrasound only, for poor neurodevelopmental outcome, particularly acystic PVL. So we wouldn't even do an MR on this baby.

Q. Where does that occur? Do you see that on 8/31 where you start seeing the cystic changes?

A. Yes, ma'am.

Q. Okay.

A. I think it's 8/31. Yes, is when we first start seeing them. I think that's what I said there.

Q. So what you just referred to -- I don't think I can repeat your words, but when he starts showing the poor prognostic indicators, that's what you're talking about as of 8/31/01?

A. That's true. And particularly when it goes from there and progresses to the point that it's not until -- what? -- November 15th that the cysts start decreasing in size somewhat, and the ventricles appear stable then.

Q. What's the significance of that time frame?

A. That -- the extent and the severity of the imaging findings also correlate with outcome. We don't have imaging after that on this baby to see what the final injury pattern is -- I don't guess. I don't guess there is any.

Q. Actually, there were some MRIs that were done later in time --

A. Oh, there were. Okay.

Q. -- when he was transferred to the Cleveland Clinic for care.

A. Okay. But the prognostic indicators that are used, according to the practice parameter evidence-based, are on the predischarge imaging findings. Those

correlate -- those have been shown with outcome measures two, three, four years out. The worse it is on that predischarge near term, or whenever they get released, imaging study, the worse their neurodevelopmental outcome status is.

Q. Is there some significance that between -- that the cyst did not decrease, nor did you have -- did the ventricles stabilize until November 15th?

A. Well, we started seeing them on the 31st. And we go, gee, another -- what? -- two-and-a-half months before we show them that they're beginning to stabilize. As long as you see -- the longer that cystic phase lasts and it's changing and you're getting more and larger cyst, you're getting an evolution of more brain injury. It doesn't mean there is ongoing insults. It could be. These babies, you know, to get them to term or near term is a huge struggle. To get them to the point to where you have them off the ventilator, they can breath on their own -- and, of course, they have terrible lung disease. And they've got to be able to eat and feed and not have any apnea spells or brachycardia spells. That's classic. You have to get their brain, the intact part of their brain, to a point where it's mature enough that either this baby can breath on its own, feed on its own, or it's going to have some help. About the time we get to that point is

where we image these babies before they go home. We do the predischarge MR. And our previous studies have shown and others is that the MR findings then or the ultrasound, the worse the findings, the poorer the prognosis neurodevelopmentally, period. It's been shown in all the literature. So that's why I comment on that. Yeah.

Q. Okay. Let's take the date of August 31st, 2001. That's where you, in your opinion, there is really the first evidence of cystic change as a result of PVL.

A. Yes. I think that's correct. Yes. Yeah.

Q. Okay. Now, if we use this evidence-based -- what's the word? -- medicine you were referring to, that's two to six weeks, that cavitation?

A. That's true.

Q. All right.

A. It's a two to six week. Like I said before, the more severe, the longer duration of the insult, the earlier they show up, and the worse they are. So does that put more toward the two week than the six week? It does. Evolution.

Q. I don't understand that.

A. Okay. As I said earlier, it has been shown that the less severe insults, shorter duration, the PVL commonly is small cysts. Okay. The worst ones are the larger cysts.

Q. Right. I understand that.

A. And they will -- and the PVL -- you can read this in Barkovich. He beautifully summarizes the literature. The smaller cysts evolve a little slower and show up. The big ones often come earlier because it is such a severe insult that you have necrosed brain. You're not just going along with a little bit of ischemia, and there is some injured tissue, and then it takes a period of days, weeks, by way of what's called apoptosis. Are you familiar with that term?

Q. No.

A. Okay. That's another term we have to consider here that's classic part of this injury. There is without with oxygen or blood flow to the brain, if it's severe enough, you get immediate necrosis, cells die, tissue dies, shows up earlier on imaging, evolves to worse injury faster. That's true for term. That's true for preterm.

Here's the other part that's much more elusive for us to detect and that I'm talking about. Okay. You have some drops in oxygen. You have some drops in profusion. You get injury to the cells, but they don't die right away. They're nuclei -- the nucleus of the cell that runs the cell, you know, it's like the engine. It gets a few damaged parts which they can't replace. And over a period of time -- you know, that's where the DNA is

that is the source of the cell and how it survives and how it functions -- it gets damaged. So over a period of time of several hours, depending on the damage, to days, to months, that cell slowly dies. And so those are the little cysts that you get as opposed to, bang, terrible episode, period of no oxygen, no blood flow, necrotic brain, earlier necrosis, big cysts early. So that's when you read in Dr. Barkovich's book and you go to the literature, and you see that two- to six-day range.

Q. Two to six week?

A. Two- six-week range, that's what they're talking about.

Q. So using that analysis of what Dr. Barkovich has written --

A. It's not his primary research. It's others that he's quoting. But he --

Q. What he summarized.

A. Yes.

Q. You're saying, because of the size of the cystic changes we see on 8/31, we're probably closer to initial insult of two weeks rather than six weeks?

A. Yeah. Yeah. You're in that range. But those are the general trends observed in the literature. And, again, based on just like everything else, the way injury evolves in the brain from a lack of oxygen or blood flow

is based on severity and duration, and it's the same for PVL.

Q. This initial cyst that Dr. Siegel points out on the August 3rd ultrasound, is that involved in the cystic changes you see on August 31st of '01?

A. Yes. Yeah.

Q. That area is involved?

A. Yes.

Q. So isn't it possible, plausible, that that is an extension of an initial PVL cyst.

A. Oh, it's possible. Yeah. It's possible.

Q. You don't think it's more likely than not, though?

A. No.

Q. Okay.

A. No. And the imaging doesn't allow me to say it's more probable than not because of the evolution of this. And it's a small cyst. It disappears. You go, "Man, if that's all we have, this kid might be doing lovely. And it's early. Hey, we have some hope here."

And then, bang, much later you get this terrible injury.

Q. Is ultrasound known to have some limited specificity in showing PVL and cystic changes related to PVL?

A. Yes, in two areas. When you go look at the neuropathological correlations with ultrasound for PVL, the correlations are poor with the noncystic type. The correlations are great, almost 100 percent, with the cystic type. So that's why when you're in the cystic phase and you have the cystic phase and evolution of that, that's why that's much more reliable on imaging because it correlates.

Q. Okay.

A. It's the noncystic phase. It includes the early evolution of cystic PVL starting with edema. But it also includes that other noncystic PVL white matter injury I was talking about that we're dealing with now that's not this category.

Q. And that goes back to the point you were making earlier as to why we rely on the cystic formation in dating these rather than the noncystic changes?

A. Yes. Yeah. See that's a very broad range, two to six. That tells you we're not that precise.

Q. Right.

A. But there are some tendencies, as I described earlier, yeah.

Q. Why don't we go through the next film.

A. Okay.

(Discussion off the record.)

BY MS. REID:

Q. Dr. Barnes, we've had the August 7th ultrasound up. And, again, this is the ultrasound film that was marked at Dr. Siegel's deposition.

A. Yes, ma'am.

Q. She -- first of all, on the fourth image on the first row and the first image on the second row, she has some yellow markings on that; is that correct?

A. Yes.

Q. What's your understanding of what's being identified on those films?

A. Well, now that we may have right and left sorted out --

Q. Right. Correct.

A. -- the top row, fourth image of the ultrasound, August 7th, 2001, and the time stamp on that is -- military time is 20:35:34 -- that identifies the specific image -- has a yellow arrow pointing to the left subependymal hemorrhage that shows areas of increased echoes and some areas of decreased echoes, very similar to the first study. The ventricles are larger than on the first study. And there is still a little bit of increased echo just adjacent to that in the subependymal and periventricular region, although we don't see that little dot or cyst as well on the coronal. And I thought she

marked the sagittal image.

Q. I thought she marked -- she did.

A. There it is. Is that it?

Q. There it is.

A. And she has also marked first image, second row. That's on reader's left. That time stamp is 20 hours, 35 minutes, 57 seconds. And, again, she has a yellow arrow on the mixed echo germinal matrix hemorrhage on the left. So I agree with her findings and comments on that.

Q. As it relates to the germinal matrix hemorrhage, you agree?

A. Yes.

Q. Okay.

A. And she has on the second film, second row, third image from reader's left, the time stamp says -- that's the August 3rd. I'm sorry. You know, I thought -- these are both August 3rd.

Q. Isn't there a second?

A. I'm sorry. This is August 3rd that we looked at. This is --

Q. August 7th.

A. -- August 3rd that we also looked at where she marked -- this is the second page of August 3rd where she marked the sagittal images. And on the second page -- we didn't go over this yet -- second row, third image from

reader's left, the time is 16:11:08. She has the yellow arrow on the sagittal -- like a side view on the germinal matrix hemorrhage that is mixed echogenicity. She didn't mark -- oh, here. We didn't go over these on the first study either, and I apologize.

Third row, first page of ultrasound images, again, August 3rd, 2001, time stamp 16:07:10, sagittal image which matches up in the sagittal view or side view with the coronal or front view that we talked about earlier with regard to this area of increased echoes. And the central part of it has relatively less echoes. That's what we were discussing before: Can that be cystic PVL versus periventricular hemorrhagic venous infarction. It's amazing I can still say that. Then she also marks other images on that same film of that same study that are just showing some of the mixed echoes within the germinal matrix hemorrhage on the left.

I apologize. But she did mention that on the August 7th study she did see -- maybe I don't have that exhibit. Let me see. I think I only had three films. And the exhibits I've been talking about are IA, IB, and then this exhibit is not --

Q. Flip that.

A. Oh, is that how it works?

-- is 3. Maybe there is another exhibit because

in her deposition she talks about very clearly that she sees it on two views on August 3rd, and I agree. She only sees it on one view, as I recall that she said that, on 8/7. I see it also. I'm pretty sure it's there on that, but let me see if I can show you on my set. Let's see if we can find it. Okay. Here's my copy of August 7th, 2001 ultrasound. The two top rows are mostly the coronal images. Then starting at the last image on the second row and then the third, fourth, and last row are all sagittal images. So let me see if I can find that left -- a little tougher to find, that area that we were talking about before; but I thought I saw it. Like she says in her deposition, we may be missing it. It may be off to the side a little bit.

Q. Because of the views that are taken?

A. Yes, that's right. So maybe what she was saying is that on the second study of August 7th, maybe she only saw it on the coronal and not the sagittal.

Q. And when you say, "it," you are referring to what? The cystic formation in the middle of the --

A. Yes, the left subependymal and periventricular tiny cysts with the increased echoes around it, that we've been discussing before, the issue being cystic PVL versus hemorrhagic periventricular venous infarction. I think I said it backwards.

Q. Hemorrhagic comes first in that?

A. Periventricular hemorrhagic venous infarction.

MR. BECKER: Is there another film in your packet for the same day?

THE WITNESS: Yes, I think there is.

MR. BECKER: Let's get it.

THE WITNESS: They did the kidney that day.

BY MS. REID:

Q. That doesn't have anything to do with your opinions; does it?

A. It does not. I will concede on the kidneys. Okay. You got me on the kidneys. Well, I think that's maybe the only one.

Q. The only August 7th film.

A. Because it has both coronal and sagittal images on one film. Isn't that interesting. Well, it's the same film. All right. I was expecting that she marked. Let me see. I thought she marked the sagittal, but maybe she didn't. Maybe it's this she's marking, the one we talked about earlier. So I don't see that she has marked the sagittal on there.

MR. BECKER: Let's go off the record a moment.

(Discussion off the record.)

THE WITNESS: So on the August 7th study that we're relooking at again to try to clarify Dr. Siegel's

marks, the two marks that she has on this film are on coronals or the front views, none on the side views. And from the arrows that she has, she is pointing primarily to the left germinal matrix hemorrhage; whereas, on the August 3rd study, she drew a circle around the little cystic area. On both a coronal and a sagittal on this study I don't see a circle or an arrow pointing specifically to that arrow -- area. I thought I underlined in her deposition where she said that, though.

MS. REID: Yeah.

MR. BECKER: I think, Chris, during the depo, I was particularly concerned, since she gave up one of the bases, in her opinion, there was a neuroinjury rating, I didn't spend a lot of time on that. I just wanted to focus in on the cyst that she sees. I had her circle where she saw the cyst during the depo.

MS. REID: The depo is going to speak for itself, but I think -- if we want to take the time to go through the depo, we can do that.

MR. BECKER: No. I'm not sure why we're doing this.

THE WITNESS: Do you want me to show you where it is in the depo?

MS. REID: No, no. I think I know where it is. I can't find it now.

THE WITNESS: I think I can find it. I think it's much earlier.

MR. BECKER: Let's take a short break.

(Recess.)

BY MS. REID:

Q. We were going through Dr. Siegel's deposition on the break, which led me to a question. You've made some markings in her deposition, some underlinings, some parentheses, some stars. Is there any significance to your method? I mean, rather than go through every mark you've made in the deposition --

A. No. Most of the marks are to remind me of what she said about given ultrasounds at given times. I agreed with her in terms of what the findings are. Where I disagree is what they represent and their timing.

Q. Okay.

A. So I'm not sure that there is anything other than that.

Q. All right. So when you both look at these films, you're seeing the same thing. It's just a matter of interpretation of what's causing them or what their significance is?

A. Well, that's true. That's generally true.

Q. Your ultimate opinions in this case, I guess, are summarized under the conclusions in your report?

A. Yes, ma'am.

Q. All right. First conclusion, the ultrasound show classic postnatal brain complications of prematurity.

A. Yes, ma'am.

Q. You have described for me the evolution and the process that you see, stemming from a periventricular hemorrhagic venous infarction?

A. Yes, ma'am.

Q. Is that what you're referring to in that conclusion?

A. Yes. And, more importantly, the cystic PVL.

Q. Okay. Meaning the cystic PVL occurring on August 31st in this case?

A. First visualized on August 31st.

Q. First visualized on August 31st.

A. Correct.

Q. When did you come to the conclusion that there is evidence of periventricular hemorrhagic venous infarction?

A. It was a consideration of mine to explain, particularly after I read her opinion, after I read her letter where she was pointing to this -- is how to explain that. As you can see from the series of my notes in here, I could not explain that. And it's primarily after I read her opinion. So that may tell you that I must have read her opinion after I wrote this letter. Yeah.

Q. Okay. Did you see that cyst, though, that she refers to or that black spot --

A. That dot?

Q. That dot prior to reading her report and prior to reading her deposition?

A. Yes. And I lumped it together here on this as part of the grade 1 and grade 2 germinal matrix hemorrhage of mixed echogenicity. I was lumping all that together in the subependymal periventricular region. She, in her report, decided to isolate that out as cystic PVL. That's commonly how we consider when we see a grade 2 or larger subependymal hemorrhage that extends beyond the subependymal region like this does, the primary thinking about that -- you'll read it in the textbooks. It's a classic pathophysiology -- is the complication of grade 1 germinal matrix hemorrhage, some grades 2 also, is the venous obstruction with infarction and the hemorrhagic infarction. And they tend to describe it as periventricular rather than subependymal. And the reason they do is because it extends and the starts from the subependymal region out into the periventricular region. So that's where the debate starts. And it was that way for years. It was that way for years until we got it straightened out with animal models and neuropathology in these babies when -- you know, the postmortem

neuropathology when we discovered that vein was being obstructed. You could see it in the pathology on the section that caused the secondary hemorrhage related just to a grade 1. And so it's kind of misclassified from early on as a grade 4, when it doesn't have to be a lot of intraventricular hemorrhage with that to get that. You can get it with the grade 1.

Q. They're two separate processes?

A. No. They're probably the same. They're probably the same. But just like in this case here, where if that had persisted and become a big hole, what we call porencephaly, they may have said -- they may have classified this as a grade 4 later.

Q. Okay. Ultimately, what I'm trying to get to, is you described for me the basis of your conclusion No. 1?

A. Yes, ma'am.

Q. All right. No. 2, I think it's the same conclusion stated another way. The ultrasound show a classic textbook progressive evolution of postnatal periventricular neuromalacia.

A. Yes, ma'am.

Q. Okay. You described your basis for that. And one of the primary reasons for that is that initial cyst disappears; and then, on August 31st, we again see is cyst formation.

A. There and elsewhere. More importantly, elsewhere that's much more extensive than just that area, yes.

Q. The ultrasound findings are not consistent with the antepartum hemorrhage or antepartum PVL?

A. Yes. More probable than not that's my opinion. That's, you know, for most everything that we're seeing here. That means am I at 60 percent on that? Am I at 70 percent on that? I mean, there is 20 to 30 percent there that says there may be some overlap here with one part of this injury that we see that we're talking about. And I think the dispute or the difference between myself and Dr. Siegel is about that one area. This overall brain injury, more probable than not, as number 3 says, is not consistent with antepartum hemorrhage or antepartum PVL.

Q. But we've agreed earlier that it's possible that that cyst formation that we see could be possibly consistent with an antepartum injury?

A. That portion, yes.

Q. So what you're saying is not consistent is the PVL?

A. Correct.

Q. Okay. Do you think it's not even a possibility that the PVL that we see in this child could be related to an antepartum injury?

A. The vast majority of the PVL damage in this

brain, yes. Yes.

Q. Okay.

A. Yes. That one spot is a possible. I don't think it's cystic PVL. I told you what I think it is. So based on that, more probable than not, what I'm saying is what I state in No. 3, that this is most consistent with postnatal. And No. 3, to turn that around, it's more probable than not not consistent with antepartum hemorrhage or PVL.

Q. So you're saying the same thing, just kind of flipping them around?

A. Yes.

Q. Let me ask you this to avoid going through -- really there is three more films between 8/3 and 8/31. Have you described your interpretation of the 8/7, 8/15, and 8/24 film and the significance of your opinion in your report?

A. Yes, ma'am.

Q. What's the significance of the ventricles getting larger in the 8/7 film?

A. In the 8/7 film, probably the intraventricular hemorrhage as it expands and causes -- you're familiar with the term hydrocephalus?

Q. Right. That one I know.

A. That's why I make that statement in the first

paragraph. Where is that statement? One cannot use ventricular enlargement in the presence of intraventricular hemorrhage as an indicator of atrophy, meaning tissue loss, due to prior parenchymal injury or old parenchymal injury. I mean, you can get ventricular enlargement, obviously, from loss of brain tissue. But when you have a hemorrhage in there causing the hydrocephalus blocking the flow of CFS, causing those chambers to enlarge, you can't say anything with regard to that finding representing old injury with atrophy, in my view, my opinion.

Q. Is it your interpretation of the 8/7, 8/15, and 8/24 films that there are no cystic changes apparent on those ultrasound films?

A. Yes, ma'am. You said 8/7?

Q. 8/7.

A. 8/15?

Q. 8/15.

A. 8/24?

Q. 8/24.

A. Yes, ma'am.

Q. All right. So you see it on 8/3 and then not again until 8/31?

A. Correct.

Q. These -- in Paragraph 2 of your interpretations,

the periventricular basal ganglia and thalamic echoes consistent with edema.

A. Yes, ma'am.

Q. What's the significance of that?

A. Increased echoes that are representative of edema tells you you are in the so-called acute to subacute phase if we're talking about a mechanism such as hypoxia-ischemia. The acute to subacute phase is considered somewhere between -- depending on what modality that you use, is at least 24 hours up to about 7 days, acute to subacute.

Q. Okay. So as of August 7th, are you saying we are within 24 hours to seven days of the initial insult?

A. Yeah. That's probably fair. About one to seven.

Q. And then in Paragraph No. 3, referring to the 8/15 film, those echoes are now decreasing, meaning the swelling is subsiding. And that's, in your opinion, the natural progression of the process?

A. Yes. Usually starts coming down after about five to seven days. The edema definitely comes down between 10 and 14 to where it starts disappearing if you've had no more insults. I mean no more insults significant enough to cause more injury with, you know, more edema.

Q. Do you have an opinion in this case as to whether the PVL was the result of one large insult or many small

insults, or can we make that determination with the information here?

A. No. All you know is that it's a severe injury, so now you have to look at severity and duration. And by that, meaning severe, was it -- and duration, was it one episode with a very severe lowering of blood flow or oxygen; or was it a series, and the injury is accumulative? And then over what time period are we talking? I don't think imaging can tell us that, particularly with regard -- sometimes imaging in the term can suggest, but not in the preterm, I don't think.

Q. When we talk about timing in this deposition today, you've given me a lot of ranges.

A. That's right.

Q. -- which I understand is the nature of the signs. When you come to trial in this case, are you going to give anything more specific as to here is the date I believe the initial insult occurred?

A. No.

Q. You can't do -- science doesn't allow us to do that?

A. It does not, particularly with ultrasound.

Q. All right. Is there anything -- I'm just trying to do this rather than go through all the films -- that you feel you need to add to your findings of ultrasounds

that you have noted in your report?

A. No.

Q. Okay. The findings you noted here are what support your basis that this is -- that the findings are consistent with postnatal brain complications of prematurity?

A. Yes, ma'am.

Q. Let me just ask you a couple of questions about your expert work. Obviously, you were kind enough to give me that chronology. So that gives me most of the information.

Do you still garner about one-third of your income from doing medical/legal reviews?

A. Yes, ma'am.

Q. At one point reading a deposition, you were reviewing about three new cases a week?

A. Yes. Now it's about no more than three a month.

Q. No more than three a month. When did that change?

A. Probably the last year. Yeah.

Q. Any particular reason why it's decreased in frequency?

A. I'm doing more child abuse work and a little bit less medical/legal.

Q. How often do you testify in child abuse?

A. Oh, boy, almost monthly.

Q. Really? That's not good to hear.

A. That's not good to hear.

Q. The testimony here that's listed on Exhibit 2, is that both the child abuse cases and your medical/legal work or is this all medical/legal?

A. Both.

Q. Both.

A. But you'll find that 80 percent is medical/legal. But over the last two, three years, it's increasingly more child abuse work.

Q. And is there a way I can distinguish which ones would be child abuse versus which is medical/legal?

A. Yes, ma'am. Where it says, "state versus," or the name of a state versus.

Q. Gotcha. Gotcha.

Do you remember -- it looks like -- this is just a listing starting in 1993. What year did you first start doing medical/legal work?

A. Practically the day I got out of fellowship in 1977.

Q. Do you remember why it was you first decided to get involved?

A. I went back to Oklahoma, Oklahoma Children's. And I started getting calls from attorneys, mostly defense

attorneys in the beginning in the Oklahoma area. And then after that, I did mostly defense consults for maybe the nine or ten years I was there. And I wasn't really turning down plaintiffs to review things. It was just after a while, I would get a call. And my friends, who are defense attorneys, saying, "Yeah, you need to take both. You're doing this work and just showing your bias toward one side." And I've been doing, over the years, increasing plaintiff work. Now it's about 50/50 in terms of reviews. So I do both. Whoever calls me first. I don't screen the cases, which makes some of the initial phone conferences interesting. I don't know if the plaintiff or defense sent me this case.

Q. Do you enjoy doing this?

A. I'll tell you it is very important. And as a lot of my colleagues at Packard Children's, the neonatologists, and other colleagues before that do this work, we learn so much about brain injury and all the different factors involved and particularly with regard to quality improvement. You know, I'm not just talking about risk management. I'm talking about quality improvement. And I think many or most of the neonatologists where I work and the OB people do medical/legal work, both sides, same in Boston. And let me tell you, it's very, very important. You know, for physicians who turn their backs

on these types of issues and say, "They don't exist," and "I don't talk to lawyers," and, "Don't question me about how I practice medicine" -- or vice versa on the other side of it. It's very important. And that was impressed upon me early in my career by physicians around me and attorneys who say, "You know, you're very specialized in this area. We have a lot of people testifying who are GPs or general radiologists or people outside of imaging that don't really have expertise in this area that are giving opinions."

So the group of us in the Society -- in the American Society of pediatric neuroradiology, when we finally formed that, gosh, back in the early '90s, said, you know, we've got to do something about this. That's Tom Naidich, Bob Zimmerman, Jim Barkovich, Bill Ball, just about anybody you could name out there, Marvin Nelson. Most everybody, 10 to 12 of us, are involved to try to keep it at the level of expertise that it should be. So that's kind of how it's done. Oh, my gosh, what we learned from it is important for improving medicine, if -- you know.

Q. This chronology that you've given me, this is just the list of cases where you've testified, deposition or --

A. Yes.

Q. So there's other cases that you have reviewed and maybe given a report that wouldn't be listed here; correct?

A. That I never hear from again.

Q. Do you know how many times you reviewed a case for Mr. Becker or his law firm?

A. At least a half dozen, at least a half a dozen, maybe more.

Q. Other than Dr. Siegel, have you been provided the names of any other experts or people involved in this case?

A. No, ma'am.

Q. And other than the ultrasounds and the ultrasound interpretation in the discharge summary, you've reviewed no other medical records in this case?

A. That is correct.

Q. Is that your general policy? You just review the films, the interpretations, and maybe the discharge summary?

A. Yes, just like I do at work. I try my best not to be biased by that so I can give a fair opinion to our clinicians instead of saying, "Oh, that's right. You're right about that. That supports hypoxia-ischemia. Yeah. That's the way I read my report." That's the worst thing we can do in medicine. Because we know findings are often

nonspecific in a pattern of injury and timing. You have to consider other things.

"You guys better rule out infection."

Or, "You know, this is not quite typical of hypoxia-ischemia. This could be a metabolic disorder."

"Oh, yeah."

You know, they'll come down and argue with us. We think it's this. We think -- that's fine. This is what the imaging says. You've got to work on these other things.

Q. Do you know, was it Mr. Becker himself that called you from his office?

A. I doubt if he called. It's usually an e-mail from most people because I have no time to be on the phone talking to somebody who wants me to review a case. If they call me, I say, "Look, can't talk now. Busy on the clinical circuit. E-mail me." I do everything by e-mail.

Q. Do you retain the e-mails you do on these cases?

A. No. It's really rare. It would have to be an e-mail that had a report on it, for some reason, that I didn't have in any other form or something. It's very rare.

Q. Dr. Barnes, have we covered your opinions in this case?

A. Yes, ma'am.

Q. All right. So what you anticipate testifying to at trial, we have discussed today?

A. Yes, ma'am.

MS. REID: I don't think I have any further questions.

(Whereupon, Defendant's Exhibit 3 was marked for identification.)

(Whereupon, the March 8, 2007, deposition of PATRICK D. BARNES, M.D. ended at 3:21 p.m.)

_____

PATRICK D. BARNES, M.D.

C E R T I F I C A T E

I, DARCIE L. MOORE, duly authorized to administer oaths pursuant to Section 2093(b) of the California Code of Civil Procedure, do hereby certify that the witness in the foregoing deposition was administered an oath to testify the truth in the within-entitled cause; that said deposition was taken at the time and place therein cited; that testimony of said witness was reported by me and thereafter transcribed under my direction into typewriting; that the foregoing is a complete and accurate record of said testimony; and that the witness was given an opportunity to read and correct said deposition and to subscribe the same.

Should the signature of the witness not be affixed to the deposition, the witness shall not have availed himself/herself of the opportunity to sign, or the signature has been waived.

I further certify that I am not of counsel nor attorney for any of the parties in the foregoing deposition and caption named, nor in any way interested in the outcome of the cause named in said caption.

DATED: March 14, 2007.

DARCIE L. MOORE
CERTIFIED SHORTHAND REPORTER NO. 3143