COURT OF COMMON PLEAS

CUYAHOGA COUNTY, OHIO

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MATTHEW CHASE WAGONER, ETC. et al. : CASE NO. 497179

-vs-

MARK R. EVANS, M.D, et al.

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Oral deposition of ROBERT RYAN CLANCY, M.D., was taken pursuant to Notice, held at ZANARAS REPORTING & VIDEO, 1616 Walnut Street, Suite 300, commencing at 9:00 a.m., August 25, 2006, before Micheline Brown, License No. XI 00230800, Certified Shorthand Reporter and Notary Public, there being present:

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I N D E X

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E X H I B I T S

LITIGATION SUPPORT INDEX

DIRECTION TO WITNESS NOT TO ANSWER

REQUEST FOR PRODUCTION OF DOCUMENTS

STIPULATION

(It is hereby stipulated and agreed by and between counsel that, sealing, filing and certification are waived; and that all objections, except as to the form of the question, be reserved until the time of trial.)

ROBERT RYAN CLANCY, M.D., after having been first duly sworn, was examined and testified as follows:

BY MR. BECKER:

Q. Good morning, Doctor, would you state your full name for me, please?

A. My name is Dr. Robert Clancy.

Q. And what is your business address?

A. The Division of Neurology the Children's Hospital of Philadelphia, 34th Street and Civic Center Boulevard, Philadelphia, Pennsylvania 19104.

Q. Dr. Clancy, did you bring with you your complete file that you have on this case?

A. Yes, I did.

Q. If you would, sir, would you, just kind of, go through your file and tell me what medical records, in general, you have, depositions, et cetera?

A. I'm going to move to get to the stack.

Q. That's fine.

MR. BULLOCH: It's fairly voluminous, Mike

BY MR. BECKER:

Q. And, Doctor, while you're looking at that I gather from some old depositions of yours that, at least at one point, you had a tendency to make notes on the front of the depositions of the respective experts. Did you do that in this case?

A. Yes. In all of them.

MR. BECKER: Okay. And, John, can we get an agreement that we can have the front page of each deposition marked after -- photocopied and then marked as an exhibit after the depo?

MR. BULLOCH: Sure.

THE WITNESS: So I'll just read through this. I have my billing for the case. The first letter from Moscarino and Treu that lists the material. A copy of my report that you have, my Curriculum Vitae, and these are cover letters for Dr. Wilhem's deposition, Dr. Robert Darnall, old billings from couple years ago, additional records from Dr. Cahigh, EMS report, University of North Carolina and just correspondence about the trial coming up, cover letter about Dr. Adler's deposition, cover letter about

Dr. Barry Pressman's report. The announcement of this deposition. I have one E-mail from, well, from my secretary to call Mr. Ostra, an E-mail from Mr. Ostra. The report from Dr. Raymond Redline. I have notes from the medical records which are attached

BY MR. BECKER:

Q. Those are your notes?

A. Yes, sir.

Q. Okay.

A. Just from the chronology and things like that. Another E-mail from my secretary to call Mr. Bulloch. The cover letter for Dr. Jonathan Cronin's deposition, Dr. Cronin's report, a report from Dr. Hermansen, Dr. Adler and Dr. Redline, another E-mail, the medical chart of Dr. Cahigh, the cover letter for Dr. Hermansen's deposition, the home movies; I have a medical article regarding mitochondrial disorders, receipts from old bills. I have notes that I took when I re-reviewed the -- there was two volumes of home movies, the VHS tape said 2004, I think version I or number one, something like that. The second tape was home movies 2004 number two. Report from Dr. Pressman, notes from a volume I got recently that included the

time when Matthew was, I think, ten months and had suspected meningococcal infection.

Here is the depositions that I have the notes on. So I have Dr. Adler and there are notes on the front of that. Dr. Cronin, there's notes on the front of that, Dr. Bachman's deposition, there's notes on the front of that. Dr. Joshua Alexander, there is a few notes with that. Dr. Nelson, his recent deposition, there's notes on that. I have the deposition, I guess the first deposition of Dr. Wilhem, I don't have notes on that. I have -- I have a stick-um on page 41, just to look at that, and a stick-um on page 65. Here's the deposition of Dr. Hermansen, there's notes on that; Dr. Darnall. I have the mother's deposition. I have not read it. I have no notes on it. I have the deposition of Dr. Lilien with notes on it. These are medical records, Dr. Cahigh's, this volume is sort of all the material that related to his admission to University of North Carolina, Chapel Hill, between July 25, 2000 and August 2, 2000.

Now, the rest of the medical records so this is the Developmental Evaluation Center and these are from the Dosher Memorial Hospital, so these are duplicates I believe. This is, this is the volume for

the meningitis, July 2000. This was the notes from the Fairview Hospital when he was a newborn.

MR. BULLOCH: The records you mean?

THE WITNESS: The records. And this file is the Preliminary Service Coordination Summary Recommendation. This is a lot of his pediatric and follow-up stuff. Dr. Cahigh's records. This is the Wellcare Home Health records. These are his newborn records I guess that's Parma Hospital. And then I have a batch of outpatient records from North Carolina. So this is David Bachman's report, Dr. Rhoades, a GI, Joshua Alexander, Dave Wallis, Richard Southerlin, Tracy Irwin, therapy notes, neonatal developmental clinic follow-up, social service letters and lab work.

MR. BULLOCH: Again, Doctor, two times you said, "notes," by that you mean records?

THE WITNESS: No. These are all medical records. My handwritten notes are over here; okay?

BY MR. BECKER:

Q. How many pages are your handwritten notes?

A. Well, the notes from the medical records I

think are about six pages or so and they're stapled together and then I have, just, additional notes from, again, like, looking at the movies or the home movies and some other things.

MR. BULLOCH: Doctor, not to belabor the point but I know Mr. Becker is going to ask for copies of these so you need to refer to each one and tell him how many pages, exactly, they are.

THE WITNESS: These are my original handwritten notes from reviewing the medical records and they are six pages long. I have, again, my list for the billing time.

MR. BULLOCH: And that's how many pages?

THE WITNESS: This is one page for the billing notes. I have three pages of notes from the review, there was actually four video tapes. there was the two home movies and then there was one, the professional tape, the Day in the Life of Matthew, and then there was a third tape but it was really just an edited piece of the home movies that were put together.

So anyhow these are my notes on that, that's three pages. One page of notes just reviewing the medical records from the episode of

meningococcal infection in July of -- or whenever that was, July 2000.

BY MR. BECKER:

Q. I should ask you, Doctor, how is your handwriting?

A. Not bad.

MR. BECKER: John, can we get an agreement, so we don't make this deposition four hours long, that if I can't read anything we can get a subsequent interpretation?

MR. BULLOCH: Yeah, I mean, Mike, I'm sitting here and I can read it; I'm sure you won't have any trouble reading it, but, sure. I'm sure the doctor will be happy to interpret his notes for you if you need him to.

MR. BECKER: Okay.

(Whereupon, Exhibit 1, Exhibit 2, Exhibit 3, Exhibit 4A, Exhibit 4B, Exhibit 4C, Exhibit 5A, Exhibit 5B, Exhibit 5C, Exhibit 5D, Exhibit 5E, and Exhibit 5F were marked for identification.)

MR. BULLOCH: Okay, Mike, for the record, we have Exhibit 1, Exhibit 2, Exhibit 3, Exhibit 4A through 4C and Exhibit 5A through 5F.

BY MR. BECKER:

Q. And, Doctor, I do not need you to go through each page of the exhibit, but as to Exhibit 1, tell me what that is just for the record, for two, et etcetera?

A. Sure. So Exhibit 1 are my notes regarding hospitalization for the meningococcal infection. Number 2 is my billing notes for the case. Three is a, what I call consideration, sort of like summary of the time line, the facts. Four is the notes on the home movies and the professional Day in the Life, and then the composite film, and then Exhibit 5 are the six pages of notes that I took when I originally reviewed the case couple years ago from the medical records.

MR. BULLOCH: And just for the record these were marked in no specific order of priority. They were just done between myself and the court reporter.

MR. BECKER: That's fine.

BY MR. BECKER:

Q. Doctor, I know you've been deposed before but just to be fair with you I want to review the ground rules.

This is a question and answer session under oath. It's important you understand the question that I

pose. If, for any reason, the question doesn't make sense to you or is inartfully phrased, I want you to stop me and tell me so and I would be most pleased to attempt to rephrase or restate the question. Fair enough?

A. Yes.

Q. However, unless you indicate otherwise to me I'm going to assume that you fully understood the question that has been posed and you are giving me your best and complete answer today. Fair enough?

A. Yes.

Q. Is it safe for me to assume that you did not look at the EEG strips themselves in this case, the original strips?

A. That's correct. I only read the reports.

Q. Did you ask for the EEG strips for your review?

A. No, I didn't.

Q. Did you look at the head films?

A. Yes, I did.

Q. When did you look at the head films?

A. Yesterday.

Q. For the first time?

A. Yes.

Q. Did you ask for the head films prior to yesterday?

A. Actually I did and -- but only recently and since I was meeting with Mr. Bulloch yesterday he agreed just to bring them for me to look at them then.

Q. I noticed in some of your depositions, Doctor, that you have a practice of reviewing head films with Bob Zimmerman in your department or at CHOP; correct?

A. Yes.

Q. Did you by chance review these films with him?

A. No, I didn't.

Q. Have you discussed these films with Bob Zimmerman?

A. No.

Q. Have you discussed your opinions with anyone on this case other than counsel for Fairview General Hospital?

A. No, I have not.

Q. Why did you ask for the films?

A. Just to see what they looked like, really, because there were so many different interpretations of them.

Q. Is your interpretation of these films noted in any of the notes we've marked as exhibits?

A. No.

Q. What is your opinion of what the films reflect?

A. Well, first of all, I've looked at one film and that was the brain MRI that was obtained in the -- right after birth. So that was the film taken September 8, 1999, because I thought that was the most relevant study with respect to, you know, the neonatal course.

Q. Fair enough. What was your interpretation if you have one?

A. Well, I certainly can see the lesions on the study. I'm not in a position to say if they're early neonatal fibers or coagulation necrosis with PVL but I can see what other people are pointing to. I also see that the basal gangla areas appear normal on those studies, which is obviously a relevant consideration for this child because of the type of CP that he has.

Q. We'll talk about that. Let me ask you straight up, Doctor, since it's not noted in your report, do you hold an opinion, within a reasonable degree of medical probability, as to the cause and

mechanism leading to Matthew's brain injury?

MR. BULLOCH: Objection

THE WITNESS: I do.

BY MR. BECKER:

Q. Okay. Is that opinion reflected in your report; maybe I missed it?

A. Well, I do discuss the lactic acidosis and concerns for a metabolic condition. Really, just after the deposition of Dr. Wilhem, who talks about the current condition of Matthew, that he is only taking a couple steps, and then, again, looking back at the home movies, it's really pretty clear that he's deteriorating. He's not running a stable course or, you know, gradual improvement as you expect with CP. And so putting together, again, the type of CP and the lactic acidosis, from my perspective, new knowledge, this is not a static lesion but a progressive lesion, then, that leads me to think that he has a metabolic condition such as mitochondrial disorder.

Q. All right. So that was kind of a long answer and I think you were giving me your explanation as to why that opinion is not contained in your report. Specifically, that you discern that this child, taking what the life planner says, that you're presuming that

the child has a progressive motor disease and that would cause you, because you're interpreting her comments as a progressive motor disease, you're presuming, then, that it, likely, is some type of a disorder, mitochondrial disorder, that you just outlined?

MR. BULLOCH: Objection. Mike, I don't believe he said this was a new opinion. I need to object to that and I need to object because I think there is, like, six parts to that question.

BY MR. BECKER:

Q. Well, Doctor, is your opinion about the mitochondrial disorder reflected anywhere on your report?

A. No. The word "mitochondrial disorder" does not show up but the concerns about the consistently elevated lactic acid, the mixed CP type, the fact that he has a hearing loss and so forth, because at the time I wrote this report in October 2004 it was my understanding then, again, that he had a static lesion, but I think the evidence now, is that it's not static, it's been progressive and that's why I'm refining this opinion today.

Q. Well, what evidence is it that it is progressive and not static, other than what Cynthia

Wilhem says?

A. Well, first of all, I did, after I read Wilhem's deposition, I looked back through the medical records, and let me just stand up and find the one because I think it's important.

So this is one of the medical records, it looks like it was sent to me February 9, 2006 and it's from the Developmental Center mostly. There is some insurance information, communication notes, skilled notes, but then we go to the physical -- physical therapist and I'm looking at a report, this is from Wellcare Home Health, Pediatric Physical Therapy Evaluation. And it says, July 20, 2004 but it's scratched out. Anyhow it's the PT report from Matthew Wagoner, and he's four years ten months, and under caregiver's concern it says, "Patient has regressed in motor skills". And then further along that line, and this is from -- the name of the therapist is Cathy Poulos, P-O-U-L-O-S, physical therapist, "he can ascend and descend stairs independently and demonstrated independency in going up and down one step in-home that divides two rooms. He cannot jump or hop. He attempts to run but it is a fast walk pattern."

So that, that description by his therapist,

you know, combined with the fact that the mother is saying he is regressing and actually in the Day in the Life movie it shows him requiring assistance. I remember there are little plates or something on the floor. He had to be held up to step over those and then Dr. Wilhem saying that she had seen him and then did a telephone interview of the mother more recently, I don't remember the date, saying that he couldn't take more than two or three steps. You know, when you look at the movie and then look at that they're describing two very different children.

Q. Well, is -- are you assuming that the mom is referring to motor regression or simply not advancing in academics?

A. Well, this is the quote, it says, "caregiver concerns, 'patient has regressed in motor skills.'"

Q. You're reading me a physical therapist note?

A. Yes, sir.

Q. Okay. My question, sir, was relative to the mom. What did she report, if any, relative to motor skills or was she specific?

A. Right. Just so; maybe I'm not making

myself clear. The mother is the caregiver. The caregiver is the child, the mother, and so it's the mother saying that the patient has regressed in the motor skills.

Q. Well, has any doctor documented, any physician documented that this child has regressed in motor skills?

A. Not that I know of. Again, the only way it was apparent to me was by really looking at the home videos because they're very telling. I mean, I know you've seen them and, you know, he's outside with the swings. He's got a very lurchy, you know, he looks very off balance but he's pretty much all over the yard. There's a scene when they're out by the, the red pickup truck and a basketball thing and he's in and out of that and to go from that which you can clearly see that he's abnormal. There is no doubt about that. You can clearly see he is abnormal, but he is walking without any assistive devices, you know, on rough terrain, he's outside. To go from that to, like, he falls after two or three steps, that's a big difference.

Q. This is difficult for me, Doctor, but I'm going to ask you to assume, for the purpose of most of my questions here, that this child, since I have seen a

very recent video of him, there's no difference from my perspective and there's been no major difference in the way this child moves around. If you saw a video today that shows this child just as active as he was in the Day in the Life would you retract that opinion?

MR. BULLOCH: Well, Mike, I'm going to have to object because number one, we have repeatedly asked for additional medical records. The last medical records we know we received from your office is 2004. You're under duty to supplement your discovery responses, and those films were obtained through discovery. We have not seen this film. So to the extent that you've got a late produced film, I'm going to object to its production at trial. Go head, Doctor, if you can remember what his question was.

THE WITNESS: Well, I do remember the question. So the diagnosis of a mitochondrial disorder is on fermise ground or at least it's the more classic presentation when there's deterioration. And again as a neurologist we always, sort of, have our radar out for these things. So the one article I brought is a description of, you know, all right, we have

experience with kids with mitochondrial disease and basically it says, yes, you know, the classic picture there is things like extrapyramidal CP, deafness, regression, lactic acidosis but in the families they show, there are children that behave like, sort of, conventional CP that they're static. So that they look like they have a lesion. It doesn't particularly evolve over time.

So even if his condition is static, it's still consistent with the mitochondrial disorder and, again, the lactic acidosis numbers are very -- I think they're very, important, coupled, again, with the deafness, you know, the neuro behavioral issues the child has, because when you put that all together that's mostly consistent with the mitochondrial disease and that really needs to be pursued in Matthew.

BY MR. BECKER:

Q. Well, have any of his doctors in North Carolina made that diagnosis?

A. The closest was Dr. Bachman who wrote something like, I forget the exact words, persistent lactic acidosis. I think he has a little note, I need

to pursue this, and I don't really know what happened to that at that point. I don't know if it's been pursued and I don't know, actually know, if he's been back to see Dr. Bachman for that matter.

MR. BECKER: Doctor, why don't we mark as Exhibit 6 this article that you brought with you. You only brought one article, mitochondrial disorder?

THE WITNESS: Yes, sir. So just for the record, the title is Multiple Presentation of Mitochondrial Disorders. It's from the Archives of Diseases in Childhood 1999, Volume 81, pages 209 to 215 and the first doctor's name is Nissenkorn, N-I-S-S-E-N-K-O-R-N.

(Whereupon, Exhibit 6 was marked for identification by the court reporter.)

BY MR. BECKER:

Q. Doctor, is that the only medical journal article that you brought with you today?

A. Yes.

Q. Now, are you able to rule out that Matthew's RDS and its complications including pneumothrosis is the etiology for his brain injury?

A. That's correct. I did not consider his

respiratory distress and pneumothrosis to be the cause of any of his current problems.

Q. You are able to rule that out?

A. Yes.

Q. And the basis that you're ruling it out is what, Doctor?

A. Well, first of all, if this is a quote "asphyxia type injury" and by asphyxia I'll take it to mean either hypoxic, ischemia, loss of profusion or combination of those. And taking this child's gestational age to be 35 weeks. We have a lot of experience on how children should behave acutely if they've been exposed to an asphyxial event that's powerful enough to actually cause damage, right then and there, severe enough to lead to a permanent disability.

Now, I recognize that if it's a very, very small preemie they may not show acute neurological signs. They may not show multisystem malfunction and so forth because it's a different biology at that point. But certainly by 34 weeks that if the child has cardiac arrest or pneumothorax or birth asphyxia or any other mechanism that's going to be quote "asphyxiating" and yet severe enough to cause permanent damage, then we really need to see acute neurological signs right then

and there. Not days later or weeks later, but right when the pneumothorax happened or the hypoxia was documented.

Q. Are you aware that any experts on behalf of the Plaintiffs have asserted that this child was asphyxiated?

A. Well, again, I'm not saying birth asphyxia. I don't mean at the moment of birth but you know if a baby --

Q. Excuse me, Doctor.

MR. BULLOCH: Mike, let him finish his answer.

MR. BECKER: John -- and I will let him finish but I think we ought to go back and work with some definitions because I want to make sure we're on the same page.

BY MR. BECKER:

Q. So let's start with some definitions Doctor, and maybe I'm using the wrong words in my questions. What does asphyxia mean to you?

A. Right. Asphyxia refers to an embarrassment or interruption of either oxygenation, tissue oxygenation, profusion, or both, and, again, I didn't mean to get you -- I know when we hear the word

asphyxia we always think of in the delivery room. I don't mean that at all. But realize that if someone falls into a swimming pool and they're drowning, that's a type of asphyxia; if a baby gets wrapped up in a bag and can't breathe, that's a form of asphyxia. So what I'm saying when I use the word asphyxia here has nothing to do with labor and delivery in the OR or things like that. But, you know, the issue that's on the table is that he had a pneumothorax and what did that do to his ability to deliver oxygen and blood to his brain. So I'm using asphyxia to describe, like, the oxygenation, profusion aspects. And maybe I should stay away from the word because most people immediately hear birth put the word birth in front of asphyxia. So I'll stay away from that word.

Q. What does hypoxia mean?

A. Well, if it's in reference to the blood stream it means a low oxygen reading in the blood, hypoxemia. And on the other hand, if you're referring to the oxygenation of the tissue, that's tissue hypoxia, a lack of oxygen in the tissue or lower than normal oxygen in the tissue.

Q. And what does ischemia mean?

A. Ischemia is a loss of profusion or the

actual flow of blood through the tissues of any part of the body.

Q. What does HIE mean?

A. HIE is actually a clinical syndrome of -- the operating word there is encephalopathy. So there is something pathological with the brain, encephalopathy, and the clinical signs of the encephalopathy are due to some combination of hypoxia and ischemia.

Q. And what is an EEG?

A. The EEG is the electroencephalogram or a brain wave test.

Q. And what is a seizure?

A. A clinical seizure is a sudden abnormal parasedral attack that is brought on by a temporary electrical disturbance in the cortex of the brain.

Q. And what is something called subclinical or occult seizure?

A. An occult seizure refers to the phenomenon of a child having a seizure on the EEG but for whatever reason doesn't provoke a visible clinical seizure in the child or the adult for that matter.

Q. And is that the same thing as subclinical, occult, one in the same, or is there something different?

A. No. I consider them to be the same.

Q. Is it true that 80 percent of seizures are subclinical or occult?

A. That sounds about right to me. That's about what my experience is.

Q. And it true for -- to gain an EEG confirmation of a seizure it should be done very close to the acute event causing the brain injury?

A. Well, if you're looking for seizures it should be as close to the time that you're actually, as you say, suspecting the clinical seizures. So remember that the concept is that the child did something clinical to make the doctors worry. I think the baby might be having a seizure. So then they call the EEG people in and they do the EEG and they realize that, sure enough, not only is the child having seizures, but we're only detecting, you know, 20 percent of the actual, 20 percent of the actual seizures. So that doesn't mean that if you walk up to a healthy baby and put an EEG on that they are, like, closet seizure kids or they're secretly having seizures. These kids that the EEG was done for a purpose because they have signs of HIE or it could be, you know, something other than that. It could be meningitis or hemorrhage but

something triggered the concern for seizures in the first place. Then you realize, again, you are only seeing a small fraction of them.

Q. The point of the question, Doctor, was, that if you -- your best chance of getting the EEG confirmation of a seizure is to have it done in a time that's very close to when the acute event causes the seizure, correct?

A. I do agree with that.

Q. Let's talk about the concept of autoregulation. What does that mean, Doctor?

A. Autoregulation refers to the ability of the nervous system to adjust its own cerebral blood flow to maintain it relatively constant, even though the blood pressure can fluctuate from low values to high values.

Q. What are some things that can compromise autoregulation?

A. Well, marked prematurity can certainly do it. Sepsis, I suppose there are some drugs that can do it. Children who do have HIV are, don't have the wherewithal to regulate their own blood supply. So there is a list of serious causes.

Q. How about hypoxemia from RDS, can that

compromise autoregulation?

A. Well, it depends on what degree of hypoxemia. I don't know that a modest amount of hypoxemia does. It's difficult to actually measure these things in humans. For example, when they have been done they've been done with things like Zion, radioactive Zion, blood flow. So those types of studies are not being done anymore.

Q. How about newborns that are on mechanical ventilation, are they at risk of losing autoregulation?

A. As far as I know if it's just a matter of, if their brains are normal and they're simply on mechanical ventilation I'm not, personally, aware that that stops autoregulation.

Q. What does the phrase passive pressure cerebral circulation mean to you?

A. The passive pressure refers to the behavior of a child that has lost the autoregulation. So in this place cerebral blood flow will mirror the profusion so if the blood pressure goes up, then, passively, the circulation of the brain goes up and you run the risk of actually creating a hemorrhage because there is too much blood flowing through the brain. On the other hand, if the blood pressure falls or the

profusion pressure falls then there is less blood being delivered to the brain and you run the risk of an ischemic injury.

Q. Now, I don't want to confuse things, Doctor, but you recognize that one can have a brain injury from either hypoxia or ischemia without a true classic asphyxia, correct?

A. If you're talking about a real small preemie that's true. But, again, when I originally -- you asked me about asphyxia, I said it's a combination of either hypoxia or ischemia or something together. So I don't know how I can answer the question, you know, if you're hypoxic or ischemic to me that's asphyxia. Not birth asphyxia but, you know, from a cardiac arrest or smothering or anything else.

Q. Well, just, hypothetically, I guess, because your use of, what I would respectfully say or liberal use of the word asphyxia if you have a newborn that is on mechanical ventilation and has a sudden bout of bradycardia for 15 or 20 minutes but is not -- doesn't go on to multisystem organ injury or, you know, major lactic acidosis, with a base deficit, that kind of thing, you recognize that that condition can cause brain injury?

A. No. See I would not accept that. In other words I don't see, you know, I don't think it matters if the child is five days old or one day old, or is even, in fact, freshly delivered from the labor and delivery room, but if that bradycardia was, you know, severe enough and prolonged enough to damage the brain then I do expect the majority of cases, not a hundred percent, but the majority of cases will have the multisystem but if the brain is acutely injured there has to be signs of injury.

By what magic do you permanently damage somebody and yet they are alert and active and have good tone and all those things? That's not an acceptable proposition in my mind.

Q. Would you expect to see an abnormal MRI at, roughly, eight days old if there was a mitochondrial cause?

A. Well, first of all, there can be and this is described a little bit in this paper. Some of the kids have white matter abnormalities. And I'm not saying that those lesions on the MRI scan are from that. Because, you know, as you know that, you know, the whole, the whole pneumothorax and chest tubes and the low oxygen reads were on August 25th and the child

looked okay clinically during that time, but, then, later there were other concerns, and I think very legitimate concerns, for infection when the child behaved later, I don't remember exactly, seven or eight or nine days, was less active and described as hypotonic and so forth and then they were worried about the infection. That's when they did the spinal tap and the culture of the tubes and the white blood cell counts that they didn't like. Because even though I realize they did not confirm any particular virus or bacteria or whatever, clinically they were very worried about sepsis. So that MRI finding, you know, from '98, '99 that could have been related to that. It could have been related to other things, I'm not really sure, but, again, the main reason I wanted to see the scan was to really look at the basal ganglia because that's a big part of his disability, this chondroid cytosis. I see nothing wrong with the basal ganglia and I don't think anyone described any basal ganglia lesion in the later scans. I have not looked at them myself but I wanted to at least look at the one that was done right after birth.

Q. Do you understand, Doctor, that the basal ganglia, the thalami that can be compromised secondarily

as a result of a primary white matter injury. Did you learn that in your training?

A. No. I must have been sick that day.

Q. All right. Did you learn in your training, Doctor, that for one to pick up, that you can have a thalami injury without radiographic support. In other words, there can be a microstructural injury to the thalami without MRI support? Did you learn that?

A. Well, you know, you could certainly --so let's pretend that a person has an MRI scan and they say we looked at the basal ganglia and they looked fine. And then the guy or the girl, whatever, dies and they do, you know, their post-morbid examination and they'll say, you know, we found microscopic findings in there. I'm sure that's happened, but the point is that if you have a clinically relevant finding, and, again, Matthew Wagoner's chondroid cytosis is, by no means, subtle, it's florid, then how does a microscopic thing affect, you know, that's not even visible under your best scanners and you have multiple scans and multiple people, you know, the community radiologist, your radiologist, his radiologist, no one is seeing anything there. I don't think, I don't think that's acceptable. On the other hand, we do know that kids that have

dysfunction, like kids with mitochondrial disorders, that's one of their characteristic types of, you know, disability. And they very often don't have, you know, they have functional disturbances of the basal ganglia but they don't have like, you know, scars and holes necessarily. Some of them actually, over time, will develop that. But they can be dysfunctional without being abnormal structurally.

Q. When one thinks of extrapyramidal is that pronounced correctly?

A. Extrapyramidal.

Q. Okay. You think of ball bar signs, you look for ball bar signs?

A. Children who have extrapyramidal CP, for whatever reason, often have ball bar signs, dysarthria, drooling, trouble swallowing, sometimes, I don't know if you consider reflex to be, sort of, a neurological sign of that context, but they seem to go hand and hand together.

Q. Which of those, if any, does Matthew have?

A. Well, he has -- you mean ball bar signs?

Q. Yes.

A. He has dysarthria, I really don't know about the drooling. I couldn't tell. I don't remember

reading about it. But the dysarthria is probably the most prominent one.

Q. What does that mean?

A. That he has difficulty with generating distinct speech. It's not what he says but the way he says it. The way he articulates his words.

Q. You're not suggesting that one can't have a mixed CP from hypoxic, are you?

A. No. As a matter of fact, that's a known combination in the setting of events such as terminal bradycardia. So let's say a woman is in labor and the heart rate suddenly goes down to 60 or below for an extended period of time, then they will have a pattern of brain injury that includes both the cortex, the related white matter, the deep gray structures and they are clearly encephalopathic. They will have seizures. They don't feed. They are hypotonic and when all is said and done they will have -- they may have a mixed CP picture with elements of spasticity and extrapyramidal features and dysarthria and that's certainly known but, again, it's majorly catastrophic, you know, acute profound asphyxia pictures.

Q. Well, you can get a mixed CP from, just speaking generally now, you can have a partial prolonged

hypoxia and then superimposed on that would be a sudden complete anoxia situation, correct?

A. Right, but again just to answer your original question. If a child only had the acute profound because the fact, again, we always focus on the deep gray structures and the corpus callosum and so forth but remember, they also have lesions in the perietal cortex, the white matter between the basal ganglia and the perietal cortex. So that's even without prolonged partial those kids can have a mixture because they have a combination of pyramidal traction, extrapyramidal but yes, if there is a partial prolonged asphyxia and then it's complicated by a terminal bradycardia that would be another way of getting a mixed CP pattern.

Q. You would agree, Doctor, that radiographically the way HIE injuries appear are typical global, bilateral and symmetrical?

A. I'm sorry, I wasn't listening. Could you repeat.

Q. HIE, for an HIE radiograph picture they typical appear as global, bilateral, symmetrical?

A. I think in general that's true.

Q. Can you tell me what the potential

deleterious or negative effects of pneumothorsis or bilateral pneumothorsis would be on a child with moderate RDS?

MR. BULLOCH: Objection.

THE WITNESS: Well, they embarrass the circulation. They introduce an element of respiratory compromise so that the lungs can't aerate as well. You can't blow off the CO2 as well. You can't oxygenate as well and if they are large and unattended they can affect the return of blood into the heart.

BY MR. BECKER:

Q. And if it affects the return of blood into the heart how will that impact the child's profusion to the brain?

A. Well, it has to do that through general blood pressure mechanisms. I mean, when the blood leaves the heart it's not, you know, it's just going up the aorta. Some is going to go to the arms and the body and the heart itself and some is going to the brain. So the segregate measure for that is still in the blood pressure and, for example, the color of the baby's face. So if there is a lack of oxygen going to the head, including to the face, the, you know, the baby

will desaturate and that's really what the cyanosis is, is when you have too much of deoxygenated hemoglobin. So, theoretically, it can be very serious, but it has to do its harm through measurable effects on blood pressure, oxygenation and so forth.

Q. So if it was severe it could cause the heart -- impact the function of the heart and drop in blood pressure?

A. Yes.

Q. And if there is a drop in blood pressure and if, in fact, there already may be a loss of autoregulation, what is the impact of that on the cerebral profusion to the brain?

A. The cerebral profusion will reduce. If, again this is hypothetical, if the child has lost autoregulation, for whatever reason, and there is a significant loss in blood pressure that is driving the blood to the head then, because it's a pressure passive system there will be less profusion of the brain.

Q. And that can cause an ischemic permanent brain injury?

A. Right. The pattern would be, the pattern would be an ischemia pattern. I mean the radiologists have to decide exactly -- what exactly an ischemic

pattern is. But that would be the principal mover of the damage, would be the ischemia. So that's the pattern you would look for.

Q. Doctor, I'm going read some things to you from Volpe. I want to know if you agree or disagree with them. Okay. I'm assuming you have Volpe in your library?

A. Yes, I do.

Q. And is Volpe a neurology in newborns -- the leading textbook in the subspecialty field of newborn neurology?

A. I think it probably is, yes.

Q. The perinatal brain can be deprived of oxygen by two major pathogenic mechanisms: Hypoxemia, which is diminished amount of oxygen in the blood supply and ischemia, which is diminished amount of blood profusion to the brain. Do you agree?

A. Yes.

Q. Ischemia is the more important of these two forms of oxygen deprivation. Do you agree?

A. I mean, they're both important. I don't think either one of them are trivial.

Q. Marked hypoxemia is required to produce serious changes in brain energy state in the neonatal

adult (ph). Do you agree?

A. Yes.

Q. The principal biochemical mechanism of cell death with hypoxemia, ischemia and asphyxia are, presumably, very similar, if not identical, and are initiated by oxygen deprivation. Do you agree?

A. If you're reading it accurately I will agree with that.

MR. BULLOCH: Mike, I'm going to object to you reading this out of a textbook without putting a copy in front of the doctor so that he can see the entire reference. I don't know if you're picking and choosing. If you're reading one paragraph out of five paragraphs.

BY MR. BECKER:

Q. Doctor, I'm reading from Chapter 6 on Volpe entitled, Hypoxic-Ischemic, Encephalopathy Biochemical and Physiological Aspects. Alterations and cerebral blood flow are of prime importance for understanding a neuropathological and neurologic consequences of all varieties of parinatal asphyxia and hypoxic-ischemic insults. Do you agree?

MR. BULLOCH: Objection.

THE WITNESS: Yes, I do

BY MR. BECKER:

Q. And, by the way, was Matthew a preterm child?

A. Yes. I think, you know, I don't know there is any consensus on this but I considered him to be, basically, 35, maybe 36 weeks, so that's preterm. Term is 37 or higher.

Q. Now, did you notice that there is a Dubowitz assessment of him at 34 weeks?

A. I did notice that.

Q. What weight do you give to a Dubowitz assessment?

A. You know, I don't do them anymore so I can't -- I think he was a near-term baby and as I mentioned, even for 34-week baby, that's still mature enough that if you have brain damaging asphyxia you're required to see acute encephalopathy signs.

Q. The margin of safety, at least in the preterm fetus, and at least to a lesser extent in the term fetus, is small at the lower end of the autoregulation curve and points to the vulnerability to ischemic brain injury with modest hypotension, particularly in the preterm animal. Do you agree.

MR. BULLOCH: Objection

THE WITNESS: Right, but remember what you said there, that you're at the bottom of the profusion curve. If you're at the bottom of the profusion curve that means, like, you're teetering on the edge there and then further reductions can be serious. So that's kind of hard to disagree with that.

BY MR. BECKER:

Q. The impairment of autoregulation requires only a 20-minute exposure to hypoxia and autoregulation will not recover until seven hours after restoration of neuromoxia?

MR. BULLOCH: Objection.

THE WITNESS: I don't know if is he talking about laboratory animals or human beings there.

BY MR. BECKER:

Q. I think he's talking about neonatal animals which includes laboratory animals.

MR. BULLOCH: Objection.

THE WITNESS: Well, you know, it may be true, you know, in laboratory animals that they measured them and, again, I don't know how severe the hypoxia -- or anything like that, but I think, probably, data that's not precise is not

describing humans.

BY MR. BECKER:

Q. Do you agree, Doctor, that merely because hypoxia or hypoxemia is addressed that it is likely that the impairment of autoregulation takes some time in the human to recover, if you know?

A. I don't know. Again, it's very hard to actually measure these things and that's the value of the animal models, to at least give you some concept that these symptoms don't turn on and off automatically, but I really wouldn't have no idea in the human what the true time course is for losing it or regaining it.

Q. Volpe comments that a pressure passive state of cerebral circulation was observed both in seriously asphyxiated full-term infants and mechanically ventilated preterm infants; do you agree or disagree?

MR. BULLOCH: Objection

THE WITNESS: Right, and, again, though I think you need to be very careful when he says, "preterm," because I'm familiar with his -- he's looking at this thing that, I forget the term he used, like the jitter and the cerebral profusion pressure. Those were really, at least I recall them to be quite young babies, not 35-weekers

but, I don't know, 30-week or something like that.

BY MR. BECKER:

Q. The neuropathol -- I'm up to Chapter 8 in Volpe. The neuropathological features of neonatal and hypoxic-ischemic encephalopathy vary considerably with gestational age of the infant, the nature of the insult, the type of interventions. Do you agree?

MR. BULLOCH: Objection

THE WITNESS: Yes.

BY MR. BECKER:

Q. Of particular importance in the genesis of impaired cerebral blood flow and, thereby, cerebral ischemia, is an impairment of cerebral vascular regulation in the subset of premature infants who are mechanically ventilated, often clinically unstable and a a clear exhibit of pressure passive cerebral circulation. Do you agree?

MR. BULLOCH: Objection.

THE WITNESS: I do but, again, it's important to know exactly the population of when he says pre-term how premature.

BY MR. BECKER:

Q. He goes on to state, the potential reasons

for pressure passive cerebral circulation include hypercardia or hypoxemia related to respiratory disease. Do you agree or disagree?

MR. BULLOCH: Objection

THE WITNESS: I think that's true.

BY MR. BECKER:

Q. premature infants with pressure passive cerebral circulation are at higher risk for development of ischemic white matter injury. Do you agree?

MR. BULLOCH: Objection

THE WITNESS: Yes.

BY MR. BECKER:

Q. Even in the presence of intact cerebral vascular autoregulation marks cerebral basal restriction or systemic hypertension to the natural impairment of cerebral blood flow to the cerebral blood vascular zones resulting in cerebral white matter injury. Do you agree?

MR. BULLOCH: Objection.

THE WITNESS: Yes.

BY MR. BECKER:

Q. I'm now up to Chapter 9 in Volpe. Let me ask you, Doctor, does Volpe say that you need asphyxia in any of his texts in either an hypoxemia or ischemic

process?

MR. BULLOCH: Objection. Mike, come on, you're asking him what Volpe has written in his entire lifetime now.

MR. BECKER: I'm asking if he knows whether Volpe agrees with him on this proposition that we need asphyxia?

MR. BULLOCH: Objection. I don't think he ever said that, but go on.

THE WITNESS: I actually don't understand the question. It was similar to your other question, like, can you have asphyxia without having hypoxia ischemia. In my mind asphyxia is either hypoxia or ischemia or a combination of them. So I just don't really understand the concept that you're asking.

BY MR. BECKER:

Q. Fair enough. Chapter 9 is entitled, "Hypoxic-ischemic Encephalopathy Clinical Aspects." Do you agree with, that hypoxemia leads to brain injury principally by causing myocardial disturbance and loss of cerebral vascular autoregulation with ischemia the major consequence?

MR. BULLOCH: Objection.

THE WITNESS: I think, you know, that sentence was probably read properly and it sounds reasonable.

BY MR. BECKER:

Q. The major causes or clinical settings for serious hypoxemia include postnatal respiratory insufficiency, secondary to severe RDS. Do you agree?

MR. BULLOCH: Objection

THE WITNESS: Severe RDS can cause bad changes in O2 and CO2 but Matthew's wasn't severe.

BY MR. BECKER:

Q. The major causes of serious ischemia is marked hypoxemia of any cause. Do you agree?

A. Marked hypoxemia is not necessarily good.

Q. It should be noted that the majority of infants who experience interrater hypoxic-ischemic insults do not experience overt neonatal neurologic features or subsequent neurological evidence of brain injury. Do you agree?

MR. BULLOCH: Objection

THE WITNESS: I would have to look at that whole context before agreeing with that. I just don't know what the context is. Are you talking

about modest, non-damaging, hypoxia. Obviously, you know, people lose track of the fact that if you do a venous a puncture on a fetus and actually measure their oxygen level it's, like, 30, because of their fetal blood and that's hypoxia by our usual definition, but, you know, we've all been there. All of us have gone through that low oxygen fetal stage. So I don't know that it is relevant to this case.

BY MR. BECKER:

Q. Periventricular white matter injury may disturb subsequent cerebral cortical development. Do you disagree with that?

MR. BULLOCH: Objection. Relevance.

THE WITNESS: No. I think that's probably true.

BY MR. BECKER:

Q. Well, what would you consider, what part of the brain would you consider the cerebral cortical area?

A. Well, all of the surface of the brain is the cortex. So, you know, the frontal lobes, the gray matter of the frontal lobes and the temporal lobes, parietal, occipital and so forth.

Q. Hypoxemia may lead to the disturbance of cerebral vascular autoregulation and, as a consequence, a passive pressure circulation. Do you agree?

MR. BULLOCH: Objection

THE WITNESS: Yes.

BY MR. BECKER:

Q. Under such circumstances the infant is vulnerable to superimposed ischemic cerebral injury with only modest decrease in arterial blood pressure.

MR. BULLOCH: Objection.

MR. BECKER: Do you agree?

THE WITNESS: I agree but again, you know, Matthew's blood pressures are really, really very, very good throughout that whole period.

MR. BECKER: We're going to talk about Matthew's blood pressure in a moment.

THE WITNESS: Okay.

BY MR. BECKER:

Q. Concerning detection -- one second. Concerning detection and causes of hypoxemia in the infant with hypoxic-ischemic encephalopathy very diligent surveillance is critical. Do you agree?

MR. BULLOCH: Objection

THE WITNESS: Yeah, again, I'm not sure of

the context but it sounds reasonable.

MR. BULLOCH: Mike, just to save me from making objection every time you read something from Volpe's can I have a continuing objection to this line of questioning?

MR. BECKER: Sure.

BY MR. BECKER:

Q. Are you familiar with Barkovich who has written a text, Pediatric Neuroimaging?

A. I know Dr. Barkovich. I have not read the book.

Q. So do you recognize him as one of the leading authorities in this country on newborn and pediatric neuroimaging?

A. Yeah.

Q. I'm in Chapter 4, Barkovich says that a number of different patterns of brain injury can be seen as a result of the hypoxic-ischemic episodes in neonates, infants and children. These patterns can best be understood if interpreted as being a result of three primary factors: Severity of hypotension, procuring the brain at the time of injury and duration of the event. Do you agree?

MR. BULLOCH: Mike, before he answers, since

I asked for a continuing objection as you were reading through Volpe, I'd like a continuing objection as you now read through Barkovich.

MR. BECKER: You can have one.

MR. BULLOCH: I would also like to know what edition and the copyright date is that you're reading from that particular text, because we all know --

MR. BECKER: Standard of care is not an issue here. I'm reading from Chapter 4 of the most recent edition that came out in 2005.

BY MR. BECKER:

Q. Premature infants that suffer mild to moderate hypertension typically sustain injury to the periventricular white matter with sparing of the subcortical white matter and cerebral cortex. Do you agree?

A. Yeah, and, again, I think you are talking about substantially young preemies there. Again, the term "prematurity" covers a lot of territory. Anywhere from 25 weeks to 36 weeks, technically. So I agree with the statement but, you know, with the understand that he is probably describing the very young preemie there.

Q. I will represent to you, professionally,

Doctor, he doesn't limit his age group for premature infants in these statements. In contrast, term infants who suffer similar degrees of hypertension sustain injury in the watershed portion of the cerebral cortex and in the underlying subcortical and periventricular white matter. Do you agree?

A. I think that's generally true.

Q. Classically, this change in injury pattern has been attributed to a change in location of intravascular boundary zones. Do you agree with that?

A. Well, I actually don't. I mean, that's part of the, part of the answer but it turns out there's some interesting work that actually hasn't even been published fully yet, that looks at things beyond just the simple mechanics and plumbing of the brain and this has to do with the distribution of what are called ampter receptors and ampter receptors change very much through the premature time beyond, you know, beyond term and birth. So, you know, that is sort of a new concept and it's still being studied a lot in humans and in animals. So, you know, he is writing about, sort of, like, you know, they always try to make these things fit simple mechanical models. Like, this is the area of where the profusion is lowest. It turns out there are

still other factors involved. So it's not that simple. That's my only objection to it.

Q. He says that white matter damage of prematurity is there for an injury associated with abnormal neurological outcome in premature born children and it's particularly common in those with hyaline membrane disease. Do you agree with that?

A. With severe hyaline membrane disease you get white matter injury and spastic dysplasia.

Q. Probably due to the maceration of both the brain and it's vascular system, the pattern of injury begins to change between 34 and 36 weeks cause conceptionally as the regions at the highest risk for injury extend peripherally to include the subcortical white matter and cerebral cortex in the intra-arterial boundary zones. Do you agree?

A. Right, but again this is the age group that is pertinent to Matthew so you have the subcortical white matter. You go all the way to the surface and that's the cortex so that's got to be the injury. So you have EEG abnormal, mental status changes, the white matter right underneath that, the subcortical white matter; we don't have that finding in him. So I don't disagree with the statement but, just, again, for

relevance, this isn't described in his radiology picture.

Q. And, maybe, either I misread it or you didn't understand it. I'm saying it grows and extends, by the time you get to 36 weeks and 37 weeks into that region we just described. That's what that paragraph says. Do you agree or disagree?

A. Can you just re-read it again. Maybe I heard it wrong.

Q. Probably due to maceration of both the brain and it's vascular system, the pattern of injury begins to change between 34 and 36 weeks as the regions at highest risk for injury extend peripherally to include subcortical white matter and cerebral cortex.

A. Yeah, but realize what that is saying is that for a real little preemie it's very ventricular more or less, and now, between 34 and 36, where Matthew is, injury at that age extends to the cortex and the subcortical white matter. You know, if it's ischemic, well, he doesn't have those changes.

Q. Well, if he has an ischemic pattern of injury, assume it's true, would that be consistent with what Barkovich is saying?

A. No. Barkovich is saying, you know, it's

not just deep now. It's going all the way out between 34 and 36 weeks, that's the pattern. And his white matter changes are deeper, but they're not subcortical and there is no cortical injury. I mean that's the way, that's the way I read that -- understand that paragraph.

Q. Just to move on in the sake of time, Doctor, he has a table where he is -- shows the pattern extending to the parasagittal, at 36 weeks, region and before that nothing documented as to 35 weeks and up to 34 weeks showing periventricular white matter as the pattern of injury as mild to moderate hypertension.

MR. BULLOCH: Is there a question there, Mike?

BY MR. BECKER:

Q. Yeah. My question is, assuming that's true, Doctor, do you agree with that or disagree with that?

A. You know it's fine. I really need to look at the table. It's just hard to picture what you're describing.

Q. Now, you used the phrase, earlier in this deposition, of PVL and do you mean periventricular leukomalacia or do you just mean white matter injury of prematurity?

A. Well, I may have used that non-specifically. I think of them as being -- basically, the generic term should be white matter injury because that could be around the ventricles or subcortical and so forth. PVL by definition has a location, it's around the ventricle, it's periventricular. And I'm not enough of a radiologist to be able to look at white matter lesions and say that's the location and the look of PVL versus coagulation necrosis versus infection or inflammation. To me I'm just lucky if I can see the white matter changes at all.

Q. Doctor, did you bring your current copy of your Vitae with you?

A. Yes, I did.

MR. BECKER: Can we mark that as seven please.

(Whereupon, Exhibit 7 was marked for identification by the court reporter.)

BY MR. BECKER:

Q. Doctor, are there any articles that you've written on there that deal with mitochondrial disorders?

A. No.

Q. Was that a no?

A. No.

Q. Are there any articles other than the one article about ischemia to the spinal cord that you wrote on the topic of HIE?

A. Well, my -- I have an article about the metabolic effects of seizures in kids with HIE. That is number 11. The articles about the seizures are mostly about kids. The articles about the neonatal seizures are mostly about HIE kids. I have article 17, we have continuous intracranial pressure monitoring and serial electroencephalographic recordings in severely asphyxiated term neonates. And the one you mentioned the hypoxic ischemia spinal cord injury. Twenty-four is about acquired neuropathological findings in kids with a heart defect, and those are ischemically, in term babies. So it's relevant. It's not exactly that. I think that's pretty much it.

Q. Doctor, can we agree that there is no indication of what this child's blood pressure was or heart rate was between 5:00 and 6:00 p.m. on the 25th and between 6:00 and 8:00 p.m. on the 25th?

A. I'd have to look at the records to say that.

Q. This is around the time when there was suspected pneumothorses, bilateral pneumothorses,

putting in chest tubes?

MR. BULLOCH: Objection

THE WITNESS: There was a right-sided pneumothorax and the right-sided chest tube is put in and I understand there is a dispute about the interpretation of the chest X-ray. There was a left-sided pneumothorax also or pneumoinstinum and I'm not going to have an opinion on that but that he did later get a left-sided chest tube. So this is around those times, yes.

BY MR. BECKER:

Q. Doctor, I want you to assume it's true that between 5:00 and 6:00 p.m. there is no heart rate or blood pressure or pulse ox recorded -- let me, there is no blood pressure, heart rate recorded and between 6:00 and 8:00 p.m. there is no blood pressure and heart rate recorded. Would that be what you have referred to in other depositions as, kind of, a black hole of data as to what was going on with the child during this critical period of time?

MR. BULLOCH: Objection

THE WITNESS: Well, there is no numbers written down. There is a black hole of, you know, recorded data but, you know, these babies

are under visual observation and they don't look good, they don't respond to them. I mean alarms can go off. If there was a sudden bradycardia or sudden hypoxia the kids are on machines to alarm if that happens.

BY MR. BECKER:

Q. I think we have agreed that gradually the seizure will fizzle out after an acute injury, correct?

A. I'm sorry, what did we agree on?

Q. That gradually a seizure will fizzle out after the acute injury?

MR. BULLOCH: Objection. I don't recall any discussion about that but go ahead, Doctor.

THE WITNESS: I mean when kids have seizures acutely from a stroke or hemorrhage or HIE almost always they will, you know, over hours or days or, you know, some finite period of time, they will stop the acute seizures.

BY MR. BECKER:

Q. Was this child a normal head size at birth for a 34, 35-weeker?

A. Actually recall that it was 25th percentile or so.

MR. BULLOCH: Feel free to look at the

records, Doctor.

THE WITNESS: My report recorded it to be around the 25th percentile for age.

BY MR. BECKER:

Q. And did the mom have a complaint of decreased feel of movement?

A. Not that I recall.

Q. And was there evidence of polymyodous?

A. Not that I recall.

Q. And the newborn exam when the child arrived at Fairview was normal?

A. As far as I remember, yes.

Q. So we have a normal looking brain and child at the time this child arrived at Fairview?

A. You mean normal looking exam?

Q. Yes.

A. When you said brain -- yes, as far as I recognized, that the child, you know, had the respiratory problems, which is why he was transferred, but had a normal head circumference and had normal muscle tone, activity and, I guess, alertness at that point.

Q. About day 6 of life I believe that the first note is a change in tone and change in activity.

Do you recall that?

MR. BULLOCH: What date, Mike?

MR. BECKER: I believe it's day 6 of life when they first started charting decreased tone and the decreased activity.

THE WITNESS: I, actually let me just get my notes. So I have on day 10 -- no, I'm sorry, on day 11 low tone and this is from my notes. So actually I think on day 8 there was concerns about the low sodium; on day 11 there was low tone. The next day is when they found the pneumonia was higher, 67.

BY MR. BECKER:

Q. And can increased elevated pneumonia be a reflection of encephalopathy?

A. The other way around, an elevated pneumonia can cause an encephalopathy. If you have a bad liver and you drink too much or whatever those people get pretty stuporous and that's, at least in part, from the elevated pneumonia that their liver is not clearing.

Q. Anyways, Doctor, around roughly day 6 or day 7 the child started to demonstrate some abnormal neuro signs which persisted throughout that neonatal

care. Do you agree or disagree?

MR. BULLOCH: Objection, Mike, you just said it was day 11. Now you're trying to push it back to, he said it was day 6. He never agreed it was day 6. He said it was day 11.

BY MR. BECKER:

Q. Let's say day 11 then, Doctor.

A. Well, that's what I have in my notes, is that the activity and tone was around day 11.

Q. Okay. That was noted at day 11 and did that persist?

A. Yes, it did.

Q. And does the record reflect that that was the basis for low tone and decreased activity, the basis for the concern by Doctor Lilan of a neuro injury and the reason he ordered an MRI?

A. That was my understanding. I think he got a neuro consult in there, too.

Q. Doctor, I'm going back to this mitochondrial disorder. Is there a specific test that can be done to rule this in or rule this out, specific blood work?

A. Well, there are specific tests, plural, but this is a very, very complex metabolic disturbance

so you might imagine there is a lot of chemicals that go into the normal working of a mitochondria. So there could be a genetic thing, the oxidative enzymes, transport enzymes. So the work-up, usually, is to have a neuro metabolic specialist to see the child to look for very specific things.

Q. And was that ever done with Matthew?

A. No. Just the screening test, which is the lactic acid is the screening test for them and they were positive.

Q. Have you ever suggested to defense counsel that this child should have metabolic testing?

A. I did yesterday and I am today, too.

Q. So it's your opinion, Doctor, that this child's condition is not related to any type of infection or cytokine injury?

A. I don't think -- see, I don't think that any of that explains Matthew right now. At least my understanding of cytokine injury, which is, again, it's a picture of spasticity and as the white matter is being injured so that's the long track sign. So you have spastic dysplasia or spastic quadriparesis. Again, when you look at the videotapes I think the most visible part of his disorder is the choroid cytosis and then we have

the deafness and his neuro behavioral things and those are pretty classic for mitochondrial disease. Whether or not this is progressive, my understanding from the recent stuff, we talked about before with Dr. Wilhem, is that it sounds like there is a big difference. Even if it's static, as you'll see in the description of the mitochondrial disorders, that it's a very broad spectrum of condition and that can include static lesions.

Q. Your speciality is within pediatric neurology, EEG, electroencephalograms, correct?

A. No. I mean I'm a general child neurologist and I have conditional training in EEG and epilepsy. I run the epilepsy clinic. I certainly have kids with mitochondrial disorders because some of them have epilepsy, but when I attend the, you know, the inpatient service it's whatever comes in. It could be a stroke, a hemorrhage or, you know, whatever, mitochondrial disorder. So I have, you know, 25 years of experience here at CHOP. So I've seen a lot of stuff.

Q. As to medical/legal you do about 70 percent defense, 30 percent plaintiff?

A. Something like that.

Q. You failed your boards in the EEG the

first time you took them?

A. Yes.

Q. Is it your opinion, Doctor, that the lesions that both Dr. Nelson and Pressman see on the MRI early on are not causing any dysfunction, either motor-wise or cognitively, in this child?

A. So, I mean that's a really fair question. I can't say that they're causing no issue whatsoever. I guess what I'm saying is that they certainly do not describe the predominant form of history of cerebral palsy or the deafness or the lactic acid or the behavioral stuff. So, usually, when you see white matter lesions causing spasticity there's a whole lot more there. The cyst and all that kind of stuff. So I'm not really sure if this is contributing or not. There is a possibility that there is but I don't really think so.

Q. Did they ever measure this child's serum prolactin?

A. Prolactin, I don't remember.

Q. If it was elevated what would that be an indication of?

A. Well, prolactin can go up with hormonal problems, pituitary problems, in a comatose state it can

increase after seizures. So there is a pretty broad list of things. Some drugs can increase prolactin, too.

Q. Doctor, I just want to recap here. Your opinions as to why you can rule out the complications of RDS as the etiology of Matthew's current condition, you feel there should still be, if it was hypoxemia combined with loss of autoregulation, you feel there should still be evidence of multi-organ injury, correct?

A. Yes.

Q. Do you feel that -- well, we now know that two experts both see abnormalities in early MRIs so the basis of a negative MRI that you reference in your report I suspect is being withdrawn?

MR. BULLOCH: Objection because -- well, I'll just object.

THE WITNESS: Right. I mean, I was basing that on the medical records that, you know, the hospital's reading of that and I don't think they're normal. So I can't -- I have to withdraw that.

BY MR. BECKER:

Q. Okay. And the fact that there was no seizures or documented seizures, clinically, the fact that seizures are very difficult to pick up in a newborn

and only occur, that can be appreciated clinically 20 percent of the time, that's not a strong basis for you to say there wasn't an insult in this child, correct?

A. Well, you know, you don't have to have a seizure to be asphyxiated. There are babies that are asphyxiated that, even if you didn't continue to use EEG monitoring, don't have seizures. You know, the fundamental thing, even the multisystem, that's true, I believe to be around 80 percent of cases of birth asphyxia will have multisystem but it's not a hundred. I can live without the seizures but I can't live without the encephalopathy. I mean if you damage the brain, right then and there, by pneumothorax and hypoxia and all that, so severally that for the rest of this kid's life is going to have the troubles that Matthew has, how do you do that without having any neurological signs? Not just seizures but, you know, changes --

Q. Well, when you say --

MR. BULLOCH: Wait a minute, Mike, let him finish.

THE WITNESS: So, I mean, the cardinal signs of then and there, right then and there, during the pneumothorax, the mental status, the activity, the tone, reactions to the nurses and

so forth. So that's my main objection to connecting the pneumothorses to his long-term problem.

BY MR. BECKER:

Q. Now, then and there, during that day, was he medicated with Valium and morphine?

A. Yes. He was given morphine for his irritability and I just presume it had to do with the pneumothorax. I guess it could hurt. So that -- but, again, we're talking about, really, encephalopathy. A 35-week baby there is a difference between being given a pain medication and being encephalopathic. I can't imagine they couldn't tell the difference or would miss an encephalopathy.

Q. Let's first see if we can agree that medication can mask encephalopathy, potentially?

A. Well, if anything it would mimic encephalopathy. If I gave you enough morphine, you would be unconscious and not move or whatever. It is not going to mask it. It would actually raise the concern, why isn't this person active in moving around and responsive. So it's really opposite, I would say.

Q. Well, can the irritability be a sign of encephalopathy?

A. In some circumstances, it can. For example, in meningitis the babies are irritable as a non-specific cerebral sign because, I don't know, they have a headache or the meningitis is irritating the meninges but I don't think of, I don't think of irritability as being part of, like, classic hypoxic ischemia and encephalopathy.

Q. When you say, where is the encephalopathy, are you saying that this child, if there was a hit on this child due to the hypoxemia and the vulnerability to loss of autoregulation, that this child should have demonstrated what Volpe describes as the neonatal neurological syndrome?

A. Yes. Definitely.

Q. That's what you're talking about?

A. Yes, yes and by neonatal neurological syndrome -- again, you know, if this is a tiny, tiny preemie I do not expect them to have altered mental status, seizures or whatever. They can have plenty of brain damage from hypoxia or ischemia when they are very preemie. But for this child who is near term, 35 weeks and, certainly, I know this is in the ACOG thing when they define, you know, 35, it's 34 to term. You know, if you have brain damage from an event right now, then

you must show some acute neurological signs right now.

Q. Which ACOG are you referring to?

A. The green thing, the, you know, that lays out the criteria, but I only refer to that because they, literally, state, for the neurologic signs, they recognize, if it's a very young baby, you may not see neurological signs in activity, seizures. But 34 weeks to term or post term is where that's one of the cardinal features for birth asphyxia. Again, this is not birth asphyxia but it's still a type of asphyxia. You're saying, you know, your hypothesis is impaired autoregulation, hypertension, hypoxic, well, that's a type of an asphyxial injury. So, how do you, how do you injure somebody, you know, day after birth when, if it was a day before, then you would really expect to see those prominent clinical signs.

Q. Well, let's assume that this happened, that, if you follow the Plaintiff's hypothesis here, that there was insult and, maybe, multiple insults of the combination of loss of autoregulation with hypoxemia. What specific clinical signs would you expect to see as evidence of the brain injury or the encephalopathy. What specific neuro signs in a newborn would you expect to see?

A. Again, so if it's significant enough to cause brain damage you would expect, you know, a striking degree of low tone, hypertone; a striking degree of inactivity, unresponsiveness, up to coma. It turns out that over half of the kids that are birth asphyxiated do have seizures, but not all of them, so you might have them you might not. Eighty percent of the kids will have because, again, their whole body has been exposed to this ischemia, you would expect to see 80 percent of the time, other organs involved, you know, the usual thing, the kidneys, the gut, the liver, the bone marrow.

Q. Doctor, I'm going to have to apologize for about the last two minutes I'm having problems with my sound and I have been playing with this and it's not working. So if you could just either re-state that last answer because I didn't hear it. I heard it in segments.

MR. BULLOCH: Do you want the court reporter to read back the answer.

MR. BECKER: Either way.

(Whereupon, the last answer was read back by the court reporter.)

BY MR. BECKER:

Q. What you would expect to see, a noticeable change in activity, a change in tone, and possibly multisystem organ injuries?

A. Those, yeah those are included.

Q. Now, when a child is on a mechanical ventilation does he receive any drugs to keep him calm?

A. I don't think it's automatic and some babies seem to tolerate it more than others. It's not uncommon for nurses to give drugs like Valium just to reduce the "anxiety" of the experience but I don't think it's a knee jerk reflex thing.

Q. There is a order, I think, around noon of the 25th, for staff, a stat administration, I believe, of Valium, not morphine, I think it was Valium. Do you have any understanding as to what led up to that?

A. I don't. I don't recall that.

Q. Doctor, I think I understand where you're coming from. I want to chat with you at the very end, I'm just about done. I might have 15 minutes or so or more. I want to talk about this article that you co-authored with many other physicians at CHOP. And it's entitled, "PVL is Common Following Neonatal Cardiac Surgery." Do you recall the article?

A. Yes.

Q. In fact it was published in March of 2004 and before I get into this a little bit would you agree with, at least, with Barkovich when he finally concluded that this use of the word PVL has been too liberal and it is has a very specific meaning and one should be more talking about premature white matter injury to avoid confusion. Would you agree with that?

A. Well, I probably agree that there is a lot of different terms that are thrown around. Some are better than others. But, you know, we're all talking about acute lesions in the white matter, whether they are premature babies or not. But I do understand he's got this opinion, but --

Q. In the article you say you conclude that hypoxemia and hypotension in the early post operative period, particularly diastolic hypotension, may be important risk factors for PVL, correct?

A. Yes.

Q. Now, in these children was there evidence of multisystem organ injury?

A. I don't, I mean, yes, they're all cardiac kids. So the picture -- please remember this, the context here, these kids just had cardiac surgery and --

Q. Right but what you were discovering --

MR. BULLOCH: Mike, let him answer his question, please.

THE WITNESS: Just so you understand, though, these kids all do go through a cardiac failure in the post-op period. We know that is going to happen and, you know, it's called the sag. So around 12 hours after surgery they get hypertensive and things like that. So it is in this context, and I'm not saying birth asphyxia. You just said cardiac surgery. So they do have falling blood pressures. That's why we look at oxygenation of the blood and blood pressures. Their lungs will often get stiffer. We often confuse that with their cardiac output. Now, in terms of correlating it with PVL the lowest oxygen reading and the lowest blood pressures were risk factors for those things being developed in the post-op period.

BY MR. BECKER:

Q. At the end of the article there is a communication between a doctor asking questions and someone from your group, in this case a Dr. Gaynor, responding and the doctor asked the question -- he makes a statement, the doctor not part of the study, says, in

patients with postoperative hypoxemia in PAO2 of less than 40 or post operative hypotension, both of which were great points in your study, was there a correlation when the PVL occurred, and Dr. Gaynor goes on to answer in the affirmative. My question is I'm assuming PAO2 here means TAO2 and that is the gas oxygenation of less than 40, correct?

A. Yes. That's TAO2.

Q. Now, and a diastolic blood pressure less than 35, correct, for these kids?

A. Yeah, I don't remember the number but we gave it as a cut point.

Q. And was there evidence Of THO2 of less than 40 in this child at Fairview General Hospital that is below your break point?

A. There was a single measurement below that.

MR. BULLOCH: Objection.

BY MR. BECKER:

Q. Do you know whether or not there is a correlation or -- strike that. Can you correlate, Doctor, oxygen saturation of 60 to 70 as to how that would equate into a PAO2A? Are you familiar with any formula based in your experience?

A. See I can't, that's not part of my

expertise so I can't answer that.

Q. Dr. Gaynor writes that we haven't looked for PVL in neonates without cardiac disease but patient population in which PVL was described is in the neonatal intensive care unit, in preterm infants, and it has been associated with a variety of factors with hypoxemia, with respiratory distress syndrome in these infants and with hypotension. Do you agree?

A. Right. Again, for preterm babies -- realize again Dr. Gaynor is a cardiac surgeon. He's presenting to the Society of Cardiac Surgery and that these are kids who have just had cardiac surgery, so, like, the hypoxic -- you have to keep in mind, again, these kids do have multisystem malfunction. They have bad profusion because they have just had major cardiac surgery and, of course, Matthew didn't have any blood pressure issues. He had the one reading below 40.

Q. Right, but what Dr. Gaynor is -- I guess I would have to show you the article to be fair with you, Doctor, but he is saying we haven't looked for PVL in neonates without cardiac disease but the patient population in which PVL is described he is referring to the literature, clearly, in a neonatal intensive care unit in the preterm infants that have been associated

with hypoxemia and with respiratory distress syndrome in infants that's what he's saying.

MR. BULLOCH: Objection. You're assuming what Dr. Gaynor is referring to in his question at some point in time, but go ahead, Doctor.

THE WITNESS: Yes. When we discussed this issue it's actually very important, I think, because from a population point of view there is a lot more preemies with PVL and handicapped, like, real preemies, like, 30-weekers, and here we have term kids that go into cardiac surgery and they have this PVL that shows up. It's much smaller. We don't really know how significant it is, by the way. That gives us a chance to, maybe, understand what's behind the PVL and the preemie. So, you know, I think he's referring to the literature there. I agree with that. He's talking about most of what we know about PVLs and little preemies in the NICU and all that sort of stuff but this, sort of, extends our understanding of the biology of PVL.

BY MR. BECKER:

Q. Clearly Matt was a preemie, correct?

A. He was, he was near term, 35 weeks. He is

right on the border. So, yes, he's a preemie but, you know, just a pinch.

Q. He had moderate respiratory distress syndrome and he was mechanically ventilated, correct?

A. Yes, sir, that's correct.

Q. He had, at least by reading the chart, two bouts with pneumothorsis and there was evidence of hypoxemia in his chart about this period of time, correct?

A. Yes. He had some low readings, but I don't consider them to be terribly low.

Q. Well, how low does it have to be and you use this phrase in your report of "brain damaging hypoxemia." How low does it have to be and what's your authority for it?

A. So for a near term child I guess the critical value would be around 20 and the authority for that statement has to do with my work and experience with kids that do have congenital heart disease, because there are plenty of babies floating around that have a big hole in their heart and the blue blood mixes with the red blood and they have chronic hypoxia and they have it until they undergo their cardiac surgeries days or weeks later. Now, there are some critical forms of

congenital heart disease where you'll literally have the mother give birth at our pediatric hospitals so the baby can be taken directly from her room into the cardiac operating theater, if we know that their cardiac lesion is one that will produce this profound, severe, low value.

So 20 is sort of the cut-off point for crisis, but again you're talking about someone with, you know, not just hypoxia but profusion. And Matthew's blood pressures were good. His color was consistently described as pink, his saturation were generally good. They're not perfect but they're good. We have this one point in time where you've got one PA02 value that falls low and that's lower than you want it to be. I know that, and he did have a pneumothorax, but if that triggered his brain damage then where is the clinical evidence with the encephalopathy. That's really the way I analyze this case.

Q. Would you defer to a neuroradiologist on the issue of whether one can have microstructural injury to the deep brain, the thalami, the basal ganglia and not have it appear on the MRI?

A. No, I wouldn't really, because I don't think they're in position to judge that. I'm the doctor

that says, look, I just did an examination. This kid has severe extrapyramidal features. Whether or not they see it. Like I said, in metabolic disease the basal ganglia don't work properly, they're dysfunctional, but they may appear fine. If he's going to give me some song and dance about invisible damage, I'm not going to buy that. I will accept their reading if they say it looks structurally normal but the interpretation that really is a secret, hidden, cryptic kind of injury, I think I'm the best judge of that.

Q. Well, if, in your experience, if seeing edema in the deep brain of MRIs taken, one day or two days of life and if, day of life 8, 10 or 15 they no longer see any evidence of damage on the films, yet the child has evidence of thalamic injury, is it wrong for them to conclude that the injury is there but we know that it is just not showing up?

MR. BULLOCH: Well, Mike, you just said that they're seeing an image of the thalamus, and you're talking about the neuroradiologist, and then, in the same question, you're saying they're not seeing any changes. I'm confused.

THE WITNESS: I understand his question.

BY MR. BECKER:

Q. No. I'm saying in my hypothetical, Doctor, where they, I've had this, I'm not making this up, in multiple cases. I can think of two where there have been, early day 1 or day 2 of life, clear evidence of edema deep in the brain, subsequent MRI on day 10 or day 20 showing it normal and yet, today, the child is showing mixed CP?

A. Right and here's -- and I think maybe we're not really disagreeing but I've also seen cases where, like right after birth, a day or two, you see changes. They go through a phase, later, of normalization and I don't know what that time period is, I don't remember it but then when you do follow-up scans when they are a year or two years old you do see, then, like, the permanent scaring and atrophy and signal changes of the basal ganglia. There could be a phase, like, maybe, I don't know, for argument sake, ten days, two weeks after birth where it could be the edema is gone but then when you have follow-up studies you do expect to see the changes. So we don't have changes on our first scan and I forget how old the child was then. His MRI scan was done on 9/8, so I guess he was 15 days old, day of life 15, but we have got several scans after that and they should have shown the basal ganglia

changes.

Q. Doctor, in your report when you say, I've never seen the combination arise in practice, that is, a mixed CP as a result of PVL, I think I know what you mean but better explain it to me.

A. Right. The combination there, is a combination carditis, is the classic combination of extracranial CP and deafness. You know, this kid's billow rate was never that high it was 13, 13.6 something like that, but I've never seen arise from PVL alone a picture of extrapyramidal and pyramidal CP. I expect those kids to have spastic forms and again when you look at the videotapes the choroid acidosis is not subtle it's actually extremely florid. So this is a major contribution to his disabilities from the choroid acidosis and I have not seen that on PVL.

Q. Well, where do you see the chordal, simply in his head or do you see it in his arms as well?

A. Yeah. It's actually, there is a nice scene, I think it was the first time video, where mom's got both of the kids in their little high chairs and the camera is facing them and the little girl is eating and as Matthew tries to eat, you know, his head rotates. He doesn't have the control. His hands rise and he is

trying to eat. All of these extra overflow movements are part of the choroidal acidosis. And then in the day in the life movie I think he is working with one of the occupational therapist and he's in this, they have him suspended from a sling and he's, like, supposed to be getting these forms and putting them in the molds and again his hands are rising and so forth. It's his head trunk and arms. I don't really see it in his legs. His legs doesn't seem to be as involved.

Q. How do you pronounce that again?

A. Extrapyramidal.

Q. Extrapyramidal it's got to be without purpose, correct?

A. Correct.

Q. And you're assuming that his movements while he was eating were without purpose?

A. I mean the movements, the extra movements. He was trying to eat and the movements didn't serve the purpose of eating. They would intrude or interfere with the desire to act. So that's why they are called involuntary, they're not under his voluntary control. They arise within.

Q. Did you ask to examine this child?

A. No, I didn't. I did ask for an updated

video. I mean I asked if there was an updated video

Q. So if, in fact, his motor problems are static, you would still maintain your position that this kid's problem is from an undiagnosed metabolic disease of some type?

A. Yeah, again the concept of the mitochondrial disorder as being static is well documented but, again, when you look at the whole picture of the extrapyramidal CP, the deafness, the lactic acidosis, the neuro behavioral things, that's clearly within the diagnostic category of mitochondrial. If it's progressive even deeper into that territory but I am certainly uncomfortable with that as a working diagnosis at this point.

Q. You're presuming that his lactic acidosis persists?

A. Well, first of all, and I know you will look at this paper, they don't have to have any lactic acidosis. That can be an intermittent thing, but when I read Dr. Bachman's deposition, remember, the first time they did the lactic acid, they also did a pneumonia level. Pneumonia is a very fussy thing, too. So if you have a tourniquet or whatever you can get spurious pneumonia readings. His pneumonia was very normal

thing. So that told me in the same blood draw he has a normal pneumonia level and that's a sensitive test and he's got a clearly high lactic. And then Dr. Bachman's deposition he said, well, I wanted to be sure about this so he wrote in the chart he did it in the morning. He wasn't fussy. They didn't use a tourniquet to repeat it and I don't remember the exact number. It was still clearly high. I think the first value was 21 and the second value was 17, but those are way elevated; 16 and 21 were the numbers. So I thought they did a good job of, you know, being -- recognizing where the pitfalls were in the technical aspect of drawing a lactic acid. So again that shouldn't be there. Now, whether another measurement would prove it, no. You need to go to the next level now, which would be more invasive things, genetic testing and really something that requires true expertise in this.

Q. Well, is it your experience with children with mitochondrial disorders that they generally manifest this condition in the very early newborn period?

A. Absolutely. Some -- I mean, again, when you see the paper they talk about the experiences and say what was the range of presentation, and some are

newborn and I don't even know that Matthew is manifesting his metabolic disease as a newborn. I think it's really later when they appreciate that he's got the arcing and all the rest of that. That, to me, is when it really manifests. To me the newborn period, again, when he had the pneumothorax he didn't really show anything specific neurologically and then later when he was, like, 11 days old and had the hypertonia and all that. They were worried about infection, the pneumonia, the low sodium. They had a neurologist come in and he, sort of, kind of, got a little better. He was certainly better when he was discharged from the hospital than he was at the presentation. So I don't really know when the metabolic mitochondrial disorder really declared itself, but at this point he's got all the hallmarks of a mitochondrial disorder.

Q. So, Doctor, you would urge his treating neurologist to consider that diagnosis?

A. Absolutely.

Q. And you cannot, you don't have an opinion as to the brain injury that is shown in the early MRI, how that's impacting him or can you sparse that out?

A. Yeah, I mean the white matter injury is there. Again, I don't know if that's a, you know,

again, the radiologists disagree. Pressman said it was, like, a profusion issue, like a low blood pressure issue. Nelson said it was, like, coagulation as opposed to something you get with cytokine injury. I'm not going to offer an opinion on that. Although I can see it, it's not terribly impressive in terms of there was no cyst and so forth, and at least from my reading of the following reports there is not a great deal of, you know, there is little ditzels in there now. It's hard for me to correlate that with, like, CP for example.

You know what we didn't talk about, you brought up this paper that we did with the PVL and so forth. Those kids don't have CP, by the way. I mean they may, a lot of the cardiac kids have some developmental issues, attention span issues, things like that, but it's really rare for them to have cerebral palsy. So you can have little white matter pouches and they're not -- we don't know the full significance of that. That's why I can't say from Matthew either.

Q. Well, you only looked at one film and it was the early film. You didn't see how this injury evolved?

A. No, I understand that. What I'm saying is, when I read the reports they're not talking about

cysts in the white matter, things like that.

Q. You're referring to the original treating doctor's reports, not depositions?

A. Correct, and I'm sure you know this, but the correlation with white matter injury in preemies it depends on, it's not just a touch of PVL. It's like, it is supposed to be fairly extensive and there's much higher correlation of CP if there's cysts in the white matter. If there is cysts it means the tracts have died and they have been filled with water. So we're using PVL as a general blanket term but what correlates most of the CP is the multi-cystic PVL and the kids that don't have that, I'm not saying they are a hundred percent normal, but it's less correlated with CP and to my knowledge Matthew never had cystic white matter lesions.

Q. Well, isn't it true, Doctor, that classic PVL talks, connotes cysts around the ventricle. Not within the white -- not deep in the white matter. They talk about cysts, classic PVL and cysts around the ventricles?

A. But it's cysts of the white matter that surrounded the ventricle. That's PVL around the ventricle and if you get lots and lots of it they

actually turn cystic but it's still cystic white matter changes.

MR. BECKER: That's all I have.

(Whereupon, Exhibit 8, Exhibit 9, Exhibit 10, Exhibit 11, Exhibit 12, Exhibit 13, Exhibit 14 and Exhibit 15 were marked for identification by the court reporter.)

(Whereupon, the deposition concluded 11:22 a.m.)

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

I, Micheline Brown, a Court Reporter and Commissioner of Deeds for the Commonwealth Of Pennsylvania, do hereby certify the foregoing to be a true and accurate transcript of my original stenographic notes taken at the time and place herein before set forth.

_____

Micheline Brown

Court Reporter

Commissioner of Deeds

DATED:_____

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SIGNATURE PAGE

OF

ROBERT RYAN CLANCY, MD.

I hereby acknowledge that I have read the foregoing deposition dated August 25, 2006, and that the same is a true and correct transcription of the answers given by me to the questions propounded, except for the changes, if any, noted on the attached ERRATA SHEET.

SIGNATURE

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ROBERT RYAN CLANCY, M.D.

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CASE: Matthew Chase Wagoner, et al. Vs. Mark R. Evans, M.D., et al.

DEPOSITION OF: Robert Ryan Clancy, M.D.

TAKEN: August 25, 2006

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