IN THE COURT OF COMMON PLEAS
OF SUMMIT COUNTY, OHIO

MACKENZIE LYNN TARLE, a minor, et al.,	:	
Plaintiffs,	:	
-vs-	:	NO. CV2001 05 2137
AKRON GENERAL MEDICAL CENTER, et al.,	:	
Defendants.	:	

Washington, District of Columbia

Tuesday, November 26, 2002

Deposition of:

RAOUL L. WIENTZEN, JR., M.D.

A witness, called for examination by counsel for the plaintiffs, pursuant to notice, at 1522 K Street, Suite 720, Washington, DC 20005, beginning at 9:30 a.m., before JOSEPH A. INABNET, a Court Reporter, when were present on behalf of the respective parties:

FOR THE PLAINTIFFS:
JEFFREY E. SCHOBERT, ESQ.
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FOR THE DEFENDANTS:
DANIEL FINELLI, ESQ.
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C O N T E N T S

E X H I B I T S

P R O C E E D I N G S

(The witness was duly sworn by Tom Olender, a notary public for the District of Columbia.)

Thereupon,

RAOUL L. WIENTZEN, JR., M.D.

A witness, called for examination by counsel for the plaintiffs, having been first duly sworn by the notary public, was examined and testified as follows:

EXAMINATION

BY MR. SCHOBERT:

Q. Doctor, would you please state your full name for the court reporter.

A. My name is Raoul L. Wientzen, Jr., M.D.

Q. Doctor, have you previously had your deposition taken?

A. Yes.

Q. Can you give me some idea how often that has occurred?

A. I think in the last five or six years, probably four or five times a year, prior to that,

less frequently.

Q. Have these depositions over the last number of years, have they been primarily involved in the medical legal matters such as we are here for today?

A. Yes.

Q. Obviously then you understand the nature of a deposition and know the instructions in terms of if you don't understand my question, please let me know. Don't answer the question.

And because of the medium we are utilizing here today, it is always good if we wait an extra second between responses to each other because I think sometimes we can overlap, and it is difficult. Okay?

A. Fair enough.

Q. Doctor, do you have a current CV with you today?

A. No, I'm sorry. I did not bring a CV. I came from my home rather than my office this morning, but I can get one to Mr. Finelli or Mr. Margolis.

Q. Would you do that, sir, provide them an updated CV?

A. Sure.

Q. Let me ask you this just generally. Can you tell me where you did your medical school?

A. Yes. I went to medical school at Georgetown University School of Medicine, 1968 through '72.

I then did three years of pediatric residency, also at Georgetown, left to go to Dallas, Texas where I did a two-year pediatric infectious disease fellowship at the University of Texas, Southwestern Medical school from 1975 to 1977.

And then in 1977, I returned to Washington where I entered the full-time faculty at Georgetown University Medical School.

Q. Okay. You say that you did your fellowship at the University of Texas Southwest Medical School. Is that correct?

A. Yes.

Q. Who was the chairman of the department there when you completed your fellowship?

A. When I completed my fellowship was Heinz Eichenwall (phonetic), chairman of the department.

Q. Doctor, I assume that you have published. Is that an accurate statement, that you have authored peer review journal articles and/or text material?

A. Yes, I have.

Q. Is that a fair statement?

A. Yes.

Q. Has anything that you authored would you consider to be on point or -- to any of the issues that we are here to discuss in this particular case?

A. Yes.

Q. Can you give me some identification. I do not have a copy of your CV in front of me. I may have been provided it. I have your report, but I don't have the CV. So I guess I need some way to determine what articles may be on point.

A. Okay.

Q. Or perhaps we could agree that if you provide a CV to Mr. Finelli --

A. That's fine.

Q. -- or Mr. Margolis, that you could circle those peer review journals and/or tech materials as to ones you think have some relevancy to the subject matter of today's discussion, that would save us some time.

A. That would be fair. I will be happy to do that.

Q. In a very generic sense, is there a particular area that you have concentrated your writings on that would be germane to any of the issues in this case?

Have you looked at GBS infections? Have you looked at GBS meningitis, that kind of thing?

A. Yes, I have. My CV will show some articles where have I published on a model of Group B strep sepsis, a clinical series of babies with neonatal bacterial sepsis and meningitis, which includes Group B strep. It's the most predominant

cause when I wrote that article.

Some monographs that deal with neonatal infection, so there would be several that focus on Group B strep.

That has not been the focus of my research as an academic faculty member, but it has been one of the components of my academic endeavors.

Q. Is there one area that you would say has been the primary focus of your academic endeavors in that regard, some area you have spent the majority of your time researching and writing about?

A. I think it would probably be streptococcal infections in general.

Group B strep is a streptococcal infection, but I have done work on pneumococcal disease, which is streptococcus pneumoniae, Group B strep, which is streptococcus agalactiae, and Group A strep, which is streptococcus pyogenes.

And then lastly I have done some basic science work on the streptococcal species that

inhabit the mouth and how human beings become immune to those organisms.

So strep in general, but not a focus on only one variety of strep.

Q. All right. Thank you.

Board certifications in what area or areas?

A. Sure. Pediatrics in 19, I think, 78, and then pediatric infectious diseases in 1994, which was the first time the test was offered, and then recertification in pediatric infectious disease seven years later.

Q. All right. Now, going back to my original question to you about prior involvement, in medical legal matters, to your knowledge have you been asked to give opinions in a deposition format and/or at trial in issues similar to what we have here in this case, a child that ultimately has diagnosed a GBS infection at the time of delivery?

Have you given opinion testimony in that area with that as a part -- at least part of the fact pattern?

A. I'm sure that Group B strep as a disease has been part of the cases that I have given testimony in.

I'm not sure there has been a case that parallels this case very closely wherein the question is did the Group B strep cause the brain damage or not.

Q. All right. Why don't you give me -- and I'm assuming you have been asked this question before when you have been involved in medical legal matters.

Give me some idea as to the breakdown in terms of the requests that are made either by plaintiff or defendant, first of all. Let's talk about that.

A. Sure. Probably two-thirds of the cases I do are defense cases, and about one-third are plaintiff's cases.

Q. To your recollection, have you provided any the testimony in defense cases that arise from medical care rendered in Ohio?

A. I don't recall any. I may have, but I

don't recall any.

Q. All right. Do you recall being involved in any case where the care at issue is rendered in Ohio?

A. I think years ago there was a case from Ohio that dealt with recurrent pyelonephritis in a baby that then got older, and that child then lost kidney function on one side.

And I believe the case was from Ohio, but I'm not absolutely certain.

Q. Okay. How often have you been asked to do what we are doing today, which is to give deposition testimony pursuant to opinions that you have arrived at?

A. Well, as I said before, I think you had asked me that question. I think it is four or five or six.

Q. I'm sorry. I did ask you that question. Let me back up.

How often are you just asked in general to review cases?

You told me how often you are deposed.

I'm sorry. I apologize for that.

A. Probably I review a dozen or 15 cases a year.

Q. Okay. Doctor, in your report -- I have a report in front of me, first of all, of October 4, 2001.

Do you have a copy of that report?

A. I do.

Q. Is that the only report that you have generated in this case?

A. Yes, it is.

Q. Have you at any point made any notes from any review of any materials that you have been supplied either prior to the writing of this report or subsequent to the writing of this report?

A. Yes.

Q. Okay. Do you have those notes with you?

A. I do.

Q. All right. Generally, what are we talking? About one page? Multiple pages?

A. Actually, it is on the cover letter from Mr. Finelli sending me the records, that I made the

notes on the back of that sheet that deal with events taken from the medical record.

And then on the front of that was a sort of short summary of some of the issues that I had opinions about in the case.

Q. All right. Could we -- first of all, in a very general sense, is your handwriting the type that most people can read, or are you like me; most people cannot read your handwriting?

A. I think my handwriting would be challenging for most people to read.

Q. Why don't we --

A. Sometimes it's challenging to me to read.

Q. How extensive is the writing say on the front page where you kind of summarize the issues of the case after you had gone through some of the medical records?

A. Nine or ten lines.

Q. All right. Let me -- I will ask you in a moment to maybe just read that real slowly for the court reporter's sake, but let me just establish this right now.

Were these comments that you have written done at the time you completed your initial review and prior to authoring your October 4, 2001 letter?

A. These comments of my opinions were written after I did my initial review and before I wrote the letter.

Q. All right. Since authoring the letter, have you made any other written -- you know, made any other notes or put any entries into any kind of computer or anything else pursuant to other materials you may have been sent?

A. Yes. The only additional notes I made was over the last several days as I re-reviewed the original medical records, I added to the backside of the letter more facts about the unfolding of the clinical events of the baby.

No opinion facts -- or no opinions rather, but just dates and times and numbers and things like that on this side of the record -- of the letter.

Q. And that would then comprise all the written notes that you have made at any time

leading up to today?

Is that a fair statement?

A. Well, I put a note on the top of another letter, "Get CV and circle sepsis articles."

So I did do that note, but that's the only other note that I made.

Q. Okay. Let's go ahead then, Doctor, and if you would try to read at a measured pace so the court reporter doesn't get angry at either one of us your opinion comments that you have made on the one side of that page.

A. Okay. I wrote, This baby really did have Group B strep sepsis and this in association with the last in utero hours caused asphyxia, but the variety of sepsis post birth was mild.

And then I have a hyphen, Rapid recovery of cardiovascular function without DIC, without shock.

And then I have written, If somehow the baby had been delivered earlier by plus/minus six hours, the Group B strep sepsis (if it even existed then) would not have been a damaging event.

Then lastly I have written, Antibiotics were given by the OB timely (negative Group B strep screen, no fever until 4:25 a.m.)

They are my notes.

Q. Doctor, again, my ability to write is somewhat impaired by as quickly as you read, but that first set of comments that you read to me, could you re-read that just one more time?

A. Sure.

Q. Something about baby had GBS sepsis?

A. Right. This baby really did have Group B strep sepsis, and this in association with the last in utero hours caused asphyxia, but the variety of sepsis after birth was mild.

And then I have the reasons for my thinking that it was mild.

Q. Okay. Thank you, Doctor.

Going to the other side of the page then, again, is that a pretty extensive amount of notes that you have there?

A. Yeah.

Q. I don't want to take a long time reading

it, but go ahead. How extensive is it?

A. It's basically a whole page with numbers and dates and times. I would be happy to read it. It wouldn't make much sense, I think, if you take it out of context of knowing what the case is about, but I'm more than happy to read it. It would take five minutes.

Q. What about the notes you said you wrote in the last few days in preparation for getting ready for today's deposition?

How extensive are those notes?

A. That's about maybe half of what is on this page.

Q. Why don't you read those for me, if you would, please?

A. Sure.

Q. Just those things that you have written in the last few days, that you have gotten.

A. I can tell what I have written in the last few days because it's in blue ink, whereas the original stuff I wrote is in black and red ink.

So I'll read you the blue ink.

I have ABG 6:21 a.m. 8/4 7.13.

PCO2 21.

PO2 77.

Bicarbonate 6.7.

I have normal saline, 440 ccs times 2.

5 percent abumen 40 ccs 6 a.m.

7 ccs sodium bicarbonate 6:40 a.m.

Next to the normal saline 40 ccs times 2, I have the times, 5:37 and 5:42 a.m.

Then I have, Transfer note: Heart rate 150, good respiratory effort, seizures q 2 minutes. Heart rate 140, respiratory rate 64. Blood pressure 64 over 33, mean 45, capillary fill time less than three seconds.

Then I have -- next to 8/4, I have PIP14/PEEP 4. That's PEEP 4. Rate 20. No ARDS on admission. IV at 50 ccs per kilo per day.

Then underneath the date 8/5, I have written capillary fill time 2 to 3, pulses 2+/4+.

D10 1/5 normal saline. 12 ccs an hour, maintenance fluid.

I have 8/6, MAP 43 to 47. CFT less than

three seconds. Pulses 2+/4+.

I have 8/7, room air, will not extubate because of question ability to handle secretions. CFT less than 3. Mean BP 36 to 48. Pulses 2+/4+. Transfuse for hematocrit, 26.

So they are the additions recently made to the notes originally made.

Q. And if we could then hand that document, either now or at the conclusion of the depo, to the court reporter, we will ask her to mark that as Exhibit A and ask that she --

A. Let's not get sexist.

Q. Excuse me?

A. It's a man. It's a male court reporter.

Q. Oh, I'm sorry. I apologize for that misstatement -- ask that he make a copy of your notes both front and back, and then we will attach it to your deposition. Okay?

A. Good.

Q. Now, let me ask you then, according to your October 4, 2001 report, you have, it says, reviewed Akron General medical records and Akron

Children's Hospital records; correct?

A. Correct.

Q. Since that time, what additional materials have you been provided for review?

A. I have three depositions, that of Dr. Ferris, and Dr. McKelvey, and that of Ms. Tarle, and then a series of opinions from various experts, which include Louquette (phonetic), Sweet, Hayashi, Keogh, Devoe, Painter, Simpson, Rayburn.

And I think that's it.

Q. Is that all the materials then that you had a chance to review there, or you have some more there?

A. It is all of the materials that I could find, so I reviewed what I could find.

Q. Doctor, did you review any radiology studies of Mackenzie Tarle?

A. I didn't look at any films.

I read what was in the record as to the interpretation of the films as they were done.

Q. And my assumption is you have not taken

it upon yourself to look at any placental pathology.

A. Correct.

Q. That would be outside of your expertise.

A. Correct.

Q. Is that a fair statement?

A. Correct.

Q. All right. Doctor, having gone through that list of expert reports, do you recognize any of those individuals who authored any of those reports? I mean, are they people that you, from a professional standpoint, you know, know or are aware of?

A. I'm aware of one name only, and that's Dr. Sweet's name.

I don't know him personally, but I'm sure I have read some of his writings.

Q. Okay. Is there any materials that you believe you have not yet had the opportunity to review that you need to review for any opinions you may be providing in this case?

A. I don't think so.

Q. Doctor, having reviewed your report, is it accurate for me to conclude that you are not being -- or going to render any opinions on the standard of care by the obstetricians that cared for Ms. Tarle in this case?

A. That's correct.

Q. Having had the chance to review your report and knowing that you have gone back again to relook at the records, made some additional notations about findings in the records that you thought might be relevant, is there anything in this report of October 4 that you would want to change in any way?

A. No. I think what I have written in this report still is accurate.

Q. All right. Doctor, did you do any review of any literature for purposes of preparing this report or preparing for your deposition today?

A. No, I did not.

Q. Okay. Outside of what you yourself may have authored, contributed to, which we just very basically discussed at the beginning of this depo,

is there any particular author or institution or journal writer or anybody that you would go to look at if you were concerned about issues of a GBS sepsis had occurred in utero and what effect it may have on the neurological status of that child after delivery?

A. Well, I think I would -- I think there is a huge body of literature on Group B strep sepsis, some of which I have written myself.

Probably the sentinel work came from Houston, Texas, Carol Baker's group. So I probably, if I had the need to, would review some of her writings about Group B strep.

Q. When were those writings -- to your knowledge, when were those writings authored and put into the general body of knowledge?

A. Over the last 30 years, since the early '70s when Group B strep became a common clinical problem confronting pediatricians, that's when the research really began and that's when the data set that we now have has been accumulated.

Q. Doctor, are you aware or do you know a

Dr. Jeffrey Perlman or a Dr. Shalak, S-H-A-L-A-K, or Dr. Laptook, L-A-P-T-O-O-K --

A. I know none of those.

Q. -- Dr. Geoffrey?

A. No, I do not know those doctors.

Q. All right. Doctor, would you agree there is an increasing evidence supporting an association between placental infection information in term infants and the development of cerebral palsy in early childhood?

A. I think there are some data on that issue, and I think I could probably answer your question affirmatively, that there is increasing data trying to link that condition to cerebral palsy in childhood. I don't think the data is yet complete or convincing, but there is publication on that particular issue.

Q. All right. And are you aware of particular institutions or authors or people that are attempting to investigate that link between those two things, placental infection and neurological outcomes, including cerebral palsy?

A. Yeah. I have read some of the authors. I know one is Dr. Nelson, but I couldn't tell you the rest of the people involved in that research.

I have read those papers over the years.

Q. All right. What is your understanding in terms of the linkage that they are trying to determine if it exists between those events in terms of the path of physiology of that?

A. Well, I think the theory is this, that we know -- and it's now generally accepted that much of the damage that infection causes comes about because of the production of chemicals called cytokines in response to bacterial cell wall products of various kinds. And that it's the cytokines that drive the septic shock syndrome, and it's the cytokines that cause vascular injury.

So in patients with sepsis, you know, adult patients, pediatric patients with sepsis, it's really the cytokines produced by the patient himself that causes the individual to go into septic shock.

So the theory behind the cytokine

chorioamnionitis issue is are cytokines produced in that disease, do they get into the baby, do they circulate, do they cause vascular injury, and does it cause brain damage and maybe even other organ damage.

That to me is the, you know, the heart of that research.

Q. All right. Have you yourself formed an opinion as to whether or not you believe that that process does in fact exist under those circumstances whereby there is documentation of a chorioamnionitis and ultimately a diagnosis of neurologic injury such as cerebral palsy in a child.

MR. MARGOLIS: I'm objecting. Is this a term baby?

BY MR. SCHOBERT:

Q. In a term baby. That's fine. In a term baby.

A. I don't think the data is convincing to me right now that there is a link between chorioamnionitis in a term baby or in a mother who

is going to have a term baby that day and subsequent neurologic injury, such as cerebral palsy in a term baby.

I think there is some data though that is imperfect, and who knows what ten years will bring. But right now I think it is not accepted, at least in the field of pediatric infectious disease, as a direct cause-and-effect link.

And I say that for many, many reasons, one of which is often, in at least the data that I can recall, there are other factors of the baby's birth and delivery and subsequent neonatal events which in and of themselves are known to cause cerebral palsy, such as birth asphyxia, such as severe respiratory distress and bad hypoxia, such as severe septic shock.

I mean, there are many things that go hand in hand with chorioamnionitis that intervene between then and when you make a diagnosis of cerebral palsy in an individual.

So I think the data is very imperfect. I think it's fascinating data, but at this point

imperfect. And it is a theory, not a proven cause-and-effect event.

And then lastly, I would have to say that chorioamnionitis to some degree is so common that there is going to have to be many babies born to ladies with chorioamnionitis who don't develop cerebral palsy.

And what you really need to identify is what is it about the small fraction of babies with chorioamnionitis and cerebral palsy that allows that to happen, if it happens.

Q. Let me follow up.

In terms of this concept of cytokines in a pediatric or adult population, do you believe -- is it your opinion that cytokines have been proven to exist and, you know, the path of physiology you have described to me, you know, does exist in a pediatric population and/or an adult population?

MR. MARGOLIS: And I apologize. Again, I object to the "pediatric."

I would request that a distinction be made between a premature child and a term child.

BY MR. SCHOBERT:

Q. All right. We will exclude -- well, let's be all inclusive.

Do you believe in any population of patients, whether it be a premature infant or in the general population of pediatric patients into adult, that this concept of cytokines being produced as a response to the infection and then causing subsequent vascular injury and the other sequelae, do you believe that that, based on probability, exists and occurs?

A. It's a very broad question. And the answer is cytokines can induce vascular injury.

And if you have enough vascular injury, you get septic shock. And septic shock can certainly cause organ damage, such as brain damage, kidney damage, liver damage, you name it.

But where that chain is not linked quite well is if you get cytokines without septic shock, does it cause injury. And I don't believe there is any data in older children that cytokines in and of themselves, no matter which one we are talking

about, cause brain damage.

If you get enough cytokine release, you could go into shock and then get brain damage. But cytokines are released -- every time a child has an appendix taken out, there is cytokines released. Every time a child falls out of a tree and breaks his leg, cytokines are released. Every child who gets strep throat, cytokines are released.

And no one talks about brain damage in these trivial diseases, even though there are cytokines in the blood.

So that's my basic reservation about accepting this damage is that it's not just cytokines. It has to be the quantity of cytokines, the kind of cytokines, the physiologic effect of those cytokines before you can make, you know, a causation argument for cytokines.

Because we know they don't harm you or me or people sitting in the room with you there in trivial amounts or the amounts that happen with early infections.

Q. Doctor, if -- just so I understand it, if

you develop an infection, an individual, let's say an adult individual develops an infection which leads to sepsis; at that point, are cytokines being produced as a natural product of that infection?

Is that your understanding?

A. Absolutely true.

Q. And what you are saying is that it is unclear as to whether if we simply stop at that point and these cytokines have been produced, that those cytokines in and of themselves would lead to potential neurologic -- or lead to neurologic injury.

Is that what you're telling me?

A. No. I'm not saying it's unclear. I'm saying it's clear that they don't cause neurologic injury.

Q. So typically, as you understand it, you have to have sepsis leading to shock, and then the cytokines that are produced within that process itself may lead to subsequent and additional injury or may themselves be the cause for neurologic injury once you have got shock?

MR. FINELLI: Objection.

THE WITNESS: No. I think at that point the shock is what is causing neurologic injury. We know that happens.

BY MR. SCHOBERT:

Q. So --

MR. FINELLI: I --

BY MR. SCHOBERT:

Q. -- you don't believe that there's --

MR. FINELLI: I'm sorry. Go ahead, Doctor. I interrupted.

THE WITNESS: One can induce shock in other ways than cytokine induced shock.

You can develop hypovolemic shock or cardiogenic shock without cytokines and certainly infarcts of your brain, cerebral edema, terrible brain damage.

There are no cytokines. It's the shock that's causing that process.

So I think when you deal with septic shock, it's the shock that is going to cause the brain damage if it occurs, not the cytokines.

BY MR. SCHOBERT:

Q. So is it your opinion that there is no -- there is not significant enough evidence that cytokines, whether they are produced by any process, can result in additional harm to the individual? In other words, is there any link between --

MR. FINELLI: Direct or indirect?

BY MR. SCHOBERT:

Q. -- the production of cytokines and harm neurologically to an individual?

A. Certainly if a child has -- you said under any circumstances.

Certainly under the circumstance of bacterial meningitis, where there is direct invasion of tissue, meninges, and maybe even cortical brain, with involvement of blood vessels, and then liberation of cytokines at that spot, I think, yes.

I think the cytokines are potentially directly involved in the damage to that individual's vasculature and thereby directly

involved in damage to that individual's brain.

But that's a far different piece than circulating cytokines coming from the chorioamnion into the baby from the maternal circulation causing only brain damage.

Q. All right. So, outside of that example you just gave me in terms of cytokines being produced in meningitis, is there any other -- you know, any other area where cytokines are produced by these other processes that we have talked about, how they can be produced and a variety of processes where you believe they are directly involved in harm to the vasculature resulting in neurologic injury?

A. Not unless it goes through a shock syndrome.

Q. Okay. Now, there is literature -- Mr. Margolis has objected a couple of times.

There is literature that I think has been out there concerning cytokine production with infection in preterms.

Have you read that literature?

A. Yes, I have.

Q. Do you believe that that literature has established to your -- in your opinion a linkage between those events, a preterm infant infection, cytokine introduction, neurologic injury?

A. No, I don't believe so.

I think if one looks at that literature -- and it has been a long time since I have, but I have some recollection of that literature -- one can find other intervening causes in almost all of the babies written about purporting to show cytokine direct damage from chorioamnionitis causing PVL in babies.

And by that I mean many of these babies are intubated at birth and ventilated for weeks because they have severe RDS, or they have persistent fetal circulation postnatally with profound hypoxia for a long time.

It's just very hard to tease out the subsequent neonatal course of many of these premature babies and be able to say there was nothing else but the cytokines from the

chorioamnionitis; therefore, we believe it was directly inducing brain damage, PVL, in these babies.

Now, if you send me some literature and you want me to read it, I'll be more than happy to do it. And maybe there is more than I have read recently, but that's my residue of that data.

Q. All right. Doctor, when we talk about literature, are there certain periodicals or journals that you yourself read on a routine basis?

A. Yes.

Q. What would those be?

A. Pediatric Infectious Disease Journal, Journal of Pediatrics, Pediatrics, New England Journal of Medicine, sometimes JAMA would be the major journals.

Q. Doctor, you have used the term birth asphyxia both in your report and in some of the conversation we have had here today.

Define that term as you use it.

A. Not being a neonatologist, but to me birth asphyxia is hypoxia and hypercarbia.

Q. Is birth asphyxia a process or a description of a condition at a given time?

A. I think it can be both.

Q. All right. Tell me how it can be both. How is it a process?

A. It's a process in that it evolves over time. A baby starts off with let's say lack of profusion, and the process of asphyxia starts.

If that obstruction to profusion is very brief, the baby will not have hypoxia and will not have hypercarbia and would not then be described, should the baby be seen, as an asphyxiated baby.

The other way in which the term is used, and it's a more clinically common use of the term, is in describing a way a baby is at the moment of, let's say, birth and saying the baby is floppy, has no heart rate, has no resp -- no respiratory, has no tone. The baby is asphyxiated.

And a blood gas then may be done which would show a hypoxia and hypercarbia and the effects of that on the ph, and that would be the clinical picture of a baby who has been asphyxiated

in utero.

MR. FINELLI: Just for clarification, can you tell the court reporter hypo or hypercarbia?

THE WITNESS: Hyper, E-R.

BY MR. SCHOBERT:

Q. What values, Doctor, if any, do you utilize to try to determine -- when we are talking about a description, say, at the time of birth, what values would you rely upon or clinical findings would you rely upon to say I believe asphyxia exists in this infant?

A. I think the clinical descriptors that I have just given would be commonly used to describe an asphyxiated baby, a baby with very poor tone, with no respirations, with poor color and profusion, potentially with a low heart rate or potentially even with an absent heart rate would typically describe an asphyxiated baby.

In terms of blood gas findings, the PCO2 would be elevated, usually above 45, the PO2 would be very low, under 20, and the ph typically is very low, usually less than 7.1.

Q. When we utilize asphyxia as a description of a condition that exists, say, at the time of delivery, there are many causes for asphyxia; correct, when we describe it as a condition --

A. Sure.

Q. -- at a given moment?

A. Yes. There are many in utero causes of asphyxia.

Q. And you have already agreed that one in utero cause of asphyxia could be septic shock?

A. Or just sepsis, and then the birth process is enough to asphyxiate some babies, yes.

Q. In this particular case, is it your opinion that it's sepsis plus the birth process that lead to -- well, strike that. Let me start over.

First of all, it's your opinion that Mackenzie Tarle was asphyxiated by your definition at the time of delivery.

Is that a fair statement?

A. Yes.

Q. And tell me all of the factors that lead

you to that opinion from -- I know your general categories, but tell me what you found in the clinical record that causes you to reach that opinion.

A. Well, the baby needed to be resuscitated. The baby had such poor functioning at birth, the baby needed to be very aggressively resuscitated. And the baby was found not to have an adequate pulse at birth, found to be limp and apneic at birth and to have blue color at birth.

This is all based on the Apgar score of 2 at one minute. And I believe some of the descriptions in either depositions or in the record say the baby's heart rate was less than a hundred at the time of birth.

There is a blood gas, but I believe the blood gas wasn't done until about an hour of life, 6:21 a.m., in fact, a little bit more than an hour of life, where the baby still is significantly acidotic, but is being very well ventilated and is reasonably well oxygenated at that point, having then reserved the hypoxia and the hypercarbia.

But the acidosis, which takes a while to correct, is still present.

Q. All right. Any other factors you are relying on as it pertains to your opinion concerning Mackenzie Tarle being asphyxiated at the time of birth?

A. I believe that was the description or the diagnosis of the baby throughout the hospital records at the Akron Children's Hospital and also at the General Hospital when being evaluated for the neonatal condition.

Q. All right. Now, when we talk about sepsis in utero, how do we -- what clinical factors can we utilize to rely on making a determination that sepsis would have occurred in utero in a general context?

A. Well, probably in order to know that sepsis occurred in utero, probably the most important determination is to know that a blood culture taken at about the time of birth grew the offending organism.

And in this case, we know that that was

in fact the case. The blood culture taken on Mackenzie Tarle was positive for Group B strep. And that connotes in utero involvement with Group B strep.

And that's not a rare feature of Group B strep. More than half of babies born with Group B strep disease in fact have acquired that infection in utero.

Q. Anything else that we can look at to -- in a general sense or in this case, utilize to support that Mackenzie Tarle did have Group B strep sepsis in utero?

A. The initial blood count that was taken at Akron General Hospital, which was -- I don't have the time that it was taken, but it was taken shortly after birth -- had a white count of 11,800, which is a normal white count.

But the baby had nine segs and 18 band forms, which would be commonly seen in a septic baby, that is to say the elevated number of bands. And in this case, the abnormal IT ratio would be part and parcel of a septic syndrome.

Q. Doctor, how would we define or determine if an infant, a fetus, has suffered septic shock in utero?

A. I think what one would turn to is the clinical course of events post birth to determine whether or not in utero the baby might have been with septic shock.

And in Baby Tarle's case, there was no ongoing shock syndrome in the days following birth. The baby had severe asphyxia and the effects of that, which lasted a number of hours, but subsequently had no evidence of ongoing shock, which would mean to me that the baby was not in shock in utero.

Q. Let me ask it in a generic way, and then we will get specific to this case.

How would -- do you agree with me that in a general sense, an infant could suffer septic shock in utero, a fetus could suffer septic shock in utero?

A. Certainly.

Q. We have already talked about how we might

try to diagnose sepsis in utero. My question to you is how would you, as a pediatrician, diagnose if an infant has suffered septic shock in utero in general?

What factors could you look to or, you know, evaluate to determine that?

A. Well, the in utero factor -- I mean, the only thing that jumps to mind as it related to the in utero well-being of a baby would be persistent fetal tachycardia as a response to the shock syndrome. That would be a common feature of the septic shock in utero event.

There would be no other easy way, other than putting a catheter in the baby and getting a mean arterial blood pressure, in my judgment, of knowing whether a baby had septic shock in utero until the baby is born.

And then if the baby post birth went on to have a prolonged episode of septic shock that went on for a number of days, the question would be open as to whether the baby might have been in shock in utero. And you might or might not be able

to come up with some reasons for believing such as an infant would be in shock in utero.

On the other hand, if a baby is born and post birth is not in septic shock, I think it is very strong evidence the baby was not in septic shock in utero, even if the baby was septic in utero.

That is in fact what we have for this baby, for Mackenzie Tarle's case.

Q. All right. So in your opinion, just so I'm clear on this, you believe that from the moment of delivery thereafter, there is no evidence that Mackenzie Tarle was in septic shock?

A. From the moment of birth, the baby was definitely in shock.

And whether it was because of the combination of birth asphyxia and sepsis, a small component of septic shock at that minute, I don't know. I don't know how anybody could really know.

All I can tell you is significant septic shock doesn't go away with a couple of boluses of normal saline and one bolus of 5 percent albumin.

It is a syndrome that is multiorgan in nature, persistent, and goes on for a number of days.

And Mackenzie Tarle was on room air on the day of birth, had very low vent settings, never needed ionotropic support for her cardiovascular status, was put on fluid restriction after birth, not extra fluid for ongoing cardiovascular support, but in fact fluid restriction.

Never developed respiratory failure. Never had DIC. There is just an awful lot in the record that says once the baby recovered from the shock associated with asphyxia, whether there was a part or not that was contributed by the sepsis, I don't know.

But once the septic -- I'm sorry. Excuse me. Once the asphyxic shock was gone, the baby had no more shock.

To me that means there was no septic shock in utero.

Q. All right. Again, if you could just answer my specific question. And then if you don't

understand it, tell me. If not -- if you can't answer it the way I propose it, then just tell me.

In your opinion, at the time of the initial evaluation immediate post delivery, from the first evaluations, was Mackenzie Tarle in shock?

A. Yes.

Q. All right. Next, in your opinion, was Mackenzie Tarle therefore in shock prior to delivery?

A. I don't believe she was in shock.

And by prior to delivery, let me just say within an hour of delivery. I don't -- I mean, an hour going backwards in labor time, I don't think she was in shock.

Q. Well, at any point prior to her delivery in your opinion, based on a reasonable medical probability, was Mackenzie Tarle in septic shock, whether it be one minute, one hour, or one day?

Was she ever in your opinion in septic shock prior to her delivery?

A. It is a very hard question to answer with

respect to the sequence of events that happened in the half an hour before delivery and then the immediate post delivery period.

Because we know she was septic. There is no question about that. She had a positive blood culture in the CBC that we have already talked about. And we know she was in shock.

And so the question that you are really asking me is whether or not sepsis played a part in that shock, and, therefore, would some people term it septic shock.

And looked at that way, yes, I would say it's reasonable to say she had a variety of septic shock in the few minutes before delivery, for the few minutes after delivery.

Because without her being septic, I doubt she would have been asphyxiated and therefore she wouldn't have been in shock.

So it's almost a logical argument to say, I could call it a variety, if you will, of septic shock. But it was very brief and very limited to those few minutes before delivery and several hours

after delivery.

But it was not the kind of shock that was ongoing in the hours before she came out. And I could tell you that because of the sequence of events after the third hour of life.

I hope that was directed to your --

Q. I understand.

Were there also positive cultures from her lung with GBS after her -- you know, after delivery?

A. I don't believe so.

Q. If there were positive lung cultures with GBS after delivery, what, if any, significance would that play in your assessment of what transpired?

A. Really no significant assessment.

The majority of babies with Group B strep early onset of disease actually have the lung as the focus of that infection. But in a sense, the proof of the pudding is in the eating.

What was her pulmonary physiology after birth? She was on room air on day one of life,

didn't need the ventilator except to protect her airway, did not have a whiteout of the lung, did not have elevated pulmonary pressures, all of the things that one sees with severe pulmonary involvement with Group B strep.

Again, not to say that she didn't have Group B strep sepsis. I just don't think the lung was a major component of that based on how she was to ventilate and what her pulmonary function was over the days following birth.

I'm aware of a gastric aspirate that was positive for Group B strep, but I'm not aware of a tracheal aspirate. Maybe I missed it in the record.

Q. All right. Let's go through that. All of these factors that you believe indicate to you that while she had a septic shock maybe in the short time interval that you have defined prior to delivery, while you believe that it is that short time interval.

I mean, you are telling me all of these post delivery factors cause you to come to that

opinion. So let's go through them so I have them listed as to all of the things you believe in the record support that opinion, that it was a very limited amount of time that she would have had GBS septic shock prior to delivery.

MR. FINELLI: Hey, Jeff, he also mentioned the in utero factors, which include fetal tachycardia.

MR. SCHOBERT: Well, again, guys, I mean, I'm trying to ask him the questions. If he feels that he has not been given the chance to explain...

BY MR. SCHOBERT:

Q. Doctor, do you understand my question?

You have voiced for me a number of times the things that you believe support your opinion concerning a very limited time frame in which there could have been a GBS septic shock prior to delivery; correct?

A. Correct.

Q. All right. Tell me what all those factors are just so I now clearly have them listed down here.

And I assume what you are looking at is all of the post delivery things you have tried to tell me in part about, pulmonary findings, pressors, and those things.

A. I'm confused about your question.

You want my reasons for my belief that this variety of shock that this girl had in the delivery room, whether we call it septic shock or not, did not continue after the first hours of life?

Is that your question?

Q. No. I think your testimony to me, if I have understood it, is that while you acknowledge that there was -- there is a Group B sepsis in utero, you do not believe that a Group B sepsis shock would have been existed in utero except for a very limited amount of time potentially prior to delivery and may have contributed in part, although not a substantial part, to this child's asphyxia.

Is that what you have been trying to tell me?

A. Well, I don't know if that last part

would be truly my opinion.

I think the Group B strep was a significant reason for her to be asphyxiated, as so babies with Group B strep are asphyxiated.

And that is a common reason for asphyxia. Group B strep disease, even without septic shock in utero, can be enough that makes it impossible for a baby to go through the rigors of delivery.

And, for instance, if I gave Mr. Finelli Group B strep sepsis right now and asked him to go out and run ten miles and he collapsed on the seventh mile in shock, you know, that's septic shock.

It's an unfair variety of septic shock because we have now put two burdens on his cardiovascular system which probably could handle the run without a problem or could have handled the sepsis without a problem, but together they were impossible for him to confront.

Well, that's what Group B strep sepsis does to newborn babies. It makes the ability to go through the delivery process a severe enough stress

that the babies are born asphyxiated and in shock.

And in that way, as I stated to you before, I could say in a logical sense, yeah, that's a variety of septic shock, but it's not septic shock that happens in and of itself. It's septic shock that's pushed along by some other process, in this case the delivery process.

The reasons --

Q. Well, let's --

MR. FINELLI: He's not done.

THE WITNESS: The reasons I think the septic shock, if we call it that, was brief and only related to the delivery process are reasons that I have alluded to before.

And they include the normal respiratory function of this baby from the first day of life onward. The baby needed room air and only room air within hours of birth.

The baby was on a ventilator only to protect the airway by the second day of life, not because of respiratory failure, but because the baby was seizing and they wanted to protect the

airway.

The baby's cardiovascular system was not in any way compromised to the point where the physicians dealing with this problem at Akron Children's Hospital needed to use cardiovascular support as you would have to in a baby with septic shock.

The fluid administration that this baby received was not the fluid administration for septic shock. In fact, they restricted fluids on day one of life.

If this baby was in septic shock, I submit to Mr. Finelli, he sued only part of the people who should have been sued, he should have sued the doctors in the neonatal intensive care unit who didn't treat her for septic shock because they only gave her 50 ccs per kilo on the first day of life, which would be about two-thirds of what an average baby would require just for maintenance.

They did not give her any pressors on the first day of life, as you would have to if she was in septic shock, because her cardiovascular system

was fine.

She had a capillary fill time of less than three seconds. She had a mean arterial blood pressure in the mid 40s. She had a pulse rate that normal for a baby of her age, no evidence of ongoing cardiovascular inability to deliver substrate to tissue, which is what shock is.

So lung function was fine. Cardiovascular function was fine.

Thirdly, there was no evidence in the record that she developed the bleeding diathesis that we often see in septic shock, namely disseminated intravascular coagulation.

One can see that in many, many syndromes, but septic shock certainly is -- goes hand in hand with septic shock when it occurs.

She didn't have that.

So I don't see any evidence in the record following her resuscitation that she had shock of any kind. And that's why I don't think she had septic shock in the general use of that term after delivery or prior to delivery, except for that very

localized time period during delivery and right after in the delivery room where I could agree with you in a logical way, you could call it a variety of septic shock, brief and limited and helped along by the delivery process.

Q. Doctor, Mackenzie Tarle did receive pressors, did she not?

A. On the second day of life, she received a renal dose of dopamine, three micrograms per kilogram per minute, which is not for cardiovascular support, but rather to increase renal profusion to prevent her from getting worse renal dysfunction.

Q. And, as you say, she was intubated; correct?

A. She was intubated, yes.

Q. How many days did she remain intubated?

A. I believe she was intubated for ten days. There was a brief period of trying to extubate her, but she was intubated for ten days to protect her airway.

Q. Doctor, you have reviewed the medical

records from obviously Akron Children's Hospital; correct?

A. Yes.

Q. It is your opinion that the care rendered by these physicians was reasonable and appropriate?

A. Yes.

Q. You have seen, therefore, their diagnoses that they placed in this chart at the time of her discharge from Akron Children's Hospital?

A. I think I have seen that, yes.

Q. Are you in agreement with them that she had a Group B streptococcal sepsis?

A. Yes, she did.

MR. MARGOLIS: And, Doctor, take your time and review the document that Mr. Schobert is asking you to review.

THE WITNESS: Well, I'm looking at a document here from Children's Hospital that I think is the discharge document 8/20/98 timed at 1730 hours.

BY MR. SCHOBERT:

Q. That's the date I have. I have a summary

of that document, Doctor, prepared by a Dr. Vollman.

A. I can't read the handwriting.

Could you hold up the document that you want me to look at?

MR. FINELLI: Are you looking at a discharge summary?

BY MR. SCHOBERT:

Q. I'm looking at a discharge summary that is dated 8/20/98 prepared by a Dr. Vollman.

A. I was looking at the face sheet.

MR. MARGOLIS: Take your time.

THE WITNESS: We are having trouble finding the discharge summary.

Do you know where in your records that appears?

BY MR. SCHOBERT:

Q. Doctor, as I have tried to explain -- I know it's difficult with this medium we are utilizing -- I did not bring a full set of records with me.

I have a pretty comprehensive summary

that has been prepared for somebody with my limited abilities, so I'm reading from that rather than looking at a specific document.

But I know it's in there because I have gone through this and verified it at an earlier point.

MR. MARGOLIS: Dan, it's in the Akron Children's Hospital chart.

MR. FINELLI: Yeah, I know.

We just...

THE WITNESS: Some of my Children's is mixed in with my Akron General.

MR. MARGOLIS: Just take your time because I want you to be able to review the document before you answer questions about it.

(A discussion was held off the record.)

BY MR. SCHOBERT:

Q. Doctor, take a moment, as Mr. Margolis has indicated, Doctor, and look at that document.

A. Do you want me to read the whole thing, basically, do you want me to?

MR. FINELLI: No, you don't want him to

read it.

BY MR. SCHOBERT:

Q. It's not my instruction. It's --

MR. MARGOLIS: Yes, Doctor. I'm asking that you take a moment and you review the discharge summary in its entirety because Mr. Schobert is going to ask you questions, and I want you to have reviewed it.

THE WITNESS: Okay.

MR. SCHOBERT: Maybe the court reporter can read back the question I had proposed to you at the time so we can pickup in context.

Do you have that Mr. Court Reporter?

(A discussion was held off the record.)

BY MR. SCHOBERT:

Q. Well, let me see if I can rephrase it.

Doctor, I think what I was going to ask you was, Do you agree with the diagnoses placed in the chart by the physicians from Children's Hospital that cared for Mackenzie Tarle?

And I was going to ask you first, number one, I -- the first diagnosis I see written is

Group B streptococcal sepsis.

And by your testimony, you agree that that was a diagnosis for this child; correct?

A. Correct.

Q. Number two says, Shock due to sepsis and blood loss; correct? That's what they have written down?

A. Yes, that's what they have written down.

Q. Do you disagree with them when they write that this child had shock due to sepsis and blood loss?

A. I think the conclusion about blood loss is more a logical conclusion than a proven fact in the record.

And I so that for the same -- and I would probably come to the same clinical question myself in dealing with a baby whose hematocrit dropped as much as this baby's hematocrit dropped in the several days from birth to I think to August 7, her hematocrit was 29.

That to the treating doctors suggested some blood loss in utero.

Whether that's true or not, I don't really know because sepsis, Group B strep sepsis is another reason for blood loss. And taking blood out of a baby and administering fluids and so on can cause hemodilution.

So this is -- I'm not disagreeing with the possibility of blood loss, but I don't think it's truly established in the record.

Q. Okay. How about No. 3, Delayed adaptation in the delivery room?

A. Certainly, absolutely.

Q. All right. And No. 4, Hypoxic ischemic encephalopathy. You agree that's a diagnosis?

A. Yes.

Q. Going down through the rest of those lists, is there anything on that list from the next -- from 5 through 9 that you disagree with in terms of diagnoses?

A. No. They were all diagnoses to this baby at some point during the course of her life in the first two weeks of her life, yes.

Q. Doctor, what role, if any, do you believe

a review of the placental pathology could play in determining the length of time that an in utero infection may have existed?

MR. FINELLI: Objection. He is not a pathologist.

MR. SCHOBERT: I'm just asking generally whether he believes, based on his knowledge -- from his background of pediatrics, whether that placental pathology could play a role in determination of the length of time infection may have existed in utero.

MR. MARGOLIS: Objection.

BY MR. SCHOBERT:

Q. And if you don't have an opinion, that's fine.

A. I don't have an --

Q. I just want to know.

A. I don't have an opinion about that.

Q. What about the utilization of head studies, MRI, ultrasonography, or CT imaging of an infant's brain?

Do you believe that it can play any role

in the timing or in a determination as to a causative event of neurologic injury in the newborn?

MR. FINELLI: Objection.

THE WITNESS: I don't have an opinion about the usefulness of CT scans, MRIs, and ultrasounds in trying to time Mackenzie Tarle's injury.

BY MR. SCHOBERT:

Q. Now, you made a -- one of your early notes that you read to me from the front of the transmittal letter I think said, If delivered plus or minus six hours?

A. Yes.

Q. All right. Explain to me, if you would, in a little more detail what you mean by that statement, and then I'll ask you some subsequent questions.

A. Well, I know the delivery time of Mackenzie was 5:16 a.m. And so I think what I meant by writing this is that, you know, by 11 o'clock at night, there is a fair chance the baby

didn't have Group B strep sepsis yet, which would be another way of saying if she were born, she would never have Group B strep sepsis. And any issue relative to Group B strep sepsis would be evaporated.

Or that if she did have Group B strep sepsis, it would have been so early that there would be no chance that it would participate in an event that might cause shock, brain damage, the things that we have been discussing now for the last hour and a half.

Q. Doctor, how do you make the determination when there is significant enough Group B strep sepsis that might be going on in the fetus, that the rigors of delivery -- in combination with the rigors of delivery, the child could suffer shock and the result asphyxia.

How do you determine how much sepsis is enough in order that a child cannot undergo the rigors of delivery without some resulting sequelae like this?

A. I don't know any operative way to

definitively say that.

I would think that cesarean section delivery induces less interruption of profusion of the baby's organ systems such as that cesarean section would be less of a challenge to any baby with Group B strep disease at any stage of that evolution.

And I believe --

Q. Well, you said -- go ahead. I'm sorry, Doctor. It's the medium. I apologize. Go ahead and finish.

A. And so that the delivery that I'm talking about here was a cesarean delivery. A C-section done at midnight or 11 o'clock at night, more likely than not, would have been before the baby developed Group B strep or well before any issue could probably arise that Group B strep could complicate that more lenient variety of delivery.

Q. All right. And my question then to you is give me the reasons or the, you know, what you are relying on to say that, A, you don't believe that the child would have had developed Group B

strep sepsis by 11 p.m. the evening before.

A. Well, again, I didn't say that.

I said the combination of those two things would more likely than not give rise to a health baby. That is to say, there was the significant chance Mackenzie Tarle had not yet developed Group B strep by 11 o'clock at night.

Whether that's more than 50 percent likely, I don't really know, but a significant chance the baby was not yet infected.

And if the baby were infected, it would have been so early in the disease that I would aghast at the idea that a cesarean section delivery would be a challenge to the baby who was early with Group B strep disease.

But the reason for thinking that Group B strep was less likely at 11 than it was at 5 is the average thinking about the evolution of Group B strep disease in any neonate, the early onset variety anyway.

And that is it follows suit with maternal chorioamnionitis. Membranes are ruptured over a

period of hours. Those membranes may become infected. And by about 12 hours, that gets to be a common enough thing, chorioamnionitis gets to be a common enough thing that one could pose the likelihood in a baby like Mackenzie Tarle and her mother that chorioamnionitis was established by about 12 hours after ruptured membranes. That would be average.

And then usually it takes several hours after that for a baby to become infected with the Group B strep or other organism that is causing the chorioamnionitis.

So if one just does the average progression of events, membranes are ruptured at 7:25, I think or 7:30.

MR. FINELLI: 7:45.

THE WITNESS: 7:45 in the morning. 12 hour later, it is 8 p.m. at night. Several hours after that Mackenzie Tarle is infected.

If everything follows the way it's supposed to follow, that's 11 o'clock at night. That's six hours before the baby is born.

And that's where that number comes from.

BY MR. SCHOBERT:

Q. Well, Doctor, let me see if we can just cut to the chase.

Do you have an opinion based on reasonable medical probability when this infection was -- when the organisms that resulted in infection were first introduced into the amniotic fluid?

A. Into the amniotic fluid?

Q. Well, yes.

A. The way you asked the question, I would probably think within several hours of membrane rupture.

Q. So it's your opinion -- let's ask it this way. Prior to membrane rupture, it's your opinion based on a reasonable medical probability, that there was not the introduction of a Group B strep bacteria into the uterus?

A. Yes, more likely than not.

Q. And the basis for that opinion is what?

A. Is that typically membrane rupture is

what allows Group B strep to ascend into the uterus of term pregnancies.

And that's quite different from preterm pregnancies where in fact the pre-existing chorioamnionitis can be a cause for premature rupture of fetal membranes.

This was at term, actually a little bit post term.

Q. You say typically it is introduced at the time of rupture of membranes.

The implication of that is that there are times when the GBS bacteria could be introduced even prior to the rupture of membranes.

Is that an inaccurate statement?

A. That's true. That can happen.

Q. Is there any way in the case of Ms. Tarle that you can rule out the fact that this could have been introduced prior to the rupture of membranes based on all of the materials you have reviewed?

A. I think a couple of things. The two things that would argue against this Group B strep having been there for 21 hours before Mackenzie's

delivery, rather than six or eight hours before Mackenzie's delivery, is the absence of severe Group B strep disease in Mackenzie herself, which means she probably had a pretty early infection at the time of delivery.

And then number two, the presentation of maternal fever, which I think was at about 4:30 in the morning, something like that -- let me get the right time. 4:25 was the temperature of 38.5.

I would expect Ms. Tarle to have fever earlier than that if in fact she had amniotic infection prior to the rupture of her membranes. I would have not have expected her to wait 20 hours for the fever.

Q. Any other factors you are relying on?

A. I think they are the only two.

Q. All right. Now in your opinion, based on reasonable medical probability, when did sepsis arise in Mackenzie Tarle?

A. I can't give you an exact moment in time. I could give you a range of when I think Mackenzie probably became bacteremic or septic with Group B

strep.

And that range of time would be, knowing that she was born at 5:16 or 5:17, I would say on the long end, 12 hours before birth, on the short end, a couple of hours before delivery would be the range of when it --

Q. And again -- I'm sorry. I apologize. I should look at you. I stop hearing you for a second and I think you are done. I'm sorry.

A. Those would be I think the ranges, the long end and the short end, as to when Mackenzie might have become infected with Group B strep.

Q. And, again, the factors you are relying upon for that opinion.

A. The factors are the absence of severe disease in the baby post birth, again meaning probably pretty recent infection.

And then number two, the statistical knowledge that most Group B strep in most term babies occurs after a prolonged period of fetal membrane rupture.

And we know when the membranes ruptured.

And usually there is a quiet period before Group B strep gets to anything after membrane rupture.

MR. MARGOLIS: Can we take a break?

MR. SCHOBERT: Yeah, I'm almost done, but go ahead.

BY MR. SCHOBERT:

Q. Anything else, Doctor, but before we take a quick break?

A. I think not.

(A recess was taken at 10:56 a.m. until 11:03 a.m.)

BY MR. SCHOBERT:

Q. Doctor, in your opinion, based on reasonable medical probability, at what point -- by what point did this child need to be delivered by any means to avoid neurological sequelae that this child has experienced?

A. Well, I'm not an obstetrician, but I'll answer it as I would a pediatrician looking at --

Q. Well, I'm asking you to look at it from a damage standpoint.

By when do you believe a delivery -- and

I'm not asking you to talk about obstetrical care but when you believe delivery would have -- could have contribute -- if it occurred by that point, you believe based on a probability this child would not have suffered any neurological sequelae or at least a different outcome in that regard?

A. Right. Well, I think it goes up to that period of time when the prolonged bradycardia began, whether that would be 20 minutes or a half an hour before delivery, I don't remember as I sit here, but in about that period of time.

I think cesarean section delivery would have prevented the complication of Group B strep from causing disease of the baby's brain.

Q. And, again, give me just the overview of factors in support of that opinion so I have a clear record to review in that regard.

A. Okay. I think it's pretty much what we have already talked about several times, and that is the delivery process, a vaginal delivery, because it can interfere with blood supply to babies by compression of the cord, during sepsis,

that interruption can be catastrophic.

And I think that's what happened here, and I think that's what the bradycardia in this baby's case really was reflecting, the combination of the stress of labor on top of the stress of Group B strep disease caused this baby to have very poor profusion of brain.

If you got to the baby by cesarean section before that stress was put upon the Group B strep disease, the baby would not have had brain damage.

Q. When you say the stress of labor is put upon the Group B strep -- I think that's how you said it. If I misphrased, that correct me -- but explain that path of physiology to me. I guess I want to understand what you mean when you say that.

A. Well, I really mean the stress of the delivery process, when the baby is coming through the lower birth canal and the umbilical cord is compressed, either from contractions or because of the baby's position, that poor profusion of the baby, who is already stressed with sepsis, is what

puts the baby into an asphyxial mode with poor profusion of organs, hypercarbia, hypercapnia, acidosis, and the ensuing brain damage.

Q. So what effect is the sepsis having?

Let's just say the moment before the delivery process beginning, where she starts to push, and the baby starts -- they start into that second stage of labor.

What is the sepsis doing to that fetus at that moment?

A. Well, I think it is probably taxing the baby cardiovascularly, making it harder for the baby to provide substrate to every organ.

The baby is working maximally to do that. And I think I used the example of someone doing a very arduous physical activity where your cardiac output is now sort of maximized, and then you are asked to do more.

You can't confront that extra, so you go into a failure mode, in this case shock.

Q. Okay. And this blood loss that we had talked about as being something that was in the

diagnosis at Children's hospital, would it be fair to state based on a probability that this process that's going on at that point leading up to the beginning of the pushing, the labor, the actual delivery itself, was there blood loss occurring to the child at that point?

A. Well, I don't believe there would be quote, unquote blood loss occurring to the child because of Group B strep disease.

That disease can cause hemolysis. And if it's longstanding enough, you know, a baby can be born with a very low hematocrit.

That's not the case for Mackenzie. She didn't have a low hematocrit.

The kind of hemologic features she did manifest are more consistent with the real true blood loss, like an abruptio placenta or a ruptured cord or some other significant gross bleed that deprived the baby of blood volume.

Not the kind of anemia you see with severe sepsis where the blood volume isn't changed, the amount of circulating red cells is changed.

She didn't have that.

So I don't think the Group B strep in this case is the etiology for this quote, unquote blood loss, if it occurred.

And I don't have an etiology. I know there is talk in the record about an abruption. I have no opinion about any of that, and I don't even know that for a fact she had blood loss.

Q. Well, I thought you said to me in some earlier testimony that there was evidence of a dropping hemoglobin hematocrit in her post delivery records. Is that correct?

A. That is true.

Q. And what is your opinion based on reasonable medical probability the cause of that?

A. I think there are two ways to explain it.

One is that the baby had blood loss somehow, and that blood loss could be post birth or prebirth.

It could include things like attempting to put umbilical arterial catheters in and some bleeding at that particular emergency time. It

could be bleeding into the lungs from Group B strep pneumonia. It can be bleeding into the GI tract, bleeding into the head from poor profusion and infarcts.

There can be a lot of ways in which a baby can have blood loss post birth. It could be prebirth. We already talked about abruptio. I have no idea, but obviously that's a classic cause in the way of a baby with blood loss.

The second way in which this baby could have had blood loss is the typical way in which sick babies in intensive care units develop anemia. It is iatrogenic. People have taken out so much blood over the first three days of life for chemistries, blood gases, CBCs, you name it, that they get to a point where they have made the baby anemic and have to transfuse the baby.

And on top of that, a baby with Group B strep disease, you can expect some hemolysis.

So I can't sit here and tell you amongst those possibilities what was the cause of this baby's anemia. It could have been any of those

causes.

But I would agree, the baby did develop anemia and actually had a hematocrit of I believe 26 or 29 on the third day of day of life, which obviously has to have a reason behind it.

And I think I have given you sort of a panoply of the possible reasons.

Q. Hypothetically, Doctor, if you had a child that had developed Group B sepsis in utero and had a significant -- or had a blood loss in addition to that, would that make them even more likely for some type of neurologic sequelae, the combination of those two events?

A. I think absolutely it would. I think those two events would be yet another stress on this baby's capability of delivering substrate to the brain.

And an in utero significant anemia and Group B strep and delivery could be a catastrophic combination for a baby.

Q. Now, Doctor, hypothetically if Mackenzie Tarle had been delivered vaginally at midnight

based on your opinions concerning the underlying sepsis that she may have had at that juncture -- and I know you have explained all of that, and I'm not trying to go back and recreate all of that.

But assuming hypothetically she was asked to undergo the rigors of delivery at that time, a natural delivery, do you have an opinion as to what her outcome would have been in this case?

MR. FINELLI: Objection.

THE WITNESS: I think statistically it would have been better. More likely than not, she would have had a better outcome.

Much more than that, I can't say.

BY MR. SCHOBERT:

Q. So you have no opinion as to whether or not she might have still suffered some degree of hypoxic ischemic encephalopathy based on the combination of the rigors of the labor and delivery process as well as the underlying Group B sepsis to the extent it would have existed at that time?

MR. FINELLI: Objection.

MR. MARGOLIS: With vaginal birth?

MR. SCHOBERT: With vaginal birth.

MR. FINELLI: Objection.

THE WITNESS: I guess I would agree with the way you phrased my opinion.

There would be some subset of babies like Mackenzie delivered at midnight by vaginal delivery who would not yet have had Group B strep disease.

In which case, her risk of brain damage is whatever the risk is for any baby, term baby with a vaginal delivery.

There are other babies who would have had Group B strep syndrome for, you know, one hour, and be so early in it that the vaginal delivery would not impact on the Group B strep syndrome; or, to put it better, the Group B strep syndrome would not impact on the vaginal delivery.

And you could do that almost like in an integral fashion. If you are familiar with calculus, for every hour or every increment of time where you add -- okay, now she has had Group B strep for two hours, what would that mean.

But I think in general, you would wind up

with a better outcome in Mackenzie at midnight delivering vaginally than you would at 5:30 delivering vaginally, but I can't say with more likely than not certainty it would have meant a normal Mackenzie Tarle.

Q. And, Doctor, if I understand it, in your opinion -- again, in a hypothetical, assuming that Mackenzie Tarle had not had Group B strep sepsis at any time prior to her delivery, and assuming that she had gone ahead and vaginally delivered in that time frame of 5 to 6 a.m., it's your opinion based on a reasonable medical probability she would have -- you see no reason that she would have had any asphyxia leading to potential neurologic injury.

Is that a fair statement?

MR. FINELLI: Objection.

THE WITNESS: It's a fair statement if I'm able to discount the issue of abruptio placenta that I read in the record, but which I don't find substantiated anywhere.

Clearly a baby with abruptio placenta can

have fetal distress and then be born asphyxiated.

And I know it's stated in the record that there might have been an abruptio placenta, but I don't know that, and it's not further clarified in any of the documentation that I read.

So if we exclude the abruptio placenta, then I would answer the way I have answered.

Q. Doctor, if cytokines are produced as a result of septic shock, which I think we have discussed you believe that is one way they can be produced --

A. Cytokines can cause septic shock.

They are not produced as a result of septic shock. They are the cause of septic shock.

Q. All right. So sepsis results in the production of cytokines, which then result together in septic shock.

Is that simplistically what you are telling me?

A. Yes. Can result in septic shock.

Q. Can result?

Once cytokines have been produced as a

result of the underlying sepsis, is there anything that can be done treatmentwise to stop whatever effect these cytokines may have on the vasculature of an individual?

A. There may be some experimental modalities of therapy, but there is no clinically available standard therapy that can reverse the effect of cytokines until they have gone by their half-lives.

Q. Doctor, in the discussion of cytokines, I have -- strike that. Let me ask it this way.

If you have somebody with sepsis and you begin the antibiotic therapy for the sepsis itself, what effect, if any, does that have on cytokine production?

A. I think it has two effects.

Oftentimes, there is an initial effect whereby the death and dying of the organism elaborating more cell wall products or releasing more cell wall products, it has been shown that some patients have a boost in their cytokines transiently immediately after therapy is started.

But the long-term effect of therapy is

to, a day later or two days later in the face of antibiotics, to prevent the growth of these organisms and thereby diminish the production of cytokines.

Q. Doctor, if there was a study that demonstrated that there was a significant association between abnormalities in the neurologic examination and cytokine concentrations with the highest cytokine concentrations observed in infants who developed HIE seizures -- and we are talking about term infants -- would you be in a position to dispute those findings?

MR. FINELLI: Objection.

THE WITNESS: Well, I would be in a position to review the subset of data that --

BY MR. SCHOBERT:

Q. Dispute this -- I mean, would you, based on your knowledge of this area, would you be in a position to disagree that those findings would suggest this association between those two factors?

MR. FINELLI: Objection.

MR. MARGOLIS: I'm going to object, and

I'm going to instruct -- this is Mr. Margolis.

I am objecting. I'm going to instruct Dr. --

MR. SCHOBERT: I will withdraw the question.

BY MR. SCHOBERT:

Q. Doctor, if you assumed that a study had been done where there had been a significant association developed between abnormalities and -- with neurologic exams in the recent -- in a child just delivered, and an association in those same infants of increased cytokine concentrations, what might that say to you as a pediatrician?

MR. MARGOLIS: I will object.

Doctor, he is asking you to make certain hypothetical assumptions.

THE WITNESS: Well, I would want it --

BY MR. SCHOBERT:

Q. I'm asking you to -- go ahead, Doctor. I'm sorry.

A. I would want to know a lot more about the patients than just their neurologic exam and their

cytokine level. That's certainly not the way medicine is practiced, and that's certainly not the way research is done.

I would want to know whether there are any other factors offered in those patients that might bridge the cytokine and the neurologic exam, such as septic shock, meningitis, severe respiratory distress with hypoxia, hypoprofusion of the brain with persistent fetal circulation.

I mean, many other things that might be related to the cytokines, not directly the brain damage, but some other disease that then gives forth the brain damage. That's what I would look at in those -- in that data set.

MR. SCHOBERT: Doctor, I don't have any further questions. Thank you.

MR. FINELLI: We will read and sign.

(Thereupon, Deposition Exhibit No. 1 was marked for identification.)

(Whereupon, the proceedings in the above-captioned matter were concluded at 11:21 a.m.)

CERTIFICATE OF REPORTER

I, Joseph A. Inabnet, do hereby certify that the proceedings in the foregoing matter were taken by me in Stenotype and thereafter reduced to typewriting under my supervision; that said transcript is a true record of the proceedings; that I am neither counsel for, related to, nor employed by any of the parties to the action involved in these proceedings; and further, that I am not a relative or employee of any attorney or counsel employed by the parties thereto, nor financially or otherwise interested in the outcome of the action.

Joseph A. Inabnet
Court Reporter

CERTIFICATE OF NOTARY PUBLIC

I, Thomas W. Olender, do hereby certify that RAOUL L. WIENTZEN,JR.,M.D. was sworn by me on November 26, 2002, at the offices of Olender Reporting, Inc., 1522 K Street, NW., Suite 720 Washington, D.C. 20005.

Thomas W. Olender
Notary Public in and for
The District of Columbia

My commission expires: July 14, 2007

CERTIFICATE OF READING AND SIGNING

I, RAOUL WIENTZEN, the deponent herein, do hereby certify that I have read the foregoing deposition and certify that it is a true and accurate transcription of my testimony given in the above-captioned matter, except for any corrections as noted on the enclosed errata sheet.

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RAOUL WIENTZEN