The State of Ohio,)
) SS:
County of Cuyahoga.)

IN THE COURT OF COMMON PLEAS

Gloria Maslanka,
Individually and as Parent
and Natural Guardian of
Shane Maslanka,

Plaintiff,

vs. Case No. CV-05-552424

MetroHealth Medical Center,

Defendant.

* * *

Transcript of the videotaped deposition Of RICHARD MARTIN, M.D., called as a witness on cross-examination by the Plaintiff, taken before Kathleen A. Hopkins, a Notary Public within and for the State of Ohio, at the Rainbow Babies and Children's Hospital, 11100 Euclid Avenue, Suite 3100, Cleveland, Ohio, on Monday, the 25th day of September, 2006, at 9:45 a.m., pursuant to notice.

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APPEARANCES:

On behalf of the Plaintiff:

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CURRICULUM VITA MARKED PLAINTIFF'S EXHIBIT 1 FOR IDENTIFICATION.

DR. MARTIN'S REVIEW NOTES MARKED PLAINTIFF'S EXHIBIT 2 FOR IDENTIFICATION.

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RICHARD MARTIN, M.D.,
of lawful age, called as a witness by the Plaintiff, being first duly sworn as hereinafter certified, was examined and testified as follows:

CROSS-EXAMINATION OF RICHARD MARTIN, M.D.

BY MR. BECKER:

Q. Morning, Doctor.

A. Good morning, sir.

Q. For the record, please state your full name.

A. Dr. Richard J. Martin, M-A-R-T-I-N.

Q. All right. You have had your deposition taken before, correct?

A. Yes.

Q. Just to review the groundrules, this is a question and answer session under oath. It's important you understand the question that I ask. If the question doesn't make sense or is inartfully phrased, I want you to let me know and I would be pleased to restate or rephrase the question. Fair enough?

A. I will do that.

Q. However, unless you indicate otherwise to me today, I'm going to assume that you've fully understood the question that I have posed and you're

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giving me your best and most complete answer today. Fair enough?

A. Yes, sir.

Q. Doctor, what have you reviewed in preparation for today's deposition?

A. I have reviewed the medical records of Mrs. Maslanka and her child, Shane. I have reviewed three or four depositions. I can list them for you. I think that's about it. A couple of reports.

Q. Okay. You have had an opportunity to see the pediatric infectious disease, Dr. Rotbart from Denver's?

A. I saw that report over the weekend, yes.

Q. And have you read both volumes of Michael Sherman's deposition?

A. Yes.

Q. Okay. Have you done any research in preparation for today's testimony?

A. No, sir.

Q. Okay. I'm going to start off and go through some exhibits, Doctor, what's been marked as Plaintiff's Exhibit 1. Would you identify that for the record?

A. Yes, that's an updated CV of myself.

Q. Okay. Are there any articles that you have

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authored thereon that would be potentially relevant to the subject matter of this case?

A. There are neonatology review articles that may be relevant for various textbooks. No specific scientific articles I think are immediately relevant.

Q. Okay. What, is it something you could go through right now and just put a check mark as to which articles those are, or would it take some time?

A. Not really. I think it would be, if you look at the chapters, there is general pediatric chapters, for example, a review of neonatology for Creasy and Resnik, which is an obstetrics book which is just a general neonatology text. I haven't looked at it recently. And it may indicate broad issues about neonatal care. I don't think they are specifically related to this case.

Q. Okay, fair enough.

And are there any articles that you have authored or co-authored that are not reflected on Exhibit 1?

A. No, sir.

Q. Showing you what's been marked as Plaintiff's Exhibit 2, would you identify that for me, please?

A. Yes, these are several pages of notes, as I was sent information. Save me going back to the original

chart each time, I make notes.

Q. Fair enough. May I have that, Doctor.

MR. BECKER: And, Kath, I'm just going to, so I don't walk off with these, make sure that you get the exhibits from me and include them with the deposition.

Q. Doctor, do you have the original notes with you?

A. I do.

Q. And I'd like to go through with you these notes to make sure I can interpret, understand your abbreviation and your comments.

And for the record, if you would, just kind of slowly read through each page for me, please.

A. I can do that.

It says, Shane Maslanka versus Metro. It's a male. Has asthma. That refers to the mother.

Should I editorialize?

Q. Yes, because sometimes I see some abbreviations. I want to make sure I know what they mean.

A. '04 Cleveland Clinic admission - failure to thrive, epilepsy, G tube feeding, cortical blindness.

MetroHealth admission from 8-1 to 10-15-01. Birth weight 1100 grams. White male. 32 weeks by

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dates, 27 weeks by exam. Apgars 7 and 8. Blow by oxygen at delivery. Mother A pos. Twenty-four year old mother, GBS negative, screens unknown at delivery.

That's what I thought at that time of my review.

Other screens negative; G7, P4, AB2. Treatment: Antibiotics prior to delivery.

That's, as I was reading, this is what I came up with, and some of those things, as you know, are probably not correct.

Spontaneous rupture of membranes. Rupture of membranes less than 12 hours. Vaginal vertex delivery.

Needed Surfactant times 3. Ventilated 18 days. Lasix, Aldactone, Dopamine. Nasal IMV times 4 days. Nasal cannula times 46 days. PVL grade III IVH. Hydrocephaly. PDA. Presumed sepsis. Hyperbilirubinemia. Birth weight 1100 grams, 58th percentile. Length, 36.5, 49 percentile. Head size, 26, 54th percentile. Discharge on caffeine thiazide.

Next page: Prenatal care began late, June 1. July: Enlarged kidney on antenatal ultrasound. Thirty-two weeks by dates, twenty-seven by exam. Date of birth 8-1-01, 6:31 a.m. Male infant, white.

Plus tobacco half a packet a day. SROM, which

is spontaneous rupture of membranes, eleven and a half hours prior to delivery.

UAC, UVC placed. Initial ventilator settings, a hundred percent oxygen, 20 over 4, 30 per minute, intubated for RDS. First ABG 741, 36, 260, 22.5, minus 1.6. Weaned well. Hematocrit 37 percent. Glucose is okay. CBC 4.4 times 10 to the third. Blood culture sent. Treatment: Amp/Gent. Received nasal, received, sorry, normal saline bolus to Dopamine for several days for pressa support.

8-2-01: IV Calcium gluconate for low ionized calcium.

8-3: Grade II IVH. Vent down to 35 percent. Slight metabolic acidosis-7.38 to 28/97/13.6/minus 7. Treatment sodium bicarbonate. Hypernatremia, about 154. Transfused to hematocrit of 39.6.

8-4: PDA treatment with Indomethacin deferred because of IVH. One gas with bicarb 15. Base excess minus 10.7. Treatment bicarb.

8-5: Still on Dopamine. Pink endotracheal tube secretions.

Next page. 8-6: Dopamine weaning. Needed another transfusion. Started trophic feeds. LFT's okay. Bilirubin 3.4. Fresh frozen plasma. Bloody endotracheal secretions. Abdominal ultrasound,

mild hydronephrosis. Nasal IMV from 8-8 to 8-12 to reintubated. About 8-8 Dopamine dc'd.

8-10 to 8-11: Aspirants an issue.

8-12: Needed re-intubation.

8-15: Now Grade III IVH.

8-19: PDA ligation.

8-23: Started on Dex. Weaned vent for three days, 8-23 to 8-26. Positive endotracheal aspirant to Tobramycin aerosols.

8-24: Extubated.

8-30: Nasal canula.

8-31: Reintubated for apnea, only about one day. Vanco/Gent started for a positive blood culture for coagulase negative staph. Appears to be collected on 9-2. CSF OK.

Note indicates bilateral cystic PVL on 9-5.

9-27: Nasal cannula O2.

10-8: Passed hearing screen. Some ROP.

10-15: Discharge.

Labs, next page. Labs - chemistry about 8-2 sodium increased at 154. Creatinine 0.5 to 0.7 range. BUN about 12. Bili to 4.5 on 8-9.

CBC's: 8-1, 8:00 a.m., white cell count 5.3 by 10 to the third. Corrected to 4.4. 31 percent of 4.4 equals 1.36 times 10 to the third. 28P, 3B, 58L, 11M,

NRBC's 20.

8-3: Midnight, white cell count 5.5, 6 times 10 to the third. Corrected to 3.7 - 37P, 54L, 7M. 37 percent of 3.7 equals 1.37 times 10 to the third.

8-4: 4.7 times 10 to the third. 22 granulocytes, 69 lymphocytes, that is a thousand granulocytes. Hematocrit 42. Platelets okay. 8-4 to 8-6 NRBC's relatively low. 16 on 8-4. Stayed low. For example, 8-6, 4.9 times 10 to the third - 15 granulocytes, 56 lymphocytes, 22 monocytes, total less than a thousand.

Transfused on 8-2. Reason, question mark. Hematocrit mid 30's.

By 8-8 - white cell count 8.7 by 10 to the Third. 28P, 5B, 38L, 27M.

Coags: PTT elevated 60 on 8-4, to 99 on 8-6. Normal is about 25 to 35. PT about 13 to 14. Normal is 10 to 13. Blood culture negative from 8-1.

Blood gasses basically okay. PO2 is a bit high. PCO2's in 30's and 40's. FI02 about 50 percent on 8-1. Down to 30 to 40 percent by 8-2.

8-2: Some metabolic acidosis. Bicarb down to 14. Base excess minus 11.

8-3: Bicarb in 14 to 20 range. Base excess minus 5 to minus 7. Baby A pos. Mother A pos also.

You're pretty good.

Imaging: RDS described as Grade I.

8-3: Grade I --

Q. Excuse me, Doctor. I want to interrupt on imaging. Are you simply restating the official interpretation or are you actually --

A. Yes, sir.

Q. You didn't read the ultrasounds yourself?

A. I didn't -- I don't think I've seen any images myself.

Q. Okay. Go ahead.

A. 8-3: Grade I to II IVH's. Possible increased white meta echogenicity.

8-6: Moderate right hydronephrosis, mild on left.

8-7: Head ultrasound - resolving Grade II, consistent with ventriculitis, mild increase in ventricular size.

8-15: Bilateral Grade III bleeds, mild ventriculitis, increased ventricular size. Subsequent chest x-rays show BPD with pulmonary edema.

8-31: Head ultrasound - biliteral cystic PVL, right greater than left. Slight increase in ventricular size.

9-18: PVL progressing. Increased ventricle

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size.

9-30: Ditto.

New page. Deposition - Gloria Maslanka, mother. Went to hospital evening of 7-31 with tightness of breath. Question asthma. ER to L&D to spontaneous rupture of membranes. Has seizures. G tube for feeds. Baclofen for increased muscle tone. Increased secretions.

Deposition - Dr. Razi, 5-31-06. Attending at deliver. Suggestion that info on younger gestation on antenatal ultrasound not appreciated. Decels prior to delivery. High risk perinatologist not involved initially on hospital admission of about 8:30 p.m. Not appreciated to be 26, 27 weeks.

6-28-01: Positive urine culture for GBS, but insignificant -- but no significant growth elsewhere.

Mid July, urine culture for GBS negative.

Infection risk not quite clear.

Claim seems to be that perinatology should have been involved earlier.

Deposition - Dr. Louis, L-O-U-I-S, resident in OB at time.

Admit note - Assumed about 36 weeks. No infection. GBS negative. SROM two hours earlier in evening before delivery. Says he would not have

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augmented labor if known to be 27 weeks so he could have given steroids. Would have given antibiotics if known to be 27 weeks. Had an amnio infusion about 2:00 a.m. Question mark for decels. Mother not considered infected. Probably did not get antibiotics.

Deposition - Dr. Sherman. Doubts infection. Seems to question whether SROM occurred. Will not come and comment, as will not commit as to how long pregnancy could have been prolonged.

Critical of neos: Charting. Delayed treatment of PDA to brain ischemia contributed to PVL. Says PVL first recognized at four weeks. Caused at first day or second of life. Says brain injury contributed to hyperoxia. Says hypernatremia contributed to IVH and caused by amnio infusion with saline. Believes hypotension and need for Dopamine secondary to PDA. Does not believe possible Grade II bleed -- I can't read the word -- justified withholding Indomethacin.

Presence -- does not believe --I'm sorry -- does not believe possible Grade II bleed presence justified withholding Indomethacin.

Critical of treating hypernatremia with sterile water feed drip. Says delay in PDA treatment caused bleed. Believes bleed extended to Grade III between

6th and 9th of August. Does not believe neutropenia reseptic. Critical of postnatal dex. Says with antenatal steroid IVH would not have occurred or been reduced. Says that if in utero early bleed -- says that if in utero/early bleed, would have seen signs of general bleeding.

Continued depo: Believes delayed echo and PDA closure contributed to morbidity. Believes infant would not have had IVH, et cetera, if PDA closed.

Critical of 8-3 head ultrasound that says Grade I to II bleed. Believes just Grade I. Is okay with diagnosis of Grade II on the 7th, Grade III on the 15th. Discusses cystic resolution of clots. Disputes neutropenia data.

Q. I don't see any notes off of the second volume of Sherman's depo.

A. I think when it said continued deposition, I think that probably was the second volume.

Q. Oh, okay.

A. I made the point, continued depo on the top of that last page, sir.

Q. Okay.

A. I think that was the continuation.

Q. So that was your summary of the second volume?

A. I think so.

Q. On the last page of your notes?
A. I think so.
Q. Showing you what's been marked as Plaintiff's Exhibit Number 3, would you identify that for the record?
A. These are some clinic follow-up notes.
Q. It's a letter to you from whom?
A. From Jim Malone.
Q. Okay. What's the date of the letter?
A. March 24th, '06.
Q. There is some handwriting in the upper right corner?
A. Yeah, it's my note. It says, 4/2 review of records and notes, two hours.
Q. Okay. Showing you what's been marked as Plaintiff's Exhibit 4, would you identify that for us, please?
A. I think that's an index, when the whole thing first arrived, with a gazillion.
Q. Records?
A. Records.
Q. And you've reviewed all those records that are delineated in that summary?
A. I have overviewed or reviewed all this stuff, yup.

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Q. Okay.
A. If it was sent to me. I mean, I didn't crosscheck.
Q. One more, Doctor.
Showing you what's been marked as Plaintiff's Exhibit 5, which I believe is your report in this case, would you identify it for me, please?
A. Yes, that is the report I wrote to Reminger as dated July the 17th, '06.
Q. And do you have a copy of that report at hand?
A. I do.
Q. Okay. I want to start off, Doctor --
MR. MALONE: He has written a second report.
A. There was a little extra page. You have that.
Q. I don't know that I have. Let me see it.
MR. MALONE: This was just a supplemental report that came in response to your submission of Dr. Carfi's.
A. Remember you asked earlier who is Dr. Carfi. That's when you were looking at that.
Q. Thank you for that.
Showing you what's been marked as Plaintiff's Exhibit 6, identify that for me, please.
A. Yeah, that's the supplementary sentence I sent

to Mr. Malone.

Q. Let me see that one more time.

I want to start off with some definitions, Doctor, and then we're going to talk about these medical concepts in more detail.

What does PDA mean?

A. Patent ductus arteriosus.

Q. And what does that mean?

A. It's a communication between the circulation going to the lungs and the circulation going to the systemic parts of the body. It's typically patent in a fetus and closes after birth.

Q. In a 27 weeker, when does it generally close?

A. Most patent ductuses close spontaneously and there are data on that. I think it varies a lot in time and how sick the baby is. I think there is a wide variability in when they close. I'm not sure I can give you a number.

Q. What does PVL mean?

A. PVL stands for periventricular leukomalacia.

Q. And what does that mean?

A. Well, to me that means white matter injury in the substance of the brain, most commonly in premature infants, but not necessarily.

Q. And do you restrict that concept to just simply

injury around the ventricles?
A. That's my understanding, yes.
Q. Okay. Newborn sepsis or neonatal sepsis, what does that mean?
A. Neonatal sepsis is infection which occurs in the neonatal period.
Q. And by the term sepsis?
A. Sepsis means infection.
Q. Neutropenia?
A. Neutropenia is a low total neutrophil granulocyte count in the blood.
Q. So when you use in your report the phrase or the word neutropenia, you're referring to the total neutrophil count?
A. Right. What we typically do is we take the mature granulocytes and what's called the bands, which is the immature neutrophils, and we add them together.
Q. Now, is that the same or different than absolute neutrophil count?
A. I think it's the same.
Q. Okay. What does neutrophilia mean?
A. Neutrophilia is an excess neutrophil count.
Q. IVH?
A. Intraventricular hemorrhage.
Q. GMH?

A. GMH is germinal matrix hemorrhage, I think. I don't use that term too much, but --

Q. Okay. Is there a difference between IVH and GMH?

A. Not really. IVH is graded. Germinal -- no, no, no, not seriously.

Q. NRBC's?

A. Red blood cells.

Q. ROP, retinopathy of prematurity, what does that mean?

A. That's the eye problem that can impair the vision of typically premature babies.

Q. What's the cause of ROP?

A. The cause of ROP is multifactorial, which means there is lots of things that can cause it. It's a problem of prematurity. Traditionally high arterial oxygen, just being one of the culprits, although other factors predispose babies to ROP. I don't think we quite know what really causes it.

Q. Do you have an opinion in this case what caused the child's ROP, in terms of likelihood?

A. I assume it's the prematurity. I see no other specific risk factor here that would incriminate.

Q. Specifically would it be the oxygen administration to the newborn?

A. Well, as I indicated, certainly a persistently high arterial oxygen tension in the blood has been implicated in predisposing to ROP.

In reviewing the blood gasses of this baby, while there were a few PO2's that were on the high side, this was not really different than what happens in most babies. So why some get it and why some don't, I don't think we quite know.

Q. Now, what number are you utilizing for a definition of neutropenia; 1,500?

A. Fifteen hundred or a thousand are the most commonly used definitions.

Q. Some authors utilize a thousand and your textbook uses 1,500, correct?

A. I don't remember, but it's between the two.

Q. You didn't write the chapter on neutropenia, a pediatric hematologist from St. Louis wrote the chapter?

A. Correct.

Q. Can we agree that neutrophil count can vary considerably during the neonatal period?

A. Yes.

Q. Do you have a copy of your most recent edition?

A. Yes. I think that, we just need that volume, I think.

Q. I was looking for this table in your textbook, and I thought I tabbed it, but apparently I didn't, where you have the normal ratios of, I think, bands to total, and what's, what's generally considered?

A. There are a bunch of tables in the appendix. Are you not finding it?

Q. No. I'll stumble upon it here in a minute. It's Table 44-11.

A. Do you have a page?

Q. I don't.

A. Table what?

Q Maybe I do. Maybe I do, Doctor, I'm sorry. Page 1310.

Tell me when you're there on Page 1310.

A. I'm there.

Q. Okay. I want to understand the Table 44-11 up here. This shows a, what a normal absolute neutrophil count should be?

A. Correct.

Q. Of 3,500 to 6,000, at the, within the first 12 hours of life. Did I read that correctly?

A. Yes.

Q. And what a normal absolute band count is?

A. Yes. I got it.

Q. And then the column to the far right is what

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the band to neutrophil ratio is, correct?
A. Yes.
Q. Now, if there were signs of infection, if a newborn had sepsis, would you expect the numbers to be higher or lower than what's reflected in this table?
A. The most common things we see when babies are septic is neutropenia rather than neutrophylia, and that's often associated with an elevated band count.
Q. Okay. And if the band count in this newborn was less than .14, that would be inconsistent with newborn sepsis?
A. That would be not supportive of newborn sepsis. On the other hand, I think when a baby is persistently so neutropenic as this child was, that would be quite a concern to me about sepsis.
Q. If a pediatric hematologist would opine that this child didn't have neutropenia, would you defer to a pediatric hematologist on that issue?
MS. REID: Objection.
A. I wouldn't for a couple reasons.
The first is that pediatric hematologists make pretty rare appearances in most NICU's, so I think they are rarely consulted and don't spend a lot of time there.
The other thing is that this graph and this

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table and a variety of graphs I'm sure we can find in the appendix, would indicate that having a persistent neutrophil count in the thousand to fifteen hundred range is way below normal on all normative graphs, so I would disagree with that statement, yes, sir.

Q. Would you agree, Doctor, that typically when a newborn has an infection and is septic and responds to antibiotics, a depressed white blood cell count will respond within a few days, assuming it's administered antibiotics?

A. I would anticipate an effective response, effective treatment of sepsis would result in improvement of the neutrophil count, yes.

Q. Within a few days?

A. I'm not sure I can give a specific time frame for it, but within days, yes.

Q. Would you agree, Doctor, that negative blood cultures, normal liver function and the absence of direct hyperbilirubinemia speak against newborn sepsis?

A. Let me handle those individually if I may.

Q. Sure.

A. The definitive diagnosis of sepsis is obviously made by a positive blood culture. Some would say two positive blood cultures. And that's not the case

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here, so we have a diagnosis of presumed sepsis, not proven sepsis by virtue of the negative culture.

Elevated liver function tests are quite uncommon in neonatal sepsis, unless you're dealing with a viral illness.

And the same applies for direct hyperbilirubinemia, direct hyperbilirubinemia unless you're dealing with a viral disease is a very uncommon manifestation of sepsis, so that's not something I would bring into play.

Q. Fair enough.

Would you agree, Doctor, that there are numerous conditions of prematurity that account and can account for low white blood cell count in the newborn unrelated to sepsis?

A. I'm not quite sure what they are, sir. I think neutropenia in a premie is really sepsis until proven otherwise.

A mother who is hypertensive is one reason we sometimes see neutropenic babies, but I don't believe this mom was hypertensive.

Other causes of neutropenia, while they may exist, I think they're pretty rare. There is not a striking list of causes of neutropenia.

Q. Fair enough.

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Would you agree that often with newborn sepsis one sees low Apgar scores?
A. If the baby is born septic, you may have a low Apgar score, yes.
Q. Would you expect to see a low Apgar score?
A. There is a lot that goes into the Apgar. I don't know whether I'd expect it or not, but it certainly is a clue that the baby might be septic.
Q. With newborn sepsis would you like to see, would you expect to see evidence of an asphyxiation by way of blood gasses?
A. Metabolic acidosis is the one blood gas feature I'd expect with neonatal sepsis.
Q. And with newborn sepsis would you expect to see evidence of thrombocytopenia?
A. You may.
Q. But more likely than not would you expect to see it?
A. I can't answer that. I suspect most septic babies are not thrombocytopenic, but I can't give you numbers on that.
Q. Did you say that most newborns that are born with, that are septic, do not have thrombocytopenia?
A. I suspect so, but I'm not sure I can prove that.

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Q. Would you agree that typically when there is newborn sepsis there is evidence at the time of birth of foul smelling amniotic fluid?

A. No. That's a very subjective sign. And I think it's a probably a feature of a profound chorioamnionitis, for which we don't have evidence here.

Q. And one of the features of profound chorio is funisitis, and we don't have that here, do we?

A. Correct.

Q. Just so I'm clear that, you're not opining in this case that this child had brain injury secondary to cytokines?

A. It's certainly a possibility.

Q. Well, I want to know in terms of probability if that's what your opinion is here.

A. I believe we, based on the evidence that I have, that we have a baby with white matter injury manifest in the first days of life. We have a baby who is, in my opinion, probably septic based on the persistent neutropenia, metabolic acidosis and hypertension, all of which are features of sepsis. So you have likelihood of sepsis. You have evidence of white matter injury. It is certainly possible that the white matter injury is caused by either infection

or the inflammatory response of an infection, yes.

Is that the likely reason of the white matter injury, I think that was your question. I think that would be speculation on my part. White matter injury can be caused by a variety of things. It's certainly --

Q. We talked about --

A. -- a likely association.

Q. Excuse me. We talked about causes of PVO at our last deposition, and just to make sure that we're on the same page, you can have the cytokine inflammatory response as one source and the other cause would be the typical hypoxic ischemic injury, correct?

A. Correct.

Q. And you can have a hypoxic ischemic injury from a hemodynamically significant PDA, correct?

A. You know, I know that Dr. Sherman in his deposition is taking that position. I tend to associate significant left to right shunting through a patent ductus arteriosus as a cause of pulmonary edema. I think it is, and there is the theoretical possibility of shunt, of blood being shunted away from the systemic circulation including the brain. I think that's a reasonable hypothetical argument.

Am I convinced that left to right shunt through a patent ductus in this or any other baby can lead to hypoxemic brain injury, I've not considered it very much. I think it's a possibility. I don't think it's likely.

Q. Well, is there literature that stands for the proposition that with a hemodynamically significant PDA you can get a ductal steal, it's called a ductal steal, which results in hypotension and that leads to both metabolic acidosis and ischemia, are you aware of any literature to that effect?

A. I'm certainly aware of the fact that shunting blood left to right away from the systemic circulation could lead to circulatory imbalance. I think the story is complicated. I mean, I think metabolic acidosis is not a characteristic presentation for a patent ductus arteriosus. In fact, these babies are much more likely to present with a respiratory acidosis because of their pulmonary edema than they are with their metabolic acidosis.

Hypotension can certainly aggravate shunting, but I have a problem, I guess as you can tell, with the thinking that left to right shunt through a patent ductus arteriosus will cause a metabolic acidosis and will cause hypoxemic brain injury.

Q. Or ischemic brain injury?
A. Right. I think that's an unusual line of thinking. It's theoretically conceivable, but I'm not sure it happens.
Q. And you're not familiar with any literature on that topic?
A. Well, in terms of PDA's causing brain injury, that would be very hard to come up with, you know. It's the smallest, sickest babies that have a PDA and it's the smallest, sickest babies that have brain injury and everything goes wrong in these kids. They're usually the ones that, for example, there is an association between PDA's and sepsis, so it becomes almost impossible to unravel these various risk factors.
Q. In the absence of interpartum antibiotics in a truly septic newborn, would you expect the blood cultures to be positive?
A. Certainly having positive blood cultures would be a definitive diagnosis of sepsis. And we don't have that here, hence the diagnosed of presumed sepsis. So many babies we see with sepsis don't have a positive blood culture and we presume they have sepsis. I don't know quite know how to answer your question. So would I expect it, not necessarily.

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Q. Well, how about more likely than not, if you're not giving antibiotics to the mom, that there is no intrapartum antibiotics, and if, hypothetically, the newborn is septic at birth, and you do cultures, would you expect, if there was true sepsis causing serious problems for a newborn in utero and there is no intrapartum antibiotics would you generally expect the blood culture to be positive?

A. Not necessarily. I think if the baby was, had an overwhelming bacteremia you would probably expect that, if the bacteremia were maybe more subtle, if the more long lasting, subtle infection, not necessarily.

Q. So can I infer from that answer that your opinion in this case that there was subtle bacteremia present before birth?

A. Not necessarily. I'm just trying to come up with an explanation for why the blood culture is so frequently negative. So let me reword what I said.

I mean, these babies, babies who are presumed to have sepsis, very frequently have a negative blood culture. And I don't think we quite know why. Whether it's because the volume of bacteria or the amount of bacteria in the blood is not as high, whether it's the way we sample the blood, I don't really know the answer to that. So I was really sort

of speculating at that point. I don't know why sepsis is so often associated with a negative blood culture in the baby.

Q. I want to stay with this a little bit more, Doctor. I want to make sure I understand your opinion.

A. Yes.

Q. Do you have an explanation -- strike that.

Would you expect if there was newborn sepsis and no intrapartum antibiotics given, and this was a significant newborn sepsis to cause brain injury to the child in utero, would you expect, more likely than not, to see a positive blood culture?

A. Not necessarily.

Q. I don't want to argue with you, but you answered not necessarily, which means a hundred percent, and I'm asking you for more likely than not.

MR. BECKER: Kath, could you read back my last question, please.

(Notary read back last question.)

A. And I'm telling you I don't know the answer to that.

Q. Okay. Now, do you consider the, jumping back to neutrophils for a second, do you consider the, which of the laboratory analysis do you consider more

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reliable as to whether a newborn is infected; absolute neutrophils, band to neutrophil ratio or I to T ratio, which of those three do you consider more reliable as a predictor of newborn sepsis?

A. I don't think it's possible to differentiate one as being more important than the other. I think what's more important is if you have an abnormal value that you repeat it and make sure that that abnormal value is not just a one, one shot deal, maybe a lab error or whatever, but persists.

And in this case we have one what I consider to be abnormal lab value which was just quite persistent. So I think the persistence of that phenomenon is more important to me than identifying one specific marker.

Q. So would you agree that there are many noninfectious processes that are associated with both neutropenia and neutrophilia? Did we cover that already?

A. We didn't talk about neutrophilia. Neutrophilia is very nonspecific sort of thing which I think can be a response to any sort of stress in a baby.

I think when you asked me earlier if there are many causes of neutropenia, I answered that I wasn't aware of a whole bunch of common causes of

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neutropenia.

Q. If your own textbook says that there are many noninfectious processes associated with neutropenia, would you disagree with it?

A. I probably should take a look at the sentence if you don't mind.

Q. Sure.

A. Make sure what context it's in.

Q. Page 796.

MS. REID: Does that say neutropenia and neutrophilia is the same sentence?

MR. BECKER: It does.

Q. It's the right-hand column, second or first full paragraph. I think it's the third sentence down?

A. I'm just going to look at the table to see what they're referring to.

So if I, if I may take this in the context I think it's stated, the first sentence says, neutropenia, especially in the first hours of life, and is associated with respiratory distress can be worrisome because of a strong association with GBS sepsis.

And it goes on to say, however, many noninfectious processes are associated with both neutropenia and neutrophilia. And they refer you to a

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Table 37-16. And it says clinical factors affecting neutrophil counts. And they've got 4 pluses next to maternal hypertension, which I think I answered earlier, that the first thing you might think of other than sepsis would be maternal hypertension as a cause of neutropenia, which is not apparent.

They've got a one plus associated with asphyxia, and I was not aware of asphyxia causing a decrease in neutrophils, but there is a one plus.

There is a three plus for periventricular hemorrhage, which could be consistent with this baby coming out with a periventricular hemorrhage. I was personally not aware of the fact that periventricular hemorrhage at birth causes neutropenia. And hemolytic disease we have no evidence for.

So I think that sentence probably refers more to the neutrophilia, which is the right side, I think, where you've got pluses everywhere. So I think it might be a little out of context. I don't think there is a lot of things that cause neutropenia, is my bottom line.

Q. Do you have any experience as to whether nucleated red blood cells cause a drop in red blood cell count, in RBC's?

A. I'm not specifically aware of that. I know we

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correct for the right for the nucleated red blood cells. But that's not your question, right?

Q. Right. You are not aware of that?

A. No.

Although I will say the nucleated red cell count in this baby of around 20 was not particularly elevated.

Q. Well, what's normal?

A. Normal is sort of anywhere from about zero to ten. Some of these babies have like several hundred if they're stressed, so at 20 is upper range normal maybe.

Q. On that same page, 37, Table 37-15 shows the range of normal absolute neutrophil count at birth. Do you see that, 500 to 6,000? It's in the bottom right-hand corner of Page 796.

A. Yes, I see that. I do see that, yeah.

Q. Do you agree with that, that that's a normal range; minimum and maximum?

A. This is what somebody is proposing. And they have minimum values of around a thousand at 60 hours with 500 at birth. That's very, very low.

I think this gentleman is proposing that and that's, he's entitled to propose that, but that's, I'm sure that's greater than two standard deviations below

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the mean.

Q. Doctor, it's in your textbook. Do you review the materials that ultimately are contained, I realize that you didn't write the chapter, but do you generally review each author's chapter before you include it in your final textbook?

A. Well, yeah, we do, but I think there's tables in the tables in the back where there are white cell counts. I think there maybe even an elaboration of this particular study, we can go to it if you like. So, so this is a small piece of a larger body of data which I think appear in the appendix of the book.

Q. Just for the record I want to make sure, what was your calculation for the absolute neutrophil count for, at 8:10 a.m., the blood drawn I think two hours after birth, what was your calculation?

And feel free to, at any time during this depo, feel free to look at your notes, the chart.

A. You know, there is a series, let me just in a general comment make this. There is a series of white cell counts between about a thousand and fifteen hundred and it's complicated by the fact that some have nucleated red cell corrections, some don't. Some have diffs, some don't.

If you want to go to the specific one, maybe I

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should go to the record. Is that okay?
I'm on 8-1 at 8:00 a.m, is that correct?
Q. Yeah, that's the first blood.
A. That's five. I'm not very good at math, but you've got 5.3 corrected to 4.4. 4.4, 28 polys and 3 bands, so you have to take 31 percent of 4.43. I can't do that in my head. I'm sorry.
Q. I came up with 1,373. Any reason to disagree?
A. Sounds reasonable.
Q. Sounds reasonable?
A. Yes.
Q. You feel that that's an abnormal for the --
A. We talked earlier --
Q. -- blood work done within the first six hours of life?
A. We talked earlier about the fact that a thousand or fifteen hundred is the lower range of normal that most people take. That's sort of in between.
Q. Now, is it your practice, Doctor, if you suspect a newborn sepsis, if you have a feeling that this child may be septic but you have a negative culture, is the next step that you, Dr. Martin, does is do a C reactive protein analysis?
A. We tend to, that's sort of become pretty

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fashionable. We tend to do that. Whether we were doing that in 2001, I don't remember.

Q. Okay. That helps the clinician?

A. It does.

Q. In their diagnosis, correct?

A. It does.

Q. And if you choose not to do a C reactive protein and you feel the child may well have newborn sepsis, do you engage in lumbar puncture, assuming the child is stable?

A. We routinely do a lumbar puncture if a baby has a positive blood culture. If a baby with respiratory disease does not have a positive blood culture, most of us would not do an LP. The practice varies a bit on that.

Q. But if you're not going to do an LP but your working with the presumption of sepsis, would you then institute high doses of antibiotic therapy?

A. Not necessarily, no.

Q. Now, I want to talk about steroids. Is a steroid -- is betamethasone a steroid?

A. Yes.

Q. And dexamethasone is a steroid, correct?

A. Yes.

Q. And I've heard the phrase corticosteroid, is

that the same thing?
A. There are mineral corticoids and glucocorticoids. We're talking here about glucocorticoids. So for practical purposes we're talking about the same thing.
Q. Okay. Doctor, you recognize that the advent of betamethasone for an intrapartum use is one of the most promising drugs that neonatology has encountered, correct?
A. I'd accept that.
Q. In fact, your textbook says, "The most promising specific prophylactic drugs are antenatal steroids." Do you agree with that? I'm not sure who wrote that chapter on the central nervous system, but let me tell you. Maybe it's you.
A. No, it's European authors, but I'm not going to argue with that. That sounds fine.
Q. Pierre Gressens?
A. Yeah.
Q. You agree with that?
A. Yes.
Q. It goes on to state that, "A systemic review published in 1999, showed a significant reduction in the risk of GMH-IVH diagnosed by ultrasonography." Any reason to disagree with that?

A. That statement is correct.
Q. The next sentence I find very interesting if you can find it, if you want to look at it, it's Page 931 in the right-hand column.
It also says that, "There was also a strong trend towards improved long term neurologic outcome." Do you agree with that?
A. I'm trying to think of the literature on antenatal steroids and long term outcome. And I'm not recollecting if, how strong those data are. When someone says a strong trend that usually means it looking encouraging but it's not quite there, right. And I have no reason to dispute that statement.
Q. Okay. Let's go on to the next sentence. It says, "It is not certain whether the effect," and that's the effect of the steroids, "is mainly due to increased lung maturation and therefore less severe RDS, stabilization of postnatal blood pressure, or perhaps a direct protective effect on the brain." Do you have any reason to disagree with that statement?
A. No.
Q. Would you agree that the effects of betamethasone administration on lung maturity are relatively rapid and most dramatic and effective if administered, if administration is provided to the mom

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approximately one to seven days before delivery?
A. That's correct.
Q. Would you agree that betamethasone decreases the likelihood of a hemodynamically significant PDA?
A. I don't think that's correct.
Q. Are you aware of any literature to that effect, that betamethasone has a positive influence on subsequent development of a hemodynamically significant PDA?
A. That literature is not at my fingertips. You might expect if a baby has less RDS, they would be less likely to have problems with the PDA. I imagine that's the association, rather than a direct effect.
VIDEOGRAPHER: Excuse me. I need to change the videotape.
* * *
Thereupon, a short recess was had.
Thereupon, the deposition continued pursuant to recess.
* * *
Q. Still going through your textbook, Doctor, it notes that, "Advances in neonatal intensive care and increase in the use of prenatal corticosteroids enhance fetal lung maturation and have resulted in lowering of the incidence of intercranial hemorrhage

to less than 20 percent of infants with low birth weight." Would you agree with that percentage?

A. Yes.

Q. Now, moving on to dexamethasone. Was this institution in 2001 utilizing dexamethasone to assist in extubation?

A. Yes. And in fact it's still used on occasions.

Q. Does this institution in low birth weight kids use indomethacin prophylactically?

A. Some of my colleagues may. Most of my colleagues do not.

Q. And that was the same in 2001?

A. Yes. This is one of the hardest things, indications for indomethacins is one of the hardest things we've had as a group to come to closure on, as is the case nationally.

Q. You recognize that there is some studies that show that indomethacin when used prophylactically significantly reduces the incidence of Grade III and Grade IV hemorrhage?

A. Correct.

Q. Now, I want to jump on to the topic of PDA.

Would you agree that when you see a 27 weeker that has a murmur, you want to be thinking about PDA?

A. Yes.

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Q. And when you think about PDA, you have to move quickly for confirmation via an echo, which is the gold standard?

A. Yes.

Q. That's the standard of care?

A. Yes.

Q. Do you read your own echoes or do you just rely on interpretations by cardiology?

A. I rely on the cardiologists.

Q. Would you agree that failure of the ductus to close is a common complication in infants with RDS and has been linked to the development of both BPD, bronchopulmonary dysplasia, as well as necrotizing enterocolitis?

A. That is correct.

Q. What are the clinical heralding signs or symptoms of a ductus?

A. In various combinations they would be a murmur, bounding pulses or a wide pulse pressure, and development of CO -- retention of CO2, in other words, development of a respiratory acidosis. Those are the main things that come to mind.

Q. And I have read that it's not uncommon, and particularly in 27 weekers who have hemodynamically significance PDA, to not be able to pick up a murmur,

is that true?

A. I don't know of any study that -- to answer that you'd have to know, you'd have to echo everybody, wouldn't you, those with a murmur and those without a murmur. And I don't know that that's been done.

I imagine most 27 weekers would have some patency of a ductus. But if it's not clinically significant, I'm not sure it matters.

Q. And the way you can tell whether it's clinically significant is what you just described to me about respiratory acidosis, murmur, and I forgot what the third was you just said to me?

A. Bounding pulses or wide pulse pressure.

Let me qualify what you said. I think that a murmur and bounding pulses are, certainly a murmur is a suggestion of a duct, not necessarily clinically significant. If the ventilator settings start going up and the CO2 starts going up, and the baby develops pulmonary edema, that's sort of more likely to be clinically significant.

Q. And pulmonary edema is apparent on chest x-rays?

A. Yes.

Q. And can one make a determination from an echo whether or not there is hemodynamically significant

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PDA?

A. Yes. Although I think that decision is a combination of echographic criteria and the clinical status of the infant.

Q. You would agree that a PDA should be suspected when there is a delay in clinical improvement from RDS and the infant has prolonged requirements for high ventilating setting?

A. Yes.

Q. Would you agree that indomethacin is effective in closing the ductus, particularly closer to the day one or day two of life in a 27 weeker?

A. Early use of indomethacin is thought to be more effective than delaying its administration.

Q. Now, I understand that there are some contraindications for indomethacin. And one of those which we're going to talk about today is active bleeding, correct?

A. Yes.

Q. And how do you as the clinician make a determination whether or not there is active bleeding?

A. The, obviously you look for clinical signs of bleeding.

Q. Just give me some examples of what those might be.

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A. Well, if the baby is oozing, for example, the baby is excessively bruised.
Q. Oozing from what area, any area?
A. Maybe puncture sites for example.
Q. Okay?
A. We would be concerned if the baby has a low platelet count.
Q. Okay.
A. Or as in the case of this child, we would be concerned if an ultrasound of the head has been done and shows a bleed.
Q. Okay. But just merely because there's a bleed doesn't mean it's active bleeding, does it?
A. Correct.
Q. So you as a clinician, if you're concerned whether or not you want to treat the PDA and you've got an ultrasound showing a bleed, you have to take other steps to satisfy yourself as to whether or not this is active bleeding, including coagulation studies, correct?
A. That's a consideration. I think obviously when you worry about bleeding, the place that you're most worried about bleeding is in the brain. And if you've got evidence of a bleed in the brain and the concern would be whether that's an actively progressing

phenomenon, but really the way to check that would be to do another ultrasound to see if the bleed is progressing.

Q. The next day?

A. The next day or soon thereafter, yes.

Q. Have you in your career ever had a concern, first of all, in a clinical setting where you feel the child needs indomethacin, you're not sure whether or not there is active bleeding, and then you order certain coagulation studies, have you done that in your career?

A. I'm sure I've done it. I'm not sure, if I haven't done it, the residents have done it. But I, I would not routinely order coagulation studies based on a Grade I to II intraventricular hemorrhage, let's say on day three.

Q. Okay. Have you or your residents on occasion given coagulation products or blood replacement therapy to help in the cessation of a bleeding so that indomethacin can be then administered?

A. No. I would imagine that if it's bleeding that actually required administration for example of FFP, I would be a little worried about giving such a baby indomethacin.

Q. Then you would consider ligation as an

alternative?
A. That would be an alternative.
Q. I think we talked about this earlier with PDA. Do you agree that PDA can cause diastolic hypotension?
A. Hypo?
Q. Hypo, H-Y-P-O.
A. Yes.
Q. And diastolic hypotension can lead to poor profusion?
A. Yes.
Q. And poor perfusion can lead to metabolic acidosis?
A. Yes.
Q. Can PDA cause hyperoxemia?
A. Not that's immediately apparent to me.
Q. And can PDA cause hypercarbia?
A. Yes.
Q. How does it cause it?
A. On the basis of pulmonary edema. That's easy. The flood, the lungs get, you're shunting left to right, you're getting excess blood going to the lungs and the gas exchange is impaired. In fact, a respiratory acidosis is a common manifestation of a clinically significant PDA, whereas I don't think you'll find metabolic acidosis in that context to be a

common phenomenon.
Q. We can agree, Doctor, that it could be potentially very dangerous to let a hemodynamically significant PDA go unaddressed, to a 27 weeker?
A. I'm only hesitating because of your use of the term very dangerous.
Q. It can be dangerous?
A. Yes.
Q. It can be harmful if it's --
A. Yes, yes.
Q. Would you agree that a diastolic blood pressure in the teens in a newborn 27 weeker would make one highly suspicious of PDA, particularly also in the presence of a murmur?
A. You see that's where I disagree with that statement. And I think I sort of maybe indicated earlier, I think in the context of this infant, with his neutropenia, I would associate a metabolic acidosis and hypotension more likely with sepsis than with a PDA.
Q. You can appreciate how someone might say that that could equally be attributable to PDA in day two of life rather than sepsis?
A. Yes, I can appreciate that. I think, yes, that's a possibility, yeah.

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Q. High oxygen concentrations administered during venting, can that cause a further dilation of the ductus?

A. Well, keep in mind we're talking about the arterial P02 that's a consequence of the high inspired oxygen as you know.

Q. Yes.

A. Hypoxemia is associated with opening up of the ductus, which is why the fetus in utero that has a low PO2 has a duct that's open. It follows that hyperoxemia would tend to close the duct, so I think I'm disagreeing with your statement.

Q. Okay. And you've not, and I forgot to ask you, you've not done any specific research, I know you've got a subspecialty interest within neonatology, you've done not a subspecialty research study in PDA's or in white blood cells of newborns, correct?

A. That is correct.

Q. In an unaddressed hemodynamically significant PDA increases the risk of an intracranial hemorrhage and worsening chronic lung disease, correct?

A. The later statement I agree with, that I think an unaddressed PDA is associated with a higher incidence of BPD. Whether it causes it or not is another story, but there is an association between

having a PDA and BPD. I think we've talked about that already.

Q. Okay.

A. Does a PDA cause an intracranial hemorrhage, I don't think so. If a PDA is shunting blood away from the brain, I don't see why it would cause a bleed, that doesn't, unless it's on the basis of --

Q. Ischemia?

A. Yeah, but that's not a bleed then. I think that would be more of an ischemic lesion in the brain, I think.

Q. Do you agree that PDA, a hemodynamically significant PDA if untreated is associated with worsening IVH in pulmonary hemorrhage?

MR. MALONE: I'm sorry, could I have the first part of that question read back? Did you say clinically unstable, was that?

NOTARY: Do you agree that PDA, a hemodynamically --

MR. BECKER: In a hemodynamically --

NOTARY: Significant --

MR. BECKER: PDA.

MR. MALONE: You got it?

A. I thought we've sort of already addressed that, sir. I mean, I think I'm having a problem associating

a PDA with intraventricular hemorrhage. And we've already discussed the pulmonary edema.

Q. Okay. You do feel that untreated PDA can lead to pulmonary hemorrhage?

A. Yes.

Q. And bloody secretions are classic findings associated with PDA?

A. Bloody secretions can be a manifestation of a PDA, yes.

Q. If you saw bloody secretions in a 27 weeker that would make you think about a PDA first and foremost, correct?

A. Yes.

Q. You still have problems with newborns with PDA every day? As you did, you told me last year, every day, there is not a day goes by that we don't have a PDA issue in our nursery?

A. That is correct.

Q. Next topic, PDL.

Do you, I think I covered this already, Doctor, do you agree that hypotension postbirth or in the neonatal period related to a ductus can cause PDL?

A. I wouldn't be as specific as that, so I'm not sure. I would disagree with the way you phrased it, sir. I mean, I think we've talked about the fact that

PDL can be, there are opposing camps, whether PDL is a sign of circulatory instability to a vulnerable area of the brain pre or postnatally, whether it's a manifestation of an inflammatory response pre or postnatally. So the way you worded it I didn't buy it, I guess.

Q. Would you agree that Grade III hemorrhages can cause periventricular leukomalacia?

A. I would disagree with that.

Q. Why?

A. There are people making research careers trying to figure out the relationship between IVH's and PVL. And it's very confusing.

My sense is that they're either separate, the current research would suggest that they're either phenomenon or that, if anything, the PVL causes the bleed. In other words, you are bleeding into an ischemic injured area of the brain rather than the other way around. I don't know that anybody believes that a bleed causes PVL, but it's a hot area of controversy.

Q. Okay. Would you agree that the majority of 27 weekers in 2001, assuming good obstetrical and neonatal care, were surviving without neurologic compromise?

A. Yeah, most of the data, quote, babies like for example less than 1,500 grams, less than a thousand grams, so if you narrow it down to a specific 27 week group, it's going to be hard to come up with specific data. I would say the statement is correct, but it's qualified by lack of data that specifically, okay.

Q. Back to betamethasone for a second. It, it also helps in reducing inflammation from significant chorioamnionitis, correct?

A. I was not aware of that.

Q. Are you aware of any literature that stands for the proposition that antenatal steroid treatment is associated with over a fifty percent reduction in the incidence of periventricular leukomalacia in preterm neonates?

A. I'm aware of the fact that large studies show a nonsignificant trend towards the reduction in PVL, but it's not impressive and it's not significant in almost all studies.

Q. Well, would you consider a fifty percent, over a fifty percent reduction in the incidence of PVL significant?

A. I think we're using the term significant in a different context, in that there are many papers, and I don't know which paper you're referring to, sir, but

there are many papers which have had difficulty showing an improvement in PVL with antenatal steroids. The best study of this I can cite you if you'd like.

Q. Go ahead.

A I think that the largest body of data, the first author is I think Stoll, S-T-O-L-L, Barbara Stoll, S-T-O-L-L.

Q. Okay.

A. And they found a, I believe it was a nonsignificant trend towards the reduction in PVL, which others have shown too.

Q. All right.

Are you familiar with The New England Journal of Medicine article that stands for the proposition that antenatal exposure to betamethasone is associated with a decreased risk of cystic periventricular leukomalacia among very premature infants?

A. Is that the, is that a French, French author that wrote that?

Q. Correct.

A. I haven't read it for a while. I'm aware of the paper. And that has not been substantiated by other larger series.

Q. Well, it's peer review by The New England Journal of Medicine.

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A. Yes.
Q. So --
A. I don't doubt their findings. I'm just saying that that's not necessarily held up.
Q. Okay. Let me just take a few minute break and then I'm going to go into your report, Doctor?
A. Okay.
VIDEOGRAPHER: We're off the record.
* * *
Thereupon, a short recess was had.
Thereupon, the deposition continued pursuant to recess.
* * *
VIDEOGRAPHER: We're on the record.
A. I think I was asked by you about a question about how 27 weekers do, whether they have or do not have morbidity. And I think I started by answering, you know, there is data for babies less than 1,500 grams, there is data for babies less than a thousand grams. And if you take 27 weekers, I'd have to sort dig in the literature to see how 27 weekers as a group do in terms of morbidity. And I don't have that information quite at my fingertips. I suspect about half of them do fine. I just don't exactly know what the numbers are. I didn't want to mislead you in my

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answer.
Q. Okay. This comment is in response to my inquiry about don't most with good obstetrical and neonatal care, don't most, that is 51 percent of the 27 weekers you see, survive without long term neurological outcome?
A. Right. And I, I would have to answer that I'm not exactly sure about putting a number to a 27 weeker under those conditions.
Q. How about 29 weekers, would you agree that most 29 weekers, assuming good obstetrical and neonatal care, survive without --
A. I'll go with that one and say that's okay.
Q. Okay. Thank you.
Okay. Do you have a copy of your report at hand, Doctor?
A. Yes.
Q. This July 17th, 2006 report marked as Plaintiff's Exhibit 5, is that the only report other, well, actually you have now done two reports, but is that the, up until July 17, is that the only report you wrote on this case?
A. Yes.
Q. And have you had an opportunity to review this report before today's deposition?

A. Yes.
Q. Do you want to make any changes, corrections, particularly as to the numbers on neutropenia, before we begin questioning?
A. No.
Q. Do not?
A. No.
Q. Have you checked, rechecked your math on that?
A. I did not go back to the precise math, because I think the numbers are very close.
Q. Okay.
A. If there's one thing that I would say differently today, I think I used the term mild RDS twice. Once, the first time in the context of the infant was intubated. A chest x-ray was consistent with mild RDS. I think I was referring to the x-ray report.

And then the baby received, had a pretty good course received several courses of Surfactin. And in my concluding statement I think I say he experienced mild RDS that progressed to BPD. Some might challenge that statement and say, well, if you need Surfactin by definition you've probably got moderate RDS.
Q. Fair enough.
A. It's a rather arbitrary adjective.

Q. In the second paragraph of the report you reference the spontaneous rupture of membranes occurring 12 hours prior to the vag delivery. What's the source of that information?

A. My review of the records.

Q. Can you be more specific for me?

A. Not at this time.

Q. At the end of that first paragraph you mention, quote, "It was not apparently recognized by the obstetrical team managing this patient this was a 27 week gestational infant, and neither antibiotics or antenatal steroids were administered." Do you see that?

A. Yes.

Q. Can I infer from there that you feel had it been recognized such drugs should have been administered to this baby?

A. No. I think they're just two related statements. And I don't think it's unqualified to indicate what the best prenatal management should be. Although we've already acknowledged that certainly one of the obstetricians indicates that if he or she had known they might have tried to give steroids, but I don't want to get into the prenatal management of this baby.

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Q. The first gasses on this baby, blood work, would that be inconsistent with sepsis or inconsistent with an asphyxiated baby, asphyxiated baby, the first gasses?

A. The first gas shows no signs of a metabolic acidosis, which might be apparent in sepsis. So I wouldn't say it's inconsistent, but it's not supportive of.

Q. This child was, you mentioned at the bottom of the first page, this child was reintubated August 12th. Can we agree that the reason that he was reintubated was his unaddressed PDA, the reason for the need for re-intubation was his unaddressed PDA?

A. That's a possibility. It's, he was also developing BPD. He was having some apnea. I can't specifically identify at this time exactly why he was reintubated except he was obviously needing respirant.

Q. Can we agree that this child's BPD was likely secondary to the long course of assisted ventilation and lung disease?

A. No. There is so many things that go into the development of BPD. There is immaturity, exposure to oxygen, exposure to a ventilator, potentially infection. PDA, I don't feel comfortable singling out, singling out one factor that caused this child to

develop BPD.
Q. Can we agree in retrospect, Doctor, that the lung disease that was prolonged was due to the apparent delay in treating the PDA?
A. No.
Q. I'm on Page 2 now.
A. Okay.
Q. In the first full paragraph you mention that the PDA was diagnosed on August 3rd. Do you see that?
A. Yes.
Q. And the diagnosis was made because an echo was performed, correct?
A. There was an echo performed, and I think the echo indicated that there was an intermittently open PDA. I think that's what the report said.
Q. Did it conclude that it was a large PDA?
A. I just remember them saying it was an intermittent PDA. We can check the report if you like.
Q. Go ahead.
A. "Intermittent PDA. No evidence of ductal dependent lesions. "There is a large PDA," it says that down further.

I don't know what they mean by large. Typically if a PDA is intermittent and is opening and

closing it's not large, so there is a bit of inconsistency there.

Q. Would you conclude from reading this report as a neonatologist that this was a hemodynamically significant PDA?

A. Not necessarily. I think what the consultant here is telling the neonatologist primarily is that this is an intermittent PDA. And I think as the neonatologist that's what they probably would have interpreted out of this. Which means that there is a PDA that's opening and closing, that's what I understand by intermittent to be.

Q. So if you were a treating neonatologist and you received this echo report, you would more likely than not conclude that this was not a hemodynamically significant PDA?

A. I think I indicated earlier that whether it's hemodynamically significant, or what's the term you used, clinically significant or hemodynamically significant?

Q. I think I --

A. It doesn't matter.

Q. I didn't mean to interchange it but --

A. But I mean a clinically significant PDA is one that is readily apparent on an echo and shows signs of

wide pulse pressures and some of the things we've talked about earlier, pulmonary edema, so I don't think you could just rely on the echo for that alone. And I think it depends on whether there was wide pulse pressures at the time, whether the hypertension was still a problem at the time. So I think all those factors would go into the judgment call, whether this was clinically significant.

Q. Can we agree that if this echo that was done on the 3rd was done on the 2nd, it likely would have reflected the same thing, more likely than not?

A. I have no idea. It could have closed and opened up, it could have been open all the time. It's pure speculation. I have no idea.

Q. Can we agree, Doctor, on the 2nd we have a murmur in a 27 weeker and we have some evidence of low diastolic hypotension and that should have caused the care givers on the 2nd to have ordered an echo to rule out a PDA?

A. I don't think it made any difference whether the echo was done on the 2nd or the 3rd.

Q. I'm not talking about causation. I'm talking about standard of care.

A. Typically if we have a murmur on a baby on day 2 we would order an echo.

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Q. Can we agree that it was a standard of care violation by Dr. Kumar and his residents in failing to order an echo on day 2 in this case?

A. Not really. I mean, I think most neonatologists would order an echo in such a child when they, when they notice a murmur.

Q. And to update you, the standard of care by way of Ohio Jury Instructions has changed a little bit in the last few years. It is now defined as what a diligent and skillful specialist of the same training would do under those circumstances.

Can we agree that a diligent and skillful neonatologist would likely, should order a echo on day 2?

MR. MALONE: Objection. Go ahead.

A. I would say, if at that time there was a murmur associated with, for example, the development of pulmonary edema, if the blood stained secretions were reported at that time, then the standard of care would have required you to order an echo.

If there were, if the only other feature was hypotension, and we have, we already talked about the fact that there are other reasons why this baby might have been hypotension, which was being treated, I'm not sure that there was any tremendous urgency to it.

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So I would, I don't remember whether the pulmonary edema or the secretions were bloody at that time or not, but I think certainly that would of course call for an echo at that time.

Q. If Dr. Martin is managing the patient, day two of life, 27 weeker, we have a murmur, we have evidence of metabolic acidosis, we have evidence of low diastolic hypotension, would Dr. Martin order an echo?

A. I would order an echo.

Q. And do you know Dr. Kumar?

A. I may have met him. It's not -- I don't recollect meeting him.

Q. Do you go over to Metro and consult occasionally?

A. No. That's a completely separate program. In fact, we're in competition because they've aligned their program with the Cleveland Clinic. I may go over there once a year to meet with their director and discuss giving a lecture or something, but there is no cross-coverage at any level, resident, fellow or attending.

Q. Is there any referral or consultation between the offices or between the hospitals?

A. No. Were the only echmo program in town, so they will send us babies for echmo. That's about all.

Q. When did they become aligned with the Cleveland Clinic to your knowledge?

A. I don't know. You probably read the Plain Dealer as much as I do. I know they have a combined fellowship program in neonatology with the Cleveland Clinic. I think that's been going for about two or three years.

Q. Under the umbrella of Case Western Reserve University is both UH and MetroHealth, you're both professors, people that are attendings at both institutions or professors at the same university?

A. Yes, it's a screwy system, right. They have academic appointments at Case.

Q. Okay. And at least from that relationship that UH and Metro from that perspective are sister hospitals?

A. No. They're competitors.

MR. MALONE: Cleveland Clinic shares a medical school with them, Case. It's one big happy family, Mike.

Q. Now, Doctor, and I failed to note the page on here, but you conclude that the August 3rd bleeds were at least a few days old. Somewhere in this report you mention that.

A. I'm basing that on the reports I have at hand,

that's correct.

Q. That's, your opinion is based on reports of someone else?

A. They're based on the x-ray report, the ultrasound report in the chart from the radiologist. And I think some subsequently confirmed by I think one of the defense experts, although I don't know whether I had that in my hand at the time or not.

Q. Well, prior to drafting this July report you received Mr. Malone's letter which has been marked as Plaintiff's Exhibit 3, where he is suggesting that there is expert opinion that this child had a sepsis in utero. It's on that second paragraph. Is that true?

MR. MALONE: That's my March 24th letter. Look at the date of the letter.

A. That's March the 24th. When was my report was dated?

Q. I have July.

A. Okay. So what's the question, sir? I'm sorry.

Q. I don't remember now.

I think I asked you, is it true that Mr. Malone on the second page is suggesting via other consultation with experts that there was an in utero bleed and an in utero infection?

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A. Yes, but I don't think I would have, I certainly would have considered the report of that individual in my opinions if I would have had it at hand. Based on what Mr. Malone said here, I doubt I would have paid much attention to that.

Q. I don't follow what you said. Tell me that again.

A. Okay. I don't think I would have taken much notice of that paragraph in his letter unless I had seen a report from such an individual, and I don't remember when I saw that.

Q. All right.

You mentioned that you in part are basing your opinion that this bleed was several days old based on Metro's radiographic interpretations as well, correct?

A. Yes, absolutely.

Q. Pull that out and tell me what it is about the early ultrasound report that suggests to you that this bleed is several days old?

MS. REID: Do you have at it hand, Dr. Martin?

THE WITNESS: I'm sure I do.

Q. You know, I think it's attached to Jim's letter to you. I believe the radiograph, if I'm, maybe I --

MR. MALONE: Look at the letter, see

what's attached to that.

Q. Maybe, no, maybe it's an exhibit, one of these exhibits. I saw that somewhere. Maybe not.

Sorry, Doctor. I don't want to mislead you there.

A. I know that the Metro report on the 3rd indicates concern about some white matter injury on day 3.

Q. Okay. I want to be very specific, because this is important, and if you can pull that.

MR. MALONE: You know Segal's report was in his hands before July, because that's actually dated March 24th as well.

MR. BECKER: I understand, Jim. I want to know, he mentioned --

THE WITNESS: I didn't remember that. I wasn't sure when I got that.

A. Here we go. I got it, got it, got it, got it.

MR. BECKER: Thank you, Chris.

Q. You understand the question?

A. I understand the question.

Okay. There is a statement on the 3rd of August by the radiologist at Metro which says, "areas of hypogenicity are demonstrated within the bleed suggesting that these may be of some duration, at

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least a few days old." I think that suggests, I read that as indicating that someone thinks the bleed is of several days duration.

Then there is separate --

Q. I apologize, Doctor, I misunderstood what you said. I thought you were implying in here that this bleed was antenatal in nature. In other words, the bleed occurred before the child was born. Maybe I read too much into your report.

A. My impression was that this bleed which was recognized on 8-3, at which time the baby was about two days old, was several days duration, of at least a few days old, indicated that this bleed either occurred prior to birth or around the time of birth.

I was also concerned about the fact that the echogenicity of the white matter is slightly increased which suggests to me some sort of a PVL kind of lesion, which we all know take days to weeks to develop. The fact that that was apparent on day 3 was strongly suggestive to me of an intrauterine lesion.

Q. Would you defer to a pediatric neuroradiologist on that issue?

MR. MALONE: What aspect of the issue?

MR. BECKER: Well, let me have his --

MR. MALONE: Because what he's just said

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is fundamental pathophysiology.

MR. BECKER: Let me have his answer back, Kath, please.

(Notary read back last answer.)

Q. Let me be specific, Doctor. On the opinion that echogenicity of the white matters takes days or weeks to develop, would you defer that issue to a pediatric neuroradiologist?

A. No. I would defer to them evaluation of the lesion. The concept that PVL takes, takes weeks to manifest on an imaging study, that's widely held, widely accepted.

Q. Are you talking about the end cystic formation takes weeks to develop?

A. That takes even longer, but just the, just the visualization of echodensity is thought to take days to weeks. Plus the cystic change when it occurs then follows upon that.

Q. And what's your authority that it takes days to weeks for echogenicity to appear, can you cite me anything?

A. Not off the top of my head.

Q. Now, how unusual is it for a 27 weeker to have an antepartum brain bleed?

MR. MALONE: You said brain bleed?

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MR. BECKER: Yes.

MR. MALONE: Excuse me.

A. I think about 20 percent or so of premies of this gestation will have intraventricular hemorrhage, some of them occur prior to birth, some occur at the time of birth, some just after birth. I don't think my report indicates that I know when this bleed occurred. I think it occurred a few days prior to its recognition, being recognized. I don't think I feel strongly that I know that this bleed occurred prior to birth.

Q. Okay. You mention in your report that ventriculitis is seen or at least reported. Can we agree that the ventriculitis is likely just a reaction to the blood being present in the IVH?

A. I think that's reasonable, yes.

Q. Can we agree, Doctor, that there are some times when it's the neonatologist's actions or inactions that can cause the development of ROP?

A. Everything is possible, but I think, you know, ROP has multifactorial etiology and it is unusual that you can implicate specific physician action or inaction in its course.

Q. Well, you want the oxygen concentration to be maintained in a 70 to 80 percent ballpark area?

A. Yes. So that's millimeters of Mercury, not percent.

Q. I'm sorry.

You certainly don't want it over a hundred percent or a hundred?

A. You try to keep it below a hundred, that's right.

Q. If it's over a hundred that creates the risk of ROP?

A. Traditional thinking is that hyperoxcemia may be a risk factor in ROP, and that's probably correct. I think in this particular case, I don't think the hyperoxemia was any different to what we would see in most of our babies as we try to adjust their ventilator settings.

Q. In the last paragraph on Page 2 you mention that, "Shane suffers from severe morbidity associated with preterm birth and its complications." I think it's the first sentence in the last paragraph. Do you see that?

MR. MALONE: Page 2?

MR. BECKER: Page 2.

A. I agree with that statement.

Q. And I just want to understand what you mean by that.

A. That the problems he has --
Q. Today?
A. Yes. -- are the recognized complications of a preterm birth, of being born prematurely.
Q. At 27 weeks without the benefit of betamethasone, correct?
A. I, I don't feel real comfortable, you can tell, the way you're expressing it. I think he was born prematurely and he has PVL, he has an IVH, he had bad lungs and so forth, and this is the price he's paying for this.

If he would have received antenatal steroid, he still may well have all the problems he has today. I mean, I think we know, for example, it would appear in all likelihood that he had PVL already going on at that time when he was born. That's a major risk factor for cerebral palsy. Would that have been impacted by being, by betamethasone, I would maintain probably not. So --
Q. Did his newborn course worsen his PVL, likely?
A. I think likely it was already established. I mean, I, that's speculation.
Q. I didn't say whether it was established or not.

It is speculation whether it was established already?

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A. No, it's speculation what impact postnatal -- your question I think was whether postnatal events from being born prematurely aggravated the PVL.
Q. Made it worse.
A. And I say that's speculation.
Q. Okay. So --
A. I really have no idea.
Q. So what is your opinion as to was caused, do you have an opinion in terms of probability as to what caused this child's brain injury? Is it more likely the IVH or more likely the PVL or a combination which you can't separate out?
A. I would say it's more likely the PVL.
Q. Okay. And what caused, what caused the PVL in this case, if you have an opinion in terms of probability?
A. I don't know what caused the PVL. Infection may have contributed to it, but that's, I don't know that.
Q. You don't know that in terms of probability, based on what you've just said?
A. You know, I think we've talked about the fact that there is several things that can cause PVL in utero; circulatory imbalances, infection, things that we may not yet understand.

I believe this baby as I've indicated had an infection acquired in utero. Can I go the next step and say, therefore, the PVL was caused by that presumed infection. I think that gets a little speculative.

Q. And the sole basis for your conclusion that that baby was, was -- do you believe this baby had bacteremia or was actually septic before birth?

A. If I say he's septic, the assumption is that at some point there was a bacteremia, which we haven't recognized. We know that he was neutropenic from almost the beginning. I think the assumption that I'm making is that he acquired that infection from his mom prior to birth at the time of delivery, but, again, I don't know exactly. I can't tell.

Q. Okay. Is there a, is there a difference medically between the term bacteremic and septic or sepsis?

A. I think sepsis implies that you're bacteremic.

Q. Well --

A. In the case of a bacterial infection anyway.

Q. Right. I've learned that, and I certainly can be wrong, that sepsis or septic implies more of a systemic response, multiorgan response to an infection versus someone can be bacteremic, I can be bacteremic

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today.
A. And be well.
Q. And be well.
A. Okay.
Q. So I'm trying to understand your opinions as to whether or not this child in utero was merely bacteremic or actually septic, if you have an opinion?
A. I don't have an opinion. I do not have an opinion.

VIDEOGRAPHER: Excuse me. I need to change the videotape. We're off the record.

* * *

Thereupon, a short recess was had.

Thereupon, the deposition continued pursuant to recess.

* * *

VIDEOGRAPHER: We're on the record.
Q. Going to your final conclusions in your report, Doctor, where you say that, "The complications of Shane's preterm birth probably could not have been avoided by an altered course of neonatal or perinatal management," is it your opinion that his neonatal course would have been the same had tokolytics and betamethasone been applied antepartum and this pregnancy extended 10 to 20 days?

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A. No, I don't think it would have been the same. I think if he would have received betamethasone, I think the lung disease might have been diminished. Probably would have.

Whether it would have affected the intraventricular hemorrhage really depends a little bit on when that intraventricular hemorrhage occurred, so I'm not sure if that was already there or if that occurred around the time of the birth.

I'm concerned as you know that the baby was infected, and what prolonging the pregnancy would have done to that is hard to say. It could have had an adverse effect. I don't know.

I'm concerned that this baby had an ischemic brain injury or PVL, ischemic/inflammatory brain injury of PVL prior to birth.

Q. That's based on your interpretation of how long it takes for echogenicity of the periventricular region to appear, correct?

A. Yes.

Q. Okay.

A. And I don't think that prolonging the pregnancy as you describe would have improved that problem.

Q. Do you think the PDA played any part in this child's ultimate development of BPD?

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A. I don't think the PDA was a big part of this story. It obviously contributed to the pulmonary edema this baby had, and as such it may have prolonged the ventilator course. May have aggravated the BPD, it's possible. I don't think it was the major factor, a major factor.

Q. So are you saying that even had betamethasone been administered and tokolytics and this pregnancy extended to 29 weeks, you don't feel that it would have altered the outcome in this case?

A. I think it might have been improved the respiratory outcome. I don't think it would have improved the neurologic outcome.

Q. And the basis for your opinion that it would not have improved the neurologic outcome is, goes back to your interpretation that there was evidence of PVL early on and that likely was prior to birth?

A. Correct.

Q. Okay. And as to whether or not the newborn course made the PVL much worse, much more severe, would you defer to a neuroradiologist on that issue?

A. No. I think I feel comfortable, having as much expertise on that as anybody, and I think, I think what I answered was I think it's speculation as to whether postnatal events, which were difficult at

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times, might have aggravated that or not.

I think what I'm saying is that, this baby came out with PVL acquired in utero. And it is speculation as to whether some of the postnatal problems that they ran into might have aggravated that or not.

Q. Well, whether you call it speculation or not, you would, you would find, you would not have, or would you find, strongly disagree if someone opined that this child's neurologic condition is in large part from his complications at Metro in the first ten days of life?

A. Oh, I would pretty strongly disagree with that.

Q. And that goes back to your opinion because you feel an ultrasound shows a echogenicity, which is brightness, in one part around the ventricle, you feel that that means this kid was doomed to have a permanent neurological condition, correct?

A. That was a long question. Let me think what I -- when I reviewed the postnatal course of this child at Metro, I honestly could not fault the postnatal management of this baby. I might have done things a bit differently, but I saw nothing that didn't meet, nothing that deviated from the standard of care on this infant.

We know that the baby's prognosis is bad. The

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baby is doing well, doing badly. And we have evidence of an intrauterine insult to the brain. That's how I draw my conclusion.

Or was that not the question?

Q. I think, I think maybe you lost my question or I lost your answer.

MR. BECKER: Could you go back and read my question again, Kath.

NOTARY: Question: Then that goes back to your opinion because you feel an ultrasound showing an echogenicity, which is brightness in one part around the ventricle, you feel that that means this kid was doomed to have a permanent neurological condition, correct?

MR. MALONE: I think he answered that question. For the record, Kathy, I said I think he did answer that question, because it is not a simple question, it's a complex question and he gave a bit of a longer answer, but that is the answer.

Q. The answer is yes, Doctor, just so we're clear?

A. The answer, the primary -- I'm sorry, do you want to read it again? I'm sorry, I don't know what I'm saying yes to anymore.

Q. Okay, I understand?

A. I apologize.

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Q. That's all right.

MR. BECKER: Would you read it again?

(Notary read back question.)

A. No, the answer to that is no. I mean, I think the, I'm sure we see bad babies. I'm sure we see babies with lesions such as this who we worry about because they're at risk for PVL and probably do okay. So if you're suggesting that I would look at a picture like this at day 3 and say this baby is doomed to a disastrous outcome like this poor child has, the answer is no.

I'm putting everything together. I mean I'm putting together premature infant, who had this lesion, has a, I think, an appropriately managed postnatal course, has now a very bad outcome, and I'm attributing, a major contributor to that bad outcome is what we're seeing on the ultrasound, on that first ultrasound.

Q. But what you see, hypothetically, if that, what you see in the first ultrasound occurred within, on day two of life, then this, all this damage could be, have occurred in the newborn period, correct?

MR. MALONE: Well, I'm going to object. Now you're getting argumentative, Mike.

He said that some of the things in the

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ultrasound can not have occurred on day 2 of life, pathophysiologically impossible.

So go ahead and answer the question if you can, but please don't get more argumentative.

A. I believe that the imaging finding of the echodensity on day 3 points in all likelihood to an antenatal etiology, not one that occurred the previous day.

Q. And on that issue you would not defer to a nationally recognized pediatric neuroradiologist as to what that image says by way of timing, true?

A. No, that's not true. I mean, if imaging people tell me this is an artifact, I would have to trust them. I have the expert, I have some expert testimony, I think in front of me, which suggests that this is already showing some cystic change. I don't read the ultrasounds, so I, I would have some faith in what the experts say, yes.

Q. Okay. And if there was an expert that said that there is not an evidence of a cyst on the first ultrasound, assuming that expert to be true, then your opinion as to an antenatal injury would be withdrawn, correct?

MS. REID: Objection.

MR. MALONE: Yes.

A. Well, keep in mind, if I am told, if I am convinced by experts that this white matter injury doesn't exist, then obviously I would have confidence in the fact that it doesn't exist. But keep in mind that I'm still quite concerned that this is an infected baby, and I worry about an infected baby being in rather than out of the uterus for any length of time. So I don't think that should be ignored.

Q. Right. Fair enough.

But I think we previously agreed that if you have a baby where no antibiotics are given and there is a negative culture, it's likely if there was an infection it was a subtle infection versus a flurid infection?

A. I think I made a comment along those lines, but I sort of withdrew it. And I think that there are many reasons that we don't understand why we make the diagnosis of presumed sepsis and we can never get a positive blood culture. And whether that relates to, I think probably the wrong term microbiologically, but whether it determines, whether it's how many bugs you have in your blood, whether it has to do with the sampling or some other factors, I'm not in a position to know.

Q. But I'm glad you mentioned that, Doctor,

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because this concept of presumed sepsis, any time, is it true that when you have a 27 weeker born that you will most always make a presumption of sepsis and treat that patient, particularly when the GBS is reported as unknown, GBS status is reported as unknown?

A. Well, here we get into semantics. I mean, I think that there, all these babies are treated with antibiotics when they come out because we have to make sure the baby doesn't have an infection.

Q. So that's called presumed sepsis.

MR. MALONE: Please let him answer, Michael.

A. And those antibiotics are typically stopped at 48 hours and we wait for the next problem to happen. I personally wouldn't call that presumed sepsis, no, that's a baby that needs to have sepsis ruled out.

Now, unfortunately what we get into now is that we will not get reimbursed if we call something rule out sepsis, there is no such diagnosis. Now, you get into the whole business of, I think people use the term presumed sepsis to give it a diagnosis for which there might be some sort of reimbursement.

To me presumed sepsis means I assume the baby is infected, not I'm just giving 48 hours of

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antibiotics because that's what I do.

Q. Doctor, even at this institution you've had unfortunate circumstances when there's been an iatrogenic premature birth, correct? We talked about that a year ago.

A. I'm sure it happens.

Q. And you would expect, Doctor, if there was iatrogenic premature birth that there would be some kind of investigation by the medical staff, either via an incident report or quality assurance to determine what happened?

MR. MALONE: I'm going to show an objection. Whether they investigate here or don't, whether we investigate or don't is absolutely out of bounds for this lawsuit.

You don't need to answer that question.

What do you mean by iatrogenic? Doesn't that mean man made, man caused?

MR. BECKER: Yeah, that means bringing about a preterm birth. Isn't that what --

MR. MALONE: Well, what does it mean? Just tell us what you mean by the word iatrogenic, so that I'm not misguided. I think it means caused by man.

MR. BECKER: Brought about by, in this

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case it would be medical personnel.

MR. MALONE: Well, the preterm labor was not caused by medical personnel in this case. There can be no claim that it was.

Now, if you want to engage in colloquy with this witness, we've been here now two and half hours. I mean, ask more questions, but we're not going to ask questions about who investigates what or whether this is iatrogenic. I mean, we're getting too far afield now. You've covered every issue you could possibly have inquiry about in this case.

You may go a little bit further and then we're going to stop. Thank you.

Q. Do you know a pediatric hematologist by the name of Cairo, he's head of ped hematology at Columbia Presbyterian?

A. I don't know him personally, but he has a good reputation.

Q. If Dr. Cairo would say that this is not newborn neutropenia, would you defer to him?

MS. REID: Objection.

A. I think I mentioned already that I don't know that the, I believe that, pediatric or even neonatal hematologists get very, only a very, very small microcosm of what goes on in the NICU.

Q. But it was pediatric hematologist that wrote the chapter in your newborn, in your book on neonatology, correct?
A. Correct.
Q. Do you know Michael Sherman?
A. No.
Q. Going back to Dr. Kumar and the relationship or absence of relationship with Metro, you have no problems, you don't perceive any problems with you testifying on behalf of Metro, correct?
A. No, none at all.
Q. Do you consult with pediatric infectious disease doctors?
A. Yes.
Q. Who do you, is there one or just a group of pediatric ID doctors here that you consult with?
A. There is a group, and whoever is on service comes to the NICU.
Q. When you're not certain whether or not there is newborn sepsis, do you ever consult with a ped ID doctor?
A. No, not usually. I think the commonest practice would be, the baby has an infection, what antibiotics should we use, how long should we treat, does this baby or does this baby not have meningitis,

those are the usual discussions we have.

Q. Do they actually physically come to the NICU?

A. Yeah, they come about once a day unfortunately.

MR. BECKER: Okay, Doctor, I think we're done. I thank you for your time, sir. Good to see you again.

THE WITNESS: I thank you.

VIDEOGRAPHER: Doctor, you have a right to review the videotape to approve its accuracy or you may waive that right.

MR. MALONE: You can waive the viewing if you want. I want you to read the transcript though.

THE WITNESS: Okay. The viewing of this, I don't want to see myself.

C E R T I F I C A T E

The State of Ohio,)
) SS:
County of Lorain.)

I, Kathleen A. Hopkins, a Notary Public within and for the State of Ohio, duly commissioned and qualified, do hereby certify that the within-named witness, RICHARD MARTIN, M.D. Was by me first duly sworn to testify the truth, the whole truth and nothing but the truth in the cause aforesaid; that the testimony then given by him was reduced by me to stenotype in the presence of said witness, subsequently transcribed into typewriting under my direction, and that the foregoing is a true and correct transcript of the testimony so given by him as aforesaid.

I do further certify that this deposition was taken at the time and place as specified in the foregoing caption, and was completed without adjournment.

I do further certify that I am not a relative, employee of or attorney for any of the parties, or otherwise interested in the outcome of this action.

I am not, nor is the court reporting firm with which I am affiliated, under a contract as defined in Civil Rule 28 (D).

IN WITNESS WHEREOF, I have hereunto set my hand and affixed my seal of office at Elyria, Ohio, this _____ day of _____, 2006.

_____
Kathleen A. Hopkins, Notary Public
My Commission expires 1-10-10
Recorded in Lorain County, Ohio

Kathleen A. Hopkins & Associates
Court Reporters
401 Broad Street Suite 300
Elyria, Ohio 44035
440-323-5620
September 29, 2006

In re: Gloria Maslanka vs. MetroHealth Medical Center
In the Cuyahoga County Common Pleas Court
Case No. CV-05-552424

Richard Martin, M.D.
Rainbow Babies & Children's Hospital
11100 Euclid Avenue
Suite 3100
Cleveland, Ohio 44106

Dear Dr. Martin,

The transcript of your deposition taken on September 25, 2006 has been prepared. You have the right to read the transcript and correct any errors you may find in transcribing your testimony.

Enclosed is a copy of the transcript of your deposition. You may note any corrections on the yellow certificate page enclosed with the transcript. Please return the signed certificate page to our office in the enclosed self-addressed stamped envelope within 30 days.

If our office does not receive the certificate within 30 days, we will assume that you have waived your right, and the unsigned transcript may be filed with appropriate Court. You may keep the transcript of your depostion.

If you have any questions, do not hesitate to contact us at the number listed above.

Very truly yours,

Susan Muckley
Office Manager

cc: Michael Becker
James Malone