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IN THE COURT OF COMMON PLEAS
CUYAHOGA COUNTY, OHIO

No. CV-05-552424

GLORIA MASLANKA, Individually and as Parent and Natural Guardian of SHANE MASLANKA,

Plaintiff,

vs.

METROHEALTH MEDICAL CENTER,

Defendant.

DEPOSITION OF HARLEY A. ROTBART, M.D.

March 9, 2007

Pursuant to Notice taken on behalf of the Defendant at Courtyard Marriott, 6901 Tower Road, Suite 830, Denver, Colorado 80203, at 9:11 a.m., before Kelli J. Wessels, Registered Professional Reporter and Notary Public within Colorado.

APPEARANCES:

DAVID A. KULWICKI, Attorney at Law, from the Law Firm of Becker & Mishkind, 1660 W. 2nd Street, Suite 660, Cleveland, Ohio 44113, appearing on behalf of the Plaintiff.

CHRISTINE S. REID, Attorney at Law, from the Law Firm of Reminger & Reminger, 1400 Midland Building, 101 Prospect Avenue, West, Cleveland, Ohio 44115, appearing on behalf of the Defendant.

I N D E X

(Original exhibits attached; copies to counsel.)

WHEREUPON, the following proceedings were held pursuant to the Ohio Rules of Civil Procedure:

HARLEY A. ROTBART, M.D.

having been duly sworn to state the whole truth, testified as follows:

EXAMINATION

BY MS. REID:

Q. Good morning, Dr. Rotbart. Is that how you say it?

A. We say it Rotbart.

Q. Well, I want to say it the right way. Rotbart. My name is Christine Reid and I represent MetroHealth Medical Center in this case and we're here for your deposition this morning.

As a matter of housekeeping, before we started you were kind enough to show me a copy of your file materials. Is everything you've provided to me this morning, does that encompass the entirety of your file on this matter?

A. Yes, it does.

Q. So there is no other materials that you have at your home or your office that you have reviewed?

A. No, nothing else that I reviewed. I did provide Mr. Kulwicki with a handwritten chart and article this morning for his information regarding neutropenia and

25 normal levels of the neutrophil counts in premature babies

1 but that was -- I just prepared that last night to be able
2 to show to him.
3 MS. REID: Are you going to provide that to me?
4 MR. KULWICKI: You know, I'll give you the
5 article. The handwritten note was for me so that's work
6 product, but I'll give you the article.
7 MS. REID: Well, I'm going to disagree on the work
8 product. If they are notes he made in expert review of
9 this matter, I think I am entitled to them. I don't think
10 I've never not had somebody hand it over to me.
11 MR. KULWICKI: Well, it was a note to me. Let me
12 look at this article.
13 MS. REID: But you are taking the position today I
14 can't see his notes?
15 MR. KULWICKI: It is not his notes. It is a note
16 for me. It is my notes.
17 Q. (BY MS. REID) Doctor, let me ask you, tell me
18 what are in the notes? Are they analysis of whether or not
19 there is evidence of neutropenia in this case?
20 A. No. After reading the discussions in the reports
21 I wanted Mr. Kulwicki to see how absolute neutrophil counts
22 are calculated based upon this baby's laboratory results.
23 So I simply went through the eight or ten CBCs that the

child had and calculated the absolute neutrophil counts for him so he could see the method of calculations based on the

neutrophil band counts and myelocytes.

Q. Did you ultimately conclude based upon those calculations that you performed that there was evidence of neutropenia or there was not evidence of neutropenia?

A. Well, I concluded not based upon those calculations. Those were to show Mr. Kulwicki how the calculations were made and why I concluded earlier there was no evidence of neutropenia. But my opinions regarding neutropenia were not based on those notes. They were based on review of records and I had expressed those to Mr. Becker and Mr. Kulwicki in numerous previous phone conversations.

This was just a method -- since everyone has different ways of calculating the absolute neutrophil count, this is just the way my understanding is that they are calculated.

MS. REID: We can do this the easy way or the hard way. I guess I can sit here today and ask you to calculate those for me so I know how you are making the calculation or you can let me see the notes and I can see what the calculation is. It seems to me this is a silly position you are taking.

MR. KULWICKI: I'm not giving you the notes.
MS. REID: So instead I'm going to go through with Dr. Rotbart and have him make these calculations. We've

got a flight to catch --
MR. KULWICKI: It's not important.
MS. REID: Well, it is important because I need to know. Neutropenia is one of the issues in this case.
MR. KULWICKI: I'm not giving you the note. I'm not giving it to you.
MS. REID: All right. We'll do it the hard way then.
Q. (BY MS. REID) Other than this article and the notes, are there any other materials that you have related to your review of Shane Maslanka that are not in front of me today?
A. No, ma'am.
Q. What I'm just going to do and you can watch me as I do it, I'm just going to go for the record and identify what it is in your manila folder here so I'm clear exactly what you reviewed in this case. Some of them I may have attached as exhibits at the end of the deposition.
First is a February 12, 2007, letter from Mr. Kulwicki and that enclosed the expert reports of Dr. Cairo, Dr. Leonard, Dr. Martin, Dr. Barnes,

Dr. Sherman, Dr. Siegel and the deposition transcript of Dr. Richard Martin and Dr. Michael Sherman.

Next -- then we have the expert report of Dr. Cairo, expert report of Dr. Leonard, expert report of

Dr. Martin, expert report of Dr. Barnes, expert report of Dr. Sherman, expert report of Dr. Siegel, deposition of Dr. Martin, both parts of the deposition of Dr. Sherman, your expert report of February 9, 2007, correspondence to you regarding your deposition from Mr. Kulwicki.

A December 28th, 2006, letter to you from Mr. Kulwicki enclosing Dr. Leonard's report. You've got that twice. Then an August 28th, 2006, letter from Mr. Kulwicki enclosing the medical records of Shane Maslanka. And there is two volumes, but they are both related to the newborn records, correct?

A. Yes, ma'am.

Q. Was August 28th, 2006, the first letter you got regarding this case?

A. Everything is in my file so I believe that's right.

Q. Prior to that time, did you have some kind of telephone conference with Mr. Kulwicki or Mr. Becker regarding your interest in this case?

A. I believe that is true. I don't have distinct

memory. I don't believe I would have received the records unless there would have been a prior phone call asking that I review them. Thank you.

Q. Do you have any sense about how -- when the initial phone call would have occurred in reference to the

August 28th, 2006, letter that you received?

A. I don't unless the letter says, thanks for our conversation last week, I wouldn't have any memory of when the phone call had occurred. And it doesn't say that.

Q. Did you happen to bring a copy of your CV with you today?

MR. KULWICKI: I've got one. Do you want one?

MS. REID: If you have one, yeah.

MR. KULWICKI: Yeah.

(Deposition Exhibits 1 and 2 marked for identification.)

Q. (BY MS. REID) Doctor, I'm going to hand you what has been marked as Exhibit 1. It is a copy of your CV. Is that a current copy of your curriculum vitae?

A. It's pretty close. I think I added a couple of talks or papers or both sometime later in 2006 but this is essentially current, yes.

Q. All right. Are any of those additional papers or talks relevant to the issues in this case that you can

recall?

A. Well, most of my career has actually been dealing with central nervous system infections and neonatal infections so it is likely they were duplicative of other things that are in the CV, but I don't think there is anything ground breaking or new that would have been in

those.

Q. All right. I've marked the article that you have provided to Mr. Kulwicki as Exhibit No. 2. When did you pull that article?

A. Um, March 8th. That would have been yesterday.

Q. Okay. That was -- is the date on that somewhere?

A. No, it is on the bottom.

Q. Okay. Of when you downloaded it?

A. Yeah.

Q. What was the reason for you to download that article yesterday?

A. The same as the handwritten notes. That is just to explain to Mr. Kulwicki -- it just seems as if there is an enormous amount of focus in an area that there should be essentially no focus. That is this baby's white blood cell count and neutrophil count. And I have been telling Mr. Becker and Mr. Kulwicki that I consider this baby's counts to be normal and that not to be a factor in this

baby's diagnosis, but I wanted him -- again, after reading all the reports in preparation for the deposition, I wanted him to see an example of how other people have described neutropenia in newborns and how this baby's counts stack up.

Q. Okay. So this article that you provided then supports your position regarding the normalcy of white

blood cell counts which we'll discuss in a minute?

A. Well, yeah, I don't know if every line and every word is supportive or not. But it shows how people think about neonatal neutropenia and why we think about it differently for premature babies and how we think about it for newborns and why we are not surprised that with the vast majority of babies who have neutropenia never have an infection. It's just sort of an established way of thinking that I thought was summarized in there in a way that would be useful for Mr. Kulwicki to see.

Q. Okay. Can you tell me a little bit about your practice as a pediatric infectious disease doctor?

A. Following my fellowship as an infectious disease doctor that was completed in 1985 I joined the faculty and for the next 21 years I was an attending physician doing clinical consultations. I had a research lab that did basic science virology research, and I did clinical studies

and clinical trials as well. I taught and I had a growing administrative role.

And that continued with some ebbs and flows and various components of that until March of 2005 when for the first time my administrative responsibilities outgrew my others and I became more white collar than I had been in the past.

Then in January of 2006 I no longer did clinical

work because the chairman of pediatrics stepped down and in the transition I was asked to take over a number of his responsibilities and gave up the clinical time that I had been doing at that point.

We now have a new chairman and no good deed goes unpunished. I haven't been able to give back any of those responsibilities. So I continue to be mostly a bureaucrat for the past years.

Q. Do you have an administrative title at the university?

A. Yeah, I'm vice chairman of pediatrics. My academic title is professor of pediatrics and my administrative title is vice chairman of the department.

Q. This increase in administrative responsibility began in 2005?

A. Right, it had been gradually increasing. 2005 is

the time I remember I crossed over to be doing more administrative work than clinical or teaching or research and that was sort of a water shed period where, again, the job description just expanded. I've been a vice chairman since 1997. But I just assumed increasing administrative responsibilities and by 2005 the administrative responsibilities outweighed the clinical ones.

Q. So as of 2005 -- was there a particular month in 2005?

A. March.

Q. March of 2005 you spent greater than 50 percent of your time doing administrative duties?

A. Exactly.

Q. And less than 50 percent of your time doing teaching, clinical and research work?

A. That's exactly right, yes, ma'am.

Q. And then as of what month in 2006?

A. January.

Q. January of 2006?

A. I became a full-time administrator.

Q. So you as of January of 2006, you spent 100 percent of your time doing administrative duties?

A. Well, that's not true. I spent 100 percent of my time -- well, that is true. I spent 100 percent of my time

doing administrative duties and within that context I continued to teach. I continued to consult on research projects but that is sort of an add-on. I no longer do clinical work. The water shed in January of '06, was that I just wasn't able to figure out how to continue doing the clinical attending work that I have been doing.

Q. Okay. So since January of 2006 you haven't seen patients as a pediatric infectious disease physician, right?

A. I continue to provide phone consultations, but I

have not seen patients since January of 2006.

Q. Who will call you for these phone consultations?

A. Well, it's a relatively large -- I don't know that the word is following but a relatively large group of physicians around the world who I have established a relationship with over the years because of my interest in a particular group of infections, the enterovirus or sort of summer virus infections. And I continue to get calls five to ten a week about difficult to manage patients.

Q. So this isn't people within your own system calling you for consultation?

A. That too.

Q. Oh, that too?

A. That too, but it extends beyond the local region.

Q. In 2005 when your administrative responsibilities became greater than 50 percent, what were the administrative responsibilities that you took over?

A. The job description for me has since 1997 been to be in charge of promotion and tenure and disciplinary actions for the department of pediatrics. So it's sort of a provost type role for a department of 360 full-time faculty members.

Beginning in 2005 we greatly expanded that role to include career counseling, mentoring, advising so that I'm now -- not only do I do the promotion and tenure, the

paperwork, coordinating and preparing for all the faculty, but now I actually do all the counseling and mentoring myself for the faculty as well.

So I have sort of a nonstop flow of people in and out of the office who have problems with the people they work with, who want to make career decisions, who are offered opportunities that they need to weigh against their current opportunities, et cetera.

Q. Okay. Prior to 2005 when you were doing clinical work, tell me a little bit about your practice and your day-to-day responsibilities?

A. Well, I've always been at the university and at Children's Hospital so it has always been a full-time

institution-based practice of pediatric infectious diseases. I did inpatient consultative services. So I would see patients at the request of doctors. These were patients who were hospitalized, patients where the question of infection came up or the management of a diagnosed infection came up or a difficult patient who they just needed additional input on.

I also saw patients in consultation in infectious diseases clinic. It ranged from one day a week to several days a week. I saw patients in consultation in the emergency department. We have a very busy phone service where we provide consultative services. It's a training

service so we have residents and fellows and medical students and associates rotating with us making it a teaching service as well as a service -- clinical-providing service. And that balance all exceeded 50 percent or 60 percent of my time prior to March of 2005.

And I also through the year 2000 had a research lab that did basic molecular virology research and my teaching has sort of been unchanged for the past 25 years despite the new administrative roles. That has just tracked along with whatever I've been doing.

Q. Okay. Back in the prior to 2005 time period, what percent of the time would you spend in the NICU?

A. I would say probably about a quarter of my time was probably with patients -- with babies in the NICU because they just happened to be sicker babies.

Q. And when you would go to the NICU you would go there as a consultant, I assume?

A. That's correct.

Q. The attending, so the neonatologist?

A. Correct.

Q. And in your facility, in your experience, who generally makes the diagnosis of sepsis? Would it be the neonatologist and then they consult you regarding management or is it the other way around?

A. It's both. So if the neonatologist makes the

diagnosis of sepsis or raises the issue or rule out or presume sepsis they might call us for confirmation or for assistance in management. On the other hand, we would also be called for a baby who is generically sick with the query is this infection or is this sepsis. So it works both ways.

Q. All right. Is it more common -- I'll tell you where I'm coming from. Doctor Martin explains the way their group works. It's more common that the neonatologist will make the diagnosis based on their clinical impressions and then call infectious disease in for assistance and

maybe antibiotic management and things like that. Is that similar to how your facility works?

A. I think what Dr. Martin was referring to is probably true at many institutions including ours and that is that neonatologists are appropriately trained to broadly make the diagnosis of rule out sepsis or presume sepsis. And they more narrowly make the diagnosis of sepsis or proven sepsis more rarely, but they cast a wide net over sick babies to raise the concern regarding sepsis. And then within that wide net, infectious disease doctors are frequently called in to help say is this a proven sepsis, is this presumed sepsis, is this a routine rule-out-sepsis patient or is this not infection at all.

And Dr. Martin in his deposition was a little too

casual with the definition of sepsis. Sepsis is not the same as infection. There are babies for whom we're asked to consult who have infection but are not septic. Similarly there are babies for whom we're asked to consult where the diagnosis is sepsis, but we cannot prove there is infection. There is a sepsis-like syndrome but whether there is proven infection or not -- so those two terms are not interchangeable.

Q. I don't mean to interrupt you, but I'm thinking while we are on this point, why don't you provide me with

what your definition of infection is and your definition of sepsis so then we're on the same page.

A. Sure. Well, it starts with the definition of colonization before we do either of the other two. Colonization means germs that live on a baby's -- and we'll talk about babies here -- germs that live on a baby's body or in a baby's body all the time and they may or may not be causing harm. But that is colonization.

Infection is when germs that are either innate to the baby, that is they are either living in or on the baby or come in from the outside and get to a place in the baby's body where they are not supposed to be as opposed to colonizing and begin to cause harm.

And then sepsis is a clinical syndrome which is oftentimes caused by infection. A clinical syndrome of

multi-organ system involvement, severe illness and life-threatening illness where the babies have signs of involvement of not only multi-organ systems but vital signs and sort of basic core body functions. So sepsis is a syndrome which is frequently caused by infection. But many, many infections never progress to sepsis.

Q. Okay. Now, I think it's clear from your report and my understanding of your opinion that you do not believe that Shane Maslanka had any evidence of sepsis,

correct?

A. Correct.

Q. Did he have any evidence of infection?

A. Maybe I shouldn't have said so quickly correct to your first question, so let me go back for a second. Our job in reviewing a baby's records to determine whether there's infection or sepsis is to form a gestalt based upon the entire picture. And I don't think it's fair to say that Shane Maslanka didn't have any evidence of infection or any evidence of sepsis. I don't think that's a fair statement.

Q. Okay.

A. I think what is a fair statement is when all the evidence is weighed there are far better explanations for what this baby had and that the likelihood that this baby had either infection or sepsis falls well below reasonable

medical probability.

Q. Okay. So what you're saying is that there may be some signs or symptoms that are consistent with sepsis but when you look at the picture in totality you to a reasonable degree of medical probability do not believe a diagnosis of sepsis or infection would be appropriate?

A. That's correct.

Q. Okay. Speaking of that, did you review any

depositions of any of the care providers?

A. No.

Q. Okay. Did you ask to see the deposition of the neonatologist, Dr. Kumar?

A. I did not.

Q. Would his observations of this child being the hands-on physician who you know treated Shane be important to you?

A. Well, I think I was able to derive what the observations and the opinions were from the medical records. If I thought that those -- my opinions of their interpretations were incomplete, I would have asked for additional information. But I used the medical record. I think it was pretty complete.

Q. We can agree the clinical assessment of the physician that is treating this baby is important in the analysis of what was going on at the time?

A. Well, I mean the treating physician is always important.

Q. Sure.

A. But we have the benefit in doing what I'm doing here of having the retrospective scope active and being able to look back at the totality of the medical record and from that I believe I understand what the doctors were

thinking when they were thinking it in chronological order as it evolved. And I also have the benefit of being able to put all the evidence together and see whether or not what they were thinking was reasonable and whether their actions reflected concern or lack of concern regarding infection and sepsis. So I think I was able to get most of that from the record.

Q. Okay. Just so we're clear, as we sit here today, it's my understanding that your opinion in this case is going to be limited to whether or not Shane's neurologic deficits are related to infection, correct?

A. Correct.

Q. You're not here to provide any opinions regarding whether the care and treatment provided in the NICU at MetroHealth Medical Center were appropriate or not?

A. Correct.

Q. You're not going to provide any opinions, I assume, on the obstetrical care that was provided to Gloria

Maslanka in this matter?

A. Correct.

Q. Let's talk for a minute about your experience with expert work. How long have you been doing this type of stuff, how frequently?

A. Um, I've been doing this since 1982 or 1983, I

think was the first case. I look at five or six cases a year. I am deposed twice a year. Some years maybe three times. Some years it's once. I do about 40 percent of the reviews and 40 percent of the testimony turns out to be on the patient's side and about 60 percent turns out to be on the defense side. And that is a pretty fair reflection of just the percentage of phone calls that request it.

I turn down a lot of cases from plaintiff's attorneys and from defense attorneys where I think that I am not the right person. And I'm turned down when my opinions don't coincide with what someone is looking for many times.

Q. All right. Have you in your work as an expert ever been asked to put together a list of all the cases you have reviewed and who you have reviewed them for?

A. There was a Colorado case recently where I was asked do that. It was a four-year look back. And I don't have that with me, but I can provide that for you.

Q. Can you provide that to Mr. Kulwicki for me?

A. Yes.

Q. Was it a federal case?

A. No, it was a Colorado case and Colorado requires that.

Q. Maybe that is new laws here.

Have you reviewed cases for the law firm of Becker and Mishkind in the past?

A. You know, I think I have. I can't remember when or what the details were. I think on one or two occasions in the past and I can't remember how recent past, but I think I have looked at cases for them before.

Q. Have you reviewed any other cases that involve this question of whether or not there is neonatal sepsis?

A. For Mr. Kulwicki?

Q. For anybody in general?

A. Oh, sure. Sure. Over the years that question has come up a lot.

Q. Would any of those cases be listed on this list?

A. Probably. There is probably some in the past four years.

Q. So is -- do you put on the list you created, is the subject matter part of the list?

A. Yes.

Q. How much do you charge for review, deposition, trial?

A. 450 an hour for review. 550 an hour for deposition and trial. And if the trial is out of Colorado, which this would be, $4500 a day instead of the 550 an hour.

Q. Do you know any of the expert witnesses that have been identified in this case?

A. I don't believe so.

Q. Ever heard of anybody in your academic world?

A. I think I've seen or heard about Dr. Martin's textbook, not about Dr. Martin himself. I don't know Dr. Leonard and I have not heard of him. And I don't think I know any of the others.

Q. Okay. You have not reviewed the medical records of Gloria Maslanka, correct?

A. Correct.

Q. You read in the expert reports particularly of Dr. Leonard that he believes there is evidence of viral infection at the time she presented for labor and then after the delivery. Do you agree or disagree with that or have any opinion?

A. I have an opinion.

Q. Okay.

A. The -- August is the peak season for enterovirus infections, summer cold viruses. It is a group of viruses that I've spent a lot of time studying and thinking about

and writing about including a book on the subject.

Q. That's listed on your CV, I assume?

A. It is.

Q. Okay.

A. Doctor Leonard is correct and that is if mom had sneezing and sniffling and cold like symptoms she probably did have an enterovirus infection during August because that is the most likely cause of sniffling and sneezing, a summer cold that is tough to shake.

Q. Low grade fever as well?

A. Correct. Correct.

Q. All right.

A. So it is very possible that mom had a summer cold.

Q. Okay. So if we assume the symptoms that he points out in his report are correct, you wouldn't disagree that there's evidence of viral infection?

A. That she had a cold, correct.

Q. I assume, though, you differ in your opinion as to whether or not that has any relevance -- I'm sensing this and I could be wrong, that you disagree as to whether or not the existence of a cold or viral infection has any significance to Shane Maslanka's course?

A. I could say with certainty that it has no significance in Shane Maslanka's course.

Q. Okay. What do you base that on?

A. Well, again this is -- Dr. Leonard has touched on an area that I've spent my career working on. And I would

challenge him to show that mom's summer cold with an enterovirus has ever or could ever produce the type of disease, type of illness that Shane Maslanka had. There are clear-cut, well-described and many times overpublished descriptions of what happens to babies in the womb and near the time of delivery of women who have this type of summer cold viral infection. And none of them fit the description of Shane Maslanka.

Q. Are there studies -- strike that.

The studies you just referred to, are any of those listed in your CV that you published?

A. Yes, most of them.

Q. All right. Can you just take a look through here briefly and point to them?

A. You bet. You want numbers or names?

Q. Numbers are fine. We'll make it easy on the court reporter.

A. Okay. Page 14 in the bibliography section on original articles and referred journals; No. 9, No. 14, No. 15, No. 17, No. 20, No. 23, No. 26, No. 27, No. 28, 32, 34, 35. The title of Article 35 is Profile of Neonatal Enterovirus Disease and Identification of a High Risk Group.

Number 37, 38, 39, 41. That one is titled,

Neonatal Enterovirus Infection, Virology and Serology and Effects of Intravenous Immune Globulin. 42, that one is titled, Diagnosis of Neonatal Enterovirus Infection Reaction by Polymerase Chain Reaction.

Number 43, 44, 45. That one is titled, Adverse Effects of Maternal Enterovirus Infections on the Fetus and Placenta. 47, 48, 51, 53, 56, 57, 59.

Q. I have a lot of reading to do.

A. Be careful what you ask for. 60, 61. Then on Page 19, Section B, Books, Number 1, the book is entitled, Human Enterovirus Infections.

Then on Page 19, Section C, invited book chapters and scholarly reviews, No. 1, 2, 5, 7, 8, 11, 12, 15, 16, 17, 18, 19. These are all chapters in pediatric and infectious disease textbooks, neonatal infections and enterovirus infections in general.

Number 20, 22, 24, 27, 28, 29, 30, 31. That one is titled, Enteroviral Infection of Neonates and Infants. 32, 33, 35, 36, 37, 38, 40, 41, 43.

Then on Page 23 under letters and book reviews, No. 6. And then on Page 23 there is the beginning of a lengthy listing of abstracts which I think I probably can save you the time. Those are generally preliminary reports that were then followed up by the papers that I already

mentioned.

Q. Fair enough. I don't know if you can simply describe kind of the conclusions you've drawn in the research you've done on this issue, but let me just ask you a couple of questions. I'm just trying to save myself reading all those articles, I guess.

Have you determined that if a mom has enterovirus at the time of delivery it can pass to the fetus?

A. Yes.

Q. Okay. But what are the complications then that you see in the neonatal period if indeed that occurs?

A. It can pass at a couple different times. One is in the period prior to the baby's birth. That's uncommon. That would be a transplacental passage where it passed from the mother's blood through the placenta across the placenta into the baby's bloodstream. That occurs but occurs rarely because the placenta is a good barrier.

The much more common time is after rupture of membranes or during the birth canal -- during delivery through the birth canal when the baby is exposed to the virus, swallows the virus, becomes infected from maternal secretions, from maternal fluids as opposed to via the blood.

The effects are very similar from both presentations on the newborn. The enteroviruses are

tropic. That means they are attracted to or they hone in on specific organ systems in the newborn.

There are two general patterns. The first involves the heart. The second involves the liver. When the babies have neonatal enterovirus infections involving the heart, the central nervous system, the brain can also be involved. When babies have infection involving the liver, the brain, the central nervous system can also be involved.

In both of those situations and both of those patterns of illness the brain involvement is typically insignificant or trivial. On rare occasion when the liver disease is severe or the heart disease is severe, there can be significant gray matter encephalitis in the baby's brain.

There is no circumstance that I'm aware of in any of the babies that I've reviewed, reported on, been contacted about, called on where a baby developed exclusive white matter disease. And particularly I'm not aware of any situation where a baby developed brain disease and brain infection without concomitant -- it's a word that starts with an S that means balanced. I'll think of it in a minute. But a concomitant and balanced effect on one of the other two organ systems, either the liver or the heart.

That is if a baby is born with severe cardiac

involvement and myocarditis and severe brain disease, that's a consistent pattern for newborn enteroviral disease. If a baby is born with severe liver disease and liver failure and severe brain disease that is consistent with neonatal enteroviral disease.

I have never seen of, never heard of, never cared for a baby who had exclusive white matter brain disease in the absence of those other findings due to the enterovirus.

Q. Do you believe that Shane Maslanka did have an enterovirus?

A. No, I don't.

Q. Why do you say that?

A. Because he didn't demonstrate any of those findings that I just described for you. That is Shane Maslanka's brain injury was not a gray matter injury and the baby did not have liver disease and the baby did not have myocarditis. So there was really no patterns, none of the patterns that we are used to seeing that were consistent with this baby's presentation.

Q. When you -- when a neonate acquires an enterovirus, can they become septic? Is that picture you just pointed out, the heart and liver involvement, would that be defined as septic or sepsis?

A. Yes. And in fact, neonatal sepsis is not just a bacterial process and enteroviruses can cause a sepsis like

picture and accompanying either the heart, parentheses brain, or the liver, parentheses brain, pattern of infection. It can also be a total system wide deterioration that includes disseminated intravascular coagulation, profound acidosis and shock and the babies can die of that neonatal sepsis like picture. When they do, the dominant finding either clinically or at autopsy is profound heart or liver involvement, parenthetically the brain can be involved or gray matter of the brain in either of those patterns. So, yes, sepsis can be a part of those. Pneumonia can be a part of those. Again, it would be very unusual to have pneumonia absent one of the primary presentations of either liver or heart.

Q. And having enterovirus?

A. Having enterovirus.

Q. Other types of viruses can cause sepsis other than the enterovirus?

A. The answer is, yes, but very rarely. Herpes, for example, is a well known cause of disseminated infection and sepsis in a newborn. It's well known but it is fortunately uncommon.

Enterovirus as a cause of sepsis is also uncommon. There is only 500 or a thousand cases of that a year -- of that enterovirus that I just described. The number of cases of herpes is probably in that same range of severe

neonatal sepsis due to herpes. Chicken pox virus can cause a severe and overwhelming infection and sepsis in a newborn. Parva virus can cause what is called a fetopathy where a baby is -- a fetus is infected and develops severe incompatible-with-life findings as a fetus and is a stillborn but not born with sepsis. Congenital rubella and congenital CMV are viruses that have distinct presentations of rashes, brain involvement, bone involvement, physical anomalies that are apparent, unique patterns of calcifications in the brain, for example. And some of those babies are sick enough to be classified as septic at birth.

But far and away, far and away, bacteria are the leading causes of neonatal sepsis. Virus can do it. Among the most common are the enteroviruses that cause the cold that Dr. Leonard thinks, and I don't disagree with it, that mom may have had. Doctor Leonard is very mistaken in thinking that this infection was a viral sepsis.

Q. Because there's no evidence of heart and brain or liver and brain involvement?

A. In the case of enterovirus there is no evidence of herpes infections -- in the case of herpes infections, exactly. So for Dr. Leonard to say that this is a virus because mom has a cold and therefore this baby is born with white matter brain damage, is a -- there just is -- it is

true, true and completely unrelated. That is baby had white matter brain damage, mom had a cold. There is no connection between the two. Let me just say -- I'm sorry.

Q. Go ahead. No. No. No, this is your chance to say what you need to say.

A. The last thing I would say about that is one of the things you'll notice if you decide to read all of those articles, which I understand your hesitation to do, is that there are between 10 and 30 million enterovirus infections every summer, every summer in the United States. Which means of the 4 million newborns who are born every year, let's say that a third of them are born in the summer months when enteroviruses are prevalent. So 1.2 or 1.3 million births during the summer months, you would expect a very large percentage of those moms to have colds because of the overwhelming number of enterovirus infections circulating in the community. The summer cold is just so ubiquitous.

Just on a pure epidemiologic population-based study, the conclusions that enteroviruses can cause brain damage is illogical. But then when you add in all of what we know about the patterns of enterovirus disease, there is just no way to make that conclusion.

Q. Do you agree with the proposition that infection itself or the inflammatory response to infection can cause

white matter injury?

A. No. And that's a much longer answer than the word no, but I'll try to summarize it in a couple of sentences.

Q. Okay.

A. As you know, because you asked the question, there is a hypothesis and a conjecture that inflammation of the mother can damage the baby without it actually causing infection in the baby. That is there can be a bypass mechanism whereby a mom can have an inflammatory response, a baby can have brain damage and it doesn't involve the baby ever becoming infected. That is no germ passes. There is just simply the inflammation in the mom that somehow damages the baby.

And I use the words hypothesis and conjecture because right now the data to support that are just that. They are hypothetical. That is there has been found a very tenuous association when looked at in a very limited fashion between things like maternal chorioamnionitis and the development of cerebral palsy. It is what is classically called in science an epi phenomenon. That is we have two observations that are true and we are trying to determine if they are related.

Q. Right.

A. So if you were born in 1920 and you ate pickles,

you have a three times greater likelihood of dying this

year than if you weren't born in 1920 and ate pickles. Now, is that three-fold increase risk in dying because you ate pickles or because you are 87 years old. And right now the cytokine hypothesis or the inflammatory response hypothesis is saying it's pickles.

Now, it may turn out some day that pickles are the causative of death. And it may turn out some day that cytokines are in fact responsible for some cases of cerebral palsy. But we are so far from being able to say that the two true observations, that is babies that are born with cerebral palsy and moms have a high incidence of chorioamnionitis are related. That -- and certainly not only are we far away on the population basis, but we haven't even come close to being able to say in an individual baby this is a baby that is infected.

And, again, the population -- the numbers are affected -- the numbers are overwhelming. 30 percent of women have histologic chorioamnionitis, 30 percent of all women at the time of delivery. Cerebral palsy is a much rarer disease than 1.2 million cases. So if all histologic chorioamnionitis caused cerebral palsy you would expect a huge number of babies born with cerebral palsy and you don't have it.

The other disconnect is that even that association, the inflammation and cytokines and brain

damage association doesn't apply to premies. It's a full-term phenomenon. So that the studies from the CDC, Karen Nelson studies and others that have looked at the inflammatory response have failed to find the association in prematures which is a remarkable indictment of the hypothesis because you would expect prematures to be so much more susceptible to everything, to any kind of inflammatory or other challenge if there was truly a relationship between the inflammatory response and babies -- and moms and baby's brain damage, premature should be the most affected.

But yet when the studies that are founded on the full-term babies were repeated in premature babies by Karen and others, they haven't been able to find it. So they don't apply to this baby because this baby is premature and at 1100 grams there is no study that can show this baby can be affected by -- even on the population basis by maternal inflammation. But I don't believe it can be said yet for any baby. That is the reason for the quick answer of no when you asked about it before.

Q. What about if there is truly infection, infection in the baby, do you agree that infected mom, infected baby

can lead to white matter damage in the brain, infection itself?

A. White matter damage, yes. But the way it works,

the way it happens is if mom has an infection and baby becomes infected and the baby becomes so hypotensive that the shock is so profound that it's not that the baby's brain is affected -- it is not that the baby's brain is infected -- I didn't mean affected. It's not that the baby's brain is infected, it is that the baby's circulation is so compromised --

Q. You need hypotension to cause the ischemia to cause --

A. Precisely. And the hypotension to the extent of such severe compromise of circulation to the baby's brain that the baby would have essentially a septic shock like picture. And where that has been seen, where brain damage -- white matter damage has been seen in newborns who are infected is when they have developed septic shock. Classically, for example, with Group B strep.

So Group B strep has actually been shown to be a cause of such profound shock in babies that it can cause periventricular leucomalacia. It can cause cerebral palsy. Not because it was Group B strep meningitis or Group B encephalitis, but because it was Group B septic shock the

baby is so profoundly sick.

And, again, I agree with Dr. Leonard. I don't believe there was any evidence in this baby there was bacterial infection. Because bacterial infection is a

profound finding in a 1100-gram baby. I think Dr. Leonard's conclusion that if this was going to be an infection it was going to be a virus was partially correct. That is, it's not a bacteria, but I think it's not a virus either.

Q. And your basis for saying it is not bacterial infection is because the clinical course was not of a profoundly ill child?

A. That's right. In addition to the fact the blood cultures were negative, et cetera, et cetera.

Q. Right.

A. This was not the timing, tempo, severity or pattern of severe bacterial infection.

Q. I'm going to get to your report and how you describe that in a minute, but just let me just ask you a couple more questions.

You note in your report in the second paragraph you have substantial personal research and interest during your career regarding infections of the neonates. What does that mean?

A. Just the articles that I read to you.
Q. Okay.
A. Many of those papers and many of the studies that we've done both laboratory and in the clinic have been neonates.

Q. But is there something personal to this? Everybody has a story and I'm just curious.
A. I have a story, if you want to hear it. When I was a trainee I took care of a baby who died of overwhelming Echo Virus 11 infection, that is an enterovirus. This was a baby for whom we couldn't do anything, couldn't even make the diagnosis until a week after the baby died, had no treatment and didn't know what killed the baby. And it had a very profound effect on me and sort of directed my research from that point on.
Q. Okay. Actually, do you have a copy of your report, it might be easier.
A. Do you want it?
Q. I want just for you to refer to it. I have my copy here. That way we are both on the same page. I'm just going to ask you some questions about the final paragraph on the first page of your report.
It's your opinion, I'm surmising, from that final paragraph here that you believe that all the complications,

neurologic complications that Shane suffers from are related to complications of prematurity; is that correct?

A. Correct, yes, ma'am.

Q. Now, Shane was born at or about 27, 28 weeks, kind of depending on how you make the calculation. Do you believe he would have had the same complications had he

been born say at 29 weeks or do you have an opinion one way or the other?

A. It's hard for me to do that. I'm not a neonatologist. To know what the difference is, for example, in a risk of PDA or RDS in a 27-weeker versus a 29-weeker or 27-weeker versus 30-weeker, I'm probably not the best person to answer that.

Q. The complications that you refer to include respiratory distress syndrome, PDA requiring surgical closure and intraventricular hemorrhage. You also include elevated red blood cell count, which we are going to talk about in a second.

As it relates to the PDA, do you know of any literature that links the existence of a PDA and/or the management of a PDA in the neonatal period to causing neurologic damage?

A. I wouldn't be able to cite you literature. The issue as it is with septic shock, the issue is one of blood

volume and profusion. And insofar as flow abnormalities through a PDA or fluid restriction as a result of a PDA or fluid restriction as a result of intraventricular hemorrhage might have an impact. I couldn't say. All I could say is the reason one might link PDA to brain damage would be as a result of blood flow to the brain. Again, I'm not the right person. I can talk about germs.

Q. No, fair enough. That's why I want to know how far --

A. Not that far.

Q. So you would defer to the neonatologists in this case, either Dr. Sherman or Dr. Martin, regarding the neurologic implications of a PDA?

A. Again, without naming names, someone other than me I would defer to regarding the complications of a PDA.

Q. So these complications you list here are based on what you see in premies in the NICU, not based on some researcher or study you have performed?

A. That's correct. Again, the generic observation is premature babies are very subject to white matter brain disease and premature babies have a panoply of risk factors that predispose them to that. And my job, as I interpret it, in this case was to see if infection was one of those risk factors and I do not believe it was.

Q. Okay. Did you take a look at the ultrasound reports as you were reviewing the brain ultrasound reports as you read the records?

A. I just read the reports, yes.

Q. Did they have any significance to you in your opinions here?

A. Well, they explain the baby's neurologic adverse outcome, but I can't draw any conclusions from them as to

when the insult occurred or how it occurred or what it was.

Q. So you can't say one way or the other whether there was any insult in utero or exactly what time any insult occurred during the neonatal period?

A. Correct.

Q. Do you have any opinion in this case, Dr. Rotbart, as to what the cause of the premature birth was?

A. No, I don't.

Q. Do you accept that infection is one of the leading causes of premature delivery in this country?

A. Not as you phrased it. But if I can rephrase it to say that bacterial infection of the mom, that is chorioamnionitis or interuterine infection as the term is sometimes used, due to bacteria, have been associated with premature births in some studies. Now, I can't go so far to say infection in general because the association with

viruses are very tenuous. But bacterial infection I can say.

I also cannot go so far to say that it is the leading cause or even among the leading causes because the studies are controversial. But it certainly has been implicated, that is bacterial inflammation, bacterial infection has been implicated as a cause of premature delivery.

Q. So bacterial infection or inflammation has been

correlated with premature delivery?

A. Yes.

Q. Let's talk about the elevated nucleated red blood cell count that you also state is a predictor of neurologic damage. Do you have any research or literature on that point?

A. No.

Q. Okay. Do you accept that there is literature out there on both sides of that point? There is some literature that says there is no predicted value of elevated nucleated red blood cell count or neurologic damage and then there is some literature that says there is?

A. I'm sure there is. I don't know enough about that literature. What I do know is we do not see large

elevation of nucleated red blood cells in infected babies. Baby's primary problem is infection. Nucleated red blood cells are not one of the harbingers or predictors of that. But I have seen studies linking nucleated red blood cells to neurologic damage in the context of asphyxia or hypoxic ischemic encephalopathy. And I'm sure, because it's true for every study, I'm sure there are studies that have found the opposite also.

Q. Is there a normal range for a 27-week or 1100 grams of nucleated red blood cell counts?

A. There probably is.

Q. Is there a specific number in Shane's chart that you are referring to when you talk about the elevated nucleated red blood cell count?

A. I can't remember. I'd be happy to look back. I can't remember if there was one count.

Q. Yeah, if you could do that, I would appreciate it.

A. The numbers that I was probably referring to are those from August 1st and August 3rd of 20 and 49 respectively. I think those would have been the ones. The subsequent nucleated red blood cell counts are lower and, again, without being an expert in that seemed reasonable to me.

Q. Okay. Do you know how long the elevated nucleated

red blood cell counts need to persist in order to have this link between that elevation and neurologic damage?

A. No.

Q. So this to me is just kind of in addition to the main thrust of your opinion?

A. Yeah. Maybe I should have phrased it better by saying that the presence of nucleated red blood cells is not an association that we see with infection. That probably would have been more within my area of knowledge of nucleated red blood cells than the way I phrased it.

Q. Do you agree, Dr. Rotbart, that Shane Maslanka did

have evidence of neutropenia?

A. Yes, the baby's white blood cell count was low. But the baby's white blood cell count was on the low side, but the baby's neutrophil count was on the low side. Neither of which was below my understanding of what we see with premature babies who are not infected.

Q. Does this get to the calculations you made previously?

A. Yes.

Q. Well, let's just get into this issue now that we're here. Tell me -- well, do you know off the top of your head what the range is, what we look for? And we're talking about neutropenia now.

A. Yes.
Q. What is the laboratory value that we look for in making a diagnosis of neutropenia in a premature baby like Shane?
A. If you flip to that paper, I think I can do it by heart, that paper actually summarizes a number of other papers. There is a graph on the second page of the paper.
Q. We're at the birth weight of less than 1500 grams?
A. Less than 1500 grams. So if you look at the cutoff values that this author of this particular paper used and they are based upon sort of the classic studies of neutropenia in very low birth weight infants. Those

numbers are a pretty good guideline.
And at no time during this baby's post-delivery life did the ANC that the baby had drop below those norms with the exception of two blood counts that were done by automated differential where the ability to count bands and myelocytes in immature cells is not adequate. So that on all the manual differentials wherever there was a white blood cell count that was then counted manually, the baby always had absolute neutrophil counts above those cutoffs. So it was never neutropenia.
Q. You've read how Dr. Martin calculated the ANC. He adds the polys and the bands to a percentage and then puts

them against the white blood cell count?

A. Right. Again, that's the reason that I showed my calculations the way I understand they should be done to Mr. Kulwicki because everybody came in with a different formula for the way they should be calculated.

Q. Let's go through that. You sure you don't want to just show us these notes?

MR. KULWICKI: I'm sure.

Q. (BY MS. REID) Did you go through every lab value?

A. Just until, I think, through the 8th or the 9th. So there were eight or nine of them.

Q. Let's go through them because I want to understand this, exactly how you made the calculations.

A. Sure.

A. Okay. So these pages are Bates numbered, but I'll read you the values that were used that I used.

Q. Okay.

A. August 1st at 0810 hours. So at that point the baby would have been about an hour and a half old.

Q. Right.

A. The corrected white blood cell count was 4.43.

Q. Right.

A. That number then is multiplied by .31 which is the polys plus the band.

Q. Okay.
A. I don't have a calculator but that number is 1300 something. It is above the cutoff of 500 which is the threshold for babies in the first six hours of life.
Q. All right.
A. And then we can do it for each one, but I can tell you that with each one of the subsequent ones are the exact same result. And that is if you look at the hour that it was drawn, calculate the age of the child. I think the next one was at 42 1/2 hours of life that the CBC was done. And if you look at that value which is 5.56. And I think that that number was corrected in the notation below to 3.73. So do you see where --
Q. Yes.

A. -- the corrected count is 3.73. If you take 3.73 and multiply that by 39 plus 15 or 54. So by .54. You also get a number that is well above the threshold of a baby that is at 42 hours of life which is less than 1500 or something like that. Again, you've got the paper in front of you.
Q. Right.
A. I just went through each of those and the last one that I did was -- it was either August 6th or August 8th. And there were no counts using that method that ever fell

below the normal neutrophil threshold with the exception of the two automated CBCs for which we do not have a manual differential. One of them on August 4th at 0500 and the other one August 6th at 0405. And both of those were done by a very inaccurate automated differential method where you can't count bands, myelocytes, promyelocytes, et cetera.

Q. Well, how do you even calculate the ANC on those counts, the automated counts without --

A. Well, because the automated count -- you see where it says gram percent?

Q. Gotcha.

A. Gram percent is instead of looking at the actual cells -- and I'm not a hematologist, but my understanding is that that is just a much less accurate way of looking at

it. But if you do multiply, for example, on the 8-6, 0405, if you multiply .146 times 4.88 it comes out to a number which if I remember correctly is below the threshold on there. But it's only on the two automated counts that that happens. When the cells are actually looked at there are no neutropenic lab counts for the baby's entire life.

Q. What did you do -- and I'm forgetting, is there a corrected white blood cell count in all of these?

A. No, there isn't. There is for many. When one is

provided, I used it. When there isn't one provided, it is because the nucleated red blood cell count had dropped below the level that the lab has to correct. The corrected white blood cell count is because of the nucleated red blood cells.

Q. Right.

A. So when the nucleated red blood cell count drops below whatever level the lab is no longer tricked by --

Q. There's no reason to correct --

A. -- they no longer correct it.

Q. So if it wasn't corrected I assume you used the white blood cell count that was present?

A. Right.

Q. So basically what you're saying is based upon your calculation here -- and I think you make the calculation the same way Dr. Martin does.

A. It is possible. Several people gave different formulas.

Q. I think his formula is the same. The question becomes whether or not the value that you determined is truly neutropenia?

A. I can't say yes to that because I don't remember if we even came up with the same values.

Q. Okay.

A. We may have used the same method but at some points I'm not sure that he -- whether he -- for example, on one of those there was a myelocyte or promyelocyte. That is an even more immature cell, but it should be counted in the formula for calculating ANC, because it's a neutrophil. And I don't remember if he used that or not. I didn't go back and track his calculations. I just made my own to determine whether or not the baby ever fell below the threshold.

Q. And the myelocyte you referred to -- there was one lab value I think that had a myelocyte?

A. I'll find it for you. I closed my book.

Q. I think it was 8-6 at 1720.

A. That's correct. There was one on 8-6 and then there was a promyelocyte on 8-12, but I didn't make calculations for that date because by then the total white count was up over 13,000. And, again, there was another

white myelocyte observed on August 14th and again that was not -- we are up over 19.8 white cells and I didn't make a calculation for that ANC either.

Q. So your opinion then to a reasonable degree of medical probability, based upon these calculations, that there was no evidence of neutropenia in the neonatal period?

A. Correct.

Q. Do you agree with the premise, though, that persistent neutropenia can actually be a sign of sepsis?

A. No, that is actually a very important next point.

Q. Okay.

A. If you don't lead me there, I will try to get there myself. And that is that exactly the opposite pattern of what you just said is consistent with sepsis. That is when neutropenia is due to sepsis it is transient. It occurs at the time the sepsis is diagnosed. And when you look at all babies who are neutropenic, the medium number of neutropenic white blood cell counts that they have, that is how many abnormal CBCs do they have in a neutropenic range is one.

That is babies who are neutropenic as a result of sepsis are not persistently neutropenic. They are transiently usually once neutropenic and then with treatment their neutrophil count rapidly bounces back.

And that goes to the part of the rest of my report where I talk about the pattern of neutropenia in infection is one of rapid response, not of persistence. So that if in fact this baby had persistent neutropenia, which he didn't, but if in fact this baby had persistent neutropenia it would even speak more strongly or equally strongly

against there being infection, because it is the noninfection associated neutropenia that persists. Infection associated with neutropenia is transient. It's fleeting. It's usually just once.

Q. Is that because of antibiotic response?

A. Well, it's because of response. Now, in many cases -- all cases babies are receiving antibiotics. Again, is that true, true and related or true, true and unrelated. The baby's -- the observation that is true is that when infection occurs neutropenia is fleeting. Usually one abnormal CBC. Whether that's because we're fixing it or because the baby is fixing it, is impossible to distinguish. All those kids are treated. So how do we know if it is pickles or old age.

Q. What is the significance then in general of a persistent neutropenia?

A. It suggests a noninfectious cause. That is when you look at -- if you read that paper you'll see that it is the noninfection -- first of all, most babies with

neutropenia do not have infection. The majority of the babies with neutropenia are due to something else. Of those babies who are neutropenic due to infection and those babies that are neutropenic because of noninfection the persistent neutropenia occurs in the noninfection. So the

persistence of neutropenia suggests that the neutropenia is not related to infection because the infectious neutropenia responds quickly.

Q. And is there something in the pathophysiology of neutropenia that leads you to that conclusion or is that just based on this study and what we've seen in babies with neutropenia?

A. Right. Well, it is not just this study. This study has nothing to do with my report, which I wrote before ever seeing that study. But I wrote my report because that's just what we know about neutropenia. Neutropenia resulting from infection is a transient fleeting phenomenon.

As I was looking for cutoff values to show Mr. Kulwicki for neutropenia yesterday, this study just happened to say the exact same thing that my report had said and that is it's the neutropenia of noninfections that persist. The neutropenia of infections do not.

Now, you asked why. And is it physiologic or is -- it is probably physiologic. And that is that if you

have an infection that transiently suppresses your neutrophil count, the response to the infection is the body mounts a neutrophil response. So that if it's infection causing your neutropenia, initially the body quickly says,

we can't afford to be neutropenic because we have an infection.

Q. So march those neutrophils out?

A. That's right.

Q. Um, how about the concept of the low white blood cell count in and of itself or can we not separate that from the neutrophil count?

A. You can't separate it.

Q. Okay.

A. Leukopenia in and of itself is not a predictor. It's neutropenia that is a predictor. In this case it's a neutropenia that didn't exist. And it is a neutropenia that if it did exist, as Dr. Martin suggests in a persistent state, it predicts against infection.

Q. This is something we hear in cases a lot involving infection that when you have a low -- you can get a suppressed white blood cell count due to massive infection?

A. Absolutely.

Q. Okay. But you're just saying that would be an initial response, it wouldn't be a persistent response?

A. Exactly. That's exactly right.

Q. Do you believe that Shane had evidence of anemia?

A. Again, I'm neither a hematologist or neonatologist, but every premature baby has evidence of

anemia. That is we do so much blood drawing on babies and their blood volume is so low that premature babies, especially 1100-gram babies, are always in the NICU and always requiring transfusions.

There was no evidence in this baby, in contrast to what Dr. Leonard suggested, that this baby's bone marrow was depressed. This baby had anemia based on the basis of frequent blood drawing but there was no evidence of bone marrow suppression.

Q. You don't see any evidence of thrombocytopenia?

A. There was a single platelet count of 127,000. There was one of 147,000, which is normal. 127,000 is a low platelet count, but barely low. And it was never sustained. That is it never occurred again. So, again, we're talking about patterns.

The pattern of thrombocytopenia that results from infection is a progressive one where the platelet counts drop, drop, drop, drop and keep dropping sometimes until needing transfusion and are frequently associated with coagulation abnormalities, which this baby did not have.

So that syndrome of disseminated intravascular coagulation we see in babies who are septic but with low

platelet counts, but they are also bleeding from every vena puncture site because they can't clot at all. Their

platelets are being consumed in the process of trying to deal with their overwhelming sepsis. This was a transient value which happens in prematures and newborns all the time and is of no significance.

Q. Do you have to have evidence of thrombocytopenia -- let me put it another way.

Do all premies have -- with sepsis have thrombocytopenia?

A. No, I wouldn't say all to anything. But it is a contributing -- that is a real thrombocytopenia that is a thrombocytopenia with 40,000, 20,000, 50,000 that lasts from days to a week is seen in many babies with sepsis including this enterovirus sepsis that we talked about before where thrombocytopenia is very frequently seen. You don't have to have it, but the absence of it speaks against infection.

Q. Metabolic acidosis, how do you define that?

A. Metabolic acidosis is an imbalance in the pH of the baby's blood. This baby had metabolic acidosis. It's various cutoffs that can be measured by the bicarbonate on the electrolytes or it can be measured by blood gas, the bicarbonate in the CO2. This baby had metabolic acidosis.

Q. Can we agree that metabolic acidosis can be a sign

of sepsis?

A. Yes.

Q. What do you believe the metabolic acidosis was related to in this case?

A. What the doctors believed it was due to and that is a fluid imbalance. The baby was dehydrated and either because of the dehydration -- and some of the dehydration, I think, was intentional to try to protect the baby's brain after the hemorrhage was diagnosed and perhaps in trying to help manage the PDA to prevent congestive heart failure.

But when you dehydrate a baby intentionally or unintentionally it decreases profusion to the tissues and lactic acid builds up and an acidosis results.

Q. The issue of the blood culture that you point out in your report, we can agree that you can have sepsis with a negative culture?

A. Yes.

Q. And why is that?

A. Well, in the rare cases where it's a virus, as Dr. Leonard points out, viral cultures -- unless you request a viral culture -- in fact, the routine cultures don't look for viruses. So it could be because it's sepsis due to overwhelming enterovirus or overwhelming herpes. That was not the case in this baby.

Then there are about 20 percent of babies who have

a clinical sepsis. That is everything about them is septic and yet we don't grow anything from the blood. And there are a number of different reasons. And I think Dr. Leonard summarized them pretty well. That is that some bacteria may not grow well. Some babies we may not put enough blood into the tube to try and grow it.

But we do know there are about 20 percent of babies who have a clinical diagnosis of sepsis and fit the patterns that we understand for the sepsis and their blood cultures don't agree and we treat them as if they had sepsis. Again, I don't believe that was the case in this baby.

Q. You mentioned in your report that you believe there's a -- numerous conditions of the prematurity that account for the low white blood cell counts. You know what I'm referring to?

A. Yes, of course.

Q. What are those conditions that you're referring to in your report?

A. Well, more than half of babies who have neutropenia at 1100 grams do not have infection. So the majority, greater than half of babies, of very low birth weight babies who are truly neutropenic -- this baby was not neutropenic, but more than half of babies, as I said in the report, do not have an infection. So that means by

definition there are other causes.

Now, there have been some that have been referred to in reports and depositions by others that are clearly true. So preeclampsia or eclampsia or maternal hypertension is associated with neutropenia that's not infectious. Asphyxia is reported to cause neutropenia, which is not related to infection.

And then presumably there are others because I don't think that of the more than half of babies of this weight who are neutropenic and do not have infection we can explain all of them on the basis of mom's blood pressure or on the basis of asphyxia or on the basis of any other C in the A, B, C listing so there are other causes. I'm not expert enough in the noninfectious causes of neutropenia to be able to list causes, but there are others.

Q. Now, you're saying in your response now -- you're talking about -- and I just want to make sure my terms are correct -- that more than 50 percent of babies with neutropenia have no infection?

A. Of low birth weight. If you look at all babies with neutropenia, babies -- neonates with neutropenia, 75 percent of babies do not have infection. Only 25 percent of all babies with neutropenia are found to have an infection. 75 percent are not. There is a higher proportion of very low birth weight babies, under 1500

grams, for whom neutropenia is associated with infection, but it is still less than 50 percent.

Q. When you use the word "neutropenia," is that synonomous with what your report describes as a white blood cell count of less than 5,000?

A. White blood cell count of less than 5,000 is leukopenia. Neutropenia is what we're talking about as a predictor of infections in this case. And, again, when leukopenia exists, it's either because there is too few lymphocytes or there's too few neutrophils. Those are the two components. So if we are talking about a baby who has low neutrophils and leukopenia, it's the neutropenia that is causing the leukopenia.

Q. Gotcha. This antibiotic response that you mentioned in your report, have we discussed that, your opinion on that?

A. Yeah, again, if a baby is neutropenic because of bacterial infection then the neutropenia will respond in the context of the baby receiving antibiotics. You asked whether it's due to the antibiotics. I can't say it's necessarily due to, but it is in the context of the treatment that we provided to the baby.

Q. You mentioned the fact that normal liver function speaks against infection. Does that go back to what we were talking about before?

A. Yes, so -- just, again, just tracking this, in the context of a summer virus infection that mother may well have had, as Dr. Leonard points out, one would expect concomitant findings in the baby that involves the liver, that involves direct hyperbilirubinemia that would involve cardiac manifestations and the other pattern.

When Dr. Martin was asked the question, wouldn't you expect to see elevated liver function tests in a baby with sepsis, he said not unless it's a virus. Well, that's what Dr. Leonard says it was.

And when he was asked, wouldn't you expect to see direct hyperbilirubinemia, again, I think probably because of what I wrote, he said, no, not unless it's a virus which is what Dr. Leonard said the baby had. So I agree with Dr. Martin that you would expect to see, if this was a virus, one of those findings which you did not see here.

Q. If it is bacterial, though, you don't necessarily need those components, correct?

A. Correct. Although, you may well see it. So the babies, for example, who have bacterial sepsis will also often have a direct hyperbilirubinemia, but it's more common with viral disease.

Q. We talked about earlier, I think, specifically as to whether inflammation or infection can cause white matter injury to the brain. Certainly neurologic deficits can be

as a result of infection, correct, in general?

A. Right, you can have -- if the brain is infected by virus or bacteria brain damage can result, absolutely.

Q. And that's why you were called in here was to say whether or not infection played any role in the neurologic sequelae in this case?

A. That's correct.

Q. I'm assuming that based on your expertise you can't quantify which of the complications caused which portion of the injury to Shane Maslanka's brain?

A. I cannot, no.

Q. Whether it is the PDA, the IVH, anything like that?

A. Correct. All I can say is I don't believe any of it was caused by infection. But what caused it or how it broke down in terms of importance, I can't tell you that.

Q. You say none of it was caused by infection because you see no evidence of infection?

A. Correct. I put the word "no" in parentheses because nothing is absolute, but the picture of this baby in the composite is not a picture of an infected baby.

Q. I understand. Looking at the whole picture there may be some signs and symptoms of infection. I think we said this before, but looking at the whole picture, you don't see an infected baby?

A. Correct.

MS. REID: Let's just take some time to go through my notes. Let's just go off the record for a minute.

(Whereupon, a short recess was taken.)

Q. (BY MS. REID) Doctor Rotbart, did you review the pathology reports in the case?

A. I did not.

Q. Do you have any sense as to whether or not they showed any type of inflammatory process or anything significant?

A. There was reference in a number of the reports to chorioamnionitis.

Q. Okay. But you didn't take that into consideration -- that wasn't something you reviewed or relied upon for your opinions in this case?

A. Well, I didn't review the pathology reports and as we discussed before, that's not a finding of viral infection. And it's not -- it is a finding with bacterial infection. It is present in 30 percent of women. So that it really is very difficult to make an association of that with anything.

Q. Okay. As we've gone through, I admit in not the most organized fashion, I think we've covered your report and what I at least perceive to be your opinions in the

25 case, but I want to give you the fair opportunity if there

1 is something that I missed that you intend on focusing on
2 to let me know that?
3 A. No, I think so we've covered everything. Really
4 this is just a case that if you'll pardon the expression
5 fails the smell test. That is the timing and tempo and
6 severity and pattern of illness in this baby is not an
7 infectious disease.
8 Q. To summarize, too, just what you told me at the
9 outset of our deposition, we can agree that since March of
10 2005 through the present you do not spend 50 percent of
11 your time in the active clinical practice of medicine
12 and/or teaching, fair?
13 A. That's right.
14 MS. REID: That's all I have. Thank you.
15 MR. KULWICKI: Doctor, in Ohio we have a process
16 whereby you can review the transcript before it becomes
17 your official word. I would suggest you do that just
18 because of the terminology that has been used here is
19 pretty complicated.
20 THE DEPONENT: Sure, happy to do that.
21 (Whereupon, the deposition concluded at
22 10:46 a.m.)
23

WHEREUPON, the within proceedings were concluded at 10:46 a.m. on March 9, 2007.

I do hereby certify that I have read the foregoing deposition and that the same is a true and accurate transcript of my testimony, except for attached amendments, if any.

_____
HARLEY A. ROTBART, M.D.

( ) No changes ( ) Amendments attached

SUBSCRIBED AND SWORN TO before me this _____ day of _____, 20_____.

_____
NOTARY PUBLIC
Address_____

_____

My commission expires _____

23
24
25

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

I, Kelli J. Wessels, Registered Professional Reporter, appointed to take the deposition of:

HARLEY A. ROTBART, M.D.

certify that before the deposition the deponent was duly sworn to testify to the truth; that the deposition was taken by me on March 9, 2007; then reduced to typewritten form, by means of computer-aided transcription; that the foregoing is a true transcript of the questions asked, testimony given, and proceedings had.

I further certify that I am not related to any party herein or their counsel and have no interest in the result of this matter.

IN WITNESS WHEREOF, I have hereunto set my hand March 21, 2007.

_____

Kelli J. Wessels
Registered Professional Reporter

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25

ESQUIRE DEPOSITION SERVICES
Registered Professional Reporters
303 E. 17th Avenue, Suite 565
Denver, CO 80203
303-316-0330

March 21, 2007

Harley A. Rotbart, M.D.
University of Colorado Health Sciences Center
4200 East Ninth Avenue, Box C-227
Denver, CO 80262

Re: Maslanka vs. MetroHealth Medical Center
Deposition of: HARLEY A. ROTBART, M.D.
Date of Deposition: March 9, 2007

Dear Dr. Rotbart:

Enclosed is a complimentary copy of your deposition.

It was agreed at the time of the deposition that you may use the copy transcript as a convenience in reading and signing your deposition.

Also enclosed is the original signature page and form of amendment for changes, if necessary. All changes are to be made on the amendment form only. Please sign the original full-size signature page and amendment form, have them notarized, and return to this office by April 21, 2007.

You may retain the copy transcript. The only pages necessary to return are the original signature page and amendment form.

Please call if you have any questions regarding this procedure.

Sincerely,

ESQUIRE DEPOSITION SERVICES

Enclosures

cc: David Kulwicki, Esq.; Christine Reid, Esq.

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Registered Professional Reporters
303 E. 17th Avenue, Suite 565
Denver, CO 80203
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Christine S. Reid, Esq.
Reminger & Reminger
1400 Midland Building
101 Prospect Avenue West
Cleveland, OH 44115

Re: Maslanka vs. MetroHealth Medical Center
Deposition of: HARLEY A. ROTBART, M.D.
Date of Deposition: March 9, 2007
Trial Date:

Enclosed is the above original transcript...

____ signed, no changes

____ signed, with changes, copy enclosed

____ not signed, notice duly given March 21, 2007, pursuant to the Rules of Civil Procedure

____ not signed, notice duly given March 21, 2007, since trial is set for

____ to be signed in court or signature pages to be returned to court on date of trial

____ signature pages/amendments to be returned to above counsel

____ signature not required

21 _____ sent via UPS _____

22 _____ hand-delivered by courier _____

23 Sincerely,

24 ESQUIRE DEPOSITION SERVICES

25 cc: David A. Kulwicki, Esq.