IN THE COURT OF COMMON PLEAS

SUMMIT COUNTY, OHIO

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Mackenzie Lynn Tarle, a Minor by and through her next friend and natural mother, Michele Tarle, et al.,	:	
Plaintiffs,	:	
vs.	:	Case No. CV 2001 05 2137
Akron General Medical Center, et al.,	:	
Defendants.	:	

- - -

DEPOSITION OF MARK H. LUQUETTE, M.D.

- - -

Thursday, November 14, 2002
3:15 o'clock p.m.
3242 West Henderson Road
Columbus, Ohio

- - -

SUSAN M. OLSON
PROFESSIONAL REPORTER

- - -

ANDERSON REPORTING SERVICES, INC.
3242 West Henderson Road
Columbus, Ohio 43220
(614) 326-0177
FAX (614) 326-0214

APPEARANCES:

DANIEL M. FINELLI, Attorney at Law
Finelli & Margolis, P.L.L.
730 Leader Building
526 Superior Avenue
Cleveland, Ohio 44114-1902

On behalf of the Plaintiffs.

JEFFREY E. SCHOBERT, Attorney at Law
Post Office Box 5521
3737 Embassy Parkway
Akron, Ohio 44334

On behalf of Defendants Edward Ferris, M.D., and Summit Obstetrical and Gynecological Associates, Inc.

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THURSDAY AFTERNOON SESSION
November 14, 2002
3:15 o'clock p.m.

- - -

STIPULATIONS

- - -

It is stipulated by and between counsel for the respective parties herein that this deposition of MARK H. LUQUETTE, M.D., a Witness herein, called by the Defendant under the statute, may be taken at this time and reduced to writing in stenotypy by the Notary, whose notes may thereafter be transcribed out of the presence of the witness; that proof of the official character and qualifications of the Notary is waived.

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P R O C E E D I N G S

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MARK H. LUQUETTE, M.D.,

being by me first duly sworn, as hereinafter certified, deposes and says as follows:

EXAMINATION

BY MR. SCHOBERT:

Q. Doctor, please state your full name.

A. Mark Luquette, L-U-Q-U-E-T-T-E.

Q. All right. And, Doctor, your current business address?

A. Is 700 Children's Drive.

Q. Now, that's Columbus, Ohio?

A. Columbus, Ohio. Yes. 43205.

Q. Who do you work with or for at this point?

A. I'm an employee of Pediatric Pathology Associates of Columbus, P-Path for short. It's a wholly-owned subsidiary of the hospital.

Q. All right. How many -- To the best of your knowledge, how many members are there in the Pediatric Pathology Associates?

A. There are nine pathologists. But I think some of the other doctoral staff are also part of

the corporation. I don't know the exact number of people.

Q. When did you join that group?

A. First joined the group in 1993.

Q. Okay. Obviously, you must have made some change or -- because I'm sitting here looking at a report that's got Magee Womens Hospital on top.

A. Correct. I worked for Children's Hospital for eight years.

Q. All right.

A. And then I took a job at Magee Womens Hospital in Pittsburgh, Pennsylvania, to go back to my wife's home town. And about eight or nine months of being there, I resigned over failure of the university to honor recruitment commitments and took a job back here at Children's Hospital with the same group that I was working with when I left.

Q. Tell me a little bit about that. While you were there, who were you employed by in Pittsburgh?

A. I got two paychecks when I was in Pittsburgh. One of which came from the University of Pittsburgh Medical Center, and the other which came from the University Physicians Practice Group.

So I had -- The politics of it are that I had both university privileges as in University of Pittsburgh, like the undergraduate university, and also had the University of Pittsburgh Medical Center privileges.

Q. Okay.

A. And I got paychecks from each of those people.

Q. Okay. What was your job duties for the eight months you were in Pittsburgh?

A. Well, I was in Pittsburgh for 11 months.

Q. Oh, 11 months. Okay. For the 11 months --

A. I turned in the resignation at eight months. When I was in Pittsburgh, my job title was director of developmental and perinatal pathology.

Q. Okay. And your current title, as we sit here today, is there a specific --

A. My current title is -- job description title is staff pathologist. And my administrative title is director of perinatal pathology and informatics.

Q. And informatics?

A. I-N-F-O-R-M-A-T-I-C-S.

Q. Okay. Let's -- Did you bring with you today a copy of your CV?
A. No.
Q. All right. You will --
MR. FINELLI: I'll get you one.
BY MR. SCHOBERT:
Q. Give me just, then, a brief background where did you do your undergrad. and medical school?
A. Well, my undergraduate degree was a BS in biology done at the University of New Orleans.
Q. And when did you complete that?
A. 1980.
Q. All right. And then?
A. Then I went to medical school at the Louisiana State University Medical Center in Louisiana.
Q. Okay. And when did you complete your medical school?
A. 1985.
Q. All right.
A. And I did two years of medical school, one year what they call a year of fellowship, and then another two years of medical school. I participated in a program which allowed the medical

students to take a year out of medical school to do a pathology-like internship to see if they like the field of pathology, which actually counted as a year of residency credit.

Q. Okay. Where did you do that at?

A. I did that at the University of Alabama in Birmingham.

Q. All right. Then what after you completed medical school?

A. Then I went and did a residency in pathology -- well, anatomic pathology, at the University of Washington. Well, no, that's backwards. Washington University Medical Center in St. Louis at Barnes Hospital.

Q. And how many years? Was that a three-year program?

A. That was a four-year program.

Q. But you got one year of credit, so you did it in three or --

A. I chose not to take advantage of that and did the full four years.

Q. All right. So then you would have completed that about 1989?

A. That's correct.

Q. All right. Then what happened?
A. From 1989 to 1993, I did a two-year fellowship in pediatric pathology at the Children's Hospital of Pittsburgh.
Q. All right. And you said '89 to '93?
A. I'm sorry. '89 to '91.
Q. All right. Okay. So then you completed your two-year fellowship. And then what?
A. Then I took a faculty appointment at UTMB Galveston.
Q. Okay.
A. Which stands for the University of Texas Medical Branch in Galveston.
Q. And what did you do there?
A. I was a staff pathologist and also with an emphasis on perinatal pathology.
Q. Okay. And you did that from, what, '91 to '93?
A. '91 to '93.
Q. Okay. So that brings us up to current.
A. That's correct.
Q. All right. Board certified?
A. Board certified in anatomic pathology and pediatric pathology.

Q. When did you receive your certifications?
A. If my memory serves me correct, anatomic would have been 1990 and pediatric path. in 1993.
Q. Describe for me, I guess, your clinical practice currently. I mean, what are -- not clinical, your -- well, describe for me what you are professionally doing right now in terms of practice.
A. I spend approximately three quarters of my time doing clinically-related work and about a quarter of my time doing research.
Q. All right. Let's talk about what you do during that -- for your clinically-related work. Just give me some idea of how you break your time down.
A. It's a mixture of autopsy and surgical pathology.
Q. Okay.
A. And included within the surgical pathology is consulting for developmental pathology at the university, well, Ohio State University.
Q. Well, is all of your work, whether it be autopsy or surgical pathology in the field of pediatrics alone, or do you do more than that?
A. It's all in the field of pediatric

pathology, which encompasses reproductive biology. So I work with specimens all the way from shortly after conception to teenagers and adults.

Q. When you try to break it down between obligations for autopsy versus surgical pathology, is there any way to break that down percentagewise?

A. It changes every year when they make the schedule based on how many people are in the practice. It tends to be half and half.

Q. All right. When you do the autopsy portion of it, are you at times, therefore, doing autopsies, say, on stillbirth fetuses that die in utero or children that die shortly after delivery?

A. Well, yes. The politics are a little more complicated. If it's a near term baby or neonat., those tend to go into general autopsy service. If it's a first or second trimester loss, they generally give those to me outside of the autopsy service.

Q. All right.

A. Because I do the developmental and the perinatal work there, I pick up those specimens outside of my usual service rotations. I pick them up all the time, whenever they come in.

Q. So in terms of the population that you perform autopsies on, then, just so I'm clear on that, how would you describe that, the make-up of that population that comes to you that doesn't go to the general autopsy service?

A. You mean within the Children's Hospital?

Q. Yes.

A. Well, if it's a first or second trimester fetus, most of those are going to come to me.

Q. Okay. Is there anybody else in the children's group or in your group at Children's Hospital in Columbus who does that work other than you?

A. If it's considered a perinatal case, it's mine.

Q. Okay. All right. What else makes up that population of autopsies?

A. Well, there are trimester stillbirths, neonatal deaths, infant and child deaths, and adults that die from childhood diseases, which are a very small part of it.

Q. In the population of third trimester stillbirths or neonatal deaths, could you list for me the most common reasons or causes of death that

you ultimately determine?

A. If it's an intrauterine demise in the third trimester?

Q. Yeah, let's start with that.

A. Well, it's -- you know, a heterogenous population of causes. Placental problems, umbilical cord problems make up a large part of it. Some of it is intrauterine infections. Congenital defects.

Q. All right. Within the population of intrauterine infections, when you have that as a diagnosis, do you have the opportunity to try to make determinations as to what the bacteria or the cause of the infection is; the organism?

A. Well, that's a part of doing the autopsy to try to figure out the cause of death, yes.

Q. What would be the common findings that you might see that would lead you to a conclusion that you have an intrauterine infection as the cause of death in a third trimester stillbirth?

MR. FINELLI: Are we talking stillbirth or antepartum?

MR. SCHOBERT: I said stillbirth.

THE WITNESS: Well, you do a detailed exam which involves a number of studies. First of

all you do your external examination of the fetus. But then when you open the fetus, you would take blood cultures. We might culture the lungs. If you thought it was viral, you might culture the liver. So you can culture various organs. You also take samples of various organs and look at them under the microscope to see if there are signs of infections. Some types of infections produce specific findings, and others don't.

Q. Okay. When you culture, you said from the lungs, like sputum from the lungs or culture blood might be some of the things you would culture?

A. I can't culture sputum, because if the fetus is stillborn, it can't cough it up. But we sterilize the surface of the lung and then take a scalpel and try to sterilely remove the piece of a lung and put it in a sterile bottle and send it to the lab for culture.

Q. In those cases of third trimester stillbirths that you have performed autopsies, has GBS ever been the organism that is cultured out from the blood or from the lung?

A. Yes.

Q. All right. How frequent does that occur?

A. The question is too general for me to answer.

Q. All right. Is Group A strep, is that an organism that -- within the realm of --

A. It's a recognized cause of neonatal sepsis, yes.

Q. Now, is there any way to distinguish within, you know, the fetus injury that may be caused by one type of organism, such as Group A strep, versus other bacterial organisms? I mean, is there any way to distinguish other than the culturing specific to that organism?

MR. FINELLI: You're still talking stillborn?

MR. SCHOBERT: Yes.

THE WITNESS: Culture is the definitive test. Although now in contemporary times, there are genetic mechanisms. You know, ways to study DNA that could be used. But in common practice, culture is the way that you do it.

BY MR. SCHOBERT:

Q. All right. In the realm then of neonatal deaths, is GBS also recognized as one of the organisms that can cause infection and result in

death?

A. Yes.

Q. The -- What other -- Assuming you had a case, such as that, a neonatal death, and you cultured out GBS. What other findings might you see that would be consistent with infection as a cause of death?

A. Beyond the culture?

Q. Beyond the culture. I mean, what other findings might you see in performing the autopsy that would be consistent with infection as the cause of death?

A. One of the chief -- It's a series of negatives, as well as positives.

Q. All right.

A. One of the chief problems in culturing anything from an autopsy situation is contamination. So the mere presence of a positive culture has to be taken in a guarded fashion. Because of possibilities of contaminants, you can't place all -- culture can't be the only finding if you necessarily want to prove that the infection was what caused the death. You want to find other things. It could be inflammation in the placenta.

It could be inflammatory cells in the lungs. Those are examples of evidence that there's an infection.

Q. Okay.

A. If you find nothing in any of your microscope slides and no other evidence for infection, if the only evidence of infection you have is a positive culture, then you're on shaky ground.

Q. From your expertise, if you've made a determination that infection has led to neonatal demise, what is the mechanism that is generally associated with acquiring an infection leading to death?

A. In the neonatal period?

Q. Yes.

A. Well, the fetus either is infected in utero or intrapartum or shortly after birth. And as a result of that infection, gets septic and dies.

Q. And other than what we've already talked about in terms of finding, you know, culturing various parts of the organism to determine if you have a bacterial infection, is there any other pathological finding that would lead you to, you know, be consistent with your diagnosis of

intrauterine infection?

A. Well, it depends on what the type of intrauterine infection it is.

Q. Let me ask it a different way. If you have an intrauterine infection, let's assume GBS as the organism, and it's led to death in the neonatal period, can it demonstrate itself as having an effect on the brain? Can sepsis lead to, you know, pathological findings in the brain?

A. Through the mechanisms.

Q. Right. All right.

A. Not directly.

Q. I understand. All right. First the mechanisms that will lead to injury to the brain.

A. Well, if the individual is septic and they go into cardiac arrest or cardiac failure, then the blood stops circulating in the body, and then there's no blood to the brain and there can be damage to the brain.

Q. And what kind of -- from a pathological standpoint if you're looking at an autopsy, somebody that has had this process occur, what types of findings might you see in the brain?

A. In a dead individual?

Q. Yes.

A. If -- A septic individual may or may not have brain damage. And it may or may not be part of what you find in a dead person; died of sepsis. There are a lot of different ways that sepsis can kill someone. If a septic individual had brain findings, they could either be findings of anoxia, you know, a lack of oxygen getting to the brain. Alternatively, if the individual was septic, they could develop what's called an intravascular coagulopathy, which means they form blood clots within their vessels and they could shower the brain with small blood clots or just small blood clots in their brain leading to hemorrhages in their brain. There's a variety of different things that could happen.

Q. If anoxia occurs and there's a lack of O2, how might that demonstrate itself hypothetically at autopsy?

A. Well, typically if the individual has been alive -- We're talking about an alive born person that later dies?

Q. Yes.

A. They typically have what's called an

ischemia reperfusion injury. So they have a period where their brain is starved for oxygen followed by a period when oxygen is restored to their brain and they develop death of a specific population of nerve cells in their brain.

Q. Is that generally a global finding?

A. There can be some -- if it progresses enough, they may get swelling in their brain. That might be more global. But the ischemia reperfusion injuries, specifically referred to as ponto subicular necrosis -- that's P-O-N-T-O, subicular, S-U-B-I-C-U-L-A-R -- ponto subicular necrosis is necrosis or death of the neurons in the pons, which is part of the brain. And the subiculum is another part of the brain. So death of those ponto subicular cells is indicative an ischemia reperfusion injury.

Q. Okay. How about to other organs in the body? If sepsis is the mechanism of death, what types of findings might we see to liver, kidney, heart, lung?

A. Well, each case is different. If there was a profound episode of hypoxemia, which means not enough oxygen in the blood, organs like the liver

and kidneys could get starved for oxygen and essentially die. So the individual could go into liver and kidney failure.

Q. So from a pathological standpoint, what would you anticipate seeing hypothetically if that occurred?

A. It would depend on the time interval between when the injury occurred and the time that individual died. So you may see nothing, or you may see changes in the microscope that shows the cells are all dead.

Q. Okay. Do you know why there was a determination made at some point to culture for a Group beta strep at generally 36 weeks in the pregnant female?

A. Which pregnant female?

Q. Any pregnant female.

A. I'm not familiar with the office practices of an OB-GYN.

Q. Okay. That's fine. All right. Looking at your report, I have a report dated January 16th of 2002. Correct?

A. That's correct.

Q. Have you produced any other notations,

writings, reports, or anything else related to this particular case based on review of any materials?

A. I just have the notes that I initially took to generate the report, which consists of a large Post-it note.

Q. Okay. Could I see that for just a moment? All right. And just if you could explain for me 98-261 refers to what?

A. That refers to the case number that was on the slides that I reviewed.

Q. Okay. And then your note itself has numbers 1, 2, and 3. What does that refer to?

A. Those would be the slides that I looked at.

Q. All right. So to your knowledge, there was three total slides that you had available to review?

A. That's correct.

Q. Okay. From your -- from everything you've had the opportunity to review in this case, was it your belief that those would be the only slides available to review from the placental pathology?

A. I need to refer to the original pathology

report.

Q. Whatever you need to refer to.

A. Okay. Unless I'm missing it, the pathology report which identified those slides as belonging to this patient -- Oh, here it is. I'm sorry. Right at the edge of the page. It just says -- it does say block three. And that means that there should be three slides.

Q. I'm not necessarily questioning you didn't. I just want to make sure that from your understanding you had all the material.

A. I have three slides to look at. The report says that there were three sides. It's possible that further examination of the specimen and more blocks of slides were produced that aren't reflected in this report that could have been reflected in an addendum report that I was not privy to.

Q. All right. Then the additional notations that appear, say, next to No. 1, what type of referencing is that, or what does that all refer to?

A. Well, those are abbreviations that I use. UC stands for umbilical. And times two, meaning that there were two pieces to look at. AF is acute

funisitis. And UV means umbilical vein.

Q. All right. So acute funisitis is one of the findings made from slide No. 1 from the specimens of the umbilical cord?

A. That's correct. Yeah, the specimen -- Yes.

Q. Okay. What is acute funisitis?

A. Acute funisitis means that white blood cells from the fetus have gone through an umbilical vessel into the issue that's immediately around the umbilical vessel.

Q. I'm sorry. Could you say it one more time?

A. Okay. The umbilical cord contains three blood vessels. And those three blood vessels are not touching each other. They're separated in space by a gelatinous material call Wharton's jelly. And then there's like a skin, if you will, around the outside of the umbilical cord. And probably less than a quarter of the surface area, if you were to make a cut across the umbilical cord, probably less than a quarter of the surface area would be actually filled with blood vessels. The rest of it is gelatinous material called Wharton's jelly. So

if -- you know, circulating through those umbilical blood vessels is fetal blood. And in that fetal blood can be white blood cells that belong to the fetus. If they're responding to an inflammatory signal, they can go through the blood vessel wall into the Wharton's jelly that surrounds them.

Q. Okay. And how is it that something gets into the Wharton's jelly? I mean, what is it that's in the Wharton's jelly creating this response of these white blood cells?

A. Well, the umbilical cord is in the amniotic cavity. So if there are bacteria in the amniotic cavity, fragments of the bacteria that trigger inflammatory responses are sensed by the fetal white cells, and the fetal white cells go after those signals, so they move out of the blood vessel through the Wharton's jelly in an attempt to get out of the cord into the amniotic cavity.

Q. You say these white blood cells in this case would be produced within the fetus?

A. That's correct.

Q. Okay. So getting back then, and I appreciate your patience, to the acute funisitis, it describes the process of what? The production of

the white blood cells or finding of white blood cells?

A. It essentially is the fetus's response to the infection.

Q. Is there a period of time that must occur in terms of the infection being present or at least -- you know, in contact with the cord before the fetus will produce white blood cells in response to that?

A. In humans I don't know that that's known with certainty. What we know about the human acute inflammatory response is that the first white cells in general will show up at the site of infection at about 12 hours. Precisely how long a fetal umbilical cord has to be in contact with a bacterial substance or whatever for the fetus to respond is not really known.

Q. So there is no parameters you could put on it, no less than or no greater than, based on your knowledge and experience?

A. In round figures, I would say that the contamination would have to be there for at least 12 hours. But does that mean it couldn't be 11?

Q. I understand.

A. You can't say, because this is a biologic response.

Q. Right. Now, in terms of when you looked at that slide, was there a certain amount of white blood cells that you have to see in order for you to determine if that process is going on, or is it just simply a matter of if you see just any amount it's acute funisitis?

A. Well, there are three blood vessels in there. And if the white cells are inside the blood vessel, they would be circulating. If they start to go through the blood vessel wall, you just call it a vasculitis or inflammation of the vessel. If they get through the vessel wall and into the Wharton's jelly, then it's funisitis. But without any further qualifications saying funisitis doesn't tell you if you're seeing one, two, or three blood vessels involved or, for that matter, if there's a couple dozen white cells going through the vessel at one point or thousands of white cells going through the blood vessel walls.

Q. So the number of white cells you see doesn't give any -- have any significance?

A. In terms of using the term funisitis, if

you're very liberal with it, you could say if you saw two or three or four white cells in the Wharton's jelly.

Q. Okay. How do you use the term then when you -- How do you define what is enough to be called acute funisitis?

A. If I see just a few white cells, convincing white cells in the Wharton's jelly, I will call it funisitis.

Q. From your review of these slides, do you recall whether you can quantify how many white cells you were seeing within the Wharton's jelly?

A. Well, since there's no significance to quantifying it, I don't take note of that.

Q. All right. Fair enough. What is the significance, then, to you of a finding of acute funisitis in this particular case?

A. Well, in any case it means that the fetus is responding to an infection.

Q. Okay. What was the second thing you read here? Umbilical vein?

A. All right. That was the blood vessel. There are two arteries in one vein in the umbilical cord. The only place I saw the inflammation was in

the umbilical vein.

Q. Okay. What's your next one say there?

A. FM is fetal membrane.

Q. Okay.

A. And I have on the focal AI, which means that I saw acute inflammation in the fetal membranes, but that it was focal.

Q. All right. Explain that, if you would. I mean --

A. Well, the way the examination is done is that the baby is in the amniotic sac. And imagine it like a bag. And to examine it, you take a scissors, and you cut down the bag and then you roll the membranes. And essentially it comes out like a jelly roll. And then after you put it in formaldehyde overnight and it hardens up, you make a slice of it. So when you look at it under the microscope, it looks like a jelly roll. If you're to find inflammation all throughout the membranes, that would be diffuse. If you find it only in a few spots, that's focal.

Q. All right.

A. In this case, the inflammation was focal.

Q. And what, if any, significance does that

mean in terms of looking at the overall process that was going on for this fetus at the time of birth?

A. Some people could interpret that that means that the infectious process is just in its very starting point. But the truth is that the amount of inflammation that you see really doesn't tell you much about how long it's been there.

Q. All right. Now, typically when you see that finding of focal inflammation, are you -- can you characterize what the cause of the inflammation is? I mean, whether it's a bacterial or some other source or cause for that inflammation?

A. When you have acute inflammation in the fetal membranes, the assumption is that it's a bacterial infection.

Q. All right. What is typically then the source of the bacterial infection?

A. They're ascending vaginal infections.

Q. Can you tell, again, anything about the length of time that there may have been this focal inflammation caused by this -- or assuming -- the assumption being caused by some bacteria by the findings that you saw, could you tell how long it had been going on?

A. With these findings, I would say that the -- it took at least 12 hours for the neutrophiles to get there. How much time beyond that is -- it's not possible to say.

Q. Is there no window that you can put on the outside? In other words, the best you can say is it took at least 12 hours, but I can't tell you how long after that it may have been ongoing?

A. That's correct.

Q. Okay. What did you have down here next right underneath there?

A. Triangle is a delta, which is short for change. And no MV, or maternal vascular change. One of the good places to look at the mother's blood vessels is in the fetal membranes. And I made a notation that I didn't see any abnormal maternal vessels.

Q. Okay. What else did you write then under No. 2?

A. AI, which is acute inflammation. And then C, chorionic, PLT, plate, width, OC, line of the width, and FSVV is fetal stem vessel vasculitis.

Q. All right. Explain that if you could.

A. Well, the umbilical cord contains three

blood vessels. And then when it reaches the placenta, it spreads those vessels out over the top of the placenta where they branch into the part of the placenta that faces the fetus, which is referred to as the chorionic plate. And so the -- we call those stem vessels, because they're kind of like the early branches of the tree, if you will.

And just like the process that we explained in the umbilical, how the fetal blood vessels responding to the infection come out of the umbilical vessels, they also come out of the stem vessels. The same mechanism. But these are just branches of the umbilical vessels.

Q. Right. So what you're seeing is, again, the reaction to the white blood cells coming out? Don't let me say it. You tell me. What is it that you're seeing, then, just so I'm clear?

A. It's the exact same scenario. The white cells coming out of the umbilical vessels and grow branches of the umbilical vessels. It's the same process.

Q. All right. Again, my question is having seen that process in slide 2, apparently, right? -- can you put any timing as to either the minimum

amount of time it takes for that to occur and/or a maximum amount that that process might be ongoing from what you saw in the slide?

A. You rarely ever see the fetal response without seeing the chorioamnionitis. So when either occurs simultaneously, we with the maternal response, or there is some time interval between the maternal and the fetal response, I'm not aware of any literature that answers that question.

Q. Okay. All right. Your next line?

A. That says term maturation. That means that chorionic villi, which are the little fingers that -- the fetal tissue that is surrounded by the mother's blood that actually acts kind of like the lung for the baby exchanging the nutrients and gases and -- those villi, they're either developed appropriately, or they haven't. And so the term maturation means that they're developed appropriately for what you would expect for a baby that's at the end of their gestation. A normal gestation period, 38 to 42 weeks. I guess a simpler way of putting it is that indicates that the placenta is normally developed.

Q. Okay. What did you write next?

A. Large arrow up means marked. VCCV, vascular congestion of the chorionic villi with three foci suggestive of red infarcts, but not basal.

Q. All right. Let's start with the first term. Again, that it indicates?

A. Marked.

Q. Marked?

A. Uh-huh.

Q. All right. What's the significance of that finding? Is that a finding you made, first of all, that there was vascular congestion, you said, of the chorionic villi?

A. Uh-huh.

Q. All right. What's -- The chorionic villi exists where? Are they on the chorionic plate that you were talking about?

A. Let me think about it. Without giving an entire anatomy lesson on the placenta, you start out at the umbilical cord, and then they talk about how the vessels spread out over the top of the placenta. Imagine the placenta as a disc. And those blood vessels keep branching and become progressively smaller. And as they're branching, think of it like

a tree. Imagine the trunk of the tree being the umbilical cord. And as you get out to the smaller branches, imagine the bark being the outside and the -- then there's whatever you call the wood pulp on the inside of each branch. And the blood vessels would be traveling up the trunk and down through all this wood pulp. When you get to the very finest twigs out in the end of the tree, those would be the chorionic villi. And those are surrounded by mother's blood so that the baby's blood that's circulating through those villi can come as close as it can get to mother's blood to exchange nutrients and waste products.

Q. All right. So what's the significance then of the finding that there's -- what did you call it again?

A. Well --

Q. Vascular congestion?

A. It means that if I look at the diameter of the blood vessels in these villi, the diameter is wide. So they are congested with blood.

Q. All right. Is that the way they normally should be, or is that an abnormal finding at that term maturation?

A. It's often an incidental finding. By itself it's often an incidental finding.

Q. All right. In the context of this particular case and your other findings, is there significance to that finding?

A. Well, vascular congestion can be an incidental finding. But it is also part of one of the earliest changes that occurs when the placenta is starved for oxygen.

Q. Okay.

A. And when you -- You're probably familiar with the term like a myocardial infarct, where the heart gets an infarct and a piece of the heart muscle dies. Well, you can have infarcts in the placental, as well; where if a piece of the placenta is starved for oxygen, that part of the placenta dies.

And there are a variety of changes that you can see under the microscope. Progression of changes that occur in that process of the placental tissue being starved for oxygen and subsequently dying. The very first finding that you can get is called a red infarct.

Q. All right.

A. And the red infarct is -- looks something like vascular congestion, except it's exaggerated to the point where it's difficult to see the villi individually. If normally you were looking at villi, like a bunch of checkers in a grid, and if you pushed all the checkers together to the point where you couldn't tell one checker from the next, that would be extreme vascular congestion of the villi that's giving you a microscopic appearance of a red infarct.

In this case, it was hard to know if it was a red infarct or not, because these lesions weren't found at the base of the placenta near the mother's side of the placenta, but they were found in the middle of the placenta. If they're found at the base and they're wedge shaped, you can be sure they're an infarct. If they're found in the middle, they may be an infarct; they may not.

Q. All right. Did you come to a conclusion in this case whether they were an infarct or not?

A. My conclusion was that I couldn't tell if they were an infarct or not.

Q. If it's an infarct, assuming that to be true, is there anything that -- in terms of the

process, the time that it takes for this process to occur, can you make any conclusions about timing of how long it takes to get to that point?

A. I don't know if anybody knows how long it takes to involve the changes of a red infarct. It's generally accepted that it's a recent event.

Q. All right. And recent being defined as?

A. Since vessels can expand very rapidly, the change -- I'm speculating based on what I know about --

MR. FINELLI: Don't speculate.

BY MR. SCHOBERT:

Q. I don't want you to speculate. All I want you to tell me what you know to be something you can say to a reasonable degree of medical probability.

A. I'm not sure that anybody can -- it's even in the book anyway.

MR. FINELLI: If you don't know, don't speculate.

BY MR. SCHOBERT:

Q. All right. So there was a possible red infarct. And then what was this over here? I'm sorry. Three --

A. Foci.
Q. Okay. So there were three possible foci of red infarcts? Is that what you're saying?
A. Uh-huh.
Q. If, in fact, it's a red infarct, is there any way to determine the cause of the oxygen -- As I understand you said it's oxygen depravation that can lead to this kind of a finding. Is that what you told me?
A. Right. I mean, just looking at the slide, you can't tell how it happened.
Q. All right.
A. But there could be associated findings that would explain it.
Q. Were there associated findings in what you saw that would tend to lend an explanation as to if that is a red infarct?
A. Not in what I had to examine, no.
Q. What was your third one, then?
A. It says little or no acute inflammation in chorionic plate. And then it says: Fetal stem vessel vasculitis. And that's congestion of chorionic villi. So in the first slide it was umbilical cord and fetal membranes. And in these

two slides, two and three, it was placenta. So both of them have vascular congestion. Both of them had inflammation of the fetal stem vessels. And in slide three, there was very little inflammation of the chorionic plate itself.

Q. Okay. And what significance, if any, do you draw from those findings?

A. I already can tell from this section here that there's inflammation in the chorionic plate. This is just merely a matter of recording what I'm seeing. And it's consistent with what I've seen in the other two slides. It doesn't add or detract from what I already know by looking at slides one and two.

Q. All right. Now, going back to your report, then, when you use the term then chorionic villi under diagnosis, explain that diagnosis just so I'm clear.

MR. FINELLI: The term chorionic villi?

BY MR. SCHOBERT:

Q. It says, No. 2, chorionic villi. And you're finding, just so I'm clear, how you use that term or why you made that finding.

A. Well, I don't see a No. 2.

Q. No. I'm sorry. I'm just looking right there.

MR. FINELLI: You want to know what chorionic villi means?

MR. SCHOBERT: Well, I just want to know -- He says my diagnosis is. And then it says chorionic villi as one of the things listed.

THE WITNESS: I can answer the question.

BY MR. SCHOBERT:

Q. All right.

A. There is a standard what we call pathology/morphology way of making a diagnosis. So I say arm, left, upper, excision, nevus, like a -- you know, mole. Okay? So in the placenta, the anatomic structures that I use to describe a placenta is I separate into three compartments. I say something about the chorionic villi, I say something about the fetal membranes, and I say something about the umbilical cord. So starting the sentence with the words chorionic villi is just telling me that this is the anatomic location of tissue that I'm going to tell you about.

Q. All right. Fair enough. Now, under fetal membranes, then, your diagnosis is acute

chorioamnionitis. I'm sure we must have discussed it as we went through that, but is that then the process we were talking about under what slide number?

A. In slide No. 1, where is says focal acute inflammation, that's what you see in a slide. And when you see acute inflammation in the fetal membrane slide, the diagnosis is acute chorioamnionitis.

Q. All right. And define chorioamnionitis so that I know exactly how you define it.

A. Chorioamnionitis is the presence of acute inflammatory cells in the subamnionic connective tissue.

Q. All right. So going down to your conclusion, then. These findings, in my opinion, cannot be viewed as etiologic for cerebral damage with any degree of certainty.

Explain that for me, if you would.

A. What I mean by that statement is that taken as an isolated finding, that is there's an individual with acute chorioamnionitis, you cannot conclude with any certainty that there's going to be brain damage just because there's chorioamnionitis.

Q. Am I correct in understanding that the -- Strike that. Let me ask it this way. From your review of this pathology, do you believe that there was an infectious process going on in the placenta?

A. In a way the question doesn't make sense, because the infection is not really in the placenta.

Q. All right. The infectious process affecting the placenta?

MR. FINELLI: At what point in time?

BY MR. SCHOBERT:

Q. Well, at the time that these slides are produced. I mean, do these slide evidence, in your opinion -- Can you tell from these slides whether there was infection within --

MR. FINELLI: At the etiology?

BY MR. SCHOBERT:

Q. At the etiology of the chorioamnionitis.

A. Well, pretty much by definition, acute chorioamnionitis means that there's a bacterial infection.

Q. Right. But -- So you are -- your pathology -- The review of these slides indicates that there was an infection that could be -- you could determine there was infection going on from

what these slides tell you?

A. Right. But you have to be careful to qualify that. Okay? There's subclinical infection, and there's, I guess you'd say clinical infection. In other words, you can have bacterial exposure and inflammation of the placenta with absolutely no problems to the fetus. And, in fact -- I can't give you a percentage, but I'd say most of the times I got a placenta that's got a lot of chorioamnionitis in it, the fetus is just fine. In a term fetus.

Q. Do you have, on those cases, the opportunity to get clinical information about the status of the fetus and whether or not the fetus has had blood cultures or sputum cultures, and whether or not those cultures are positive?

A. Most of that, from my personal experience, comes from practicing in places where with the pathology report, I usually have a copy of the delivery records that has the patient's APGAR scores. So when the baby is at term, between 38 to 42 weeks gestation, more likely than not when I see chorioamnionitis, the APGAR scores are usually normal. Eights or nines.

Q. All right. But have you ever seen

situations where you think chorioamnionitis and you have seen low APGAR scores?

A. That does happen, yes.

Q. Have you had the opportunity in those cases to go and do further clinical investigation or review the clinical records of the fetus or the neonat to determine if there's been blood cultures done at or near the time of --

A. I don't follow the cases like that, no.

Q. Assuming hypothetically there were positive blood cultures taken within an hour -- the blood taken within an hour of delivery that had GBS as the organism, and you had findings such as you've described here --

MR. FINELLI: I'm going to object to this, because he's not talking about clinical medicine. Basically his report states his findings on review of the pathology slides. That's the confines of his report.

BY MR. SCHOBERT:

Q. Can you rule out the findings from the placenta as having -- being etiologic for cerebral damage in this case? Can you say with any degree of certainty? I guess, my question to you is: Can you

rule it out based on your findings from your review of these slides, can you rule out the fact that those findings are not the cause of cerebral damage in this child?

A. I misplaced where you put the word fact in there.

Q. Well, you used the term --

A. I think to answer that I can reiterate the question and answer, I think. But basically by seeing the inflammation on these slides, can I be sure that they weren't the cause?

Q. Yes.

A. No.

Q. What other materials do you have in that folder in front of you today, Doctor?

A. I have a communication signed by Janice M. Lage. A letter about this deposition and its location. Cover letter that came with the slides. Copy of my report. Copy of the pathology report that accompanied the slides to identify them. Copy of a communication to Mr. Schobert from Alias Challen [phonetic]. That's what's in here. Oh, wait. A report.

Q. Right. That's a transmittal letter.

A. That's the transmittal letter. Another transmittal letter.

MR. FINELLI: You're going to the depo.

BY MR. SCHOBERT:

Q. All right, Doctor. Let's turn back -- Well, first of all, is that all the materials you've had an opportunity to see or review or -- outside of the pathology itself?

A. The ones I know about. If I have anything else, it's not been unpacked.

Q. Okay. Going back then to the report of Dr. Lage, do you know Dr. Lage?

A. No.

Q. All right. You've had a chance, I assume to review this report?

A. Yes.

Q. In order to try to cut this short, are there findings that Dr. Lage has made, since it appears she also reviewed three slides, and we'll make the assumption she's reviewed the same slides that you had, that you disagree with in terms of what she has reported as findings or the conclusions that she has drawn?

A. Yes.

Q. All right. Why don't we take them in a chronological fashion, if you want to point them out to me.

A. Okay. Well, first I would not assume that I saw the same slides that she saw.

Q. Okay.

A. And she concluded that the findings indicated an ascending amnionic fluid infection of at least 72 hours duration. I don't know how she made that determination. I don't know what literature supports that.

Q. All right. So it's your position that from those three slides -- well, it's your determination in general, nobody can make that particular finding from looking at pathology slides? Or is it more specific that these slides themselves can't allow you to draw any conclusions?

A. It's my understanding that the literature doesn't support one's ability to precisely time an infection like she has in this report.

Q. All right. You use the word precisely time. I mean, let me ask the question in a generic sense. Is there any -- do you have any knowledge, either from your own experience or your review of

the literature, where one can attempt to time to any degree the length of time an infection has existed from review of the pathology, such as this?

A. In several attempts of reading the literature, I haven't been able to find that. It's very vague. People use words like recent, and they don't qualify them.

Q. All right. So is it your belief that based on everything that you've had the opportunity to review, you've never seen where anybody has demonstrated or written about the ability to time to any degree the length of time an infection may have been evident, based on review of pathology?

A. Some of that is direct, and some of it is inferential from related subjects on chorioamnionitis.

Q. Okay. Well, let me ask the question in a different way, then. You do not have an opinion, yourself, then, as to the length of time that an infection may have been present?

A. Beyond how long I know it takes the inflammatory cells to get there, I don't think I would say that it's my expert opinion that no one can tell me if the inflammatory cells had been there

18 hours, 24 hours, 48 hours, or 72 hours. That distinction cannot be made as far as I understand the process and what's been written about it.

Q. All right. So what you told me earlier is that you can generally stick by the rule that it would take at least 12 hours, but you can't go and time it past that point?

A. Well, right. I mean, this statement here suggests that there's no way that this was 48 hours. It had to be at least 72. Well, I think that's patently incorrect.

Q. Okay. Any other basis for your opinion that it's patently incorrect, other than what you've had a chance to tell me?

A. No.

Q. What other statements do you take exception to either in terms of findings or conclusions in this report you've reviewed?

A. The second paragraph says the placenta shows chronic villitis. I did not make that observation.

Q. All right. How would you define the term chronic villitis?

A. It's a term that's used loosely. And

it's very subjective. But to me it means that there are chronic inflammatory cells in the chorionic villi.

Q. And when you use the term chronic, is there a definition you apply to the term chronic?

A. Chronic inflammatory cells refers specifically to a type of white blood cell. Usually including lymphocytes and macrophages. Lymphocytes and monocytes.

Q. So you need to find each one of those in order for it to be termed chronic --

A. Well, what they call mononuclear inflammatory cells, which is generally lymphocytes and monocytes or macrophages. One or both of those types of cells has to be present in the tissue for you to consider it chronic villitis.

Q. And in a generic sense, can those cells be seen when they exist through what you do as a placental -- or as a pathologist looking at a placenta?

A. Yes. But the term chronic villitis doesn't give you much information. It could be one villi with a few inflammatory cells in it, or it could be half of the villi in the specimen. It's an

unqualified observation. If it were present in half of the villi, I would assume that if she found a few inflammatory cells in one villus, if I had seen that, I probably would have disregarded it as a finding of no significance and would not have applied the diagnosis of chronic villitis to it.

Q. So is it your definition that you need to see a certain quantity of those cell types, those inflammatory cell types before you could term it chronic?

A. What I mean is if I see even one convincingly inflamed villus -- if I saw a villus that had two or three lymphocytes there, I wouldn't say anything about it. If I found a significantly inflamed villus like at low power I could see it full lymphocytes, even one villus, I would call it chronic villitis. But in my microscopic description, it would state I only saw one villus with chronic inflammatory cells in it, as opposed to the diagnosis that might be like marked or diffuse chronic villitis, which would further be described as having numerous villi inflamed.

Q. All right. I understand. What is the time that's generally required for the production of

that cell type?

A. We know even less about that than we know about chorioamnionitis. I mean, chronic inflammatory responses, based on what we know, generally take on the order of two, three, four days or more to form. But the majority of times you make the diagnosis of chronic villitis, it's an incidental finding of no significance.

Q. So, generally, it could take two, three, four days for those types of inflammatory cells to be produced?

A. To arrive in the tissue.

Q. To arrive in the tissue so that they would be there at the time that you're reviewing it under a microscope?

A. Right.

Q. And there's also, then, the determination in your mind that there has to be -- depending on the quantity of those you see, you might call one -- definitive one the chronic, but then you would in your microscopic define it as being a singular finding, rather, unless you saw it more often that you could call it a more generalized finding. Is that --

A. That's correct.
Q. All right. And if I understand it, you didn't see any of these inflammatory cells when you reviewed the slides you had to review?
A. That's correct.
Q. All right. What else do you disagree with?
A. Well, the statement is also made there is meconium in the amnion and chorion from the extraplacental membranes. I did not make that observation.
Q. All right. Is there any -- If, in fact, that does exist, have you seen that finding before when you've looked at placentas?
A. I actually do research in this area. And I can tell you that you can find a little bit of meconium in most placentas if you look hard enough. So the statement made in a vacuum is almost meaningless. In the context of -- when meconium is meaningful, it usually comes with an additional qualifier, that you have a reactive amnion. The amnion cell layer usually become reactive. The presence of a little bit of meconium in absence of reactive amnion is a null finding. Usually you

state if you see meconium in macrophages -- First of all, it just says it's meconium here. When it's significant, it's usually present within inflammatory cells called macrophages. And to make sense out of it, you really would have to say how many of them there are. And, again, you can find rare pigmented or meconium macrophages in lots of placentas. But the ones that tend to be more significant are the ones where you find numerous pigment latent or meconium latent macrophages. Those are also usually visibly green to the eye.

Q. Why are those significant when you do find them?

A. Well, they indicate that the meconium was there long enough for the cells to go over and ingest it.

Q. Okay. Is there generally any, to your knowledge, clinical significance attached to that when it occurs?

A. Well, that's a whole other lecture. But, I mean, basically we can have a light staining of the fluid with meconium. We can have a heavy staining of the fluid with meconium. Meconium can be passed right immediately prior to the baby being

born and then nothing happens. Or the meconium can be passed at an earlier point in time and be in utero with the baby for a while. There's a lot of variables. This individual may have seen some meconium there. But if she looked at the same slides as I did, since it's a subjective evaluation, I may have -- if there were meconium there, there wasn't enough for me to think it was worth listing as a finding.

Q. So the difference between -- I mean, you're taking exception to her report could be either one, you didn't see any at all, or what you saw, you didn't, based on your knowledge and experience, feel was significant enough to report?

A. That's correct.

Q. Am I saying that fair?

A. That's fair.

Q. All right. Go on. What else?

A. That's also the finding of this pathology report, that they did not see meconium.

The discussion about the baby's head circumference as being 45th percentile while the body weight is 80th percentile being diagnostic of intrauterine growth asymmetry is factually

incorrect.

Q. All right. Why?

A. There are two types of growth retardation. Symmetric and isometric. And in symmetric growth retardation, the baby is proportionally small, which means the head, arms, legs, and organs are all small to the same degree.

Q. All right.

A. In asymmetric growth retardation, the head is usually the normal size, and the body is small. And the most common reason for that is a placental problem. So this doesn't fit the definition of asymmetric growth retardation, because she's saying that the head is smaller and the body is larger. And that's backwards. In asymmetric growth retardation, the head is normal, and the body is small.

Q. Okay.

A. So it's wrong.

Q. All right. What else?

A. So the term -- so the statement this is diagnostic of intrauterine growth asymmetry between the baby's head and his body length, I disagree with.

Q. Well, let me ask you this. Is it typical to have a head circumference measured at the 45th percentile with length and weight measured at 80th percentile in a baby at this maturation?

MR. FINELLI: Objection. I mean, he's a pathologist. He's not a neonatal --

MR. SCHOBERT: I know. But he just took exception to the rule, so I'm just asking.

THE WITNESS: No. I make that assertation at the autopsy table all the time. If I did an autopsy on somebody who had a 45th percentile head and an 80th percentile body, I would consider that normal.

BY MR. SCHOBERT:

Q. Okay. What else?

A. Again, the statement about 72 hours is made at the bottom.

Q. Okay.

A. And then the statement that the chronic villitis has been present for days -- associating the baby's small head size to villitis that I didn't see, again, I take issue with that.

Q. All right. Did your findings include a finding of vasculitis of the placenta surface

vessels?

A. The term I used for that is fetal stem vessel vasculitis.

Q. Okay. Outside of then the opinions we've explored in your report or your opinions as it pertains to Dr. Lage's report, are there any other opinions you're aware of that you intend to be expressing at the trial in this case?

A. No.

Q. Let me ask you just some generic questions, then, Doctor. How often have you been asked to get involved in review of medical/legal matters?

A. I'd say on the average two to four times a year.

Q. All right. And for how many years?

A. Ten years.

Q. All right. How often have you been asked to do what we're doing here today, to give a deposition?

A. Fewer than a half a dozen times.

Q. Total?

A. Total.

Q. Have you ever been asked to do this by

any attorney for cases that were in Ohio other than the one obviously today?

A. Not that I recall. Since I do so few of these over such a long period of time, you know, if I did another one in Ohio, it must have been long enough ago that I don't remember.

Q. All right. Do you recall any of the attorneys that you may have worked with in the past who have consulted with you, asked you to review placental pathology?

A. Yeah, I can remember the names of the some of the attorneys.

Q. Can you tell me some of those names?

A. I've reviewed some cases for Bruno & Bruno in New Orleans, Louisiana, which is where I grew up.

Q. Well, that worked out well.

Do you know whether they were representing plaintiffs or defendants?

A. Plaintiffs.

Q. Okay. Who else?

A. I've done work for Kirby & Edwards, which is now Kirby & Holt in North Carolina.

Q. You seem to have some good geographical

spots to pick. And, again, are those representing plaintiffs or defendants?

A. In those cases, plaintiffs.

Q. All right. Any other attorneys?

A. This firm here, Finelli & Margolis, although I can't tell you how many times I've reviewed documents for them.

Q. All right.

A. And another group, I believe it's Glenn & Sonoma.

Q. And where are they out of?

A. I'm not sure.

Q. All right. But you were representing plaintiffs or defendants?

A. Plaintiffs.

Q. In this -- Well, for Mr. Margolis or Mr. Finelli, how often -- do you have any idea how often they've asked you to consult and review cases on behalf of their clients?

A. I'm not sure. Two, three, maybe.

Q. Have you ever given a deposition in any of those cases that they've asked you to consult on?

THE WITNESS: I don't believe I've given a deposition for your firm before.

MR. FINELLI: And I'll stipulate on the record that this is the first case he's reviewed for us ever.

BY MR. SCHOBERT:

Q. All right. Doctor, then when you're asked to do this, how do you charge? What are your fees for review, the time that's spent initially reviewing versus today versus if you're asked to come to trial?

A. Well, usually I would get a phone call and there would be a discussion of the case. That might be 20 or 30 minutes. And they decide that they do or don't want to send me slides and medical records to review.

Q. What type of things would you discuss in that case to make the determination whether to send you the slides?

A. Well, just the nature of the case. Whether it's a type of case that I would have the expertise to review.

Q. Okay. Go ahead then.

A. And then I would receive slides or records to review. I wouldn't charge for the time that I initially spent talking on the phone to

decide whether or not it's something that I should look at. But when I received the slides, I generally charge about $250 an hour to review slides or documents. And it's not always a direct reflection of how much time I spent. I might only charge somebody for two hours of work if I spent four. I sort of give an hourly charge based on what I think the work was worth. When I send the bill, it's just $250 on an hour times so many hours. For a deposition, I charge $300 an hour with a two-hour minimum. So it's $600 if you stay under two hours. And after that, it's 300 an hour, which I can break into 15-minute increments or something. I'm not a barracuda. And for a trial, I haven't done a civil trial. I've testified in three criminal trials where I don't get to charge anything related to my work. And I haven't testified in a civil trial. So I'll probably set that fee if and when that ever happened. If I did it today, I would have to go consult people on what the going rates are. But it would probably be on the order of about $2,000.

MR. FINELLI: We can let you know.

THE WITNESS: I gather that it averages around upwards of $2,000. I know there are some

people who have a high opinion of themselves that charge exorbitant fees, but the judges get mad at them for doing that.

BY MR. SCHOBERT:

Q. Now, Doctor, from the placental pathology that you looked at, and I apologize if I've asked you, can you determine the etiology of how this infection occurred? In other words, where it originated, where it would have gone to first, second, and third? Do you understand my question? I know it's poorly worded, and I'm very tired. It's a been a long three weeks. But if you understand it. Or else I'll try to rephrase it.

A. Well, since there is chorioamnionitis in the placenta and then the fetus either got that beta strep in the womb or the birth canal.

Q. Okay. If it got it in the womb, what would be the mechanism that would most likely occur? How would that -- Is it an ascending bacteria that gets into the uterus? I mean, how does that occur?

A. In general, the bacteria would be in the amniotic fluid and would be breathed into the lungs.

MR. SCHOBERT: All right. I don't have any further questions thank you.

MR. FINELLI: We'll read and sign.

(Signature not waived.)

- - -

And, thereupon, the deposition was concluded at approximately 4:40 p.m.

- - -

State of Ohio :
SS:
County of Franklin:

I, MARK H. LUQUETTE, M.D.,do hereby certify that I have read the foregoing transcript of my deposition given on November 14, 2002; that together with the correction page attached hereto noting changes in form or substance, if any, it is true and correct.

_____
MARK H. LUQUETTE, M.D.

I do hereby certify that the foregoing transcript of the deposition of MARK H. LUQUETTE, M.D. was submitted to the witness for reading and signing; that after he had stated to the undersigned Notary Public that he had read and examined his deposition, he signed the same in my presence on the _____ day of _____, 2002.

_____
Notary Public

My commission expires _____

- - -

CERTIFICATE

State of Ohio :
SS:
County of Franklin:

I, Susan M. Olson, Notary Public in and for the State of Ohio, duly commissioned and qualified, certify that the within named MARK H. LUQUETTE, M.D., was by me duly sworn to testify to the whole truth in the cause aforesaid; that the testimony was taken down by me in stenotypy in the presence of said witness, afterwards transcribed upon a computer; that the foregoing is a true and correct transcript of the testimony given by said witness taken at the time and place in the foregoing caption specified.

I certify that I am not a relative, employee, or attorney of any of the parties hereto, or of any attorney or counsel employed by the parties, or financially interested in the action.

IN WITNESS WHEREOF, I have set my hand and affixed my seal of office at Columbus, Ohio, on this 20th day of November, 2002.

SUSAN M. OLSON, Notary Public
in and for the State of Ohio
and Professional Reporter.

My Commission expires July 2, 2007.