In The Matter Of:

Miller v.
R.E. Sedwick, M.D., et al.

Robert R. Clancy, M.D.
April 6, 2001

Foster Court Reporting Service, Inc.
Serving the Legal Profession Since 1939
117 South 17th Street
1800 Architects Building
Philadelphia, PA 19103
(215) 567-2670

Original File RC040601.V1, 145 Pages
Min-U-Script® File ID: 1906230888

Word Index included with this Min-U-Script®

IN THE UNITED STATES DISTRICT COURT
FOR THE WESTERN DISTRICT OF VIRGINIA

CODY MILLER, an infant, :
by his parents and next :
friends, LEONARD and :
DONNA MILLER :
Plaintiffs, :
: C.A. No.
vs. : 5:00CV30060
R. E. SEDWICK, M.D., :
et al., :
Defendants. :

Philadelphia, PA, April 6, 2001

Pretrial Examination of
ROBERT R. CLANCY, M.D., held in the offices
of Kevin H. Wright & Associates, The
Widener Building, Suite 240, at 10:00 a.m.
on the above date before *Susan Marie*
Migatz, a Registered Professional Reporter,
an Approved Reporter of the U.S. District
Court, and a Notary Public of the
Commonwealth of Pennsylvania.

FOSTER
COURT REPORTING SERVICE, INC.
1800 Architects Building - 117 S. 17th St.
Philadelphia, PA 19103
(215) 567-2670

APPEARANCES:
GERALD R. WALSH, P.C.
BY: GERALD R. WALSH, ESQ.
(4020 University Drive, Suite 200
Fairfax, VA 22030-6802)
Attorneys for Plaintiffs
TIMBERLAKE, SMITH, THOMAS & MOSES, P.C.
BY: C. J. STEUART THOMAS, III, ESQ.
(The Virginia Building
25 North Central Avenue
Staunton, VA 22402-0108)
Attorneys for Defendant R. E. Sedwick,
M.D.
WHARTON, ALDHIZER & WEAVER, PLC
BY: CHARLES F. HILTON, ESQ.
(100 South Mason Street
Harrisonburg, VA 22801-7528)
Attorneys for Defendants Rockingham
Memorial Hospital; Carolyn Beckwith,
R.N.; and Stacey Lam, R.N.

INDEX OF REQUESTS

Page 3

...ROBERT R. CLANCY, M.D.,
after having been duly sworn, was examined
and testified as follows:
BY MR. WALSH:
Q: Sir, what is your full name and
your current professional address?
A: My name is Dr. Robert R. Clancy
and I am a senior physician at the
Children's Hospital of Philadelphia here in
Philadelphia.
Q: What is your specialty in the
practice of medicine, Doctor?
A: I'm sorry?
Q: Your specialty.
A: I'm a pediatric neurologist.
Q: The CV that has been provided in
this case by you is accurate, sir?
A: Yes, it is.
Q: Tell me what the Irish-American
Pediatric Society is, sir.
A: It's actually an interesting
little group that alternates their
professional meetings between Ireland and
the United States and it's a chance for the
Irish physicians from the two countries to

Page 4

meet. It's also nice to be able to go to
Ireland and have a tax writeoff, I suppose,
for the trip, because you can go there and
it's a medical meeting, it's kind of a
club in a way.
Q: Do you know if they need a
general counsel?
A: I would suspect that they do.
Q: Do you have any applications for
that position?
A: Well, you know, I could send you
one.
Q: Would you?
A: Sure.
Q: I would be delighted.
How do you define "birth
asphyxia" as you will be using it in your
opinions today?
A: In the general sense, birth
asphyxia would be an abnormal condition in
which there is an interruption of blood
supply and oxygen to the baby.
Q: And is that how you will be
defining the term if you use "birth
asphyxia" today?

Page 5

A: Yes.
Q: Prior to January 30, 1993, was
Cody Miller's brain normal?
A: That's the day of his birth?
Q: Yes.
A: All right. I believe it was,
and the reason is basically that the baby's
body had grown adequately, the child's head
circumference was 35 I believe at birth and
that's a normal value, that the mother had
reported that the infant was active inside
her and there was never any alarming
reduction in activity. There was not an
excessive amount of amniotic fluid. One of
the jobs of a healthy baby is to swallow
their own amniotic fluid and if they have a
neurologic problem that prevents them from
swallowing, the fluid builds up and you
have too much of that.
Q: What's that called?
A: Polyhydramnios. And then,
finally, the child did not have any source
of orthopedic problems at birth. If the
child had been inactive, there might have
been a contracture where the arm couldn't

Page

open up.
So I think that structurally
the baby's brain was normal in terms of no
birth defects, there was nothing
dysmorphic, and as best as I could tell,
the brain had been developing normally
through the course of the pregnancy.
Q: Have you found any reliable
indications of neurological abnormalities
that preceded the labor and delivery of
Cody Miller?
A: No.
Q: Would you describe for me as
best you can the principal mechanisms of
asphyxia in an infant?
A: I suppose one way of looking at
this is that the process of asphyxia has
many roads that lead to it, so there are
different ways that children can get into
that condition of being asphyxiated. There
are specific etiologies, like the uterus
ruptures or the cord prolapses or the
placenta tears away from the lining of the
uterus.
But setting aside like the

Page 7

tiny details of how it got to be that way, asphyxia itself has two general forms, two different kinds of asphyxia, and this is what I had included in my report.

The most common type I believe by far goes by the name of partial prolonged asphyxia, and the notion here is that whatever is happening to the child occurs in a gradual fashion and it's stretched out over time; and if there is enough reduction of blood supply over a long enough period of time, it will reach into that baby's body and affect the brain, and in that particular situation also affect other organs in a pretty striking way.

The clinical picture with this is it's not specific but it's stereotyped. You see the same clinical picture each time. So there are obviously neurologic signs. The child is unresponsive or comatose, lethargic. There are seizures. Typically the brain will have some kind of swelling so the sutures can be split open, the fontanel could be

Page 8

bulging. The CAT scans that are obtained after birth show a certain pattern. And assuming that the baby survives that type of asphyxia, when all is said and done, they do develop CP, but it's a specific type of CP, again, the most common type, which is spastic quadriparesis.

Oftentimes with those babies, because the whole brain has been injured, the brain becomes small over time. They have a secondary microcephaly or acquired microcephaly. And most of the time the children are mentally retarded as best as you can measure that in a handicapped child. So that's one kind or one flavor of acute asphyxia.

The second type goes by the term of abrupt total asphyxia, and it's much less common. Probably the best example would be like a cardiac arrest. So we're talking about a birth asphyxia here, but there's nothing to say that a newborn who is fine at birth and then maybe at a day of age has a cardiac arrest, this could produce the same clinical picture.

Page 9

Again, there are different pathways that lead to this. So the notion here is that it's not a gradual process. It's a precipitous process. It's a catastrophe. It's the bottom drops out on the heart rate or perfusion.

And because it's so brief, the amount of time that it takes to actually reach into the brain and do its damage is less, and the typical figures that are cited — I know people always refer to the Myers articles and so forth — but the notion there is that if you create a situation where there's this abrupt total asphyxia, the damage begins after about 10 or 11 minutes. So there's a brief grace period where if you restore the circulation, you can escape injury. But just, again, for the beginning of the process of damage, about 10 to 11 minutes.

After these children are born, again, a lot of the neurologic signs are similar. So, sure, they're hypotonic, they have seizures, coma, whatever. So in that very superficial sense they don't look

Page 10

that different from the first group.

But there are actually important differences that show up in terms of when you do image them, they do not have the brain edema. I'm not really sure why that is, but just as an observation, the one group has prominent edema, the second group does not.

If you can examine specific brain structures, it turns out that sort of the glass jaw for this particular type are the deep gray structures, like the thalamus and basal ganglia. And, again, when all is said and done and they survive and grow up, yes, they have CP, but they have a fairly uncommon type of CP called extrapyramidal. The extrapyramidal includes the children who have choreoathetosis and involuntary movements.

So those are the two big categories. Then, of course, there's always overlap with these in the sense that there might be a baby who has been chronically asphyxiated and then, say, at the end of labor the heart stops, so you

Page 11

had a chronic asphyxia and then on top of that is the acute total asphyxia, and that can result in a mixture of the two where both things overlap.

Q: Dr. Clancy, I thank you for that informative precis on the kinds of asphyxia. However, you may have misunderstood my question.

A: Okay.

Q: My question was: Tell me what the principal mechanisms of asphyxia are in an infant. By that I mean: What is it that causes those kinds of asphyxia, the pathways that take you to the end being partial prolonged asphyxia or acute total asphyxia?

A: Okay.

Q: Would you do that for me?

A: Sure. In the first type, the partial prolonged, the notion would be, again, something gradual over time. So I'll simply cite something like abruption of the placenta, as an example, as a mechanism that as the abruption grows, the amount of blood getting to the baby from

Page 12

the mother becomes progressively smaller and so that stretches out over time.

That's probably the most obvious, simple example. I think with —

Q: I don't want you to restrict yourself to the most obvious, simple example of the principal mechanisms of asphyxia in an infant, Doctor. I'd like a little more than just the obvious, simple examples, please.

A: The other situation might be if the mother herself had vascular disease and she had long-standing, say, hypertension during the pregnancy and the placenta had become damaged and was atrophied or calcified and so forth, so then in the course of labor there would be a protracted reduction in blood supply. I don't think I can sit here and list all of the different maternal pathologies that create placental pathologies, but as a mechanism placental disease would do it.

Cord prolapse can do either — or let's put it this way: There can be cord compression. That's not

Page 13

necessarily a cord prolapse, but anything that would, again, interfere with circulation to the baby.

So those would be the three principal things: disturbances of the cord, disturbances of the placenta, or the connection between the uterus and the placenta for the chronic case.

Q: Doctor, let's deal with the category of inadequate perfusion of the maternal aspect of the placenta as one of the mechanisms by which an infant is asphyxiated. All right?

A: Okay.

Q: Tell me what you know about inadequate perfusion of the maternal aspect of the placenta insofar as that particular process or mechanism would cause asphyxia in an infant.

A: I think probably the best example would be if the mother had chronic hypertension and so if the mother had chronic hypertension, through the course of her pregnancy herself had renal problems, liver problems, bone marrow problems, that

Page

typically is also reflected in the appearance of the blood vessels in the placenta, and as a result the placenta may be too small or the baby may be small because there's chronically a restriction of growth, and that would be one way that a child could have chronic asphyxia.

Q: Have you told me all the conditions involved in inadequate perfusion of the maternal aspect of the placenta that might be principal mechanisms of asphyxia in infants, Doctor?

A: As far as I can think of right now, yes.

Q: Would impaired maternal oxygenation be one of the principal mechanisms of asphyxia in an infant?

A: By your question you mean if the mother herself is hypoxic, impaired maternal oxygenation?

Q: Yes.

A: I suppose it could be if the mother had a respiratory disease or was hypoxic for some reason.

Q: Well, what reasons would there

Page 15

be, Doctor, that would cause impaired maternal oxygenation to be a principal mechanism of asphyxia in an infant?

A: I suppose if she had lung disease, you know, asthma or some other type of pulmonary condition, if the mother had heart disease and couldn't circulate her own blood, that could do it.

Q: Any more than what you've just said, Doctor?

A: Well, circulation is basically heart and lungs, so it's two big categories of heart disease, however many there are, and pulmonary disease.

Q: Okay. In the category of impaired fetal oxygenation, are there principal mechanisms of asphyxia that you put in that particular category, Doctor?

A: I'm not sure I understand your question.

Q: Do you recognize one of the categories of principal mechanisms of asphyxia in an infant to be impaired fetal oxygenation?

A: By definition, right. I mean,

Page 16

if the baby is going to be asphyxiated, there has to be some impairment of oxygen or circulation.

Q: What would be some of the subsets of impaired fetal oxygenation which would cause an infant to be asphyxiated?

A: Well, we're still getting back, though, to how the baby receives their circulation, so it's whatever pathological processes are going to disturb the placenta or the umbilical cord or more specifically the vein returning from the umbilicus to the baby.

Q: Well, that's what I'm asking you to tell me more specifically about, Doctor. What would be the subsets of disease processes or conditions that would fall under impaired fetal oxygenation that would be principal mechanisms of asphyxia in an infant?

A: Okay. So prolapse of the cord or some kind of a compression of the cord would embarrass the circulation between the placenta and the baby. And, again, if it's the mother side or the baby side, rupture

Page 17

of the uterus, the baby is now outside the uterus, the placenta is separated from the wall of the placenta, that would embarrass circulation to the baby. If the baby was septic in utero, then in a way the baby is in shock, so it's not so much that the placenta is not doing its job but the baby's circulation internally is collapsing because they're in shock. Or if there was heart disease within the baby, congenital heart disease, and maybe fetal arrhythmia based on congenital heart disease.

Q: Would there be anything else you can think of, Doctor, in that category?

A: Well, infections can produce spasms of blood vessels of the umbilical cord. But they also create direct toxic effects on the unborn baby as well.

Q: Anything else in that category, Doctor?

A: I can't think of anything.

Q: All right, Doctor.

MR. HILTON: Gerry, this article that was brought in to us just a minute ago is an article Dr. Clancy sent me

Page 18

yesterday.

THE WITNESS: Yesterday.

MR. WALSH: I thought this was Zimmerman. That's why I put it aside.

MR. HILTON: No. This is from Dr. Clancy in support of his opinions.

MR. WALSH: Thank you.

Q: (Continued) I will deal with that later, Doctor.

A: Sure.

Q: As soon as I complete my application for the Irish-American Pediatric Society.

A: I'll sign the card myself.

Q: Excellent.

MR. HILTON: Are you Irish?

MR. WALSH: I have been told I am.

Off the record.

(Discussion off the record.)

BY MR. WALSH:

Q: Doctor, how many episodes, *separate, distinct episodes, of bradycardia* occurred with regard to Cody Miller before

Page 19

the severe terminal episode at 0219 on the 30th of January that you refer to?
A: Let me just get my notes out so I don't say the wrong thing.
Q: Surely. And let me tell you, Doctor, that when this is all over, if you would be so kind as to make copies of your notes and provide them to whichever counsel you want to and they can send them to me.
A: I would be happy to.
MR. HILTON: While he is looking through that, Gerry, just as a point of clarification, it looks like the dates on this cover sheet on this fax from Dr. Clancy are April 4, but I think something must be off. I first saw this yesterday morning when I walked into my office. I don't know if the fax was off or somebody delayed sending that or whatever. But I didn't hold it on you from February 4 until today.
THE WITNESS: February 4?
MR. HILTON: Excuse me; April 4.
MR. WALSH: We will deal with that in due time.
A: (Continued) All right. I have three episodes of bradycardia prior to the terminal episode of bradycardia.
Q: What caused each of those discrete episodes, Doctor?
A: Oh, I don't know. What caused the bradycardia?
Q: What caused each of the three discrete episodes of bradycardia you just mentioned to me?
A: I am not sure what caused them.
Q: When did they commence, according to your understanding of the facts?
A: Okay. I have panel numbers written here, so let's do it maybe that way.
Q: Go ahead, sir.
A: The first is Panel 90356, and I think this was right after the mother had been given the epidural. She herself had low blood pressure after that, and then there is this period of bradycardia. And I think this was a couple minutes long, but let me just look.
Well, I think this was like two to three minutes.
Q: In length?
A: In duration; yes.
Q: Duration. Length and duration would be the same?
A: Yes.
Q: All right. Would you go to the second episode of bradycardia that you told me about?
A: The next I have is Panel 90382, and actually bear with me, I want to look at the nurses notes, because this is commented on in the nurses notes, too.
THE WITNESS: If you know where that's at, you might save us some time.
MR. HILTON: Okay.
The nurses notes begin on Page 50024 if yours are in order. What time are you looking for?
THE WITNESS: 1:20-ish.
MR. THOMAS: 50034.

Page 21

BY MR. WALSH:
Q: If you would be so kind, Dr. Clancy, as to simply tell me what you're looking at when you're going through the process of answering the question, please.
A: I am looking at two source documents. One is the fetal heart rate tracing itself and the other are the nurses notes in the labor and delivery area that are giving text describing events.
Okay. So this is at 1:30, and that time is actually marked on the strips, and there's also a note from the nurses that's literally dated 0130. The nurses note says: Fetal heart rate began to decelerate below baseline. Bottomed out at 45 and very gradually began to return to baseline. Entirety of deceleration lasted seven to eight minutes. Okay? So that's the text from the nurses chart.
I'm now looking at Panel 90382. To the left of this moment the fetal heart rate baseline has been fluctuating 150 plus or minus some and

Page 23

indeed as the nurse says, it drops down, I can't tell if it's literally 45, but that looks like a good reading, and it's back up again to around 120, actually, just in a couple minutes. So let's see here. Maybe three to four minutes to get back to 120. I know the nurse says seven to eight, but I guess she means completely and totally stable to baseline.

Q: Stay where you are with those papers, Doctor —

A: Yes.

Q: — because I am going to ask you some questions now rather than have you put them back and pull them out again. All right?

A: Sure.

Q: When you used the term "episodes of bradycardia" in your opinion letter or report, how were you defining "bradycardia" when you used it?

A: Slow heart rate.

Q: Well, what's a slow heart rate, Doctor?

A: Do you want to know what the

Page 24

cutoff is?

Q: I want to know what your criteria are when you use that term, because I have no way of understanding what you mean by "bradycardia" when you say there were several episodes of bradycardia. So you need to tell me exactly what you meant, Doctor.

A: Okay. So the first definition would simply be reduced from baseline, slower than usual for this baby. So, for example, the absolute number, even 90 would be slow for a baby but would be fast for me. So it's not simply a number that fits everybody. So the first notion is that it's a reduction from baseline.

Q: Can I stop you there —

A: Yes.

Q: — while we stay on these points?

The first concept of bradycardia would be a reduction from the normal baseline of the baby; correct?

A: Yes.

Q: How much of a reduction from the

Page 25

normal baseline of the baby does it have to be before you would characterize it as bradycardia, Doctor, in any instance where you are dealing with the baseline interpretation of it?

A: Probably below 90 beats per minute would get my attention.

Q: So are you telling me that with regard to determining bradycardia, as a definition, when related to the fetal heart rate below the normal baseline of the baby, if it were below 90, that would be bradycardia?

A: Yes.

Q: All right. Now, are there any other circumstances or categories of interpreting bradycardia that you would be using other than that one?

A: I use a heart rate of 60 not just as bradycardia but something that worries me as a neurologist. Okay? Part of that has nothing to do with obstetricians and newborn babies but, rather, the personal experience I have in the cardiac unit for the newborns.

Page 26

So, for example, in a cardiac unit with newborn infants where they have heart defects, it's not that uncommon to see bradycardia and so forth. When there is a sustained heart rate below 60, they know and I know that that can open the door to trouble for the babies.

So having a heart rate of 90, for example, yes, that's bradycardia; but all things being equal, that shouldn't hurt you. When you fall below 60, then there is the possibility of low blood pressure and so forth, because the actual flowing of the blood through the baby, what's called cardiac output, depends directly on the heart rate in a newborn. For you and I it's different. We can change how much blood leaves the heart with each pump of the heart. But newborns do not. They have a fixed volume per contraction. So if you drop their heart rate, you are reducing the amount of water flowing through the pump, if you will, or blood flowing through the pump.

Q: Do you have any other categories

Page 27

of bradycardia other than when it's measured as a drop below the baseline to 90 or in the neonatal environment, particularly with cardiac patients in the neonatal area, 60? Do you have any other bradycardic definitions?

A: No. I realize that for the obstetricians they classify the different bradycardias in relationship to the temporal events with contractions, so is it after the contraction or simultaneous or if it's variably related, but I am not an expert in that and I am not going to opine about whether this is a late decel or a variable decel.

Q: Do you profess in public or professional settings to be an expert in the interpretation of electronic fetal monitor tracings, Doctor?

A: Not directly. I don't do it contemporary to any mother giving birth. No one ever wants a neurologist to know about it. It is relevant to my understanding of the baby simply because it's held out to be a sign of neurologic

Page 28

well-being. If there is variability, the heart rate is under the influence of the brain. So if the brain is basically dead, there's nothing to tell the heart to beat faster or slower. So you are pretty much stuck with the fixed heart rate. So I am aware of what the issues are. But, again, I'm not at the bedside reading these things for deliveries and contemporary experiences.

Q: Have you ever testified in your many court appearances as an expert in the reading and interpretation of electronic fetal monitor heart tracings?

A: I actually do, but I always couch it that I can read numbers off a heart tracing and I know where the critical values are. I am not going to slug it out with an obstetrician about is that a Type 1 or a Type 2 dip and things like that. But if it's something like a profound bradycardia, I mean, I can read and I know where the critical values are. So I'm kind of saying yes and no at the same time.

Q: I understand.

Page 29

Let's stay with the definition that I expect you will tell me is most applicable in this case and that's the fall of the fetal heart rate from the normal baseline to below 90 during the labor process.

A: Okay.

Q: That's what you're using in defining "bradycardia," aren't you, Doctor?

A: Yes.

Q: All right. You are not using the neonatal cardiac patient below 60 as bradycardia, are you?

A: No. I mean, they're both bradycardia.

Q: I know they are. But are you using that parameter in your opinion letter?

A: No. I mean, I guess what I'm trying to say in the opinion letter is that there were periods before the terminal bradycardia where it was bradycardia, definitely bradycardia, and we talked about one of them a minute ago, we are talking about the middle one right now, and that

Page 30

these were bradycardia that were below 90. In fact, they went down to 45, which gets my attention for sure. But then they were relatively brief and they recuperated.

Q: All right. With regard to the definition of "bradycardia" when related to a drop in the fetal heart rate to 90 from a higher normal baseline, how long does the fetal heart rate have to continue below 90 to be bradycardic in your definition?

A: Oh, I've never thought of it that way. In other words, how long is it to be below 90 before you say you qualify for bradycardia?

Q: Yes, Doctor.

A: I don't know that there's a definition. I mean, I don't have a definition for that.

Q: Okay. Would it be your understanding insofar as the definition of "bradycardia" in a fetus during labor that if there was a normal baseline in the 140's and the heart rate dipped below 90 even momentarily, that that would be a bradycardic event in that fetus' labor?

Page 31

A: I suppose, yes. I mean, like I say, if it goes below 90, at that moment it's bradycardic. Whether or not it's important is obviously a different question.
Q: I am only asking that you work with me so I know what you're talking about, Doctor.
A: Okay.
Q: You are light-years ahead of me in this area.
A: I don't think so.
Q: We'll see.
In the definition of "bradycardia," when you use it, do you make it more descriptive than simply bradycardic? In other words, do you describe it in more detail, such as mild, moderate, severe, whatever other words you might use, if you do use those words?
A: I don't have any classification system or scheme for that. I suppose the two parameters that you would be interested in would be the duration in time and the magnitude. But I don't have any criteria

Page 32

for if it's below such-and-such for this period of time, that's mild. I don't have that.
Q: I need you to look at the fetal monitor tracing which you have before you for the 0130 event of bradycardia which we have been talking about and tell me as best you can how long the fetal heart rate stayed below 90 from the time it went below 90 until it appeared above 100.
A: Okay. Let me find my pen.
There it is. Thank you.
Well, let me give you a couple answers. Maybe I should just show you so we are —
Q: No, don't show me anything right now.
A: You don't want to see anything?
Q: I just want you to answer verbally on the record. Thank you for suggesting that, though.
A: Okay.
Q: What I want is an answer to that question in minutes if you are able to do that for me.

Page 33

A: I am just scribbling on the chart here so I can give you a good answer.
Q: Not my chart so I don't care, Doctor.
A: Okay. So let me state your question back to be sure I heard it right. The period of time that the child was below 90, the duration of time below 90, and recovery to 100?
Q: Yes.
A: Okay. Slightly under five minutes.
Q: Now, for the same event of bradycardia at 0130 that you categorize or describe as the second event, tell me what the normal baseline of the fetal heart was at the point in time before it began to drop and went to 90.
A: Okay. Basically I'd say it was 150, let's say 140 to 150. It was a pretty decent baseline. The heart rate slightly goes up before it starts to fall to 90.
Q: What does that mean to you, when the heart rate slightly goes up before it starts to fall to 90, Doctor?

Page 34

A: Sometimes acceleration can be a compensation, again, to try to increase circulation.
Q: Is that what you're telling me it means to you?
A: It could.
Q: Is that a guess or do you want to say that that's what you believe it is?
A: Well, I'll back off of saying that's what it is. But there is some acceleration to like 160-170 before it starts to fall to 90.
Q: I want you to look at the fetal monitor tracing that you have before you regarding the second bradycardic event at 0130 and tell me what you contend is the normal baseline at that point in time where it goes down below 90 as you've used that concept in your definition, sir.
A: So, again, the baseline before the bradycardia to 90 was 140 to 150.
Q: All right. And when it got to 90, Doctor, on the fetal monitor tracing, having dropped from the normal baseline of 140 to 150, how long did it remain below 90

Page 35

before it returned to the normal baseline of 140 to 150, if you can give me that answer in minutes or seconds or both?
A: Okay. Six and a half to six and three-quarters minutes, just shy of seven minutes. And that's getting back to the 140 to 150 baseline.
Q: And that's using your definition of "bradycardia," isn't it, Dr. Clancy?
A: Well, no. My definition of bradycardia was under 90 and what you have not asked me is how long did it take to go from below 90 back to 90. You asked me to go back to 100 first —
Q: All right.
A: — and then you asked me to go up to 140 to 150.
Q: All right. So your definition of "bradycardia" is below 90 from the normal baseline; correct?
A: Yes.
Q: All right. Let's then take the next step. I thank you for pointing that out to me. In this bradycardic event at 0130 tell me how long you calculate the

Page 36

fetal heart rate was below 90 after reaching 90 from the normal baseline of 140 to 150 and until it again reached 90, which would be your definition of "bradycardia," wouldn't it, Doctor?
A: Yes.
Q: All right.
A: Four minutes. There were four minutes in which the heart rate was below 90, either on the way down or the way back.
Q: In accord with your definition of "bradycardia" that you have already shared with me; right?
A: Yes.
Q: Now, Doctor, would you as a pediatric neurologist consider that that was a prolonged period of bradycardia in this particular instance?
A: Not really. I mean, below 90 for four minutes should be tolerable to a baby in the womb or in the cardiac unit.
Q: Well, I am talking about in the womb.
A: Right. But it's the same

Page 37

babies. So I'm just saying that if we were standing at the bedside where you could see a baby like this —
Q: Doctor, I need you to focus with me on a fetus in the womb. I am not here on a cardiac neonate in the ICU or the NICU. So if you would focus with me on the fetus in the womb, that would help us move along.
A: Well, my experience as someone who can actually see these children in the real world makes me a valuable person to be sitting here and give these kinds of depositions, because no one is inside this womb with this baby to know how —
Q: Doctor, I don't have the slightest intention to demean, disparage, or insult you, sir.
A: No, no.
Q: But I am here on a purpose in a particular case and all I ask you is to answer this question for me so we are on the same wavelength.
In this case where you have identified a four-minute period of

Page

bradycardia using your own definition, do you consider that to be a prolonged period of bradycardia in this particular setting?
A: No.
Q: Why do you not consider it a prolonged period of bradycardia in this particular setting?
A: From my perspective, prolonged would have to be, again, long enough to damage I guess is maybe what we're at odds about here, and this would not be long enough to damage.
Q: Doctor, if there was a prolonged period of bradycardia using your definition, would a fetus undergoing a prolonged period of bradycardia suffer severe fetal hypoxia?
A: I'm sorry; ask the question again, please.
MR. WALSH: Read it back to Dr. Clancy, ma'am.
(The court reporter read the pending question.)
A: (Continued) Well, that's a complicated question. Would he have severe

Page 39

asphyxia? Maybe not.
Q: Severe fetal hypoxia is the
exact terminology of my question, Doctor.
A: Right. Then I'm sorry; would
you please read it again?
MR. WALSH: Read it back.
A: (Continued) It's actually a
complicated question.
Q: You think it is, Doctor?
A: Yes, very much so.
Q: All right. It will be read back
to you, sir.
(The court reporter read the
record as follows:
"QUESTION: Doctor, if
there was a prolonged period of bradycardia
using your definition, would a fetus
undergoing a prolonged period of
bradycardia suffer severe fetal hypoxia?")
A: Not necessarily, no, not under
90 as the definition.
Q: What would you consider to be a
prolonged period of bradycardia using your
definition, Dr. Clancy?
A: Again, my definition would be

Page 40

bradycardia 60 or below for 10 or 11
minutes or more. And, again, I'm looking
at this as the neurologist looking at
injury as opposed to simply stress.
Q: All right. Doctor, I thought
that we had established that when you were
using "bradycardia" in your statement in
your report that there were earlier
episodes of bradycardia, that that
"bradycardia" was being defined by you to
be a fetal heart rate that goes below 90
from the normal baseline and then returns
to the normal baseline. Is that correct?
A: Yes, the definition of
"bradycardia" for me is under 90 beats per
minute.
Q: Was I correct in stating what I
just stated to you, Doctor, in that last
question?
MR. HILTON: Can you reread
the question for me?
MR. WALSH: Go right ahead.
(The court reporter read the
preceding question.)
MR. HILTON: Thank you.

Page 41

A: Or back above 90. Again,
bradycardia is below 90. All right? So
you start with the normal baseline. The
heart rate falls. When it goes below 90,
there's a label for that. It's called slow
heart rate, bradycardia. When it goes back
up above 90 again, it's back into the
normal range, so he's no longer
bradycardic. It may take another couple
minutes to get back to the baseline,
though.
Q: All right. So the definition of
"bradycardia" you are using in your report
is the fetal heart rate at the normal
baseline for the fetus drops below 90 and
at the point that it's below 90, that's a
bradycardic heart rate?
A: Yes.
Q: And the bradycardia continues
until the heart rate rises above 90?
A: Yes, I agree with that.
Q: All right. Now, using that
definition, Doctor, which I want you to
use, tell me in the 0130 episode of
bradycardia how long the heart rate was

Page 42

below 90 and continued below 90 until it
rose above 90.
A: Four minutes.
Q: Can you do that for me?
A: We did that already. That was
four minutes.
Q: I don't mean to prolong this
deposition with unreasonable questions,
Doctor.
A: I understand.
Q: I need to understand what we're
talking about.
A: That's what I pointed out, that
below 90 to hitting 90 again was four
minutes.
Q: I thought that's what you said,
but I'm just a little country lawyer up
here trying to get educated, sir, so help
me out.
Now, the next question is:
Isn't that the definition you were using in
your report?
A: Yes. These are periods of
bradycardia.
Q: Okay. Now, if we take it as a

Page 43

given that at 0130 there is a four-minute bradycardia according to your definition and your calculation, you don't consider that to be prolonged?
A: No, I do not.
Q: What would you consider to be prolonged bradycardia according to your definition in the fetus in utero situation, Doctor?
A: Okay. Again, 90 is bradycardia by definition. Okay? From my point of view as a neurologist, not just for definitions but looking at injury, it has to be bradycardia to the tune of 60 or below for 10 or 11 minutes or more. That to me is injurious bradycardia.
Q: So can we use as we go forward in this deposition your characterization of injurious bradycardia as being 60 or below?
A: Yes.
Q: And bradycardia without any adornment, the definition we have been struggling with, below 90?
A: Below 90, yes.
Q: Taking the definition of

Page 44

injurious bradycardia, how long would the bradycardia below 60 have to be before you would describe it or characterize it as prolonged injurious bradycardia?
A: 10 or 11 minutes.
Q: Where do you come up with this 10- or 11-minute time parameter, Doctor?
A: This is actually a figure that is part of our general neurological education within neonatal neurology. It's part of textbooks. It's certainly in Fenichel's book and Volpe's book. And it originally all stems from the Myers monkey studies: that for severe bradycardia, that the onset of damage begins 10 to 11 minutes after the cord is clamped or whatever.
Q: Is the severe bradycardia that you just mentioned relating it to the Myers study defined as below 60 in the Myers study?
A: No. That's not a heart rate study; that's a cord study.
Q: Okay. Well, tell me what heart rate study in the medical literature it is that you are relying on or referring to

Page 45

when you say that 10 or 11 minutes of injurious bradycardia is prolonged.
A: I guess the full answer to your question is from the Myers studies it says if you have block blood supply through the cord for 10 or 11 minutes, damage starts. From clinical studies we know that when you drop the heart rate below 60, it is like having your cord clamped. It's the closest thing, because no one is walking around clamping cords on babies. I believe that the paper that I furnished you with also uses that as a value. If I can put my hands on it, I will show you.
Q: Pasternak?
A: Actually, I don't remember which one. I'll look there first.
Where's my paper?
MR. HILTON: This one?
THE WITNESS: No.
Q: Phalen?
A: Yes. Let me see this one.
Yes, this is the Phalen paper.
Q: Mark that copy with the yellow

Page

Hi-Liter where you find that reference, Doctor, that you indicated was the basis for your thinking in this area.
A: Okay.
Q: Doctor, you have been kind enough to mark on Page 20 of the Phalen article in the Journal of Maternal/Fetal Medicine, 7:19-22 (1998): "an acute prolonged fetal heart rate deceleration of approximately 60 beats per minute from a normal baseline fetal heart rate"; correct, sir?
A: Yes.
Q: Now, would you be so kind, Doctor, as to continue to look at the Phalen study —
A: I've found my copy.
Q: Oh, you have? All right, sir — and take a moment and locate where in Phalen it sets forth the 10- to 11-minute time parameter that you have mentioned.
A: It does not say that.
Q: What does it say with regard to the time parameter for the acute prolonged fetal heart rate deceleration of

Page 47

approximately 60 beats per minute from a normal baseline fetal heart rate that you're referring to?

A: Right. There's actually two descriptions of the time. It's all on the same page, Page 20. In Table 1 they are simply listing how these particular babies in the study had their problems, and here it's loosely defined as a prolonged fetal heart rate deceleration alone. And I took that to mean in the absence of other events like uterine rupture.

And on the same page under the "Results" section in the first paragraph, last line, it says: "The mean duration of the fetal heart rate deceleration was 32.1 plus or minus 9.1 minutes with a range of between 19 minutes and 51." And as far as I can remember, that's the only two time descriptors in here.

Q: Let's move on to the third episode of bradycardia, Doctor, and if you can tell me when that was, please.

A: Sure.

Page 48

MR. HILTON: Can I ask for a point of clarification? I thought we only talked about one episode of bradycardia at 0130.

MR. THOMAS: Two so far.

THE WITNESS: We did the first one and this is the second one.

MR. WALSH: The first one was Panel 90356 that he referred to.

MR. HILTON: Thank you.

A: (Continued) Okay, I am looking at Panel 90392 from the fetal heart rate records.

Q: How long was the fetal heart rate in that instance below 90 according to your definition, Doctor?

A: Well, I don't know partly because I think the paper was changed here and so the graph is actually missing in this period of time.

Q: How long was the fetal heart rate bradycardic according to your definition at that point in time using only the fetal heart tracing paper that you do have access to? Do you understand that question, Doctor?

Page 49

A: Right, I mean, actually, at no time is the child bradycardic at that point simply by the chart because there is no pen deflection here at this point.

Q: All right. Then what's the basis for your belief that there was a bradycardic event at that point in time, at or about Panel 90392?

A: Because written in text is: Audible deceleration to 40 to 50.

Q: Does it say anything about duration, Doctor?

A: No.

Q: Have you formed any belief as to the duration of that bradycardic event which, according to your definition, was injurious bradycardia?

A: Well, again, there is no information present. The total duration of the gap would be about two minutes. I think when the pen stopped writing, it looks like it was two minutes after 12:00, and then the graph comes back on line just before 2:04.

Page 50

MR. HILTON: Two minutes after 2:00?

THE WITNESS: Two minutes after —

MR. HILTON: You said two minutes after 12:00.

THE WITNESS: I'm sorry; I confused myself with the line there. It's 2:02, two minutes after 2:00 p.m.

MR. HILTON: A.m.

BY MR. WALSH:

Q: Doctor, it's not p.m.

A: I'm sorry; I'm getting myself confused.

Q: That's all right. I don't want you to be confused, Doctor.

A: All right. At 2:02 the graph falls apart and is restored by around 2:04. So whatever that was, and I don't know how low it was except by what's written in, it was a two-minute time period.

Q: What is the fetal heart rate at when the fetal heart monitor tracing is restored, Doctor?

Page 51

A: It's basically 70 for a few moments and then it's 90 and above as soon as the graph comes back on.
Q: I am going to have to ask you to turn back to the 0130 time period, Doctor. I didn't ask you a question I should have.
A: Sure.
Q: When you find that, tell me.
A: I have it.
Q: Was there any period of injurious bradycardia according to your definition of fetal heart rate below 60 in that particular event of bradycardia?
A: Yes, there was.
Q: Tell me the amount of time that is in the injurious bradycardia or below 60 parameter, sir.
A: Okay. About two and a half minutes.
Q: Would you describe for me, Dr. Clancy, the effect on the fetus during that two and a half minutes of injurious bradycardia in terms of effect on its status of well-being?
A: Okay. It certainly is a stress

Page 52

on the baby. But, again, the purpose of defining it over time is that this should not leave any lasting mark on the child. That's the whole notion of the model, is that you have to be exposed to that for a longer time, 10 or 11 minutes, before the damage begins. So this two-minute period would not damage the child.
Q: Well, if you would, Dr. Clancy, we know that there was a period of bradycardia according to your definition at that point in time and there was a two-and-a-half-minute period of injurious bradycardia according to your definition; correct?
A: Yes.
Q: I want you to take the totality of the bradycardic event, including the simple bradycardia as well as the injurious bradycardia, if I can simply characterize it that way for the purpose of this question, and describe for me your understanding as a pediatric neurologist of how the fetus was being affected in that period. What was happening to it, to its

Page 53

blood flow, to its ability to carry on its functions, any chemical insults or reactions? Can you describe for me what was going on in that time frame, sir?
A: Well, there's obviously a host of responses that the baby would have and they would be basically physiological and biochemical.
The physiological responses would be turning on the so-called protective reflexes of the infant, and that has to do with redirection of the circulation to protect the brain and the heart as sort of the quarterback of the body, so to speak.
And there would be biochemical changes of a simple nature. One would be that the bloodstream would be more acidic, at least at that moment. And there would probably be compensation, the buffers, the acid-based buffers, in the body. There would probably be some release of stress hormones: cortisol, adrenalin.
Assuming that the threshold for damage has not been breached, which is,

Page

again, below 60 for more than 10 to 11 minutes, those changes would remain as biochemical changes and physiologic changes but would not be translated into actual tissue damage.
Q: Would any of those changes you have just described continue after the period of injurious bradycardia had passed?
A: This is not like a light switch turning on and off. So the moment that the heart rate goes above 60, there certainly is a recuperative process to restore the balance again to the child.
Q: Would you describe that for me, please, the recuperative process to restore the balance to the child?
A: It would be the same thing like if you finished running a race and you stopped: Your heart is still beating, and over a few minutes your heart rate will come back down to a normal value. So it's really just the reverse of the process that got the child into that situation in the first place.
Q: Well, what happens to the

Page 55

chemical imbalance, any particular component of chemical imbalance; how long does that take to go back into its complete balance state that it was in before it became deranged?

A: Generally it would be a few minutes. I mean, obviously you have to look at each individual one. You know, cortisol itself has a half-life in the body of maybe several hours. So once the molecule has been released, it's going to be in your circulation for a while. In terms of new release of cortisol, that's shut off right away. But, I mean, these are the adaptive mechanisms we all have to react to stress, meaning physical stress, and health issues.

Q: Now, was there a period of injurious bradycardia at the last time that we talked about?

A: Again, hard to say because of the pen's not writing. By the text —

MR. HILTON: We are going back to the paper change now, Gerry?

MR. WALSH: Oh, yes; I'm

Page 56

sorry.

Q: Doctor, if I don't make it clear, you let me know. I thought you were on track here. But I am on the time around 2:00 a.m., and I'll frame this question again so we're exact, Doctor.

At the bradycardic event or episode of around 2:00 a.m. or shortly after 2:00 a.m., if you assume that the heart rate is in the 40's to 50's for a couple of minutes, would you please describe the effect of that period of injurious bradycardia on the fetus for me?

A: It's the same discussion. So I'm going to take it that by the nurse's observation the heart rate fell to the 40's or 50's, which is below the level that's potentially injurious; and it could have lasted up to two minutes, and it would have been the same idea that the body responds by sending out its stress reactions and adaptive reactions; and then if the stress is removed from the system, equilibrium is achieved again.

Q: All right. Is the nature/the

Page 57

type of stress that the fetus undergoes in periods of injurious bradycardia the same no matter what the etiology of the bradycardia is, Doctor?

A: Well, yes and no.

Q: Explain that, please.

A: Yes. In terms of what's happening for either of these cases, the partial prolonged or the acute sort of thing, it's all circulatory. It's all hypoxic ischemia. What's different about it is where the attack is on the brain itself. And, again, I don't think it's understood exactly why one model produces the edema and the seizures and the other model produces the basal ganglia injury.

MR. WALSH: Mark that, please.

A: (Continued) Again, the nature of the insult is the same, but the location is different depending on the model.

Q: Now, Doctor, I'm a little confused, and let me tell you what my confusion is in your answer.

A: Okay.

Page 58

Q: You've mentioned partial prolonged asphyxia and acute total asphyxia in your answer and my question was more directed to the mechanisms of the bradycardia, what caused the bradycardia, and would the bradycardia being caused by interruption of the umbilical circulation or altered placental gas exchange or inadequate perfusion of the maternal aspect of the placenta or impaired maternal oxygenation or impaired fetal oxygenation impact the fetus in that period of injurious bradycardia differently depending upon what the stimulus was that caused the injurious bradycardia. Do you follow me?

A: No.

Q: You don't?

A: I really don't.

Q: All right. Is it your position that —

A: I think I'm being dense today, but go ahead.

Q: Life is a learning experience, Doctor.

A: Okay.

Page 59

Q: Is it your position that injurious bradycardia as you define it is a heart rate below 60 caused by something that brings about that low heart rate?

A: Well, let's be concrete rather than talk in generalities. Okay?

Q: Yes.

A: Let's say it's the cord has a spasm for whatever reason and because the cord goes into spasm, the baby's heart rate drops below 60. Whether it's the cord that goes into the spasm or the cord that prolapses or there is a uterine rupture, I don't think that matters as much if the end result is all of a sudden the baby's heart rate goes below 60 for 10 or 11 minutes. I don't think it cares what led up to that.

Q: You said I don't think it matters as much. Are you prepared to say it doesn't matter at all, Dr. Clancy?

A: I will as long as I understand, is that the question you were asking me?

Q: Yes.

A: So, again, just to understand it, there are different ways that can lead

Page 60

to this acute total asphyxia. In my opinion the end result only matters on how low the heart rate is and how long it was. It doesn't matter to me whether it's cord spasm, whatever.

Q: Any of the mechanisms —

A: Any of the mechanisms.

Q: — would not change your view as to there being different stressors or effects on the fetus simply because of the source of what caused the injurious bradycardia?

A: Right. And as an example, in the paper we just mentioned —

Q: Phalen?

A: Yes, Phalen's paper — they give a list of all these children had the same kinds of scans, the same kinds of clinical picture, and yet one was a cord prolapse, one was a severe bradycardia, one was a uterine rupture, but they all ended up looking the same in this clinical syndrome.

Q: Doctor, are you telling me that the bradycardic episode at approximately 2:00 a.m., shortly after 2:00 a.m., has

Page 61

anything to do with the partial prolonged asphyxia in this case?

A: I'm sorry; just ask it one more time.

Q: Sure. In fact, I'm going to rephrase it a little to make it more artful —

A: Okay.

Q: — if I am capable of that. Are you saying that the injurious bradycardic episode that occurred in the period just after 2:00 a.m. on January 30 is part of or actually is an indication of partial prolonged asphyxia occurring?

MR. HILTON: Object to the form of the question.

MR. WALSH: What is the objection to the form, Frank?

MR. HILTON: It seems to imply that Dr. Clancy has testified here today that he believes that injury occurred at the time of the bradycardic episode shortly after 2:00 a.m. and I don't think that's his testimony.

Page

MR. WALSH: Okay.

Q: (Continued) Doctor, I know you have been through this a lot of times and I am trying to do it as civilly as I can, and my learned colleague has a right to object when he does, but when he objects to an objection to form, I can either ignore him or ask him what the objection to form is and then I can decide whether I want to change the form of the question. In this case, I don't think his objection is very good, so I am going to ask you to answer the question that I am going to have the court reporter read back to you.

A: Okay.

(The court reporter read the record as follows:

"QUESTION: Are you saying that the injurious bradycardic episode that occurred in the period just after 2:00 a.m. on January 30 is part of or actually is an indication of partial prolonged asphyxia occurring?")

A: (Continued) Okay. Two parts to the answer. One is, no, I don't consider

Page 63

this to be partial prolonged asphyxia. And the second thing is I guess there's a slight distinction between an episode of injurious bradycardia and an injurious episode of bradycardia. When I said 60, I really probably should have said that's potentially injurious. If it's below 60 long enough, that has the potential to hurt you. But it's not that when you go below 60, that the clock starts ticking for injury, because I don't believe that.

Q: Now, with regard to the three episodes of bradycardia which you refer to in your report as happening before the severe terminal bradycardia at approximately 2:19, were those episodes arrests of uterine blood flow or arrests of umbilical blood flow?

A: Well, neither. I mean, these are the baby's cardiac rate. Okay? I mean, you take it at face value, this is what the heart rate is. Now, whether that's a reflection of something going on in the umbilicus or in the cord itself, I can't tell you. All I can tell you is that

Page 64

the heart rate fell.

Q: I am going to reframe that question, Doctor, because your answer indicates to me that I may not have made it clear.

Are these bradycardic episodes that we have been talking about, the three that you've testified about, the result of uterine blood flow being compromised or interrupted or are they the result of umbilical blood flow being compromised or interrupted?

A: I mean, I still can't distinguish between which of those because it's really ultimately one system that delivers — I mean, the placenta gives to the cord gives to the baby. So all we are looking at is the baby. So I can't tell by that.

Q: Well, the mechanics are different, aren't they, Doctor, if it's interruption of uterine blood flow or interruption of umbilical blood flow?

A: Right. But you did ask me before do I know why these bradycardias

Page 65

occurred and I said, no, I really don't know.

Q: Doctor, this is not a question. I am asking you it in a different form because oftentimes I find that in this process different questions will help to define more information, so bear with me.

You just don't know and aren't able to tell me?

A: No, I don't, I truly don't know.

Q: But you do know what caused the 0219 severe bradycardic event?

A: I have an opinion about that one, yes.

Q: We will get to that.

A: Okay.

Q: Tell me, Doctor, your understanding of the ways in which uterine blood flow can be reduced.

A: Okay. First of all, the mother has to supply the blood to the uterus. So anything, again, that's going to disturb the mother, like hypotension in the mother, will reduce supply to the placenta, and all

Page 66

the things that give rise to maternal hypotension.

Q: So hypotension as a general category?

A: Yes. Of course, that would get into anything, into just low blood pressure versus low volume or chronic disease in the mother so her blood vessels aren't nourishing the placenta.

The second would be I guess the placental abnormalities itself, the placenta is too small or if it has abnormal blood vessels within the placenta.

And third would be the cord.

Q: Well, if something was wrong with the cord, wouldn't that be an umbilical blood flow reduction rather than a uterine blood flow reduction?

A: Yes, that would be a reduction, right.

Q: Now, Doctor, I want to ask you specifically: Can you tell me what mechanical events with regard to perfusion of the uterus would take place that would reduce or stop blood flow to the uterus?

Page 67

A: I guess the most common thing is just each contraction increases the pressure within the abdominal cavity and so there is reduction of blood flow to all babies during each contraction and that's physiological reduction.
Q: Well, are you suggesting that that can be defined as a mechanical reduction of blood flow to the uterus?
A: In the sense that it's a pressure/physical relationship as opposed to something that's inflamed or separated.
Q: How is blood supply to the uterus provided?
A: From the mother to — oh, well, to the uterus? It's from the aorta, from the aorta, to the uterus; and then I believe it's the uterine arteries that attach the placenta to the walls of the uterus.
Q: Well, are there any other ways in which there is blood flow between the uterus and the vascularization of the mother other than what you've just told me?
A: Well, ultimately it's going to

Page 68

be the mother's aorta that's going to supply the arteries that will take her blood to the placenta. I can't name any more detailed arteries for you.
Q: What is the general nature of the blood perfusion process for the uterus, Doctor? I mean, how does it take place is my question.
A: How does what take place?
Q: The perfusion of the uterus take place.
A: The same as any other organs are perfused. The maternal blood is under pressure and the pressure moves the blood through the tissues, whether it be her kidney or her limbs or the placenta.
MR. HILTON: Gerry, can we go off the record for just one second?
(Discussion off the record.)
(Short recess.)
BY MR. WALSH:
Q: Doctor, have you an awareness from your review of this case if there was an umbilical cord blood gas taken and tested?

Page 69

A: Oh, let me look.
Q: Would you?
A: I have a little table with all the blood gases. Let me just find it.
Q: Take your time.
A: Okay. This baby was born at 2:41 and as far as I see, the earliest blood gas was obtained something like 0300 or 0307. So that would not have been an umbilical cord gas.
Q: That's not answering my question.
A: Okay.
Q: Do you know of any umbilical cord blood sample taken that was tested for gases?
A: No, I don't.
Q: Okay. You are working with the 0300 draw of blood sample from an umbilical artery catheter or some other source?
A: Some other. I don't know what the source was.
Q: Some other source, but arterial?
A: I presume so.
Q: Now, Doctor, I want you to give

Page

me the strongest case you can make within a reasonable degree of medical certainty for your opinion that Cody Miller's acute total asphyxia was due to umbilical cord spasm brought on by chorioamnionitis, please.
A: All right. Let's see. So there's three parts to the answer, because first we have acute asphyxia, then we have spasm, and then we have chorioamnionitis.
All right. So the first part of the answer is that the child matches the clinical picture for acute total asphyxia, and that was the point of the two articles that I provided you with.
Q: Tell me what you mean by "the child matches the clinical picture" for that.
A: The first part of the clinical picture is the way in which the asphyxia occurs, and in this particular child we have this severe drop in heart rate for a prolonged period of time. So that's the essential ingredient for the mechanism for this form of asphyxia.
The second thing is we have

Page 71

the acute picture of the asphyxia. In other words, yes, there are obvious neurologic problems with the tone and coma and abnormal EEG and seizures. But we have virtually sparing of all the other organs so that the only real damage is done to the brain. That's one of the themes that runs through this idea of the whole clinical picture of acute total asphyxia.

Then the third part of that is that Cody Miller has an uncommon type of cerebral palsy with choreoathetosis that matches the findings on the MRI scan, which agrees with where the damage is supposed to be if in fact it's acute total asphyxia.

Q: How does it agree with where the damage is supposed to be, Doctor, as you've talked about, the MRI scan indicating that?

A: Yes. In this particular syndrome of acute total asphyxia the brunt of the damage is in the deep gray structures, the thalamus, the basal ganglia; and when that part of the brain is injured, the effects are extrapyramidal CP, otherwise known as choreoathetosis in this

Page 72

child.

Okay. So in terms of the clinical picture, we have a very low heart rate for a long period of time, we have encephalopathy, absence of a multisystem malfunction, the damage on the MRI scan in the location that you would associate with acute total asphyxia, and then the clinical picture of choreoathetosis that you would expect to be in this clinical picture as well.

Q: Have you finished your answer to that question?

A: Well, your question actually first was acute total asphyxia due to spasm from chorioamnionitis. So that's Part A.

Q: All right. Can I ask you a question or two on Part A so that we can keep it manageable?

A: Yes.

Q: What do you mean when you say a very low heart rate for a long period of time?

A: Okay. The period of time would go from the onset of the terminal bradycardia really through the resuscitation in the newborn period.

Q: You will have to tell me what that is, Doctor.

A: The fetal bradycardia began at 2:19 a.m., continued until the baby was delivered.

Q: At 2:41?

A: At 2:41.

Q: Correct? You agree with that?

A: Well, I should be more careful, I guess.

Q: Yes. I don't mean to trap you, but I think you will find that's correct.

A: No, no. I think that's correct. They take the fetal monitoring off at some point, so we don't have numbers up to 2:41. The monitoring is removed.

Q: When is the monitoring removed, Doctor?

A: Let's see. I need to do a little counting here.

About 2:26. At 2:26 the monitor is off. At the moment of birth the heart rate is below 100.

Page 74

Q: Now, how do you know that?

A: I think the Apgar score was 1.

Q: Doctor, that Apgar score is not at the moment of birth, is it?

A: All right.

Q: Would you now tell me what you think the heart rate was at the moment of birth?

A: I don't know. That's a good point. It was at one minute.

Q: All right. While we're on Apgars, why don't you tell me what the components of the Apgars were at one minute, sir, as you understand it?

A: Yes. I have to go back and dissect that out.

Q: Go ahead and do that, sir. I don't mind if you get help, either.

A: I have it. The Apgar was 1 for heart rate, zero for everything else, and that was at one minute.

MR. HILTON: Gerry, that's from Page 2 of the labor and delivery summary.

MR. WALSH: Thank you.

Page 75

Q: All right. We have the one-minute Apgars, Doctor.
A: Yes.
Q: Now, what was the heart rate at one minute that gave it a 1 on the Apgar score?
A: No specific value is given. To get a value of 1, though, you have to be at least between zero and 100. If it's above 100, you get 2.
Q: Staying on this point, this Category A for the moment and the Apgars, what were the five-minute Apgars, Doctor?
A: 3.
Q: Total?
A: A total of 3.
Q: Can you tell me the individual components?
A: Let me look. I think it was 2 for heart rate.
Right. Actually, the five and the ten are the same. It's 2 for heart rate, meaning that it's over 100, and 1 for color, adding up to 3.
Q: When did resuscitative efforts

Page 76

including oxygenation and chest compressions begin, Doctor?
A: A little hard to say. I don't see where it's explicitly stated.
Q: Well, do you have a time in your notes that you are using to work from as when the resuscitation effort with oxygenation and cardiac massage began?
A: Let me just look here.
Q: Sure.
A: I take it it was right after the birth. I mean, what the records say is that the child was profoundly depressed and then patient resuscitated by the anesthesiologist with bag and mask. They don't give a precise timing on it.
Q: Doctor, do you have any understanding from any source about the resuscitation — namely, oxygen being provided and cardiac massage being provided — in this neonate before the one-minute Apgar scores were taken?
A: No, I don't know how the two overlap.
Q: The same question for before the

Page 7

five-minute Apgar scores were taken.
A: Correct. I don't know where the resuscitation is in that whole first ten minutes.
Q: All right, sir. Thank you.
Staying on this point, tell me, please, what you understand the intrauterine resuscitation efforts were for this fetus in the period between 0226 and 0239.
A: 0226.
Q: I tell you 0239 because that's my recollection of when the incision was made, Doctor, the first incision.
A: Okay. So between the time when the monitor stopped and 39 is the onset of the incision?
Q: Yes.
A: I am not sure what was done.
Q: Well, what was the condition of the fetal well-being as of 0226 according to what you read on the electronic fetal monitor tracing?
A: Basically right before 2:26 the child is in an extended bradycardia around

Pag

60.
Q: When you say "extended," can you tell me how extended, Doctor?
A: Well, for several minutes.
Q: Can you tell me what "several minutes" is when you use that term?
A: About six minutes at that point.
Q: Is it at all of significance or importance to you, Doctor, in your analysis of this case and the reaching of your conclusions, to know what the intrauterine resuscitation efforts were for this fetus between 0226 and 0239?
A: Not really. I mean, I was asked to look at this from a causation point of view and so whether or not there were attempts to resuscitate or successful attempts or feeble attempts or wonderful attempts, that doesn't really change my opinion about what happened to the child. The child was asphyxiated.
Q: Doctor, when was it that you contend that the child was first undergoing actual acute total asphyxia which continued

Page 79

unchanged and unabated?
A: Well, the asphyxia begins with the bradycardia. The damage begins after the 10 to 11 minutes.
Q: And I'm asking you, Doctor, as the expert witness pediatric neurologist in this case, to tell me within a reasonable degree of medical certainty when in this fetus' time in utero was the acute total asphyxia as you describe that present and continuing thereafter unabated.
A: Okay. So I define acute total asphyxia in this setting as heart rate below 60, and that starts then at just before 2:21.
Q: Have you finished your answer, Doctor?
A: Yes.
Q: I'm sorry; I thought you might have been still working on it.
A: No.
Q: All right. So the acute total asphyxia according to your belief within a reasonable degree of medical certainty or probability starts at 0221; correct?

Page 80

A: Yes.
Q: And does it continue for a period of time thereafter until the acute total asphyxia is no longer affecting the baby and ongoing?
MR. HILTON: I'm sorry; can you read that question back?
(The court reporter read the pending question.)
MR. HILTON: Objection to the confusing form of the question. Go ahead and answer it.
Q: (Continued) Then, Doctor, I don't want you to answer it if it's confusing to you, sir, please.
A: I think I know what you're asking.
Q: Okay.
A: Once it hits 60 beats per minute at 2:21, it basically remains at or below 60 as long as we have information.
Q: And how long do we have information, Doctor?
A: We have specific information only for five minutes because at 2:26 the device is removed.
Q: And you are not in a position to give me an opinion within a reasonable degree of medical certainty if the acute total asphyxia as you have defined it continued after 2:26 for any specific period of time; is that right?
A: Well, actually, I am. In other words, I certainly cannot tell you what the heart rate was because there is no information on that. The fact that when the child is born, he is asphyxiated and has the acute picture, I know that this process was significant for the baby.
Q: And when did that process take place in terms of when it began and how long you can state within a reasonable degree of medical certainty based upon the evidence it continued?
A: Okay. Well, again, as I said before, when you're below 60, that's potentially injurious. You have to be there for 10 to 11 minutes before the onset. So 10 minutes after 2:21 would be 2:31, and the beginning of the damage in my

Page 81

Page 82

opinion would have occurred around that time.
Q: The beginning of the damage would have occurred around 2:31, Doctor?
A: Yes.
Q: All right. That's what I was trying to get to —
A: Okay.
Q: — so I could ask you some more questions, because I wasn't sure in your report what you meant.
A: Okay. It's not in there.
Q: All right. Would you kindly tell me, Dr. Clancy, what you understand the intrauterine resuscitation efforts were for this fetus in the time frame from 2:21 to 2:31 on 1/30/93?
A: I don't actually have an opinion about that. I never considered that.
Q: No, I am not asking you for an opinion, Doctor. I didn't ask you that question. My question is — and I will restate it rather than having it read back — tell me what your understanding is of what actually was taking place in terms

Page 83

of intrauterine resuscitation for the fetus in the time frame from 0221 to 0231 on January 30, 1993.
A: Right. I didn't review that information still.
Q: You did not read those medical records in that time frame, Doctor?
A: Oh, I'm sure I did. But, in other words, that's not pertinent to my opinion as to what caused this. So if it was in one ear, it's out the other right now. I just don't remember what was done or what wasn't done.
Q: Well, once acute total asphyxia begins, as you say it did begin at 0221, does it continue without abatement even in the face of effective intrauterine resuscitation efforts or can the acute total asphyxia be affected and diminished in its impact on the fetus with effective intrauterine resuscitation, sir?
A: Well, I mean, if there is a way to reverse the process, for example, if it was cord positioning and you repositioned the mother, that could stop the process.

Page 84

So you are not guaranteed to have that happen by trying to resuscitate, but I suppose it's possible that if you restored the circulation to the baby, that it would interrupt the asphyxial damage.
MR. WALSH: Mark that also, please, Madam Reporter.
Q: Let's go on to Category B of your answer, Doctor, please.
A: Okay.
Q: Just give me a subheading for B, please.
A: Okay. B is going to be spasm of the umbilical cord.
Q: All right. Would you lay out your bases for that particular point, Doctor?
A: Actually, this in my mind is connected to the chorioamnionitis. But the point there is simply that the umbilical veins can go into spasm when there is an infection, and that ties us into Part C, which is that there was an infection of the umbilical cord and the placenta.
Q: Do you want to go into C then?

Page 85

A: Yes. I think maybe they're really linked together.
Q: Okay. Let's do C with the understanding that they work together in your concept of it, sir.
A: All right.
Q: And that's the infection caused by the chorioamnionitis?
A: Correct.
Q: Go ahead and tell me about that, sir.
A: Basically this was a pathological diagnosis and there were no other specific diagnoses other than the chorioamnionitis. In other words, there was not a diagnosis of abruption or infarction of the placenta, and that was also true from the obstetrician. I don't think they saw any other mechanism. So, at any rate, this was a pathological diagnosis; it was not a clinical diagnosis. The mother did not demonstrate the cardinal features of clinical chorioamnionitis.
Q: Which are?

Page

A: Fever over 100, leukocytosis, foul-smelling amniotic fluid, tenderness probably would be the main findings.
Q: Did the fetus demonstrate any of the clinical findings of infection due to chorioamnionitis?
A: I don't think so.
Q: Did you check?
A: Yes. I mean, the spinal fluid and the CBC's and so forth. I mean, they're always under the directive to, quote, rule out sepsis, and it's a necessary reflex because you never know at the beginning. But I don't believe and I don't think anyone there thought that the baby was septic and that was the basis for the problems.
Q: Okay. Now, do you have any other statements that you believe are pertinent to B or C, the spasm of the umbilical cord or the chorioamnionitis which caused the spasm?
A: Right. No. That's my basic opinion, is that there is chorioamnionitis and there is involvement of the umbilical

Page 87

cord as well. I have my personal knowledge about this causing spasm. I also furnished this paper which I know you didn't receive before today. But be that as it may, it describes how infection causes spasm in the vessel.

Q: Tell me when the spasm first began in the umbilical cord.

A: I don't know the timing of that.

Actually, let me go back, if I could.

Q: Sure.

A: You asked me before about do I know the cause of the first three and I said, no, I don't.

Q: Yes, that's what you said.

A: And the reason I answered that way was because all we know is that the heart rate went down. We don't know was it spasm from infection, was it the positioning of the baby, or whatever. Any of those could be possible. But to say I know the specific one, no, I don't know the specific one.

Page 88

The reason I think that the terminal one was from the infection was simply that they didn't describe any occult cord prolapse in the baby at that time of birth. So that is a possible mechanism. There was no evidence for that. What there was evidence for was the pathology of the placenta and the umbilical cord.

Q: Okay. Well, share with me all of the specifics that you can state regarding the pathology of the umbilical cord that support your opinion.

A: Okay. This is Page 50018.

Q: Can you tell me what the title of the page is, Doctor, since I don't have those numbered pages?

A: This is from the Department of Pathology, Rockingham Memorial Hospital, and this is the pathology report for — the patient is the mother — Donna Jean Miller. The microscopic diagnosis is diffuse acute chorionitis.

Q: Are you finished your answer, sir?

A: Yes.

Page 89

Q: Here again, I have to tell you that I don't think you've answered my question.

A: Okay.

Q: My question is: Tell me what facts you have and what information you are relying on to tell me that it is the umbilical cord pathology that supports your opinion that there were spasms caused by chorioamnionitis.

A: Well, spasm is a functional term, it's a physiological term, first of all, so that's not going to show up on any path report. Like a muscle spasm, if you look at the muscles under a microscope, the muscles look the same, there's nothing different about the way they look. The spasm is a functional disturbance. So I'm putting together the infection and the clinical picture for the spasm because blood vessels go into spasm.

Q: All right. Is there a microscopic evaluation of the umbilical cord in the Rockingham Memorial Hospital pathology report?

Page 90

A: It's not described. It's listed as part of the specimen, but it's not described.

Q: Okay. Have you reviewed Dr. Keough's microscopic assessment of the umbilical cord slice?

A: No.

Q: Tell me how many spasms of the umbilical cord there were that were implicated or involved in the decrease or blockage or cessation of perfusion through the umbilical cord.

A: At any time or do you mean at this moment?

Q: Well, at the moment or moments which preceded the onset of acute total asphyxia which you say happened at 0221 and continued at least until 0231.

A: I have no way of quantifying them other than to say I believe there was at least one.

Q: And tell me when that one spasm occurred.

A: Well, prior to the onset of the bradycardia.

Page 91

Q: Can you state that within a reasonable degree of medical certainty, Doctor?
A: Well, I can certainly state that the spasm had to precede the bradycardia.
Q: Right.
A: But how long before I wouldn't know.
Q: You can't give me any time period when the spasm that you believe occurred in the umbilical cord prior to 2:21 occurred; is that right, sir?
A: I guess the question is how fine a point to put on this.
Q: I want you to put as fine a point on it as you are capable, Doctor.
A: Well, you know, within a couple minutes of the bradycardia, but not an hour before, I mean, it's not going to have a delayed effect. It would be in close proximity to the bradycardia.
Q: All right. Go out on a limb, Doctor, and give me a time that you think it happened.
A: This would be pulling a number

Page 92

out of the air and I'm not really sure that's what you want.
MR. HILTON: Object to the form of the question.
Go ahead.
Q: Isn't that what you've done, Doctor, is to come up with the conclusion, that sometime before 2:21 when the acute total asphyxia was in being, that a spasm had happened beforehand that caused it? Correct?
A: Yes.
Q: All I'm asking you to do is tell me when that spasm occurred, Doctor.
A: Well, again, whether you say it's a spasm or a cord prolapse or the elbow's on it, it's hard to know that. All we have here is a heart rate. So I think it would be within a minute or so of the onset of the bradycardia, whatever the event was.
Q: Are you now saying, Doctor, that the precipitating event of the decrease or blockage of the blood flow through the umbilical cord might have been due to a

Page 93

cord prolapse or an elbow being on it?
A: Well, certainly those were the same considerations for the earlier bradycardia and that's why they put the mother in a different position, gave her oxygen and fluids and so forth. And those heart rates always responded in a short period of time. So any time there's a bradycardia, there's always possible causes/possible reasons for it, and that's why they responded the way they did when the previous three bradycardias occurred.
So my point is that at the moment of the bradycardia, whether it be in this case a spasm or an elbow or a prolapse, I don't know how much before them. That was what your question was. I would think a minute or so.
Q: No, no. My question wasn't that at all, Doctor. My question had nothing to do with a cord prolapse or an elbow being on the cord.
My question is this: Are you able to state when prior to 0221 on January 30, 1993, the time which you have

Page

told me is when the acute total asphyxia was present for the first time, that the umbilical cord spasm you have said resulted in the acute total asphyxia occurred? Do you understand that question?
A: Yes.
Q: All right. Would you answer that, please?
A: Okay. Again, we don't know, whether it be a spasm or any other mechanism, that exact onset. I think it would be a conservative estimate to say within a minute or so of the bradycardia the event occurred.
Q: Well, Doctor, what is it that caused the acute total asphyxia to result from the bradycardia?
MR. WALSH: Strike that.
Q: Are you saying that the acute total asphyxia was caused by bradycardia?
A: Yes.
Q: Severe injurious bradycardia; correct?
A: Yes.
Q: Are you saying that the severe

Page 95

injurious bradycardia was caused by a spasm in the umbilical cord brought on by chorioamnionitis?

A: Yes.

Q: Are you saying that the severe injurious bradycardia was caused by a cord prolapse?

A: No. Was the bradycardia from prolapse? No.

Q: Are you saying that the event in the range of 2:19-2:20 that we have been talking about of severe injurious bradycardia was brought on by something like an elbow compressing the umbilical cord?

A: No, I'm not.

Q: There is no question in your mind that the severe injurious bradycardia was brought on by a spasm of the umbilical cord and you can state that within a reasonable degree of medical certainty?

A: Within a reasonable degree of medical certainty I can.

Q: And that's a spasm that happened about a minute before the bradycardia

Page 96

began, Doctor; is that right?

A: That's my opinion, yes.

Q: All right. Now, how long did that spasm continue, Doctor, once it began?

A: I have no way of knowing.

Q: Why don't you have any way of knowing, Doctor?

A: Well, first of all, we lose the information on the heart rate, and that's the only gauge we have on that flow through the umbilicus.

Q: All right. Have you answered?

A: Yes.

Q: Where on the umbilical cord did the spasm occur?

A: It would be the venous system.

Q: I didn't make that clear. Thank you for the answer, but I am going to ask another question.

A: Okay.

Q: The umbilical cord is a certain length attached to the placenta and then the insertion in the umbilicus of the baby; right?

A: Yes.

Page 97

Q: Where on that longitudinal umbilical cord did this spasm occur, Doctor?

A: I don't know that.

Q: Why don't you know that?

A: Why did you ask that? I mean, there's no way of knowing that.

Q: Well, how would I know you wouldn't know that?

A: Because it's a functional reaction; it's not an anatomical reaction.

Q: What do you mean "it's a functional reaction"?

A: A blood vessel that goes into spasm is a functional disturbance. Okay?

Q: Okay. Which blood vessel went into spasm in the umbilical cord, Doctor?

A: The vein.

Q: How many veins are there in this umbilical cord?

A: One.

Q: What happened to the vein that went into spasm in the umbilical cord, Doctor? What went on with it in terms of the process of spasm?

Page 98

A: Veins react to their environment. For example, if there's infection in the environment or chemical mediators, the size of the veins will change, just like if we walk outside, our hands may get blue, or if we blush, our face may get red, depending on how the veins dilate or contract. So veins have the capacity to change their shape and their tension depending on their environment. So with contraction of the vein or spasm of the vein, flow through the vein is reduced or stops.

Q: And once a spasm begins, does it take something, some action, to cease the spasm in the vein of the umbilical cord, in this instance, from continuing?

A: I suppose that when the trigger is removed, when the inflammation is removed, that the vein will eventually relax. But that's not like a minute-to-minute thing; that can go on for a considerable period of time.

Q: Where do you get that information, Doctor?

Page 99

A: From the paper that you haven't read yet.
Q: The paper I received this morning?
A: Yes.
Q: Well, was this a spasm of the umbilical vein which amounted to a total occlusion of the umbilical vein?
A: Well, a spasm will not actually occlude it. I mean, it will reduce flow completely or markedly. But to me occlusion is like, you know, hardening of the arteries where there's physical stuff in there.
Q: Oh, I see. Is this spasm that you contend occurred in the umbilical vein a spasm which constricted the umbilical vein so that there was no blood flow, oxygenated blood flow, through the umbilical vein?
A: It certainly constricted the flow. To say zero flow, I don't think anyone could answer that. But reduced flow, yes.
Q: Tell me how much of the flow of

Page 100

the oxygenated blood through the umbilical vein occurred at the site of the spasm, Doctor. And you can give me that in your best estimate of percentage of the decrease in normal blood flow.
A: I would estimate that more than 90% reduction would have to occur to produce a bradycardia.
Q: How do you go about this intellectual process of estimating that information, Doctor?
A: Okay. This comes back to the Myers experiments and the point there was that they actually did a series of umbilical cord clampings to see what level of reduction of flow was necessary to produce the end results. And the figure was when there was 90 or more reduction in flow for the 10 or 11 minutes, this had an effect on the fetal monkey's heart rate.
Q: What were they clamping, sir?
A: The umbilical cord of the fetus, the monkey's fetus.
Q: In the Myers study did they clamp only the umbilical vein?

Page 101

A: No. You clamp the cord so that the arteries and the vein are all included.
Q: In this case you are not testifying within a reasonable degree of medical certainty that there was any spasm in the umbilical arteries, are you?
A: No.
Q: Do you have any evidence whatsoever that there was any spasm in the umbilical arteries at or around the time of the onset of the acute total asphyxia?
A: No, there is no evidence.
Q: What was it exactly that caused the umbilical artery to go into spasm and reduce the blood flow of oxygenated blood —
A: You said artery. You mean vein?
Q: Vein to go into spasm — strike that. I'll make it clear.
What was it about the chorioamnionitis that caused the umbilical vein to go into spasm and reduce the oxygenated blood flow by 90%?
A: The process is that within

Page

inflammation there is a great deal of change in the environment where the infection is. The most obvious thing would be the pus cells and so forth. But what's created in the environment are vasoactive substances.
The simplest example for you and I would be something even like a beesting. All of a sudden it's red and puffy because there's inflammation there. The body responds to a foreign substance, whether it be an infection or a beesting or whatever.
So here with the infection is created within that environment vasomotor mediators. This is the same thing even for people in our age, like heart attacks are more common when you're sick.
Q: How does that agent/substance/whatever it is that is causing the spasm of the umbilical vein get to the site where it causes the spasm?
A: Well, first of all, it's in the *amniotic fluid, so it's part of that whole*

Page 103

pocket of water. The umbilicus is bathed in the amniotic fluid. It also inserts into the amnion, so now it is physically attached.

Q: What's the amnion that it's physically attached to something? What are you talking about?

A: In other words, the cord itself is physically attached to the placenta.

Q: Yes.

A: So one way of how do you get the infection there is, well, they're together, they're connected. Plus everybody's in this sea. The amniotic fluid, it's infected, too. So the infection comes through the cord. The cord is bathed in infection.

Q: Well, is the infectious pathogen in the amniotic fluid? Is that what you're saying?

A: Yes. I mean, it's in the amniotic fluid. It's in the chorion. It's in the amnion.

Q: Well, how does it get from the chorion to the umbilical vein?

Page 104

A: Again, the whole cord is bathed in this fluid so it invades from outside, in other words, from the outside walls in, and also by direct extension, so-called funisitis, directly from the placenta.

Q: Do you have an opinion as to what route the infectious pathogen took to cause the spasm?

A: No.

Q: Do you have an explanation as to why the umbilical arteries didn't go into spasm?

A: Well, I don't know how you would recognize spasm in the arteries. In other words, the arteries are leaving the baby going to the placenta. So if there had been, I'm not sure what sign you would find to detect that. The vein is taking everything back to the baby, so if you have a spasm there, now you are cutting off the good blood and the good oxygen supply.

Q: Well, if it was an infectious process and a hostile pathogen insofar as what is normal and it did come into one of the two umbilical arteries, it would

Page 105

inflame those, wouldn't it?

A: Yes, I think it certainly could.

Q: And it could be determined on microscopic evaluation if it was present, couldn't it?

A: It could, yes.

Q: And the same holds true for the uterine vein: that on microscopic examination this sort of pathogen, if present, could be found; correct?

A: You mean umbilical vein?

Q: Umbilical vein, yes. Thank you, Doctor.

A: Sure. Right. I mean, you could see inflammation of the vein.

Q: Okay. You talk about "The pathological condition of chorioamnionitis can cause abnormalities of the umbilical cord" in your report. What do you mean by that, Doctor?

A: Funisitis or cord inflammation.

Q: And in particular you say: "It can cause irritation of blood vessels in the umbilical cord leading them to go into

Page 106

spasm." What do you mean by that, sir?

A: What we have been talking about, the venospasm of the umbilical vein.

Q: Anything else?

A: No.

Q: And you say: "When this occurs, there is a sudden and sharp reduction of blood flow between the baby and the placenta, as occurred in this case." What do you mean by that?

A: The 90% reduction.

Q: Was this spasm of the umbilical vein similar to a compression of the umbilical vein by trauma by something, an elbow being placed on it, a prolapse?

A: It could have the same effects as spasm.

Q: And when there is this spasm or compression of the umbilical vein, what do you see the fetus' reaction to be in the areas that you can assess?

A: Well, this is really not different than the other question; in other words, when we looked at the bradycardia charts and you asked me what happens to the

Page 107

baby. So it's a stress. There's the physiological response to stress. There's the biochemical response to stress. So this is all mediated through the cardiovascular system of the child. So whether it be an elbow or a prolapse or a spasm, if the baby gets bradycardic, then they have to turn on their protective reflexes to mitigate against that.

Q: When in the 0200 time frame do you see a fall in the heart rate below 100, Doctor?

A: What was the panel on that?

MR. HILTON: 2 o'clock is 90392.

A: (Continued) I'm sorry; you will have to ask your question again.

Q: Was there a fall in the fetal heart rate below 90 in the time frame of approximately 0200? By that I mean on either side of it, Doctor, just to —

A: Yes; at 2:02.

Q: Does that continue through the end of the tracing that's available to you?

A: Well, again, I have a normal

Page 108

tracing and then it disappears and all I have here says: Deceleration to 40 to 50. So if it's 40 to 50, certainly it's below 90 at that time.

Q: No. I'm sorry, Doctor; I didn't make myself clear.

You see a heart rate below 90 at about 2:02; is that what you told me?

A: No. I mean, the graph itself is not below 90 anywhere along that time.

Q: Oh, when you said the heart rate was below 90 at 2:02, where did you get that factual information?

A: Because the nurse writes in: Deceleration to 40's and 50's.

Q: It's not shown on the graph?

A: It's not shown on the graph.

Q: All right. Thank you.

Now, tell me what you understand happens to the fetal circulatory system in terms of the circulation of blood when the blood flow in the umbilical vein is reduced as you say it is reduced by this spasm.

A: So what happens to the

Page 109

cardiovascular system? Probably the earliest thing would be that the coronary arteries of the baby's myocardium dilate, they're trying to get whatever blood they can into the heart, and there is a shunting of the venous blood away from internal organs such as the GI tract, liver, kidneys, spleen. So in a sense they're robbing Peter to pay Paul.

Q: Now, Doctor, when there is reduced umbilical blood flow through the umbilical vein by this spasm that you contend occurred and caused injurious bradycardia which went into acute total asphyxia, is there redistribution of blood flow to fetal organs?

A: There's redistribution from fetal organs.

Q: Well, what do you mean "from fetal organs"?

A: From the GI tract and the liver and the kidneys to the myocardium and to the central nervous system.

Q: Okay. Well, is the redistribution of the blood flow different

Page

in this acute total asphyxia than that which is observed during maternal hypoxemia?

A: You are going to have to be more specific than that.

Q: Do you know what maternal hypoxemia means in the context of a mother with a fetus in the stage of gestation such as this one was?

A: I do understand what maternal hypoxemia is.

Q: Tell me what you understand maternal hypoxemia is in that setting.

A: Low oxygen.

Q: Low oxygen in the mother?

A: Right.

Q: Which then has an impact upon the uteroplacental perfusion; correct?

A: Well, has an impact on the oxygen delivery to the — I mean, perfusion is how many gallons of blood go there.

Q: All right.

A: Hypoxia is just the gas tension.

Q: When there is hypoxemia based in the mother, the fetus gets less oxygenated

Page 111

blood; correct?
A: Yes.
Q: All right. Now, is there a
difference, Doctor, between the
redistribution of blood to fetal organs
when there is reduced umbilical cord flow
because of a spasm as compared to reduced
flow to the fetus because of maternal
hypoxemia? That's my question.
A: All right. So let me answer it
this way: Probably, no, there is no
difference; and the only reason I'm being
fussy about this is that, again, you're
saying hypoxia.
Q: Hypoxemia.
A: Hypoxemia or whatever.
Q: Well, it's a term of art, isn't
it?
A: As opposed to ischemia. Okay?
Big difference between the two.
Q: I know. That's why I'm asking
the question.
A: All right. Well, if there's no
alteration in perfusion to the baby, it's
purely oxygen, I'm less sure what the organ

Page 112

rearrangements are.
Q: Well, that's why I want you to
answer this question —
A: Okay.
Q: — Doctor, as the expert witness
in this case on the outcome of this child,
and I am going to ask it again to be
eminently fair to you.
Is there a difference in the
redistribution of blood flow to fetal
organs between reduced umbilical blood flow
caused by blockage of the umbilical vein by
a spasm compared to the mother having
maternal hypoxemia and that blood being
delivered to the fetus? Can you answer
that question?
A: Yes. I don't know the answer.
I have never seen a case of simply pure
maternal hypoxemia, period. I mean, I've
just never seen that.
Q: Do you know what happens in that
situation that I have just asked you about
with regard to either an increase or a
decrease in the flow of blood to the brain
when the reduction of blood flow is due to

Page 113

an umbilical problem where the flow is not
normal?
A: I have to hear that question
again.
MR. WALSH: Read it back to
Dr. Clancy.
(The court reporter read the
pending question.)
A: (Continued) That implies to me
you are saying now the mother has hypoxia
and there is —
Q: No.
A: That's what the question says.
Q: Then I'm glad you corrected me.
Doctor, assume this
situation: the blood flow to the fetus is
affected by a spasm such as you've
described. How is the blood flow to the
brain of the fetus affected in that
instance as compared to how it would be
affected if there was maternal hypoxemia?
A: Okay. And the answer to that is
also I don't know because I've never seen a
case of pure maternal hypoxemia.
Q: All right. How is the blood

Page 114

flow increased or decreased to the heart in
that same situation?
A: With pure maternal hypoxemia?
Q: I will say the question again,
Doctor, I was trying to use shorthand.
A: All right, go ahead.
Q: With reduction of umbilical
blood flow, as you posit in this case,
which is due to a spasm in the uterine
vein, how is the blood flow to the heart
either increased or decreased when compared
to maternal hypoxemia?
A: The same thing. You know, maybe
I can save you some questions. But since
I've never seen a case of maternal
hypoxemia alone, I won't know what it does
to the head, the heart, or any other part
of the baby. As far as I know, it doesn't
exist.
Q: All right. Are you saying that
maternal hypoxemia does not exist?
A: Not as an isolated hypoxemia.
Q: All right.
A: I mean, that would require
putting a mother in a room full of nitrogen

Page 115

but with normal blood pressure and normal perfusion. Well, how do you do that? You know, that's not part of reality.
Q: Okay. Is it your position in this case that when there was this spasm that you say happened and there was a 90% reduction in the flow of oxygenated blood through the umbilical vein, that certain organs of the fetus got less blood and were damaged?
A: That's what happens in all people or all babies.
Q: Which organs got less blood and got damaged?
A: Well, I didn't say they got damaged; I said they got less blood.
Q: All right. Were there any organs that got less blood and got damaged in this setting of Cody Miller?
A: No. In this baby nothing was damaged.
Q: What happened to the brain?
A: Well, I thought you were talking about multiorgan damage.
Q: No. Is the brain an organ?

Page 116

A: Start again.
Q: No. I am just asking you. I am not trying to be flippant, Doctor.
A: Of course it is.
Q: I am really not.
A: All right. Go ahead.
Q: The brain is an organ?
A: Yes, it is.
Q: Did the brain get less oxygen than normally?
A: Yes, it got less oxygen and it was damaged in the process.
Q: Did other organs of the body other than the brain get less blood than normal?
A: I think the kidneys got some less perfusion because there was reduced urine output after birth.
Q: Can you tell me if the kidney, the gastrointestinal tract, or the spleen got less blood during this time with spasm than they would normally get, Doctor?
A: I think that is true.
Q: Can you tell me if the heart and the adrenals got less blood during this

Page 117

period of spasm than they normally would have gotten, Doctor?
A: I think the whole baby got less blood than he normally would have had.
Q: Did the whole baby get less blood in the same proportion of reduction of the amount of normal blood supply that separate organs would have gotten with normal blood supply?
A: I think the notion is that everyone is cut back because the whole supply to the baby is less. Some get worse than others.
Q: Tell me which ones get worse than others.
A: Again, the kidneys, the liver, and the bone marrow, the spleen have further reduction, a disproportionate reduction; and, yes, the heart is reduced as well as, yes, the brain is reduced as well, but proportionately they're better off. And that's the idea of protecting the organism, the person's body.
Q: All right. Is there any one organ that suffers a significant reduction

Page

of blood flow when there is reduced umbilical blood flow as you say happened during the spasm?
A: Is there any one organ what?
Q: That gets significantly less blood flow than normal when there is the reduced umbilical blood flow that you say was occasioned by the spasm of the uterine artery — or the umbilical artery.
A: Well, again, I think the whole body is reduced, so all the organs have reduction. When you say "significant," I'm not sure what you mean by "significant."
Q: I mean by "significant" does any particular organ get significantly less blood than any other organ? Is there one organ singled out to have a significant reduction in the flow of blood to it?
A: I guess the only thing I can say is that when there's one organ that takes it on the chin, it's more likely the kidney than the other organs.
Q: Doctor, after Cody's birth were there any conspicuous signs of an acute neonatal encephalopathy?

Page 119

A: Oh, of course.
Q: Can you tell me what there was?
A: Basically seizures, abnormal eye movements, unresponsiveness, low muscle tone. He needed mechanical ventilation. These are all the visible signs of encephalopathy.
Q: Was there multisystem malfunction of organs?
A: No, not other than oliguria, reduced urine flow.
Q: Are there any other reasonable causes for Cody Miller's neurological disorder that has been diagnosed?
A: Aside from asphyxia?
Q: Yes, any other reasonable causes.
A: I didn't see any.
Q: What was the nature of the brain damage that Cody Miller did receive as a result of this insult you've posited happened, Doctor?
A: An ischemic insult, infarction of the deep gray structures.
Q: Was that bilaterally in the

Page 120

brain or was it one-sided?
A: It was bilateral.
Q: Was there brain swelling?
A: Not that I know of.
Q: Did you look for it, Doctor, in the records?
A: Yes.
Q: What is the frequency known to you in your field of medicine of the presence of acute total asphyxia in human infants?
A: The proportion of all cases?
Q: Yes.
A: Very small.
Q: Can you explain that any further, Doctor?
A: Oh, maybe one in ten asphyxiated children in my personal experience fit this picture.
Q: Was Cody Miller's brain damage in the deep brain?
A: Yes, the deep gray structures.
Q: Why did it happen to his deep brain, the damage?
A: Right. That's basically the

Page 121

nature of the beast. I mean, it's nothing unique to Cody Miller. It's any infant in that situation, that's the vulnerable areas.
Now, the physiology of this is supposed to be that in acute prolonged hypotension, that the deep gray structures are in a watershed and they're like at the end of the trail. They're also packed with what are called glutamate receptors which are liberated in hypoxia. So they are spilling out these toxic transmitters which are thriving the brainstem.
Whether that's the whole mechanism or not, I guess in a way, operationally, even though we may not know why it settles in there, we certainly know that it does settle in there. The pathology tells you that.
Q: Was the brainstem of Cody affected?
A: It was clinically. In other words, the fact that the child needed a machine to breathe for him, was pooling saliva, had to be suctioned, would say that

Page 122

that part of the brain is not working.
It's very difficult to image the brainstem. I mean, my brainstem is the size of my pinky, so a newborn's brainstem is very small. It's hard to resolve those areas. So I don't believe they ever said that the brainstem looked abnormal on the MRI, but clinically the child's brainstem was functionally abnormal after birth.
Q: Was there any renal dysfunction due to this acute total asphyxia, Doctor?
A: There was some renal malfunction in the first day or so of life. The child did have low blood pressure then, hypotension. Of course, you have to perfuse the kidneys to make urine. So once the blood pressure was improved, the child's urine output was fine. The creatinine count was never abnormal.
I looked in the labs for red blood cells in the urine and in the laboratory I don't see it. If there is a notation somewhere by a nurse, that's possible, but I didn't see it.
Q: Why were you looking for red

Page 123

blood cells in the urine, Doctor?
A: Because sometimes the kidney
will get leaky if it's injured and then it
leaks the red blood cells that are filtered
through the blood into the urine.
Q: What seizures did Cody Miller
have postdelivery?
A: I'm sorry?
Q: Postdelivery what seizures did
Cody Miller have?
A: Postdelivery? I mean, the
simplest answer is simply newborn seizures,
and the reason I answer it that way is that
they are fairly pleomorphic. They can look
different ways. They describe unusual
movements, unusual eye movements and so
forth. So they were different appearances.
Q: What was the time of onset of
those, Doctor?
A: I have to look here. I'm sure I
have it labeled here.
MR. HILTON: Do you want me
to help him?
MR. WALSH: Sure. Go right
ahead.

Page 124

A: (Continued) Okay. Well, I
think I want to look at the nurses notes,
too. This is the doctor's admission notes
and it simply says as part of the hospital
course shortly after birth, but they don't
give a specific time.
Again, I have now the
newborn physical examination which is dated
January 30 of '93 but has no time on it
which states: Initially very depressed,
later some movement and respiratory effort,
later tremulous with seizure activity.
Again, no specific time.
Another untimed entry
describes: Admission Data: Term infant,
asphyxia, seizure.
Hang on here.
Okay. Here we go. Now,
here's the nurses notes and the timing here
is 0314 and it says something like
quivering lips, eyes twitching. Now, I
don't know if that's the first recorded
seizure, but it's the first one that's
timed that I saw.
Q: More specifically, Doctor, have

Page 125

you already reviewed these records and
determined in your own mind based upon the
facts in the records when the onset of
seizures was first noted?
A: Not to the minute. I mean, I
knew it was very early after delivery.
Q: All right. And what's the
significance of the time in which the
seizures became observable and the
nature/frequency of the seizures?
A: Well, the fact that they're
there tells you that there's something
acute going on, whether it be asphyxia or
bleeding or infection or whatever else. So
it says there's something serious going on
with the brain.
Typically seizures begin
within the first 24 hours of life in
asphyxia and I don't particularly get too
wrapped up in the exact moment of onset to
count back 12 hours or anything like that.
Q: Okay. Was there any bleeding
that you think caused these seizures,
Doctor?
A: No.

Page

Q: Was there any infection that you
think caused these seizures?
A: No.
Q: When you went through this case,
how did you analyze the records before
reaching your conclusions and opinions
which are in your report? Just give me a
shorthand version of how you analyzed the
case.
A: I often work backwards
actually. In other words —
Q: What do you mean?
A: Meaning that I actually looked
at what the child's status was now, meaning
he has a normal head circumference,
relatively preserved —
Q: Did you actually work backwards
in this case?
A: Yes.
Q: All right.
A: — the cognitive ability and the
athetosis, and then the scan was deep gray,
and then I realized this was not your
average birth asphyxia where the kids are
microcephalic and spastic and all that.

Page 127

And then when I read through the perinatal records, there it was, the terminal long bradycardia, and I knew that that was the tie-in.

Q: When you got to that point, did you in your own mind run through a process of differential diagnoses to see what would be on your list of most probable causes of that untoward event?

A: I mean, to do a fair review you have to.

Q: Did you, Doctor, is my question.

A: Yes.

Q: And what was your thought process on your differential diagnoses as to the most likely cause that you would back as the reason?

A: Well, I guess my focus was always on the cord and the amniotic fluid, the chorioamnion, because I had seen the pathology; and the other part was I guess the doctor who delivered the child didn't see any other placental abnormalities. So I do have positive evidence for infection

Page 128

because it's there microscopically and I have the absence of other factors by the eyewitnesses there. So I thought that that was suitable to exclude prolapse and other things that were on the list but there was no evidence for them.

Q: Well, other than prolapse, what else was on the list of differential diagnoses for a cause, Doctor?

A: Compression. I mean, not just a prolapse through the vagina but compression by a presenting part or —

Q: Yes. What else?

A: There's weird things like there's hematomas —

Q: No, I am not asking for weird things.

A: No, I am saying there are other things like there are hematomas of the cord or true knots and there can be fistulas and there's all kinds of things.

Q: Mechanical obstructions?

A: Yes.

Q: So you have kind of told me what your differential diagnoses amounted to?

Page 129

A: Yes.

Q: Have you read Dr. Stevenson's deposition?

A: I did last night.

Q: Do you have any significant substantial disagreements with his thought process and findings?

A: I was actually a little confused. I wasn't convinced I really understood what his opinions were. I mean, I don't think anyone is saying that the child was not asphyxiated. It was not clear in my mind, when he was talking about acute asphyxia, if he was trying to distinguish between these two models that you and I have been talking about as opposed to something that happened a month ago. That was my confusion with that.

Q: I see. All right.

In terms of the fetus, what is circulatory decentralization?

A: I believe what you're referring to, if it's my same understanding, is this idea of redistribution of blood flow from the center, from the internal organs, to

Page 130

the heart and the head.

Q: Have you ever read the medical literature on circulatory decentralization, Doctor?

A: Yes.

Q: Have you ever written on that point?

A: I don't think I've directly written on it.

Q: With regard to Dr. Pasternak's article which you had attached to your report, would you tell me what it is that is in there that you believe is support for your opinions, as I understand that you did indicate that was the situation? You do actually say "copies of three medical articles which support my opinions." I am not interested in the opinion on life expectancy, Doctor. I am not even going to ask you a question about that, sir. I am interested in the opinion on proximate cause of the damage.

A: So your question is what's the value of this paper to me in this case?

Q: In supporting your opinions,

Page 131

Doctor.
A: Right. It provides a peer-reviewed document that describes the syndrome and that when I have analyzed the facts of the Cody Miller case, that the blueprint of this child's records, profound bradycardia lasting a long time, all that we just talked about, match this description; and in fact the outcome of these children, both with respect to sparing of the other organs, the location of the damage, the type of cerebral palsy that results, even some of the conditions that gave rise to this, thrombosis of the cord, rupture of the cord, and so forth, that there is a foundation in the medical literature that matches my experience and my opinions about the case.
Q: It's more of a general confirmation of your opinions as you see it?
A: Uh-huh.
Q: That's a yes?
A: Okay.
Q: No. That's a yes?

Page 132

A: Oh, you want me to give you —
Q: Well, see, you said "uh-huh" and she can't take that down, Doctor. That's not a useful response.
A: Sorry.
Q: I am not making you say anything, but was that a yes you were saying, that it's a general confirmation of your opinions in your view?
A: Yes, it is.
Q: That's what I was trying to say.
A: Okay.
Q: Doctor, briefly tell me what you've reviewed in this case while I look at this article I got, and you can simply keep reading into the record until you run out of things to read into the record as I use my Evelyn Wood technique on this article.
A: All right. Two volumes of medical records, including the mother's maternal records, the baby's newborn records and followup records.
MR. HILTON: I think he

Page 133

wants you to list all of that stuff, too.
THE WITNESS: Okay.
A: (Continued) I have an article from Phalen called "Intrapartum Fetal Asphyxia...," et cetera; an article by Pasternak called "The Syndrome of Acute Near-Total Intrauterine Asphyxia."
I have a copy of my report; a report from Judith Poole; a report from Jean Bolan; actually, a copy of an e-mail from you, Mr. Walsh.
Q: Me? My goodness.
A: You didn't send it to me.
MR. THOMAS: You mailed it to Frank.
A: (Continued) That I have just giving me a heads-up.
Q: I must have been asking for an advanced copy of my application, Doctor —
A: I see.
Q: — in my mind.
A: Plaintiff's Supplemental Answers to Interrogatories; seven pages of handwritten notes; a sheet from followup from the child's pediatrician; a document

Page

called "Complaint for Medical Negligence," and there's two of these; a report from Medical Rehabilitation Resource Consultants.
I have read the father's deposition, Leonard Miller; the mother's deposition, Donna Miller; Richard D. Stevenson's deposition; an article or a paper about umbilical vein spasm.
Q: Can I see that?
A: And these are two copies of my CV. I wasn't sure if I had provided that before.
Q: Where did the article in Obstetrics and Gynecology, Scott Hyde, the leading author, come from, Doctor?
A: From the library.
Q: You got it?
A: Yes.
Q: All right. Anything else you have, Doctor, that you reviewed?
A: Yes; a report from James O'Leary, and that's it.
Q: Have you learned any facts about this case which you have utilized in

Page 135

formulating any of your opinions, Doctor, that are facts that are not in these documents that you have told me about, such as getting facts in letters from counsel or a phone call from somebody, talking to somebody, saying, Doctor, I want you to know about this A, B, and C?

A: No. The only reports I've had are from your experts.

Q: All right. Do you know Dr. Zimmerman, the radiologist?

A: Yes.

Q: You have written together in collaboration on matters, haven't you?

A: Yes.

Q: How long have you known him?

A: 20 years.

Q: Professionally or professionally and socially?

A: Professionally.

Q: All right. What is it in the article "A Model of Bacterially Induced Umbilical Vein Spasm, Relevant to Fetal Hypoperfusion" that is relevant to your opinions or support for your opinions, Doctor?

Page 136

A: Basically because it provides a model for the clinical picture. The clinical picture is that when there is chorioamnionitis, that the blood vessels behave like there's spasm. This actually takes healthy women who have just delivered, so it's a human being study, they cut the umbilical cord after the baby is delivered and then directly in a sense pour on the germs to see how the blood vessel responds. You can actually see the vessels go into spasm. So that's the relevance of that.

Q: Have you personally read any of the EEG tracings?

A: No.

Q: Do you have any intention to read any of them in order to firm up any of your opinions?

A: No. I don't think they'll be that helpful.

Q: You've been in cases before with Mr. Hilton, haven't you?

A: Yes; a couple.

Page 137

Q: Tell me how many, Doctor.

MR. HILTON: One, I think.

A: One.

Q: George Mays versus Rockingham Memorial Hospital?

A: Oh, Mays, yes.

Q: Is there some reason that wasn't on your list of cases, Doctor?

A: If it was already over with, it would be out of my office.

MR. HILTON: I think it was more than four years ago, Gerry.

MR. WALSH: '98. His deposition was taken April 9, 1998.

MR. HILTON: Okay. My mistake.

A: (Continued) No. But, again, once a case is done, it leaves my office. So what I have on my list here are any things that I can put my hands on.

Q: Where does it go when it leaves your office?

A: In the trash.

Q: Well, doesn't somebody keep your schedule and appointments, Doctor?

Page 138

A: No, I mean, not really. I don't necessarily have a record of, you know, the cases.

Q: Do you have a secretary?

A: Among five people; yes.

Q: Does she have a calendar of your schedule for being in depositions today or being at —

A: No.

Q: Do you keep a calendar?

A: She does not. My secretary is at the University. She wouldn't keep it.

Q: Do you keep a calendar of where you're going in depositions and trials?

A: Yes.

Q: Do you keep the back copies of those calendars?

A: No.

Q: Do you get the money, the checks that are sent to you by the people who engage your services —

A: Yes.

Q: — and negotiate them?

A: Yes.

Q: Do you get the 1099's at the end

Page 139

of the year and then report on your income tax that income?
A: Yes.
Q: Do you keep your income tax returns for a certain period of time as required?
A: Yes.
Q: And you have those 1099's with income tax records for the last four years, don't you?
A: I do. But, again, for a case you might get a check from an insurance company and it's St. Paul's, and I'm not going to be able to reconstruct from that who the case was.
Q: Well, Doctor, have you attempted to do that, to look at the 1099's that you have from the various people who have engaged you to see what cases they're for?
A: What I did was what I thought was a reasonable effort to answer this and the answer is no, of course.
Q: All right. That's all I'm asking.
A: All right.

Page 140

Q: I'm not quarreling with you.
A: Okay.
Q: These cases represent the active cases you had in your office when you were asked to prepare the list; is that it?
A: Well, they're still alive. They're not active and ongoing right now. You know how things come and go. But those are still unsettled cases, if you will.
Q: How did you go about preparing the list?
A: I went from file to file and wrote down the names.
Q: When you use the term "anoxia," what does it mean to you?
A: Total lack of oxygen.
Q: To what? To where?
A: Well, in other words, zero. Hypoxia is less than normal. Anoxia is without oxygen, zero.
Q: And hypoxia is what?
A: Low oxygen. Hypo-oxia, less than normal oxygen.
Q: And what is hypoxemia to you?
A: A measurement in the bloodstream

Page 141

of reduced oxygen.
Q: And what is ischemia to you?
A: Reduction of perfusion, the cc. of blood per minute per gram of tissue.
Q: Do you remember the last deposition you gave prior to today, Doctor, in general?
A: Let me think. Yes.
Q: Where was it?
A: In Philadelphia.
Q: Who was it for in terms of the person retaining you?
A: It was for Gerard Mitchell, Stein, Mitchell & Mezines, and it was a case of kernicterus.
Q: Pending in the District of Columbia or Maryland?
A: It must have been Maryland. I think it was like Gaithersburg, Maryland, or something.
Q: How many cases have you done for Gerry Mitchell?
A: Five or six.
Q: Do you have any other cases for

Page

Mr. Hilton at this time?
A: No.
Q: Do you have any other cases for Mr. Thomas at this time?
A: No.
Q: Do you have any requests outstanding to obtain for you additional information or material for your review in this case, Doctor?
A: Like for other medical records or something?
Q: Yes, just to get more information.
A: No. I thought I had a pretty good set of medical records.
Q: Do you have any other opinions regarding the proximate cause of Cody Miller's neurological deficit other than you've already shared with me, Doctor?
A: No.
Q: Have I asked you all that — I won't even continue that question.
A: That's a trick question.
Q: No, I am not going to do that.
MR. WALSH: All right. I'll

Page 143

take a copy of that with mustard and relish, and I'm finished.

THE WITNESS: Very good.

MR. WALSH: And I got you out of here by 1:20.

THE WITNESS: Excellent.

(Witness excused.)

(Whereupon the examination adjourned at 1:20 p.m.)

Page 144

COMMONWEALTH OF PENNSYLVANIA :
COUNTY OF PHILADELPHIA : SS.

I, Susan Marie Migatz, a Notary Public in and for the Commonwealth of Pennsylvania, do hereby certify that the witness was by me first duly sworn to testify the truth, the whole truth, and nothing but the truth; that the foregoing examination was taken at the time and place stated herein; and that the said examination was recorded stenographically by me and then reduced to typewriting under my direction and constitutes a true record of the testimony given by said witness.

I further certify that I am not a relative, employee or attorney of any of the parties, or a relative or employee of either counsel, and that I am in no way interested directly or indirectly in this action.

In witness whereof, I have hereunto set my hand and affixed my seal of office this _____ day of _____, 2001.

Susan Marie Migatz
Registered Professional Reporter

Page 145

I have read the foregoing transcript of my examination given on April 6, 2001, and it is true, correct, and complete, to the best of my knowledge, recollection, and belief, except for the list of corrections, if any, attached on a separate sheet herewith.

Date ROBERT R. CLANCY, M.D.

Sworn to and subscribed before me this day of , 2001.

Notary Public