IN THE COURT OF COMMON PLEAS

OF CUYAHOGA COWNTY, OHIO

LONNIE HURT, ET AL.,

Plaintiffs,

vs. Case No.

THE MT. SINAI MEDICAL 198452

CENTER, ET AL.,

Defendants.

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Deposition of SAMUEL J. HORWITZ, M.D., a Witness herein, called by the Defendants for examination under the statute, taken before me, Julie Gentile, a Registered Professional Reporter and Notary Public in and fur the State of Ohio, by agreement of counsel, at Rainbow Babies and Children's Hospital, 2074 Abington Road, Cleveland, Ohio, on Friday, December 27, 1991, at 8:55 o'clock a.m.

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APPEARANCES:

On behalf of **the Plaintiffs:**

Nurenberg, Plevin, Heller & McCarthy, by

MARSHALL [redacted] NURENBERG, ESQ.

HARLAN M. GORDON, ESQ.

JAMIE R. LEBOVITZ, ESQ.

1370 Ontario Street-First Floor

Cleveland, Ohio 44113

621-2300

On behalf of the Defendant The Mt. Sinai Medical **Center:**

Reminger & Reminger Co., L.P.A., by

THOMAS R. KELLY, ESQ.

The 113 **St.** Clair **Building**

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APPEARANCES: (cont'd)

On behalf of the Defendants Dr. Samuels and Dr. Demio and Emergency Practice Associates:

Jacobson, Maynard, Tuschman & Kalur, by
JEROME S. KALUR, ESQ.
North Coast Building
1001 Lakeside Avenue, Suite 1600
Cleveland, Ohio 44114-1192
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On behalf of the Defendant Dr. Washington:

Jacobson, Maynard, Tuschman & Kalur, by
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MR. KALUR: The record should reflect that this deposition is being taken under Rule 26 solely for purposes of discovery and that it's being taken by agreement of counsel as to time and place.

SAMUEL J. HORWITZ, M.D., of lawful age, called for examination, as provided by the Ohio Rules of Civil Procedure, being by me first duly sworn, as hereinafter certified, deposed and said as follows:

EXAMINATION OF SAMUEL J. HORWITZ, M.D.

BY MR. KALUR:

Q. Would you just for the record state your full name and your professional address, sir?

A. My name is Samuel J. Horwitz, and my address is University Hospitals of Cleveland, 2074 Abington Road.

Q. Dr. Horwitz, I've been furnished a document dated February 12, 1991, consisting of some 16 pages, which is ultimately signed by you on page 16. Is that your only writing pertaining to this case and with respect to a request to review matters by the plaintiffs?

A. This is the February 12, 1991 --

Q. Yes.

A. That is the only written report that -- I've got some handwritten notes for my own benefit, but nothing else that I've secluded.

Q. Let me make it simple so we don't have to ask several questions about drafting a report. I've asked you to draft reports myself before.

A. Right.

Q. This one takes a rather unusual format, and I wonder if you could explain to me how it came about that this format was used instead of the traditional letter process.

A. I'm not sure I know the whole mechanism in which this was done, but I was asked by the attorneys to talk with me about Lonnie Hurt as my patient, and I did so. And then I had not at that time undertaken an extensive review of his record, simply talked about his medical condition, and then they sent me this to complete and requested that I complete this form. I did not draft this form. I did not set it up. And I answered the questions.

Q. Was it explained to you why an authored report by you as is customary was not needed?

A. It was not explained. I assumed that it might follow the fact that I had stated that I was the treating physician and in my own mind was not an independent expert but was actually the treating physician and was going to take that kind of approach to it. Maybe that's the reason this was done this way, but I really didn't even question it. I've had these sent before, so it didn't mean anything to me.

Q. Who first contacted you for representing the plaintiffs in this case?

A. I can't remember exactly, but I think it was Mr. Lebovitz. Mr. Lebovitz.

Q. Have you had any discussions with Mr. Nurenberg before today about this case?

A. Yes.

Q. When did those occur?

A. Those occurred -- I can't tell you the exact date, but probably within the last -- I'm guessing -- about nine months or so I've seen him on two or three occasions that he's requested.

Q. Concerning this case?

A. That's correct.

Q. And was Mr. Lebovitz present on those occasions or are we dealing with separate visits here?

A. I think some were joint and there were some separate visits from him as well.

Q. Instead of my asking you a whole bunch of separate questions, would you give me the background as to how many visits there have been as you can recall and what went on at those visits, who was present?

A. I would say there have been probably three or four visits with Mr. Nurenberg and Mr. Gordon and Mr. Lebovitz and Mr. Lebovitz and Mr. Gordon maybe singlely or together maybe two or three. Some of them were very brief to show me something or bring something, but together there were probably three or four. I would say four at least.

Q. Now, your -- I'll use the term report, although it's a question and answer thing. We'll call it your report of February 12, 1991. It refers only to medical records. Have you been furnished any of the

depositions in this case?

A. No, I have not.

Q. Have you at any time requested any of the depositions?

A. No, I have not.

Q. Have you been advised orally that the depositions of the residents both in the ER and in the pediatric department at Mt. Sinai have been obtained?

A. I'm aware that depositions have been obtained because of a statement in a deposition in discussion and also in reports from expert depositions are alluded to, but I have never seen them or actually had any knowledge of specific contents.

Q. Did you get any type of a report of what Dr. Demio or Dr. Samuels had stated in their depositions about the goings-on on the 14th and 15th?

A. No report whatsoever,

Q. What expert reports have you been given?

A. I've been given a report from Dr. Chalab, a report from Dr. Blaise Congeni. There is one -- I've forgotten the gentleman's

name from University of Indiana. This morning just before we started I was shown a report from Dr. Mark Gibson,

Q. Those three?

A. I'm trying to think if there are any others. I think those are the ones. I don't recall any other reports. If there's another one, I may have forgotten it.

Q. The plaintiffs have given us a report from a Dr. Todd at Children's Hospital in Denver. Have you seen that?

A. There is one from a Dr. Rigotti, a rehabilitation person. That may have been the one.

Q. That's not the rehab report.

A, No, I've not seen this report.

Q. There's another one from a Dr. DeHart at West Penn Hospital in Pittsburgh.

A. I've not heard of him. I've not seen that.

Q. Dr. Raff, R A F F, from University of Louisville?

A. Never seen it. Don't know him. There was one other -- somebody. I've forgotten his name. Maybe Matthews.

Q atthews is the rehabilitation

A. Yes, I saw that one.

Q So you have this report from Dr. Calabretta that you've been given?

A. Yes.

Q. May I see that, please?

A There is also -- I ran through a report from a -- addressed to a Mr Kalwer from a Michael May.

Q. So you've seen Dr. May's report?

A Right I think those are all that I have been given.

Q. So you have an opinion in this case that there was a herniation of the brain stem?

A. I have an opinion that there was a herniation of the brain, which involves the brain stem, not specifically the brain stem.

Q. When did that herniation occur in your opinion?

A. The herniation was -- I can't tell the specific time when it started, but its manifestations are observable when the pupil failed to react. Fixed pupil on the right side. That's the time when it was clinically manifested.

Q. There are all kinds of observations about the pupils and dilatation of the pupils in this case. I wonder if you could look in the medical records and tell me the first time you feel that there were clinical manifestations of brain herniation.

A. Yes. I just want to be specific about looking at two areas. The earlier record that I can find right now and that I have noted is the physician note of April 15, 1990, 7:45 p.m. That's 19:45 hours and that is the note that records the pupillary abnormality.

Q. I see in that note writing that indicates eyes right pupil dilated. I can't read what it says.

A. To six millimeters.

Q. To six millimeters. Left pupil dilated to approximately five millimeters.

A, Yes, That's not able to be reactive to light. That's correct.

Q. Of course what preceded what I read was fontanel flat.

A. Yes.

Q. And then it says funduscopy. No papilledema.

A. No papilledema.

Q. What in your opinion was causing these clinical signs -- this clinical sign or signs -- strike that.

Is there anything else in what we've just read other than the dilatation of the pupils that indicates to you that there clinically was evidence of herniation as of 7:45 p.m. on 4/15?

A. Specifically of herniation that's it at 7:45.

Q. Is there anything in the findings that we've just gone through that indicates that is a negative for an indication of herniation starting with fontanel flat?

A. No.

Q. If, in fact, this clinical sign of herniation of the pupils as exhibited in this progress note did indicate a herniation, do you hold an opinion as to anatomically what portion of the brain was herniated?

A. Well, the portion of the brain that would have to be herniating would be the cerebral hemispheres pushing down and compressing the brain structures down to either

compress the stem or compressing the third nerve because right now all you can see is the third nerve function involved. It can either be the core going down or it could be the stem or it could be the cerebral hemispheres actually compressing the nerve itself.

Q. So one couldn't tell from this whether it was, for example, an uncal herniation or a tonsillar herniation?

A. You can't -- well, it's not tonsillar herniation at this point. But uncal herniation versus a central herniation -- it would be difficult to tell, Uncal herniation is usually pretty unilateral to start with and sometimes they combine, but right now it's difficult. Not enough there.

Q. What clinical signs after this 7:45 p.m. entry and up until the time of transfer to UH indicate to you or do you find supportive of your conclusion that there was a herniation?

A. The subsequent findings. And I'll give you -- we would have to go back to the nurses' notes, The sign is what they called a form of Cheyne-stokes breathing and then

qualified that. I'll try and find the time when that was recorded.

Q. 2:25 a.m. there's an entry of Cheyne-stokes breathing I see.

MR. GORDON: And 1:30.

MR. KALUR: Yes. Mr. Gordon is right. 1:30 a.m. I see one.

A. Yes. And that is the 1:30 a.m. respiration 48 with respiration similar to Cheyne-stokes without apneic episode. That's the one.

Q. That's another symptom that would indicate to you herniation?

A. That is correct.

Q. So we have one at 7:45 p.m. related to dilatation of both pupils and then at 1:30 a.m. Cheyne-stokes breathing reports begin. Any other signs --

A. Well, it's not really Cheyne-stokes, but something similar.

Q. What the nurse records as that anyway.

A. Yes.

Q. Anything else in these records at Mt. Sinai that indicates to you from a clinical

perspective that herniation was or had occurred?

A. No. Those are the specific ones.

Q. Do you hold an opinion as to what was the causative agent for herniation in this case on the evening and early morning of the 14th and 15th of April, 1990?

A. The causative agent in the widest sense is meningitis -- intercranial infection.

Q. What is the pathophysiology of that process, if you can tell me?

A. I don't think the pathophysiology is totally clear. There are a number of factors that get involved. One is the inflammatory process affecting blood vessels, the inflammatory vessel affecting the superficial layers of the cortex, disruption of blood brain barrier, disruption of tight junctions that increase permeability, possibly release of "toxic substances" from bacteria or from destroyed white cells. Those are all facets that lead the brain to swell.

Possibly in addition there may be a factor of increased production of, cerebral spinal fluid further increasing pressure or

obstruction to flow and absorption of cerebral spinal fluid increasing the intercranial pressure, and then there is the presence of extra cerebral fluid effusions which are occupying space. In this case they appear to be subdural effusions of fluid more than likely.

Q. Subdural effusions?

A. Effusions, yes, all of them occupying more space in the head. It's a complicated mechanism, but I don't think anybody really knows why you get all of the swelling that you do except you get it.

Q. Would some of the inflammatory response in your opinion be due also to a reaction of the killing mechanism between the antibiotics and the bacterial cells?

A. That's probably correct as one of the mechanisms except you can see it without that, and being specific about this case the pupillary inequality at 7:45. At least it's noted there. This had to have taken place in a period prior to that to do this. It doesn't usually happen in five seconds as far as we know.

And antibiotics were standard around about that time. We can look at the exact time, but it's in the region of seven something. So for the antibodies to absorb and get there and do that it could be a factor, but I think the process is already there before that factor, but I can't exclude that the body's antibodies are just sitting there.

Q. It could be a supplemental factor?

A. It could be. That is correct.

Q. For example, we might have the pupillary response to an inflammatory process going on and then as the antibiotics were increased in frequency we could have other manifestations of herniation develop?

A. Well, I'm going to be clear that the pupillary response -- and maybe you could just read it. Pupillary response is not the response to inflammation. It's the response to pressure.

Q. The pressure is from an inflammatory response, isn't it?

A. That is correct, but it's not inflammatory reaction in the pupil or its nerve. That's why I'm trying to be very

specific.

Q. I didn't mean to imply that.

A. I know, but the way the question came I wanted to be sure.

Q. Could we then have a supplemental increase in intercranial pressure from the killing process of the antibiotic and release of endotoxins?

A. Yes. I don't know that's all endotoxin, but release of substance.

Q. From the killing process?

A. Yes.

Q. When you're using the term herniation, are you referring to the protrusion of brain tissue through the bony area which is at the base of the skull?

A. No. That's tonsillar herniation.

Q. Maybe you could tell me exactly what you're referring to.

A. This is transtentorial herniation.

Q. And is this process of herniation when it occurs a permanent one or does it recede when the inflammatory process recedes?

A. Let me put it this way. It doesn't usually -- if you don't treat it, it can't

recede because you die. Okay? So of itself you're unlikely to survive long enough for it to recede, but having been treated it certainly can recede and will recede if you survive.

Q. The treatment is by the giving of Mannitol or other antiinflammatory drugs?

A. Its treatment is administration of Mannitol, other drugs if necessary, and the use of hyperventilation.

Q. When in your opinion did this child first require the administration of Mannitol and hyperventilation?

A. When do I think it required it?

Q. Yes.

A. 7:45 p.m.

Q. Do you hold an opinion that standards of appropriate medical care -- we'll put aside what happens before 7:45, but let's assume that one had the information available in the chart -- a reasonably prudent physician had the information available in the chart as of 7:45 and the information from seeing this child that's made known to us here. Would standards of prudent care require that Mannitol be administered at that time and

hyperventilation by ambulance to this child?

A. It is my opinion that at 7:05 when the pupils were unreactive that the physician or physicians taking care of the patient need to make an immediate determination as to why those pupils are unreactive and that is there is -- I would see even a remote likelihood or a reasonable likelihood that the pupillary nonreactivity is due to rising intercranial pressure and potential herniation, Mannitol and/or hyperventilation or one of the other measures should be given immediately -- immediately on an emergency basis. So if, but or maybe.

Q. Does that mean in this circumstance with what was known here it should have been given?

A. In this circumstance it means that it needed a minute or two at most to assess whether this was the situation and then it should have been given immediately, so I would say 7:47 if this is recognized at 7:45. Two minutes was enough. I'm being arbitrary with two minutes, but what I'm trying to say is it should have been given right away.

was given earlier than 5:45 a.m.?

A. I have reason to believe that the note was written at 4:45.

MR. **NURENBERG:** 5:45.

Q. 5:45.

A. I'm sorry. Did I not say 5:45?

MR. NURENBERG: **You** said 4:45.

A. I have reason to believe no later than 5:45 when the note was written, but knowing the way this works in an intensive situation it could have been given at 5:20 and at 5:45 the nurse wrote it down, I will accept it was 5:45 because that's the time it was written, but I know the way this works. It was not done later. That I know for sure.

Q. The Rainbow transport team arrived at 1:55 a.m.

A. Yes.

Q. There was a little elevator traffic then. The child was brought back off the elevator. Nurse claims more Cheyne-stokes breathing as she interpreted it. At 2:30 there's a recording of the child being given Valium, intubation by the RB & C doctor, bagging, and then at 2:45 the child left via

stretcher and presumably I guess we could figure 15 minutes to get over here.

A. Longer than that by the time you drive, park and get through the tunnels and get up here.

Q. 3:15 the child is in the hospital. Why did it take a couple hours over here? Don't tell me Rainbow was below the standard of care.

A. Are you asking should we have given the Mannitol here?

Q. Yes.

A. Definitely.

Q. Does the Mannitol operate in these types of situations in a relatively rapid manner to reduce intercranial pressure?

A. It does.

Q. Is there a time which you would consider to be significant to reduce intercranial pressure in order to avoid morbidity in cases of pneumococcal meningitis?

A. I'm not sure I understand the question, Could you repeat it?

Q. Let me try to rephrase it in a clearer form.

If I document -- what I'm getting at is I take it you believe that increased intercranial pressure from the inflammatory reaction to pneumococcal meningitis requires relief as rapidly as possible.

A. That's correct.

Q. And is there any type of a correlation between how long the herniation exists and the sequelae that will result?

A. Right. That's a very good question and --

Q. It took me two times.

A. And earlier you did discuss pneumococcal meningitis. I'm just going to say this statement applies to pneumococcal meningitis as well as to any other context of raised intercranial pressure and a fixed pupil and that is there is no experiment done in humans or indeed in animals that I know of in which you can set up a specific level of herniation and then watch it for X period of time and see what your outcome is. No one could do that experiment, so we can't give you high figures from literature that anybody can say I sat on a herniation for an hour and guess

what. The mortality was twice as high. You can't do that.

So what we have is clinical observation and the writings of people who have learned that herniation is a disastrous situation and personal observations,. and those are if you're going to get recovery, you must treat very, very quickly. When you go beyond a period that extends in an acute situation like this, that extends beyond minutes, you're running into a serious question whether you have any recoverability.

From what I've seen in treating cases my opinion would be that if you have fixed pupils in a meningitis like this and you go beyond 30 minutes, it's extremely doubtful that you are going to get a favorable recovery. If you go within several hours, the chances are close to negligible that you're going to get any meaningful recovery. You can't say you'll throw in the sponge at 20 to 29 1/2 minutes. But once you go beyond minutes and into hours,. it's nonmitigated in my experience and most people feel that way. It is a red flag -- a big red flag.

Q. You used one term. You say when the pupils are fixed --

A. Yes.

Q. So we're not passing in the night here, at 7:45 they're referring to pupils dilated.

A. And fixed. Well, they're talking about nonreactive to light. That's the same term. And then later, just so we don't have to go through this, there are questions that the left pupil is reactive and it's not reactive, but the right is fixed. And actually somewhere in the nurses' notes I think they're transposed, but that doesn't mean it's a fixed pupil. That's the point. Nonreactive to light. That's the observation.

Q. If the observation that the pupil or pupils did not react to light at 7:45 is taken with the right pupil dilated to six millimeters and the left pupil dilated to five millimeters, if I understand what you're saying, within 30 minutes after that time it would be extremely unlikely that you are not going to have severe neurological damage?

A. Yes, in this situation.

Q. Do you hold an opinion as to whether or not the lumbar puncture that was done in this case was in a direct cause and effect relationship to the herniation?

A. I have an opinion.

Q. What is that?

A. My opinion is, firstly, the lumbar puncture should not have been done at that time. Secondly, that the lumbar puncture certainly at that time did not do the patient any good. Thirdly, that I do not know how much it contributed to further compromising the patient. I don't know how much harm it actually caused. It certainly didn't do good and I wouldn't have done it, but I don't think that it specifically harmed it. There's no way to measure that.

Q. I think what you're telling me is that you don't have an opinion as to whether that precipitated the herniation or whether it would have occurred anyway.

A. No, no. The herniation was there. The herniation was there. I'm just saying I don't know that it further aggravated an already bad situation. I can't tell you. I

wouldn't have done it

Q. Would that be speculation to tell if it aggravated an already bad situation or not?

A. It would be speculation. On general principals it shouldn't have been done Whether it actually hastened things, I think you can't tell. There's no way of quantifying that.

Q. I think you've probably had a chance to look at the lumbar puncture results.

A. Yes.

Q. In this instance the protein and CSF was listed as 293 and the glucose as 18. What significance are those findings to you in this situation?

A. Would I just check? Was it 18 or 13? I want to be sure.

Q. It looks like 18.

MR. POLITO: It's 18.

A It s 18. Okay. That's fine.

Q. Can you tell me what significance those findings are to you other than the obvious indication of meningitis?

A The protein elevation as such is

abnormal. It indicates that there is some process that is resulting in increased exudation of protein into the spinal fluid. Of itself it's abnormal, but it doesn't tell me what the abnormality is. The low sugar is clearly abnormal and in the context of just the sugar it could have any number of meanings except it's abnormal.

Q. How about in the context of the comparison of the protein level?

A. There's a pathologic process going on around in the meninges. Of themselves those two things do not -- I could give you a long differential diagnosis. Given those two things there are lots and lots of diseases that could do them.

Q. Let me be more specific as to where I'm going. The 18 would you agree from your clinical experience with meningitis is a rather low number?

A. You mean --

Q. If we take 100 children who have meningitis -- bacterial meningitis -- and look at their initial lumbar punctures, where would 18 fit in? Low? High? Medium?

A. I don't know any specific figures of how many are at 18, but it's common enough to say that that's, you know, everyday -- it's everyday with meningitis. It can be lower than that. It can be higher than that. Of itself it's just -- it's helpful in making the diagnosis. No more, no less.

Q. Any prognostic significance to these type of values with meningitis?

A. There are people who have said that if it is lower, it may be worth prognosis. Others say if it is higher, it may be worth prognosis. The fact is there are so many variables in this particular equation that it is a very, very limited value.

I don't know any of us here who would go out to a parent or even to a colleague and say because this is 18 this is what's going to happen. I'm just going to say the kid has got meningitis, and we do not use that in any major interpretive way.

Q. Let's shift away from the glucose and protein. Isn't -- maybe I'm not going that far away from it. Isn't the degree of leukocytic response in a CSF indicative of a

child's ability to ward off the disease on its own?

A. No.

Q. In a child who is less than six months old do they have less of an ability to ward off meningial infection than a child of an age greater than six months?

A. In general the ability to resist infection is better after six months because of a number of factors. In a general sense you tend to do better, but six months is not some magical figure. The much more magical figure is the first six to eight weeks of life. I think if I had meningitis, I'd rather have it at a year than five and a half months. It's not a big difference at that point. The big difference is five and a half months and five days.

Q. What about white blood cell count from serum -- white blood cell count in response to bacterium? If the white blood cells don't rise to high levels but stay normal, is that any reflection of immunological response of that individual?

A. It may indicate either overwhelming

infection or it could indicate that the individual doesn't have a good reactivity to the infection.

Q. If it were overwhelming, wouldn't you expect to see actually a low white blood cell count from serum?

A. It may be low. It may not rise. I think I would worry about the fact that it doesn't go up. This infant isn't reacting very well.

Q. I believe this child had -- I believe the first time it got a CBC over at Mt. Sinai it had, if my memory serves me right, 5,400 on white blood, which is in normal range, and that's taken on the 15th.

A. Yes.

Q. Does that tell you anything with respect to what you believe would have been the white blood cell count if it had been taken on the 14th?

A. That would be speculative. I really don't know that at all.

Q. Making a diagnoses of otitis media is not really part of your present job description, is it?

A. Well, I don't know that anybody has written a job description saying I can't diagnose.

Q. I didn't say can't. You might be pretty good at it, but that's not the thing you usually do here, is it?

A. Yes, I do.

Q. You pick it up occasionally?

A. I see children everyday, and I see they have ear infections all the time when they come to my office because I'm seeing them for seizures and mom says he's got a runny nose and is tugging at his ear and he's running a low grade fever, I'm not going to say I -- I look at the ear and if it looks infected, I call the pediatrician and tell him what's there and he would follow it up. I do it quite frequently. It probably happens several times a month. I look at ears all the time, but I don't actively give the antibiotics and follow up as if I'm an everyday treating otitis doctor. I'm not.

Q. Since you have been given depositions in this case to review, I'm going to ask you to assume some facts as true so I can fully explore your opinions in this case

and understand them when you mount the witness stand.

I want you to assume that when Lonnie Hurt first presented on the 14th to Dr. Demio in the emergency room that he examined the child's ears and found them to exhibit a dull redness or at least the tympanic membranes to exhibit a dull redness and that he considered them to have very slight abnormalities but not to be indicative of an ongoing infectious process but rather to be indicative of a resolving otitis media that had undergone a full course of antibiotic therapy.

I want you to further assume that that view was concurred by the attending -- strike that. By the resident who saw the child that day.

A. Dr. Demio is not a resident? I don't know who Dr. Demio is.

Q. Dr. Demio is the attending ER physician, my client in this case.

A. Okay.

Q. And the next day Dr. Samuels was the attending ER doctor and he examined the child's ears and also concluded that this was

not an active ongoing otitis media but was also the result of a resolving otitis media and that Dr. Demio on the first day included within his differential -- didn't write it down -- that this child had been subject to a viral syndrome and that had caused the elevation in temperature that had been noted but was not noted at that level at the time the child was in the ER on the 14th.

Just assuming those facts and that those three physicians determined that this was not based on their examination of the ears an ongoing active otitis media situation, does that information alter your opinion that there was bilateral otitis media in this child?

MR. GORDON: Objection. Go ahead.

A. The written -- it's a difficult question to answer right now.

Q. I try and give you difficult questions.

A. I understand The written description they have in the record as I had read the description of the ears to me would have indicated active otitis If they would say that they really looked at it and it

doesn't look the way they wrote it, they were there. I was not there. It doesn't correlate in my opinion with what were the only written notes I have on the child, which sounds to be more in that context, However, I'm the first to acknowledge that interpreting ears is difficult in the best of hands. Given six good pediatricians it's not always easy to see what one calls dull redness as opposed to bright redness in the ears, which is a common infection. The ears are the hardest things to look at, and I accept that. It is difficult to interpret.

So it's a matter of judgment at that point. Explanation of a viral infection is in the differential diagnosis. It is never in the record that that was considered from what I can see. The diagnosis that I see is Candida infection, and Candida will not cause the patient's symptoms, his reported temperature elevation at home, his marginal temperature in the emergency room albeit after a dose of Tylenol. So neither would -- well, I'll just leave it at that.

Q. Let me focus on what's not in the

record but what is in the depositions.

First of all, even what's in the record there is no diagnosis of otitis media on the 14th; is that right?

A. That's correct.

Q. What we do have is an observation in that record written by the resident and not by Dr. Demio that he saw a redness in the ears and that I believe he saw a less than clear landmark, I believe, Are we in agreement there?

A. That's what he said.

Q. Now, I want -- if, in fact, there was then another examination by an attending -- an experienced attending physician who at that point found a dull redness in the ears but found no other significant abnormalities and concluded from his examination and based on his experience that this was a resolving otitis media and he also had the history from the mother of the course of antibiotics that had been concluded -- if you assume those facts to be true, do you hold an opinion that Dr. Demio under those circumstances with a conclusion that he had reached, although not written in

the chart, of viral syndrome was below the standard of care in releasing that child that night without antibiotic care?

MR. GORDON: Objection.

A. I don't have all those facts written in front of me, but as I pointed out it is a judgment of the physician who looks at the ears. He saw them. I assume he didn't write it down, but assuming he did do it and he saw them he had a decision -- a judgmental decision to make. And what I'm saying is the record doesn't reflect his decision. It also doesn't reflect an alternative diagnosis that shows us that the child was sent out with a consideration of what the actual diagnosis was beyond thrush.

So he made that judgment. If indeed he saw there was -- that this was incorrect and the ears unequivocally in his opinion were not infected, then he could conclude two things. One, that it's not the ears causing the fever -- the recorded fever. And, two, I think he would have to conclude that it's not the oral thrush causing the fever. Then he would be left with a fever as

yet undetermined and you would have a differential diagnosis of what that fever is including viral infection, and he may decide that that's the most likely depending on all those findings. If that is indeed true, it would not be unreasonable.

Q. Now, we're using the term fever here and I'm --

A. Yes.

Q. Is that a term you usually use in talking to your medical colleagues or just with us poor lawyers here today illiterate medically?

A. Their temperature elevation.

Q. Afebrile?

A. Febrile, afebrile, pyrexia if you're in the British world and occasionally if you're in the American world.

Q. We had a rectal temperature in this child of 100.2.

A. It's .6.

Q. It might be .6.

A. 100.6.

Q. 100.6 in the ER on the 14th.

MR. GORDON: Triage nurses' notes.

Q. Is an elevated temperature in a child who was this old having been born at about 36 weeks gestation -- isn't an elevated temperature in these children considered to be 102 rectally?

A. That is usually considered to be a fever. That's close to what you're saying, and as I pointed out this particular fever is in the marginal range. Most babies, if you look at them, are not shooting up to that level. If they're up there, you look at the baby and decide. You also have to take the factor into account this is a moment in time to be very specific. This is a moment in April. We don't know how cold it was. You take the child outside. Temperatures often drop. This baby was given Tylenol.

So all you can say is the temperature at the time recorded is certainly not a rip-roaring temperature. I would agree it is at the stage where you would note that it is there and you wouldn't be horrendously alarmed by that as a measurement, but the history is much more significant.

Q. For example, in the literature that

I've read I've seen references to whether antibiotics should be initiated on the suspicion of a bacteremia in infants and I've seen the figure of a fever over 102 and a white blood cell count of 15,000. Do you subscribe to that view?

A. I will tell you this. That I have -- I subscribe to the view that every month I ask what's new because this has changed so often from my infectious disease colleagues. I think everybody is having a hard time knowing what the appropriate criteria is for treating possible bacteremia and the changes. I do not know what it was specifically in 1990 at that time and even from place to place it has varied, and I think although people set these limits, it becomes a matter of judgment. Their practices have changed and changed and changed over the years with us.

Q. A few moments ago when I asked you about the temperature of 100.6 you said that under those circumstances when it's not rip-roaring I think was the word you used, you have to look at the baby and decide. When you say look at the baby and decide, what do you

mean?

A. What I meant is you have to look at the baby and that you have to take into context the whole infant's clinical picture. One,, its past history. Two,, the history of the complaint, what the mother brought the baby in for, the circumstances surrounding the arrival, prearrival what's being done, what's being treated and your examination of the baby. All of those are factors in making a decision. And obviously you have guidelines, but you can't make specific rules for each of those things. It depends on taking the global picture of what you're going to do with that.

Q. Within that global picture of looking at your examination of the baby is the reaction of the baby as being alert and active significant in that evaluation process?

A. Everything is significant.

Q. Does that have a particular significance to a pediatrician to find an alert and active child --

A. Absolutely.

Q. -- under these circumstances?

A. Absolutely.

Q. Would that be the type of circumstance which would weigh in the process of looking at the temperature of 100 F and determining whether it is, in fact, related to an ongoing process that requires antibiotic care or not?

A. It would all weigh in the alertness, again, taken in context with the whole picture -- what you heard of the past, what you're seeing now, the drugs that were given -- it's not one factor, but obviously a physician is going to be influenced whether a child is alert, you're completely out of it or nonalert. I mean those things are part of the judgment call.

Q. I want to shift now to the next day I'm going to go to the emergency room, but I want to jump back again to our herniation area because there's something I forgot about.

The chil was supposed to have had seizure like activity in the early morning -- actually on the 15th just before a transfer Was that also related to an ongoing herniation process in your opinion?

A Seizures per se are not a symptom

of herniation, so the seizures are not caused by the herniation process. That's not one of the symptoms.

Q. Would inflammatory process as it relates to the meninges stimulate a seizure in some instances?

A. Absolutely. I mean 20 to 25 percent of infants with meningitis are going to have seizures, but it's the same process causing both. The infection leads to the swelling in herniation. The infection leads to the inflammation and seizures, But the swelling and herniation does not cause the seizures.

Q. I see what you're saying.

What about this process that the mother describes at approximately, according to her, 6:00 a.m. in the morning on the 15th of the eyes rolling back in the head? Do you have an opinion whether that was a seizure like activity in this child?

A. I have an opinion.

Q. What is that?

A. My opinion is that it was a seizure.

Q. Is that a so-called febrile seizure or would it be related to the inflammatory process for meningitis?

A. Febrile cannot be -- let me back up. Febrile seizure by definition is a seizure provoked by fever in the absence of intercranial infection. Once you've got infection in and around the brain, you'd be smarter than Solomon if you figured out it was the high fever and the infection of the brain is causing the seizure, but nobody even thinks about that because you assume it is the direct meningitis effect causing it. The fever is such -- is extremely unlikely. So as long as it's not a febrile seizure to make it simple.

Q. Therefore, you conclude that meningitis existed as of 6:00 a.m. on the 16th in this child?

A. Yes.

Q. Do you hold an opinion as to what was the earliest that the meninges were invaded by the infectious process in this child?

A. I don't know how you can answer that specifically. None of us can time that, but you know it was there at 6:00 a.m. because

the child was seizing already and the ongoing course is very typical. You can go back in time, but I don't think you can be sure. I mean I can't tell you it was there at 4:00 a.m. I can't tell you it was there on the 14th when they went to the emergency room. 1 can't tell you it was not there on the 14th. There's just no way of knowing the moment it's starting and proliferating.

It could have been the 14th. It's pure speculation to say it was there, and I'm not going to do that.

Q. That would be speculation one way or the other on the evening of the 14th whether this child had meningitis?

A. It's pure speculation. I'm not going to even hazard a guess because there's no way I could know that.

Q. Is it fair to assume that by the time meningitis reaches the stage where the inflammatory action is going to lead -- can lead to a seizure that it certainly has been -- there's been an invasion of the meninges that's at least taken hours as opposed to minutes?

A. I don't know that, I really don't

know. It just can't be done to actually figure that out, I can tell you some anecdotal experience only to say we have seen children come in with a sudden fever and have a convulsion and we have tapped them and the fluid looked clear and we have assumed it was a febrile seizure and then to our horror 12 hours later the spinal fluid is growing out bacteria and we've treated the child for meningitis and it was clear that at that point the child actually had a seizure before you even saw cells or low sugar.

So experientially we've actually seen that kind of thing, but to ask you at what moment it gets into the meninges to get the seizure, you can't tell. There's just no way you can ever know that.

Q. It leads me to another question. You just gave us some anecdotal experience. Have you had children -- infants. We'll stay with apples and apples here.

Have you had infants who have come in the first time with a report of a seizure at home and you do a tap and you find meningitis and you initiate promptly antibiotics who

despite that go on to suffer devastating neurological injury?

A. I have had that circumstance,. yes.

Q. And you had, I take it, the opposite, too, where you intervened with antibiotics after the seizure was first reported and the child went on to have a normal sequelae.

A. Yes.

Q. The sequelae were normal?

A. That's the majority, yes.

Q. Why does it happen one way in one case and -- strike that, Let me try to phrase it in a more definitive manner.

What differentiates those children? Why does some proceed onward, although they both had a seizure before care was initiated, one to do well and one not to do well-- an infant?

A. There would be several factors that could be involved. The first would be how long prior to starting treatment the child was seen and diagnosed. In other words, how many days of illness prior to the seizure. So the actual duration of infection is going to make a

difference.

The child may be at home for three days before the first seizure. That's going to make a difference with fever I'm talking about -- an illness. It's going to be very different with a child who went to bed around midnight, started crying and had a seizure, went to the hospital and got treatment. The time of diagnosis and treatment is very, very important. That's number one.

Number two, I think it depends on the organism. They are more or less virulent organisms.

And, three, it's going to depend on the resistance of the host. If it's a healthy child versus a chronically sick child or the immunologic ability, that's going to make a difference.

And, fourth, it is going to be probably the volume of infection that enters the spinal fluid where one gets massive infection right away and the other one gets a more gradual or less heavy load of infection.

And then there are individual factors very hard to understand like given two

children with the same duration of fever and the same time of seizure and one swells like crazy and gives you all sort of problems and the other one looks like how could train, you know, a nonskilled person in using antibiotics and they leave the hospital in ten days like it was a trivial disease I really don't know why those two differences No theory as figured out.

We know that pyrogens gets some swelling, but why one goes pervastatingly into herniation and does it fast and the next one never even approaches it, I can't tell you The factor of treatment is important, but it happens irrespective of that.

Q. With respect to Lonnie Hurt can we agree there was no scientific basis for determining when he first developed a bacteremia or meningivis?

A. We don't know when he developed his bacteremia or meningitis. There's no way of giving a specific time on that.

Q. Is there any way to determine his specific ability -- strike that.

Is there any way for you to give us your opinion with respect to the virulence of

the organism that he had?

A. I can give you opinions. Pneumococcus is a lousy organism, but nothing in the spinal fluid is a good organism. It's all a relative thing. And it is a virulent organism. It's a bad customer.

Q. What can you tell us specifically about Lonnie Hurt's individual ability to resist, in other words, his host's response to infection as compared with other infants of his age?

A. I don't know that I can give you anything specific about that. I mean he mounted reasonable normal responses. He put cells into the spinal fluid. He showed the elevation and protein that whatever the mechanism is, he had that. He dropped his sugar indicating the transport system was there. He did not generate a good leukocyte response in his blood, which might indicate either he had a very high fulminating infection or he didn't mount a good resistance. That's as much as I can say about him as a host.

Q. What about the volume of the inoculum of pneumococcal bacteria in his CSF?

Is there any way to compare that or to tell how much he received?

A. Not specifically that I know.

Q. NOW, turning to the 15th again I'm going to ask you to assume some things that are not in the record but I believe will be in evidence in this case and I want to see what your opinions are in respect to that assuming these to be accurate and true as supplemental to the record. That Dr. Samuels, who is the attending ER doctor on the 15th, first saw this child somewhere between about 1:40 p.m. and 2:00 p.m. and that before he saw the child Dr. Klein, who was the resident in the ER at Mt. Sinai, had already seen the child and the child had been seen by a pediatric resident in a consult also before Dr. Samuels, the attending, arrived on the scene and that Dr. Samuels was advised by Dr. Klein, the ER resident, that a decision had already been made to admit the child to the hospital and to do a workup in light of the child's presenting symptoms and the history of the eyes rolling back as given by the mother and that Dr. Samuels as the attending and based on the known

protocol in those situations presented that a lumbar puncture would be done on the pediatric floor within a very short time after transfer and administration and that antibiotics would also be started as soon as the IV could be started and that they were having trouble starting it in the emergency room and that, in fact, he learned the next day from Dr. Klein that the pediatric resident had, in fact, promised to do a lumbar puncture as soon as the child hit the floor just as Dr. Samuels had anticipated would be done.

Given that additional scenario of facts to be true and in light of what you have read in the record, do you hold an opinion in this case as to whether Dr. Samuels fell below the standard of care in now performing a lumbar puncture in the ER or starting antibiotics in the ER?

MR. GORDON: Just note an objection.

A. You've given me a complicated chain of command, and I'll answer it very simply Given the child's presentation a lumbar puncture should have been done as early as was

possible and start antibiotics. The decision of whether it's done in the emergency room or the child is whisked to the floor and it's done five minutes later is an internal arrangement. I'm not going to say what I am used to, what our hospital does, because it might be in a given situation one place is more comfortable than another in doing it.

Five minutes is not the issue of life or death. Hours are unequivocally unacceptable, so we are talking about do you do it right away or you do it upstairs. Now, if somebody told me today that we do it in the emergency room but they're overloaded and can we run the kid up to the ward and get moving, I'd say get the fastest way to do it. I don't care where you do it. Just do what you have to do.

So the answer to your question is the spinal tap should have been done at the earliest possible opportunity and their internal arrangement is mt. Sinai's internal arrangement. It should have been done, period.

Q. So it wouldn't be unreasonable under those circumstances for Dr. Samuels to

rely on the protocol that this would be done as soon as possible up on the floor, would it?

MR. GORDON: Objection.

A. I don't know what he relies on and what the circumstances are. I'm simply saying get whatever arrangement they made to do the spinal tap and it had to be done there and then an antibiotic started if appropriate.

Q. I understand that. I certainly don't think there will be any disagreement with that among reasonable people. The point is your report seems to imply based on reading the medical records alone here that not doing the lumbar puncture in the ER by Dr. Samuels was below the standard of care, and what I'm getting at is if we add these other things to it, is that below the standard of care for Dr. Samuels?

A. If you gave me the added things that an arrangement be made to do it very, very promptly -- and I'm being very, very specific. I'm not taking three residents examining him and two medical students and a nursing report. That's not appropriate. But if he had been whisked to the floor and done by arrangement, I

have no problem with that as long as we are very specific that it's minutes of difference

Q. Does your conclusion with respect to reasonableness also apply to starting an IV as soon as possible on the floor when they race into difficulty in the ER and wanted to get the child up?

A. It is exactly the same thing. Antibiotics should have been started at the earliest opportunity. Whether it is three minutes' difference is irrelevant to me. Just get it done.

Q. The antibiotic that was actually prescribed probably for the wrong reasons in this case was Cefataxime. Was that an appropriate antibiotic to prescribe for meningitis -- suspected meningitis in this cse?

A. This was Cefataxime.

Q. Yes.

A. I will only tell you that I do not prescribe the antibiotics at the present time and that you would have to look at what was recommended in 1990 at that time and what the resistance organisms were and decide whether it was appropriate, and I'm not going to address

it. I really don't do it, and I'm not going to address it.

Q. Do you have an opinion as to if, in fact, the order had been written stat. and the drug had been administered on a stat. basis, let's say within 15 minutes after the order was written when the child hit the floor, whether we would have a different outcome in this case?

A. I have an opinion.

Q. What is that, sir?

A. My opinion is that the outcome to a reasonable degree of probability would have been better than it is now.

Q. Can you quantify that opinion to the extent, €or example -- and I'm not trying to be facetious at all, but I'm trying to put it in medical terms. Could Lonnie Hurt write with a pencil, for example? Or obviously I know that one, but can you quantify it in any physical parameters of improvement over what he has now that care has been initiated within a half hour of the child reaching the floor?

A. No, I think it would be scientific speculation to even begin to do that. I can answer it to say that every single person who

treats meningitis or teaches on the team says the earlier you treat it, the better the outcome. And everybody recognizes that each minute that ticks by becomes precious. Nobody says you've got to go crazy in one minute, but hours make the difference in most people's opinion.

Again, I will not quantify because there's no scientific evidence in which you can ever do the study. So we just know from clinical observation the longer it goes, the worse the outcome is. The worse patients are the ones that have had the longest delay before treatment. That's a general sense. It's intuitive. But percentages -- there's no way I can answer that in a specific way.

Q. You seem to be saying to me that the shorter delay between disease onset and administration of antibiotics, the better the neurologic outcome; is that right?

A. With a few exceptions, yes.

Q. But if one takes the whole spectrum of neurologic normalcy down to the other side of the coin of basically Lonnie Hurt's condition as it exists today, you can't

quantify anywhere in a time line whether he would have any specific defined improvement in his condition by earlier care?

A. You can't define exactly what he wouldn't have had as a complication because things happen, but you can only find in a general way that everybody will agree the results of that are better. But to say one walks versus nonwalk, you can't do that.

Q. And if I understand what you're saying correctly, you don't hold an opinion that even if he had received the drug that was prescribed in a prompt manner after admission that he would be normal today?

A. You can say that --

Q. I always hate that term normal.

A. If he had received the drug in -- I'm thinking of the word. Not rapid.

Q. Expeditious?

A. Expeditious manner and all other management had been carried out appropriate to his condition, then to a reasonable degree of probability he would have been normal or certainly more normal than he is today. But you can never say that one would be guaranteed

normal with meningitis. I mean that's foolhardy. We know that's not true. How could you?

Q. But your conclusion is probably that if -- I think he was admitted about 3:00 in the afternoon. Let's say by 3:30 he got his first dosage of this drug Cefataxime.

MR. NURENBERG: I don't think so.

MR. KALUR: I said if he had.

MR. NURENBERG: I'm sorry.

A. Yes.

Q. That in all probability it would have turned out with proper care to be normal; is that right?

A. Yes.

Q. Now, that probability -- can you quantify that down to a percentage basis?

A. I can qualify it down to a percentage basis in a very general statement. If you take infants who have meningitis with a common organism like pneumococcus and the diagnosis is made reasonably promptly and treatment is instituted promptly and complications are handled expeditiously, the mortality rate is one probably no more than a

couple of percent, that the morbidity rate long term is probably in the region of 12-15 percent depending on the series; that in that morbidity rate there is a spectrum from very mild morbidity to profound morbidity, but that the profound morbidity is more probable to occur in infants who have had delay in diagnosis, delay in therapy or the delay in management complications.

Again, it is not absolute, but those are the percentages. You're going to take the adequately diagnosed and adequately treated and at least 80 percent could be expected to come out normal.

Q. Is that 80 percent figure based on your own personal experience or a combination with literature?

A. In combination with modern literature our own experience. I'm being very specific that we are excluding newborn infants. I want to make that quite clear. We're not talking about the first month of life. That's a totally different problem.

Q. We're talking about a child of the approximate age of Lonnie Hurt.

A. That's correct.

Q. And so I'm clear your conclusion of probable normalcy in this case is based upon statistical experience but is not related specifically to Lonnie Hurt, is it?

A. It's related to two factors. One is statistical experience and, secondly, experience from myself and other colleagues that children who tend to do very badly are, again, late diagnosis, preexisting conditions, untreated complications. But when a child comes into the hospital and is still reactive and has a normal or near normal neurologic exam except for depression of the level of consciousness, those figures stand up very high that child is expected to do well. When the child comes in already lousy, he's in a deep coma and is static epilepticus and has been going for days, you know that equation is going to go to the right of a bad result, You can predict that immediately. But most kids who come in are like Lonnie.

Q. On the 15th?

A. Coming in on the 15th who would have had one seizure who still may be

lethargic, but there is still some reactivity of the child to stimulation. There is no paralysis, overtly recognized pupils. Eighty percent of those kids are going to walk out of this hospital and will earn their living attending colleges or be lawyers or doctors, that's correct, and there is going to be some that will die and some that will be damaged, but those will be less likely to be the ones who came in looking reasonable,

That's why we've come from 100 percent mortality in meningitis to this kind of figure. And everybody emphasizes aggressive and expeditious management so that you move that curve off to a higher and higher percentage of normalcy. It has changed significantly over the years with better management.

Q. But even if we take this group that comes in with maybe only some lethargy on arrival as opposed to being comatose and the real advanced stages of the inflammatory response, there is still about 20 percent of those who are either going to die or have some degree of permanent damage, correct?

A. Ones that came in looking reasonably good?

Q. Yes.

A. No. I would disagree with that. It's far too high.

Q. What would your figure there be?

A. The mortality figure of that group should be incredibly low. Probably -- I'm going to give you a generous 1 percent.

Q. And the morbidity?

A. And the morbidity is going to be -- Long-term morbidity probably around 10 percent or less.

Q. So we've got about maybe at the most 12 percent?

A. Of recognizable morbidity.

Q. Death or some type of morbidity?

A. Right.

Q. But when those children come in the door, you have the 100 percent of all those children, there's no way for you to tell which 8 or 9 go to one side of the room and which 10 go to the other side of either death or some morbidity, is there?

A. No. We've excluded the one who

comes in looking totally --

Q. We're talking now about the group that doesn't come in advanced stages of disease.

A. Right. You can't look at them and say this has been 15 hours, You're looking good. I will tell you for sure you will be in the 90 versus the 10. You can't because we know some complications that are permanent morbidity are irrespective of early diagnosis and they account for a fair percentage of those. It's just the luck of the draw and have nothing to do with the way they are diagnosed or managed.

Q. You talked earlier about the Tylenol that had been given. Were you aware that the Tylenol that had been given by the mother was less than half of suggested dosage?

A. It was .4.

Q. Yes.

A. Well, according to -- I happened to look up this point, and according to the PDR -- and I was looking at the recommendations of the manufacturer. The dose for a four-month-old is to .4 and four months and up it's to .8. And

this child is a couple months premature, You can take it to any way you want to call it.

Q. Did you determine how long the mother reported giving that Tylenol to the child before the child came to the hospital on the 14th?

A. I don't remember specifically. I don't know.

Q. If the ER physicians were told a time they believed would negate the influence of Tylenol and reducing fever, would that be of significance to you in evaluating their response to the child's 100.6 fever?

MR. GORDON: Note my objection.

A. You would only modify it to the extent of saying that given the whole picture at this point they examined the kid and assuming the Tylenol had not worn off, he looked alert and he didn't have a major fever, they would still have to make a decision on all the other factors involved -- their findings, the history when he was brought there, et cetera.

Q. Had you been informed or have you determined from your review of the records that

there was also an upper respiratory infection in this family before this date?

A. Yes.

Q. Have you ever seen any of the records of the aunt or the grandmother?

A. No, not at all.

Q. Can this type of infection that Lonnie Hurt had be spread by upper respiratory infections?

A. Well, which type of infection are you talking about?

Q. I'm talking about bacteremia first and then meningitis pneumococcal.

A. You have to take the sequence in which he got it. He has got pneumococcal meningitis. The pneumococci got into his spinal fluid from the bloodstream. To a reasonable degree of probability they got into the bloodstream from some source where he was colonized. He may have picked up the pneumococcus from somebody and seeded it into his bloodstream. He could have had a source in him for some time that for one reason or another it broke down and seeded into the bloodstream.

I don't think we can tell whether he got it four days before from his grandmother or didn't, but the pneumococcus got into the spinal fluid from him. Where he got it and colonized, you don't know.

Q. Don't these children who develop pneumococcal bacteremia and meningitis often have a history of URIs in the days preceding the diagnosis of bacteremia or meningitis?

A. They often do because they can have an upper respiratory infection, a secondary infection with pneumococcus and then going through. So that's a perfectly acceptable scenario.

Q. Have you been advised in reaching your opinions that the mother never told anyone at the emergency room on the 14th that she had relatives who were confined to Mt. Sinai with what she termed at that time pneumonia?

MR. GORDON: Objection.

A. I do not have that information.

Q. Assuming that otitis media was, in fact, something that should have been diagnosed on the 14th -- the real signs and symptoms of it were there and it could have or should have

been diagnosed at that time -- what would be the care that should have been rendered at that point with a suspicion of otitis media causing the child's symptoms in your opinion?

A. If you think those are the symptoms, you would proceed to give what you think is an appropriate antibiotic based on the history.

Q. Orally or IV?

A. It would depend. If the child is able to keep down oral medication, then usually you're not going to give an IV. If he's vomiting profusely, you might have to, but usually you're going to try to give it orally and you'll pick an antibiotic that you think is safe, usable and is not -- like let's say he had been treated in the last week and it recurred. You might not pick that same antibiotic. You might go to something else.

Q. With a child with Amoxicillin would you go back to Amoxicillin within a reasonable standard of care within the circumstances?

A. I would tell you that right now there are enough changes in recommendations for epidemics. Bugs come up. If I were in that

situation, I'd say right now what I would do is I'd call up one of the people who treat it everyday. I would say, what are you doing now? Because, as I mentioned to you, I don't often prescribe it.

Q. Am I out of your area of what you're competent in?

A. Yes. I will ask because I don't do it often enough and they keep changing the recommendations depending on what's being resistant and what you've treated with before. So I don't presume to be doing it everyday and I call.

Q. Is there any way of knowing to a reasonable probability that had oral antibiotics been started at 11, 12 at night six hours before the first seizure occurred that meningitis would have been prevented in this infant?

A. You can't say with absolute certainty it would have.

Q. With reasonable likelihood can you even say that?

A. You can't say that with likelihood because you don't know when the meningitis

started. I was told you I can't tell you the specific hour. I can only tell you in the general sense if you treat and you get bacteremia, you might prevent the meningitis, but to say absolutely specifically you would have, I don t think you can say that categorically.

You don't even know the meningitis wasn't there already. I was intimated that And the oral antibiotic would not cure the meningitis. It would modify it, but it wouldn't cure it.

Q. Or perhaps mask the symptoms for awhile?

A. That's been said. It hasn't been a major problem for most of us because half the kids in the world get antibiotics and then the next day they come in and people diagnose meningitis. That's not so unusual. I think it masks very little if you know what you're looking at.

Q. Looking through your report now, I'm on page 3 and you have your initials there and then there's been a change with the word UN written before t e word common.

A. Yes.

Q. 1 take it this was typed up at the Nurenberg office and sent back to you and you made a change.

A. No. This was typed up at my office and this was an error and put in.

Q. So the answers were typed here?

A. Yes.

Q. And when you proofread it, you picked that up and just corrected it manually?

A. You know I'm trying to remember the circumstances. I will tell you this was done and I think I was going away or something and either I proofread it or it was gone through and sent back to me. I can't tell you that I remember it specifically, but certainly the statement of common is totally incorrect. I would never make that statement.

Q. Dr. Horwitz, you conclude near the end of your answers to these questions that this child has a greater than 50 percent probability of surviving into early adulthood or beyond early adulthood. That was written by somebody else in their question. You didn't define your understanding of early adulthood.

would you explain what for me?

A. Twenties.

Q. Early adulthood. My son is going to be 21 I guess theoretically he's an adult.

A. He is.

MR. NURENBERG: He can vote at 18.

Q. That's early adulthood?

A. Yes.

Q. How far does that go? Twenty-five? Thirty?

A. I'm just going to tell you I implied the early twenties and I'll leave it at that. I can't be more specific. I mean that is what I would expect.

Q. You've unfortunately seen children that are this devastated before from meningitis?

A. Yes.

Q. It really doesn't matter what they're devastated from, I suppose, as long as they're this devastated as to their life expectancy; is that correct?

A That's correct.

Q. Will he have to be in your opinion institutionalized during his life expectancy?

A. That is a decision that his family will have to make depending on their resources, their ability tu handle the situation, so it is not a medical decision.

Q. Well, I suppose we can make hospitals like homes if there's enough money, but --

A. I'm not sure that -- the Lonnies are not unusual in terms of neurologic damage who stay home and are managed and their family supports them, and the support system we provide -- I would not like to use the term hospital. I think it's incorrect.

Q. Are you aware of any long-term studies that have indicated that out of 100 children similarly damaged that 51 will be alive at age 21?

A. The studies we all know come from one or two from California and some old studies and some of them institutionalized children. It's a huge difference. Nobody can take an exact Lonnie and say, we've got the perfect 100 Lonnies and we've studied them in East Cleveland given the following setup.

Q. Well, they have studied children

with mental retardation and quadriplegia.

A. You have got such a diverse population and you've got different social circumstances. You've got different levels of medical care going into them. There are enough variables that you can take the group and publish it. We agree with that. But even there the experience is different from what we have because this is Cleveland and it is opposed to an institution, et cetera. And we've seen the worse figures and the better figures from the literature. We understand the variables. We're basing them on our own experience and the best estimate given the present level of medical care and provisions that we can come up with. We can't give you a Lonnie Hurt treated in exactly the Cleveland situation in 100 patients and what their survival will be.

Q. I don't want to belabor the point, but aren't you in fairness to some extent guessing on this point?

A. Intelligent estimate.

Q. There's a thin line between that and a guess, isn't there?

A. A guess, I think, is when you just play the lottery. An intelligent estimate means we take care of hundreds of children and the circumstances and it's very individualized, and that's why I'm saying it's not a guess.

Let's say, for instance, that you were giving me the most neglectful mother in the world whose sole purpose is getting out to get her cocaine fix tonight.

Q. Short survival for the child?

A. It would be quite different. And she had no access to health care and didn't bother with it and didn't have individual physicians like Dr. Mortimer and myself and the people in the intensive care unit. So those two circumstances would be so different, so it's not quite a guess. That's why I said it's an intelligent guess.

Q. So quality of care would be expected to figure into life expectancy?

A. There's no question about it.

Q. But are there any figures available with the best possible care how long these children live?

A. No, I don't think you can come up

with a specific figure again because you say these children. You are talking -- when you talk about handicaps, it's apples and oranges. There are so many.

Q. Certainly there's a severely handicapped category that's recognized.

A. Severely. And it depends on tube feeding. It depends how bad the seizure, the drug interactions, I mean you're putting many, many things in that the next one may not have, so that's why I'm telling you I'm giving you a general ballpark figure.

Q. The radiological studies in this case at University Hospitals indicated infarctions, didn't they?

A. Which ones are you talking about? Yes. I have a copy here.

Q. There's an MRI impression and report from 4/18.

A, Yes.

Q. Approximately two days after he came into this hospital. Before I ask you about that impression I did notice in the chart the child was changed to Penicillin after four days. Do you know why that was done?

A. I don't know the specific reason, but when they grow pneumococcus, it's usually very sensitive to Penicillin and it is a cheaper drug, effective and probably less side effects than what they were using, so they went to it. There's nothing unusual about that.

Q. Was there a difference in response to the two antibiotics -- the one that was given for the first four days and then to the Penicillin in your opinion?

A. I would have to really review the sensitivities and whether there was a specific reason, but from my recollection it happened so often to change over that unless there were recommendations all over saying this is a resistance bug and documented, it wouldn't ring any bell with me other than it's common procedure. More the rule than the exception.

Q. The impression on the MRI reads, probably infarct involving the thalami bilateral in the left internal capsule.

A. Yes.

Q. Do you have an opinion, first, as to when that infarction of the thalami came into existence?

A. I have an opinion.

Q. What is that, sir?

A. It is my opinion to a reasonable degree of probability that those infarctions occurred during the process of herniation and vascular compromise.

Q. That would be then between approximately 7:45 p.m. and --

MR. NURENBERG: 1:30.

Q. -- the next morning?

A. No. Closer to 7:30. Closer to 7:30.

Q. P.m. on the 15th?

A. Also to 7:45 on the 15th when the pupillary actions became unreactive.

Q. Do you hold an opinion as to whether the immediate IV administration of antibiotic had it occurred at about 3:30 p.m. without supplementation by hyperventilation and Mannitol would have prevented the herniation?

A. That's a question you would have to divide up and say the patient was given antibiotics. Whether he would have them at 3:30, there would be no reason to give him either Mannitol or hyperventilation. Neither

of those substances would be indicated and should not be used,

Q. Let me back off the question and say this. Had he gotten the antibiotics by 3:30 and they be continued at appropriate dosage levels, would that have prevented the increased -- would that have prevented the inflammatory response that led to the herniation at or around 7:30 p.m.?

A. Again, that's very difficult to give a specific answer, I'm going to go back to say that the earlier you give it, the better results in a general sense. We know that herniation was manifested at 7:45. We know the antibiotics were just gotten in. I don't know how we can be sure either way, but it's really unanswerable in that sense. I still in the general sense think you should give it, but I don't see how you can answer that question. You can't say the herniation wouldn't have happened, but you can say if you give the antibiotics, an inflammatory condition subsides somewhat or is controlled, you're probably less likely to get the excessive swelling and herniation. Again, with the caveat that there

is still the problem that we don't know how much product release from the antibiotics is a factor and so all of those things weigh in.

But everybody who treats meningitis without exception still ascribes to the fact that the major complications are less common when you treat expeditiously, so that is to be the standard.

Q. The MRI also shows ischemic changes throughout the white matter, Have you ever seen the film itself?

A. Yes.

Q. You agree with that?

A. Yes.

Q. Is that due to the herniation and a cutoff of blood flow?

A. Yes.

Q. Or a reduction of blood flow I probably should say.

A. Yes.

Q. Are you telling us then in your opinion that the die was cast for cellular brain damage as of the time this herniation purportedly occurred and that really -- I'll leave it at that first.

A. The die was cast when the herniation -- not the moment of herniation becoming clinically manifest. The die was cast after a reasonable time of minutes, not an hour or two. It was not treated expeditiously. At that point you get into the irreversibility of the situation. But the die was not cast at 7:45 the minute the pupils dilated. That's the time to intervene.

Q. Don't many children who have pneumococcal meningitis as infants have devastating neurological injuries without herniation?

A. Absolutely.

Q. How can *you* tell us in this case had herniation not occurred that these same sequelae would not have occurred?

A. You have to look at the clinical picture here, and that is in the absence of herniation the children that suffer devastating sequelae will occur under several circumstances. One is if they have overwhelming bacteremia and shock and they drop the bottom out of their blood pressure.

Q. Hypertension?

A. Okay. The brain may simply be destroyed on an ischemic mechanism. That was not the case here.

The second reason they may undergo devastating neurological outcome is if the diagnosis and, therefore, treatment is delayed for days. Then the pathology is very clear that you get a vasculitis with vascular occlusions, both venous and arterial. You get multiple infarctions all over the brain, and they're devastating because of multiple infarcts. At that stage is a late stage. That is not a first or second day problem. Multiple infarctions with total devastation.

The third reason it might be devastated is if they have seizures that are uncontrollable, and uncontrolled for an extended period the seizure may cause some destructive changes.

Those are the mechanisms that some come out devastated. I'll leave it at that.

Q. Did this child go on to have multiple infarctions during the care at University Hospitals?

A. This child had a massive

infarction, more than multiple infarctions. This child had a probable infarct recognized at 6:00 a.m. on the 16th, which is telling you that there was at least one recognized infarct going back at least eight hours.

Q. That I understand. I understand what you believe is related to the herniation. But I'm asking later after the herniation was relieved --

A. I think --

Q. -- were there infarctions then?

A. After the herniation was relieved what you saw over the next few days was the total -- this is not multiple infarction. This is just total infarction of the whole brain, and that is not a picture you see in a meningitis of a day or two's duration without herniation. You simply don't see it. You can see an isolated infarct. You may get two infarcts even on first or second day. I've seen that. But total destruction is simply not a first or second day problem. And this was going on already. I mean clinically you know it was going on.

Q. What about these bilateral cystic

hydromas? Were they a product of the herniation also in your opinion or a later process of the disease?

A. You know, they're given different names here. Let's not get hung up on cystic hydroma.

Q. I'm using the term you used. You signed it.

A. I know, That's what the report said dictated by a new intern, and I was glad he did it for me. I'd have to review it. You can call it a cystic hydroma. It's a fluid collection, and the hydroma is rather a specific term. It's a subdural -- it's a fluid collection probably subdural, and I don't think it is due to the herniation. I think it was there prior to the herniation.

Q. You also -- you qualified this. You said this was dictated by the resident, Dr. Wenger, I guess.

A. Nicest guy in the world. Say nothing bad about him.

Q. You read it before you signed it. I take it you agree with what he's got in it or you wouldn't have signed it. Or am I wrong?

A It was adequate for the purpose that it was done. There was nothing in there that is going to effect. If you tell me everything is specifically accurate, probably the lab values are Some of these concepts aren't, but that's --

Q. I'm not going to play that game of trying to find a factual error that way.

A. No. In fairness let me just say that Dr Wenger dictated this as a floor resident after the patient was discharged from the intensive care unit and everything in the sequence he sort of picked up by reading and so on where are going to be inaccuracies.

Q. He has written here on 4/21, the CAT scan revealed bilateral cystic hydromas with acute hemorrhage within the right hydroma.

A. Yes

Q Did you agree with that interpretation whether we call it a subdural or a hydroma on the CAT scan of 4/21?

A. Those are the reports I have no reason to dispute them.

Q. Then it goes on to say, it, being

the CAT scan of 4/21, also revealed acute infarcts in the right middle cerebral artery distribution.

A. I would have to take each CAT scan and look and ask you what the specific questions were.

MR. LEBOVITZ: Do you want to see the CAT scan report?

MR. KALUR: We can look at the interpretation. I don't think we have the films here.

MR. LEBOVITZ: We do.

MR. KALUR: Good.

A. Which one are you referring to?

Q. This appears to be the first error because it looks like a 4/20 scan.

A. Well --

Q. But it won't matter.

A. There is a 4/20 scan.

Q. If we've got it here. Well, it says, acute infarcts to the right middle cerebral artery distribution. Do you agree that they were acute?

MR. LEBOVITZ: I think you're on a different page.

A. Which scan are you looking at?

Q. I'm back to the discharge summary now.

A. Oh, I don't know what he's saying. The acute --

Q. Yes, he says acute infarcts.

A. Oh, acute in the sense that they are not two weeks old. Yes.

Q. You would attribute the term acute in this case to relate back to the 15th?

A. Let me be clear. This is an infarct seen on the first scan of the 16th, and I think it was slide 11. I found it a hard time to see it, I sort of convinced myself, But I've been through it with Dr. Kaufman, and that would indicate one recognizable infarct,

Now, we know that infarcts take some hours to become visible on a CAT scan. And then you see the progression through the skin, but it's still acute. We're not talking about some three weeks remote. That's the word acute.

So all of it is evolving and it shows more and more as time goes on, and that's pretty typical. But the extent of it would

show on one of the scans. It shows diffuse white matter, and what I'm trying to show is total destruction of the white matter. There's nothing left.

Q. In the discharge summary that Dr. Wenger dictated and you signed over it indicates that there was found to be in the CAT scan of 4/21 -- apparently it's a 4/20 CAT scan, but in any event, middle cerebral artery infarctions.

A. Yes.

Q. We'll just leave off the word whether it's acute or not at this point. To what do you attribute those middle cerebral artery infarctions?

A. All of that is attributable -- all of that is attributable to the decreased flow that occurs during herniation process when you can't profuse and you get diffuse or multiple scattering of the infarctions in that situation. So this is all attributable to that.

Q. How can you rule out vasculitis between the 16th and the 20th as being causative of that given that it takes a few

hours at least to show on a CAT that there's an infarction? We're talking about four days.

A. You're telling me that vasculitis occurred after the 16th and caused these infarcts?

Q. How can we rule that out as a possibility?

A. What if you look at the clinical picture of the patient at that time? Everything is consistent with gross brain infarction. But if you tell me there were one or two small additional infarcts, I think the whole thing was grossly infarcted anyways. The clinical picture was there. But did he have one or two out of that middle cerebral artery? I -- you're adding an extra thing after the fact when you already know what happened before the fact. I don't know why you would want to add it after the fact.

Q. If you don't have herniation in these cases, as you said earlier vasculitis, you said, can cause infarctions, so maybe this would have occurred in any event, and I want to get your view on that.

A. This child had fixed pupils. There

is no infarct that causes fixed pupils in this situation. There isn't. Also as I pointed out to you infarctions occur with vascular inflammation generally late in the course of the disease untreated. Isolated infarcts are known to occur either early or later in the course despite adequate treatment. Massive infarction to destroy the whole brain like this does not occur in an early treated case with an infarction. It simply doesn't happen.

But if you're asking would one infarct have occurred because of vasculitis, the answer is yes, but it would be a drop in the bucket compared to what's happened to this child's brain.

Q. I may be done. Just give me a second here.

Do you have an opinion as to whether hospitalization of this child was required by appropriate medical standards even if the diagnosis of otitis media was an obvious one on the 14th given all the other --

A. On the 14th would I have hospitalized the child?

Q. Yes.

A. No. No.

Q. Given the signs and symptoms that existed at that time and, again, presuming that otitis media was a proper diagnosis, was there a reason to do a lumbar puncture at that time in accordance with proper medical standards?

A. The doing of a lumbar puncure at that time would have been an absolute judgment call on the part of the emergency room physician. If he had diagnosed otitis and treated it and decided he had an adequate cause and was satisfied and explained the history of fussiness, fever and the child looked good, I would say it would have been unnecessary to do the lumbar puncture at that point.

If the mother really termed the kid as extremely fussy and he had vomited and there was a marked change in this child and he thought that the history was ominous and not explained by the otitis, his judgment might have led him to a lumbar puncture.

I think we don't have those facts, and given what I see I would have loved to have the lumbar puncture because I would have known at that point. But given the emergency room

you see a child vomiting and a little sleepy, mother said had a fever and had some otitis, I think unless the history told you more you would probably not do the lumbar puncture.

MR. KALUR: Those are all the questions I have, Dr. Horwitz.

MR. POLITO: I just have a few.

EXAMINATION OF SAMUEL J. HORWITZ, M.D.

BY-MR. POLITO:

Q. Doctor, you indicated you had some handwritten notes. Could I see those?

A. Yes. I can't promise you can read them.

MR. KALUR: Just one thing before you get going and you're going to get into that.

EXAMINATION OF SAMUEL J. HORWITZ, M.D.

BY-MR. KALUR:

Q. While you look at those notes, I wonder if you could read for the court reporter your consult note so we will know exactly what it says and I don't have to guess at some of the words.

A. Sure.

Q. I can probably read 90 percent, but

I'm afraid of that 10.

A. This is a consult note of April 16, 1990, that's headed peds neuro. The note is written by Dr. Rahel Berrhene, R A H E L B E R R H E N E, and I signed it. So it's her writing actually, but I'll be glad to read it.

Q. You didn't write a consult note then?

A. No. She presented the patient to me.

Q. I didn't think that was your writing.

A. No, that's not my writing.

Q. All right. Then I'll struggle with it.

A. I'll be happy to read it for you.

Q. If you can. Some of it I couldn't read, but maybe you can read it. Maybe you're used to it.

A. Seven-month-old, former 32 week, transferred from Mt. Sinai with bacterial meningitis.

Presented with fever and irritability of two days' duration followed by seizure activity on the day of admission. He

received Cefataxime on admission to Mt. Sinai and a spinal tap was performed a few hours after admission. There's some symbol I don't know. It could be consistent with or something.

Q. I think that's blood culture. BC?

A. No. It's something see of clinical deterioration because of -- it's probably what it is, but, again, I don't know her abbreviation.

Transported from Mt. Sinai following endotracheal intubation for irregular breathing pattern in the face of an isophoria.

Q. What does that mean?

A. Unequal pupils. (Right greater than left.) With minimally reactive pupils and obtundation.

Since admission to PICU he has had two self-limited seizure activities which were aborted with Valium. He also was loaded with IV Phenobarb and Dilantin.

Exam today at 24 hours after initiation of antibiotics revealed on Phenobarb, Dilantin, Valium. No other sedation. Temperature 30. Can't read it. The

copy could be 38. Pulse 184. Respiration vent. (BP equals 141 over 72 with arrow to 100.)

Minimal spontaneous activity. Occasionally draws up both legs less than arms. I think that's an arrow down between legs and arms.

Q. Yes.

A. No posturing. Withdraws from noxious stimuli.

Question mark right eyelid to the ptotic and somewhat swollen lid.

Q. Does that mean ptosis of the eyelid? Is that what ptotic means?

A. Yes.

Q. Is that a reflection of pressure on the third or fifth cranial nerve?

A. It could be dysfunction of the third -- of the elevator of the third nerve. But it also could be due to the swelling of the lid. And she said it is swollen. Lid swollen. Can't elevate it.

Q. Why would you still have this finding 24 hours after the initiation of antibiotics and at least 12 hours after the

giving of Mannitol and long after the giving of hyperventilation to that child?

A. After you've compressed the nerve -- third nerve outflow it may take -- if it's transient compressed, it's been a few minutes and you relieve the pressure, it can usually come back. If it's been some hours, it may be so compressed that it never ever comes back, is destroyed, or it may come back partially or very extended. So it's perfectly acceptable.

Do you want me to carry on?

Q. Yes.

A. Right pupil five millimeters left, three millimeters right. Both nonreactive. Disk flat right side. No spontaneous breathing over the vent. and then DTRs as indicated. And there's a little man figure to show that they're three plus. Has peritoneal (increasing tone on repeated pass of flexion and extension.) Babinski plus bilaterally with positive -- I don't know. And EG underlined, Multiple epileptic form discharges. It's either left or right. I can't read if it's an L or T. Parietal temporal. There's some numerals there. Oh, okay. Two hertz, one

hertz. THZ, lHZ, and then an arrow which changes to three hertz, a spike and slow wave discharges sporadically on the right side.

CT underlined. Bilateral subdural effusions. No mass effect? Mild enlargement of ventricular spaces,

Assessment: Global depression (paucity of spontaneous movement.) It's abbreviated, It's MVT. (No spontaneous breathing over the vent.) Concerning global depression. It should really read global depression concerning and then she's got the parentheses between the two statements. Continuous seizure activity on EG. Also portends an unfavorable prognosis.

Bilateral ocular palsy with depressed sensorium and loss of respiratory drive may indicate a brain stem lesion, vasculitis and thrombosis.

And then recommendation. Would continue to monitor; respiratory support, anticonvulsants, and a few dots. Needs repeat EG tomorrow.

And then there's a line, which is in my handwriting. Agree with above. Also MRI

when clinical condition permits. And then it's signed by Rahel Berrhene and countersigned by me.

Q. When you say agree with above, are you agreeing with the recommendation or more than that?

A. Agreeing with the general assessment.

Q. What about -- first of all, this was a resident who wrote this?

A. Dr. Berrhene was a third-year resident who was actually the person who transported the patient from Mt. Sinai. In that month she was doing an elective with me and, therefore, would see all of the consultations ahead of me, present them and then write it up. And I would pretty much -- she might write it up before I got there or she might write it up after we've seen the patient and then I would countersign that. It was what we had talked about.

Q. Was she a neurology resident?

A. No. She's a third-year pediatric resident.

Q. Her conclusion that bilateral

ocular palsy with depressed sensorium and loss of respiratory drive may indicate a brain stem lesion, vasculitis and thrombosis -- do you agree with that?

A Yes She should have qualified it. What we are implying is that the brain stem had been damaged leading to vascular occlusion. Thrombosis and vasculitis was used in the sense of sort of more than inflammatory If I had my way, I would have her write it a little more specifically to avoid confusion.

She and I talked about this for hours as we did with a medical student there that morning. I probably spent two and a half hours in there at least on this case both reviewing and teaching, so going through all this with a very new, fresh medical student.

Q. RJM?

A. Is Dr. Aronberg's daughter and, therefore, I had to be very nice and thorough with her or her daughter would be critical of me. Junior medical student and well written, but obviously a lot of it out of context with this case But that's part of the learning experience She's very good.

MR. KALUR: I'm sorry. Go ahead. Thank you.

MR. POLITO: I'm going to have these marked. There's no need for you to read them, but if we could just have them masked.

MR. NURENBERG: We can have them marked, but they're staying with the doctor.

MR. POLPTO: Then we'll want copies of them.

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(Thereupon, Deposition Exhibit 1 was mark'd for purposes of identification.)

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Q. Doctor, handing *you* what has been marked as Defendant's Exhibit 1, can you identify what those are?

A. This?

Q. Yes.

A, Oh, this is a yellow note pad with handwritten notes from me relating to the medical record.

Q. And they consist of how many pages, doctor?

A. Six.

Q. Mr. Kalur asked you if you had seen deposition summaries of a few physicians. Wave you ever seen a deposition summary of Dr. Washington?

A. I have not seen a deposition summary of any physician including Dr. Washington.

Q. Do you know Dr. Washington?

A. Yes, I do.

Q. You indicated the pupillary changes seen at 7:45 were due to brain pressure.

A. Yes.

Q. If it was not due to brain pressure, doctor, what are other causes -- what could this also be due to?

A. It could be due to somebody having given the infant Atropine in the eyes or a very large dose of an Atropine like drug administered parenterally.

MR. KELLEY: Administered what?

THE WITNESS: Parenterally.

A. A unilateral dilatation transient could occur after a severe seizure. Very rare, but it happens. An inflammation of the third nerve by the inflammatory process could do

that. It would be incredibly uncommon and I've certainly never seen it to be bilateral and for so many hours to go without showing on third nerve manifestations. Maybe the first time, but it wouldn't persist for hours. So there's really not much else that could do this.

Q. Did the child's neurologic status change from 7:45 up until the time he was transferred to RB & C?

A. The neurologic status changed because,. one, the pupils became unreactive. That was one change. The second is there was a respiratory change, and that respiratory change is a neurologic basis. That was the second basis.

Q. That occurred about 1:30?

A. Yes.

Q. Any other changes?

A. As best I can extrapolate from the records, this child was less responsive to the mother later in the course of the evening as opposed to on admission* Beyond that you can't say because the admission records really do not indicate a detailed neurological examination at any time during the course. It's simply not in

the records, so you can't say what the change was because it wasn't recorded before or after.

Q. You indicated you believe in this case this child had a transtentorial herniation.

A. Yes.

Q. Could you define that for me?

A. That means there is pressure from above and the cerebrum pushes down and the brain stem pushes through the tentorium jamming up all the structures then.

Q. Was that noted on the initial CT?

A. No.

Q. Was it noted on the subsequent CT?

A. It was never noted on the CT.

Q. Was it noted on the MRI?

A. No.

Q. What is the basis then, doctor, of your opinion that this child had a transtentorial herniation?

A. The clinical picture of the child that evolved is absolutely typical for transtentorial herniation. It's the classic picture described. It's accepted neurologic syndrome. It's perfectly clear.

Q. What signs and symptoms would you expect or what signs and symptoms did the child have when he reached RB & C that would be suggestive of transtentorial herniation?

A. The things I mentioned before. He had the pupillary dilatation. He had a diminished level of consciousness.

By the way, can I just make -- I want to make a comment after that. Diminished level of consciousness and he had respiratory change. He also didn't have normal motor activities. Actually he had withdrawal activity which wasn't well specified.

Q. Was increased intercranial pressure noted when he reached the floor at RB & C?

A. You can't note intercranial pressure.

Q. Did the clinical exam done at the time the child reached RB & C indicate the child had increased intercranial pressure?

A. I don't know how to answer the question because you can't tell raised intercranial pressure just on one side. The picture is of raised intercranial pressure.

Q. So when the child hit RB & C, in

your opinion it was suggestive of increased intercranial pressure?

A. In my opinion it was suggested way beyond increased intercranial pressure.

Q. And radiographically also suggestive of increased intercranial pressure?

A. I've qualified already that the radiographic appearance did not show the herniation.

Q. At any point?

A. It did not show the herniation at any point, that's correct.

MR. GORDON: I think he's saying it went down by that time.

MR. NURENBERG: I think so.

Q. You said, doctor, at 7:45 you would have treated the patient with Mannitol and you used the term other drugs. What other drugs are you speaking of?

A. Mannitol, Hydrocortisone steroids. Hyperventilation is not a drug. And if the child was the least bit agitated at that point, I would have given some sedation to keep him calm so as not to push up intercranial pressure.

Q. I'm not sure you answered this, but I just want to be clear.

Had these drugs been given at that time can you quantitate the amount of damage the child would have suffered?

A. I don't think I was asked that question specifically had he been treated, I will only tell you that to a reasonable degree of probability that child would have been normal or certainly infinitely less damaged than he is now.

Q. At 7:45?

A. That's correct.

Q. And what's the basis of that opinion?

A. The basis of that opinion is that the sign of herniation at 7:45 -- the first sign is your indicator it is happening. It's the window of opportunity that is in all cases of herniation the time where clinical experience throughout the United States and the world indicates if you're going to make a difference, you get in, And all of us have had the experience of turning around kids at that point -- getting in early and coming out with

perfectly normal survivors. I mean I've written a paper on that and have been one of the people extensively quoted. This is a standard of care that is now accepted. It is the practice that we teach. And we know that if you don't relieve it, you die, and we know if you get in early -- that's why you do pupillary checks, There's no other reason to do them.

And it's part of the vital signs. A blown pupil to a neurologist or neurosurgeon is like a siren going off,. and we tell everybody else to take care of it -- to act and call immediately for help. I mean it's a standard thing that I teach someone. I don't know how many hundreds or thousands of times I've taught this thing.

Q. And what's the window of opportunity, doctor, in your opinion?

A. The window of opportunity is minutes at the most favorable, hours with luck, but, as I've pointed out, I've seen kids who in precious minutes have gone by and it became irreversible. It's a very short window. Everybody agrees with that. It should be acted

on -- in fact, let me just qualify that and say before our intensive care units were as organized as they are today when we admitted any meningitis, not only did we do the pupillary checks I'm talking about at this hospital, but the Mannitol was drawn up in a syringe and strapped to the bedside next to the intravenous because of the determination to get in there so there's no running around looking for the darn stuff and drawing it up. Minutes are literally precious.

MR. POLITO: I have nothing else, doctor.

MR. KELLEY: My understanding is Mr. Goldwasser through previous communications with you has preserved his right to reconvene this deposition.

MR. NURENBERG: At his expense at a time when I can rearrange the doctor because he had to be out of town. We will ask Mr. Horwitz to make himself available so long as the same questions aren't gone over.

MR. KELLEY: And Gary will review the transcript obviously before he makes that decision.

MR. NURENBERG: So I guess that's it then. I think I'd like to have the doctor have the opportunity to read the deposition.

(Whereupon, the deposition was adjourned at 11:20 o'clock a.m.)

CERTIFICATE

The State of Ohio,)

SS:

County of Cuyahoga.)

I, Julie Gentile, a Notary Public within and for the State of Ohio, duly commissioned and qualified, do hereby certify that the within named witness, SAMUEL J. HORWITZ, M.D., was by me first duly sworn to testify the truth, the whole truth and nothing but the truth in the cause aforesaid; that the testimony then given by the above-referenced witness was by me reduced to stenotypy in the presence of said witness; afterwards transcribed, and that the foregoing is a true and correct transcription of the testimony so given by the above-referenced witness.

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

I do further certify that I am not a relative, counsel or attorney for either party, or otherwise interested in the event of this action.

IN WITNESS WHEREOF, I have hereunto set my hand and affixed my seal of office at Cleveland, Ohio, on this 9th day of January, 1992.

Julie Gentile

Julie Gentile, Notary Public
within and for the State of Ohio

My commission expires September 21, 1995.