IN THE CIRCUIT COURT OF THE SEVENTH JUDICIAL CIRCUIT

IN AND FOR ST. JOHNS COUNTY, FLORIDA

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JAMES and MAUREEN LONG, individually and as parents and natural guardians of Christina Long, a minor,

Plaintiffs,

vs.

ANGELITA CORRAL, M.D.; ROBERT LEE, M.D.; WARREN O. WHITLOCK, M.D.; FLAGLER HOSPITAL, INC.; AIR AMBULANCE ASSOCIATES; JUAN LARROUDE, Y.D.; JOYCE HENUBER, R.N.; SALLY ROBB, R.N.; SHARON HELLMAN, C.N.M.; ALICE ROEHRIG, R.N.; S. PADRON, Y.D.; and N. NORTHINGTON, R.N.,

Defendants.

No. 88-492-CA
Division P

DEPOSITION OF ANTHONY JAMES BARXOVICH, M.D.

APRIL 23, 1990, 9:16 A.M.

Reported By:

Christine M. Niccoli, CSR No. 4569

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E X H I B I T S

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BE IT REMEMBERED that, pursuant to Renotice of Taking Deposition Duces Tecum, and on Monday, April 23, 1990, commencing at the hour of 9:16 a.m. thereof, at the TNIVERSITY OF CALIFORNIA SAN FRANCISCO MEDICAL CENTER, Long Hospital, 505 Parnassus Avenue, Room L-305, San Francisco, California, before me, CHRISTINE M. NICCOLI, a Notary Public in and for the County of San Mateo, State of California, there personally appeared ANTHONY JAMES BARKOVICH, who was called as a witness by the Defendant herein, was examined, and testified as is hereinafter set forth.

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A P P E A R A N C E S

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ANTHONY JAMES BARKOVICH,

having been duly sworn, testified as follows:

MR. NOECKER: Would you state your full name for the record, please.

THE WITNESS: Anthony James Barkovich.

EXAMINATION BY MR. NOECKER

MR. NOECKER: Q. Dr. Barkovich, my name is Scott Noecker. I represent Flagler Hospital. If at sometime as we go through this you don't understand my question, I want you to please ask me and I'll do my best to repeat so it's decipherable to you.

What's your address, business address?

A. Neuroradiology Section at UCSF, and it's 505 Parnassus in San Francisco.

Q. How long have you been with the university?

A. I've been here for almost four years. I spent a large portion of that on the clinical faculty while I was doing my army obligation at Letterman Hospital here in San Francisco, and I've been full-time faculty since the beginning of October.

Q. Have you ever been in private practice?

A. No.

Q. As an associate professor, which I think your CV indicates, --

A. Yes.

Q. -- what are generally your duties and

responsibilities under that title?

A. Well, my responsibilities are to take care of the -- or to perform the clinical duties of a neuroradiologist here at University of California, and we cover Long and Moffitt Hospitals where we're now as well as the VA Medical Center at Fort Miley and an imaging center on California Street called San Francisco Magnetic Resonance Center; and while doing that we teach the fellows and the radiology residents, neuroradiology fellows and radiology residents in neurology.

Q. Okay. Breaking it down in percentages, how much of your work week is spent in actual teaching duties?

A. Well, any time I'm doing clinical work I'm also teaching because we never do anything without a fellow or a resident at our side. So I'd say probably, oh, 50 percent of my week is clinical, slash, teaching; probably 25 percent is research, and 25 percent is administrative.

Q. Okay. So are you salaried by the state?

A. Yeah.

Q. Okay. Do you have any other income other than your salary you receive from the state?

A. Well, I receive income from lectures on neuroradiology, as well as I make some money from consulting in medical-legal cases.

Q. Who is your immediate supervisor here in the

radiology department?

A. Dr. David Norman, who is the chief of neuroradiology.

Q. In your practice have you ever had your staff privileges at all curtailed, or have you ever been disciplined in any nature?

A. I have not.

Q. Have you ever been investigated under peer review?

A. No.

Q. You board certified?

A. Yes.

Q. What year were you board certified?

A. **1984.**

Q. Pass that on the first try?

A. Yes.

Q. You've given me a copy of your CV before we started this deposition. Is that up to date and current?

A. It's real- -- I mean, you know, within a couple of months.

Q. Okay. Do you have any other articles or presentations that you've made that need to be added to this?

A. Yes. There are a few.

Q. Okay. Are any of those articles even remotely related to either the facts or the medicine involved in

the Long case?

A. There are nonrelated to the case that aren't on the CV.

Q. Okay. What states are you currently licensed to practice in?

A. Just in California.

Q. Of the articles and research activities, presentations, that are listed in your CV that you've given me, can you tell me which ones deal with, however remotely, either the facts or the medicine involved in this case?

A. Well, there are -- there's a chapter of my book that -- Chapter III in my book deals with asphyxic damage in premature and term kids, and there's a paper that's in press for the AMERICAN JOURNAL OF NEURORADIOLOGY that deals with MR patterns of asphyxic injury in children.

Q. Why don't you go ahead and mark the ones -- put a circle or a check.

MR. NOECKER: And we'll have this -- you remember, if you would, we'll have this one attached to the deposition.

A. Those are the only two that really pertain perhaps -- see, where is that one. There's one over here that may have some remote -- There it is. So I've checked three references that are related in some way.

Q. Is that research activity that's listed on page

12 with neonatal asphyxia related to this case?

A. I -- In remotely, I guess, yeah. We're still waiting to get the decent funding on that. That's more of a grant proposal than an actual research activity. We've done just very little preliminary stuff on that project, and I don't think we have enough data to reflect in any way on this case.

Q. And is your answer equally applicable to the subparagraph b under that where you're speaking of patterns of asphyxic brain damage is retrospective study in attempt to correlate patterns of brain damage with the duration and severity of the insult and with the gestational age of the patient at time of the insult?

A. That's actually in that paper that's in press.

Q. Okay.

A. The neonatal pig one we haven't done anything on to speak of except to show that we can -- that we have adequate technology here to do a -- we're still waiting for funding from the NIH.

Q. Do you know why plaintiffs' counsel contacted you in this case?

MR. BARNHART: Objection. Speculation.

You can't read my mind.

A. I have no idea why he contacted me.

Q. Who -- Let me don't put the cart before the horse. Who did contact you first with regard to the Long

case?

A. I think it was someone who works in Mr. Barnhart's office called up -- Linda Gordon called up and asked me if I'd be willing to review some films. And since that's what I look at, -- MRs and CTs and ultrasounds is what I do for a living -- I said I would be happy to look at it for them.

Q. Is she the same -- strike that.

Was your first contact, to the best of your knowledge, from Palm Beach Medical Consultants?

A. Yeah, to the best of my knowledge.

Q. When was that first contact?

A. I can't -- Maybe it's -- Within the last year, I believe.

Q. Do you have any correspondence that memorializes that first contact?

A. I don't . . . No. This (indicating) is very recent. Everything I have on this, I think, is in here. I probably no longer have that.

Q. Do you know when you were first provided with any information to review in the case?

A. October 25th, 1989.

Q. And who did you receive that information from?

A. Linda Gordon at Palm Beach Medical Consultants.

Q. And what information did you receive in that first package?

A. I received three sets of films. The first was a series of copies of ultrasound films, dated the 5th of September 1984; the second was a CT scan, dated the 23rd of May 1985; and the third packet was a CT scan and an MR scan, both of which were dated 17th of May 1988.

There was also a set of skull films in here, plane films of the skull that were dated 18th of May 1988; and at that time I also received some medical records from Flagler Hospital on Baby Girl Long, dated the 2nd of September and the 3rd of September 1984.

Q. That's all the information you -- that you received in October of '89?

A. Yes.

Q. Have you subsequently received any other information?

A. Just some additional medical records.

Q. When did you receive those --

A. Oh.

Q. -- and what did you receive?

A. In the -- Within the last week Flagler Hospital records on Maureen Long on Christina's delivery, St. John's County Rescue Service Air Ambulance Associates reference Baby Girl Long, and Angelita Corral, M.D., records on Baby Girl Long and Plantation General Hospital records on Baby Girl Long.

Q. Is your practice here at U.C. related solely

and limited solely to radiology and neuroradiology?

A. Yes.

Q. I notice your CV says you are professor of radiology as well as pediatrics and neurological surgery.

A. Yes.

Q. Are you board certified in pediatrics?

A. No, I'm not. And this is kind of a -- what's -- it's sort of an honor more than anything else that -- I do participate a lot in the pediatrics department in conferences and the like because I do basically pediatrics neuroradiology here in addition. I do general general neuroradiology, but I'm here largely as a pediatric neuroradiologist, and that's why I was hired here.

And the neurological surgery stuff is because I participate -- I review either officially or unofficially the scans of every pediatric patient in -- who undergoes neurological surgery in the department. So the surgeons also wanted to honor me with an appointment in their department.

MR. BARNHART: Come on in.

MR. SCHODER: Excuse me, guys. We've been out seeing most of San Francisco.

(Recess taken.)

MR. NOECXER: Q. Do you have any actual duties, though, within the pediatric department or the

neurosurgical department?

A. In terms of caring for patients, no.

Q. Okay. And I presume you don't teach pediatrics to the pediatric residents. Do you?

A. I teach pediatric neuroembryology and pediatric neuroradiology to them, but I do not teach pediatrics to them.

Q. How many times have you been retained by Mr. Barnhart's firm to either review material or serve as a witness?

A. This is the second time.

Q. Okay. Remember what the other case was?

A. Case's name was McLaughlin.

Q. McLaughlin?

A. Yes. It's M-c-L-a-u-g-h-l-i-n.

Q. That was the plaintiff?

A. Yes.

Q. Do you know who the defendants were?

A. No.

Q. Remember when that was?

A. Few months ago, two months ago. That's when -- Actually, they retained me about a year ago. I take that back.

Q. Has your deposition been taken in that --

A. That was about two months ago.

Q. Has your deposition been taken in that case?

A. That was about two months ago, that deposition.
Q. Oh. Do you remember what attorney took your deposition?
A. I do not. I'm sorry.
Q. Do you have any idea where that case was filed?
A. Florida.
Q. Okay. You don't remember the city that --
A. No. I'm sorry. I really don't.
Q. What kind of case was that?
A. It was similar to this one, a question of birth asphyxia.
Q. What were your findings?
A. There was a sonogram that showed acute cerebral edema that a week later had abated, and there is a follow-up MR scan that showed damage to the cerebral cortex and the subcortical white matter in the intervascular boundary zone, what we called the water shed zones; and I thought it was suggestive or compatible with some brain damage, asphyxia.
Q. Did that case involve any radiologist or neuroradiologist as defendants?
A. I have no idea.
Q. Okay. You didn't render any standard-of-care opinions in that case?
A. No.
Q. Your opinions in this case were limited to

what? Causation?

A. My opinions were limited to interpreting the radiographs and come to some conclusions as to the type and time of injury and perhaps the cause of injury.

Q. What have you been asked by Mr. Barnhart or anyone from his firm or Palm Beach Medical. Consultants tc do in this case?

A. Similar. I'm not an expert in obstetrics; I'm not an expert in pediatrics; and I have no opinion as to the level. of obstetrical or pediatric care or neonatal care that was given here.

MR. BARNHART: Scott, Dr. Barkovich is going to look at the head films and read it. That's it. That's his role.

MR. NOECXER: Q. What's your fee arrangement with Mr. Barnhart?

A. Mr. Barnhart pays me by the hour.

Q. How much?

A. $400.

Q. How many hours have you put in on the case to date?

A. To date? Counting this morning?

Q. Yes.

A. About two.

Q. How much are you charging me?

A. $500 an hour.

Q. Not as bad as most.

A. What?

Q. Nothing.

MR. BARNHART: He said it was not as bad as most.

THE WITNESS: Is that right? Maybe I should raise my fees.

MR. NOECKER: Q. Are you affiliated with any expert-witness service?

A. No.

Q. Okay. Have you ever been?

A. No.

Q. You know what I'm referring to, these different services that list doctors to help attorneys find people to testify in medical malpractice cases?

A. I assume -- I know. I assume that that was what you were referring to. I have never been in one of those, and I have no intention of ever belonging to one of those.

Q. In the last year how many times have you either served as some expert or consultant in a medical-legal matter?

A. In the last year, probably about 20 or maybe 25 times.

Q. Have you ever worked for any other Florida lawyers?

A. I just don't recollect. I don't think so.

A. Yes.

Q. How many med-mal cases did you take on, let's say, the year prior to that, 1988?

A. Probably a lot less. Maybe ten. Probably less. Five, ten.

Q. What about '87?

A. I don't think I did any at that time.

Q. Okay. What percentage of these have been for the plaintiffs or for the defendants?

A. Probably about 80 percent for the defense and maybe 20 percent for the plaintiffs.

Q. Have you ever worked with a Florida defense firm?

A. I think I just told you that I don't think I've been retained in Florida, so -- other than this.

Q. Have you ever given any other depositions on facts in medicine related to this case?

A. No.

MR. FACCIOLO: I'm sorry I came. Hi, guys.

MR. NOECXER: Q. Have you ever testified in court?

A. Yes.

Q. Where?

A. Once in Seattle and once in San Rafael, which

is just ten miles --

Q. Was that --

A. -- ten miles north of here.

Q. Was that for the plaintiff or the defendant?

A. Both were for the defendant.

Q. Do you remember who the lawyers were you worked for in those cases?

A. Yeah. The one in Seattle's name was Joel Cunningham; and the one in San Rafael, the lawyer was Robert Lynch, L-y-n-c-h.

Q. Is he a San Rafael lawyer?

A. No. He works out of San Francisco.

Q. Have you prepared any kind of written reports or letters, any sort of memoranda, notes in this case?

A. No.

Q. You haven't ever been sued for malpractice, have you?

A. I have not.

Q. I think you listed for me the -- all the information and all of the records that you have received from Mr. Barnhart; and they've come in two packages, once last week and once on -- in October of 1989?

A. That's correct.

Q. Have you reviewed any other medical literature in your efforts to evaluate this case?

A. Nothing in addition -- No, I haven't reviewed

anything specifically, other than my normal reading as pertains to my job.

Q. Well, since you got this case, have you read anything that's been particularly enlightening as to your understanding of this case?

A. No.

Q. Have you brought with you everything that Mr. Barnhart's provided you?

A. Yes. It's all right here.

Q. Have you come to any conclusions?

A. Well, it would be easier for me to explain any conclusions if I could just go through the x-ray studies that have been provided to me chronologically.

Q. That's fine.

A. Okay. Well, the first studies in time that were provided for me --

Q. I'm sorry. I didn't hear what you said.

A. The first studies, speaking in terms of the timing, okay, that were provided to me were sonograms, ultrasound exams that were dated the 5th of September **1984.** And these images were obtained through the anterior fontanel, which is an area where there's no bone in the top of the skull so that the sound waves can get through to the brain.

And these images are remarkable for an area of what we call hyperechogenicity. Now, all that means,

kind of a fancy term for saying that the area produces more echoes of the sound waves. And the area where that has the more echoes is right around the lateral ventricles or what we would call the periventricular region. And this hyperechogenicity is most commonly seen as a result of edema, which is increased fluid in the spaces between the cells: and it's more -- most commonly occurs as a result of injury to the brain.

Now, there are two different intensities of hyperechogenicity in this area. There's a kind of diffused hyperechogenicity around the ventricle, and within that broader area there are several focal areas that are much darker or even more echogenic; and my interpretation of this is that there's some periventricular edema with probably some foci of hemorrhage within it. And the hemorrhage -- there is a -- the most echogenic-presumed hemorrhage is most prominent posteriorly around the trigones of the lateral ventricles and anteriorly around the frontal horns of the lateral ventricles.

MR. BARNHART: Would you tell us which actual study. Is that marked out so we can --

THE WITNESS: It's not on these -- On sonograms what you do is: It's a real-time exam so that you're looking at the brain; and whenever you get a good image, you just shoot it. And it's done on -- usually done on

sometimes -- usually with kids in the neonatal ICU -- intensive care unit. They take the machine up with the videotape and then you bring the machine back down with the videotape and you play the videotape on the camera, and then you shoot the images at that time. So it's not like a CAT scan or an MR scan where you go methodically through the head and get images in a particular order.

MR. BARNHART: Oh.

THE WITNESS: How methodical it is depends how methodical the person who actually does the scanning.

MR. BARNHART: For the record, we're using the middle two images and the one on the lower right-hand corner. Is that the three you talk about?

THE WITNESS: Yeah -- well -- Yes.

MR. BARNHART: Sorry to go through, but you wanted to make a --

THE WITNESS: And all the images show basically the same thing. The ones that we just pointed out are predominantly in the coronal plane, and that's where this "COR" in the upper left stands for. And coronal just means cutting the brain from front to back or imaging the brain from front to back, And the abnormal areas are substantiated or verified, I guess, on saggital images.

Saggital images means cutting the brain from side to side. And when you cut the brain from side to side you can again see in the periventricular area

dorsally around the trigone zone hyperechogenic areas and within it some areas that are even more hyperechogenic or even more echogenic. So everything that we see on the coronal images is verified on the saggital images. So if I see it on two planes, you're sure it's not some sort of srtifact and it's real.

Now, there are basically two conditions in which you see this kind of pattern on sonograms in a young kid. And the first one is if there's a meningitis with the ventriculitis, okay. And what happens is: The infective process starts in the ventricles and spreads out in the adjacent brain and causes edema and hemorrhage and cavitation. However, from reading through the records, I saw that they did a lumbar puncture and that there was no evidence of meningitis. So it's extremely unlikely that this was a result of infection.

The other cause for this in a premature kid -- and this goes up through about 35 or so weeks of gestation, gestational age -- you see this pattern due to asphyxia. And it's theoretically because the water shed areas of the brain, those areas that are the most susceptible to asphyxic damage are in the periventricular region in premature infants; and it stays in the periventricular region up through about 35 weeks gestational age, and then it starts moving peripherally out to the surface of the cerebral cortex.

So since we know this child was 33 or 34 weeks, this is a common -- this is where you would expect asphyxic damage to occur. And on the basis of these scans I would predict that there was ischemic damage to the brain resulting in this increased number of echoes around the ventricles and that the areas that are even more echogenic are probably little areas of hemorrhagic necrosis. So there's probably some edema and ischemia with some superimposed hemorrhagic necrosis of the brain out there.

MR. NOECKER: Q. What is the significance of your finding of the hemorrhagic necrosis?

A. Well, I -- there are people -- I don't think it's well-proven, but there are people --

Q. You don't need to put those away. I need to follow up OR a few of the things, ?

A. Certainly. There are a few people that think that the areas that undergo hemorrhagic necrosis suffer or are damaged more severely than the other areas. So that's -- I guess that's my interpretation of the sonograms.

Q. At -- As you look at the sonograms, can you come to any conclusion as to when the hypoxia occurred?

A. Well, from this degree of edema, we know that edema is maximum between about two and five days after n insult. So, you know, again, you need comparison studies

to really firmly state whether this is maximal edema or whether it might be on the down side or on the up side. But I would say that probably between about the -- let's see, these were done on the -- around noon on the 5th of September. So I would say sometime between about the 1st of September, maybe the 31st of August, and the 3rd, maybe as late as the 4th of September.

Q. And how do you reach that conclusion?

A. Well, as I said, edema is maximal within about two to five days. And by about a week the edema's usually fairly minimal if there was an insult to the brain, and more than a week you don't usually see it. As a matter of fact, I've seen a lot of cases where a week out you don't see any edema anymore. You have to look real hard for it.

And it takes a while for it to be generated. You know, you have an insult and then there's damage to the vessels; and then the blood -- or the serum, actually, from the blood vessels starts leaking out into what we call the interstitial space, which is the space between the cells. So that takes a while. So you usually don't see it for about 24 hours. And then after about five or six days you usually stop seeing it. So if you use that kind of time frame, it comes out to around, you know, between about the 31st or 1st and about the 3rd or 4th of -- 31st of August, 1st of September, and about

.he 3rd, maybe 4th of September.

Q. Okay. Is there anything that you read in any)f the ultrasounds that would cause you to think that any)f this was caused prior to August 31st?

A. I don't see anything on here. You know, if it 'ere -- the things I'd be looking for would be some sort)f a cavitation, okay, because it takes a while if you iave an area of brain damage for it to actually cavitate ind form a hole, if you will; and there's no evidence of :avitation here yet. So I don't think it was before that :ime. I would be expecting to see further evolution of :hese changes by now.

Q. Other than what you've already told me, is :here any other conceivable cause really for the pattern :hat you're seeing there on the sonogram?

MR. BARNHART: Let me object. "Conceivable cause" is not a legal standard. Go ahead.

MR. NOECKER: Q. Well, it conceivably could be in a differential diagnosis?

A. It's a real specific pattern. It's not a good pattern for metabolic disease, which is much more diffuse. It's not a good pattern for an -- you know, for a direct kind of injury or for most inflammatory processes.

Really about the only things that cause this kind of periventricular damage in a premature newborn are

infection and asphyxia, hypoxic ischemic injury. I would expect the rest of the brain to be a lot more affected than most other processes. It's very localized and, therefore, seems very specific.

Q. As you know, we have Hemophilus flu as infection is an issue in this case.

A. Right.

Q. Have you ever seen a correlation between these sort of patterns and a -- that kind of prenatal infection?

A. Well, in -- if you had H flu meningitis, okay, H flu, or Hemophilus influenza meningitis, can certainly devastate a brain; and I've seen that. But number one, it affects the brain more diffusely than this. If -- just compress vessels or get into vessels and narrow vessels and cause strokes, but it tends to -- when it surrounds a vessel like that and invades a vessel and causes strokes, it tends to be more peripheral. It tends to involve the cerebral cortex and not the periventricular region. When I say, "the periventricular region," you think of what we call a ventriculitis where the infection involves the ventricles and then spreads out in the periventricular area. But in both of those cases a parent should have a grossly positive lumbar puncture. And meningitis is not subtle on lumbar puncture. And for -- to have this kind of an infection

n the face of a normal lumbar puncture, I think, is ssentially impossible.

(Discussion off record.)

MR. NOECKER: Q. If you had an anoxic episode, say, ack in the fourth or fifth month of pregnancy, what kind f a pattern would you expect from that? Is that where ou would expect to see the holes?

A. Well, in the fourth or fifth month --

Q. I'm obviously using that as an example. If .t's something more --

A. Well, what you have to remember is that the pattern of damage that you're going to see is going to lepend, to a large extent, on the age of the fetus at the :ime. So an anoxic event that -- at 8 weeks will give a :otally different pattern than an anoxic event at 15 veeks, or an anoxic event at 25 weeks which would be different than what you see at 34 weeks. So I can't say, €or example, such and such. You tell me the time, and I'll tell you what you'll see.

Q. All right. Is there anything that you would see in any -- anoxic intrauterine event prior to 32 weeks --

A. Okay.

Q. -- that's -- that you don't see any evidence of on these films?

A. Let me -- Repeat your question so I'm sure I

understand what you mean.

Q. Okay.

A. Is there --

Q. Go ahead.

A. Is there something that I would expect to see that I don't see?

Q. Yes. On any sort of anoxic event at any time during the pregnancy up through 32 weeks.

A. Well, that's a really complicated question. But -- okay. I don't see any schizencephaly; I don't see my lissencephaly.

Q. Why don't you explain what they are as you go along.

A. Okay. I'll tell you, up to about 20 to 22 weeks, if there was an asphyxic event, you'd expect to see congenital brain malformation, okay. Might be a genesis of the corpus callosum: might be a neuronal migration anomaly. If it was early enough it might be a holoprosencephaly, which is a very severe anomaly.

When you start getting up to about -- and I don't see any evidence of a congenital brain abnormality. So then you start getting into the time when you start getting to 23, 24, 25, 26 weeks; and at that time you'll see a loss of brain tissue but you don't see any scarring yet because the brain -- up in about 27 or 28 weeks the brain doesn't have the ability to form scar in response

to injury. So at about 24, 25, 26-week injury you'll see loss of brain tissue; you'll see expansion of the ventricles; but you won't see any scarring around the ventricles. And we see -- on later films on the MR we see scarring. So that means that it was at least **28** weeks.

Now, if there were an injury at **28, 29,** 30, even **31** weeks, -- let's see, the kid was -- we don't know exactly how old she was when she was born, but take **33** or **34, I** would say up until even **32** weeks I would expect to see some further progression of these areas of hemorrhagic necrosis in that I'd expect you to start to see some cavitation within them. So if you put the **MR** together with sonogram, I just don't see how this could have occurred back then. **It** looks like something that was subacute at the time of this scan.

And I don't see anything else, such as a congenital brain malformation or enlargement of the ventricles, at this time in response to a prior injury that would suggest something that happened at **24** to **28** weeks. So to answer your question, I don't see anything here that indicates some prior injury or insult; and I don't see anything on the other studies that we haven't talked about either.

Q. So it's your testimony that you're looking at possibly two, three days before birth up until two, three

days post birth as your window of --

A. Yes.

Q. -- in which this damage could have occurred?

A. Yes.

Q. Is there anything to indicate here that this was caused by either acute hypoxia or a chronic hypoxia?

A. I would say that these findings are a lot more compatible with a more chronic nature of hypoxia.

Q. Explain to me what you mean by "chronic."

A. Okay. Here is my definition of acute and chronic. I like to differentiate these cases into two categories. What I call acute total anoxia, and what that usually refers to is either a complete cardiorespiratory arrest with no blood and no oxygen getting to the brain or perhaps a severe abruption, again with no blood getting to the brain. You see a different pattern in that than you see in some kid who's had hypoxia or hypotension for a prolonged period of time. And this looks more like the type of damage you see with hypoxia or hypotension for a prolonged period of time.

(Telephonic interruption.)

Q. When you speak of prolonged period of time, do you have an opinion as to how long she suffered a chronic period of hypoxia?

A. I don't have enough data to say whether -- I mean, I would say it was probably at least half an hour,

but it could be up to probably a couple of days. I just can't -- I don't know. I don't have the -- I don't have the knowledge to be able to say that.

Q. Is there something that you could be provided with to further define your conclusions in that regard, or is it you're deferring to another expert?

A. I don't think -- I don't think anybody knows. I don't think anybody -- There's a combination of factors, okay. It's how severe the hypoxia and hypotension is conpounded upon how long it was. I mean, so if you have a more severe asphyxia for a shorter period of time or a more -- less severe asphyxia for a longer period of time, they'll probably -- you'll probably end up with a very similar picture.

Q. After reviewing the medical records in this case that Mr. Barnhart provided you, did those help you come to your conclusions? And if so, what in there did you base your opinion on?

A. They really didn't help me a lot. Again, I'm not here to talk about any other clinical care. I'm just here to talk about what I can glean from the x-rays.

MR. BARNHART: Scott, you may want to go through the MRIs because a lot of these questions deal with all of them.

MR. NOECKER: Okay.

Q. Why don't we do that. Go to the next films,

and why don't you just for the record tell the Court Reporter what you're working on.

A. Okay. Okay. Well, chronologically the next films were a CT scan from Shands Hospital, and this is dated the 23rd of May 1985.

Q. Did you -- Before we go on, did you read any of the reports that accompanied any of these films?

A. No.

Q. Is there any reason you didn't?

A. Because I -- it wouldn't have affected what I had to say, and I don't like to do that. I -- frankly. I just don't like to read in these cases what other people have said. I don't want to clog up my mind.

Q. Okay.

A. Okay. The CT scan from May of 1985 is -- I'd like to dispense this real quickly because it's just awful, okay. It's a very -- It's an old machine. It's a poor-quality scan. There's a lot of movement by the patient which distorts it even further.

And I will say that the ventricles are big and they have kind of an irregular contour which suggests the sequela of periventricular brain damage. And we saw periventricular brain damage on the sonogram. So that's -- unless you have specific questions, I just assume move on. There's nothing on this film to indicate that there was any change between the appearance of the

brain at this time and the appearance of the brain on the scan in 1988.

Q. Okay. Fine.

A. Okay. And now I'm putting up the -- some films from an MRI scan, magnetic resonance imaging scan, which is dated the 17th of May 1988. And there are four sheets of films from this MR scan. And there are three of them that show most of the findings, and so I'll deal primarily with them, and I'll get -- okay.

Now, the way this scan was done, the advantage to MRI -- or MR we call it now -- is that you can image the brain in any plane, okay. You can image it from side to side. You can image it from front to back. You can image it from top to bottom. You can image it obliquely, and this was done pretty standardly.

This was one sequence which what we call a saggital sequence imaged from side to side. There's one sequence we call an axial sequence imaged from top to bottom, and then there's a sequence that's called the coronal sequence which is imaged from front to back.

The major finding on the saggital image, the side-to-side image, is that the corpus callosum, which is the white matter tract or set of white matter tracts that connect the left hemisphere of the brain to the right hemisphere of the brain, is much too thin, okay, which --

Q. What's the significance of that?

A. When you you see a thin corpus callosum, it's strongly suggestive that there's been damage to most of the white matter tracts. Okay. Those are the axons that connect one part of the brain to another. The axons are the little fibrils that extend out and connect one neuron through another. And when you see a thin corpus callosum Like this, strongly suggest that there's been a lot of damage to the cerebral white matter, to the axons.

And in -- that impression is really verified on the coronal and axial scans. I will look at the coronal scans, cut the brain from front to back. The first finding is that almost all levels of the brain -- here we are in the front: here we are in the back: so it goes from front to back this way.

Q. Why don't you -- When you're referring to those, let's -- for the record let's read them from left to right; that would be the 1, 2, 3 across, 4, 5, 6, 7, 8, 9, 10, 11, 12.

A. So I'll refer to it as "row" and "column."

Q. Why don't you -- and if you're -- okay.

A. There are actual y image numbers on each of these.

Q. That's fine.

A. So what we are looking at is series No. 3 which is coronal, okay. And in series No. 3 on all of the images you can see the lateral ventricle. I'm pointing

but image No. 3. You can see the gray matter from the cerebral cortex comes all the way down to and essentially touches the lateral ventricle. Now, there should be a thick layer of white matter between the ventricle and the cortex. In a normal brain there's always a layer of white matter. And when you see the cortex coming all the way down to and touching -- essentially touching the lateral ventricle it means that there's been a severe insult to the cerebral white matter which is in the periventricular region.

As we come from the back to the front, -- I'm now looking at images 6 and 7 and 8 -- you also see some area of hypointensity, some dark signal adjacent to the ventricle in the white matter.

Q. What's the significance of that?

A. Okay. Now, the white matter is normally white. This is damaged brain.

MR. BARNHART: Can you see that?

MR. NOECKER: M-hm.

A. Okay. This is damaged brain. And you can see it on both sides on several images. And as we come forward (indicating), we still see it. So there's -- you can see some residual damaged brain, but the most striking finding is that there's just not enough brain. There's not enough white matter here. All the way from the back of the brain to the front of the brain the white

matter's missing in the central portion. Now, peripherally, we still see white matter. What we call subcortical white matter is still present. But the periphery -- the central white matter is gone.

And the -- now I'm going to look at sequence 2, okay, or series 2; and these are axial scans to the brain. And one thing you'll notice here is that the cerebral ventricles are now relatively bright as opposed to on these -- in series 2 where the ventricles are dark. That's because this is done slightly differently. This is what we call a T2 weighted image.

What we do is just change the parameters that we use to obtain the scan, and what you do is: You make water or tissues with a lot of water look bright. Now, on these scans the white matter should look dark, and what we see is -- around the ventricles we see a lot of very bright tissue. This is tissue with increased water in it, and it's what we call gliosis.

Q. Called what?

A. G-l-i-o-s-i-s. Gliosis is a fancy word for scar, okay. When the brain is injured, it responds by increasing the number of astrocytes, and the astrocytes there send out more process in the damaged area. Now, another name for astrocytes is glial cells.

So when you get a proliferation of astrocytes the medical term is gliosis. Basically what it is is

scar. And the scar is all in the periventricular area, which is exactly where we saw the damage on the sonogram. And this is exactly the place and exactly the same pattern of damage that you see in kids who have asphyxic injury in the premature-age group up through about 34, maybe 35 weeks. So those are the -- really the significant findings on the MR.

There's -- The CT done the same day really just shows the same findings, only CT isn't as good for showing the gliosis. So all we see is a paucity of white matter and -- and then I guess that's really about it.

So those are the findings, and the findings of all of the scans are consistent with an insult to the brain -- insult to a premature brain, such as this child was, and consistent with that -- although the CT and MR Scans are real nonspecific as to the timing. The sonogram is strongly suggestive of this occurring in a window of time of about maybe, oh, one or two to five or six days before it was obtained, which would be right around the time of birth.

Q. And there's a reason for that is because you didn't find any of the holes that you would have anticipated had an anoxic event occurred prior to that time?

MR. BARNHART: Objection to the form. He gave a great more deal of testimony than simply not finding

holes.

A. You can -- I'll take that. There are certainly holes that can occur at that time; but there weren't any holes in the brain at the time of the sonogram, which indicates that there wasn't -- that the damage that occurred didn't occur weeks before the time the sonogram was done. It takes basically a week or two -- couple of weeks for those holes to appear. We didn't see any evidence at the time of the sonogram.

Q. Okay.

A. And there are holes in the brain here. There's no question about it. But, you know, you can time when they occurred from the sonogram. I would say that if you didn't have that sonogram, that it would be much more difficult to time the insult because I'm -- just on the basis of the CT and MR, it could have occurred any time after about 27 or -- 27 weeks; but the sonogram shows changes that are very specific for a recent insult to the brain.

Q. Do you have an opinion after looking at the MRs and the CTs as to how you would expect or how Christina -- how you would expect Christina to manifest this in either motor or intellectual deficits?

A. Well, with this type of damage, these kids almost always have significant motor deficits and they tend to be spastic. With this much damage I would expect

ıer to basically be quadriparetic. You know, again, she may have a little more function in her arms or in her Legs or on one side or the other, but basically she should be spastic and quadriparetic, and she probably would have significantly more motor dysfunction than intellectual dysfunction because usually these insults get the motor tracts, and intellectual function's usually normal or near normal. I would expect that she may have had initially some visual problems because the optic radiations are in the area that are affected, but usually -- I co-authored a paper on it -- the deficits -- that deficit improves with time.

Q. I want to cover that a little bit -- in a little bit more detail as to what you just related as to her potential optic problems.

A. Yeah.

Q. Are there any indications there of frontal lobe abnormalities?

A. Sure.

Q. Okay.

A. I don't know what that relates to.

Q. Explain in a little more detail, which I think what you started to explain there, as to optic problems.

A. We did a paper. I was, I think, the fourth author on the paper out of the ophthalmology department here where we looked at kids with hypoxic-induced

cortical blindness. It's one of the references I checked fo'r you. And what we found was that the most -- the area that's most sensitive in picking up these problems are the optic radiations. And the optic radiations run right back along the occipital horn to the lateral ventricles. And that area looks damaged.

Mow, when we looked at these kids who have so-called cortical blindness we found that although some of them had diminished visual acuity initially, that a lot of them came back and eventually developed good visual acuity. But again, the ophthalmologist talked about the visual acuity. I reviewed the scans for him blindly and looked at four or five different areas of the brain, gave them the results of which areas were the most severely affected, and then they statistically correlated it with the kid's vision. And I just -- I would refer you to that article.

Q. Okay.

A. "Reviewing the scans blindly" was a bad choice of words.

MR. BARNHART: No pun intended.

MR. NOECKER: Q. Are there any other radiographic exams that you have recommended that she undergo?

A. No.

Q. Okay. Do you see any evidence of any scarring of the optic nerve from any prenatal infection --

A, These scans aren't --

Q. -- on anything that you reviewed?

A. Well, no. There's not enough information on these scans. In order to look at the optic nerve you'd need to do the scan differently; and, you know, I can't even tell you if the optic nerves are atrophic or not on the basis of the scan. The cuts are much too thick. I just -- It's just not done. It wasn't done for that purpose.

Q. I understand. At this stage of her development could those -- could that exam be done if you were -- if you wanted to determine whether or not there had been any optic nerve scarring?

A. Well, you could look at the optic nerves specifically, and you could get some information as to whether there's scarring or not: but I'm not sure you could determine whether the scarring was a result of infection or perhaps asphyxic-type injury.

MR. BARNHART: Scott, off the record.

(Discussion off record.)

MR. NOECKER: If somebody else wants to ask you some questions, I want to look back through my notes. I think I'm just about done.

MR. BARNHART: Anyone else?

MR. BULLOCK: My turn next? I have to look at the guest list.

MR. BARNHART: Go ahead.

MR. BULLOCK: Dr. Lee didn't come. Didn't care anymore.

EXAMINATION BY MR. BULLOCK

MR. BULLOCK: Q. Dr. Barkovich, inquiring on -- on behalf of the physician Dr. Whitlock, is it true that the only type of infection you considered was a meningitis, that is to say, in trying to determine to your satisfaction what might have caused this ischemic incident?

A. Well, I think that's the main thing that you want to rule out with that kind of acute picture. There are a lot of congenital infections that can involve -- so-called TORCH infection, taxoplasmosis, rubella, herpes, encephalitis, syphilis, cytomegalo virus. There's a lot of them. But most of those are not acute at this time. Most of the ones -- The general -- The general feeling about those viruses -- and, you know, I have a lot of about it again in that textbook is that if they occurred during the first trimester they're devastating. If they occurred during the second trimester they are rather less so. And if they occurred during the third trimester they're relatively minor. And you add to the fact that this -- you know, again, without the ultrasound, I think you can make a stronger case for this being some sort of a congenital infection, perhaps.

But it's ongoing at the time on the 5th of September. It's still acute.

Q. Now, did you find in the records you reviewed that this child was thought to be septic at the time she was --

A. Yes.

Q. -- immediately post birth?

A. Right.

Q. And that that was subsequently confirmed?

A. Yes.

Q. And a septicemia does not necessarily involve a meningitis or any, per se, infection of the brain tissue, does it?

A. No.

Q. What is a septicemia in medical terms?

A. Well, it's a generalized infection.

Q. That the blood is carrying infective organisms everywhere it goes?

A. Yes, yes.

Q. Does that include in the case of Christina that the blood was carrying infective organisms throughout her brain tissue?

A. Well, it was -- you have to differentiate the blood from the brain because there's a blood-brain barrier, and everything that's in the blood doesn't get into the brain. As a matter of fact, the brain is the

only organ that has an in-tact barrier between the blood vessels and the parenchyma, and only certain small molecules get through there. Glucose gets through this and a few others. But other than that, there has to be active transport across the barrier *so* --

Q. Do the waste products of bacteria go through the brain barrier and enter the brain tissue?

A. Endotoxin can, I believe.

Q. Endotoxin produces tissue swelling in the area served with the blood?

A. Yes.

Q. Tissue swelling is probably what causes the ischemia that was injurious to Christina, was it not?

A. Yes.

Q. So the swelling that you see that shut down the nourishment of that area of brain could have as easily been produced by endotoxic wastes of blood-born infection as by lack of oxygen, could it not?

A. Well, to the best of my knowledge, Hemophilus influenza does not produce endotoxin.

Q. Is it harmless to brain tissue, its waste products?

A. Well, when we see H flu infections, Hemophilus influenza infections, we don't see this. When we see H flu meningitis, we see terrible brain destruction. But in patients with, say, an H flu pharyngitis or pneumonia,

it's not common or I don't recall seeing brain damage without actual brain infection.

Q. Now, you've taken us back to sites of infection with H flu in your experience, have you not, including the lungs with an H flu pneumonia, the meninges with a meningitis; but have you seen cases in which the septicemia has a widespread carrying of H flu, that is, throughout the bloodstream?

A. Yes.

Q. And that is -- do you know of any localized H flu infection that was demonstrated in Christina at any time?

A. Im trying to think back through the records. I didn't -- No, I don't believe so.

Q. So essentially and crudely put, her blood was full of H flu, but there was no particular organ which this was found to be specifically infected with it?

A. That's what I recall.

Q. And H flu does have an endotoxin?

A, It does? I'm asking a question. It does? Because I didn't believe that it does.

Q. If we assume that the ischemic changes you've observed in Christina's brain were due to a chronic hypoxia or low oxygen level, would that be consistent with several hours of blood oxygen of less than 40?

A. Well, I think I already answered that question

:hat I think there were several combinations of blood oxygen level and duration that could cause this.

Q. Anywhere from a half an hour to several hours, you felt?

A. Well, I think --

MR. BARNHART: I think it was a half hour to several days.

THE WITNESS: Yeah.

MR. BULLOCX: Q. I was trying to get a more definite answer than -- but you'll stand with what the record says so far?

A. Yeah, I don't have a more definite answer. If I did, I would have given it the first time.

Q. And high potential would be a low blood volume and, therefore, a lessened ability of the blood to transport oxygen to the brain cells?

A. Well, not necessarily a lower blood volume, but it can be due to lack of pumping of the heart. It's just less blood getting to the brain. It doesn't necessarily mean that there was less blood volume. But yeah, less blood and, therefore, less oxygen getting to the brain.

MR. BULLOCX: I have no further questions.

(Discussion off record.)

EXAMINATION BY MR. FACCIOLO

MR. FACCIOLO: Q. Doctor, when you looked at the earliest studies of Christina's brain they indicated

enlargement of the lateral ventricles, did they not?

A. No, not the sonogram, which was the earliest study.

Q. Did you have a CAT scan study from Plantation Hospital of September the 23rd?

A. No.

Q. And you were provided no report of that CAT scan by Dr. Ayjee (phonetic)?

A. I haven't -- I haven't seen the report, and I haven't seen the CAT scan.

Q. Excluding any other contributing condition or pathology, would you expect an injury that occurred secondary only to a hypoxic ischemic injury at this time of life, 31st weeks' gestation in the fetus, to create a significantly marked dilatation of the ventricles on one side of the brain and obviously to a much lesser e'xtent on the other?

A. I've seen cases like that. There's actually a report out of the University of Cincinnati; the senior author was Myers, M-y-e-r-s, where he showed experimentally that you can get asymmetric brain damage and hypoxic ischemic injury in monkeys, and he undertook that after he saw a couple of examples in humans of asymmetric brain damage. So I guess I'm not that surprised.

What surprises me more than that is that the

amount of brain damage seems very symmetrical in 1985, May of '85, the first CT scan that I was given, and in September of '88. So I guess I'd be a little puzzled if there was marked asymmetry of the ventricles at that time and not later.

Q. If we assume that condition to exist, have you any explanation for the process that would explain such a change from the early scans to the later scans, if in fact the early scans demonstrated marked dilatation on the right side of this child's brain and then the later scans showed a more symmetrical damage to --

A. Well, I would prefer to see the scans rather than comment on a hypothetical scan. I can come up with a couple of scenarios that could do that, but, you know, -- but I don't -- but I'd like to see the scan because a lot of times the scan is done a little asymmetrically, and it makes one ventricle appear bigger than the other one when in fact it's not. A lot of times there's some low density in the white matter around the damaged ventricle, which people misterm as an enlarged ventricle. So I would just -- I guess I would prefer to see the scan before I would render judgment on that.

Q. Based on the scans or studies that you've had an opportunity to have reviewed, would you anticipate that the motor loss suffered by this child would be basically symmetrical or that she would have a

substantially greater damage or -- near the left or the right side of her body?

A. Based on these scans, I would expect it to be roughly symmetrical. Yeah, I would expect it to be roughly symmetrical. Again, you know, as I stated earlier, sometimes one side of the body's a little more affected than the other, the arms more or the legs more than the arms. But I can't on the basis of this scan predict that one side would be significantly more affected than the other, no.

Q. With the sonogram, the earliest sonogram that you had available to you, -- which was dated when?

A. The 5th of September.

Q. The year of the child's birth?

A. Yeah.

Q. -- could you exclude from that sonogram the presence of some degree of hydrocephalus?

A. I think so. The ventricles are very small. I don't think there's any degree of hydrocephalus there.

Q. If within two weeks the ventricles of the right side became significantly enlarged, would some degree of hydrocephalus tend to explain that pathology?

A. Well, it's real hard to explain hydrocephalus enlarging one ventricle and not the other. So I don't think so. I mean, hydrocephalus is a symmetric process usually, and every once in a while you can get a -- very

ocalized lesions. It's usually a tumor at the outlet of one of the lateral ventricles that will cause enlargement of one ventricle and not the other, but it's real common. I don't think that since we have follow-up scans that don't show a tumor, it's unlikely that there was one there.

MR. FACCIOLO: Thank you, Doctor. That's all.

MR. BARNHART: No questions.

MR. SCHODER: Doctor, I have one.

THE WITNESS: Sure.

EXAMINATION BY MR. SCHODER

MR. SCHODER: Q. My name is Tony Schoder, and I represent Dr. Padron.

With regard to this window in which you've indicated that the sonogram reflects edema which would have taken place wherever hypoxic event had caused it would take place one, two days before and up to three or four days after birth, -- or is that the right time? Or whatever time you said.

A. Yes, that's approximately right.

Q. Okay. Somewhere in the first of the two explanations you gave you said something about 24 hours, that you would not expect to see anything on a sonogram until at least 24 hours after the hypoxic event. What was that about?

A. It takes a while to produce the edema or for

the edema to become severe. What happens is that when you have -- I mean, I'm talking about asphyxic damage here as a rule. The asphyxia causes damage to the blood vessel because the blood vessel is not getting enough oxygen either, and so it stops performing its metabolic activities, the cells and the blood vessel wall. And so the blood-brain barrier that I talked about starts to deteriorate, and fluid from in the blood vessel starts to leak out into the area between the cells; interstitial space we call it. It takes a while for that to happen. But -- and exactly how long it takes, I guess, is a matter of debate.

But you don't usually start seeing edema on a sonogram. If -- We usually start seeing it in a day or so. And I don't think 24 hours is rigid. You know, you could probably -- you could probably see it as soon as 12 hours.

Q. Could you see it as late as 36 or 48 hours after a hypoxic incident?

A. You'd see it at that point, but the onset would -- which would almost certainly be before that because within about 48 hours the edema starts becoming maximal; and then it stays maximal for -- like I said, from about two to five days, and then it starts to abate. So I'd be surprised if you didn't see it for 24 -- for -- I would be surprised if you didn't see it for 36 or 48

hours.

But none of those numbers are absolute. And I think, you know, -- I think, as I said, you know, the scan was probably five or six days to one or two days after the -- after whatever event occurred. And -- but I'm not saying, you know, bang bang.

Q. Is there any way other than looking at those sonograms as to whether or not any of the edema seen on the sonogram had occurred in fact within two days of taking of the sonograms that you know?

A. There's no way to tell.

MR. SCHODER: Thank you, Doctor.

MR. WIESENFELD: Doctor, have you formed any conclusions or opinions in this matter in addition to what you've already expressed during the course of this deposition?

THE WITNESS: No.

MR. WIESENFELD: Thank you.

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(WHEREUPON, Defendant's Exhibit A was marked for identification.)

(Whereupon, the deposition of ANTHONY JAMES BARKOVICH concluded at 10:45 a.m. on April 23, 1990.)

---ooo---

I declare under penalty of perjury that the foregoing is true and correct, including any changes I made in my answers today.

Dated _____ at _____
(City, State)

_____
ANTHONY JAMES BARKOVICH, M.D.

Subscribed to before me this ___ day of _____, 1990.

_____
NOTARY PUBLIC, STATE OF CALIFORNIA

PRINCIPAL OFFICE: COUNTY OF _____.

STATE OF CALIFORNIA)
) ss.
COUNTY OF SAN MATEO)

I, CHRISTINE M. NICCQLI, Certified Shorthand Reporter, License No. 4569, and a Notary Public in and for the County of San Mateo, State of California, do hereby certify:

That the deponent in the foregoing deposition was by me duly sworn and that this transcript is a true record of the testimony given and of any changes made by said deponent.

IN WITNESS WHEREOF, I have hereunto set my hand and affixed my seal of office this 3rd day of May, 1990.

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CHRISTINE M. NICCOLI
Notary Public-California
SAN MATEO COUNTY
My Comm. Exp. July 12, 1991

Christine M. Niccoli
NOTARY PUBLIC

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Date: May 4, 1990

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RE: LONG v. CORRAL
REPORTER: Christine Niccoli
DEPOSITION DATE: 04/23/90
TRIAL/ARB. DATE: --

Please find enclosed herewith the transcript of your deposition taken in the above-referenced matter for reading and correcting.

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After reviewing your testimony, we request your subscribing to same on the enclosed original signature page in the presence of a Notary Public.

Please return said enclosures to this office at your earliest convenience. Thank you for your cooperation.

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Marital Status: Married, May 24,1986 to Karen K. Jernstedt
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1973	Ames Research Lab U.S. Atomic Energy Comm.	Research Assistant
1974-1977	Chemistry Dept. Univ. of CA, Berkeley	Research Assistant Teaching Assistant
1974-1989	U.S. Army	Medical Student Intern Radiology Resident Neuroradiology Fellow
1989-present	University of California San Francisco	Associate Professor of Radiology, Pediatrics and Neurological Surgery in Residence

EDUCATION:

DATE	SCHOOL	MATOR	DEGREE
1966-1970	Redwood High School Larkspur, CA		
1970-1974	Univ. of CA, Davis	Chemistry	B.S.
1974-1977	Univ. of CA, Berkeley	Chemistry	M.S.

DEFENDANT'S EXHIBIT A
BARKOVICH 4/23/9

DATE	SCHOOL	MATOR	DEGREE
	RESEARCH - Synthesis and characterization of the electron energy levels of strained, cyclic, unsaturated hydrocarbons		
	Passed Oral Examination for Ph.D., was advanced to candidacy €or Ph.D., but did not complete dissertation		
1976-1980	Geo. Washington Univ. Washington, D.C.	Medicine	M.D.
1980-1981	Letterman AMC §an Francisco, CA	Radiology	Intern
1981-1984	Letterman, AMC San Francisco, CA	Radiology	Resident
1984-1986	Walter Reed AMC Washington, D.C.	Neuroradiology	Fellowship

Included in fellowship:

Four weeks	Johns Hopkins Hospital, Baltimore, M.D.
Six weeks	Children's Hospital NMC, Washington, D.C.
Six weeks	Hospital for Sick Children, Toronto, Canada
One hour weekly	Armed Forces Institute of Pathology

MILITARY HISTORY:

1976-1980	Served in U.S. Army Reserves as a medical student
1980-1989	Served as Major in U.S. Army

BOARD CERTIFICATION:

National Board of Medical Examiners, 1981

American Board of Radiology, 1984

LICENSURES:

California G 46445

PROFESSIONAL SOCIETIES:

The Radiology Society of North America
The American Society of Neuroradiology (Senior Member)
The Phi Kappa Phi Honor Soaety
Western Neuroradiological Society

AWARDS:

Edward F. Kraft Scholarship Award, U.C. Davis, 1971
President's Undergraduate Research Fellow, 1973-1974
U.S. Atomic Energy Commission Summer Research Fellow, 1973
Honorable Mention, Exhibit, RSNA Annual Meeting, **1983**

ACADEMIC POSITIONS:

Instructor in Radiology, F. Edward Hebert School of Medicine, Uniformed Services University of Health Sciences, 1984-1987

Assistant Clinical Professor of Radiology, University of California, San Francisco, 1986-1989

Associate Professor of Radiology, Pediatrics **and** Neurological Surgery in Residence, University of California, San Francisco, 1989-present

Assistant Clinical Professor of Radiology, Uniformed Services University of Health Sciences, 1987-1988 ?

PRESENT POSITION:

Associate Professor of Radiology, Pediatrics and Neurological Surgery in Residence, University of California, San Francisco

Consulting Pediatric Neuroradiologist, Division of Pediatric Neurosurgery, Univ. of California, San Francisco

TEACHING:

Radiology Residents
UCSF -- One hour-long lectures per month
Letterman Army Medical Center -- One lecture per month
Informally, neuroradiology readout, four hours per day

Neurology/Pediatric Residents
UCSF -- One hour-long session per week
Informally, via consultation, one hour per day

Teaching contd

Neurology/Pediatric/Neurosurgery/Radiology Residents
Biweekly one hour Pediatric Neuroradiology conference at UCSF

Neurosurgery/Radiology Residents, Neuroradiology Fellows (Pediatric)
Neuroradiology Grand Rounds, four times a year

APPOINTMENTS:

Member of Editorial Advisory Board, American Journal of Neuroradiology
Training and Standards Committee, American Soaety of Neuroradiology

INVITED LECTURES -- UCSF:

Congenital Anomalies of the Brain. UCSF Annual Neuroradiology Course, January 1987

Anomalies of the Spine. UCSF Annual Neuroradiology Course, January 1987

Normal and Abnormal Myelination. UCSF Annual MRI Course, October 1987

Practical Pediatric Neuroradiology. UCSF Annual MRI Course, October 1987

Neuro MR: A New Window to the Pediatric Brain. UCSF Radiology Grand Rounds, February 18,1988

The Effect of MR on Pediatric Neuroimaging. Annual UCSF Diagnostic Radiology Course, March 1988

MR of Normal Brain Maturation. UCSF Annual Clinical MRI Course, October 1988

New Concepts in Developmental Brain Anomalies. UCSF Annual Clinical MRI Course, October 1988

Developmental Spine Anomalies. UCSF Annual Clinical MRI Course, October 1988

MR of the Phakomatoses. UCSF Annual Diagnostic Radiology Course, March 1989

INVITED LECTURES :

CT Anatomy of the Temporal Bone. Armed Forces Institute of Pathology Otolaryngology Review Course, May 1985 and April 1986

CT of Temporal Bone Pathology. Armed Forces Institute of Pathology Otolaryngology Review Course, May 1985 and April 1986

Invited Lectures contd.

Metabolic and Destructive Brain Disorders. Neurology/Neurosurgery Grand Rounds, Mary Hitchcock Clinic, Dartmouth Medical School, Hanover NH, October 1988

Developmental Brain Disorders. Radiology Department, Mary Hitchcock Clinic, Dartmouth Medical School, Hanover, NH, October 1988

Pediatric Neuroradiology: The Maturing CNS. Refresher Course (with Thomas P. Naidich, MD), Annual Meeting of the RSNA, Chicago, November 1988

Developmental Brain Anomalies. Barrows Neurological Institute Postgraduate Radiology Course, Phoenix, AZ, May 1989

Developmental Spine Anomalies. Barrows Neurological Institute Postgraduate Radiology Course, Phoenix, AZ, May 1989

Metabolic Diseases of the Brain. Barrows Neurological Institute Postgraduate Radiology Course, Phoenix, AZ, May 1989

MR of Normal Brain Development. Children's Hospital of Los Angeles, Los Angeles, CA, October 18,1989

Supratentorial Tumors of Childhood. ASNR categorical course on Brain Tumors, Los Angeles, CA, March 17,1990.

MR of the Pediatric Spine. Society for Magnetic Resonance Imaging Categorical Course. Washington, DC, February 24,1990.

PROGRAMS DEVELOPED AND DIRECTED:

Program Director, Pediatric Neuroradiology Course. Hyatt-Regency-Embarcadero Hotel, San Francisco, CA, October 22-23,1988.

Program Director, Pediatric Neuroradiology Course. Hyatt-Regency-Embarcadero Hotel, San Francisco, CA, October 7,1989

PUBLICATIONS:

WGL Aalbersberg, **AJ Barkovich,** RL Funk, RL Hilliard and KPC Vollhardt. Transition metal catalyzed acetylene cyclizations. 4, 5-Bis-(trimethylsilyl) benzocyclobutene, a highly strained, versatile synthetic intermediate. J Am Chem Soc, 97,5600 (1975)

Publications contd.

AJ Barkovich and KPC Vollhardt, 1, 5, 9-cyclododecatriyne. Synthesis, and conversion to intermediate 1, 2:3, 4:5, 6-Tricyclobutabenzene, J Am Chem Soc, 98, 2667 (1976)

AJ Barkovich, ES Strauss and KPC Vollhardt, Hexaradialene, J Am Chem Soc, 99, 8321 (1977)

AJ Barkovish, ES Strauss and KPC Vollhardt, Unusual rearrangements in the C12-H12 Manifold: 1, 5, 9-cyclododecatriyne 1, 2:3, 4:5, 6-Tricyclobutabenzene, Hexaradialene, Israel J Chem, 20, 225 (1980)

DF Sobel, **AJ** Barkovich and SM Munderloh, Metrizamide myelography and postinyelographic computed tomography: comparative adequacy in the cervical spine, AJNR, 1984; 5:385-390

AJ Barkovich, FJ Wipgold and RE Brammer, False negative MR in an acoustic neurinoma, AJNR, 1986; 7:363-364

AJ Barkovich, FJ Wippold, JL Sherman and CM Citrin, Significance of cerebellar tonsillar position on ***MR,*** AJNR, 1986; 7:795-800

AJ Barkovich, CM Citrin, PM Klara, FJ Wippold and J Kattah, MRI of cysticercosis, West J Med, 1986; 145:687-689

JL Sherman, **AJ** Barkovich and CM Citrin, MR of syringomyelia: New observations, AJNR, 1986; 7:985-995

JL Sherman, CM Citrin, **AJ** Barkovich and BJ Bowen, MR imaging of the mesencephalic tectum: Normal and pathologic variations, AJNR, 1987; 8:59-64

AJ Barkovich, JL Sherman and CM Citrin, MR of post-operative syringomyelia, AJNR, 1987; 8:279-287

JL Sherman, CM Citrin and AJ Barkovich, MR of syringobulbia, J Comput Asst Tomogr 1987; 11:407-411

FJ Wippoid, CM Citrin, AJ Barkovich and JL Sherman, Evaluation of MR of spinal dysraphism with lipoma, comparison with CT, Pediatric Radiology 1987; 17:184-188

AJ Barkovich, SH Chuang and D Norman, Anomalies of neuronal migration: MRI, AJNR 1987; 8:1009-1017, AJR 1988; 150:179-187

Publications contd

SR Lambert, CS Hoyt, JE Jan, **AJ Barkovich** and O Flodmark, Visual recovery from hypoxic cortical blindness in childhood: CT and MRI predictors, Arch Ophthmol 1987; 105:1371-1377

AJ Barkovich, D. Norman, MR of schizencephaiy, **AJNR** 1988; 9:297-302, AJR 1988; 150:1391-1395

AJ Barkovich, BO Kjos. Normal post-natal development of the corpus callosum as determined by MR. **AJNR** 1988; 9:487-491

AJ Barkovich and D Norman, Anomalies of the Corpus Callosum: Correlation with further anomalies of the brain. AJNR 1988; 9:493-501, AJR 1988; 151:171-179

AJ Barkovich, D Jackson, BO Kjos and D Norman, MR of normal brain maturation at 1.5T. Radiology 1988; 166:173-180

AJ Barkovich. Abnormal venous drainage in migration anomalies. **AJNR** 1988; 9:939-942

AJ Barkovich, D Norman. Absence of the septum pellucidum: a useful **sign** in the diagnosis of congenital brain malformations. AJNR 1988; 9:1107-1114. AJR 1989; 152:353-360

AJ Barkovich, SW Atlas. MR of intracranial hemorrhage. Radiological Clinics of North America. 1988; 26:801-820

AJ Barkovich. Techniques and methods in pediatric MR. Seminars in CT, Ultrasound, and MR. 1988; 9:186-191

SR Pollei, RS Boyer, S Crawford, HR Harnsberger, **AJ Barkovich.** Disorders of migration and sulcation. Seminars in CT, Ultrasound and MR 1988; 9:231-246

N Raghavan, **AJ Barkovich,** MSB Edwards, D Norman. MR imaging in the tethered cord syndrome. AJNR 1989; 10:27-36

AJ Barkovich, TH Newton. MR of aqueductal stenosis: Evidence of a broad spectrum of tectal distortion. AJNR 1989; 10:471-476

AJ Barkovich, BO Kjos, D Norman, MSB Edwards. New concepts in posterior fossa cyst-like malformations obtained by rnultiplanar imaging. AJNR 1989; 10:977-988

Y Okada, S Aoki, AJ Barkovich, K Nishimura, D Norman, BO Kjos, RC Brasch. Cranial bone marrow in children: Assessment of normal development with MR imaging. Radiology 1989; 171:161-164

Publications contd

AJ Barkovich, E Framm. D Norman. MRI of septo-optic dysplasia. Radiology **1989; 171:189-192**

MA King, AJ Barkovich, VV Halbach, **GB Hieshima, MS Edwards.** Traumatic monocular blindness associated with carotid injuries in children. Pediatrics 1989; **84:128-132**

AJ Barkovich, DE Jackson, Jr, R Boyer. Band heterotopias: a newly recognized form of migration anomalies. Radiology **1989; 171:455-458**

S Aoki, WP Dillon, AJ **Barkovich,** D Norman. Marrow conversion prior to pneumatization of the sphenoid sinus: MR assessment. Radiology **1989; 172:373-375**

S Aoki, Y Okada, K Nishimura, **AJ Barkovich,** BO Kjos, RC Brasch, D Norman. Development of brain iron in childhood and adolescence on T2 weighted images at 1.5T. Radiology **1989; 172381-385**

RA Filly, JD Cardoza, RB Goldstein, **AJ Barkovich.** Detection of fetal CNS anomalies: **A** practical level of effort for a "routine" sonogram. Radiology 1989; 172:403-408

S Aoki, **AJ Barkovich,** K Nishimura, BO Kjos, T Machida, P Cogen, D Norman, **MSB** Edwards. neurofibromatosis type I and II: Cranial MR findings. Radiology **1989; 172527-534**

AJ Barkovich, DE Jackson. MR of normal and abnormal myelination. MRI Decisions **1989;** 3:17-25

AJ Barkovich, N Raghavan, SH Chuang. MR of lumbosacral agenesis. AJNR **1989;** 10:1223-1231

A.J.Barkovich. Apparent atypical callosal dysgenesis: Analysis of MX findings in six cases and their relationship to holoprosencephaly. AJNR 1990; 11:333-339

PAPERS IN PRESS:

R. Tien, **AJ Barkovich,** MSB Edwards. MR in the evaluation of pineal region masses. AJNR March, 1990

PK Maurer, AJ Barkovich. Locations of cervical disc herniations: implications for surgical approach. Neurosurgery

AJ Barkovich, SH Ckuang. MR of unilateral megalencephaly. AJNR

Papers in press contd.

AJ Barkovich, CL Truwit. MR of perinatal asphyxia: correlation of gestational age with pattern of damage. AJNR

CL Truwit, **AJ Barkovich,** A Gear-Martin, Nittibri, D Norman. Loss of the insular ribbon: A new CT sign of acute infarction. Radiology

CL Truwit, **AJ Baxkovich.** Intracranial lipomas: A new unifying concept. (AJNR)

PAPERS SUBMITTED FOR PUBLICATION:

BO Kjos, R Umansky, **AJ Barkovich.** MR of the brain in children with developmental retardation of unknown cause (submitted to **AJNR)**

DA Kaku, AJ **Barkovich,** RJ Shanahan, CM Greco. MRI suggesting leukodystrophy in Leigh disease. (Submitted to Annals of Neurology.)

AJ Barkovich, MSB Edwards, PN Cogen. MR and CT of congenital nasal masses. (submitted to AJNR)

CHAPTERS:

AJ Barkovich. The effect of MR on Pediatric Neuroradiology. Diagnostic Radiology, 1988; AR Margulis, **CA** Gooding, eds. pp. 399-406

AJ Barkovich. Imaging of the Phakomatosis. Diagnostic Radiology, 1989. AR Margulis, CA Gooding, eds. p. 315-329

TEXTBOOKS:

AJ Barkovich. Pediatric Neuroimaging. Raven Press, 1989

PRESENTATIONS:

G Sze, F Pardo, J DeGroot, W Kucharczyk, W Olsen, J Kucharczyk, **AJ Barkovich,** G Krol, D Norman M Brant-Zawadski, TH Newton. Magnetic resonance-anatomic correlation in the pituitary fossa. Western Neuroradiological Society Annual Meeting, Palm Springs, CA 1986

N Colombo, I Berry, J Kucharczyk, W Kucharczyk, J DeGroot, **AJ Barkovich,** T Larson, G Sze, W Peck, M Brant-Zawadski, D Norman, H Newton. MRI of the posterior pituitary bright spot. Western Neuroradiological Society Annual Meeting, Palm Springs, CA 1986

Presentations contd.

J Kucharczyk, **I Berry,** J DeGroot, M Wedland, M Mosley, G Sze, W Kucharczyk, N Colombo, **AJ Barkovich,** D Norman, T Newton. The chemical nature and functional significance of the posterior pituitary "bright spot" Western Neuroradiological Society Annual Meeting, Palm Springs, **CA, 1986**

AJ Barkovich, SH Chuang. MR of migration anomalies of the CNS. Western Neuroradiologicai Society Annual Meeting, Palm Springs, CA, **1986**

AJ Barkovich, **SI3** Chuang and D Norman. Anomalies of neuronal migration: MRX, Presented at the 25th Annual Meeting of the American Society of Neuroradiology, New York, **1987**

AJ Barkovich and D Norman, The callosal key to brain anomalies. Presented at the 25th annual meeting of the American Society of Neuroradiology, New York, **1987**

SH Chuang, **AJ** Barkovich, DC Harwood-Nash. Unilateral Megalencephaly. American Society of Neuroradiology Annual Meeting, New York, 1987

T Larson, **AJ Barkovich,** J DeGroot, D Norman, TH Newton. MR of the Cerebellum. American Society of Neuroradiology Annual Meeting, New York, **1987**

AJ Barkovich, BO Kjos, D Jackson, D Norman. MR of Normal Brain Maturation. Western Neuroradiological Society Annual Meeting, Scottsdale, AZ, **1987**

T Larson, **AJ Barkovich,** MS Edwards, D Norman, TH Newton. MR of the Chiari I, 11, and 111 malformations. Western Neuroradiological Society Annual Meeting, Scottsdale, AZ, **1987**

AJ Barkovich, 3O Kjos, D Jackson, D Norman. MR of normal brain maturation. Presented at the annual meeting of the Radiological Society of North America, Chicago, **1987**

CF Dowd, SW Atlas, WF Hoyt, **AJ** Barkovich, D Norman. Visual Pathway Gliomas in Neurofibromatosis Patients: MR Characteristics. Presented at the annual meeting of the Society of Magnetic Resonance Imaging, Boston, March, **1988**

SW Atlas, AS Mark, E Fram, **AJ** Barkovich, D Norman. Aqueductal Stenosis: Evaluation with gradient echo aquisition rapid MR imaging. Presented at the annual meeting of the Society of Magnetic Resonance Imaging, Boston, March, **1988**

AJ Barkovich, D Norman. The tectal key to aqueductal pathology. Presented at the annual meeting of the American Society of Neuroradiology, Chicago, May, 1988

Presentations contd.

AJ Barkovich, BO Kjos, MSB Edwards, D Norman. New concepts in posterior fossa cysts in children. Presented at the annual meeting of the American Society of Neuroradiology, Chicago, May 1988

DK Haas, S Chuang, DC Hanvood-Nash, AJ **Barkovich,** J Laidley, L Becker, R Coates. MELAS syndrome: Multiple recurrent strokes in children with diagnostic muscle mitochondrial findings. Presented at the annual meeting of the American Society of Neuroradiology, Chicago, May, 1988

N Raghavan, **AJ** Baxkovich, D Norman. MR of the tethered spinal cord syndrome: New findings. Presented as a poster at the annual meeting of the American Society of Neuroradiology, Chicago, May, 1988

WP Dillon, **AJ** Barkovich. Asymmetric fusion of the septum pellucidum: A cause for "pseudo" unilateral obstructive hydrocephalus. Presented at the annual meeting of the Western Neuroradiological Society annual meeting, San Diego, Oct, 1988

DE Jackson, Jr., **AJ** Barkovich. Band Heterotopias: **A** newly recognized form of migration anomalies. Presented at the annual meeting of the Western Neuroradiological Society, San Diego, **CA,** October, 1988

AJ Barkovich, S Aoki, K Nishimura, D Norman. Cranial MR of neurofibromatosis I and II: two distinct diseases. Presented at the annual meeting of the ASNR, Orlando, March, 1989

RD Tien, AJ **Barkovich,** D Norman. The value of MR in the diagnosis of pineal region tumors. Presented at the annual meeting of the ASNR, Orlando, March, 1989

RD Tien, AJ Barkovich, D Norman. The value of MR in the diagnosis of pineal region tumors. Presented at the annual meeting of the ASNR, Orlando, March, 1989

RD Tien, **AJ** Barkovich, TM Newton. MR and CT evaluation of pituitary infundibuluin lesions. Presented at the annual meeting of the ASNR, Orlando, March, 1989

EXHIBITS:

S Munderloh, AJ Barkovich, and N Duenas. A New Contrast Agent for CT of the Liver and Spleen. Shown at the Radiological Society of North America Annual Meeting, Chicago, 1983

Exhibits contd.

M Brantley, AJ **Barkovich,** *FJ* Wippold and JM McCabe, MR of Intracranial Vascular Malformations. Shown at the Radiological Society of North America Annual Meeting, **Chicago,** 1986 and at the annual meeting of the American Roentgen Ray Society, Miami, 1987

RESEARCH ACTIVITIES:

1. Neonatal Asphyxia

   a. Effects of myelination on the pattern of asphyxic brain damage in neonates. We are inducing asphyxia in neonatal pigs prior to, during, and after myelin formation in the cerebrum. Hypothesis: the increased metabolic activity of myelin syntheses renders actively myelinating areas more susceptible to asphyxic damage. We will follow myelination using known signal changes in the white matter on MR imaging and the known disappearance of the phosphomonoester peak on in vivo MR spectroscopy.

   b. Patterns of asphyxic brain damage. **A** retrospective study is under way in an attempt to correlate patterns of brain damage (as detected by ***MR*** imaging) with the duration and severity of the insult and with the gestational age of the patient at the time of the insult. The MR assessment of the degree of brain myelination of the neonatal and infant brain has been deduced (Barkovich, et. al., Radiology 1988; 166:173-180).

   c. Eventually, we hope to study the effects of calcium channel blockers (known to attenuate the effects of ischemia in adults) on perinatal asphyxia.

2. Syringohydromyelia

   Myelination is the most effective modality in the detection of syringohydromyelia and in the evaluation of its treatment (JL Sherman, AJ Barkovich, and CM Citrin, AJNR 1986; 7:985-995, AJ **Barkovich,** JL Sherman, and CM Citrin, AJNR 1987; 8:279-287). Technological advances in the past year have made it possible to quantitate CSF flow in the subarachnoid space and, consequently, within the syrinx cavity. We are presently comparing CSF flow at the foramen magnum in patients with obstruction to CSF flow at the foramen in patients with obstruction to CSF flow at that level with and without syringes to normals in an attempt to discover exactly what alterations of flow are necessary to cause syrinx formation.

3. Normal and abnormal brain development

   MR imaging allows gross pathological examination of the brain in vivo in any plane. When proper techniques are used, exquisite anatomic detail can be

obtained. This anatomic information can be combined with **known** embryologic data to deduce additional facts pertaining to embryology and developmental pathology **(Barkovich** and Norman, **AJNR** 1988; 9:493-501, **Barkovich** and Norman AJNR 1988; 9:297-302, **Barkovich** et al., Radiology 1989; 171:455-458).