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1 IN THE COURT OF COMMON PLEAS OF CUYAHOGA COUNTY, OHIO 2 3 KEVIN KISS, a minor, by and through his next friend 4 and natural mother, Anne Kiss, et ai., 5 Plaintiffs, 6 vs. 7 ANDREAS MARCOTTY, M.D 8 et al., 9	 BRUCE H. COHEN, M.D., of lawful age, called for examination, as provided by the Ohio Rules of Civil Procedure, being by me first duly swom, as hereinafter certified, deposed and said as foilows: EXAMINATION OF BRUCE H. COHEN, M.D. BY MS. TOSTI: Q. Doctor, would you please state your full name for us. A. Bruce Howard Cohen. Q. And what is your home address? A. 26525 Amhearst Road, Beachwood, Ohio, 44122.
DEPOSITION CF BRUCE H. COHEN, M.D. Monday, February 26, 2001 Deposition of BRUCE H. COHEN, M.D., called by the Plaintiffs for examination under the statute, taken before me, Karen M. Patterson, a Registered Merit Reporter and Notary Public in and for the State of Ohio, pursuant to notice and stipulations of counsel, at the offices of Cleveland Clinic Foundation, <i>9500</i> Euclid Avenue, Cleveland, Ohio, on the day and date set forth above, at 2: 10 o'clock p.m. 23 24	 Q. Is that a single-family home, doctor? A. That's an apartment. Q. Do you have an apartment number? A. 106. Q. And is your current business address here at Cleveland Clinic's main campus? A. That's correct. Q. At the time that you rendered care to Kevin Kiss, was your business address also here at Cleveland Clinic's main campus? A. Yes. Q. In April of 1998, were you seeing
2 APPEARANCES: 3 On behalf of the Plaintiffs: 4 Becker & Mishkind Co., L.P.A., by JEANNE M. TOSTI, ESQ. 5 Suite 660 Skylight Office Tower 1 660 West Second Street 6 Cleveland, Ohio 44 113 (16) 241-2600 7 On behalf of the Defendant Andreas 8 Marcoury, M.D.: 9 Mazanec, Raskin & Ryder Co., L.P.A., by 10 D. CHERYL ATWELL, ESQ. 100 Franklin's Row 11 34305 Solon Road Cleveland, Ohio 44139 12 (440) 248-7906 13 On behalf of the Defendant Cleveland Clinic Foundation: 14 Roetzel & Andress, by 15 ANAC CARULAS, ESQ. 16 1375 East Ninth Street 17 Cleveland, Ohio 44114 (216) 623-0150 18 On behalf of the Defendant Signature Eye 19 Associates: 20 Ulmer & Berne LLP, by 21 POD Bond Court Building 1300 East Ninth Street 22 Cleveland, Ohio 44114 (216) 621-8400	 4 1 patients anywhere besides the main campus of 2 Cleveland Clinic? A. I may have also been seeing patients 4 out at Kaiser Beachwood, but I'm not sure. Q. Would that have been on a consulting basis or on a regular basis? A. Consulting. That would be probably 8 one afternoon every one to two months, but I 9 can't remember when I stopped going out to Kaiser 10 Beachwood. 11 Q. And who is your current employer? 12 A. The Cleveland Clinic Foundation. 13 Q. And was that also true at the time 14 that you rendered care to Kevin Kiss? 15 A. Yes. 16 Q. Aside from the professional services 17 that you provide for Cleveland Clinic Foundation, 18 do you provide for Cleveland Clinic Foundation, 19 other entity? 20 A. I do occasional consulting work with 21 drug companies. 22 Q. Any particular drug company? 23 A. Rhone-Poulanc Rorer. I no longer do 24 consulting work with them. 25 Q. So you currently are not consulting

1 (Pages 1 to 4)

 5 1 with them? 2 A. Correct. 3 Q. And in the time period when you 4 rendered care to Kevin Kiss, were you providing 5 professional services for any other entity 6 besides Cleveland Clinic? 6 A. No. 9 A. No. 9 A. Have you ever had your deposition 9 taken before? 10 A. Yes. 11 Q. How many times? 12 A. I don't remember. 13 Q. Approximately, doctor. 14 A. A dozen. 15 Q. How many of those times were in a 16 medical negligence case? 17 MS. CARULAS: Note my objection. 18 I'll have a continuing line of objection to this, 19 but go ahead. 20 A. Ail but one. The other one was a 21 rape case, and one was a murder case, all in the 22 context of a medical basis. 23 Q. In those other instances that were 24 medical negligence cases, were any of those cases 25 ones in which you were named as a Defendant in 	 I would also ask that you give ail of your answers verbally because the court reporter cannot take down head nods or hand motions, and, also, at some point defense counsel may choose to enter an objection. You are still required to answer my question unless your counsel instructs you not to do so. Do you understand those instructions? A. I understand. Thank you. Q. Now, doctor, we were talking about some of the depositions that you have given in the past. Have you given any depositions in a medical negligence case in the last year? A. Yes. Q. Can you tell me what the name of the Plaintiff was in those cases? A. One. Q. What was the allegation of negligence in that one case that you gave deposition last year? A. Anesthetic negligence. Q. Is that case still pending?
 6 1 the case? A. No. Q. Was Cleveland Clinic named as a Defendant in the case? A. No. Q. What was the reason that your 7 deposition was being taken, and by that I mean 8 was it as a medical expert, a fact witness? A. It was a medical expert and others 10 were I was the treating physician. 11 Q. But your care was not called into 12 question in those? 13 A. No. 14 Q. I want to go over some of the ground 15 rules for deposition. I'm sure counsel has had a 16 chance to talk with you. This is a 17 question-and-answer session. It's under oath. 18 It's important that you understand my questions. 19 If you don't understand them, let me know, I'll 20 be happy to repeat the question of rephrase it. 21 Otherwise, I'm going to assume that you 22 understood the question and that you're able to 23 answer it. At any point, if you would like to 24 refer to the medical records that counsel has 25 provided you with, feel free to do so. 	 A. Yes, it is. I would be happy to provide you with the name, but it escapes me at this point. Q. So in any of the other cases where your deposition was taken, was your care ever called into question? A. No. Q. Now, you also informed me that you had acted as a medical/legal expert in some medical negligence proceedings. A. Yes. Q. How many times have you acted as a medical/legal expert? A. My guess would be about ten. Q. How many in the last year have you acted as an expert on? A. I think one. Definitely one and possibly two. Q. in the instances where you have acted as a medical/legal expert, were you providing expert opinions for the Plaintiff or the Defendant in the case? A. It's about 50/50 split.

^{2 (}Pages 5 to 8)

 Q. What was the allegation of negligence in the case that you provided opinion testimony for in the last year? A. It was a case where a child underwent an anesthetic for a fractured arm, awoke from surgery and then had a downhill neurologic course following the surgery. It was alleged that something happened during anesthesia that caused his neurologic decline. The evidence of the case suggests the child had an underlying illness called a mitochondrial cytopathy. Q. In the instances in which you acted as a medical expert, how many times has your deposition been taken? A. My guess is about ten. Q. Have you given trial testimony as an expert? A. In a medical/legal case, once. Q. In that was for Plaintiff. Q. In any of the instances that you have acted as a medical/legal expert, have any of those cases involved issues dealing with papilledema? 	 Ohio. Q. At the time that you rendered care to Kevin Kiss, did you have any additional medical license? A. No. Q. Has your license in Ohio or any other state ever been suspended, revoked or called into question? A. No. Q. Are you board certified in any particular areas of medicine? A. Yes. Q. Which areas? A. Pediatrics, and a board in neurology with special competence in child neurology. Q. And when did you obtain your board certification in pediatrics? A. 1989. Q. And when did you obtain your board certification in neurology? A. 1990, Q. Did you pass both of those on your first attempt? A. Yes. Q. Where do you currently have hospital
 A. Not that I can recall. Q. Any dealing with increased intracranial pressure? A. The trial case dealt with a child with a brain tumor in which there may have been a question of increased intracranial pressure, but I do not recall. That was about ten years ago. Q. Do you recall what the allegation of negligence was in that case that went to trial? A. The Plaintiff alleged that the pediatrician failed to make a timely diagnosis of the brain tumor. Q. Do you recall the name of the Plaintiff in that case? A. I donot. Q. Doctor, you are currently licensed to practice medicine in the State of Ohio; is that correct? A. Yes. Q. And were you also so licensed at the time that you gave care to Kevin Kiss? A. Yes. Q. Are you licensed in any other states besides Ohio? A. I have no active licenses outside 	 privileges? A. The Cleveland Clinic Foundation. Q. Do you have any hospital privileges at any other hospital A. No. Q affiliated with Cleveland Clinic? A. You'd have to ask the general counsel at the Cleveland Clinic. I don't know that answer. This is the only place I practice medicine. Q. Was that also true when you rendered care to Kevin Kiss, that you had privileges at Cleveland Clinic's main campus? A. Yes. Q. Have your hospital privileges ever been questioned, suspended or revoked? A. No. Q. Have you ever been denied hospital privileges A. No. Q anyplace? You have to wait until I finish my question before you answer or she'll have difficulty taking us both down at the same time. A. Thank you.

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4 (Pages 13 to 16)

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 Q. There were both inpatient as well as outpatient records from Cleveland Clinic. Did you review both the inpatient and the outpatient records? A. Yes, I did. Q. Did you review any deposition testimony in preparation for this deposition? A. No, I did not. Q. You haven't seen Dr. Luciano's or Dr. Kosmorsky's deposition? A. I have not reviewed them. Q. Have you reviewed any summary of those depositions? A. No. Q. And I believe there was also some records? M. I don't recall seeing those records. I may have seen them and have certainly forgotten what those records would have shown. Q. Have you done any type of a literature review? A. I have not. Q. Since this case was filed, have you 	 MS. CARULAS: Note my objection. A. In the context of any information in a textbook is accurate, I would consider it to be reliable but do not necessarily prescribe to everything said in any textbook. Q. As you sit here today, is there any particular publication that you believe has particular relevance to the issues in this case? And I'm asking if there's one that you know of, as you sit here today, that you feel has particular relevance. A. I donot. Q. Have you participated in any research dealing with the subject matter of papilledema? A. No. Q. Any with increased intracranial pressure? A. No. Q. Do you subspecialize in any area of pediatric neurology? A. The majority of patients I see have either brain tumors or mitochondrial cytopathies. Q. And what is a mitochondrial
 discussed this case with any physicians? A. No, I have not. Q. And other than with counsel, have you discussed it with anyone else? A. No, I have not. Q. And aside from the clinical notes that appear in the Cleveland Clinic records, inpatient and outpatient records, do you have any other notes or file referencing Kevin Kiss? A. No. Q. Doctor, is there a textbook in your field of practice that you consider to be the best, most reliable? MS. CARULAS: Note my objection. Go ahead. A. There's not a single textbook that I consider to be the best or most reliable. Q. Is there any that you refer to more often than not in your practice? A. I tell the residents to get a book written by Fenischerl which I think does a very nice job overviewing the many topics of child neurology. Doy you consider the material in that book to be reliable? 	 20 1 cytopathy? A. It is a biochemical disorder of energy metabolism. Q. How often in your practice do you see patients with arachnoid cysts? A. Maybe one patient a year with a symptomatic arachnoid cyst. Q. Have you referred children to surgery for fenestration of arachnoid cysts? A. Yes. Q. Are there any complications that are associated with arachnoid cysts? A. There can be. Q. And what are those complications? A. Pressure on the brain that can cause deformation of the brain structure itself and problems resultant to that. Q. Would you agree that headache is one of the early signs of increased intracranial pressure? A. Headache can be one of the signs of increased intracranial pressure. Q. Is it one of the early signs of increased intracranial pressure.

5 (Pages 17 to 20)

21 1 Q. After fenestration of an arachnoid 2 cyst, can increased intracranial pressure recur? 3 A. After fenestration of an arachnoid 4 cyst, the intracranial pressure can remain as it	 23 1 papilledema? 2 A. Papilledema is a sign. 3 Q. When you observe the inner portion of 4 the eye, is there anything that indicates to you
 5 was before the fenestration. It can decrease and 6 come back to normal, and if scarring takes place, 7 or the fenestration closes up again, conceivably 8 pressure can recur. 9 Q. Do patients who have undergone 10 fenestration require long-term followup with 11 neuro-imaging evaluation because increased 12 intracranial pressure can recur? 13 A. Usually not. 14 Q. What is papilledema? 	 5 that the papilledema is early rather than a 6 chronic or later form? 7 A. That's a difficult question to 8 answer. Papilledema can be graded as mild, 9 moderate or severe. But the rapidity at which 10 papilledema forms is based on the process at 11 hand. For example, if someone gets shot in the 12 head with a bullet, you can get papilledema 13 developing within minutes of that injury. In a 14 situation of chronic increased intracranial
 A. Papilledema is a physical finding that one sees in the back of the eye that can be a result of a number of processes, the most common of which would be increased pressure inside the brain. Q. Isn't it true that one of the earlier signs of increased intracranial pressure is swelling of the optic disc? A. Could you rephrase the question? You used a negative when you asked the question. You said "isn't." Could you just rephrase it without 	 pressure, papilledema can take months to develop. Q. Doctor, if you see blurring of the optic disc margins, is that any indication as to whether this is something that is early or in a chronic stage? A. No. Q. When you observe the inner eye, what are you looking for, what signs or symptoms, characteristics? In evaluating for papilledema, what are the observations that you look for? A. Central disc elevation, blurring of
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 the "isn't" because that confuses me. Q. k one of the earlier signs of increased intracranial pressure swelling of the optic disc? A. Optic disc swelling can be a sign of increased intracranial pressure. It can occur early in the course; it can occur very late in the course. Q. How is papilledema diagnosed? A. By using an ophthalmoscope and looking in the back of the eye with that ophthalmoscope. Q. Now, in the course of your practice, do you ever perform ophthalmologic examinations to look for papilledema? A. Looking for papilledema using an ophthalmoscope is part of the neurological examination. Q. So the answer to my question is, yes, that you do that? A. It's a ·· I want to stay away from the term whether or not I do an ophthalmologic examination. Q. What are the early signs of 	 disc margins, hyperemia and bleeding. We also look for pulsations of the veins or lack thereof. Q. And the signs that you just mentioned, can those be present whether it is an acute problem or a chronic problem? A. Yes, it can. Q. Would you agree that, when papilledema is found to be present, that the patient should be monitored closely to determine if the condition is progressing or if it's stable or resolving? MS. CARULAS: Note my objection. Go ahead. A. When one diagnoses papilledema, the first thing the physician must do is investigate as to why the papilledema is occumng, and then if it is a treatable disease, to treat it. Once the disease is treated, papilledema can take months to disappear, even with adequate treatment of the underlying process. Q. Would you agree, though, that the papilledema should be monitored to determine whether it is resolving? A. The papilledema should be monitored. Q. Have you ever referred a patient to

6 (Pages 21 to 24)

1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25	 Manage. Q. If there was a question as to whether or not a patient should undergo an optic nerve fenestration, is that something that you as a neurologist would determine, or would that be something that an ophthalmologist would recommend? A. I would not determine that. Q. From your perspective, if that was a consideration, whose decision would it be to 	 MS. CARULAS: Note my objection. Go ahead. A. It can be. Q. Are there any complications associated with papilledema? A. Papilledema is a clinical finding. The complications are generally related to the underlying cause of the papilledema. So if the cause of the papilledema is a large brain tumor, the complication of the underlying disease can be death of the brain tumor. If the cause of papilledema is a disorder called pseudotumor cerebri, the complications of that may be quite a bit different. So, again, papilledema is a clinical sign. very much like wheezing is a clinical sign. Wheezing can be due to asthma, which is generally a harmless condition, or lung cancer, which is a fatal condition. It's up to the doctor to determine what the underlying cause is and treat that appropriately. Q. Doctor, isn't it true that blindness may result from persistent papilledema? A. Again, I'm just going to ask you to rephrase that question without the negative.
16 17 18 19 20	Q. If there was a question as to whether or not a patient should undergo an optic nerve fenestration, is that something that you as a neurologist would determine, or would that be something that an ophthalmologist would	 16 sign, very much like wheezing is a clinical 17 sign. Wheezing can be due to asthma, which is 18 generally a harmless condition, or lung cancer, 19 which is a fatal condition. It's up to the 20 doctor to determine what the underlying cause is
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1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25	 particular procedure for papilledema? MS. CARULAS: Please note my objection. Go ahead. A. I don't have an answer to that question, because I don't know the context in which it's being asked. Q. Have you ever referred a patient for an optic nerve fenestration? A. I think I have asked an ophthalmologist on a couple of occasions to consider whether it would be helpful, and, to my knowledge, none of my patients have had optic nerve fenestrations, so the answer was no. Q. They would give you their recommendation then after evaluating the patient? A. The answer is yes, but when I send a patient to a surgeon, it's generally with the question, do they have a surgical procedure that could be helpful in the context of the disease, and then they offer an opinion, and then we decide generally together whether or not it would be reasonable to proceed with this. Q. Is a finding of papilledema cause for 	 Q. Can persistent papilledema result in blindness or vision loss? A. Again, papilledema is the sign of what the underlying process is. Q. What is optic atrophy? A. Optic atrophy is the result of some process which destroys the optic nerve. That process can be due to a brain tumor; that process can be due to increased intracranial pressure; that process can be due to a stroke of the optic nerve itself, and there are probably a dozen other causes of optic atrophy. Q. Can papilledema that is persistent cause optic atrophy? A. Patients with underlying neurologic conditions that result in papilledema can result with optic atrophy later on in the course of that underlying condition. Q. Iwant to make sure I'm understanding what you're saying. The papilledema does not result in optic atrophy, it doesn't cause optic atrophy? A. That's correct. That's correct. Q. Increased intracranial pressure can cause optic atrophy, correct?

7 (Pages 25 to 28)

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 A. Optic atrophy can be seen as a result of a number of neurologic factors, including increased intracranial pressure. Q. Doctor, if a patient has papilledema and in some instances an optic nerve fenestration is done, isn't that done in order to relieve the papilledema to prevent optic atrophy? A. One could do an optic nerve an optic sheath fenestration to relieve intracranial pressure in the context of pseudotumor cerebri. Outside of that disease model, I don't have any Tve never referred a patient for an optic sheath decompression, nor do I really know anything else about it. Q. Does the risk of optic nerve damage increase with the duration of papilledema? A. Not necessarily. I say that based on the fact that I have a number of patients who have had papilledema for many, many years and never develop optic atrophy or visual loss. Q. When a patient does develop optic nerve atrophy, are there any visible changes that can be seen on funduscopic exam in the structures of the eye? A. Optic nerve atrophy implies that you 	 ability to perceive objects throughout what we would throughout space. So if one closes one eye and imagines the visual field of one eye, one can picture a clock with the central portion of the clock representing the center of the visual field and the numbers of the clock representing the periphery of the visual field. I don't know if I've answered your question, because I forgot your question, so could you restate it. Q. I just asked you what visual field testing was. A. So the testing would attempt to determine whether or not there were segments missing from that field. Q. Do you do visual field testing in your practice? A. We can do visual field testing in the office, and it's called visual field testing by confrontation. Q. What does that mean? A. We generally ask the patient to cover one of their eyes with their hand, ask the patient to stare at our nose, and then with our outstretched hands placed halfway between
 30 see those visual changes. Q. What would you see when you look into the eye if there is optic nerve atrophy? A. The optic nerve would appear smaller than normal and generally paler than normal. Q. When a young child develops a visual field defect gradually, is a child always aware that the vision is being lost? A. The child may not be aware the vision is lost. Q. Now, in regard to persistent papilledema, would the treatment be first to treat the underlying cause of the papilledema? A. That is correct, because, again, papilledema is a sign. 	 32 ourselves and the patient, we also close the appropriate eye, we wiggle fingers at different points in space and the patient tells us whether or not they can perceive the wiggling fingers. We assume that our visual field is intact when we test a patient's visual field. So we'll wiggle fingers at 6:00 o'clock, 3:00 o'clock, at noon, at 9:00 o'clock, to see if the patient can perceive the periphery as well as we can. Q. Is visual field testing helpful in monitoring a patient with persistent papilledema? A. It's one of the one of the techniques one can use to help monitor patients with increased intracranial pressure or
 papilledema is a sign. Q. Are there any treatments specifically for papilledema, irrespective of the cause, anything that can be done just to relieve papilledema? A. If I understand your question correctly, there is no treatment for papilledema. papilledema. Q. What is visual field testing? A. The visual field is a concept of the 	 15 with increased intracranial pressure or 16 papilledema due to increased intracranial 17 pressure. 18 Q. And what are you looking for in a 19 patient that has increased intracranial pressure 20 and papilledema? 21 A. Well, what we look for is preserved 22 visual field, that the patient's peripheral 23 vision is as good as ours. 24 Q. And in some instances, can increased 25 intracranial pressure result in a patient

8 (Pages 29 to 32)

 developing a visual field defect? A. In some instances it can. Q. Now, if a patient is found to have persistent papilledema, would you agree that the patient should be followed closely for signs of optic atrophy? MS. ATWELL: Objection. MS. LOESEL: Objection. MS. CARULAS: Note my objection. A. Could I ask you to repeat the question? (Record read.) Q. By that, optic nerve atrophy. A. If one has optic nerve atrophy. A. If one has optic nerve atrophy. Mit neally due respect, doesn't seem to be relevant. Maybe I just don't understand your question. Q. If a patient has persistent papilledema, you would agree that the patient should be followed to determine whether it is remaining stable, becoming chronic or resolving; correct? I think you told me yes to that 	 see using an ophthalmoscope. A visual field deficit is a finding one would see using one of a number of visual field tests. Q. But does optic nerve atrophy result in visual field defects? A. Many patients with optic nerve atrophy do have visual field defects. Q. So if you're doing visual field testing and you find that there is a defect in the visual fields, would that give an indication of optic atrophy? A. No. Optic atrophy is something one sees with the ophthalmoscope. A visual field loss is a finding someone would see by visual field testing, and they're two very different parts of the examination. Q. But if you're doing visual field testing, wouldn't it raise a concern for optic atrophy? MS. ATWELL: Objection. MS. ATWELL: Objection. A. If one finds a visual field deficit, would one would want to do an opth if one finds a visual field deficit, one would want to
 A. Patients with underlying processes that result in papilledema need to be followed by a physician for all aspects of their underlying condition. Q. If the underlying condition was increased intracranial pressure resulting in papilledema, in that type of a patient, would you agree that the patient should be Followed closely for signs of optic nerve atrophy? MS. CARULAS: Note my objection. MS. ATWELL: Objection. A. I apologize. Could I have the question again? (Record read.) A. The patient needs to be clinically followed and, again, the optic nerve atrophy would be an end result of a worst-case scenario of chronic increased intracranial pressure and resultant papilledema. Q. But, doctor, if you're doing visual field testing on a patient, would that give you an indication that the patient may be developing optic atrophy, if there's a visual field defect? A. Optic atrophy is a finding one would 	 Jook in the eyes to see if there was some underlying finding, which could be optic atrophy or could be a number of other problems. Q. Do you have an independent recollection of Kevin Kiss as you sit here today? Do you remember him? A. I remember the case. I don't remember what the child looks like. Q. Do you remember some of your involvement with his treatment? A. Yes, I do. Q. Doctor, please feel free to look at the records if that will help you. When is the first time that Kevin Kiss came under your care? A. It would be April 14th, 1998. Q. And how is it that he came to see you on April 14th of 98? A. Dr. Luciano asked for my opinion regarding his medical situation. Q. Did he speak to you directly about Kevin? A. To my knowledge, he did not. Q. Do you know how you were contacted for that consultation! A. My recollection is that his nurse

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 37 1 contacted me and asked if I would see him. Q. Were you given any information about Kevin at the time that you were asked to see 4 him? A. I probably had access to his medical 6 file. 7 Q. And what is your understanding as to 8 why Kevin was referred to you by Dr. Luciano? 9 A. My understanding is that he had 10 problems following his initial surgery. Those 11 problems included irritability, mood disturbance 12 and intermittent headaches as well as hearing 13 noises in his right ear. And I can deduce that 14 Dr. Luciano wanted my opinion regarding what was 15 going on. 16 Q. Did Kevin aiso have vision problems? 17 A. Based on my examination, he had 18 papilledema at the time of that visit. 19 Q. In the history that was given to you, 10 were you told that he had disturbed vision? 11 A. I don't see it in the notes. 12 Q. Doctor, I see a note dated April 14th 13 of 98. I think it's timed at 10:30 in the 14 morning. 25 A. Yes. 	 A. Yes, I do see that. Q. Is it likely that that's information you also were aware of at the time that you saw Kevin? A. That would be correct. Q. Tell me what other history you were aware of in regard to Kevin when you saw him on April 14th of 98. You mentioned he had ear noises. We've just discussed the vision problems. What else? A. Problems with behavior and emotion. He had been in psychological counseling for that problem. He had been on Diamox to help relieve the intracranial pressure. He had headaches, complaints of blurry vision and double vision. Q. How were the headaches described? A. The headaches were described as . right-sided, occurring every day, worse in the morning and evening. They were sharp, triggered by activity. Q. And were they almost continuous also? A. Yes. I'm sorry, almost continuous. Q. How long had Kevin been having headaches when you saw him?
 38 1 Q. It says outpatient visit, Dr. Cohen. 2 Is that a note from you? 3 A. Yes. The majority of handwriting in 4 that note is from one of my residents, but that 5 note does reflect his visit with me. 6 Q. And did the resident see the patient 5 before you or with you? 8 A. The resident I don't recall in 9 this situation. As a general rule, the resident 10 sees the patient before me. 11 Q. Does the resident then provide you 12 with the information that is obtained on his 13 visit? 14 A. That is true, at which time I go back 15 in, review the relevant history, I do my own 16 examination, report any differences I may find 17 from the resident's examination. 18 Q. Do you add or make notations in the 19 chart if you disagree with something that the 10 resident has done? 11 A. That would be correct. 12 Q. Now, at the beginning of this note, 13 doesn't it state in the first line, referred for 14 headache, disturbed vision, having noises right 15 ear? 	 40 A. My chart note indicates two months. Q. And did he indicate anything made them worse? A. I thought I mentioned activity. Activity made them worse. Q. And you may have, doctor. A. I'm sorry. Q. Doctor, did the behavior changes that were described coincide with the start of the headaches and the vision problems that he had? A. I don't know that for a fact. The chart note indicates that both problems started about two months previously. Q. Now, you indicated that he was on Diamox. It was your understanding that the Diamox was to decrease intracranial pressure; is that correct? A. That's my assumption. Q. Now, doctor, I believe in the organization of the resident's note about halfway down the page, it says I believe tried Diamox for a month, didn't make a big change? What type of change is the resident referring to there? A. My assumption is it didn't make a

^{10 (}Pages **37** to 40)

41	43
 change in his clinical symptoms. Q. The headaches, the vision, the ear noises, those types of things? A. That would be correct. Q. Now, I believe there's also a notation in this note that he was sleeping very little at night. Did that coincide with the start of the headaches and the ear noises and the vision problems? A. I don't know. I did not record that information. Q. Now, did you do a physical examination when you saw him on the 14th? A. Yes, I did. Q. Did you find any deviations from normal that you felt were significant in your physical exam? A. I thought he had papilledema. I noted three plus papilledema. I also noted, as did the resident, that he had normal visual fields. The resident noted that, that his eye movements were normal and that there was no 	 Q. I'm still not seeing exactly. I see CNS, I see normal, nerves 2 and 12 intact. A. That means cranial nerves 2 and 12 intact. Q. Below that is what you're referring to? A. It says NL visual field. Q. Ail right. What type of visual field g testing was done on April 14th of 98? A. Confrontation. Q. Were you present when the resident did it? A. I was not present when the resident it did it. Q. Now, you did your own funduscopic exam of Kevin's eyes; is that correct? A. I did my own funduscopic exam, and-I did do other parts of his exam. I documented some of those that weren't document dabove, and I specifically and didn't document others that were documented above. Q. When you did the funduscopic exam, you found that Kevin had grade three papilledema or three plus papilledema? A. That's correct.
 42 1 because that would indicate that, despite the 2 fact that I thought he had increased intracranial 3 pressure, that I didn't think the increased 4 intracranial pressure was so high that he was in 5 physical danger. 6 Q. What physical danger are you 7 referring to? 8 A. If intracranial pressure is too high, 9 children can permanently injure their brain 10 through a process called herniation. And the 11 lack of those abnormal physical findings told me 12 that I didn't have to be concerned of that 13 problem occurring at that time. 14 Q. Now, you referred to visual field 15 testing. Can you tell me where that is 16 referenced? 17 A. In the resident's note, halfway down 18 that last page, that date, it says normal, 19 abbreviated NL, visual field. 20 Q. Show me which page. I'm not on the 21 right page. 22 A. (Indicating.) 23 Q. I just want to see the top of the 24 page. 25 MS. ATWELL: Under CNS. 	 Q. What does grade three or three plus papilledema indicate? A. That's a notation for me. Since I don't know, or am not familiar with, any of the grading systems an ophthalmologist may use, I tend to think of papilledema as a range from mild to severe. And I use the designation one plus, two plus, three plus or four plus to indicate to me the severity of papilledema. So three plus would indicate a swollen disc. He had lack of venous pulsations, blurred disc margins without hemorrhages. Q. And is that what you saw when you did the funduscopic exam on Kevin? A. That's what I saw. Q. Do you know, when you saw him on April 14th of 98, how long his papilledema had been present? A. I do not know the answer to that question. Q. Were there any signs that would indicate to you that the papilledema was acute or chronic? A. I don't have the ability to tell that based on a physical finding.

11 (Pages 41 to 44)

 45 Q. Now, following your evaluation of Kevin on the 14th, what were your impressions? A. My impression was that the history and constellation of clinical signs were consistent with the increased intracranial pressure. Q. And what data did you have to support your impression of increased intracranial pressure? What were you relying on to come to that impression? A. The papilledema and the history, the context of which I was seeing the child for. Q. And what, in particular, in his history? A. The fact that he had an arachnoid cyst, the fact that he had an operative procedure for treatment of the arachnoid cyst, that the symptoms had persisted, that he had failed a trial of Diamox, and that he had papilledema. Q. When you say the symptoms persisted, are you speaking of the headache, the blurred vision, the ear noises, the emotional disturbances? A. Certainly the headache. Q. And, in actuality, didn't he have 	 47 1 well for awhile after his surgery? You mentioned 2 that some children will have symptoms after 3 surgery, but he had his surgery in December. The 4 history that you have here is about a two-month 5 history of these other symptoms, isn't it, which 6 would be February to April? 7 A. Dr. Luciano's chart note dated 8 January 22nd, 1998 indicated that his headaches 9 started two weeks prior to that. So that would 10 be about January 8th. 11 Q. So the chart note on January 22 12 indicates that January 8th would have been about 13 the time his headaches started and about the time 14 his double vision started? 15 A. The history I obtained on April 14th, 16 say in two months, was again the history obtained 17 from the parent or parents at the time of the 18 visit. It's a history that we write down. 19 Q. When you saw him on the 14th, what 10 was your plan of care for him? I think you 21 mentioned to refer him to Dr. Luciano for 22 surgery. 23 A. From what I remember, I either called 24 Dr. Luciano or Dr. Luciano's nurse and suggested 25 that surgery would be my choice as to what to do.
 46 some relief from his symptoms after the surgery? A. I think he had relief of some of the symptoms for a period of time and then the symptoms came back. Q. And then they came back? A. Yes. Q. The fact that he was relieved of symptoms and then they came back, would that add any weight to your concern that this was increased intracranial pressure? A. Following a surgical procedure such as fenestration of arachnoid cysts, children can have irritability, mood disturbances; you can have headaches after a surgical procedure. Speaking against the diagnosis of increased intracranial pressure is the fact that the CAT scan really hadn't changed up until that point. It was my clinical impression that he had increased intracranial pressure, and my opinion was that I put down in the chart to transfer him to Dr. Luciano's care for surgery, that I thought he had been given an adequate trial of Diamox and all attempts to avoid a shunting procedure had been done. Q. In actuality, didn't he do pretty 	 48 1 Q. Now, doctor, under your note, you have is that treat what does the TX stand for? 4 A. Transfer. 5 Q. Transfer to Dr. Luciano for surgery. 6 A. Yes. 7 Q. Then underneath that, you have eye 8 consult. Is that for visual fields? 9 A. It says eye consult visual field. 10 Q. Now, the type of surgery that you 11 were referring to in your plan, was that for the 12 shunting procedure? 13 A. From the best I can remember, I 14 wasn't convinced in my own mind whether the 15 subdural hygromas were what needed to be shunted 16 or the arachnoid cyst. And so I had to rely on 17 Dr. Luciano to make the correct decision. 18 Q. But it was to relieve the increased 19 intracranial pressure? 20 A. It was to relieve the increased 21 intracranial pressure, correct. 22 Q. Now, doctor, you indicated also eye 23 consult for visual field testing; is that 24 correct? 25 A. That's what I put in my chart note.

12 (Pages 45 to 48)

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 49 1 Q. Why did you advise an eye consult for 2 visual field testing? 3 A. I think as part of the longitudinal 4 care of the chronic, long-term needs of a child 5 with papilledema and intracranial pressure, it 6 would be good to know what his visual field was, 7 is and will be in the future for educational 8 purposes, for example. 9 Q. At the time that you saw him on the 10 14th, were you of the opinion that he had chronic 11 long-term papilledema? 12 A. No, I didn't have an opinion 13 regarding that. In fact, from the notes, I 14 really wasn't worried about his vision at that 15 time. We had noted normal visual fields, and it 16 was •• I would have •• again, I would have put 17 that in there as part of the longterm 18 management, not because I saw an acute problem 19 that needed to be dealt with at that time. 20 Q. So you were looking to have an 21 ophthalmologist manage the papilledema over the 22 long term? 23 A. No. I was looking for an 24 ophthalmologist to help us out with a detailed 25 visual acuity examination, for example, to look 	 51 Q. That was based on the test that was done by the resident at the bedside? A. By the resident and by me at the bedside because that would have been something I would have routinely done as part of assessing a child. Q. Well A. In other words, did I indicate in the chart? No. There's certain things I would repeat, certain things I wouldn't repeat. Q. When we talked about it before, you indicated this was done by the resident and that you weren't in the room when the resident did it. A. That's correct. Q. Are you telling me you repeated it? A. Yes. Q. There's two visual field tests done at the bedside on April 14th of 98, one by you and one by the resident; correct? A. That would have been correct. Q. Now, when did you anticipate that this eye consult would actually be done? A. I have no expectation that it would be done at any point in the immediate future. I
 at the papilledema and tell me whether it was there or not, to get visual fields. Again, this was not meant to be an emergent consult. It's just something that we ought to get along the way. Q. Did you expect that the visual field testing that an ophthalmologist would do would be different than the one that was done by your resident physician at the bedside? A. Ophthalmologists do visual fields by both confrontation and by a much more formal method which, frankly, is sometimes difficult or impossible to do in a seven-year-old, so a lot of the ophthalmologists will do confrontational visual fields as well. Q. What were you expecting the ophthalmologist in this case to do different than what had already been done already for Kevin? A. Frankly, nothing, with the addition of possibly getting a detailed visual acuity examination. Q. Do you know whether Kevin had any visual field defects at the time that you saw him? A. He did not. 	 just wanted to get it get something documented at some point. Q. Now, you were referring him to surgery, so did you anticipate it would be done before surgery or after surgery? A. I didn't anticipate it would be done before surgery because it's hard to get things arranged that quickly. Q. Did you make any arrangements for an eye consult for Kevin for visual field testing? A. I do not remember. Q. How would you normally go about doing that, to make arrangements for visual field testing for a child that you had seen? A. The key point in this child's care was to treat the underlying cause of the increased intracranial pressure. The visual field testing that was to be done could have been done either as an inpatient or as an outpatient. So when a child is not feeling well after surgery, trying to get them to cooperate with an eye exam sometimes can be difficult, so we would probably arrange for it to be done at some point as an outpatient when he was at home and feeling a little better.

13 (Pages **49** to 52)

53	55
1 Q . Did you make any such arrangements	1 atrophy had already occurred.
2 for Kevin to have it done as an outpatient after 3 he was home?	2 Q. Had already occurred when you saw him 3 on June 9th of 98?
4 A. I do not remember.	4 A. Essentially had already occurred by
5 Q. Did you come across anything in the	5 June 9th, 98. Probably it occurred long before l
6 chart that told you you did that?	6 ever met the child.
7 A. I do not remember. I don't know if I 8 did it or if someone else did it.	7 Q. Do you know when it occurred? 8 A. No. I do not.
8 did it or if someone else did it. 9 Q. Did you inform Dr. Luciano that Kevin	8 A. No, I do not. 9 Q. Who accompanied Kevin when you saw
10 should receive an eye consultation for visual	10 him on April 14th of 98 to the visit?
1.1 field testing?	11 A. Could I have the last couple
12 A. I do not remember.	I2 questions and answers read?
13 Q. Do you know if Kevin ever received an 14 eve consult as a result of your recommendation?	13 Q. Yes. 14 (Record read.)
14 eye consult as a result of your recommendation? 15 A. I know he was seen by an	15 A. My answer to your question was not
16 ophthalmologist. I don't know if it was a result	16 entirely complete and doesn't reflect accurately
17 of my recommendation and Dr. Luciano's	17 my feelings about this. Do you mind if we just
18 recommendation or some other person's	18 take a break so I can get some water?
19 recommendation. 20 O. When was he seen by the	19Q.Sure. Would you like to correct your20answer? I mean, if you would like to go and
21 ophthalmologist, that you're aware of?	21 correct your answer, you can go ahead and correct
A. I had seen Kevin on June 9th, 1998,	22 your answer if you're uncomfortable as to what it
23 and my history obtained at that time was that	23 is you said.
24 after his shunt was placed, meaning the surgical	24 MS. CARULAS: Or do it when you come 25 back if You want to take a break.
25 procedure done on April 15th, 1998, he was 90	25 back if 100 want to take a break.
54	56
1 percent back to normal in four days, and in two	1 A. Yes. It's difficult for me to answer
2 weeks was a hundred percent. He was back at full 3 activities, and he was making straight As in	2 your question in the context of everything
	A summounding that
	3 surrounding that. 4 O Which question are we referring to?
4 school. 5 On my examination, I noted mild optic	4 Q . Which question are we referring to?
 4 school. 5 On my examination, I noted mild optic 6 atrophy and no papilledema. I sent him to see 	4 Q. Which question are we referring to? 5 A. The question as to when the 6 question that implies that the visual loss may
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14 (Pages 53 to 56)

 previously. A. When we reread what I had said, I realized I had said something that didn't reflect my feelings about the situation. Q. And if you would like to correct it, please feel free to do so. A. The question as to when the visual loss took place is, I think, the crux of this entire matter. The child had normal vision prior to the surgical procedure done on April 15th, 11 1998. That had been documented in an eye exam done by Dr. Marcotty back in February. The visual fields done in my office were normal. He had papilledema. Children with papilledema generally do not have visual loss with the papilledema. This child was destined to lose his vision at some point in time. That point in time may have been back in November 1997. The process of papilledema leading to optic atrophy with visual loss is one that is well documented in textbooks, but in fact rarely occurs in clinical practice. We all read in textbooks that we need to monitor patients with increased intracranial pressure for visual loss. In fact, we rarely see visual loss as part of our 	 17th on the 16th, it was noted that his pupils were equal and reactive to light indicating that the cranial nerves were functioning. Q. Well A. It doesn't Q. It doesn't indicate visual field defects, though; correct? A. That's correct. Q. Now, doctor, you indicated that Kevin, and correct me if I'm misstating what you said, but that Kevin was destined to lose his vision at some point in time. Am I stating what you said correctly? A. I'm basing that statement on the fact that he in fact did lose his vision, that the vast majority of patients in this exact situation would not lose their vision. So that comment that he was destined to lose his vision was based on the a priori knowledge that he in fact did lose his vision. Q. You said that the rapid shift of cranial structures can result in vision loss; correct? A. One of the mechanisms of visual loss that may have been relevant in this case may have
 practice. I have patients with chronic increased intracranial pressure due to pseudotumor cerebri, and they don't lose their vision. The cause of the visual loss could be due to chronic increased intracranial pressure, it could be due to the relief of that intracranial pressure, the shift of brain contents from the displaced position they were in caused from the arachnoid cyst to a more natural position. The arachnoid cyst creates pressure that exerts itself on the brain over the course of many months to many years, which slowly displaces brain contents, including the optic nerves, and the relief of that pressure can also lead to a rapid shift which can then result in visual loss. In fact, eye exams documented in the chart following the surgical procedure dldn't indicate any problem. Q. What eye exams are you referring to? A. I had made a note on April 18th, awake, alert, feels better, no headache, examination plus papilledema, otherwise okay neurologic exam, the day we discharged him from the hospital. One of the doctors indicates normal cranial nerves two through 12. On April 	 been the relief of the cyst pressure. Q. And if that was indeed the cause, how soon after the relief of the pressure would you expect to see the vision loss occurring? A. I don't know the answer to that question. Q. Would you expect it to occur in a relatively short period of time, within days of the time of the surgery and the shunting procedure? A. It could have occurred within days to weeks following the shunting procedure. Q. Now, it's your opinion that his vision was normal when you saw him on April, I believe it was, 16th of 98? A. That's correct. Q. He did have impairment when you saw him then in June, on June 9th; correct? A. He had optic atrophy on June 9th. I did not do a visual acuity. Q. Did you do visual field testing on June 9th? A. Let me find my note and I'll answer

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 61 1 your question. I did not document a visual field 2 check at that time. 3 Q. Now, when Kevin was admitted to the 4 hospital, you saw him during that admission; 5 correct? 6 A. Yes, I did. 7 Q. We just looked at some of those 8 notes. And one of the notes that you wrote was 9 on April 17th of 1998; correct? 10 A. That's correct. 11 Q. Now, I believe Kevin was actually 12 admitted on the 14th. Did you see him any time 13 between the 14th and the 17th? Your note is 14 written on the 17th, and that's the first one 15 that I see. 16 A. Yes, I saw him on the 16th. 17 Q. Okay. 18 A. There's a three-line note about 19 two-thirds of the way down the page. 20 Q. Okay. Yes, I see that. So you saw 21 him on the 16th? 22 A. Correct. 23 Q. And this was after his surgery; 24 correct? 25 A. It was after his surgery. 	 63 Q. Did you feel at that point in time, because he still had papilledema after the shunting procedure, that he should be referred to an ophthalmologist and followed to determine if the papilledema was indeed resolving? A. On that day, I did not refer him to an ophthalmologist. I knew he was going to be seen in followup by either the surgeon, myself or both. Q. Well, my question was in regard to an ophthalmologist. Did you feel that when Kevin was discharged from the hospital he should have been followed by an ophthalmologist for his papilledema? A. No. Q. Why not? A. Because he would be seen by a neurosurgeon and/or myself or both of us. Q. And you felt that the evaluation done by the neurosurgeon or yourself would be adequate to follow the papilledema? A. That is correct, because an ophthalmologist can't treat papilledema or visual loss. Only the surgeon can treat the underlying disease process.
 62 1 Q. Did you check him for papilledema at 2 that point in time when you saw him on the 16th? 3 A. I did not. 4 Q. When you saw him on the 17th, did you 5 check him for papilledema? 6 A. I did, 7 Q. Now, would you have expected that his 8 papilledema would have been relieved by the 9 shunting procedure? 10 A. No. It can take many months for 11 papilledema to disappear. 12 Q. Did you have any concerns that the 13 papilledema would cause some type of visual 14 problems because it was persistent? 15 A. The answer to that question is no. 16 The papilledema was going to resolve because his 17 pressure had been adequately relieved. 18 Q. Did he still have increased 19 intracranial pressure when you saw him on the 20 17th? 21 A. My answer to your question would be 22 no, he did not have increased intracranial 23 pressure. His intracranial pressure was gone. 24 Papilledema can take many, many months to form. 25 And it can take many, many months to resolve. 	 Q. Then why, when you saw him before his surgery, did you want him to be referred to an ophthalmologist for visual field testing? MS. CARULAS: Note my objection. I think we've been through that at length as to why he felt long-term, but go ahead. MS. TOSTI: I'm seeing a disparity in his answer. At least it's sounding like it, and Iwould just like him to clarify that. MS. CARULAS: Objection. MS. CARULAS: Objection is noted, but go ahead. A. A child with papilledema from increased intracranial pressure is at risk to develop some visual loss. When the child goes back to school, we would like to document that the vision is normal so that he can properly read, for example. The purpose of documenting normal vision or abnormal vision has to do with the completeness of the medical care he receives, not to circumvent or change the practice of what had gone on or what goes on in the future. Q. So, doctor, and I just want to understand what you're saying, once he had this

16 (Pages 61 to 64)

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 available for Kevin's papilledema; is that correct? A. Papilledema is the sign. The disease was an arachnoid cyst and increased intracranial pressure. We may be dancing around a term here, but it makes it difficult to answer your question because at no point was any doctor treating the papilledema. We were treating the increased intracranial pressure, we were treating the arachnoid cyst. I use the term "we" as all of his treating physicians. Q. Once the shunting procedure was done and the papilledema persisted, was there any other treatment open to Kevin to relieve his papilledema? MS. ATWELL: Objection. MS. LOESEL: Objection. A. Papilledema will persist for many weeks to months following adequate relief of intracranial pressure. So the fact that it existed one day, two days, three days, five days, two weeks or two months following the shunt placement on April 15th is not relevant. What is relevant is that his pressure was relieved. 	 A. That's correct. Q. You also saw him on the 18th; is that correct? A. I saw him on the 18th. Q. Did you do a funduscopic exam on him on the 18th when you saw him? A. I did. It showed papilledema. Q. Was the papilledema at the same gegree that it was when you had seen him prior to his shunting procedure? A. My assumption is that it probably would be. Q. So it looked similar to you then? A. That would be correct. Q. Now, at the time that you saw him on the 18th, did you feei that he was at risk for optic nerve atrophy? A. I don't know if I asked myself the question on April 18th whether or not he would be at risk for optic nerve atrophy. If you ask me the question today, the answer would be that a child who had undergone this series of events would be at small risk of optic nerve atrophy. Q. Now, doctor, you spoke earlier about relieving increased intracranial pressure causing
 66 1 the process of the arachnoid cyst and the 2 increased intracranial pressure, and we have the 3 separate process, albeit related, to the fact 4 that this optic nerve underwent slow compression 5 over the course of many, many months, followed by 6 a procedure that relieved that pressure. 7 It will never be clear as to what 8 point this child's vision was destined to be 9 lost. It could have been due to the chronic 10 effect of papilledema that began many months 11 before the discovery of his arachnoid cyst, and 12 the pressure had the pressure been adequately 13 relieved by the fenestration back in 1997, he 14 could have lost vision subsequent to that. In 15 fact, the pressure was relieved in April of 98, 16 and he lost vision subsequent to that point. 17 Q. Do you have an opinion as to whether 18 his shunting procedure was done in a timely 19 manner to relieve his increased intracranial 20 pressure? 21 A. Yes, I do. 22 Q. What is your opinion? 23 A. It was done in a timely manner. 24 Q. When you saw him on the 17th, he 25 still had the papilledema; correct? 	 a shifting of structures that can ultimately result in vision loss. How many times have you seen that occur? A. Once. Q. One time besides Kevin? A. No. Q. This is the only time? A. Kevin. I've taken care of many patients before Kevin and many patients after Kevin with increased intracranial pressure, and this is the only case that I can recall where the result was optic atrophy and visual loss. Q. In the patients that you have cared for, how many patients have you seen that have developed optic atrophy as a result of increased intracranial pressure? A. This would be the case that comes to mind. We have patients with brain tumors who have pressure of their optic nerve with the tumor who develop optic atrophy. But in terms of patients with visual loss secondary to an arachnoid cyst or increased pressure, this case, in my mind, is unique.

17 (Pages 65 to 68)

 69 1 Q. Now, Kevin was discharged, I believe, 2 on the 18th with papilledema. 3 A. That's correct. 4 Q. Should he have received evaluation 5 and followup by an ophthalmologist after 6 discharge? 7 MS. ATWELL: Objection. 8 MS. LOESEL: Objection. 9 MS. CARULAS: I'm going to object 10 because we have been over it and over it. But go 11 ahead. 12 A. Well, he did. Do you mean 13 immediately after discharge? 14 Q. Yes. 15 A. No. 16 Q. At what point should he have received 17 ophthalmologic evaluation after discharge? How 18 long a time period? 19 A. At some point in the future, when it 10 was when he had recovered from the steroid 11 taper and after he was followed up by Dr. Luciano 12 and myself. 13 Q. Can you give me some type of a time 14 range for that, when you think that ophthalmology 15 followup should have been done after discharge? 	 Q. You didn't provide them with any type of written instructions to obtain ophthalmology followup after discharge, did you? A. To the best of my knowledge, I did not. Q. Do you know, or have any knowledge, of Dr. Luciano doing that with the family? A. I wouldn't know. Q. What was the plan of care at the point of discharge for Kevin on April 18th of 198? A. Generally, the surgeons will see the children back in the outpatient department at some point, one to two weeks later, to make sure the wound is healing well and for a checkup. And generally we see the patient back again for a CAT scan one to two months later to make sure everything is going okay. Q. Now, when you saw Kevin on June 9th of 98, was that for the followup that he was to have with you? Was that the scheduled followup? A. My guess would be that was the scheduled followup. Q. Now, when you saw him on that June 9th date, you indicated that you noted he did
 A. Sometime within a few months. However, had I seen him on June 9th and his optic nerve looked completely normal, I don't know if I would have even sent him back to Dr. Marcotty. Q. At the time of his discharge from the hospital on April 18th, 98, did you believe that Kevin had a chronic or persistent type papilledema? A. On April 18th when I sent him home, I felt that his increased intracranial pressure and arachnoid cyst had been adequately cared for. At that point, the papilledema was present, but if the shunt remained functional, it would be my opinion that the papilledema would disappear over the next few months. And the vast majority of patients would have normal optic nerve findings and normal visual findings following the resolution of the papilledema. In Kevin's case, when he was seen six weeks later, the papilledema resolved, but instead of a normal looking optic nerve, he had optic atrophy. Q. So you don't recall advising the family to get ophthalmology followup after discharge, do you? A. I don't recall one way or the other. 	 have some mild optic atrophy. Did he also still have papilledema to the level that he had when you saw him in the hospital? A. No. His papilledema was entirely gone. Q. And I believe you told me, you answered this already, that you did not do visual field testing at that time at that June 9th visit; is that correct? A. No, I did not. Q. Now, is there a reason why you referred him to Dr. Marcotty instead of someone at the Cleveland Clinic when you saw him on June 9th? A. Yes. Q. What was that reason? A. That he had seen Dr. Marcotty in the past and that we try to establish a continuing line of care within a specialty. So in my mind, no one knew his eyes better I should say no ophthalmologist knew his eyes better than Dr. Marcotty, so there would be no one better to follow him than Dr. Marcotty. Q. Did you have any additional plan of care for him other than referring him to Dr.

18 (Pages 69 to 72)

73 75 1 Marcotty? I O. And what was the reason that Kevin came to see you on that date? 2 A. I put down return visit PRN, which is 2 3 a way of saying as needed. So I didn't establish 3 A. My impression was that it was part of 4 any particular time that I wanted to see him 4 a followup visit, and I remember the parents had 5 back. What I really wanted is for Marcotty to 5 a lot of questions regarding the visual loss as a take a look at his eyes and tell me what was result of everything that had gone on. 6 6 7 7 Q. Did you do a physical exam of Kevin going on. 8 How soon did you want him to see Dr. 8 on that date? Ο. 9 Marcotty? Did you indicate that to Kevin's 9 **A.** It looks like my physical exam was 10 10 limited to looking at his eye, seeing optic parents? A. I didn't indicate it in the chart, 11 11 atrophy left eye greater than right, and a partial Marcus Cunn pupil on the left. 12 no. Under the situation, it would not have been 12 13 an emergency, but it would have been a statement 13 Q. What is a Marcus Cunn pupil? 14 like I would like you to see Dr. Marcotty as soon 14 A. That indicates that there is an as can be conveniently arranged. 15 injury to the left optic nerve. 15 Q. Did you have any evaluation by Dr. 16 Q. When you saw Kevin on June 9th, did 16 17 he voice any concerns about his vision? 17 Marcotty at the time that you saw him on October 18 No. 18 I 5th of 98 or by any other ophthalmologist from Α. 19 Q. Did you find that odd for a child of 19 the Cleveland Clinic, any other input? his age not to be able to tell you that there was 20A. Looks like -- yes, it looks like Dr. 20 vision problems there? 21 Kosmorsky from the Cleveland Clinic saw Kevin and 21 A. I would not find that odd. 22 22 had felt that his vision in his good eye was Now, did you speak with Mr. and/or 20/20, and his vision in his left eye was that he 23 Q. 23 24 Mrs. Kiss on the 9th and tell them of your 24 could detect hand motion, but nothing more than 25 that. 25 findings? 74 76 A. I put down here that I discussed the Q. Did you have any conversations with 1 Dr. Kosmorsky about Kevin's vision? 2 care plan management, prognosis for 20 minutes. 2 So it's my assumption that I spent a reasonable 3 3 Not that I recall. A. amount of time discussing with them my findings. 4 Any with Dr. Marcotty? 4 Q. 5 Was it your impression when you saw 5 A. I recall a phone call from Dr. Q. 6 him on the 9th that Kevin had some major vision 6 Marcotty. I don't recall what date the phone 7 call was, but my assumption is that it was after 7 loss? 8 A. I did not know that. If I did know 8 he had seen Kevin back in June. 9 that, I didn't document it. 9 Did you have any discussions with Dr. 0. Luciano about Kevin's vision loss? 10 Q. Did you indicate to Kevin's parents 10 at all that there was some vision loss? 11 A. Not that I recall. 11 You did not talk with him after you A. I don't have a recollection of that, 12 12 Q. 13 saw Kevin on June 9th of 98 to tell Dr. Luciano 13 but from the context of what's in the note, I that you had found some optic atrophy? 14 probably would have expressed some concern that 14 A. I can't imagine that I didn't discuss 15 15 there could be vision loss. 16 this with him, but I honestly do not recall any Q. And when you saw him on June 9th, 16 17 were there any signs of increased intracranial 17 discussion regarding that finding. In other pressure at that time? words, it would be my practice to call him up and 18 18 say this is what I found, but I don't recall 19 A. None. 19 20 having that discussion, nor any subsequent 20 Q. Now, you saw Kevin again after this 21 June 9th, 98 visit; is that correct, I believe in 21 discussions. 22 22And, doctor, it was your opinion that October? Q. 23 23 **A.** I know I saw him again after that. Kevin's vision loss was secondary to his 24 Let me refer to my note. Yes. October 15th, 24 increased intracranial pressure and papilledema; 25 25 correct? 1998.

19 (Pages 73 to 76)

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 A, It's my impression that his visual loss was a cumulative effect of the chronic increased intracranial pressure due to the arachnoid cyst and/or relief of that pressure. Q. Well, doctor, in your clinical note of October 15th, 98, under the P part of your note, didn't you state secondary to increased intracranial pressure and papilledema? A. That's what I put in the chart note, yes. The chart note doesn't reflect a complete set of opinions regarding what may or may not have transpired. Q. Now, that note of October 15th, 98 says you had a frank discussion about the visual loss, and that was with Kevin's parents? A. Correct. Q. Do you recall the content of that discussion that you had with Kevin's parents? A. Only in general. Q. Can you tell me what you do recall from that discussion. A. What I recall is that the parents were obviously upset that their son had developed severe visual loss in one eye. The question any reasonable parent would have is did something 	 1 such as putting him on Diamox to try to reduce 2 the intracranial pressure. 3 Q. Doctor, do you have an opinion, if 4 his shunting procedure had been done sooner, 5 whether it would have made a difference in regard 6 to his vision loss? 7 MS. CARULAS: Objection. 8 A. I don't have an opinion because I 9 don't think we can know whether or not the visual 10 loss is due to the long-standing papilledema or 11 to the relief of that papilledema or, more 12 likely, a combination of both. 13 Q. When you had the conversation with 14 Kevin's parents on October 15th of 98, was 15 anybody else present for that conversation 16 besides you and Kevin's parents? 17 A. I don't recall. My resident had seen 18 the patient prior to me seeing the patient, but I 19 don't recall if the resident was in the room at 20 the time, nor do I recall if Kevin's parents had 21 any other adult member of the family there, nor 22 do I recall if Kevin was even in the room at the 23 uny other adult member of the family there, nor 24 Q. At any point that you discussed 25 Kevin's case with Dr. Luciano, did you ever
78 1 wrong happen to my child, was there a medical error. 3 The discussion we had was almost 4 exactly the content of the discussion we're 5 having right now; a review of everything that had 6 happened to him up to this point, a review of all 7 the possibilities, a review of the fact that he 8 may have been destined to lose his vision prior 9 to medical involvement in this case in the first 10 place because relief of pressure can be damaging 11 to the optic nerve, that he may have been 12 destined to lose his vision at some point in time 13 prior to the ultimate shunting procedure done on 14 April 15th, and that, in my opinion, there was 15 very little, if anything, anyone could do to have 16 protected him from this rare, but obviously very 17 terrible, situation that he lost vision in one 18 eye; that the timing of the second surgical 19 procedure was appropriate because we all know 20 that placing a shunt is a procedure that has 21 risks, including the risk of something else 24 going wrong like bleeding, and that a careful 25 surgeon would have done an alternative treatment	 discuss whether he agreed that Kevin's vision loss was secondary to increased intracranial pressure and papilledema? Was that ever discussed with Dr. Luciano? A. I don't think so. Q. Aside from the dates that we just looked at, the ones that we've just previously reviewed, did you see Kevin on any other dates? A. Not that I recall. Q. Did you speak with Mr. and Mrs. Kiss on any other dates other than the dates that you saw him in the hospital and the visits that you had with him in the Clinic? A. There may have been a phone call somewhere in the middle, but I don't recall if there in fact was. Q. Do you have any criticism of any of the health care providers that provided care to Kevin? A. No. Q. Do you blame Kevin or his family in any way for the vision loss that he suffered? A. No. MS. TOSTI: I don't have any further questions for you, doctor, but other counsel may

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February 26, 2001

BRUCE H. COHEN, M.D. Kiss, et al. vs. Marcotty, M.D., et al.

81 1 have some. 2 MS. ATWELL: I have just a few. 3 EXAMINATION OF BRUCE H. COHEN, M.D. 4 BY MS. ATWELL: 5 Q. I'm the attorney for Dr. Marcotty; my 6 name is Cheryl Atwell. 7 Who was the resident who was with you 8 on April 14, 19981	 83 I AFFIDAVIT 2 I have read the Foregoing transcript from 3 page 1 through 82 and note the following 4 corrections: 5 PAGE LINE REQUESTED CHANGE 6 7
 9 A. The resident whose name I cannot read 10 could be identified through the Cleveland Clinic 11 Foundation medical records office. There's ways 12 to identify that resident, because all residents 13 have their signature on file. I don't recall who 14 that resident was, nor can I read that signature. 15 Q. Looking at the resident's note on 16 April 14, if you don't mind if I come and stand 17 by you, there's just a couple words I want you to 18 try to read. I don't want you to try to read the 19 whole thing because we can read most of it. On 20 April 14, the resident, on the first page, midway 	8 9 10 11 12 13 14 15 16 17 18 BRUCE H. COHEN, M.D. 19 20 Subscribed and sworn to before me this
 21 down, circled the number one, and then he wrote a 22 word. 23 A. It looks like panic attack past six 24 weeks. 25 Q. Got it. On the very bottom part of 	21 day of, 2000. 22 23 24Notary Public
 82 that first page, there's a word, and I can't read that word. I'm just pointing to it. A. Well, it says let me just try to Q. I know I've got a copy and that makes a difference, too. Emesis negative. It follows it. Before that, there's something. A. It looks like association. It looks like associations, but associations with increasing intracranial pressure. Emesis negative, et cetera, etcetera. MS. ATWELL: That's all I have. Thank you. MS. LOESEL: No questions. MS. CARULAS: You have the right to read over the transcript and make sure it's been taken down accurately, and I would recommend you do that. THE WITNESS: Okay. MS. TOSTI: Thank you, doctor. (Deposition concluded at 4:15 o'clock p.m.) (Signature not waived.) 	<pre> 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1</pre>

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Family:	Son-Jordan Benjamin Cohen, born 10-01-92 Daughter-Arielle Gaila Cohen, born 4-6-94

Education and Training:

Pediatnc Neuro-Oncology Fellowship. Children's Hospital of Philadelphia 34th & Civic Center Blvd Philadelphia, PA 19104 July 1, 1987-May 31, 1989

Pediatric Neurology Fellowship: Neurological Institute and Babies Hospital Columbia-Presbyterian Medical Center 630 West 168th Street New York, NY 10032 July I, 1984-June 30, 1987

Pediatnc Residency Children's Hospital of Philadelphia 34th & Civic Center Blvd Philadelphia, PA 19104 June 17,1982-June 30, 1984

Medical School: Albert Einstein College of Medicine 1300 Moms Park Avenue Bronx, NY 10461 Degree: M.D. June 1982



College:

Washington University Lindel and Skinker Blvd. St. Louis, MO 63105 Degree: A.B., Chemistry, Summa Cum Laude May, 1978

Hospital Appointments:

1985-1987	Assistant Attending Physician. Harlem Hospital Medical Center, New York, NY
1987-1989	Consultant in Neurology to Neuro-Oncology Clinic, Children's Hospital of Philadelphia, Philadelphia, PA
1987-1989	Consulting Neurologist The Neurofibroinatosis Clinic, Children's Hospital of Philadelphia, Philadelphia. PA
1988-1989	Instructor in Pediatrics, Children's Hospital of Philadelphia, University of Pennsylvania, Philadelphia, PA
1989	Clinical Associate, Department of Neurology, The Cleveland Clinic Foundation, Cleveland, Ohio
1989-1991	Assistant Staff, Department of Neurology, The Cleveland Clinic Foundation, Cleveland, Ohio
1991-	Staff, Department of Neurology, The Cleveland Clinic Foundation, Cleveland, Ohio
1992-	Associate Professor, Division of Neurology, Department of Pediatrics, Ohio State University College of Medicine
1999-	Head, Section of Pediatric Neurology: Cleveland Clinic Foundation, Cleveland, Ohio

Awards and Grants:

American Society of Clinical Oncology Travel Award, American Society of Clinical Oncology Meeting, San Francisco, May 21-24, 1989.

American Cancer Society, Clinical Oncology Fellow Grant 1988-1989.

Peter Preuss Foundation Grant, 1987-1988.

Eilene L. Schneider Award (Pediatric Department, Albert Einstein College of Medicine), May 1982.

Research Grant, Department of Anesthesia, Albert Einstein College of Medicine, 1979.

John C. Sowden Memorial Award (Chemistry Department, Washington University), April 1978.

St. Louis Chemical Council Award, March 1977.

Boards:

Fellow of the National Board of Medical Examiners, July 1, 1983. No. 270210.

Fellow of the American Board of Pediatrics, June 2, 1989, No. 39726. Recertified on 3/11/96 through 2003

Fellow of the American Board of Psychiatry and Neurology with Special Qualification in Child Neurology, March 1990, No. 720.

Memberships:

Child Neurology Society, 1988.

American Academy of Neurology, Fellow 1999, Active Member 1990. Junior Member 1985

Alpha Omega Alpha, Albert Einstein College of Medicine, 1981

Sigma Xi North American Scientific Society), Associate Member, 1978

American Medical Association, 1978

American Epilepsy Society, 1991

Committee Appointments.

NCI Investigator Number: 22513

1989-1992	Brain Tumor Autologous Marrow Transplant Committee (CCG-9883/9004), The Children's Cancer Study Group
1989-1995	Medulloblastoma/PNET Committee (CCG-9892), The Children's Cancer Study Group
1989-1993	Clinical Care Committee, The National Neurofibromatosis Foundation
1990-1992	Bone Marrow Transplantation Committee, The Children's Cancer Study Group
1990-1994	High-Grade Astrocytoma/Autologous Marrow Rescue and Irradiation Committee (CCG-9010, CCG-9922), The Children's Cancer Study Group
1991-	Medical Professional Advisory Committee, Achievement Center for Children, Cuyahoga County
1993-1996	Treament of Children with low-stage medulloblastoma Standard-dose cranial spinal irradiation vs reduced dose cranto spinal irradiation plus adjuvant chemotherapy with cisplatin, cyclophosphamide and vincristine (CCG-9014), The Childrens Cancer Group
1994-1999	Brain Tumor Strategy Committee, Childrens Cancer Group
1994-1996	Chairman, Task Force on Malignant Infant Brain Tumors, Childrens Cancer Group
1995-1996	Task Force on Neurofibromatosis, Childrens Cancer Group
1995-	Practice Committee, Child Neurology Society
1995-	Chairman, CPT Coding Subcommittee of the Practice Committee, Child Neurology Society
1996-	Vice Chairman, Practice Committee, Child Neurology Society
1997-	Chairman, CCG-99703C Infant Brain Tumor Study, Children's Cancer Group
1997-	Member, Board of Trustees, United Mitochondrial Disease Foundation
-------	--
1998-	Member, Board of The Accreditation Council for Pediatric Neurosurgery
1499-	Steering Member, The Neuroscience Committee, Children's Cancer Group
1999-	Chairman, Low-Grade Astrocytoma Discipline Committee, Children's Oncology Group
1999-	Brain Tumor Strategy Committee, Children's Oncology Group
2000-	Chairman, Compliance and E&M Committee, Child Neutology Section, Amencan Academy of Neurology
2000-	Professional Advisory Board, The Gathenng Place A Wellness Community for Those Touched by Cancer

Ad Hoc Reviewer

Annals of Neurology Neurology Medical and Pediatric Oncology Journal of Neuro-Oncology Muscle and Nerve

Original Papers:

1. Y. Chen. B. Cohen, P.P. Gaspar: Rearrangement of bis(trimethylsilyl) silylene (Me₂Si)Si in the gas phase; new silylene-silylene interconversions. Journal of Organometallic Chemistry 1978;195.

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6. Bruce H. Cohen, Roger J. Packer: Tumors of the central nervous system. In, <u>Practical Pediatric Oncology</u>. G. J. D'Angio, D. Sinniah, A.T. Meadows, A.E. Evans, J. Pritchard (eds), London, Edward Arnold, 1992.

7. Roger J. Packer, Bruce H. Cohen: Neurologic Emergencies. In, <u>Practial Pediatric Oncology</u>. G. J. D'Angio, D. Sinniah, A.T. Meadows, A.E. Evans, J. Pritchard (eds), London, Edward Arnold, 1992.

8. Earl Zimmerman and Bruce H. Cohen: Congenital 'Tumors. In, <u>Merritt's Textbook of Neurology. 9th Edition</u>. Lewis P. Rowland, (ed), Williams & Wilkins, Baltimore, 1995.

9. Bruce H. Cohen: Headache as a symptom of neurologic disease. In, <u>Seminars in Neurology</u>. A.D. Rothner (ed). 2:144-150,1995.

10 Bruce **H.** Cohen and James Garvin. Brain Tumors In <u>Roudoloh's Pediatrics. 20th Edition</u> Rudolph, Hoffman, Rudolph (eds), Appleton & Lange, Stamford, 1900-1920, 1996

11. Bruce **H.** Cohen, Roger J. Packer: Neurologic Aspects of Childhood Brain Tumors. In, <u>Child Neurology</u>. Augustine Legido (ed). Awaiting publication.

12. Bruce H. Cohen: Headaches. In: <u>Practical Strategies In Pediatric Diagnosis and Therapy</u> Kliegman (ed), W.B. Saunders Company, Philadelphia. 574-589, 1996.

13. Bruce H.Cohen and Roger J. Packer: Tumors of the Fourth Ventricle. In, Cohen Alan (ed), Surgical Disorders of the Fourth Ventricle, Blackwell Science, Cambridge, Mass, 1996.

14. Roger J. Packer and Bruce **H.** Cohen: Germ Cell Tumors and Pinealoma. In: <u>Handbook of Clinical Neurology</u>. Vinken & Bruyn (eds), Elsevier Science. 1998.

15. Arie Weinstock and Bruce **H.** Cohen: In, Luders (ed), <u>Epileptic Seizures: Pathophysiology and Clinical Semiology</u>, W.B. Saunders Co.

16 Bruce H. Cohen and John C. Andrefsky: Increased Intracranial Pressure. In: Maria BL (ed), <u>Current Management in Child</u> Neurology, B.C. Decker Inc., Hamilton, Ontario, 325-330, 1999.

17. Bruce H. Cohen, Elizabeth C. Dooling, A. David Rothner. Abnormal Signs and Symptoms (Ataxia, Bell's Palsy, Nystagmus, Papilledema, and Ptosis) In: Derschewitz RA (ed), <u>Ambulatory Pediatric Care 3rd Edition</u>. Lippincott-Raven, Philadelphia, 822-829, 1999.

18. Bruce H. Cohen: Approaches to Brain Tumors in Children. The Neurologist 5(2):75-89;1999.

19. Deborah R.Gold, Roger J. Packer, Bruce H. Cohen. Chemotherapy for Primitive Neuroectodermal Tumors. Neurosurg Focus 7 (2):Article I, 1999 (http://www.aans.org/journals/online_j/august99/7-2-1.html)

Books and Monographs:

1 Bruce H. Cohen, Bruce R Korf, Jane N Pugh, eds Neurofibromatosis 2 Jane Novak Pugh Conference Series, Volume 4 The National Neurofibromatosis Foundation, New York, 1992

Other Publications:

1. Pamelyn Close, David Friedman, Antonia Uri: Viral-Associated Hemophagocytic Syndrome. Proceedings of the Tumor Board of the Children's Hospital of Philadelphia. Quoted statements of the proceedings. Medical and Pediatric Oncology 1990; 18:119-122.

2 Bruce 11. Cohen Clinic Notes The Plain Dealer, April 9, 1991

3. Bruce H. Cohen: Book Review. Neurofibromatosis: Phenotype, Natural History, and Pathogenesis, 2nd ed., by V.M. Riccardi. Neurology 1992;42:2308.

4. Bruce H. Cohen: Medical Questions. Akron Beacon Journal, December 15, 1992.

5. Bruce H. Cohen: Medical Questions. Akron Beacon Journal, August 15, 1995.

6. Bruce H. Cohen: Nonepileptic Movement Disorders. Audio-Digest Pediatrics Volume 42, Number 07. April 9, 1996.

7. Bruce H. Cohen: Mitochondrial Cytopathies: Evaluation and Management. United Mitochondrial Disease Foundation Newsletter. Volume 2, Issue 3, 1997.

8. Bruce **H.** Cohen, John Shoffner, Glenn DeBoer: Anesthesia aiid Mitochondrial Cytopathies. United Mitochondrial Disease Foundation Newsletter. Volume 3, Issue 1, 1998.

9. Bruce **H.** Cohen: Strokes in MELAS and Mitochondrial Cytopathies. United Mitochondrial Disease Foundation Newsletter. Volume 3, issue 3, 1998.

10. Featured in a front-page article on Rett Syndrome. The Chronicle Telegram (Elyria, Ohio). October 3, 1999.

11. Bruce H. Cohen: Book Review. Neuro-oncology: The essentials, edited by Mark Bemstein and Mitchel S. Berger.

Presentations:

1 Central nervous system melanotic neuroectodermal tumor of infancy (CNS-MNTI) Value of chemotherapy in management American Academy of Neurology, Section on Neuro-Oncology, New York, NY, April 9, 1987

2. Brain tumors (BT) in children under two years: Treatment, survival, and long-term prognosis. American Academy of Neurology, Section on Child Neurology, Cincinnati, OH, April 1988.

3. Gadolinium-DTPA (GAD)-enhanced MRI imaging in childhood brain tumors. American Academy of Neurology, Section on Neuro- Oncology, Cincinnati, OH, April 1988.

4. Neurologic and magnetic resonance imaging abnormalities in symptomatic and asymptomatic children with neurofibromatosis type 1: Incidence and significance. Child Neurology Society, Neuro- Oncology Section, Halifax, Nova Scotia, September 1988.

5. "Phosphorous magnetic resonance spectroscopy (³¹P-MRS) in childhood brain tumors. American Academy of Neurology, Session on Neuro-Oncology, Chicago, IL, April 13-19, 1989.6. Ototoxicity of cis-platin in children with brain tumors receiving cranial irradiation. American Society of Clinical Oncology, San Francisco, CA, May 21-23, 1989.

7. "Poor-risk" medulloblastoma: Improved three-year disease- free survival after treatment with chemotherapy. Child Neurology Society 1989 Meeting, San Antonio, TX, October 11-13, 1989.

8. MR imaging of the brain in children with neurofibromatosis types I and II: Focal areas of abnormal signal intensity. The Radiological Society of North America, Chicago, IL, November 28, 1989, Section of Neuroradiology.

9 Integrated MRI/³¹P MRS studies of large pediatric brain tumors The Radiological Society of North America, Chicago, IL, November 28, 1989

10. High-dose chemotherapy with bone marrow rescue in children and young adults with recurrent high-grade brain tumors. Proceedings of the Fifth International Symposium on Autologous Bone Marrow Transplantation, Omaha, August 1990.

11. Progressive visual loss: A rare manifestation of familial cavernous angiomas. Child Neurology Society 1990 Meeting, Atlanta, GA, October 18-20, 1990.

12. Murine antiepidermal growth factor monoclonal antibody (alphaEGFr MAR 425) for the imaging and treatment of childhood brain tumors. Child Neurology Society 1990 Meeting, Atlanta, GA, October 18-20, 1990.

13 Outcome of children with medulloblastoma/primative neuroectodermal tumors of the posterior fossa in the modern ern Improved survival with adjuvant chemotherapy Child Neurology Society 1990 Meeting, Atlanta, GA, October 18-20, 1990

14 Clinical Overview Neurofibromatosis Type 2 Neurofibromatosis Symposium, Boston, April 28, 1991

15 Cerebral Gangliogliomas in Childhood Presentation, Pathologic Vanability, Treatment and Outcome Child Neurology Society 1991 Meeting, Portland OR, October 1991

16 Lack of benefit of intravenous gammablobulin in treatment of intractable childhood epilepsy (ICE) in children with and without IgG-subclass deficiency Child Neurology Society 1992 Meeting, New Orleans LA, October 1992

18. Neurofibromatosis 2 in young patients: *An* "aggressive" disorder. Child Neurology Society 1992 Meeting, New Orleans LA, October 1992.

19 Epilepsy and Metabolic Disease Amencan Epilepsy Society 1992 Meeting, Seattle, WA, December 6, 1992

20 Neurofibroniatosis 2 in young patinets Imaging Expenence Amencan Society of Neuroimaging, 16th Annual Meeting Orlando, Florida, February 3, 1993

21 Phase I trial of intrathecal 4-hydroperoxycyclophosphamidefor neoplastic meningitis. AACR, April 9, 1993.

22. Paroxysmal Non-Epileptiform Disorders. American Academy of Neurology Annal Course. New York, NY, May 1, 1993.

23. Randomized trial of CCNU, vincristine and prednisone versus "8-in-1" chemotherapy in the treatment of supratentorial primitive neuroectodermal tumors (S-PNET): A Childrens Cancer Group Study. Child Neurology Society Meeting. Orlando, Florida. October 14-16, 1993.

24. Segmental neurofibromatosis. Child Neurology Society Meeting. Orlando, Florida. October 14-16, 1993

26. Treatment of "poor-risk" medulloblastoma with radiation and chemotherapy: 5-year progression-free survival. Ann Neurol 1993,34:270. Child Neurology Society Meeting. Orlando, Florida. October 14-16, 1993.

27. Kotagal P. Bourgeois, B, Cohen B, Wyllie E. Infantile spasms in a child with brain tumor: Seizure-free outcome after resection. Epilepsia 1993;34:99.

28. Progression-free survival of children with "poor-risk" medulloblastoma after treatment with radiotherapy and CCNU, vincristine and cis-platinum chemotherapy. 6th International Symposium on Pediatric Neuro-Oncology.

28. Response to tamoxifen in patients failing conventional therapy for malignant glioma. American Association of Neurological Surgeons 62nd Annual Meeting. San Diego, CA, April 9-14, 1994.

29. Paroxysmal Non-Epileptiform Disorders. American Academy of Neurology Annal Course. New York, NY, May 2, 1994

30. The Role of Adjuvant Chemotherapy in the Treahnent of Brain Tumors. International College of Surgeons. Cleveland, Ohio, June 11, 1994.

31. High-Dose Thiotepa and Etoposide with Autologous Marrow Rescue (ABMR) for Children and Young Adults with Recurrent Central Nervous System (CNS) Tumors. International Symposium of Pediatric Neuro-Oncology, Houston, TX, August 3, 1994.

32. Paroxysmal Non-Epileptiform Disorders. American Academy of Neurology Annal Course. Seattle, WA, May 8, 1995

33. Neurologic Causes of Headaches. Amencan Academy of Neurology Annal Course. Seattle, WA, May 8, 1995.

34. Clinical Usefulness of Magnetic Resonance Imaging in Pediatric Headache. Cleveland Clinic Foundation 20th Annual Neuroscience Residents' Day. Cleveland, Ohio, May 25, 1995.

35 External Ophthalmoplegia and Spindle Coma in Combined Carbamazipine and Primidone Overdose Case Reprt and Review of the Literature Cleveland Clinic Foundation 20th Annual Neuroscience Residents' Day Cleveland, Ohio, May 25, 1995

36 Benign Brainstern Lesions in Children with neurofibromatosis -1 Cleveland Clinic Foundation 20th Annual Neuroscience Residents' Day Cleveland, Ohio, May 25, 1995

37. Benign Brainstem Lesions in Children with Neurofibromatosis type 1. The NNFF International Consortium for Molecular Biology of NF1 and NF2. Philadelphia, PA, July 14-16, 1995.

38. Spinal Neurofibromatosis: NF1, NF2, or neither? The NNFF International Consortium for Molecular Biology of NF1 and NF2. Philadelphia, PA, July 14-16, 1995.

39. Clinical Usefulness of Magnetic Resonance Imaging in Pediatric Headache. Child Neurology Society Annual Meeting, Baltimore, MD, October 27, 1995.

40 Brainstem lesions in children with Neurofibromatosis type 1 Child Neurology Society Annual Meeting, Baltimore, MD, October 27, 1995

41. Early results of reduced-dose radiotherapy plus chemotherapy for children with nondisseminated medulloblastoma: **A** Children's Cancer study. Child Neurology Society Annual Meeting, Baltimore, MD, October 27, 1995.

42. Results of Linac-based stereotactic radiosurgery for newly diagnosed glioblastoma multiforme. LINAC Radiosurgery Meeting sponcered by The Florida Neurosurgical society. Lake Buena Vista, FL, December 6-10, 1995.

43. Linac-based stereotactic radiosurgery for newly diagnosed malignant gliomas. Academy of Neurology Annual Meeting, San Francisco, CA, March 26, 1996.

44. Seizures In Patients with Cancer and Brain Tumors. Symposium on Epileptic Seizures: Pathophysiology and Semiology. Seventh International Cleveland Clinic-Bethel Epilepsy Symposium. Cleveland, Ohio May 12, 1996.

45, PSG and MSLT in Kleine-Levin Syndrome and Periodic Hypersomnia. Americcan Professional Sleep Society. San Francisco, CA. June 13, 1997.

46. Is Whole Brain Radiation Therapy Needed for al Patients with Newly Diagnosed Brain Metastases Undergoing Stereotactic Radiosurgery? Submitted to ASTRO.

47. Adhalin deficiency in a patient with limb-girdle muscular dystrophy. *Y* Zhang, B Cohen, K Levin. 22nd Annual Neuroscience Residents' Day, Cleveland, OH, May 22, 1997.

48. Radiographic and clinical characteristics of children treated at the Cleveland Clinic for acute deisseminated encephalomyelitis. S Friedman, B Cohen. 22nd Annual Neuroscience Residents' Day, Cleveland, OH, May 22, 1997.

49. Defects in oxidative phosphorylation manifesting as psychotic encephalopathy in an adult. R Dukkipati BH Cohen. 22nd Annual Neuroscience Residents' Day, Cleveland, OH, May 22, 1997.

50 Incidence of concussion in high school football players W Langburt, N Akhtar, K O'Neill, B Cohen 22nd Annual Neuroscience Residents' Day, Cleveland, OH, May 22, 1997.

51. Mucopolysaccharidosis in an 18 year old man presenting with stroke. K O'Neill, B Cohen. 22nd Annual Neuroscience Residents' Day, Cleveland, OH, May 22, 1997.

52. Is Whole Brain Radiation Therapy Needed For All Patients with Newly Diagnosed Brain Metastases Undergoing Stereotactic Radiosurgery? American Society for Therapeutic Radiation Oncology. Orlando, FL. October 21, 1997.

53. ICD-9 and CPT Coding for the Child Neurologist. Breakfast Seminar. Child Neurology Society Annual Meeting. Pheonix, AZ, October 30, 1997.

54. Thalidomide for the Treatment of Plexiform Neurofibromas in Neurofibromatosis Type 1. Breakfast Seminar. Child Neurology Society Annual Meeting. Pheonix, AZ, October 31, 1997.

55. Disorders of Oxidative Metabolism in Children Undergoing Video-EEG monitoring. 1997 American Epilepsy Society Annual Meeting, Boston, MA. December 10, 1997.

56. Defect in Mitochondrial Function Manifesting as Psychotic Encephalopathy. 1st Mitochondrial Medicine Conference San Diego, CA, February 21, 1998.

57. Fatal Status Epilepticus In A 9-Year Old Girl Due to A Defect Mitochondrial Electron Transport Chain Complex II. 1st Mitochondrial Medicine Conference. San Diego, CA, February 21, 1998.

58. Disorders of Oxidative Metabolism Presenting as Paroxysmal Non-Epileptic Disorders. 1st Mitochondrial Medicine Conference. San Diego, CA, February 21, 1998.

59. Systemic T cell adoptive immunotherapy of malignant gliomas. AACR, New Orleans, LA. March 1998.

60. Reversible Cardiomyopathy/Myopathy Due to a Disorder of Long Chain Fatty Acid Metabolism. 23rd Annual Neuroscience Residents' Day, Cleveland, OH, May 14, 1998.

61 The Spectrum of Mitochondrial Complex I Deficiencies. A Biochemical and Clinical Study. American Academy of Neurology Annual Meeting, Toronto, CA, April 21, 1999

62. Polarographic and Spectrophotometric Analysis of 80 Patients with Mitochondrial Cytopathies: A Clinical, Biochemical, and Pathologic Study. Euromit 4. Cambridge, Great Britain, September 16, 1999.

63. Well Known Neurological Conditions That Have An Associated Electron Transport Chain Deficiency. Euromit 4. Cambridge, Great Britain, September 17, 1999.

64 Neurocognitive Status in children with medulloblastoma following reduced-dose craniospinal radiotherapy and chemotherapy Child Neurology Society Annual Meeting, Nashville, TN, October 14, 1999

65. Characterization of Biochemical and Clinical Features of Children with Electron Transport Chain Complex III Deficiency. Child Neurology Society Annual Meeting. Nashville, TN, October 16, 1999.

66. Making Sense of Respiratory Chain Analysis. Mitochondiral Symposium. National Institutes of Health. Bethesda, MD, March 14,2000.

67. Clinical Aspects of Mitochondrial Cytopathies in Children and Adults. Mitochondrial Cytopathies 2000 – Professional Conference. Cleveland, OH, June 1, 2000.

68 Clinical Aspects of Mitochondrial Cytopathies in Children and Adults Mitochondrial Cytopathies 2000 – Family Conference Cleveland, OH, June 2, 2000

- 69. Case Presentations in Mitochondrial Medicine. Mitochondrial Cytopathies 2000 Professional Conference. Cleveland, OH, June 3, 2000.
- 70 Overview Of Scientific Presentations Mitochondrial Cytopathies 2000 Family Conference. Cleveland OH, June 3, 2000

71.

Media Appearances:

1 Live at Five, Channel 3 News, Discussion about the report concerning the increase incidence of childhood brain tumors June 18, 1991

- 2. Live at Five, Channel 3 News, Discussion about Neurofibromatosis. February 23, 1993
- 3 CBS Evening News, Discussion about Brain Tumors. March 13, 1995.
- 4. WJW TV-8 Evening News, Adrenoleukodystrophy. June 2, 1995.
- 5 WERE 1300AM Health Care 95, One-hour discussion about brain tumors and neurofibromatosis November 15, 1995
- 6 WERE 1300AM Health Care 96, One-hour discussion about pediatric neurology April 3, 1996
- 7. New York Times, Antineoplastin Therapy. July 25, 1996.
- 8. WERE 1300AM Health Care 96, One-hour discussion about pediatric neurology. September 18, 1996
- 9. Channel 5 Evening News, Mitochondrial Cytopathies, July 20, 1998

Invited Lectures:

1. The Child with a Brain Tumor, Symposium for Educators of Elementary and High School Students with Cancer, Philadelphia, PA, December 1987.

2. Neurologic Aspects of Neurofibromatosis. Neurology Grand Rounds, Hospital of the University of Pennsylvania, February 4, 1988.

3 Long-tenn Neurologic Effects of Cancer Therapy. Symposium for Teachers and Parents, Learn Conference, Philadelphia, PA, April 29, 1988.

4 Pediatric Brain Tumors Pediatric Grand Rounds. The Cleveland Clinic Foundation, May 24, 1988

5. Neurologic Effects of Therapeutic Irradiation: Neurology Grand Rounds, The Cleveland Clinic Foundation, May 25, 1988.

6 Long-term Neurologic Effects of Cranial and Spinal Irradiation Symposium for Teachers and Parents, Learn Conference, Philadelphia, PA, November 11, 1988

7 Learning Disabilities in Neurofibromatosis Type 1 10th Annual Meeting, The National Neurofibromatosis Foundation, Inc, Philadelphia, PA, November 12, 1988

8. The Child with a Brain Tumor. Symposium for Educators of Elementary and High School Students with Cancer, Philadelphia, PA, December 2, 1988.

9. Learning Disabilities in Neurofibromatosis: Meeting of the Ohio Chapter of the National Neurofibromatosis Foundation, Inc., Cleveland, OH, April 9, 1989.

10 Research Progress in Neurofibromatosis: 1989 Northeastern Regional Seminar, Sponsored by Singles Helping Others Princeton, NJ, Apnl 29, 1989

11. Neurofibromatosis: Pediatnc Grand Rounds, The Cleveland Clinic Foundation, September 12, 1989

12. Neurofibromatosis Update: Neuro-ophthalmology Grand Rounds, The Cleveland Clinic Foundation, September 22, 1989.

13. Cancer in Neurofibromatosis: The Cleveland Clinic Cancer Grand Rounds, The Cleveland Clinic Foundation, October 3, 1989.

14. Management of Nervous System Neoplasms in Children with Neurofibromatosis. The Children's Cancer Study Group, Neurology Committee and Brain Tumor Strategy Committee, Denver, November 4, 1989.

15. The Role of Actinomycin-D in Future Pediatric Brain Tumor Trials. The Children's Cancer Study Group, New agents Committee, Denver, November 4, 1989.

16. The Neurologic Aspects of Neurofibromatosis Type 1. Neurology Grand Rounds, The Cleveland Clinic Foundation, February 19, 1990.

17. Clinical Aspects of Neurofibromatosis Type 1 and 2. ENT Grand Rounds, The Cleveland Clinic Foundation, March 3, 1990.

18 The Medical Approach to the Patient with Neurofibromatosis Medical Grand Rounds, The Cleveland Clinic Foundation, March 29, 1990

19. New Chemotherapeutic Approaches in the Treatment of Children with Brain Tumors. Pediatric Grand Rounds, Mount Sinai Hospital, Cleveland, Ohio, March 30, 1990.

20. The Dying Child: End of Life Decisions. Pediatric Grand Rounds, The Cleveland Clinic Foundation, May 1, 1990

21 Association of Cancer in Neurofibromatosis Types 1 and 2 The Ohio Chapter of The National Neurofibromatosis Association Cleveland, OH, June 3, 1990

22 Pediatnc Brain Tumor Clinic Report. The Cleveland Clinic Cancer Center Grand Rounds June 12, 1990

23. Childhood Brain Tumors. EEG Technologist Rounds. The Cleveland Clinic Foundation. October 5, 1990.

24. The Neurologic Assessment of Children and Adolescence with Psychiatric Symptoms. Pediatric Neurology Update. The Cleveland Clinic Foundation. October 31, 1990.

25. Unusual Brain Tumors. Pediatric Neurology Update. The Cleveland Clinic Foundation. October 31, 1990.

26. Management of Minor Head Trauma in Children. Pediatric Neurology Update. The Cleveland Clinic Foundation. October 31, 1990.

27. Medical Aspects of Mental Retardation in Children. Professional Association for Retardation. 1990 Convention. Columbus, Ohio. November 5, 1990.

28. Medical Aspects of Epilepsy in Children. Professional Association for Retardation. 1990 Convention. Columbus, Ohio. November 5, 1990.

29. Headaches in Children. St. John's Westshore Pediatric Workshop. Mamott Hotel, Cleveland, Ohio. November 14, 1990.

30. The Neurological Assessment of Children Presenting with Psychiatric Symptoms. Neuroscience Grand Rounds, The Cleveland Clinic Foundation, November 26, 1990.

31. Pediatric Brain Tumor Case Reports: Brainstem Ganglioglioma and Cauda Equina Primitive Neuroectodermal Tumor. Cancer Center Grand Rounds, The Cleveland Clinic Foundation, November 27, 1990.

32. Craniopharyngioma in Children: Controversies in Diagnosis and Treatment. Mount Sinai Hospital, Department of Pediatrics, Grand Rounds. December 7, 1990.

33. Neurologic Aspects of Neurofibromatosis Type 1. Neurofibromatosis Symposium, Ft. Lauderdale, FL, March 9, 1991.

34. Neurologic Aspects of Neurofibromatosis Type 2. Neurofibromatosis Symposium, Ft. Lauderdale, FL, March 9, 1991.

35. Neurologic Exam in Children with Cancer. Pediatnc Oncology Nurses, Rainbow Babies and Children's Hospital, Cleveland, OH. March 21, 1991.

36. Ethical Issues in Neurology. John Jay High School Science Symposium, Cleveland OH. March 22, 1991

37. Neurologic Causes of Psychiatric Symptoms in Childhood. Pediatric Grand Rounds, The Cleveland Clinic Foundation, April 2, 1991.

38. Anticonvulsant Medication in the Difficult Patient with Seizures. Comprucare Symposium, The Cleveland Clinic Foundation, May 4, 1991.

39. Learning Disabilities in Neurofibromatosis. The Ohio Chapter Annual Neurofibromatosis Symposium, Cleveland, Ohio, May 5, 1991.

40. Management Strategies for Children with Craniopharyngiomas. Pediatric Grand Rounds, The Cleveland Clinic Foundation, May 21, 1991.

41. Actinomycin-D Neurotoxicity. Rainbow Babies & Children's Hospital Tumor Board, May 30, 1991.

42. High-Dose Chemotherapy with Bone Marrow Rescue in Children with Recurrent Malignant Brain Tumors. Neuroscience Grand Rounds, The Cleveland Clinic Foundation, June 24, 1991.

43. The Clinical Aspects of Neurofibromatosis Types 1 and 2. Special Lecture. Great Lakes Rehabilitation Hospital and The Harnot Medical Center. Erie, PA, July 17, 1991.

44. Effects of Neurosurgery and Radiotherapy on Patients with Brain Tumors. Caring Touch Conference. Ritz-Carlton Hotel, Cleveland, OH, September 29, 1991.

45. Adjunctive Therapies in Children with Brain Tumors. Pediatric Rehabilitation Conference, sponcered by Great Lakes Rehabilitation Hospital. Hilton Hotel South, Cleveland, OH, January 18, 1992.

46. Neurofibroniatosis 1992 Update: NF1 and NF2. Neuroscience Grand Rounds, The Cleveland Clinic Foundation, February 24, 1992.

47. Molecular Genetics and Neurofibromatosis 1: An Introductory Discussion for the Pediatrician. Pediatric Grand Rounds, The Cleveland Clinic Foundation, March 3, 1992.

48. Ethical Aspects of Anencephaly. Medical Ethics, Shaker Heights High School. March 19, 1992.

49. Neurologic Conditions in Children and Adolescents Presenting with Psychiatric Problems. Psychiatry Rounds. March 24, 1992.

50. Advances in the Treatment of Brain Tumors in Children. Child Neurology Course, The American Academy of Pediatrics. New York City. April 14, 1992.

51. Medical Management of Adults with Neurofibromatosis. Annual Meeting of The Ohio Chapter of The Neurofibromatosis Foundation, Cleveland, OH. April 26, 1992.

52. Recurrent Brain Tumors in Children. Pediatric Tumor Board, Rainbow Babies and Childrens Hospital, Cleveland, OH. July 23, 1992.

53. Neurologic and Behavioral Outcome in Children with Craniopharyngiomas: Is There a Case for Less Agressive Surgery? Health Hill Hospital Grand Rounds, Cleveland, OH. September 1, 1992.

54. Treatable Neuromuscular Diseases. Update 1992. Pedatric Grand Rounds, Cleveland Clinic Foundation, Cleveland, OH. September 22, 1992.

55. Epilepsy and Metabolic Disease. Epilepsy Grand Rounds, Cleveland Clinic Foundation, Cleveland, OH. January 27, 1993.

56. Epilepsy and Metabolic Disease, Pediatric Grand Rounds, Metro Health Center, Cleveland, OH. February 4, 1993.

57. Overview of Current Phase II and II Studies in Childhood Brain Tumors. Childrens Cancer Group Affiliates Meeting, Columbus, OH, February 12, 1993.

58. The Treatment of High-Grade Astrocytomas with High-Dose Chemotherapy and Autologous Marrow Rescue, Cancer Center Grand Rounds, Cleveland Clinic Foundation, Cleveland, OH. February 23, 1993.

59. Paroxysmal Non-Epileptic Disorders. American Academy of Neurology Annual Course. New York, New York, May 1, 1993.

60. Neurologic Causes of Leaning Disabilities. 1993 Meeting of The Ohio Learning Disabilities Association. Cleveland, OH October 7, 1993.

61. Paroxysmal Non-Epileptic Disorders. Epilepsy and Related Disorders in Children, Pediatric Neurology Course, The Cleveland Clinic Foundation. November 3, 1993.

62. Pediatric Brain Tumors: Advances of the Last Decade. Metro Hospital, Pediatric Grand Rounds, Cleveland, OH December 9, 1993.

63. Pediatric Brain Tumors: Advances of the Last Decade. Fairview General Hospital, Pediatric Grand Rounds, Cleveland, OH. December 17, 1993.

64 Pediatnc Brain Tumors Overview, Advances and Effects of Therapy Mt Sinai Hospital, Pediatric Grand Rounds. Cleveland, OH May 20, 1994

65. Leptomeningeal Carcinomatosis. Neurology Grand Rounds. The Cleveland Clinic Foundation. October 12, 1994.

66. Paroxysmal Non-Epileptic Disorders. Epilepsy and Related Disorders in Children, Pediatric Neurology Course, The Cleveland Clinic Foundation. November 2, 1994.

67. Progress in Pediatric Brain Tumors: 1985-1995. Pediatric Grand Rounds, The Cleveland Clinic Foundation. January 24, 1995.

68. Leptomeningeal Carcinomatosis: New Treatments. Cancer Center Grand Rounds. The Cleveland Clinic Foundation January 27, 1995.

69. Leptomeningeal Carcinomatosis: Impact of New Treatments on Pediatric Cancers. Ohio State University/Childrens Hospital of Columbus CCG Affiliated Meeting. Columbus, Ohio, February 16, 1995.

70. The History of Chemotherapy for Brain Tumors. Fronters in Neuroscience Course, The Cleveland Clinic Foundation. Mamott Hotel, February 26, 1995.

71. Metastasis Cerebrales. Curso Internacional de Actualizacion en Neurologiia. Hospital Central Militar, Mexico City, Mexico. March 7, 1995.

72. Tratamiento Actual de los Tumores Cerebrales Primanos. Curso Internacional de Actualización en Neurologiia. Hospital Central Militar, Mexico City, Mexico, March 7, 1995.

73. Thalidomide as a Potent Inhibitor of Angiogenesis: Possibilities for Utility in Neurofibromatosis. Presented at The NNFF International Consortium for Molecular Biology of NF1 and NF2, Philadelphia, PA. July 16, 1995.

74. Paroxysmal Non-Epileptic Disorders. Intensive Review of Pediatrics, The Cleveland Clinic Foundation. July 19, 1995

75. The Hypotonic Infant. Review Course in Pediatric Neurology. Cleveland Clinic Foundation. November 29, 1995.

76. Thalidomide as a Potent Inhibitor of Angiogenesis: Possibilities for Utility in Neurofibromatosis. Department of Neurology Grand Rounds, November 22, 1995.

77. Pyruvate and Mitochondrial Metabolism. Epilepsy Grand Rounds. The Cleveland Clinic Foundation. March 7, 1996

78. Paroxysmal Non-Epileptic Disorders. Intensive Review of Pediatrics, The Cleveland Clinic Foundation. July 16, 1996

79. Mitochondrial Cytopathies and Oxidative Stress: its Not Just for Kids Anymore. Neurology Grand Rounds. The Cleveland Clinic Foundation. October *30*, 1996.

80. Mitochondrial Cytopathies in Pediatrics. Pediatric Teaching Conference. Rainbow, Babies and Children's Hospital, April 17. 1997.

81. Paroxysmal Eon-Epileptic Disorders. Intensive Review of Pediatrics, 'The Cleveland Clinic Foundation. August 1997

82. Mitochondrial Cytopathies in Pediatrics. Pediatric Grand Rounds. The Cleveland Clinic Foundation. September 23, 1997.

83. Rationale Strategies for Treating Plexiform Neurofibromas and Optic Gliomas in Patients with NF1. Neurofibromatosis Symposium. Ohio Chapter NF Foundation, Cleveland, OH November 9, 1997.

84 Defect in Mitochondrial Function Manifesting as Psychotic Encephalopathy International Mitochondrial Medicine Conference, San Diego, CA February 21, 1998

85 Fatal Status Epilepticus In A 9-Year Old Girl Due to A Defect Mitochondrial Electron Transport Chain Complex II International Mitochondrial Medicine Conference, San Diego, CA February 21, 1998

86. Disorders of Oxidative Metabolism Presenting as Paroxysmal Non-Epilepic Disorders. International Mitochondrial Medicine Conference, San Diego, CA February 21, 1998.

87. Dystonia and Oxidative Phosphorylation. Orthopedic Grand Rounds. Cleveland Clinic Foundation. March 2, 1998.

88. Mitochondrial Cytopathies in Adults: Its not Just for Kids Anymore. University Hospitals of Cleveland, Department of Neurology Grand Rounds. March 6, 1998.

 New Treatment Strategies for Adult High-Grade Astrocytomas. Rhone-Pollene Rorer Seminar. Cleveland, OH July 9, 1998.

90. Paroxysmal Non-Epileptic Disorders. Intensive Review of Pediatrics, The Cleveiand Clinic Foundation. August 1998.

91. Treatments for Adult High-Grade Astrocytomas. Rhone-Pollenc Rorer Seminar. Columbus, OH September 23, 1998.

92. Mitochondrial Disease in Children. Pediatric Anesthesia Nursing In-Service Seminar. Cleveland Clinic Foundation, September 28, 1998.

93. What do Department Chairmen need to know about CPT Coding? Annual Professors of Child Neurology Meeting, Toronto, Ontario, October 21, 1998.

94. Energy Metabolism in Malignant Glioma: Possibilities for Therapy. 1st Annual Cleveland Clinic Foundation Brain Tumor Symposium. Naples, FL. February 16, 1999.

95. Chemotherapy for Plexiform Neurofibromas. 1" Annual Cleveland Clinic Foundation Brain Tumor Symposium Naples, FL. February 17, 1999.

96. Newborn Metabolic Emergencies. Combined Metro-CCF Neonatal Lecture Series. Metro Health Medical System, March 23. 1999.

97 Treatments for Adult High-Grade Astrocytomas The Role for Implantable Chemotherapy Delivery Systemis Rhone-Pollenc Rorer Seminar, Harrisburg, PA, May 5, 1999

98. Chemotherapy and Malignant Astrocytoinas: The Failure of Past Therapies and the Potential for Success with New Treatment Modalities. Sacred Heart Hospital 1st Annual Neuroscience Symposium. Allentown, PA, May 6, 1999.

99 Overview of Mitochondrial Cytopathies. Clinical, Pathologic, Biochemical, Genetic and Treatment Survey Cleveland Clinic Foundation Seminar Cleveland, OH, May 16, 1999

100. Mitochondrial Diseases: Recognition of Abnormal Mitochondrial Function. Pediatric Grand Rounds, Rainbow, Babies and Children's Hospital. June 17, 1999

101. Medical Therapeutic Strategies for Neurofibromatosis I: Cis-Rctinoic Acid, Interferon Alpha and Thaiidomide Pediatric Grand Rounds. University of Florida at Gainsville, Gainsville, FL. July 9, 1999.

102. Chemotherapy and Malignant Astrocytomas: Molecular Genetics, Therapeutic Failures and Hope with New Treatments. Danville, PA. July 27, 1999.

103. Management Strategies for Low-Grade Astrocytomas. Current Management of Neurologic Disorders. Cleveland Clinic Foundation. Cleveland Maniott Hotel. Cleveland OH. August 13, 1999.

104. Paroxysmal Non-Epileptic Disorders. Intensive Review of Pediatrics, The Cleveland Clinic Foundation. September 1, 1999.

105. The Muscular Dystrophies. Intensive Review of Pediatrics, The Cleveland Clinic Foundation. September 3, 1999.

106. The Clinical Manifestations of Neurofibromatosis I. The Brain and Learning: Educational Implications of Neurologic and Psychiatric Disorders. 4th Annual CCF Learning Assessment Clinic Lecture Series. Cleveland, OH. December 3, 1999.

107. Making Sense of Respiratory Chain Analysis. NIH Minisymposium on Mitochondrial Diseases. National Institutes of Health, March 14, 2000.

108. Disorders of Oxidative Phosphorylation and their Relationship with Epilepsy. Epilepsy and Sleep Disorders Grand Rounds. Cleveland Clinic Foundation, March 23, 2000.

109. Adult Presentations of Mitochondrial Disorders. Internal Medicine Grand Rounds, University Hospital's of Cleveland. April 18,2000.

110. Clinical Presentations of Mitochondrial Cytopathies in Children and Adults. Mitochondrial Cytopathies 2000. Cleveland, OH. June 1,2000.

111. Mitochondrial Cytopathies 2000. Pediatric Grand Rounds. Cleveland Clinic Foundation. October 17,2000.

112. Mitochondrial Cytopathies: An Overview for Occupational and Physical Therapists. Cleveland Clinic Foundation. October 12,2000.

113. Commonly Asked Questions by Parents and Caregivers. UMDF Ohio Chapter Meeting. October 21, 2000.

114. Overview of the Clinical Aspects of Mitochondrial Cytopathies. Cleveland Clinic Neuroscience Nursing Conference, Cleveland Ohio. November 16,2000.

1 15 ICD-9 and CPT Coding for Neurologists in 2001 Children's Hospital of Michigan-Wayne State University Medical Center Neurology Grand Rounds December 15, 2000

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