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IN THE COURT OF COMMON PLEAS
OF CUYAHOGA COUNTY, OHIO

KEVIN KISS, a minor, by and through his next friend and natural mother, Anne Kiss, et al.,

Plaintiffs,

vs. Case No.

ANDREAS MARCOTTY, M.D et al., 402593

Defendants.

DEPOSITION OF 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, 9500 Euclid Avenue, Cleveland, Ohio, on the day and date set forth above, at 2:10 o'clock p.m.

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APPEARANCES:

On behalf of the Plaintiffs:
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On behalf of the Defendant Andreas Marcotty, M.D.:
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by
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On behalf of the Defendant Cleveland Clinic Foundation:

Roetzel & Andress, by
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On behalf of the Defendant Signature Eye Associates:
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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 sworn, 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.
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

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patients anywhere besides the main campus of Cleveland Clinic?
A. I may have also been seeing patients 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 one afternoon every one to two months, but I can't remember when I stopped going out to Kaiser Beachwood.
Q. And who is your current employer?
A. The Cleveland Clinic Foundation.
Q. And was that also true at the time that you rendered care to Kevin Kiss?
A. Yes.
Q. Aside from the professional services that you provide for Cleveland Clinic Foundation, do you provide professional services for any other entity?
A. I do occasional consulting work with drug companies.
Q. Any particular drug company?
A. Rhone-Poulanc Rorer. I no longer do consulting work with them.
Q. So you currently are not consulting

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with them?
A. Correct.
Q. And in the time period when you rendered care to Kevin Kiss, were you providing professional services for any other entity besides Cleveland Clinic?
A. No.
Q. Have you ever had your deposition taken before?
A. Yes.
Q. How many times?
A. I don't remember.
Q. Approximately, doctor.
A. A dozen.
Q. How many of those times were in a medical negligence case?
MS. CARULAS: Note my objection. I'll have a continuing line of objection to this, but go ahead.
A. Ail but one. The other one was a rape case, and one was a murder case, all in the context of a medical basis.
Q. In those other instances that were medical negligence cases, were any of those cases ones in which you were named as a Defendant in

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the case?
A. No.
Q. Was Cleveland Clinic named as a Defendant in the case?
A. No.
Q. What was the reason that your deposition was being taken, and by that I mean was it as a medical expert, a fact witness?
A. It was a medical expert and others were -- I was the treating physician.
Q. But your care was not called into question in those?
A. No.
Q. I want to go over some of the ground rules for deposition. I'm sure counsel has had a chance to talk with you. This is a question-and-answer session. It's under oath. It's important that you understand my questions. If you don't understand them, let me know, I'll be happy to repeat the question or rephrase it. Otherwise, I'm going to assume that you understood the question and that you're able to answer it. At any point, if you would like to refer to the medical records that counsel has provided you with, feel free to do so.

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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. No.
Q. How many depositions have you given in the last year?
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?

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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. In the last year, for the Defendant.
Q. Well, in the ten or so times.
A. It's about 50/50 split.

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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. Was that for Plaintiff or for Defendant?
A. 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?

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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

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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. 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

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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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Q. In the time period of April through October of 1998, did you hold any administrative positions at the Cleveland Clinic Foundation?
A. I served on committees.
Q. Did you hold any titles other than staff physician at the Cleveland Clinic?
A. I was on the medical record committee.
Q. What about department head or anything like that?
A. No, I wasn't a department chief.

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

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Q. Doctor, counsel has provided me with a copy of your resume which we have marked as Plaintiffs' Exhibit 1, and I would ask if you would just identify Plaintiffs' Exhibit 1 for the record for us, just indicate to us what that document is.
A. This is a copy of my curriculum vitae. It was updated on December 6, 2000.
Q. Is it current and up-to-date, as far

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as you know?
A. No.
Q. Are there any additions or corrections that you would like to make to it?
A. There are no corrections or additions relevant to anything we could possibly be talking about today. I've given a couple more talks on mitchondrial disease. I've had another abstract or two accepted completely unrelated to anything that we would be talking about today.
Q. Doctor, did you do part of your training here at the Cleveland Clinic?
A. No.
Q. When did you first become affiliated with the Cleveland Clinic?
A. I joined the staff on June 1st, 1989.
Q. Have you been employed by the Cleveland Clinic since that time?
A. Yes, I have.
Q. And, doctor, your curriculum vitae has listed on it numerous publications. Do any of those publications deal with the subject matter of arachnoid cysts?
A. Not to my knowledge.
Q. Do any deal with the subject matter

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of papilledema?
A. The word papilledema may be mentioned in the body of some of those pubiications, but none of them deal with the diagnosis or treatment of papilledema.
Q. Any deal specifically with increased intracranial pressure?
A. I co-authored a book chapter.
Q. Is it listed in your curriculum vitae?
A. It should be.
Q. Would you please find it for us, and I would like you to indicate which chapter you're referring to, and please mark it with -- I don't know, are they numbered?
A. Yes.
Q. Just circle the number, please.
A. Page 12, article 16.
Q. Any of the publications that you have listed on your curriculum, do any of those deal with visual field testing?
A. Not to my knowledge.
Q. Now, you also have a number of presentations that are listed on your curriculum vitae. Do any of those deal with the subjects

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that I just mentioned: papilledema, increased intracranial pressure, arachnoid cysts or visual field testing?
A. I gave a talk in 1995, page 15, talk 33, Neurologic Causes of Headaches, in which the issue of increased intracranial pressure may have come up. But I don't recall exactly.
Q. Do you have any syllabus or handouts from that presentation?
A. I do not. That's all that I see.
Q. I'd like you to tell me what you have reviewed for this deposition.
A. I've looked over my chart notes. I've looked over some of the chart notes in the file.
Q. Now, Kevin Kiss received some care that was outside of the Cleveland Clinic from Signature Eye Associates, Kids in the Sun. I think there was an emergency room visit at Southwest Hospital. Did you review any of those documents that you recall?
A. I think I saw some document from Dr. Marcotty, but I don't recall if it was a letter or handwritten note or whatnot. I don't recall anything from Kids in the Sun.

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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 from a psychological counseling service of Kevin Kiss. Have you seen any of those records?
A. 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

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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.
Q. Do you consider the material in that book to be reliable?

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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. Any with arachnoid cysts?
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

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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?
A. It can be one of the early signs of increased intracranial pressure.

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Q. After fenestration of an arachnoid cyst, can increased intracranial pressure recur?
A. After fenestration of an arachnoid cyst, the intracranial pressure can remain as it was before the fenestration. It can decrease and come back to normal, and if scarring takes place, or the fenestration closes up again, conceivably pressure can recur.
Q. Do patients who have undergone fenestration require long-term followup with neuro-imaging evaluation because increased intracranial pressure can recur?
A. Usually not.
Q. What is papilledema?
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

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the "isn't" because that confuses me.
Q. Is 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. I examine the eye as part of the neurologic examination.
Q. What are the early signs of

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papilledema?
A. Papilledema is a sign.
Q. When you observe the inner portion of the eye, is there anything that indicates to you that the papilledema is early rather than a chronic or later form?
A. That's a difficult question to answer. Papilledema can be graded as mild, moderate or severe. But the rapidity at which papilledema forms is based on the process at hand. For example, if someone gets shot in the head with a bullet, you can get papilledema developing within minutes of that injury. In a situation of chronic increased intracranial 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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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

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an ophthalmologist for evaluation, management and followup for papilledema?
A. I've referred patients to ophthalmologists for confirmation of my diagnosis of the clinical finding of papilledema and for followup. I do not recall if I've ever asked the surgeon to manage the papilledema --
Q. Is that --
A. -- asked an ophthalmoiogist to manage the papilledema.
Q. Is that something that you as a neurologist would manage?
A. Depending on what the underlying disease was, it could be something that I would 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 determine whether a patient should undergo that

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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 concern in a patient?

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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.

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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. I want 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?

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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 -- I've 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

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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.
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. There's treatment for the causes of papilledema.
Q. What is visual field testing?
A. The visual field is a concept of the

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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

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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 due to increased intracranial pressure.
Q. And what are you looking for in a patient that has increased intracranial pressure and papilledema?
A. Well, what we look for is preserved visual field, that the patient's peripheral vision is as good as ours.
Q. And in some instances, can increased intracranial pressure result in a patient

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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 -- optic nerve atrophy would be a deleterious effect of optic nerve papilledema. So the question, with really 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 before,

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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. LOESEL: 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

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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 and lead to evaluation for optic nerve atrophy?
MS. ATWELL: Objection.
MS. LOESEL: 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

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look 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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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 him?
A. I probably had access to his medical file.
Q. And what is your understanding as to why Kevin was referred to you by Dr. Luciano?
A. My understanding is that he had problems following his initial surgery. Those problems included irritability, mood disturbance and intermittent headaches as well as hearing noises in his right ear. And I can deduce that Dr. Luciano wanted my opinion regarding what was going on.
Q. Did Kevin aiso have vision problems?
A. Based on my examination, he had papilledema at the time of that visit.
Q. In the history that was given to you, were you told that he had disturbed vision?
A. I don't see it in the notes.
Q. Doctor, I see a note dated April 14th of 98. I think it's timed at 10:30 in the morning.
A. Yes.

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Q. It says outpatient visit, Dr. Cohen. Is that a note from you?
A. Yes. The majority of handwriting in that note is from one of my residents, but that note does reflect his visit with me.
Q. And did the resident see the patient before you or with you?
A. The resident -- I don't recall in this situation. As a general rule, the resident sees the patient before me.
Q. Does the resident then provide you with the information that is obtained on his visit?
A. That is true, at which time I go back in, review the relevant history, I do my own examination, report any differences I may find from the resident's examination.
Q. Do you add or make notations in the chart if you disagree with something that the resident has done?
A. That would be correct.
Q. Now, at the beginning of this note, doesn't it state in the first line, referred for headache, disturbed vision, having noises right ear?

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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?

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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 big change. What does that refer to, 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

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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. Yes, I did.
Q. Would you tell me what those were.
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 increase in tone. And those are important

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because that would indicate that, despite the fact that I thought he had increased intracranial pressure, that I didn't think the increased intracranial pressure was so high that he was in physical danger.
Q. What physical danger are you referring to?
A. If intracranial pressure is too high, children can permanently injure their brain through a process called herniation. And the lack of those abnormal physical findings told me that I didn't have to be concerned of that problem occurring at that time.
Q. Now, you referred to visual field testing. Can you tell me where that is referenced?
A. In the resident's note, halfway down that last page, that date, it says normal, abbreviated NL, visual field.
Q. Show me which page. I'm not on the right page.
A. (Indicating.)
Q. I just want to see the top of the page.
MS. ATWELL: Under CNS.

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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 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 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 documented above, 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.

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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.

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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

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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

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well for awhile after his surgery? You mentioned that some children will have symptoms after surgery, but he had his surgery in December. The history that you have here is about a two-month history of these other symptoms, isn't it, which would be February to April?
A. Dr. Luciano's chart note dated January 22nd, 1998 indicated that his headaches started two weeks prior to that. So that would be about January 8th.
Q. So the chart note on January 22 indicates that January 8th would have been about the time his headaches started and about the time his double vision started?
A. The history I obtained on April 14th, say in two months, was again the history obtained from the parent or parents at the time of the visit. It's a history that we write down.
Q. When you saw him on the 14th, what was your plan of care for him? I think you mentioned to refer him to Dr. Luciano for surgery.
A. From what I remember, I either called Dr. Luciano or Dr. Luciano's nurse and suggested that surgery would be my choice as to what to do.

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Q. Now, doctor, under your note, you have -- is that treat -- what does the TX stand for?
A. Transfer.
Q. Transfer to Dr. Luciano for surgery.
A. Yes.
Q. Then underneath that, you have eye consult. Is that for visual fields?
A. It says eye consult visual field.
Q. Now, the type of surgery that you were referring to in your plan, was that for the shunting procedure?
A. From the best I can remember, I wasn't convinced in my own mind whether the subdural hygromas were what needed to be shunted or the arachnoid cyst. And so I had to rely on Dr. Luciano to make the correct decision.
Q. But it was to relieve the increased intracranial pressure?
A. It was to relieve the increased intracranial pressure, correct.
Q. Now, doctor, you indicated also eye consult for visual field testing; is that correct?
A. That's what I put in my chart note.

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Q. Why did you advise an eye consult for visual field testing?
A. I think as part of the longitudinal care of the chronic, long-term needs of a child with papilledema and intracranial pressure, it would be good to know what his visual field was, is and will be in the future for educational purposes, for example.
Q. At the time that you saw him on the 14th, were you of the opinion that he had chronic long-term papilledema?
A. No, I didn't have an opinion regarding that. In fact, from the notes, I really wasn't worried about his vision at that time. We had noted normal visual fields, and it was -- I would have -- again, I would have put that in there as part of the longterm management, not because I saw an acute problem that needed to be dealt with at that time.
Q. So you were looking to have an ophthalmologist manage the papilledema over the long term?
A. No. I was looking for an ophthalmologist to help us out with a detailed visual acuity examination, for example, to look

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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.

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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

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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.

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Q. Did you make any such arrangements for Kevin to have it done as an outpatient after he was home?
A. I do not remember.
Q. Did you come across anything in the chart that told you you did that?
A. I do not remember. I don't know if I did it or if someone else did it.
Q. Did you inform Dr. Luciano that Kevin should receive an eye consultation for visual field testing?
A. I do not remember.
Q. Do you know if Kevin ever received an eye consult as a result of your recommendation?
A. I know he was seen by an ophthalmologist. I don't know if it was a result of my recommendation and Dr. Luciano's recommendation or some other person's recommendation.
Q. When was he seen by the ophthalmologist, that you're aware of?
A. I had seen Kevin on June 9th, 1998, and my history obtained at that time was that after his shunt was placed, meaning the surgical procedure done on April 15th, 1998, he was 90

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percent back to normal in four days, and in two weeks was a hundred percent. He was back at full activities, and he was making straight As in school.
On my examination, I noted mild optic atrophy and no papilledema. I sent him to see Dr. Marcotty for eye examination. I had sent him to Dr. Marcotty because he had been seen by Dr. Marcotty in the past. That was the child's eye doctor.
Q. But between the time that you saw him on April 14th of 98 and the time that you saw him on June 9th, 98, did he see anyone for visual field testing that you are aware of?
A. Not that I'm aware of.
Q. Had you anticipated that he would see someone after you saw him on the 14th and before you saw him again on the 9th of June?
A. I had seen him in the hospital, followed him in the hospital, and he had done very well in the hospital. So I can't tell you if I told the family no need to see the eye doctor or not. It's a moot point as to whether or not he saw an eye doctor or not, because whatever process occurred leading to optic

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atrophy had already occurred.
Q. Had already occurred when you saw him on June 9th of 98?
A. Essentially had already occurred by June 9th, 98. Probably it occurred long before I ever met the child.
Q. Do you know when it occurred?
A. No, I do not.
Q. Who accompanied Kevin when you saw him on April 14th of 98 to the visit?
A. Could I have the last couple questions and answers read?
Q. Yes.
(Record read.)
A. My answer to your question was not entirely complete and doesn't reflect accurately my feelings about this. Do you mind if we just take a break so I can get some water?
Q. Sure. Would you like to correct your answer? I mean, if you would like to go and correct your answer, you can go ahead and correct your answer if you're uncomfortable as to what it is you said.
MS. CARULAS: Or do it when you come back if you want to take a break.

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A. Yes. It's difficult for me to answer your question in the context of everything surrounding that.
Q. Which question are we referring to?
A. The question as to when -- the question that implies that the visual loss may have taken place at some point in time, and I think that's the crux to what we're all here talking about.
And so I have an opinion regarding that, and the opinion stated as, quote, I don't know or I'm not sure, unquote, doesn't reflect accurately what my feelings about that are.
Q. Would you like to correct it?
A. I would like to take a break and correct it.
Q. All right.
A. Thankyou.
(Recess had.)
Q. Doctor, if you would like to add to or correct whatever answer that you gave previously to my question in regard to, I believe it was, if you had an opinion as to when Kevin developed his vision loss, please feel free to answer that question or add to your answer

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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, 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

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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 didn'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

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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

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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. 14th.
Q. 14th of 98. Is that correct?
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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your question. I did not document a visual field check at that time.
Q. Now, when Kevin was admitted to the hospital, you saw him during that admission; correct?
A. Yes, I did.
Q. We just looked at some of those notes. And one of the notes that you wrote was on April 17th of 1998; correct?
A. That's correct.
Q. Now, I believe Kevin was actually admitted on the 14th. Did you see him any time between the 14th and the 17th? Your note is written on the 17th, and that's the first one that I see.
A. Yes, I saw him on the 16th.
Q. Okay.
A. There's a three-line note about two-thirds of the way down the page.
Q. Okay. Yes, I see that. So you saw him on the 16th?
A. Correct.
Q. And this was after his surgery; correct?
A. It was after his surgery.

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Q. Did you check him for papilledema at that point in time when you saw him on the 16th?
A. I did not.
Q. When you saw him on the 17th, did you check him for papilledema?
A. I did,
Q. Now, would you have expected that his papilledema would have been relieved by the shunting procedure?
A. No. It can take many months for papilledema to disappear.
Q. Did you have any concerns that the papilledema would cause some type of visual problems because it was persistent?
A. The answer to that question is no. The papilledema was going to resolve because his pressure had been adequately relieved.
Q. Did he still have increased intracranial pressure when you saw him on the 17th?
A. My answer to your question would be no, he did not have increased intracranial pressure. His intracranial pressure was gone. Papilledema can take many, many months to form. And it can take many, many months to resolve.

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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.

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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 I would just like him to clarify that.
MS. ATWELL: 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 shunting procedure done, there was no treatment

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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.
We have two processes here. We have

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the process of the arachnoid cyst and the increased intracranial pressure, and we have the separate process, albeit related, to the fact that this optic nerve underwent slow compression over the course of many, many months, followed by a procedure that relieved that pressure.
It will never be clear as to what point this child's vision was destined to be lost. It could have been due to the chronic effect of papilledema that began many months before the discovery of his arachnoid cyst, and the pressure -- had the pressure been adequately relieved by the fenestration back in 1997, he could have lost vision subsequent to that. In fact, the pressure was relieved in April of 98, and he lost vision subsequent to that point.
Q. Do you have an opinion as to whether his shunting procedure was done in a timely manner to relieve his increased intracranial pressure?
A. Yes, I do.
Q. What is your opinion?
A. It was done in a timely manner.
Q. When you saw him on the 17th, he still had the papilledema; correct?

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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 degree 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

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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 tumor on their optic nerve and involvement 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.

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Q. Now, Kevin was discharged, I believe, on the 18th with papilledema.
A. That's correct.
Q. Should he have received evaluation and followup by an ophthalmologist after discharge?
MS. ATWELL: Objection.
MS. LOESEL: Objection.
MS. CARULAS: I'm going to object because we have been over it and over it. But go ahead.
A. Well, he did. Do you mean immediately after discharge?
Q. Yes.
A. No.
Q. At what point should he have received ophthalmologic evaluation after discharge? How long a time period?
A. At some point in the future, when it was -- when he had recovered from the steroid taper and after he was followed up by Dr. Luciano and myself.
Q. Can you give me some type of a time range for that, when you think that ophthalmology followup should have been done after discharge?

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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.

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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 98?
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

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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.

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Marcotty?
A. I put down return visit PRN, which is a way of saying as needed. So I didn't establish any particular time that I wanted to see him back. What I really wanted is for Marcotty to take a look at his eyes and tell me what was going on.
Q. How soon did you want him to see Dr. Marcotty? Did you indicate that to Kevin's parents?
A. I didn't indicate it in the chart, no. Under the situation, it would not have been an emergency, but it would have been a statement like I would like you to see Dr. Marcotty as soon as can be conveniently arranged.
Q. When you saw Kevin on June 9th, did he voice any concerns about his vision?
A. No.
Q. Did you find that odd for a child of his age not to be able to tell you that there was vision problems there?
A. I would not find that odd.
Q. Now, did you speak with Mr. and/or Mrs. Kiss on the 9th and tell them of your findings?

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A. I put down here that I discussed the care plan management, prognosis for 20 minutes. So it's my assumption that I spent a reasonable amount of time discussing with them my findings.
Q. Was it your impression when you saw him on the 9th that Kevin had some major vision loss?
A. I did not know that. If I did know that, I didn't document it.
Q. Did you indicate to Kevin's parents at all that there was some vision loss?
A. I don't have a recollection of that, but from the context of what's in the note, I probably would have expressed some concern that there could be vision **loss.**
Q. And when you saw him on June 9th, were there any signs of increased intracranial pressure at that time?
A. None.
Q. Now, you saw Kevin again after this June 9th, 98 visit; is that correct, I believe in October?
A. I know I saw him again after that. Let me refer to my note. Yes. October 15th, 1998.

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Q. And what was the reason that Kevin came to see you on that date?
A. My impression was that it was part of a followup visit, and I remember the parents had a lot of questions regarding the visual loss as a result of everything that had gone on.
Q. Did you do a physical exam of Kevin on that date?
A. It looks like my physical exam was limited to looking at his eye, seeing optic atrophy left eye greater than right, and a partial Marcus Cunn pupil on the left.
Q. What is a Marcus Cunn pupil?
A. That indicates that there is an injury to the left optic nerve.
Q. Did you have any evaluation by Dr. Marcotty at the time that you saw him on October 15th of 98 or by any other ophthalmologist from the Cleveland Clinic, any other input?
A. Looks like -- yes, it looks like Dr. Kosmorsky from the Cleveland Clinic saw Kevin and had felt that his vision in his good eye was 20/20, and his vision in his left eye was that he could detect hand motion, but nothing more than that.

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Q. Did you have any conversations with Dr. Kosmorsky about Kevin's vision?
A. Not that I recall.
Q. Any with Dr. Marcotty?
A. I recall a phone call from Dr. Marcotty. I don't recall what date the phone call was, but my assumption is that it was after he had seen Kevin back in June.
Q. Did you have any discussions with Dr. Luciano about Kevin's vision loss?
A. Not that I recall.
Q. You did not talk with him after you saw Kevin on June 9th of 98 to tell Dr. Luciano that you had found some optic atrophy?
A. I can't imagine that I didn't discuss this with him, but I honestly do not recall any discussion regarding that finding. In other words, it would be my practice to call him up and say this is what I found, but I don't recall having that discussion, nor any subsequent discussions.
Q. And, doctor, it was your opinion that Kevin's vision loss was secondary to his increased intracranial pressure and papilledema; correct?

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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

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wrong happen to my child, was there a medical error.
The discussion we had was almost exactly the content of the discussion we're having right now; a review of everything that had happened to him up to this point, a review of all the possibilities, a review of the fact that he may have been destined to lose his vision prior to medical involvement in this case in the first place because relief of pressure can be damaging to the optic nerve, that he may have been destined to lose his vision at some point in time prior to the ultimate shunting procedure done on April 15th, and that, in my opinion, there was very little, if anything, anyone could do to have protected him from this rare, but obviously very terrible, situation that he lost vision in one eye; that the timing of the second surgical procedure was appropriate because we all know that placing a shunt is a procedure that has risks, including the risk of the shunt not working, the risk of anesthesia, the risk of shunt infections, the risk of something else going wrong like bleeding, and that a careful surgeon would have done an alternative treatment

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such as putting him on Diamox to try to reduce the intracranial pressure.
Q. Doctor, do you have an opinion, if his shunting procedure had been done sooner, whether it would have made a difference in regard to his vision loss?
MS. CARULAS: Objection.
A. I don't have an opinion because I don't think we can know whether or not the visual loss is due to the long-standing papilledema or to the relief of that papilledema or, more likely, a combination of both.
Q. When you had the conversation with Kevin's parents on October 15th of 98, was anybody else present for that conversation besides you and Kevin's parents?
A. I don't recall. My resident had seen the patient prior to me seeing the patient, but I don't recall if the resident was in the room at the time, nor do I recall if Kevin's parents had any other adult member of the family there, nor do I recall if Kevin was even in the room at the time.
Q. At any point that you discussed Kevin's case with Dr. Luciano, did you ever

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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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have some.
MS. ATWELL: I have just a few.
EXAMINATION OF BRUCE H. COHEN, M.D.
BY MS. ATWELL:
Q. I'm the attorney for Dr. Marcotty; my name is Cheryl Atwell.
Who was the resident who was with you on April 14, *19981*
A. The resident whose name I cannot read could be identified through the Cleveland Clinic Foundation medical records office. There's ways to identify that resident, because all residents have their signature on file. I don't recall who that resident was, nor can I read that signature.
Q. Looking at the resident's note on April 14, if you don't mind if I come and stand by you, there's just a couple words I want you to try to read. I don't want you to try to read the whole thing because we can read most of it. On April 14, the resident, on the first page, midway down, circled the number one, and then he wrote a word.
A. It looks like panic attack past six weeks.
Q. Got it. On the very bottom part of

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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. CARULAS: So we won't waive.
Send it to me and 1'11 send it on to him.
MS. TOSTI: Thank you, doctor.
(Deposition concluded at 4:15 o'clock p.m.)
(Signature not waived.)

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AFFIDAVIT

I have read the Foregoing transcript from page 1 through 82 and note the following corrections:

PAGE	LINE	REQUESTED CHANGE

BRUCE H. COHEN, **M.D.**

Subscribed and sworn to before me this _____ day of _____, 2000.

_____
Notary Public
My commission expires _____.

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CERTIFICATE

State of Ohio,)
) SS:
County of Cuyahoga.)

I, Karen M. Patterson, a Notary Public within and for the State of Ohio, duly commissioned and qualified, do hereby certify that the within named BRUCE H. COHEN, M.D. was by me Rrst duly sworn to testify to the truth, the whole truth and nothing but the truth in the cause aforesaid; that the testimony as above set forth was by me reduced to stenotypy, afterwards transcribed, and that the foregoing is a true and correct transcription of the testimony.

I do further certify that this deposition was taken at the time and place specified and was completed without adjournment; that I am not a relative or attorney for either party or otherwise interested in the event of this action.

IN WITNESS WHEREOF, I have hereunto set my hand and affixed my seal of office at Cleveland, Ohio, on this 5th day of March 2000.

Karen M. Patterson, Notary Public
Within and for the State of Ohio

My commission expires October 7, 2004.

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WORD INDEX

CURRICULUM VITAE

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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
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Pediatric Neurology Fellowship:
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Pediatnc Residency
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June 17,1982-June 30, 1984

Medical School:
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Degree: M.D. June 1982

College:

Washington University
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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 Ncuro-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 cranio 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

1Y99- 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

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

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3. Roger J. Packer, Larissa T. Bilaniuk, **Bruce** H. **Cohen,** Bruce H. Braffman, Angela C. Obringer, Robert A. Zimmerman, Kathy R. Siegel, Leslie N. Sutton, Peter J. Savino, Elaine H. Zachai, Anna T. Meadows. Intracranial visual pathway gliomas in children with neurofibromatosis. Neurofibromatosis 1988;1:212-222.

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5. Leslie N. Sutton, Robert E. Lensinski, **Bruce** H. **Cohen,** Robert A. Zimmerman: Localized "Phosphorous magnetic resonance spectroscopy in large pediatric brain tumors. J Neurosurg 1990;72:65-70.

6. Roger J. Packer, Jeffrey C. Allen, Joel L. Goldwein, Joseph Newall, Robert A. Zimmerman, John Priest, Tadanori Tomita, David E, Mandelbaum, **Bruce H. Cohen,** Jonathan L. Finlay, Leslie N. Sutton, Giulio D'Angio: Hyperfractionated radiotherapy for children with brainstem gliomas: A pilot study using 7,200 cGy. Ann Neurol 1990;27:167-173.

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11. **Bruce H. Cohen,** Allen M. Kaplan, Roger J. Packer: Management of Intracranial Neoplasms in Children with Neurofibromatosis Type 1 and 2. Pediatr Neurosurg 1990-91;16:66-72.

12. **Bruce H. Cohen,** Patrick Zweidler, Joel Goidwein, Roger J. Packer: Ototoxicity of cis-platinum in children with brain tumors receiving cranial irradiatioii. Pediatr Neurosurg 1990-91;16:292-296.

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14. John C. Egelhoff, Douglas J. Bates, Jeffrey S. Ross, A. David Rothner, **Bruce** H. **Cohen:** Spinal MR findings in neurofibromatosis types 1 and 2. AJNR 1992;13:1071-1077.

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16. **Bruce** H. **Cohen.** Metabolic and degenerative diseases associated with epilepsy. Epilepsia 1993;34:S62-S70.

17. Roger J. Packer, Leslie N Sutton, Roy Elterman, Beverly Lange, Joel Goldwein, Stacy Nicholson, Lynn Mulney, James Boyett, Guilo D'Angio, Kaethe Wechsier-Jentzsch, Gregory Reaman, **Bruce H. Cohen,** Derek A. Bruce, Lucy B. Rorke, Patricia Molloy, Janice Ryan, Debra LaFond, Audrey E. Evans, Luis Schut. Outcome of children with medulloblastoma treated with radiation and cisplatinum, CCNU, and vincristine chemotherapy. J Neurosurg 81:690-698, 1994.

18. Ellis BD, Kosmorsky GS, **Cohen BH.** Medical and surgical management of acute disseminated encephalomyelitis. J Neuroophthalmol 14:2 10-213, 1994.

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21. Prakash Kotagal, **Bruce H. Cohen,** Joseph F. Hahn. Infantile Spasms in a child with brain tumor: seizure-free outcome after resection. J Epilepsy 8:57-60, 1995.

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31. John H. Suh, Gene H. Barnett, Jason W. Sohn, Patrick A. Kupelian, **Bruce H. Cohen.** Linear accelerattor-based stereotactic radiosurgery for recurrent and newly diagnosed acoustic neuromas. Submitted for publication.

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35. Packer RJ, Goldwein J, Nicholson HS, Vezina LG, Allen JC, Ris MD, Muraszko K, Rorke LB, Wara WM, **Cohen** BH, Boyett JM. Treatment of children with medulloblastomas with reduced-dose craniospinal radiation therapy and adjuvant chemotherapy: A Children's Cancer Group Study. Journal of Clinical Oncology. 17(7):2127-36, 1999 Jul.

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38 Roger J Packer, **Bruce H. Cohen,** Kathleen Coney. Intercraniai germ cell tumors. The Oncologist 5:312-320, 2000.

39 Feliz Rosenow, Prakash Kotagal, **Bruce H. Cohen,** Carolyn Green, Elaine Wyllie Multiple sleep latency test and polysomnography in diagnosing Kleine-Levin syndrome and periodic hypeisoinnia J Clin Neurophys 17 5 19-522,2000

40. Sarah Rollins, Richard A. Prayson, James T. McMahon, **Bruce H. Cohen.** Diagnostic yield of muscle biopsy in patients with clinical evidence of mitochondrial cytopathy. Am J. Clin Pathol, submitted for publication.

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2. **B.H. Cohen,** D.C. DeVivo, J. Garvin, Handler MS, Hays AP, Carmel P: Central nervous system melanotic neuroectodermal tumor of infancy (CNS-MNTI): Value of chemotherapy in management. Neurology 1987;37(Supp. 1):302.

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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 Apnl 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 I 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 Hamot 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 Leaniing 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 Hosptial 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 Actualizacion 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 Hosptials of Cleveland, Department of Neurology Grand Rounds. March 6, 1998.

89. 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: Possibiiities 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 Systenis 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. Cleveiand 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

12/06/2000