THE STATE OF OHIO,)
) ss:
COUNTY OF CUYAHOGA.)

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IN THE COURT OF COMMON PLEAS

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MARGARET PEACOCK, Administratrix of the Estate of LARRY PEACOCK, Deceased,

Plaintiff,

vs.

UNIVERSITY HOSPITALS OF CLEVELAND, et al.,

Defendants.

Case No. 297578

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Deposition of JAMES MICHAEL KOCH

Wednesday, April 30, 1997

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The deposition of JAMES MICHAEL KOCH, called for cross-examination by the Plaintiff under the Ohio Rules or Civil Procedure, taken before me, Priscilla A. Hefner, Notary Public in and for the state of Ohio, pursuant to agreement, at the offices of Jacobson, Maynard, Tuschman & Kalur, 1001 Lakeside Avenue, suite 1600, Cleveland, Ohio 44114, commencing at 12:15 p.m., the day and date above set forth.

APPEARANCES:

On behalf of the Plaintiff:

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JAMES MICHAEL KOCH,
being first duly sworn, was examined and testified as follows:

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DIRECT EXAMINATION

BY MS. SPERANDO:

Q. Doctor, could you state your full name for the record, please.

A. James Michael Koch.

Q. What is your profession?

A. I am acardiologist.

Q. Doctor, some preliminaries before we start. My name is Maria Sperando. This is John Martin. We represent the plaintiff in this case.

We will be asking you some questions. If at any time you don't understand a question that I ask, can we have an agreement that you will tell me, and this way I can rephrase it in a way that you will understand it?

A. Okay.

Q. If at any time you need to refer to any of the records, I would ask that you do that, rather than guess; is that fair?

A. Okay.

Q. And you need to verbalize your answers,

because it is hard for her to take down "uh-huh" "uh-uh" or a nod or shake of the head.

A. I will do my best.

Q. All right. Number one, sir, have you had an opportunity to read Doctor Herskowitz's report?

A. Yes, I have.

Q. I have a copy of your report. And I would like to know as a result of having read Doctor Herskowitz's report whether you would like to make any changes, amendments, additions to your report.

MR. HUPP: objection. I think it is overbroad. But, go ahead.

THE WITNESS: I hadn't considered that. I did not review my report in light of his report.

BY MS. SPERANDO:

Q. Having read his report, do you have any additional opinions or changes in your own opinions as a result of having read his report?

A. No. I don't think so.

Q. Tell me very briefly what precisely you have read in this case in order to have formed an opinion.

A. Let's see. I read Doctor Herskowitz's report. And I read the letters from Doctor Wilber,

Page 5

Doctor Warshall, I think. I read the depositions of Doctor Biblo, Doctor Boulware. I read the medical record; that is, the in-patient admission from 5/8, I think it was, and also the emergency room records from both the admission and the subsequent admission to Meridia. And I read the autopsy.
Q. Anything else, sir?
A. I think that's all.
Q. Is it fair to say then, sir, that you have not read Doctor Boulware's records?
A. Oh, I am sorry. I read his office records. Yes.
Q. Do you have a CV? Have we been provided With a CV?
MR. HUPP: Yes, we do. Off therecord.
---oOo---
Thereupon, a discussion was had off the record.
---oOo---
BY MS. SPERANDO:
Q. In your report dated December 20,1996, you say on the second page in the fourth full paragraph that Mr. Peacock was subsequently seen in follow-up office visits with Doctor Boulware. And then you

Page 6

say, well, "I don't have any records of these visits"; is that right?
A. At the time I wrote this, that's correct.
Q. So, is it fair to say, then, at the time you formed an opinion in this case you had not reviewed Doctor Boulware's office Visits; is that right?
A. That's fair.
Q. I take it you subsequently have reviewed them.
A. Yes. I can't tell you a date exactly, only that it was subsequent to December 20. It was before I read Doctor Herskowitz's report, so it would be between December 20 and February 13.
Q. You have not reviewed the slides in this case, have you?
A. No.
Q. And you have not reviewed the cath itself or any of the other tests that were taken, just the reports?
A. Correct. I didn't see the hard copy, just the reports.
Q. Have you referred to any sources other than those that we have discussed, such as articles, textbooks, treatises, in support of your opinion here today?

Page 7

A. Not specifically; in other words, I didn't look up something to make this opinion. In fact, at the time I wrote this, I don't think I looked at anything. I do a fair bit of reading on all this sort of thing. So, I am well read in that area. But, I can't tell you that I looked specifically for this case.
Q. Do you have an area of specialty in cardiology, doctor?
A. I am an interventional cardiologist. But, I am also the director of cardiac rehabilitation at Saint Vincent Charity Hospital. And I am also the head of the medical quality assurance committee. So, those are the things that I have special interest in now. But, interventional cardiology would be my - technically, my subspecialty.
Q. I take it you are board certified in cardiology; is that right?
A. Yes.
Q. When did you become board certified?
A. 1991.
Q. Did you pass the exam on the first try?
A. Yes, I did.
Q. So, you are an interventional cardiologist. Howdoyoudefine that?

Page 8

A. An interventional cardiologist is a cardiologist who does general clinical cardiology, but also has the training and the skills and the expertise, I should say, to intervene; that is, to mechanically fix or treat coronary *artery* disease. That means interventions including placing stents or inflating balloons in the coronary arteries, that sort of thing, to relieve coronary artery disease.
Q. So, what is the type of cardiologist that you are not - if you understand what I am saying -- interventionist versus what?
A. Versus noninvasive, maybe. Probably the biggest distinction in cardiology is invasive versus noninvasive. Noninvasive cardiologists simply don't do anything that is invasive, nothing like cardiac cath or electrophysiology or anything else that would imply that you place some device in the body.
On the other hand, I do noninvasive cardiology, also. I read echocardiograms. I do stress testing. I treat hypertension and other problems like that
Q. What are the invasive techniques which you do?
A. Cardiocatheterization, intervascular ultrasound, percutaneous transluminal coronar angioplasty -- PTCA -- placement of stents,

Page 9

directional atherectomy. I also do transesophageal echocardiography, which is a semi-invasive thing. But, also, noninvasives do that.
Q. When did you graduate from medical school?
A. 1985.
Q. From which medical school?
A. University of Cincinnati.
Q. Where did you do your residency?
A. At the Cleveland Clinic Foundation here.
Q. Do you have any advanced training after the residency?
A. Yes. In 1988, I did a four-year fellowship at Deaconess Hospital, Harvard Medical School in Boston.
Q. And you finished that program in 19 –
A. 1992. And then I was a clinical instructor of medicine at Harvard Medical School before coming to Cleveland to be in private practice.
Q. And how long have you been in private practice?
A. It will be five years in August of this year.
Q. Have you ever been sued?
MR. HUFP: objection.
THE WITNESS: Ever been named in a suit? I have been named in a suit., but

Page 10

dropped.
BY MS. SPERANDO:
Q. And how many times have you been named in a suit?
A. Once.
Q. What was that about?
A. It was a gentleman who came in with an emergency, an acute myocardial infarction. We treated him aggressively, attempted to open up his right coronary artery. That failed. And he ended up going to bypass surgery, did quite well -- congratulated us on the 6:00 news – but, then subsequently had some problems with his right lower extremity. It's kind of a long story.
He had a balloon pump in the right leg that caused some ischemia in the right leg. His right leg didn't heal properly. And he sued us about a year later.
Q. What were the allegations of negligence made against you?
A. I can't tell you specifically, since I was never – I received a copy of the lawsuit. Actually, I never actually technically received *the* copy. I never even got it by certified mail. I was told about it. I never met with anybody. I was

Page 11

never deposed. I was never asked for anything. And I was simply dropped. So, I am not one hundred percent certain why.
Q. Have any claims been made against you or have you received any letters with regard to dissatisfied patients?
A. No.
MR. HUPP: objection.
BY MS. SPERANDO:
Q. Is this your first deposition?
A. No. This is my second deposition.
Q. What was the first deposition involving?
A. The first deposition was involving the case of a patient who presented to an emergency room with chest pain.
Q. Were you acting as an expert in that?
A. Yes.
Q. Whom were you testifying on behalf of?
A. On behalf of the defendant.
Q. Who was *the* lawyer in that case?
A. The lawyer for?
Q. The defendant.
A. The defendant? I guess it was Steve.
MR. HUPP: Yes.
THE WITNESS: Steven Hupp, for

Page 12

therecord.
BY MS. SPERANDO:
Q. Do you have a copy of that deposition transcript?
A. I don't have it with me. I could probably produce one.
Q. Okay. Did you testify at trial?
A. No.
Q. Do you know how Mr. Hupp came to know of you?
A. How did I get to –
MR. HUPP: If you don't know, whatever.
THE WITNESS: I don't remember, to be honest with you. I don't remember.
BY MS. SPERANDO:
Q. Have you been retained by any other attorneys other than Mr. Hupp?
A. Asked for expert testimony, you mean?
Q. In a medical malpractice case.
A. Yes.
Q. In how many other cases have you been retained, as opposed to giving testimony?
A. Two others besides the two that we mentioned, so this and the prior deposition plus two others.
Q. Were you retained by the defense attorney or

Page 13

the plaintiff's attorney in those cases?
A. Defense attorneys, both.
Q. And who were the defense attorneys?
A. One was John Jackson. And the other is Susan Massey, M-a-s-s-e-y.
Q. Does either of those attorneys work for this firm?
A. John Jackson does. I don't know who Susan Massey works for.
Q. Was your opinion With regard to Mr. Jackson's case that the defendant doctor did not fall below the standard of care?
A. Yes.
Q. And with regard to Ms. Massey's case, did you advise her that you believe that the defendant doctor did not fall below the standard of care?
A. There was not a doctor that was being sued in this case.
Q. Who was it?
A. It was nursing personnel.
Q. And did you advise Ms. Massey that the nursing personnel did not fall below the standard of care?
A. Correct.
Q. Have you ever been retained by a plaintiff's attorney at all?

Page 14

A. No.
Q. Have you ever been retained by a defense attorney where you have advised the defense attorney that his or her client did, in fact, fall below the standard of care?
A. No; not retained. No. I take that back. I actually did -- I take that back -- in 1993. And that would be another case. And I can't even remember who the lawyer was, now that I'm thinking about it. So, that would be a fifth time that I have actually reviewed charts for somebody.
I had a brief review. And I don't even recall who it was for. But, it was somebody who -- I advised them that it would be a good idea to settle
Q. And had you been retained by the defense attorney?
A. Yes.
Q. Do you have those records in your office or somewhere that they are accessible to you so that you could tell me the name --
A. I probably do, probably. Probably, the chief of medicine had asked me to look at it for somebody. And I suspect the files are at least in his office.
Q. So, doctor, I would ask that you do that and

Page 15

relay that information to your attorney.
A. If I can find it, I will.
Q. Are there any cardiology textbooks which you find to be authoritative?
MR. HUPP: objection.
THE WITNESS: Well, I certainly read cardiology texts. And I think that the information is reliable. But, they are all equally reliable, I should say.
BY MS. S P E W :
Q. Which are the cardiology texts on which you rely?
A. I own a Braunwald's, a Hurst's, a text by Kim Eagle. I own a couple of texts that I authored chapters in -- Bernard Gersh on Acute Myocardial Infarction. I own some cardiology texts on laser cardiology. I mean, it is almost too numerous to count. 1have a fairly big library of cardiology texts.
Q. That brings me to my next question, doctor. The publications -- your publications -- do any of them involve any of *the* topics we are going to be discussing today with regard to your opinion?
A. In a peripheral sense they do. I authored a chapter or was second author of a chapter on

Page 16

triggers of myocardial infarction in Gersh's Text on Acute Myocardial Infarction.
Q. How do you spell that last name, please?
A. G-e-r-s-h, Bernard Gersh.
Q. And the name of the text is Acute Myocardial Infarction?
A. Yes.
Q. Anything else?
A. Not that I can really recall that has much to do with this.
Q. The one case in 1993 where you gave an opinion that the defendant doctor had fallen below the standard of care, do you remember the facts of that case?
A. I don't really. I can only recall that it was a vascular surgery patient, I think. And to be honest With you, I am not even *sure* -- to be honest with you, I would have to look back to even tell you that I even billed anybody for that. But, I know I reviewed the case. And I would have to go look at the whole record. It wasn't something I really did very commonly. So, I would have to look back before I could give you a right answer.
Q. In a patient who is experiencing sudden death, can you tell me what is the significance of

trembling? And I will modify that. In a patient who is experiencing sudden death as a result of a cardiac problem, what is the significance of trembling, if any?
A. First, are you using sudden death as a definition -- sudden death?
Q. Yes.
A. I wouldn't necessarily think there is any specific significance to trembling.
Q. Having read Doctor Herskowitz's report, and confining this question only to what he concluded with regard to the findings he describes, did you disagree with any of the findings enumerated by Doctor Herskowitz?
MR. HUPP: what portion?
BY MS. SPERANDO:
Q. I believe before he gets to his conclusions.
MR. HUPP: The medical and autopsy findings section?
MS. SPERANDO: *Yes;* the medical and autopsy findings, right, before he gets to any -
MR. HUPP: I am going to object just for the record, because I think - it is four and a half pages. But, go ahead.
THE WITNESS: I was going to say,

he has a lot of - his whole report is basically findings. I have not reviewed specifically the slides of which he makes a big deal here. As far as everything else, I think without going through sentence by sentence there was generally statements taken from the records. So, as far as the statements were *taken* from the records, they appeared to reflect what was in the records.
BY MS. SPERANDO:
Q. So, nothing stood out in your mind as not being in conformity with what you understood the facts to be as you read the report?
MS. CARULAS: Just note my objection. I think that is difficult for him to do --
MS. SPERANDO: If he read paragraph three and said, "Gee, I don't remember seeing that or I don't agree with that" --
THE WITNESS: I can't honestly answer that for every statement in here, because I would have to look through them each individually. In general, what I would say is that I thought he accurately reflected what happened.

BY MS. SPERANDO:
Q. Do you have any expertise in reading slides?
A. No.
Q. So, would it be fair, then, to say that as to Doctor Herskowitz's conclusions in paragraph four with regard to what the slides show, that you would not be able to comment on those conclusions?
A. I can't comment on them from the standpoint of looking at the slides and telling you that I agree or disagree with what the slides show. Some of the implications of the slides I think I could comment on in general, although -
MS. SPERANDO: Excuse me. We are going to have to take a break here. *Sorry.*
---o0o---
Thereupon, the previous answer was read back by the court reporter.
---o0o---
BY MS. SPERANDO:
Q. Okay. I am not talking about the implications. I am talking about specifically what he says the slides show. You cannot comment on that?
A. Can't comment on that.
Q. Doctor, with regard to your CV, which I have

just been handed, I note that you were an instructor in medicine at Harvard. Did you ever teach cardiology?
A. Yes. To be an instructor of medicine meant to be in that department in which you were employed. And I was employed in the division of cardiology.
Q. Whom did you teach?
A. Fellows; cardiac fellows. I also rounded with residents -- you know, teaching rounds in the coronary care unit. Predominantly, fellows, though -- fellows in cardiology.
Q. Were you yourself a fellow in cardiology as you were teaching other fellows?
A. Yes. It starts there. In 1990, you were required to do a two-year cardiology fellowship. You will note that I did a four-year cardiology fellowship, because I did years of intervention and then subsequently intervention slash research. As a senior fellow in your third and fourth year, one of the most dominant parts of that load is teaching. So, you teach first- and second-year fellows.
Q. And since you did your fellowship, have you done any teaching in an academic setting?
A. Yes. I am currently a teaching physician at Saint Vincent Charity Hospital. We have a clinical

Page 21

rotation in what we call the cardiac medical unit with second-year residents and senior residents from Cleveland Clinic.

Q. At this time, as a member of the clinical department review committee of the emergency room at Saint Vincent's, what does that consist of?

A. Each of those clinical department review committees – CDRC committees -- are quality assurance committees. And I am the chairman of the medical committee. And basically, we review any quality problems or quality markers.

We have certain statistical criteria that we look for. JCAH, the Joint Commission on American Hospital Accreditation, requires you to have these type of committees in the hospital to review any number of clinical criteria to make sure that everything is basically reviewed and being done properly.

Q. And as the medical director of cardiac rehabilitation, what are your duties and functions regarding that position?

A. Basically, I am ultimately the person who is responsible for anybody that exercises in the cardiac rehabilitation program. Although I may not be their attending physician while they are there, I

Page 22

am the last line of responsibility to make sure that they are exercising safely and properly.

Q. And as to, "the last line of responsibility," if you can be more definitive for me, what does that mean?

A. In other words, cardiac rehabilitation is ordered as a prescription by an attending physician. Therefore, that attending physician is technically responsible to clear a patient, to bring them in, to give the proper diagnosis, before the patient is brought to cardiac rehab.

If a patient is in cardiac rehabilitation, though, and experiences any sort of trouble or there are questions as to what would be the right thing for them to be doing, and the attending physician is not either available or hasn't given us adequate documentation, then it would be up to me to make sure that I review that patient's case and ascertain whether exercise is appropriate and, generally, whether the program is appropriate for that patient.

Q. But, you do not make the initial decision as to whether the patient will be in the program or what type of exercise the patient will be allowed to do?

A. I do make the initial decision in the exercise

Page 23

prescription for any of my patients who are enrolled in the program. And currently, I am one of the leading prescribers to this program. There are programs all over the city. But, at this point in time, I do have a large number of patients in cardiac rehabilitation for whom I have specifically written the exercise prescription.

Q. And, with regard to the other physicians, you do not make decisions for their patients?

A. In general, I don't.

Q. Let's focus on this cardiac rehab program at Saint Vincent's. What is the purpose of it? Why would you send a of to them?

A Cardiac rehabilitation as it is defined in this program is an exercise program which is specifically designed for patients who have coronary artery disease. The primary gc l f the ı i secondary prevention for coronary artery disease.

The patients who are enrolled under my egis (phonetic) are those patients who have been shown to have coronary artery disease, and generally, if you look at guidelines and so on and so forth, fit specific diagnoses reflecting coronary artery disease. Therefore, you have a population of patients who have undergone evaluation, some of whom

Page 24

have undergone therapies and who are currently undergoing therapies, who are now enrolled in cardiac rehabilitation as secondary prevention to make sure that coronary artery disease does not clinically – I should say to reduce the risk that coronary artery disease has for them down the road.

Q. And how does this cardiac rehabilitation program do that?

A. Well, there are three phases. Phase one is what is known as in-hospital rehabilitation. It includes exercise guidelines. It includes education. It includes a certain amount of even dietary and other recommendations. But, it is basically done in the hospital.

Phase two cardiac rehabilitation is really the program that we are involved in – that I am involved in as a medical director. That is that once a patient leaves the hospital, they are given an exercise prescription, which is designed to help reduce the risk of further clinical problems due to coronary artery disease.

There is also phase three of the program, which is a less well monitored program.

Phase two is a program in which we monitor cardiac rhythms during exercise, in which we check

Page 25

blood pressure before, during, and after exercise. In other words, there is a fairly heavy clinical involvement of special nurses in the program to make sure the patients are monitored.

Phase three is an unmonitored program.

Q. What is phase three about, then?

A. Phase three is simply an exercise program without the monitoring.

Q. That they do at home or wherever?

A. Well, you can do it at home. But, phase three is technically – it is called phase three, because it is done in a hospital setting, so the patients come back to the same program, come back to the same equipment and everything. They are simply not monitored anymore. And I li differential is probably actually defined by Medicare. Medicare pays for phase two, and they don't pay for phase three. That is really probably the biggest distinction to a patient.

Q. When a patient is not monitored, there are, however, health care providers, such as trained nurses, available, so that if they should have a cardiac event of some sort, there is immediate assistance available?

A. Right.

Page 26

Q. Can you tell me, given everything that you know about Mr. Peacock, did he qualify as someone who would have benefited from or who was eligible for this cardiac rehabilitation program at Saint Vincent's?

MR. HUPP: YOU are saying in retrospect or at what time?

BY MS. SPERANDO:

Q. All right. After the stress test, immediately after the stress test and --

THE WITNESS: would he have qualified for cardiac rehab?

MS. CARULAS: Note my objection.

THE WITNESS: IS that what you m asking?

MS. SPERANDO: Yes.

THE WITNESS: I don't think I could have gotten Mr. Peacock into cardiac rehab phase two as we define it, and particularly as Medicare guidelines, insurance guidelines define it for us.

BY MS. SPERANDO:

Q. Are these guidelines by -- you said Medicaid?

A. Medicare, Medicaid – any social programs, any insurance programs – they all feel pretty much the same.

Page 27

Q. Aretheywritten?

A. Oh, yes. I don't have a copy of those, but, yes, they are written down.

Q. Do you have access to those guidelines?

MR. HUPP: objection.

THEWITNESS: . I suspect I could get them. The clinical director of cardiac rehabilitation certainly has to fulfill those guidelines in the sense that that's how they enroll patients and get paid. So, I am sure the guidelines are available.

BY MS. SPERANDO:

Q. And does Saint Vincent's have its own guidelines separate and apart from Medicaid guidelines?

A. We certainly have policies for rehab. But, they are not guidelines that really specifically are designed to figure out who can enroll.

Q. So, the guidelines by which Saint Vincent abides or to which it adheres are the guidelines promulgated by Medicaid?

A. Pretty much – Medicare.

Q. Medicare.

A. Most of our patients are Medicare, not

Page 28

Medicaid, although Medicaid has similar guidelines. You could pick any insurance company or Medicare and Medicaid, and they have certain guidelines, certain diagnoses, criteria, and so on and so forth.

Q. So, it is your testimony that Mr. Peacock's cardiac condition and status as known after the stress test and his admission to the hospital would not have qualified him for admission to the cardiac rehabilitation program pursuant to the guidelines by which you operate at Saint Vincent's?

A. Correct. I don't think he would have fulfilled the guidelines by which we enroll patients in phase two cardiac rehab.

Q. Maybe you can tell me what the guidelines are and how he would not have fit in.

A. In general, it is pretty simple. Patients fit into the guidelines either -- Medicare has specifically three diagnoses. And that's really how -- I hate to hang my hat on Medicare, but that's who typically take the lead on this issue. Medicare guidelines state that one of three diagnoses will qualify a patient for cardiac rehabilitation. And, in general, we require documentation of exercise stress testing to put the patient into rehab, Those three diagnoses are post bypass surgery, chronic

Page 29

stable angina --
Q. Post bypass surgery, which knocks Mr. Peacock out of that?
A. Right. Chronic stable angina and myocardial infarction.
Q. So, if someone has left ventricular hypertrophy and coronary artery disease but no documented evidence of a myocardial infarction, they are not eligible for the program?
A. That's right.
Q. That is eligible pursuant to where Medicare would pay; is that right?
A. And most insurance companies follow the lead of Medicare. Yes.
Q. Obviously, however, if a person wanted to pay himself –
A. If a patient wanted to pay, then they would be welcome to enroll in the program. Unfortunately, the cost is prohibitive.
Q. With regard to the evidence of a -- what is it called -- a sub-Q myocardial infarction?
A. Non-Q.
Q. Does that count in terms of putting a person in the category of someone who has suffered an MI?
MR. HUPP: objection.

Page 30

THE WITNESS: If I could make a diagnosis of a non-Q wave myocardial infarction, they could possibly be enrolled in cardiac rehabilitation phase two.
BY MS. SPERANDO:
Q. Now, let me then focus you in on that. Do you believe that Mr. Peacock suffered a non-Q wave myocardial infarction?
A. No.
Q. Tellmewhynot.
A. I think Mr. Peacock's presentation was of syncope, which admitted him to the hospital -- was accompanied by a number of clinical findings. And certainly, a suspicion for cardiac involvement was raised.
However, there is no definitive data that says he had a myocardial infarction. He had a clearcut stress test that showed, frankly, he didn't. And that's a very good physiologic way to look for this.
Q. Well, let's put the stress test to one side. And let's talk about the findings, specifically, the enzyme findings, the EKG findings, and the echocardiogram findings.
A. Okay.
Q. With regard to those findings, was there any

Page 31

evidence at all -- let me put it this way: Was there any evidence at all that he had suffered a non-Q wave myocardial infarction?
A. No.
Q. None at all, nothing that would even suggest it?
A. No. I think that his presentation -- let me go specifically through those. Electrocardiogram is equivocal. In other words, he has left ventricular hypertrophy findings, if I recall correctly. And any time you have that finding on an EKG, certainly, the EKG has abnormalities which are equivocal. So, it is an abnormal EKG. In that sense, that doesn't give us any definitive evidence for a non-Q MI.
Q. Let's get some definitions straight. You keep using the word "definitive." And we here in the legal business -- and I understand you guys in medicine, too, very rarely talk about anything that is definitive; am I right?
A. Right.
Q. Now, legally, when I ask you a question, I am talking about within a reasonable degree of medical probability. I am sure you have heard that phrase.
A. Right.
Q. Which means a 51 percent likelihood. When I

Page 32

use the phrase "Consistent with," that does not necessarily mean – in fact, does not mean "equal to."
A. Okay-
Q. So, the question very specifically is: Was there any evidence that was consistent with a non-Q wave myocardial infarction?
MR. HUPP: objection. That has been asked and answered.
THE WITNESS: Again, a non-Q wave myocardial infarction does have a definition. It means that you evaluate a patient. You feel that they have had a myocardial infarction, damage to the muscular tissue, the heart tissue, and that despite that interpretation of all the data in front of you -- clinical presentation, the electrocardiogram – you don't have electrocardiographicevidence of a myocardial infarction. So, right away, EKG by definition tells us that it doesn't give any supporting evidence, so –
BY MS. SPERANDO:
Q. Let's take it one at a time. The EKG?
A. No evidence of a non-Q wave myocardial

Page 33

infarction.
Q. Theenzymes.
A. The enzymes, as I recall, were elevated into a range of 400 or so.
Q. 426,460, and 445.
A. Okay. Are you working from Doctor Herskowitz's –
Q. I am indeed.
A. I will assume that that data is accurate. Those enzymes -- creatine phosphokinase enzymes are elevated in all three cases. Interestingly enough, they are basically all equally elevated. In other words, there is no rise and fall in those numbers. There is nothing about those numbers that tells me clinically that that is consistent with an acute myocardial infarction, non-Q wave or Q wave.
The MB fraction is two plus. My understanding -- and, again, this is different in everyone's system. We don't use a two plus MB system at Saint Vincent's Charity Hospital. We didn't use it at the Clinic. We didn't use it at Deaconess Hospital. However, at University Hospitals, they use a two plus system.
The two plus -- again, all being equal -- interestingly enough, tells you that there is no

Page 34

basic rise and fall or change in these enzymes which would give you a hint that this is actually consistent with myocardial infarction. Those are equivocal. Those are equivocal under any circumstances. They don't tell you that there is cell tissue death. In a patient who has been exercising as hard as Mr. Peacock exercised – he was playing tennis – I wouldn't be surprised to see CPK elevation, and I wouldn't be surprised to see MB fraction.
To define a non-Q wave myocardial infarction, I would have liked to see much more significant MB fractions in those enzymes, particularly given his level of exercise.
Q. Distinguish for me what a non-Q wave myocardial infarction is versus your run of the mill myocardial infarction.
A. Sure. I started to do that.
Remember what I was saying about the electrocardiogram? The electrocardiogram in terms of defining myocardial infarction relies on a Q wave. A Q wave is a negative deflection of the electrocardiographic signal. A non-Q wave myocardial infarction means that you look at the electrocardiogram and see no electrocardiographic

Page 35

definition of a myocardial infarction. It doesn't mean -- so, it means there is tissue death. There is cell death, but no electrocardiographic evidence of that cell death.
Q. So, by definition, then, with a non-Q wave MI, you wouldn't expect to see any EKG findings?
A. I wouldn't expect -- that's right.
Q. But, a non-Q wave MI basically means there has been heart muscle death?
A. Right.
Q. Now, describe the significance of elevated CPK enzymes in terms of myocardial infarction.
A. CPK is an enzyme that is found in every muscle in the body. Any muscle that is damaged will produce in the bloodstream an elevation of CPK here are really three isoenzymes -- if you will, i the laboratory, bands of ti kinase t are c d n th lab test that help o t tell what kind f muscle g lar age l. There is an MM band, a BB band, and an MB band.
MM teclinically w s s I s to be l for muscle, B . br i although a BB d s not rily li ati . f a stroke. So, here are combinations of these things the idea being that the MB fraction is reasonably specific for he muscle damage.

Page 36

So, you look for an elevation of CPK, but you also look to tell whether that CPK came from the heart. And certainly, where there is a high level of MB, you would say it came from the heart. However, low levels of MB can accompany any muscle damage. This is a low level of MB. So, it doesn't tell me specifically that this is heart muscle damage.
Q. Well, what is the normal range of a CPK, just a CPK enzyme which you would expect to see if there had been no muscle damage?
A. No muscle damage at all? Somebody who is sedentary comes in --
Q. Likemyself—sedentary.
A. I don't know what you've been doing today. But, generally, you would expect to see that less than 200 or so. Labs are different.
Q. Let's go to this particular lab. I know labs are different.
A. I don't know their upper limit.
Q. Let's do that, because I think it's important.
A. Do we have that?
Q. Does anybody have the records for the

Page 37

hospital?
A. Here is their normal range: Zero to 225.
Q. So, zero to 225. So, then, you would agree with me that CPK's of 426, 460, and 445 are elevated?
A. Yes. Those are elevated.
Q. So, we know that there has been some muscle damage as a result of these elevated CPK's?
A. Right.
Q. Now, further defining where this muscle damage has come from, we look to the band, the MB; is that right?
A. Right.
Q. What is the normal for an MB?
A. Their defined normal range is negative; in other words, zero.
Q. And what would be considered, then, elevated?
A. Anything over zero would be technically elevated, by their laboratory.
Q. If you can tell me what is the range that they describe elevated bands in, how high can it go?
A. Well, for instance, they use two plus equals greater than international 15 units.
Q. What does that mean?
A. The international unit is a measurement of

Page 38

activity. It is helpful where you have low levels of enzymes to tell whether you actually have leakage from the heart. You would like to see at least some absolute amount of enzyme. You would like to see the fraction, number one, how much percent is that fraction. And you would like to see the absolute amount.

The definitions get hard. For instance, if someone gets hurt or has muscle damage or has trauma, and their CPK enzymes are grossly elevated, the percentage of the MB fraction may not help you tell whether it is the heart or not. By the same token, in the low numbers, the percentage may not help you much. That's where an absolute number of international units would help you tell-whether there is a myocardial infarction. And by definition, most of the time, we hang our hat on an absolute number of greater than 25 units -- 25 international units as being indicative of myocardial infarction.

So, in other words, there is an elevation here. But, again, you can see that with generalized muscle injury, vigorous exercise -- weight lifters all the time -- anytime you check a weight lifter's CPK after a workout, it is going to be elevated.

Page 39

In other words, trauma to the muscle, even Vigorous exercise, can elevate the CPK. what you would like to see then is, what does the MB band specifically tell you? And you base that on numbers like this, in which case they say -- their number two plus is just sort of a generalized range greater than 15 units and greater than three percent, which is equivocal. Whereas, four plus greater than 25 international units -- so, they have correctly picked that number to tell you that if it is greater than 25 international units, you know that you have myocardial infarction. You know that you have muscle death from the heart.

So, they have used their system, two plus, four plus, to tell you whether the enzymes are very specifically related to the heart, equivocally related to the heart, or clearly not related to the heart.
Q. So, the most you are willing to say is that the elevation of two plus for the MB part of the elevated CPK enzymes is equivocal with regard to whether there is heart muscle damage?
A. Yes. I will go one step further to restate what I said before in that the CK enzymes here also don't behave in a pattern that would make you think

Page 40

that it is myocardial infarction.
Q. What is the pattern that would make you think that it is myocardial infarction?
A. Generally, CK enzymes will peak in an 8- to 12-hour period and disappear in 24 hours. So, there is a rapid rise and fall if it is due to the heart.

Part of that has to do with the patency of the vessels. If the vessels have complete blockage, that may be a little slower. If they are patent, then it is usually very rapid.
Q. when were these CPK's taken; do you know?
A. One on admission, I know.
Q. At what time?
A. May 8 at 22:00, then May 9 at 9:00 am., and May 9 at 5:00 p.m. And you can see that over the course of that 20 hours, they basically are all the same level, which is a lot more consistent with generalized muscle trauma, Vigorous exercise, that sort of thing, particularly in a large individual.
Q. Well, the two plus, however, of the MB indicates that for whatever reason, the muscle tissue that was damaged was the heart muscle tissue.
A. No. Any muscle can give you an elevation of MB. Any muscle damage can give you some elevation

Page 41

of MB. So, what you want to distinguish, again, is, number one, is it a high level of MB's; is there a significant percentage of MB? And, number two, what is the pattern of that MB rise and fall? And he has neither of those to support heart attack.
Q. Is it fair to say that when you get these findings, especially when they are in your opinion equivocal, that you are supposed to plug them into the clinical picture?
A. Absolutely these are plugged into the clinical picture.
Q. So, plugging these findings into the clinical picture, would it be fair to say that we have a gentleman who has a significant degree of left ventricular hypertrophy?
A. This doesn't have anything to do with left ventricular hypertrophy.
Q. No. They found out when they did all these tests, plugging these figures into what they knew at the time when they took the tests, that he had a significant degree of left ventricular hypertrophy.
A. Maybe I misunderstood your question. Could you restate your question.
Q. 1mean, by history and by what you know about that particular individual, when you have findings

Page 42

from the lab or from a radiograph, you need to plug those findings into your clinical picture.
A. Right.
Q. And what you know about that individual.
A. Right.
Q. So, we know about Mr. Peacock at that time, as a result of the tests that were taken, that he had a significant degree of left ventricular hypertrophy, correct?
A. We know that, but not from this particular lab test.
Q. 1understand that. I am taking this information and plugging it into what other things that we know in order to make what are on their face equivocal findings – to give them more meaning.–
A. Right.
Q. So, we plug that into the fact that we know he has a significant degree of left ventricular hypertrophy.
A. We know that from echocardiography.
Q. And we also know that he has vessels that are to some degree clogged.
A. He has evidence of coronary artery disease; right.
Q. Tell me what that is.

Page 43

A. Cardiac catheterization – they documented a stenosis in the left circumflex artery, I believe, of about 80 percent. 1don't have that cath report. Let me just take a look here.
By cardiac catheterization, they described the circumflex artery as abnormal, nondominant, with an 80 percent stenosis in the distal circumflex artery.
Q. Is that the only vessel that was stenosed?
A. It is the only vessel that actually has any clinically significant stenosis. There is certainly irregularities as described, up to 40 percent in the right coronary artery posterolateral branch, here (indicating).
Q. So, those are the two arteries that we are talking about that are stenosed to any degree?
A. It looks like, right – the distal circumflex and the right posterolateral.
Q. We also know that this is a man who has had significant hypertension over a period of time.
A. Yes.
Q. We also know that this is a man who has end-organ disease as a result of his coronary artery disease, correct?
MR. HUPP: In retrospect?
BY MS. SPERANDO:

Page 44

Q. No; at that time.
A. No; not at that time.
Q. Did you read Doctor Boulware's deposition?
A. I did. But, you asked me if he had end-organ disease as a result of coronary artery disease. Do you mean as a result of hypertension?
Q. Okay, Hypertension.
A. Yes. He has end-organ disease as a result of hypertension.
Q. We also know that this gentleman suffered a syncopal episode while engaged in vigorous activity.
A. Yes.
Q. Do we have any other information that is significant with regard to Mr. Peacock's clinical status as of the time that he is discharged from the hospital?
A. We had a Holter monitor that was fairly unremarkable. We have an echocardiogram that describes, as you said, left ventricular hypertrophy, and otherwise very good systolic function, no evidence of a segmental wall motion abnormality.
Q. Plugging these CPK enzyme results and MB results, which you say on their face axe equivocal,

Page 45

into what we know about Mr. Peacock's clinical status -- the LVH, the stenosis of the arteries, the significant hypertension over a period of time, the end-organ disease as the result of the hypertension, and the syncopal episode while engaged in vigorous activity -- within a reasonable degree of medical probability, does the CPK and MB results then become more supportive of the fact that there was, in fact, some myocardial damage or death of heart tissue?

A. I would say, either standing alone or with all that other data, there is no evidence of myocardial infarction from those enzymes.

I would add one other thing to this; and that is, you mentioned syncopal episode with vigorous exercise, but also recalling that this gentleman had no other symptoms prior to that -- and that is important. He had no clinical syndrome prior to this presentation.

Q. What are you talking about specifically?

A. I don't have any symptoms or signs of trouble prior to this.

Q. Were you aware of the fact that he was reported to have experienced dizziness immediately prior to the episode?

A. I did see one note in there that somebody

Page 46

documented dizziness, which is a very nonspecific term in this setting. But, I also saw multiple notes that stated that he was not really having prior symptoms. What I am referring to actually is even before this episode. This isn't a man who came in complaining of anything that would suggest coronary artery disease.

Q. So, with regard to all of this evidence, then, it is your opinion within a reasonable degree of medical probability -- that is, greater than 51 percent likelihood -- that these elevated enzymes indicate that he suffered muscle damage as a result of exercising; is that correct?

A. In a layman's sense, yes, muscle damage, meaning, yes, he must have worked awful hard playing tennis or he may have had vigorous exercise which caused enzymes to be elevated. That's right.

Q. You believe that, notwithstanding the fact that he had been exercising at least 24 hours before this last CPK was taken?

A. Well -- and that's part of the reason I interpret it this way -- the fact that they stay so elevated for that period without any other pattern that helps me indicate that it's the heart makes me think it is muscular.

Page 47

Q. Because only when it is heart damage do the enzymes go all the way up and all the way down in a fairly defined pattern like that?

A. Right; exactly.

Q. Fair to say, doctor, that when you have this left ventricular hypertrophy, that means in laypeople's terms, a thickening of the left ventricle; is that right -- the left side of the heart; is that right?

A. Right.

Q. And in this case Mr. Peacock's left ventricle was approximately twice as thick as an ordinary person's heart -- his left ventricle?

A. I don't recall it was twice as thick.

Q. Take a look at --

A. Yes. His posterior wall -- again, your autopsy report will give you a better feel for that. Left ventricular or posterior wall thickening by echo suggests that it is 16 millimeters, with their normal range 6 to 11. We actually use in my lab 8 to 12. So, there is a little bit of difference there. A 16-millimeter thickness is a moderately thickened left ventricle wall.

Q. So, that would be about twice as thick -- the range, you said, is 6 to 14?

Page 48

A. No, up to 12 in my -- but 11 in their lab.

Q. 6 to 11?

A. They are using 6 to 11.

Q. So, if it is 16, would it not be fair to say that it is approximately two times as large as it is supposed to be?

MS. CARULAS: objection.

THE WITNESS: You can't say that it is supposed to be a particular thickness. There is certainly a range there. And I don't even know what this gentleman's baseline thickness was. And in a heart like -- I understand with his body habits and being a relatively heavily muscled person, he may naturally have had an upper limit of normal or even exceeded normal at baseline.

But, it is clearly thickened. This is a thickened heart, what I would call moderately thickened. This is not a severely thickened heart.

BY MS. S P E W .

Q. Can you tell me, sir, what was the degree of hypertrophy on the first EKG that was taken by Doctor Boulware in 1986?

A. EKG's don't give you a degree of hypertrophy.

Page 49

They infer with – actually, fairly insensitively, they infer left ventricular hypertrophy. But, that's a nonspecific finding, particularly in 1986, when this gentleman was only 35 or 36 years old. It's almost impossible to use an electrocardiogram to define left ventricular hypertrophy in a 35-year-old.

Q. You can only do that with an echo?

A. Defining left ventricular hypertrophy is best done by the echo. It's not the only way. It is best done with the echo if the patient is alive.

And the EKG evidence is nonspecific, and only becomes somewhat applicable as the patient gets older. So, in young individuals – I am sure if we checked your EKG it might very nicely have high voltage, because you are a young person. And we may not be able to infer hypertrophy from that. If you were over 40 years old, we might be able to do that.

Q. So, before this echo was done in the hospital there was no evidence of the degree of thickening of his left ventricle?

MR. HUPP: objection.

THE WITNESS: He is a 36-year-old, who now comes in with an EKG, whose voltage -- who, by voltage criteria,

Page 50

would suggest left ventricular hypertrophy. But, again, that is a relatively insensitive way to do it. It is certainly a definition. It is certainly a clinical – a scenario used.

BY MS. SPERANDO:

Q. I am talking about the degree of the –

A. You wouldn't be able to tell, no, particularly in a young person.

Q. So, in layperson's tern, then, this thickened left ventricle would require more blood to nourish it and provide oxygen to it in order to keep those –

A. That's an interesting question. In terms of left ventricular hypertrophy, there are a number of thoughts about oxygen requirements. A resting heart or an exercising heart always extracts one hundred percent of the available oxygen that it can extract. So, a hypertrophied heart or a nonhypertrophied heart will extract oxygen with the same ability.

Q. I am not asking that ability. I am asking about requirement.

A. The question is, how much blood does it actually require? It is almost impossible to say

Page 51

that it actually requires more blood.

Q. How about more oxygen? Oxygen is what is basically the nourishment supplied by the blood; am I right?

A. Yes, basically; right. So, in a sense, you could say that.

Q. Let's talk, then, about oxygen requirement. It is fair to say that if you have more muscle mass, in the form of thickened muscle mass, that it would require more oxygen in order to sustain it; is that fair to say?

A. You could theorize that. Yes.

Q. Well, theorize or not -- is that just theoretical?

A. Yes. You can't define that.

Q. I am not asking basically to defme it. As a proposition, is it fair to say that the more heart muscle you have in the form of a thickened heart muscle, the more oxygen it would require to sustain it?

MR. HUPP: objection.

Are you saying that is something that is acceptable in the medical community?

BY MS. SPERANDO:

Q. correct.

Page 52

A. I think it is probably generally acceptable.

Q. And would it be also fair to say that when you have stenosis of the distal left circumflex and the – was it the right coronary?

A. Yes.

Q. That – again, in layperson's terms – that the heart is not getting – as a result of this stenosis or closing, that the heart is not getting as much blood or oxygen as it would otherwise get if these vessels were not closed?

A. No. That's my point – is that a 70 percent stenosis might be considered flow limiting. Or 80 percent; I am *sorry*. But, the 40 percent stenoses would not be considered flow limiting stenoses.

Q. So, then just with regard to that one vessel, is it fair to say that the blood that the heart would normally receive from that one vessel is being limited as a result of the 80 percent stenosis?

A. No. It is not fair to say that, because the point is that you have -- there is a difference between anatomy and physiology here. Anatomy says there is an 80 percent blockage there. Physiology says the question is whether it is getting enough blood.

Q. No. I didn't ask you enough. I said as much

Page 53

as it normally gets,
MR. HUPP: Let him finish his answer.
THE WITNESS: My point is that it could get as much as it normally would even in the face of what we see anatomically to be an 80 percent stenosis. It could get adequate blood supply, as much as it needs.
BY MS. SPERANDO:
Q. How does that work?
A. *Your* heart extracts a maximum amount of oxygen, even at rest. So, the idea is that it does receive more blood to do exercise, to perform. In any heart in which there is a stenosis defined by anatomy, there are other ways, number one, other than directly, to get flow through that in a forward flow direction.
I am trying to explain this as best I *can.* That tissue gets blood supply from vessels that we don't appreciate on coronary angiography: And the vessels that supply that might not be what you *see.* What you see on coronary angiography are called epicardial vessels. They sit on the surface of the heart. Luckily, those are the ones that get coronary artery disease, and not the true resistance

Page 54

vessels, or *the* vessels that actually supply blood to tissue.
So, you are asking if a stenosis of 80 percent is clearly going to limit flow to muscle to that area. I can't tell you that it is. And the difference is between you what you see anatomically on a cardiac catheterization and what you see physiologically as that muscle performs and actually takes the blood.
Q. I am simply referring to that one vessel, 80 percent stenosed. Is it fair to say that the blood flow through that vessel to the heart is limited?
A. Could be limited. Yes. It could be.
MS. CARULAS: Objection.
BY MS. SPERANDO:
Q. I am not saying could be. Is the likelihood that a vessel that is 80 percent stenosed is going to not transport as much blood as it otherwise would have?
A. Right. That's the likelihood, with greater than 51 percent. Is that what you are asking me?
Q. Yes.
A. Yes.
Q. Now, when an individual is exercising vigorously -- and by "vigorously," I mean both

Page 55

singles and doubles tennis -- is the requirement of oxygen by the heart muscle increased?
A. Yes.
Q. So, now, in a person such as Mi. Peacock, with, as you describe it, moderately severe left ventricular hypertrophy, and one vessel that is 80 percent stenosed and limiting the blood supply, with an increased requirement of oxygen during vigorous activity, why would you say that that person is not at risk for a sudden cardiac event such as a malignant arrhythmia or ischemia?
A. If you take any person with that description -- left ventricular hypertrophy and an 80 percent stenosis of a vessel -- and *ask* me are they at risk for an event, the answer is yes. They are at risk.
Are they at a significant risk? That is not defined by the anatomy you just gave me. Do you see what I am saying?
Q. I understand the language you are using. 'When you say **risk,** that they *are* at **risk,** how would you defme the **risk?**
A. Well, risk is defined prognostically. In other words, given set criteria or given set information, can I prognosticate to say is this patient at risk for something? There is nothing

Page 56

prognostic about an 80 percent stenosis in the left circumflex coronary *artery.*
Q. I am not asking you --
A. Okay.
Q. I am not asking you to -- I can't quite think of the word right now. I am not asking you to divide or separate each of these things that we are talking about.
A. Iunderstand.
Q. I am asking you to take this patient as an entity. And what you said was, taking him as an entity, there is a risk of a malignant arrhythmia or an ischemic event for him. But, you said your could not say that it was a significant risk or you did not know the degree of the risk. Is that fair to say?
A. In that individual patient, that's correct.
Q. Now, my only question to you, sir, is, how would you describe the **risk?** If you can't say that it is significant, how would you describe it?
A. **Again,** risk is not disease. **Risk** is the potential to ultimately develop some **disease.** The way to **look** at these patients is to define as best you can what their anatomy is and what their physiology is and to correlate that with their

Page 57

presentation and their symptoms and so on and so forth and then to prognosticate.

Unfortunately, there is not anything prognostic about — there is nothing significantly prognostic or easily prognostic about a person with left ventricular hypertrophy and 80 percent circumflex disease. If you take either of those criteria or both of them together, they each carry some risk, which is increased for those things. I think that is well recognized. People with left ventricular hypertrophy do have an increased risk of sudden death. And people with coronary artery disease have an increased risk.

However, in an individual patient, then, you need to take those things and apply some other criteria of the risk factors — age, sex, functional capacity, all those sorts of things. And then you could prognosticate to say what that person's risk means.

So, in and of themselves, I think I am in agreement with you that there is an increased risk in those things. But, they don't in an individual prognosticate for me what that patient's likelihood of developing disease is or an event is.

Q. Okay. And what would do that? Specifically

Page 58

in that case what would enable you to prognosticate?

A. I think in this gentleman's case you have a stress test, which I think is in cardiology one of the best prognosticators there is. And he performed on a stress test. And that helps to prognosticate.

Q. Does that remove the risk to zero?

A. No.

Q. What does it lower the risk to?

A. It doesn't change the risk of left ventricular hypertrophy and coronary *artery* disease.

Q. No. The risk we are talking about is a risk of a significant arrhythmia or an ischemic event.

A. Iunderstand.

Q. How does — let's say this particular stress test --

A. It changes the risk profile for that individual. And where it does that is it translates this anatomy into a patient, via functional capacity now gives us a complete picture of anatomy and physiology picture of this patient's risk of an event.

Q. How do we translate that to Mr. Peacock in terms of risk, now that we have the stress test?

A. I think his stress test is powerful prognostication that says — he exercised to 12

Page 59

mets, ten and a half minutes, both of which tell you that this man is capable of fairly high level exercise, certainly about average or above average for his age and sex, and that having exercised to that degree, his risk of an event from a cardiac -- or his risk of a cardiac event is over -- it depends on whose data you believe -- but, is over the next four to five years actually excellent.

Q. What is the risk, doctor, if you can quantify? Let me make it much more basic. Does it put the risk at zero?

A. No. No one's risk is zero given those findings.

Q. As best you can quantify for me, with these stress test results, assuming they are valid, what does it put his risk at?

A. If you look at data from the coronary artery surgery study, in which people had severe coronary artery disease, if they were able to exercise into the fourth stage — which he did — of exercise, their four-year survival was one hundred percent. If you look at —

Q. What study are we talking about?

A. Coronary artery surgery study.

Q. Where do I get my hands on it?

Page 60

A. It was a national registry that was surgery patients collected from 1976 to 1986. If you look under anything in the Med Line under coronary surgery study you will find, even in 1996, articles published about that patient population. That is a well known study.

Q. So, they are saying in this study that people with Mr. Peacock's anatomical findings and his ability to do the stress test the way he did it — they have a four-year survival rate of one hundred percent?

MR. HUPP: Objection.

THE WITNESS: Individuals are -- again, we are trying to balance, again, between findings on a study and the individual. But, patients who can exercise — even patients with severe coronary artery disease who can exercise into the fourth stage generally in that study had a one hundred percent survival rate of four years.

BY MS. SPERANDO:

Q. Since we are not relying on statistics, we are talking physician to patient one on one, not statistics -- that's how you want to be treated, right?

Page 61

A. That's right.
Q. You don't want to he treated based on statistics. So, based on this particular gentleman's physiology as determined by *the* tests and based on his stress test what was his risk for an event as we have talked about after the stress test results were known?
A. I will say near zero. Will that define it close enough for you?
Q. Near zero, but it is not zero?
A. Yes.
Q. If it is not zero, does a physician have the responsibility to tell the patient that there is a risk, but it is a very small risk, however small it is?
MR. HUPP: Objection.
THEWITNESS: I think a physician has the duty to advise a patient what his risk factors for problems, disease — cardiac disease or otherwise — are and to help him modify those. To give the patient a number — is that what you mean?
BY MS. SPERANDO:
Q. No. I am saying if the risk for a sudden cardiac event if a patient engages in a certain type

Page 62

of behavior is not zero, does the physician have a duty within the standard of care to advise the patient of the risk, however small it is?
MR. HUPP: objection. Asked and answered.
THE WITNESS: I think a physician has the duty to educate the patient as best he can that he is at risk for whatever those reasons are and how to modify those risks. As far as telling them specifically all the outcomes and the chances of those, I don't know how you could do that.
BY MS. SPERANDO:
Q. Well, sir, I am not asking about all the outcomes. I am asking about a particular outcome; and that is sudden death. Does the physician — is he required by the standard of care to advise a patient that he is at risk to whatever extent for sudden death if he engages in a certain type of activity?
MR. HUPP: objection. That's the same question he just answered.
THE WITNESS: I don't think he has a requirement to advise a patient about a specific event like that. I am having trouble

Page 63

with this. In other words, Mr. Peacock presented with a syncopal episode.
Does Doctor Boulware or Doctor Biblo or any of these other doctors have some sort of — you are asking me if they have a responsibility —
BY MS. SPERANDO:
Q. Within the standard of care.
A. — To tell him that he is at risk for sudden death?
Q. Yes. If he engages in vigorous activity such as tennis.
MS. CARULAS: Objection.
MR. HUPP: same objection.
THE WITNESS: He has an obligation to educate him as to the possibilities that it is there. That would be a tough one to —
BY MS. SPERANDO:
Q. Now, let's focus on the stress test results. You understand that, doctor — that Mr. Peacock was given Nifedipine prior to the stress test; is that right?
A. Uh-huh.
Q. And that was because he had a — let me just find it — he had a blood pressure of 168 over 116

Page 64

before the stress test; is that right?
A. I think that's correct. Yes.
Q. That's what Doctor Herskowitz says. If you have some other —
A. *Yes.* I have a copy of the stress test nght here. It saysthat.
Q. *So,* when they gave him the Nifedipine. Is Procardia the same thing?
A. Yes.
Q. When they gave him that drug, would it be, then, fair to say that they were not comfortable — those people who were administering the test were not comfortable with allowing him to undergo a stress test with a blood pressure of 168 over 116?
MS. CARULAS: Note my objection. I think this has already been answered by the people themselves, meaning Doctor Effron and so forth.
THE WITNESS: I can't comment on his level of comfort in doing the test with that kind of blood pressure.
BY MS. S P E W :
Q. Why, then, is it your understanding that the ten milligrams of Procardia were given?
A. Ostensibly, it says that the blood pressure

Page 65

was elevated at rest, and after ten milligrams of Nifedipine was 150 over 100. So, I am going to – I can go with the logical assumption that he gave the Bocardia to lower the resting blood pressure.

Q. Can you tell me, sir, then, to what extent the stress test results are valid, when Mr. Peacock is not given ten milligrams of Procardia or Nifedipine before engaging in vigorous exercise?

A. I think the stress test results are clearly valid. The resting blood pressure of 168 over 116 was apparently the result of not taking any medication prior to the stress test.

Q. Wait a minute. Do you know that for a fact?

A. I don't know that, but that's what one would assume from this.

Q. Well, on what basis are you assuming it?

A. Because we instruct patients usually to hold their medicines prior to stress testing.

Q. Did you know that Mr. Peacock was instructed to take medications upon his discharge from *the* hospital?

A. Oh, yes, I am sure from the hospital he was instructed to take those.

Q. Is there anything in the record to indicate that Mr. Peacock had taken or had not taken any

Page 66

medication before he went to the stress test?

MR. HUPP: objection.

THE WITNESS: I don't actually have that here. I don't know that. I don't know that, whether he was instructed – I don't know how he was instructed prior to this stress test to prepare himself for the stress test.

BY MS. SPERANDO:

Q. So, not knowing one way or the other whether he had taken any blood pressure medication, but knowing that his blood pressure prior to the test was 168 over 116, and knowing further that with that blood pressure he was given Procardia and then had a blood pressure of 150 over 100, at which point the stress test was administered, to what extent then can you say that *the* results of this stress test would be valid if Mr. Peacock had not been administered ten milligrams of Procardia or a similar drug prior to engaging in rigorous exercise such as he was made to do on the stress test?

A. These stress test results are completely valid, even given the Procardia.

Q. Dega me. Tell me why.

A. Because the validity of a stress test result

Page 67

is based on cardiac work. Cardiac work is defined basically as elevation of blood pressure and heart rate. The statistical significance of a stress test depends primarily on reaching a maximum heart rate of 85 percent of his predicted maximum, which he did.

The fact that his blood pressure was elevated at rest didn't seem to affect his ability to elevate his blood pressure with exercise, which is a normal response to exercise. And, frankly, all these numbers axe clearly within the *limits* of a stress test and the *limits* of acceptable stress test criteria.

Q. Doctor, I am going to have to confess, I don't know anything about these tests, these results. But, as a layperson, I do understand that if blood pressure is elevated – and you can disagree with me -- that if blood pressure is elevated that means there is an increased load or pressure on the heart. Is that fair to say?

A That's what is inferred by that. Right.

Q. And when there is an increased load or pressure on the heart, that puts that person at greater risk for an arrhythmia or an ischemic event if, in fact, he has left ventricular hypertrophy,

Page 68

coronary artery disease that would otherwise cause him an ischemic event.

A. What you infer is probably intuitively correct, and it may have some significance. The heart doesn't sense blood pressure. The heart senses tension – wall tension. Wall tension and pressure are not the same thing. It has something to do with the size and shape of the heart. It does have something to do with the thickness of the heart.

And so, we infer that the tension inside the heart is actually increased, that the tension inside the heart causes stronger contractions; and therefore, the work of the heart is increased. We infer that by elevated heart rate and elevated blood pressure. Some people use *the* product of heart rate and systolic blood pressure to also infer that work. Again, it is somewhat nonspecific. But, we hope it relates the two.

Q. Okay. So, basically, the higher the blood pressure, especially after a certain point, the greater the risk of damage to the heart when you engage in vigorous exercise?

A. Oh, no. Any one of us sitting at the table can probably elevate our blood pressure to the level

this was elevated. People who are not hypertensive will achieve with each stage of exercise somewhere around seven to ten millimeters of mercury increase in their blood pressure. Assuming you start at a normal blood pressure of 120, 130, if you go five or six stages, you will hit 200 millimeters mercury. There is no damage to your heart from doing that.

Q. Like I said, I could be wrong. I understand that in a person such as Mr. Peacock, with his anatomical status and condition, that a high blood pressure puts him at risk for a cardiac event.

A. His high blood pressure chronologically, in other words, over many years, puts him at risk for a cardiac event. Hypertension -- systemic hypertension is a known independent risk factor for heart disease. Hypertension chronically over the years also puts him at risk for left ventricular hypertrophy.

Again, the disease and the risk are different. His hypertension as a single event -- in other words, walking on a treadmill and elevating his blood pressure to a level of 210 over 100 - is not a significant risk factor for anything.

Q. Why would they lower the blood pressure before they even the start the test? What is the point?

A. It is interesting that they do it. They may have some criteria for that. And I don't know what Doctor Effron's criteria is. But, in general, if someone presents at baseline for a stress test - and I think, you know, there is documentation for *this* -- with a blood pressure - I use the number 180 over 120 at baseline. If they present with a pressure below that, then they are actually statistically okay to exercise on that treadmill test. Statistically meaning - realizing that there is no report that I am familiar with - and having done this as a fellow, I haven't really looked at this in a long time. But, there is no report that anybody on a treadmill presenting with blood pressures like that ever suffered an event, even though their blood pressure is elevated.

In general, we limit people on a treadmill when their pressures start to either at baseline -- again, my limitation is 180 over 120 -- and other people will use different numbers. But, the limitation with exercise is more in the range of 250 systolic and 120 diastolic.

Q. So, my question becomes -- as a layperson - how can these test results be valid if what you have done is medicate someone and thereby affect the

results? I mean, you are testing what his blood pressure is going to be and his heart rate is going to be at the end of this stress test. That's what you want to find out, correct?

A. Yes.

Q. And yet, immediately prior to the stress test you give him drugs which will affect both the blood pressure and the heart rate. So, then you say -

A. He didn't receive any drug to affect his heart rate.

Q. Is it your contention, doctor, that a drug that affects blood pressure is not going to affect heart rate? Is that what you are saying?

A. There is no evidence that being given Procardia affected his heart rate. Procardia is not known to affect heart rates.

Q. Not at all?

A. No.

Q. So, blood pressure has no correlation whatsoever to heart rate?

A. No. That's not true. There are drugs that do affect both blood pressure and heart rate, most notably Atenolol, which he is on. But, drugs like Lisinopril, which he is on, generally do not give you reflex tachycardia. And, in fact, Lisinopril

has an effect that statistically lowers heart rate. So, even though it lowers blood pressure, it also does lower heart rate, not through the same mechanism. So, some of them do give you reflex tachycardia. Some of them do give you bradycardia. There are all sorts of combinations. Nifedipine itself isn't something that chronotropically affects the heart.

Q. You are trying to determine as a result of the stress test what his blood pressure will be and what his heart rate will be, and then you give him a drug immediately prior to the stress test that you know is going to affect the blood pressure, which drug you don't give him immediately prior to his engaging in tennis or whatever else he is going to do. So, how then can you rely on these stress test results to prognosticate what will happen in a setting where he is not given these drugs?

A. Okay. Number one, the stress test is still valid as long as your heart rate criteria reaches 85 percent of maximum. A secondary criteria might be to include blood pressure criteria. But, heart rate is the statistical criteria for this. And he achieved that. Nifedipine doesn't affect that.

The third thing is, I can't make any

Page 73

assumption about what he did or didn't receive here. So, whether he received these medicines – Atenerol and Lisinopril -- before stress testing, I have no idea. But, he certainly is prescribed those medicines on discharge from the hospital, and so ostenibly has taken them prior to exercise. I can't tell you that his status when he presented for the exercise has anything to do with his status prior to playing tennis.

Q. I am not asking you that.

A. You are asking me to predict on the basis of this stress test whether – you are saying he didn't get Nifedipine before he played tennis. I don't know what he got before he played tennis. I don't know what he got before he took this stress test.

All I can tell you is that, statistically speaking, the criteria for stress test accuracy is based on heart rate, which he achieved. It doesn't have anything to do with the thallium – and that the blood pressure is actually not a major criteria for determining whether this stress test is accurate or not. His blood pressure rate product – his rate blood pressure proauct is actually quite good, 150 times 210. I would challenge anybody in this room to reach a rate pressure uroduct of 31,500. That's

Page 74

a very high rate pressure product.

Q. What are you talking about – after he is given the Procardia?

A. Yes; well, after he has exercised. His peak exercise blood pressure is 210 over 100.

Q. But, he has just been given ten milligrams of Procardia.

A. Doesn'tmatter.

Q. Isn't the effect of the Procardia to lower the blood pressure?

A. Yes.

Q. So, if you start out with a lower blood pressure and then you engage in a stress test, does that not affect the blood pressure?

A. It affects the blood pressure, but it does not -- affect the validity of the results of this test.

Q. All you are looking at is the end result heart rate?

That's the definition of a valid test.

But, that's not all I am looking at. I am also looking at the fact that his rate pressure product -- which is maybe not the most up to date way to look at this or whatever or something that they have detailed here – his rate pressure product is the highest rate attained, 150, times his peak

Page 75

exercise blood pressure, which is 210, which is over 31,000. That is a very high rate pressure product. And for me as a cardiologist I would use that number to tell me this is clearly an adequate stress test.

Q. But, any figures that you are using with regard to the blood pressure by definition have to be invalid, because you have manipulated the blood pressure from the start, have you not?

A. "'hat's not true. That might be your intuitive sense, but it is simply not true.

Q. I'm not relying on my intuition here. It says he starts out with a blood pressure of 168 over 116. It further says he is given ten milligrams of Procardia. It further says, which resulted in a lowering of his blood pressure to 150 over 100.

A. That's an assumption that Doctor Herskowitz made. I don't know that.

Q. Take a look at the record.

A. I am reading it with you.

Q. Not Herskowitz Don't even take his word for it. Let's take a look at the record.

A. I have it right here in front of me.

Q. What do the physicians say his sequence of events were?

A. They don't actually detail his sequence of

Page 76

events. I am not willing to assume –

Q. No. Are you finished?

A. I am finished.

Q. You have to understand I am not asking you to assume anything one way or *the* other. Basically, I am asking you not to assume anything. And I don't know whether he took his medicines. And apparently, neither do you. Is that fair to say?

A. That's correct.

Q. So, then, to make any conclusions based upon any assumption of whether he did or did not take his medicines would not be accurate, correct?

A. It wouldn't be accurate, and it wouldn't have any bearing on this test.

Q. That's what I *am* asking you to do. I don't want you to make any assumptions. We need to get that straight. Secondly, just forget about what Doctor Herskowitz said. And read for me what it was his starting blood pressure was.

A. 168 over 116.

Q. What was the next thing they did as reflected by the medical record itself?

A. They said – taking this out of context, it says blood pressure was elevated at rest, and after ten milligrams of Nifedipine, the blood pressure was

Page 77

150 over 100.
Q. So, the record itself, whoever wrote that, has made the conclusion that after the Procardia was administered his blood pressure decreased, presumably as a result of the blood pressure medication, correct?
A. I think a reasonable person would assume that. However – go ahead.
Q. So, now, how can you not then say strictly with regard to the blood pressure results of this test and not any intuition that I am coming up with that the blood pressure was not manipulated with or affected before the test even started?
MR. HUPP: Note an objection. He has already answered that three times. Maybe you are not understanding it. But, that has been asked and answered. It really has.
THE WITNESS: The blood pressure has nothing to do – as it was manipulated prior to this test has nothing to do with the accuracy of this test. The accuracy of this test depends on achieving a maximum heart rate of at least 85 percent of his predicted maximum -- the electrocardiographic accuracy. Number two, his blood pressure rate product is

Page 78

adequate. I don't care where it started. I don't care the baseline. His *peak* exercise blood pressure times rate product is adequate for me to look at this test and tell you that this is an accurate test, clearly an accurate test.
BY MS. SPERANDO:
Q. So, focusing, then, on the blood pressure rate product, that does not depend upon any change in blood pressure as a result of a drug before the test starts?
A. No, doesn't depend on it. I am only interested to make *sure* he has achieved some minimum.
So, in fact, you have probably gone above and beyond achieving that minimum. You have probably gotten – you started with a lower blood pressure or whatever. It doesn't matter. You have achieved a minimum, in fact, gone way above the minimum that I would be required – and I think any reasonable practitioner would be required – to call this stress test accurate. And it doesn't matter that he received the Procardia prior.
Q. That minimum being what, sir?
A. In general, I use a minimum of 20,000 for the

Page 79

rate pressure product. I also use a minimum of six mets for his metabolic capacity.
Q. And you say in your report, doctor, that no segmental wall motion abnormalities were seen; is that right?
A. Yes.
Q. I recall having read by Doctor Herskowitz's conclusions that the segmental wall motion abnormalities -- let me just get it here for a second. He said something about -- okay. Okay.
On Page Five, at the bottom of Page Five, he says, "The amount of' – this is the third sentence from the bottom -- "The amount of permanent heart muscle damage was small, and altered wall motion could have easily been masked, particularly in Mr. Peacock's hyperdynamic thick-walled ventricle."
Do you *agree* or disagree with that, sir?
A. Well, again, I haven't seen the echocardiogram. And I haven't seen the tape. I don't h o w the quality of that tape. I can't tell you that it was masked by something technical about the study. However, in general, where there is heart muscle damage or ongoing ischemia, wall motion abnormalities are generally considered to be very sensitive. So, if there was a non-Q wave myocardial

Page 80

infarction or ongoing ischemia, I would expect to see some wall motion abnormality.
Q. I understand what you would expect to see. My question, sir, is do you *agree* with him that altered wall motion could easily have been masked, particularly in Mr. Peacock's hyperdynamic thick-walled ventricle? Is that a possibility?
A. The word "mask" is an unfortunate choice of words. Altered wall motion may not be seen or something like that. But, whether it was masked –
Q. Whether or not they use the word "mask" – but, basically, you agree with the proposition that it could have been missed, particularly in Mr. Peacock's hyperdynamic thick-walled ventricle?
MS. CARULAS: Note my objection. I think he has answered it.
THE WITNESS: Yes.
BY MS. SPERANDO:
Q. All right. Let's go and read Doctor Herskowitz's report. I would like to know what it is that you specifically disagree with. So, let's go through it as much as we *can*.
If, in fact, Mr. Peacock had experienced dizziness or light-headedness for about five minutes prior to the syncopal event, what, in your ***mind,***

Page 81

would be the significance of that?
MR. HUPP: what page are we on?
MS. SPERANDO: Page One.
THE WITNESS: Honestly, I don't know the significance of that. In the middle of exercise, that's certainly possible. The gentleman was playing two hours of tennis. He may have been fatigued. I don't know. Again, "dizziness" is a fairly difficult term to use medically. I don't have any evidence in his record that he had true vertigo. No one established that.
BY MS. SPERANDO:
Q. If he did, in fact, have true vertigo or light-headedness prior to the event, what if any significance would that have to you in terms of his having experienced a cardiac event?
A. I don't think it necessarily points to a cardiac event at all. He might be dehydrated or simply fatigued.
Q. If, in fact, he did not have any symptoms prior to his syncopal episode, what significance if any would that be to you in terms of whether he had acardiacevent?

Page 82

A. If he didn't have -- say that again.
Q. Any symptoms at all.
A. If he had no symptoms at all prior to this event, would it have --
Q. What significance would that have in terms of whether this was a cardiac event; that is, the syncopal episode?
A. Again, in a young patient with syncope, that doesn't help me.
Q. What about a complaint of shortness of breath while playing tennis that day?
A. I am not surprised he is short of breath. The man was playing tennis. I think certainly anybody who does aerobic exercise is going to be short of breath.
Q. So, no significance to you in terms of a cardiac event?
A. No.
Q. Let's go to Page Two, please. Now, apparently, the intern who was taking *care* of Mr. Peacock on May 8 when he was admitted concluded, quote, "Given cardiac enzymes, T wave inversions in V4 to V6, and two plus MB. This could very well be ischemic heart disease causing VT and syncope."
Do you agree with that?

Page 83

A. I think that that's a pretty dramatic stretch of the findings.
Q. So, you don't agree?
A. I don't think there is evidence of that.
Q. Okay, The intern goes on to say, "Doubt neurally mediated syncope, given *the* fact that the patient was exerting himself when it happened. Patient was not orthostatic and neuro exam was nonfocal." Let me just focus you in on that one sentence: "Doubt neurally mediated syncope" -
A. The fact that he was exerting doesn't rule that out.
Q. What about the fact that he was not orthostatic and neuro exam was nonfocal in conjunction with the fact that he was exerting himself when it happened?
A. I don't have the intern's definition of orthostasis. But, interns are notoriously unreliable for that. Do you have a definition for what he means by orthostasis? Again, I don't know Doctor Herskowitz's understanding of that term, either.
Q. He is quoting the intern at this time.
A. I understand. But, orthostasis is a definition.

Page 84

Q. How do you define it, sir?
A. Orthostasis in a patient like this is most easily defined as a heart rate variability as the patient is brought from a lving to a standing position. And generally, an increase of heart rate is really your most sensitive finding of orthostasis. The change in blood pressure is often what interns like to use. But, it is not generally considered to be the definition. And so -
Q. Let me ask you this: If the event happened while he was exerting himself and, in fact, Mr. Peacock was not orthostatic and the neuro exam was not focal, do those factors then mitigate against this having been a neurally mediated event?
A. No. It still very *well* could be a neurocardiogenic event.
Q. In your opinion, doctor, within a reasonable degree of medical probability, the cause of Mr. Peacock's syncope on May 8, 1994, was?
A. The cause of his syncope? I don't know the cause of his syncope, to give you a very specific definition. My opinion? Is that what you are asking?
Q. Yes.
A. My opinion is that his syncope was mediated by

Page 85

what we call or what is known as vasovagal -- it means, in other words, an abnormal reflex of some sort. It may have also been neurocardiogenic, although I don't have specific evidence of that.
Q. When you say neurocardiogenic, what do you mean?
A. There are a number of reflexes that can cause a patient to pass out, particularly given vigorous exercise on blood pressure medications after fairly long period of time, at which point whether or not they have defined orthostasis correctly – he may have been dehydrated.
Again, I don't know when this intern writes this note. If he writes the note likely several hours after the patient arrives and has been rehydrated in the emergency room, et cetera, et cetera – I am a little lost to defme what has happened necessarily to that patient.
He may have indeed had a combination of those things. And given the fact that it happened during exercise, I would likely say it is probably what I call a vasovagal event.
Q. Meaning?
[illegible] combination of hypotension
or lowering of his blood pressure – sudden lowering

Page 86

– or the slowing of the heart rate, bradycardia, would combine to make him pass out.
Q. Just so that we are on the same wavelength, does your definition of vasovagal include an arrhythmia?
A. No, not a malignant arrhythmia – bradycardia being the slowing of the heart rate.
Q. Does it include an ischemic event?
A. No.
Q. So, as we sit here today, knowing everything you know about the fact that he died while playing tennis, all the test results, the autopsy report, it is your opinion within a reasonable degree of medical probability that Mr. Peacock's syncopal episode on May 8 was not the result of some form of an arrhythmia or an ischemic event; is that fair to say?
A. That's fair to say.
Q. Doctor, how much are you getting paid per hour on this case?
A. That's a good question. In general, I charge $400.00 an hour.
Q. How many hours have you billed this for?
A. For this whole case?
Q. Yes, sir.

Page 87

A. I wish I could answer that. I am going to guess on the order of eight hours – eight to ten, I would say.
Q. Did you look at the ECG that was done on May 8?
A. I believe I did, although the ECG's that I have received are not very adequately labeled for time and so on and so forth. But, I believe we established the order of them.
Q. Can you, please, sir, refer to it and tell me if you agree with Doctor Herskowitz that there was one millimeter ST elevations in leads two, three, and AVF?
A. I did look at that in light of his ascertation. Or the presenting EKG, which is the one here, which – I can understand how someone might look at that – there is not a baseline that allows you to measure that. And I don't see one – number one, I don't see one millimeter of ST elevation. And number two, I think the baseline is a little too erratic to tell you exactly what the ST segment is doing. It looks quite nonspecific to me.
Q. Do you need a previous -- would a previous EKG in terms of baseline help you determine?
A. Not for this particular EKG.

Page 88

—oOo—
Thereupon, a brief recess was taken off the record.
—oOo—
BY MS. SPERANDO:
Q. Doctor, as I understand a vasovagal response, what happens is – in layperson's terms -- when you are exercising vigorously and then you stop suddenly, your brain is sent a message that it does not need as much oxygen, and then it sends a message down to the heart saying, "I don't need as much oxygen, and you can slow down," but what happens is that the heart slows down too much and then causes the syncopal episode. Is that basically it?
A. Basically, that's right. In other words, the heart receives a signal to slow itself down or the peripheral vasculature suddenly dilates, and the blood pressure can drop because of that. Or it may be a combination of both.
Q. That's why those people who exercise – myself being one of them, when I was -- were told, "When you finish exercising have a cool-down period and don't stop suddenly"; is that right?
A. Well, in general, we do tell people to cool down. It is not because we are afraid necessarily

Page 89

that everyone is going to have a vasovagal event, but certainly, the potential is there, particularly where someone has been charged up in an emotional, hard driving situation.
Q. Is there any evidence that you know of that Mr. Peacock suffered his syncopal event after having stopped playing tennis or stopped engaging in vigorous activity?
A. I don't know. I don't know exactly what the event was or exactly how it happened, in the sense that I know that he was playing tennis. To my understanding, he was having vigorous activity and in the course of that had passed out.
Q. So, if, in fact, Mr. Peacock was continuing to play tennis and experienced a syncopal episode while engaging in Vigorous activity, as opposed to stopping cold or short at any time, and then experienced that syncopal episode, would that not mitigate against your conclusion that it was a vasovagal episode?
A. No. That is a well described phenomenon, that people during exercise can have a vasovagal type -- or neurocardiogenic, if you will, to broaden that term. And that's been well described.
Q. Significance of a possible small pleural

Page 90

effusion that was revealed on chest x-ray while he was in the hospital?
A. Fairly nonspecific finding.
Q. What was the significance of the ST having been elevated in the inferior leads in the ECG taken on 5/8?
A. I den'? think it is defined as elevated. I am saying the baseline wanders. And I would defy anybody to tell me that that is an elevated EKG or ST segment. If you will look at the EKG carefully, you will see that lead two is actually downsloping. And the ST segment in the first lead is fully five or six millimeters above that in the last segment, as it is in lead three. And then AVF actually goes up.
It is impossible to define what the ST segment level is in those leads, but it doesn't appear to be significant.
Q. Let me review the findings of Doctor Chaffee on the second EKG taken on the Ninth, where he talks about the ventricular rate having decreased by 44 beats per minute and says -- and I am quoting -- "ST no longer elevated in inferior leads."
Do you know what he is referring to when he says, "ST no longer elevated in inferior leads"?

Page 91

A. Well, he may have interpreted it one way or the other. I am looking at these. And, again, I don't know what he read in reference to that. But, I am looking at the same two ECG's. And I don't see any defined ST elevation in these inferior leads.
And I see clearly that you have a beautiful baseline on the next EKG, which clearly has no ST elevation present. It is very difficult to compare these two EKG's and try to draw a conclusion. Is that what you are saying?
Q. Let me ask you what it was we can infer that Doctor Chaffee was concluding when he said, "ST no longer elevated in inferior leads." Would it be fair to say that he believed that at some point the ST had been elevated in the inferior leads?
A. That's what that would imply, although he also talks about a 44 beat change in the heart rate. And it is quite common knowledge that ST segments are very sensitive to rate changes. And 44 beats would certainly change the orientation of an ST segment. So, I don't know if he is implying any clinical finding by that at all.
Q. How did the second EKG change? What were the changes in the findings? And what if any significance were the changes to you?

Page 92

A. Again, comparing EKG's, you have to realize there is a change in heart rate. There is a change in his clinical situation. The changes, if at all, are nonspecific, Comparing them side by side right now as I am, it is hard for me to say that there is any specific change in these EKG's.
Q. What is the significance of a decrease in beats per minute?
A. Well, certainly, people have elevated heart rates for a lot of reasons. Anxiety could certainly be one. I am going to guess that that's the likely difference.
Q. The 24-hour Holter monitor that Doctor Herskowitz notes that the -- "ST depressions up to 1.3 millimeters including T wave inversion in channel one were noted"
What significance if any would those ST depressions and T wave inversions have for you?
A. Again, this is a nonspecific finding. And in a patient with left ventricular hypertrophy, that has absolutely no clinical significance.
Q. When he was given the stress test, it notes here that the "EKG monitoring revealed 1.5 millimeter ST depressions in leads two, three, AVF in V5 to V6 during peak exercise." Do you see where

it says that on Page Three of Doctor Herskowitz's report?
A. Yes.
Q. What significance if any is that to you?
A. No clinical significance whatsoever in a patient with left ventricular hypertrophy.
Q. Do you know why the conclusion was, "abnormal stress test"?
A. That was the conclusion?
Q. Yes, sir.
A. Let me look at this.
Q. Are you not aware of the fact that that was the conclusion?
A. I want to read exactly what it says, only because, again, extracting one sentence -- what did we do With that?
MR. HUPP: Let's use this. There.
THE WITNESS: It doesn't say abnormal stress test. I didn't think it did.
BY MS. SPERANDO:
Q. What about the handwritten report? Why don't we take a look at that?
A. Where is that? He had the stress test after he left the hospital.

Q. I am sure that there are handwritten --
MS. CARULAS: YOU wouldn't happen to have those handy that you could show them to him?
MS. SPERANDO: I wish I did. All I can tell you is that this is what Doctor Herskowitz said: The handwritten test report stated abnormal stress test. And he is putting that in quotation marks. I am assuming he got that from somewhere.
THE WITNESS: I certainly didn't see that. And I think it would be unlikely to see handwritten notes on a stress test. It would be unusual. I think that would be unusual to see a handwritten note about a stress test like this.
BY MS. SPERANDO:
Q. So, it is your understanding that there is no handwritten note that says, "abnormal stress test"?
A. Correct.
Q. He further goes on to say that the handwritten report stated, "Abnormal stress test. Above average functional capacity for age and sex. EKG changes suggestive of myocardial ischemia."
Do you agree, doctor, that the EKG changes

shown on that stress test were suggestive of myocardial ischemia?
A. No.
Q. What was the significance of the conclusion by -- let me go back one. If there is, in fact, a handwritten test report which states, "abnormal stress test," do you have any understanding as to why anyone would make that conclusion?
A. That would be almost an impossible conclusion to make from this stress test, given the fact that, as they accurately state here, the patient's baseline ECG is abnormal and precludes accurate interpretation. By definition, you wouldn't be able to define that. So, I don't know who would have written that.
Q. So, you agree, then, that the abnormal baseline ECG precludes accurate interpretation of exercise induced ST displacement?
A. Correct.
Q. Now, translate that into English for us, please.
A. Basically, there are two portions to a stress test. There is the electrocardiographic portion. And there is the thallium, or Sestamibi imaging portion, the nuclear imaging, portion.

The electrocardiographic stress test depends on meeting criteria as we talked about before. But, it also depends on a baseline electrocardiogram being essentially normal. If your baseline electrocardiogram is abnormal, there is no way to interpret ST changes accurately in the stress test. Anyone that does clinical stress testing knows that definition by heart.
Q. When they say, "abnormal baseline ECG," are they referring to the ones on May 8 and 9?
A. No. They would be referring to exactly what was in front of them at the time of the stress test. In other words, a baseline electrocardiogram is done as part of the stress test.
MR. HUPP: It is this one, if you want to look at it. It is over there.
BY MS. SPERANDO:
Q. So, they are saying that that baseline of that test done on that day was abnormal?
A. Yes.
Q. And therefore, they could not interpret the significance of the exercise induced ST displacement; is that fair to say?
A. Correct.
Q. Now, what was the significance of the abnormal

Page 97

baseline?
A. His abnormal baseline is exactly what we have talked about before. I don't have – again, I don't have that particular ECG test baseline electrocardiogramin front of me. However, the tracings that are here would suggest that there are voltage changes and ST and T wave changes consistent with left ventricular hypertrophy, which is a known confounder for this type of test.
Q. If the baseline had been normal, what is the significance of exercise induced ST displacement?
A. Well, that is a good question. It depends on the displacement. The displacement, given a normal baseline EKG, by definition has to be at least one millimeter of ST segment depression at point 08 seconds -- in other words, two of these little boxes (indicating) -- after the J point, which is the little point at which this changes direction (indicating). So, it has to be depressed at least one millimeter and needs to be flat or downsloping.
Q. What was it in this case, with this abnormal baseline?
A. Well, his is – it depends, again, where you look. If you look at peak exercise or what is defined as peak exercise – and he is looking at the

Page 98

leads V4, V5, and V6 – you would actually look at V4 and say that's a normal response. V5 is a slightly abnormal minus 1.2. And V6 is slightly abnormal.
But, if you look as quickly as one minute into recovery, which is part of the defiition of how people look at these, they actually are all within normal limits.
Q. Are there any abnormal ST segments?
A. Well, they are abnormal at baseline. And they are abnormal throughout the test.
Q. What is the significance of ST displacement?
A. ST depression is commonly interpreted as a sign of myocardial ischemia if that depression is related to a normal resting EKG and meets those criteria. In other words, a little bit of depression isn't enough to call it ischemia. So, you have to meet the criteria. Those criteria develop statistically.
So, the likelihood of a one-millimeter ST segment depression point 08 seconds after the J point, which is flat or downsloping at peak exercise, and lasting for at least a minute into recovery, would be by conventional standards consistent with ischemia.

Page 99

Q. And even With this abnormal baseline, you are saying that none of the ST depressions meet that definition; is that correct?
A. Because the baseline is abnormal, these reflect that abnormality. If you took these in and of themselves, it is virtually impossible to look at these without looking at the baseline.
Q. Would it be fair, then, to say that the EKG part of the stress test was of no significance?
A. The correct terminology would be that it is nondiagnostic. This is nondiagnostic because of baseline abnormalities.
Q. So, there may be or may not be myocardial ischemia, but we can't tell that based on the EKG portion of the test?
A. The electrocardiogram in and of itself -- the ST segment interpretation is nondiagnostic.
Q. Is there any way that we can then redo that test or retake it in order to have it be diagnostic in terms of determining whether those ST depressions reflect myocardial ischemia?
A. From the electrocardiographic standpoint, there is no way to change this test to make it reflect – statistically speaking – to give you diagnostic accuracy.

Page 100

Q. On 5/18, Mr. Peacock was *seen* by Doctor Boulware. And his blood pressure was 160 over 110. That is not too far different from the 168 over 116, which he had two days earlier before he was given the stress test. Is that fair to say?
A. Yes.
Q. You would not consider that to be under control?
MR. HUPP: objection.
THEWITNESS: I would consider that to be moderate hypertension.
BY MS. SPERANDO:
Q. On what basis, doctor, in your report, do you make the statement that it was your understanding – I am referring now to Page Two, the fourth full paragraph, where you say it was your understanding that Mr. Peacock's hypertension was aggressively treated in the follow-up office visits after be was released from the hospital, if you did not have the records of Doctor Boulware's visits?
A. I read a deposition from I believe it was either Doctor Biblo or Doctor Boulware – that questions were asked about the treatment of the hypertension, which implied that the medications had been changed and attempts were made to control it

Page 101

and that he had been seen relatively frequently.
Q. Can you tell us, with a blood pressure of 140 over 86 what Mr. Peacock's blood pressure. would have been – if it had started out at 140 over 86 what it would have been after two hours of vigorous tennis?
A. That would be almost impossible to say. Again, I don't know how Mr. Peacock plays tennis. And realizing it is – singles tennis is only a moderate activity compared to his stress test, I would say that I wouldn't expect it to be as high as –well, I don't know. I don't know what it is. I think that's very hard to say.
Q. I may have asked you this before, but I am not sure. Do you have an opinion as to whether there is any relationship between – in a patient such as Mr. Peacock -- between the blood pressure and ischemia or a malignant arrhythmia?
MR. HUPP: Objection. In terms of what? If you can answer it, go ahead; because I think that's a little broad,
THE WITNESS: I am not so sure what you mean by "relationship." In other words, if you want a hypothetical situation –
BY MS. SPERANDO:
Q. In other words, is there any relationship

Page 102

between the degree of blood pressure or the rate of blood pressure and precipitating a malignant arrhythmia or an ischemic event?
A. I am not sure exactly what you are asking me. You are talking about specifically Mr. Peacock?
Q. Correct.
A. And you are talking about whether his blood pressure precipitated an event?
Q. No. Can it? I mean, like the higher the blood pressure – is there a correlation between blood pressure in a person with his anatomical status and – a correlation between the blood pressure and precipitating a malignant arrhythmia or an ischemic event?
A. There could be. Again, it would depend specifically on whether you are hypothesizing that his blood pressure reached some malignant level.
Q. Well, then, that's basically the question. At what point does an increase in blood pressure pose a threat or a risk to precipitating a malignant arrhythmia or an ischemic event in a person with Mr. Peacock's anatomical status?
A. There is no specific number that I could pin that on.
Q. Well, would you agree in general – just in

Page 101

general – that the higher the blood pressure, the greater the risk of precipitating a malignant arrhythmia or an ischemic event?
A. No, because blood pressure naturally has to go up with exercise. And where Mr. Peacock was doing exercise – albeit singles tennis, not something extremely strenuous – the elevation of blood pressure that goes along with that would be expected. So, you would expect some elevation of blood pressure. It is pretty clear that people who do exercise elevate their blood pressure and that that isn't always a risk for any event in and of itself.
Q. I am not talking about – I don't want to play semantics with you. I want to really be very definite about what I am asking here. I am not saying that it is always going to result in a cardiac event, I am simply talking about in terms of prognosticating and focusing in on risk.
I would assume just as a generalization that if a person with coronary artery disease is sitting down, not doing anything, that his risk of a sudden cardiac event is less than it would be if he were running a three-minute mile. So, I am simply asking you with regard to high blood pressure if there is

Page 104

any relationship – the greater the blood pressure, the greater the risk of precipitating an event as we have described it.
A. The way you have asked it sounds as if you are saying there might be a linear relationship in this thing. And there is no linear relationship there in terms of increased risk. In fact – and he demonstrated quite nicely that he did 12 mets of exercise on a very vigorous stress test and raised his blood pressure to appropriately high level and performed quite nicely.
I don't think his raising his blood pressure to those levels that he has demonstrated capable of doing in and of itself presents any risk to him. He is doing six or seven mets of activity playing tennis. He walked 12 mets on a treadmill. I don't think that you can correlate a blood pressure rise in one activity necessarily to another, But, I don't see the relationship, necessarily.
Q. I am not asking you for necessarily a linear relationship. And let us for the moment ignore the stress test results. And let's focus specifically on his anatomical status. In general, is it fair to say that a person with the anatomical status of Mr. Peacock, which we have already described in

Page 105

general -- that the higher the blood pressure, the greater the risk for a cardiac event?
MR. HUPP: objection. Asked and answered. Go ahead.
THE WITNESS: Again, let me separate this a Little bit for you.
If in general Mr. Peacock runs high blood pressures his entire life, is that independently a risk for coronary artery disease and the effects thereof? Yes. That is a well known fact.
If you are asking me specifically is he at risk of elevating his blood pressure doing a specific event to a point where it presents a risk of a cardiac event – is that what you are asking?
MS. CARULAS: I am going to object, because I think this exact same question and answer took place about an hour ago. I heard the exact same discussion about chronically and one episode.
MR. MARTIN: The question has never been answered point blank.
MR. HUPP: Yes, it has.
MS. SPERANDO: Let's just try and

Page 106

get the answer again.
BYMS. SPERANDO:
Q. Specifically, with regard to correlation between high blood pressure and a person with this anatomical disease and precipitating a cardiac event, is there any relationship whatsoever?
A. Not that I am aware of.
Q. So, you are not concerned whether his blood pressure would be normal versus extremely high –
A. Define "extremely high."
Q. Well, you said there was no relationship. That's why I have to –
A. You asked me if there was a linear relationship.
Q. No. I specifically said it doesn't have to be a linear relationship.
A. I said a not linear –
Q. I think the record will reflect that – whatever you mentioned. It doesn't have to be linear.
Is there any relationship between high blood pressure and precipitating a cardiac event? Or is blood pressure simply not a factor?
MS. CARULAS: objection. Asked and answered.

Page 107

THE WITNESS: If you are looking at a general risk factor of blood pressure potentially causing end-organ damage, i.e., a myocardial infarction cardiac event, there can be a relationship. In people with malignant hypertension, that's the definition. High blood pressure caused an event.
However, in the general population, such as Mr. Peacock, even with his defined anatomy, I am not aware of any specific data, any literature, any anecdotes, any patients of mine who, because they exercised and got high blood pressure specifically, had a cardiac event. I am not sure you are getting *the* answer you are looking for.
BY MS. SPERANDO:
Q. So, then, boiled down to the essence of *your* opinion, the degree of blood pressure has no relationship to a sudden cardiac event in a person with the anatomical status of Mr. Peacock, whether it is normal, low, or high?
A. That's not what I said. There is a level of malignant hypertension in anybody, if they are capable of reaching it, at which it could cause a myocardial event.

Page 108

Q. And what is that level, sir?
A. It is different in everybody. And in Mr. Peacock, I would suggest that that is very, very high.
Q. Such as?
A. In general, I limit patients whose blood pressure reaches 250 over 120.
Q. Do we know whether Mr. Peacock's blood pressure when he was exercising without the Procardia got anywhere near 250 over 120?
A. No one would ever be able to give you any idea of that, because we also don't know whether he took Atenolol, Lisinopril, or aspirin the day of his test, either.
Q. And assuming he took those drugs on the day of his test, and assuming he took them the day that he died, how would that impact your opinion, assuming he did?
A. Assuming he took those drugs at those times, I doubt very, very seriously that he could have reached a level of malignant hypertension that would have had end-organ disease, i.e., event specific cardiac arrest -- myocardial infarction.
Q. The significance of the autopsy findings -- let me go one step before that -- do you have an

Page 109

opinion within a reasonable degree of medical probability as to what caused Mr. Peacock's death?
A. I have read the coroner's report and the emergency room report. And I would say within a reasonable probability he had sudden cardiac death, probably arrhythmic.
Q. Cardiomegaly was noted, a heart weight of 540 grams on autopsy; is that right?
A. I believe that's *right.* I am reading from Doctor Herskowitz's report -- heart, 540 grams.
Q. Doctor, do you know Doctor Biblo?
A. No. I don't know him.
Q. Have you ever met him?
A. No.
Q. Have you ever had any professional relationship with him whatsoever?
A. None.
Q. Have you had any professional relationship whatsoever or any kind of relationship with any of the physicians who have been involved in this case?
A. Let's see. I don't know any of the University physicians personally. I have talked to Doctor Effron, who did the stress test, on one occasion in the past where I referred a patient to his rehab program. You are talking everybody involved as --

Page 110

Q. Doctors Effron, Lesnefsky, Bodware, Biblo --
A. No. I don't know any of those people.
Q. Since you have been retained in this case, have you spoken to any of those physicians, including Bodware and Biblo, with regard to this matter?
A. No; never had any contact with any of those physicians.
Q. Have you spoken With anyone other than Mr. Hupp with regard to this matter?
A. No. No,
Q. Do you know Doctor Herskowitz or know of him?
A. I have met Doctor Herskowitz.
Q. When did you meet him?
A. I met Doctor Herskowitz in Chicago. Let me think here. In 1995, maybe. I am trying to think when I was in Chicago. Let's see. It may have been 1996, actually, 1996, I would guess, so last year.
Q. What was the nature of the meeting?
A. I was introduced to him. And he was at a meeting. And my wife was at a meeting, the same meeting, of the Centers for Ischemia Research, a group I met in Chicago. He was there. My wife is a member of that group. And I met him in the hallway.
Q. And did you have any conversation with him at

Page 111

that time?
A. Shook his hand and said, "Nice to meet you."
Q. Have you ever attended any lectures he has given or --
A. No.
Q. Do you know what Doctor Herskowitz's area of specialty is?
A. I honestly don't know a lot about him. But, I know he works for this McSPI group, which is a largely -- as far as I know, a group of anesthesiologists who do ischemia research on bypass surgery patients. I know that he works for them. I don't one hundred percent know his capacity. But, I understand he is not -- he doesn't do any surgery or anesthesia. He is a researcher or he is completely employed as a research by the organization.
Q. Do you know what his reputation is in the field of cardiology?
A. I don't, honestly. I would assume he is a published guy or a researcher, because that is the nature of that group. I understand he is a cardiologist. I should say that. So, I don't know that he does cardiology -- or what he does cardiac wise. I don't know clinically what he does, frankly. I know that he does research.

Page 112

Q. All right. Now, referring to Page Four of Doctor Herskowitz's report, *the* third full paragraph, can you tell me -- he goes on to describe the findings of the coroner's report.
Do you know how the coioner's findings were different from what the physicians knew who were taking care of Mr. Peacock prior to Mr. Peacock's death?
A. The coroner's finding, being this stuff before the slides, described several narrowings in the coronary arteries that were 70 percent in the left anterior descending -- is that right? Let me go back -- left anterior descending coronary artery and the right coronary *artery* showed focal distal luminal narrowing of 70 percent, which was different than what the catheterization would have implied.
Q. What did the cath imply?
A. The catheterization implied that the left anterior descending was essentially free of significant disease and that the right coronary artery had several 40 percent narrowings. And then in the circumflex coronary artery it says focal distal narrowing of 90 percent, which would be relatively consistent with what was shown on the cathreport.

Page 113

Q. Any other differences?
A. Certainly, a thickness of 1.8 centimeters anteriorally and 2.5 centimeters at the septum is different from the reported thicknesses by echocardiography.
Q. In what way?
A. I *think* we showed that the echo indicated that the thickness of the posterior wall was 1.6 centimeters, and that *the* septum was actually 14 centimeters.
Q. Were the differences between what was shown on autopsy versus what the physicians who treated Mr. Peacock knew at the time they were treating him -- were they in any way significant in terms of how you believe Mr. Peacock should have been treated?
MR. HUPP: objection.
BY MS. SPERANDO:
Q. Do you understand the question?
A. Go ahead and restate,
Q. If the physicians who were treating him knew what the coroner knew on autopsy, do you *think* that that should have changed their treatment of him in terms of a proscription with regard to vigorous exercise such as tennis?
MR. HUPP: objection.

Page 114

Relevancy.
MS. CARULAS: objection.
THE WITNESS: No. I don't think so.
BY MS. SPERANDO:
Q. Okay. Do you *agree* with what Doctor Herskowitz has said in Paragraph Two regarding the slides, Page Fou?
A. I didn't review the slides. And I haven't seen anybody else review them.
Q. So, with regard to his findings, he says, "Both of these findings are unusual and are consistent with arterial injury patterns seen in malignant hypertension."
Do you *agree* with that statement, assuming his findings are correct?
A. I don't know. I am really not an expert in that area.
Q. Okay. Now, with regard to the next page, assuming that there was, in fact -- Paragraph Four, I am referring to the second sentence -- Doctor Herskowitz notes: "There is a moderate degree of interstitial fibrosis, or scarring..."
Assuming that that was, in fact, correct, do you agree with his conclusion, which is as follows:

Page 115

"...that is most likely a consequence of longstanding hypertension." Do you agree with that?
A. I am losing you here. Show me where it talks about scarring.
Q. Paragraph Four.
A. Paragraph Four.
Q. And the second sentence: "There is a moderate degree of interstitial fibrosis..."
Do you see that?
A. Okay.
Q. "...or scarring." And then he makes the conclusion that it is most likely a consequence of longstanding hypertension.
Do you agree with that?
A. Again, it is not an area of expertise. But, it is not necessarily my understanding that longstanding hypertension causes what you would by lay terms call scarring. Certainly, myofibrullar disarray and other myocardial fibrillar changes can take place with hypertension. But, I am not aware that this is what he is talking about.
Q. Well, he makes the conclusion that the interstitial fibrosis was a consequence of longstanding hypertension. You don't agree with that?

Page 116

A. I don't know that that is true or untrue.
Q. He then talks about his microscopic findings. Assuming that they are true, he then makes a conclusion which -- at the fourth line up from the bottom of paragraph four -- his conclusion is as follows: "These microscopic findings represent clear evidence that Mr. Peacock suffered small amounts of permanent myocardial damage over an extended period of time."
Assuming that his microscopic findings, which as I understand it you cannot comment on, are correct, do you agree with his conclusion that they represent clear evidence that Mr. Peacock suffered small amounts of permanent myocardial damage over an extended period of time?
MR. HUPP: objection.
THE WITNESS: I don't know.
BY MS. SPERANDO:
Q: Okay. Do you *agree* or disagree with his conclusion that, "The more recent myocytolytic lesions are consistent with the clinical findings of a non-Q wave MI approximately three weeks prior to his death"?
A. Again, I would have to review these slides with a pathologist and look at the whole thing

Page 117

clinically to tell you what was there and what it means. Under this circumstance, I have no opinion about whether that is correct, incorrect, or consistent.
Q. Okay. If he did have myocytolytic lesions, assuming that he did, would they be consistent with a non-Q wave MI?
A. Basically, myocardial infarction equals myocytolytic lesions. They are the same words, basically. So, I suppose those are consistent.
Q. Okay. Doctor Herskowitz concluded that Mr. Peacock, number one, had severe coronary artery disease. Agree or disagree?
A. I disagree with that.
Q. How would you define his coronary artery disease?
A. I would define his coronary artery disease as mild to moderate disease, *again* realizing that the definition is not just pathologic, not just anatomic, i.e., catheterizations, but also clinical. And I would say that he had some evidence of -- my conclusion is he had mild coronary artery disease.
Q. Focusing strictly on the anatomic coronary artery disease, how would you describe it?

Page 118

A. Focusing on what?
Q. Anatomic coronary disease --
MR. HUPP: You *are* saying after death, now?
BY MS. SPERANDO:
Q. Yes.
A. That is pathologic. That is not anatomic.
Q. Okay. Pathologic.
A. I would say he had moderate disease. That is clearly not severe disease.
Q. He concluded that Mr. Peacock had severe hypertension with end-organ injury. Agree or disagree?
A. I disagree.
Q. I thought you agreed with that.
A. Severe hypertension has a defiition. It includes a diastolic blood pressure in the range of 120. He has hypertension with end-organ injury.
Q. You would disagree with the severe part?
A. Yes. That's a defied term. And that defines a level of hypertension that I don't think has been demonstrated here.
Q. Number three, you disagree that he suffered a non-Q wave MI on 5/8. We have already gone through that.

Page 119

Do you agree that he suffered ischemia induced tachyarrhythmia and syncope on 5/8/94.
MR. HUPP: And the Q wave?
BY MS. SPERANDO:
Q. He definitely disagrees with the non-Q wave.
A. Do I think he suffered ischemia induced tachyarrhythmia and --
Q. And syncope. We will start with the suffered ischemia induced tachyarrhythmia.
A. I disagree.
Q. Why do you disagree?
A. Number one, I have no evidence he had a tachyarrhythmia at all on 5/8/94. Number two, I don't have any evidence of ischemia. In fact, I have evidence by stress testing that he didn't have ischemia when he was pushed to the upper limits of his exercise capability.
And, certainly, singles tennis is about half that strenuous. I would say very likely he did not have ischemia at that level of exercise.
Q. What in your opinion to a reasonable degree of medical probability caused the arrhythmia on the day of his death?
A. On later -- 5/29?
Q. Yes, sir.

Page 120

A. Likely the cause of his death is sudden death or arrhthmiagenic.
What caused it? That's a good question. There are a lot of possibilities: Where he had, again, a stress test that didn't show me ischemia, I don't think it is ischemia. I am at a bit of a loss to tell you specifically what it could be.
Q. So, given everything that you know, including from the autopsy, you cannot tell the jury within a reasonable degree of medical probability what caused the arrhythmia which caused his death?
A. Right. Again, I haven't looked at these slides. And I certainly haven't looked at them with the aid of an expert, someone who looks at slides and *can* help me interpret the clinical scenario. That might be of some help.
On the other hand, I don't see any demonstration -- certainly, by his description, there is no acute thrombus in the blood vessels. That would be the definition of an ischemic event. That is clearly not here. So, I would have to tell you I don't see ischemia in what he has even shown me here even with the slides.
Q. Okay. What is the differential with regard to the possible causes of an arrhythmia on 5/29?

Page 121

A. Certainly, he is a guy who has left ventricular hypertrophy, so he does run some elevated risk of arrhythmia, because he has left ventricular hypertrophy. Ischemia is in the differential. I don't think that is what caused this, but it is in the differential.

Again, a neurocardiogenic event could certainly have taken place. And occasionally, they do have fatal consequences. He is taking medications. I don't know, again, his entire response to those, because we-don't know whether he has taken them or not on his exercise test. I'm not one hundred percent certain what could cause it.

Q. Okay. Out of all of the potential causes that you have outlined for us, what do you believe was the most likely?

A. In his case, I have to think he had an arrhythmia somehow related to left ventricular hypertrophy or other idiopathic, unknown conduction disease -- again, that is not predictable from the vast amount of testing he had.

Q. With regard to the syncopal event, what is *the* degree of likelihood that it was caused by an arrhythmia?

A. The degree of likelihood – which one, *the*

Page 122

first one?

Q. The syncopal event; comt.

A. In general, with young patients, the probability that a syncopal event is arrhythmic is prettylow.

Q. I am talking about this young patient with his degree of disease.

A. Yes. Again, arrhythmia is quite low. Certainly, none was documented. Certainly, he recovered without any maneuver to change a rhythm. He didn't get shocked. He didn't have any other event to help him overcome that. So, I would say that the probability it is arrhythmia was low.

Q. How low?

MS. CARULAS: Note my objection.

THE WITNESS: Statistically speaking, arrhythmia as causing syncope in this kind of patient, in a patient this age, this active, probably less than two or three percent, I would think.

BY MS. SPERANDO:

Q. Are there any sources you are relying on for that opinion, doctor?

A. I don't have any specific in front of me. But, when you look at the literature in the 1980's, there are a couple of pretty good syncope studies. And I know they were put together as a Med analysis. In that analysis, certainly, age factored into the statistical probability that arrhythmia was at work.

And so, based on age, plus the fact that this guy was a vigorously exercising patient with no symptoms, I would have to say that statistically the probability is quite low, The patient is under 70 years old, if I am remembering correctly.

Q. I am not talking about the general population or people in general. I am talking about this patient, with his anatomic and pathologic disease, what is the statistical likelihood of his having suffered the syncope as the result of an arrhythmia?

MS. CARULAS: objection.

THE WITNESS: It is the same low probability. This is an asymptomatic guy.

BY MS. SPERANDO:

Q. And if you could give me the names of the articles to which you are referring that would support that.

A. Right offhand, I don't know that.

Q. Can you tell that to your attorney after you get back to your office?

Page 124

A. Sure.

Q. Is what you are saying then as I understand you that it is your opinion within a reasonable degree of medical probability that Mr. Peacock suffered a syncopal event on May 8 as a result of a vasovagal reflex and then within a reasonable degree of medical probability, suffered a sudden death on May 29 as a result of a malignant arrhythmia?

A. That is as best as I can put that together.

Q. Does that make sense to you, doctor?

MR. HUPP: objection.

THE WITNESS: He died from one event. He had nothing near death from the other event.

BY MS. SPERANDO:

Q. I am sorry. Say it again.

A. He died from the second event. That is a big difference than not dying with a syncopal event earlier. So, to try to relate those two incidents? I have trouble doing that.

Q. What effect, if any, does the fact that he happened to be exercising when he experienced the syncopal event – in terms of the cause of the event as you have described it, what significance if any does the fact that he was exercising during both

events, playing tennis, have on your opinion?
A. Well, intuitively, you would say, well, he is exercising and had events. But, when you look at the evidence and you weigh the evidence of what happened there, I can't put those two together as related events, necessarily.
Q. Doctor, are you saying -- come on. Are you saying that a man with this kind of left ventricular disease, coronary artery disease, who happens to be playing tennis on May 8, faints as a result of a vasovagal reflex, having nothing to do with his heart, and then three weeks later happens to drop dead while playing tennis as a result of a cardiac event, and it is simply a coincidence that it is three weeks later? There is no connection?
A. Number one, I don't know whether there is a coincidence or not. I don't know that they are related.
But, I have found no connection. The fact that he dies three weeks later, the fact that he had an event three weeks later – whether it was precipitated by – and caused his death was clearly different than the event that was precipitated *three* weeks earlier, because he didn't die three weeks earlier. There is clearly a difference.

I know you are trying to show me that there is a similarity. But, I think you have to appreciate that there is a huge difference here.
Q. I don't appreciate that there is a huge difference.
A. He died in one, and he didn't die in the other.
Q. Trust me. I understand that. And that's why we are here.
But, in terms of the physiology or what is causing someone to faint, I think it is just but for the grace of God that he didn't die the first time around, and he simply fainted. I mean, I see –
A. So, you want his first event to be coincidental that he didn't die, but the second event is not coincidentally related to the first, I don't see that.
What I am telling you is that I have evidence that the first event occurred without evidence of ischemia. And, certainly, a prognostic test following that event predicted that he should do well from all standpoints. And defining his anatomy and physiology as we do with stress testing, it was not predictive of these things -- that now he suffers a second event that -- you are asking me is

it clearly related to the first? I have no evidence of that.
I have to answer you scientifically, not intuitively. And scientifically, I have no evidence of that event being related. Does that make sense?
Q. No, it doesn't, quite frankly.
MR. HUPP: Let's not get argumentative here.
BY MS. SPERANDO:
Q. Pathophysiologically, what causes someone to faint, assuming it is an arrhythmia, is the same thing that is causing someone to die, assuming it is an arrhythmia – is it not?
A. Number one, you have assumed it is an arrhythmia. That's a pretty bad assumption.
Q. I am asking you to assume that it is an arrhythmia. The bottom line is, an arrhythmia can cause someone to faint and not to die, correct?
A. That can happen.
Q. Okay. Now, freeze frame the action right there. If someone has an arrhythmia which causes him to faint, what is happening under those circumstances versus the basically same arrhythmia that is causing him to die?
Why under certain circumstances – in other

words, physiologically, what is happening to the heart that under the first scenario he just faints and then recovers and in the second scenario he dies? Can you tell us that?
A. Certainly. What I am telling you is that I don't think what happened the first time around was what happened the second.
Q. I understand that.
A. What I have stated is that statistically speaking someone presents to an emergency room dead like this is likely arrhythmiagenic or sudden death. I am not so certain when that arrhythmia was precipitated.
In other words, could he have had two syncopal episodes that were related for the same reason, neurocardiogenic or otherwise? Yes. He could have had that. Could something have been different about the second episode that caused him to die? That's entirely possible.
What I am telling you is that the fact that he died is not necessarily related to the fact that he passed out. I don't know that it is or isn't. But, I don't have any scientific evidence that it's related.
Q. But, that wasn't the question I asked you.

Page 129

A. I understand what you are saying.
Q. Let's just focus on what I asked you, doctor. Do you understand the question?
A. I think so.
MR. HUPP He answered the question.
THE WITNESS: You asked me if --
MR. HUPP Let's reask it. Let's be fair. Go ahead.
---o0o---
Thereupon, a previous question was read back by the court reporter.
---o0o---
MR. HUPP: Now, he just answered that. There was an answer to that question.
MS. SPERANDO: I didn't understand it as being responsive.
MR. HUPP First of all, was there an answer to that question?
(Brief interruption.)
MR. HUPP There was an answer to that question? Then I object. It has been asked and answered. I don't know if you can

Page 130

make your answer any clearer, but go ahead.
THE WITNESS: I think I know where you are coming from with this. If someone has an arrhythmia that they don't die from, that they actually do recover from, in general, that requires some manipulation to do that ;-'electric shock, some sort of pacing, or something – a maneuver. And that's the difference, generally, between dying and not dying.
If someone has an arrhythmia that they pass out from, that they are syncopal from, and nothing is done about it, as in, you know, your first hypothesis, they are probably going to die.
Now, you are asking me are these events related? Or are they likely related? What I am telling you is I have no evidence that they are likely related. Intuition is a lousy way to tell me that they are related.
And I have to tell you that death and syncope are not the same thing. Sudden death and syncope are not the same thing, either. And sometimes I think that is a little confused here.

Page 131

BY MS. SPERANDO:
Q. So, then, is it fair to say that if Mr. Peacock had been exercising under a monitored condition and had experienced the same arrhythmia that you believe he experienced immediately prior to his death, that if there had been monitoring and intervention, that there was a likelihood that he could have been revived?
MR. HUPP: objection.
THEWITNESS: Number one, I am not so sure he had an arrhythmia. I am saying that, statistically speaking, a patient that shows up like Mr. Peacock, dead in emergency room, probably had arrhythmia.
If he had an arrhythmia like that and he were under monitored conditions, there is some chance that you would revive him. Statistically speaking, it is not 50/50, but at least you have a chance.
BY MS. SPERANDO:
Q. Okay. Let's please continue With Doctor Herskowitz's report. We are on Page 5 --
THE WITNESS: one break, red quick.
MR. HUPP: okay.

Page 132

---o0o---
Thereupon, a brief recess was taken off the record.
---o0o---
BY MS. SPERANDO:
Q. In the last paragraph on Page 5, doctor, the first sentence, it says, "Mr. Peacock's clinical presentation on 5/8/94 was likely caused by the transient occlusion or severe stenosis of his right CA precipitated by a plaque rupture."
Do you agree or disagree with that?
A. I disagree with that.
Q. Because?
A. certainly, again, I haven't looked at the slides. But, he describes this athrosclerotic plaque in the RCA that shows a healing clot. That healing clot is certainly part of coronary artery disease, but doesn't necessarily cause ischemia when it ruptures. It certainly can. Andthat's certainly a high possibility.
But, again, there is no evidence that he had a myocardial infarction or evidence of ischemia. Plaques rupture pretty frequently in people and don't cause heart attacks and don't cause events, even. So, I don't know how – that's a real

stretch, without any evidence.
Q. So, basically, your opinion, then, is based on that fact that these CPK enzymes and MB bands do not, in your opinion, reflect ischemia?
A. Correct. And he certainly didn't have any typical symptoms of ischemia. Syncope is not a typical symptom of ischemia.
Q. But, it is fair to say that a syncopal episode can very well be caused by ischemia without showing those, quote, unquote, typical signs and symptoms of ischemia; isn't that right?
A. That would be pretty unusual to do that.
Q. But, it is possible?
MR. HUPP: objection.
MS. CARULAS: objection.
BY MS. SPERANDO:
Q. Or are you saying it is not possible?
A. I haven't Seen it, but –
Q. The question is, is it possible to have a syncopal episode based on ischemia without the typical signs and symptoms of ischemia preceding it?
MS. CARULAS: objection.
MR. HUPP: objection.
THE WITNESS: I would guess that it is.

BY MS. SPERANDO:
Q. Second sentence. I take it you disagree with that: "The ischemia which ensued triggered an episode of ventricular tachycardia, causing sudden syncope."
You disagree With the third sentence, I take it; is that right: "Upon clinical investigation --" we have already gone through that?
A. Right.
Q. "The clot in the RCA, if it ever completely occluded the vessel, likely spontaneously lysed, allowing the heart muscle to be reperfused, precluding the development of a large transmural inferior wall MI."
A. I mean, it is an interesting statement, because right there he admits there is no evidence that it ever completely occluded the vessel. And, on the basis of an isolated plaque rupture, to draw the conclusions that are drawn here of ischemia, clinical scenario, is a real stretch. So, I would not say -- I would say that the word "likely" is completely out of bounds there.
Q. Do you believe that Mr. Peacock suffered microscopic heart muscle cell injury evident at autopsy?

A. I would have to go over those slides myself with a pathologist to know.
Q. So, you have no opinion on that?
A. No opinion.
Q. We have discussed the rest of that. Okay.
He says, "It should be noted that both the echocardiogram and the left ventriculogram were performed one and two days, respectively, after hospital admission."
Do you agree with that?
A. It looks like that's when they were performed. Yes. I didn't date them.
Q. And then the conclusion: "And, therefore, if transient wall motion abnormalities were present on the first day of admission, they would have been missed."
A. It's possible that if transient wall motion abnormalities were there, they may not be there two or three days later. That's possible, if they were ever there.
Q. The second sentence: "Based on the autopsy findings, the nuclear stress imaging results, which revealed," quote,`a small persistent perfusion defect in the inferior segment on the short axis images,' unquote, according to Doctor Herskowitz,

"may reflect true injury in the inferior wall, rather than the artifact of diaphragmatic attenuation noted in the report." Agree or agree?
A. It is unlikely. And the reason is quite simply – and again, where somebody looks at – as somebody who does this frequently, reads these sort of tests frequently – we know that the finding of a defect in one set of images is not consistent -- is not diagnostic for anything and that it is very unlikely that that represents injury. Much more likely that it is diaphragmatic attenuation.
Q. It is possible that it reflects true injury in the inferior wall?
MS. CARULAS: objection.
MR. HUPP: objection.
THE WITNESS: within the limits of the test?
BY MS. SPERANDO:
Q. Yes, sir.
A. The test – that finding doesn't reflect injury. Could there be injury there that you don't see?
Q. I am talking about what he is refemng to. Let's just focus in on his opinion. I would like to know whether you agree or disagree.

So, he is talking about – "a small persistent perfusion defect in the inferior segment on the short axis images may reflect true injury in the inferior wall."

Is it possible that that is, in fact, what occurred?

MR. HUPP: objection.

THE WITNESS: YOU are asking me to change the definition of a test. The definition -- this is a test. How you interpret the possibilities beyond that test are different than what you are asking me. This test does not reflect any true injury to the inferior wall. The possibility that there is injury there – is that what you are asking?

BY MS. SPERANDO:

Q. Yes.

A. Is there possibly injury there? In any stress test it is possible that there is injury somewhere that is not reflected by the image. In this test, though, as defined, the answer is no. *See,* he has changed the definition of the test here.

Q. We have already discussed the second paragraph where he says his death was likely due to ischemia

precipitated by rigorous exercise.

It is your opinion that the arrhythmia was not precipitated by ischemia, correct?

A. I don't have any evidence of that.

Q. Let's go to the second full paragraph, the fourth line, where he says, "These types of lesions have been clearly shown in many studies to be the most prone to rupture spontaneously," referring to the lesions he had mentioned in the previous sentences. Agree or disagree?

A. Number one, I haven't looked at the slides, so that I can't tell you how thick this fibrous cap was and how to compare that. But, in general, lesions that have thin fibrous caps and lots of lipid in them – any plaques are prone to rupture, number one. It is difficult to define which ones are most prone. But, certainly, these soft plaques can rupture.

Q. The next sentence: "Another clear precipitating factor for plaque rupture is increased shear force associated with hypertension."

Agreeordisagree?

A. In general, hypertension can be a precipitating force for plaque rupture.

Q. Next sentence: "At the time of rupture of the

plaque within the vessel lumen, the surface of the plaque rupture develops a clot which begins to build and further encroach on the lumen."

Agree or disagree?

A. In general, that's the theory behind myocardial infarction.

Q. Next sentence: "In the setting of extreme exercise, acute ischemia may ensue even if the vessel does not completely occlude."

Agree or disagree?

A. It can, but that is less likely. But, that's right. You can still have ischemia.

Q. Next sentence: "The clot forming in the vessel may either transiently occlude the vessel and then spontaneously reopen or completely occlude the vessel and cause a large, transmural MI."

Agreeordisagree?

A. In general, in patients who have an MI, that can happen, although a complete occlusion of the vessel doesn't necessarily cause large transmural MI. It is not always the outcome.

Q. Going not to the next sentence, but the sentence after that: "Finding only a 40 to 50 percent lesion in the posterolateral branches of the RCA at cath is entirely consistent with the autopsy

findings of a healing thrombus."

Agreeordisagree?

A. That's consistent.

Q. Next sentence: "Spontaneous lysis of clots within coronary arteries typically demonstrate a relatively low grade underlying stenosis."

Agreeordisagree?

A. Almost all stenoses that rupture – almost all of them lyse. So, it doesn't necessarily mean it is a low grade lysis. A high grade stenosis can also have spontaneous lysis. In general, most arteries after a complete Q wave myocardial infarction are open after 24 hours regardless of the degree of stenosis.

Q. Next sentence: "The lipid-rich nature of the coronary stenosis and the shear forces generated in the vessel are what make the coronary prone to plaque rupture, not the severity of the stenosis."

Agreeordisagree?

A. That's a pretty big simplification, but certainly those are among considerations for why plaques rupture.

Q. "While receiving medical care from Doctor Boulware, Mr. Peacock's blood pressure was never under control for any significant length of time."

Page 141

Agreeordisagree?
A. Well, Mr. Peacock was only under Doctor Boulware's care for intermittent periods. And during those times, he worked hard to control that blood pressure. "Significant length of time," I have trouble with, because I am not so sure Mr. Peacock followed up with Doctor Bodware for a significant length of time,
Q. Well, you do know that he was seeing him from what, '86 to '88?
A. Yes.
Q. And then from '93 to '94?
A. So, he had five years in there where he didn't see him. And '93 to '94 is a fairly short period of time.
Q. Okay. During the periods of t h e that Mr. Peacock was seeing Doctor Boulware, did he have his blood pressure under control?
A. For those short periods of time, it was under control only several times – at several sporadic visits.
Q. So, would you agree, then, while receiving medical care from Doctor Boulware, Mr. Peacock's blood pressure was never under control for any significant length of time?

Page 142

MR. HUPP: objection.
THE WITNESS: He didn't receive care from Doctor Boulware for a significant length of time. So, the answer to that is, by your definition, no. You have defined it.
BY MS. SPERANDO:
Q. You wouldn't consider a year a significant length of time?
A. No, not in terms of trying to control this kind of blood pressure.
Q. Two years?
A. I would say two years is a significant length of time. But, during that period of time, there were times where Mr. Peacock's blood pressure was tending toward good control, but at which time he had periods where he was not tolerating the blood pressure medications, for different reasons. So, control is an odd word, I would say. You know, to say that it wasn't under significant control for lengthy periods of time is – you just don't have it to call.
Q. Okay. You would not put any degree of blame on Mr. Peacock for continuing to engage in vigorous exercise such as tennis after his stress test, would you?

Page 143

A. Would I put any – no. I think moderate activity like that is fine for him after that kind of stress test.
Q. Especially in view of the fact that he was told by his physicians that he could engage in that kind of activity, correct?
MR. HUPP: objection.
THE WITNESS: From Doctor Boulware's testimony, I don't have the idea that he was specifically told he could do that. However, what I would say is that, again, tennis is considered a moderately vigorous activity which runs about six mets. This is a guy who exercised to 12 mets. I would certainly have no trouble telling him that that was an okay activity.
BY MS. SPERANDO:
Q. "Even during his hospitalization from 5/8 to 5/11/94, he required multiple and repeated doses of antihypertensives following his cardiac catheterization to control his hypertension and was discharged with a regimen that was found to be inadequate during his first follow-up outpatient visit to Doctor Bodware."
Agreeordisagree?

Page 144

A. That's a simplification. Number one, I don't know what medications he actually received every day. I didn't review those records. And it's very evident that patients have medications withheld before procedures or even after procedures sometimes, so that during a short hospitalization – the only thing I can tell you is that before he was discharged, as I recall, his blood pressure was reasonable. I don't remember what his last blood pressure reported was. But, I don't think it was excessive.
So, clearly, at his first follow-up, assuming he was taking those medicines, his blood pressure was high. Again, I don't know whether he took the medicines or not.
Q. Assuming that he was taking the medicines -- well, the medicines were changed on the first follow-up, were they not?
A. Right.
Q. Presumably because Doctor Boulware –
A. Felt they weren't working; right.
Q. Next sentence: "At autopsy he had clear evidence of end-organ injury secondary to longstanding hypertension."
Agreeordisagree?

Page 145

A. I agree.
Q. Well, whatever his kidney slides showed, you wouldn't have an opinion on, correct?
A. I don't know what his slides showed. It wouldn't surprise me that a gentleman with coronary artery disease had arterial sclerosis in his kidneys. That's a systemic disease.
Q. "He had a remote cerebellar infarct, which, within medical probability, was due to hypertension."
Agree or disagree?
A. That's probably true.
Q. Next sentence: "He had evidence of LVH, both clinically (by echocardiography and ECG) and at autopsy..."
Agree or disagree?
A. Both the echocardiogram and the autopsy detail left ventricular hypertrophy.
Q. Okay. And he says, "...had both thickening of the LV walls grossly and severe thickening of the small coronary vessels, the latter an unusual finding consistent with severe hypertension."
Agree or disagree?
A. I would say it is not an unusual finding. But, thickening of small coronary vessels is

Page 146

consistent with severe hypertension — I take that back. Again, it is consistent with hypertension. Hypertension can be very longstanding and cause this, as Mr. Peacock's was. Again, I don't have evidence as to his hypertension was ever severe.
Q. So, you don't *think* that severe thickening of the small coronary vessels is an unusual finding?
A. No; not with hypertension.
Q. Next sentence: "The microscopic lesions in the left circumflex coronary artery, with hemorrhage into the outside of the vessel wall and the necrosis of the smooth muscle cells in the outer layer of the vessel are consistent with histologic findings of malignant hypertension."
Agree or disagree?
A. Malignant hypertension has a clinical syndrome. So, again, Doctor Herskowitz has chosen to redefine what the definition of "malignant hypertension" is,
I have no evidence that Mr. Peacock ever had malignant hypertension. The fact that he finds microscopic lesions, as he has described, and which, again, I am not an expert in looking at and describing, may be consistent with hypertension. But, I doubt that they are consistent with what we

Page 147

define as malignant hypertension.
Q. Okay. Next sentence: "These findings all suggest that with a high degree of medical probability that Mr. Peacock had poorly controlled hypertension and episodically experienced extreme elevations of blood pressure."
A. These findings have nothing to do with episodically what he experienced.
MR. HUPP: wait a second.
Objection.
THE WITNESS: They certainly are suggestive that he had hypertension. It doesn't tell you *the* degree of control. It doesn't tell you anything about the peaks. It just tells you he had hypertensive heart disease.
BY MS. SPERANDO:
Q. Last sentence there: "...the most likely scenario is that he was experiencing severe elevations of blood pressure during exercise."
Agree or disagree?
A. I think -- I disagree with that. I have clinical evidence from a stress test that that's not true.
Q. Next page. He says here, "Both physicians" —

Page 148

meaning Doctor Boulware and Doctor Biblo -- *"were* responsible to inform the patient that he had biochemical and clinical evidence of a heart attack and that he would have to limit his physical activity during the high-risk, proarrhythmic, post-MI recovery period."
I take it you disagree with that.
A. Absolutely.
Q. Okay. Now, if, in fact, Mr. Peacock had had a non-Q wave MI, at that point, if that was the case, should Mr. Peacock have been engaging in exercise such as tennis?
MS. CARULAS: objection.
MR. HUPP: objection.
THE WITNESS: Again, realizing a non-Q wave infarction is a clinical entity, not something you look at slides to tell, and you believe he had a non-Q wave myocardial infarction, you would advise Mr. Peacock to enroll in cardiac rehabilitation. That's what you would advise him. And you would advise him not to do high level physical activity.
BY MS. SPERANDO:
Q. Such as tennis?
A. Such as tennis, yes. That's moderately high.

Page 149

But, yes.
Q. Okay. If he had had an MI -- he talks here about this high risk proarrhythmic post-MI recovery period. Does that mean that after he has an MI -- assuming he did – that there is a certain period of time after that MIwhere he is especially at risk?
A. There is a certain amount of time after an MI where you are at risk for arrhythmia. And the best clinical scenario or best clinical way to look at that is during the length of hospitalization to monitor a patient.
And where Mr. Peacock had an excellent Holter monitor and no other recorded arrhythmias that I saw, the prediction would be that he would not have a high probability of arrhythmia in his post-discharge period.
Q. Okay, Well, is there statistically speaking a period of time during which a patient who has had an MI is most susceptible to having another MI?
A. Yes; or another arrhythmia,
Q. Or an arrhythmia –
A. Well, the first 24 to 48 hours after an MI, a patient has a high risk of having arrhythmias. Those arrhythmias are not generally considered to be necessarily malignant or life threatening. It is

Page 150

more arrhythmias in the days following that – i.e., 48 hours to even up to seven days – that you would be more concerned about as being predictive of arrhythmic problems.
A patient with a non-Q wave myocardial infarction does run a risk of an event. Arrhythmic events are not usually the events that we associate with non-Qwave MI's. Instead we generally associate ischemic events, i.e., a myocardial infarction, with them. So, they run the risk.
Q. What I am asking you to do, sir, with regard to a non-Q wave myocardial infarction, is answer this: What's the period of time where they run this risk for another ischemic event that's higher than it would be --
MR. HUPP: Asked and answered. Objection.
THE WITNESS: Yes. I probably didn't answer it totally. In the first couple of days you run some risk. But, *six* months, something like that.
BY MS. SPERANDO:
Q. So, if a person does have a non-Q wave MI during that six months and they want to exercise, you would put them in this program where you would

Page 151

monitorthem?
A. The programs are not *six* months. In general, I would put them in a program which lasts approximately 12 weeks, depending on who approves it. And depending on their performance and so on and so forth, you might then do the definitive prognostic test. The definitive prognostic test is a stress test.
He happened to have his stress test very early on, and it was excellent. So, in that sense, even if you thought there was a non-Q wave MI, his prognosis is excellent. It would be awful hard to tell him to -- it would be very hard to prognosticate otherwise, to tell him that he was at significant risk.
Q. "The stress test results were clearly abnormal and suggested ongoing inferior ischemia by ECG."
Youdisagree?
A. Totally disagree.
Q. You disagree about the ongoing inferior ischemia by ECG -- because the ECG was not valid, based on the fact that there was an abnormal baseline to start?
A. His ECG is completely not specific. There is no evidence of ischemia on any of his ECG's.

Page 152

Q. I am talking about the stress test.
A. The stress test ECG's are nondiagnostic. No.
Q. Okay. Then he says, next sentence: "While the ECG changes were not definitively diagnostic in the setting of his underlying ST abnormalities associated with left ventricular hypertrophy, *the* nuclear scan results of a defect in *the* inferior wall were consistent with the ECG changes and should not have been ignored."
Agree or disagree?
A. completely disagree.
Q. Well, let's take it one at a time. Do you *agree* that the nuclear scan results showed a defect in the inferior wall?
A. No.
Q. Where is he getting this from? Have you any idea?
A. In one view -- again, you are mixing definitions. And, certainly, he is clearly way out of line mixing definitions here. A small persistent perfusion defect on the short axis image – so, in one image of multiple imagings, there was a defect, which is generally when you see it only in one view – by definition, I should say an attenuation defect.

Page 153

By definition, to call this ischemia -- this defect ischemia, you have to see it on more than one image. That's why we do more than one. I don't know Doctor Herskowitz's expertise, but this is a clearcut misunderstanding of what a thallium and ECG on stress testing shows. These are way out of bounds clearcut misunderstandings.

Q. We have already gone through the next sentence. You don't feel that the fact that he was pretreated with Nifedipine in any way invalidated the results of the stress test, correct?

A. Correct.

Q. Okay.

A. Again, I think clinically that is a well known phenomenon.

Q. What was the data that they had regarding blood pressure response during exercise - vigorous exercise?

A. What's the data - I am *sorry* -- that who had?

Q. Doctors had.

A. That Doctor Effron had?

Q. Effron, Boulware, Biblo.

MR. HUPP: During the *stress* test?

Page 154

MS. SPERANDO: After the stress test.

MR. HUPP: After the *stress* test.

MS. CARULAS: what is the question?

BY MS. SPERANDO:

Q. Doctor Herskowitz says, "Allowing a patient with uncontrolled hypertension to exercise rigorously without any data as to his blood pressure response during exercise places the patient at unnecessary risk and danger." So, I would like for you to tell me what the data is or was as to his blood pressure response during exercise.

A. The data is recorded right here. His blood pressure response was to go from baseline 150 over 100 to 210 over 100. I can also tell you that there is - again, I don't mean to quote literature - but to tell you that I know there is literature that placing a patient on a stress test with blood pressures even up to levels of 180 over 120 and allowing them to exercise has never been reported, to my knowledge - that there is a complication from that exercise test.

BY MS. SPERANDO:

Page 155

Q. Okay. What literature are you relying on, doctor?

A. I am sure textbook literature. I am sure I could dig that up.

MS. SPERANDO: I am going to ask you to provide that to your attorney.

MR. HUPP: For the record, I am not his attorney. But, okay.

MS. SPERANDO: The attorney who has retained your services in this matter, Mr. Steven Hupp.

MR. HUPP: That's correct.

THE WITNESS: Esquire.

BY MS. SPERANDO:

Q. You note in your report on Page Two in the first incomplete paragraph, "Only nine isolated ventricular atopic beats were noted. Only one couplet of ventricular atrophy was noted, and there were no episodes of tachycardia" -

A. There is a typographic error. "Atopic" should be "ectopic," e-c-t-o-p-i-c. "Atrophy" should be "ectopy."

Q. Okay. What is the significance of those findings to you?

A. It is not unusual to see isolated ventricular

Page 156

beats on a Holter monitor on just about anyone. So, they are insignificant.

Q. And you say in the second *full* paragraph on Page Two, the last sentence: Thallium and Sestamibi radionuclide scintigraphy demonstrated a fixed area of inferior perfusion defect attenuation. The radiology report indicates that this is most likely attenuation"; is that right?

A. That's right.

Q. What was the possibility that it was not, in fact, attenuation, but an inferior perfusion defect?

A. The point is that that represents artifact, period.

Q. "Artifact" meaning?

A. Attenuation or other things. when seen in only one view, it is by definition artifact, meaning possibly diaphragmatic attenuation.

Q. So, there is no possibility in your mind that, in fact, it was not attenuation, but rather inferior perfusion defect?

A. That finding on this stress test, by definition, is not ischemia.

Q. Okay. You say in your report that, "Doctor Warshall's hypothesis of ischemia as the cause of the arrhythmia from which Mr. Peacock died is

Page 157

certainly interesting and agreeably has some potential likelihood"; is that correct?
A. where are we?
Q. That is the last paragraph on Page Two.
A. Okay. Let's see. Okay.
MR. HUPP: what's the question?
BY MS. SPERANDO:
Q. Well, my question is: When you say, "has some potential likelihood," what do you mean?
A. Well, I think that – he died. And at the time of his death we don't know for sure what caused his death. So, we have to think of what is the potential. As I said, in the differential of sudden death, it was ischemia. It is in the differential. There isn't any way to adequately tell you that it is one hundred percent one way or another.
I think that's why we are here today. We don't know exactly what caused his death. But, there is potential that his death – in general, syncopal death or a sudden death -- arrhythmic death has potential to be ischemic. That's in the differential diagnosis. I think in the next sentence is where I come from then to tell you why I don't think it was ischemic,

Page 158

Q. Well, but the words you used, doctor, were, "agreeably has some potential likelihood" Did you not say that?
A. Yes. I said that.
Q. So, that likelihood meaning?
A. Remember earlier I said it is unclear what the physiologic-basis of arrhythmia is.
Q. You said that.
A. And what I am saying is that he says it is ischemia, and certainly, an arrhythmic death has potentially likelihood to be from ischemia. I cannot tell you whether he was ischemic or not. I can't tell you. I believe he was not. I have evidence that he wasn't. However, Doctor Warshall sayshewas.
Q. And you cannot tell us whether it was, in fact, ischemia or not, because fair to say that you cannot rule out ischemia by what you did, by the tests that were done, correct?
A. I ruled out ischemia by those tests. The tests rule out ischemia in terms of a stress test that didn't show ischemia. So, what the likelihood is there – the second part is his own slides don't show occlusive thrombus anywhere. So, I can't tell you -- in fact, they don't show any fresh thrombus

Page 159

at all. That bothers me, that you have an autopsy result that doesn't show any fresh – which is, as far as I know, the hallmark of ischemia, and yet you hypothesize ischemia. I don't have that evidence. In fact, I have evidence that it is not there.
Q. Well, it is fair to say that you cannot rule out ischemia in the sense of saying absolutely that that is not what it was, versus saying there is no evidence for it?
Do you understand the question, doctor?
MR. HUPP: objection to relevancy.
THEWITNESS: I understand the question.
BY MS. SPERANDO:
Q. So, wait. There is no question.
So, it is fair to say, then, that with all of the tests that were done, ischemia was never ruled out as a possibility.
MR. HUPP: objection.
THE WITNESS: Ischemia was ruled out to the extent that those tests can rule it out.
BY MS. SPERANDO:
Q. "here is a difference there?

Page 160

A. Yes.
Q. Between saying --
A. Iunderstand.
Q. And when someone's Life depends on ruling it out, versus to the extent that those tests can rule it out, that could be a very big difference, correct?
A. You have taken a jump. And the jump is to prognosticate based on what you have evidence of. None of those tests prognosticate. So, to the extent that I can rule out that he has ischemia, I have ruled it out. I have to go one extra step. And that extra step is *the* stress test, which prognosticates excellent long term recovery and no evidence of ischemia.
Q. Stress tests cannot rule out *the* possibility of ischemia causing a sudden cardiac death in this patient --
MR. HUPP: objection.
Relevancy.
THE WITNESS: They cannot rule it out. But, they *can* prognosticate that it is a very, very small likelihood.
BY MS. SPERANDO:
Q. Doctor, if someone said to you, Doctor Koch,

Page 161

you have a one percent chance – one percent -- point 05 percent chance of dying suddenly from a cardiac event if you engage in playing tennis, what would your response be?

MR. HUPP: Objection. Relevancy.

THE WITNESS: If somebody told me that?

BY MS. SPERANDO:

Q. Yes.

A. Five in a thousand chances or five in ten thousand chances --

Q. Point 05, not even a percent, but a half of a percent–

A. What would I do?

That would depend on the benefit of playing tennis. If the benefit of doing exercise -- and playing tennis being doing exercise – was to mean my long term probability that I would prevent secondary event – or primary event, actually, in this patient – if I had the probability that routine exercise prevented my long term primary event, I would have to look at five in *ten* thousand the same way as I look at having a cardiac cath, which has a little more risk than that, or a stress

Page 162

test, which has slightly more risk than that.

Knowing what I know as a physician, I would have to say the benefit of exercise, for me, may outweigh that. I would have to think that tkrough, But, you have to understand, the entire philosophy of medicine is a risk versus the benefit. Nothing is risk free. I can't tell you that if you have no risk factors you won't die a sudden death tomorrow or ten minutes from now.

But, I *can* tell you that there are benefits to these things and that the benefit statistically outweighs the risk. And I think that you cannot isolate risk like that.

Q. Well, you wouldn't ever say that the only way that Mr. Peacock could get any benefit from exercising would be by playing tennis, would you?

A. No. But, that's a pretty moderate exercise activity.

Q. Wouldn't you *agree* that briskly walking is also very good exercise for cardiovascular benefit?

A. Right.

Q. And, in fact, aren't all the studies saying now you don't even have to work hard; you can just briskly walk, and you get that exercise benefit?

A. 'What we suggest -- the prescription in most

Page 163

patients is to exercise to 70 percent of their predicted maximum functional capacity; correct.

70 percent of Mr. Peacock's functional capacity is eight and a half mets. Singles tennis is not eight and a half mets, by all definitions. So, I would have no trouble telling him that he coulddothat.

Q. Doctor, the question, once again, is, if a physician said to you, "It is important that you exercise, and you can do that by briskly walking, but if you play tennis you have a point 05 percent chance of having a sudden cardiac death," would you say, "No, I am not going to briskly walk; I am going to take the chance, because I need to exercise"?

MS. CARULAS: Objection.

THE WITNESS: I would certainly weigh the risks and benefits of those two things and probably choose the highest benefit formyrisk

MS. SPERANDO: *your* nose is growing, doctor.

THE WITNESS: I don't appreciate that.

MS. SPERANDO: I am only kidding. That was just a joke, doctor.

Page 164

THE WITNESS: YOU asked me some questions that you fail to understand *the* basic definitions of. And the bottom line here is that if you don't want to understand those definitions, *then* don't ask me *the* questions. Wait.

MR. MARTIN: That's not the question before you. You are not here to –

MR. HUPP: we are over. It is 4:00. It is over. We have agreed to be here until 4:00.

MS. SPERANDO: I have agreed to be here for four hours. It started at 12:15.

THE WITNESS: Keep going.

MR. HUPP: All right. You want to take a break for a couple seconds and cool down?

THE WITNESS: No. "hat's all right.

MR. HUPP: I don't blame you.

MS. SPERANDO: Do you have a comment you would like to make, Mr. Hupp?

MR. HUPP: I was surprised that you said that. I fiid it to be unprofessional.

Page 165

MS. SPERANDO: It was just meant as a joke.
MR. HUPP: You don't know this man. You can't joke with him. You can joke with me.
THE WITNESS: I answered the question that you implied was a lie.
MS. SPERANDO: It's just a joke, doctor.
THE WITNESS: You know how seriously I take that position? I have never done this except once.
MR. MARTIN: Let's go off the record.
THE WITNESS: Let's stay on the record.
MR. MARTIN we'll stay on.
MR. HUPP: Either way you want to do it.
THE WITNESS: I respect what you are trying to do as a professional. I am happy to come here and try to find out the truth and to try to tell you what I honestly think happened. I spent a long time looking at these documents. I spent a long time

Page 166

thinking about these things. I have a lot of clinical expertise in these areas. I do this every day.
You are asking me some questions that, frankly, are a little bit tough to answer in the sense of their *sense* -- in making *sense.*
I am trying to answer them for you, so that they make sense, so there is some truth here.
I am not trying to steer you one way or the other.
I know that sometimes answers aren't exactly what you want. But, your intuitive sense has probably been steered an awful lot by what is in some of these reports. And your intuition is not working here.
BY MS. SPERANDO:
Q. I simply asked you whether if you had a point 05 percent chance of death from specifically exercising from tennis – and you twisted the answer by saying if it meant your long term Survival you would play tennis, completely ignoring the fact that there are other forms of exercise.
A. You didn't ask that question. You are the lawyer. You ask the question you want the answer to.

Page 167

Q. I asked a specific question. And you put into the question that the only form of exercise was tennis. Clearly --
A. That's the only form you asked me about.
Q. But, you specifically said that you would do it if the benefit to doing it outweighed the risk, completely ignoring the many other forms of exercise which do not pose the risk of tennis.
A. You didn't ask me about those.
Q. I subsequently did.
A. No.
Q. As I understand it, I asked you if there were other forms of exercise which you knew you could engage in without harm to you and you were told that you had a point 05 likelihood of death from engaging in tennis – and I understood you to continue to say the same answer: That you would nevertheless weigh the risk and benefits of playing tennis, notwithstanding the fact that there are other forms of exercise –
A. That is not what I answered. I said I would weigh the benefits of exercise for my risk, and exercise giving me more benefit than risk, I would choose my option that way.
MR. HUPP: For the record, he

Page 168

also talked about 70 percent of target heart rate.
THE WITNESS: Right. 70 percent of your functional capacity, which is generally considered to be 70 percent of your maximum heart rate.
MR. MARTIN: For the record, I move to strike the exchange.
BY MS. SPERANDO:
Q. It says here in your report, "A consideration of left ventricular hypertrophy as a substrate with a predisposition to sudden cardiac death is an intriguing hypothesis. However, withing the limits of available studies, this hypothesis lacks a specific predictor." Did you not tell us before that, in fact, left ventricular hypertrophy is a predisposition to sudden cardiac death or *can* be –
A. Yes. Right.
Q. When you say this hypothesis lacks a specific predictor, what are you talking about?
A. I am talking about an electrophysiologic study to predict sudden death in a person with left ventricular hypertrophy.
Q. In your third paragraph, the second full paragraph on Page Three, you say, "In any event,

Page 169

Doctor Boulware's involvement with this case includes his prior aggressive attempts to control Mr. Peacock's blood pressure."

What do you consider aggressive attempts by Doctor Boulware?

A. Well, he saw Mr. Peacock frequently, number one. Allowing for the fact that when you change blood pressure medications you have to give them somewhere between two and six weeks to even know whether they are going to be effective, his records have stated he had seen Mr. Biblo anywhere from the frequency of a month to even more frequently.

He had, by his deposition, gone out of his way to meet the guy before office hours when it was convenient for Mr. Peacock, So, he went out of his way frequently to get this guy into the office, check his blood pressure, and change his medications. And that's pretty aggressive.

Q. Are you aware of the fact that there were times when Mr. Peacock was not on any medication with the blessing of Doctor Boulware?

A. I am not aware that he was ever on no medication with his blessing.

Q. Did you read Doctor Boulware's deposition where he said on at least two occasions Mr. Peacock

Page 170

came in and was not on any medication because of the sexual disfunction that it caused and that Doctor Boulware was in full agreement with attempting nonpharmacologic measures to bring his blood pressure under control?

A. I recall something of the gist of that conversation.

Q. Do you find that to be an aggressive attempt to control Mi-. Peacock's blood pressure?

A. Yes, because, again, there are lots of approaches to controlling blood pressure. Pharmacologic is primarily the primary one. However, where patients aren't tolerating pharmacologic approaches, it is certainly reasonable to try other things and continue to try to monitor them. That's what I mean by aggressive therapy - frequent visits, a lot of feedback, a lot of attempts at different therapies.

Q. All right. Just so that I have you on the record, doctor, you believe that the exercise of tennis that Mr, Peacock was engaged in - you would consider that to be moderate activity, correct?

A. Yes. I *think* that is defined.

Q. But, you would not have any problem With Doctor Boulware or Doctor Biblo having okayed

Page 171

vigorous physical activity for Mr. Peacock after the stress test; is that right?

A. Correct. Also, noting that practically what they have defined as vigorous exercise was singles tennis - strenuous, vigorous - these are all different words. I think vigorous exercise is not necessarily implied as a level of exercise, only that they defined it as singles tennis.

Q. So, that would be - you would have no problem with his playing tennis two hours at a time, at a clip?

A. No.

Q. when you say vigorous activity, anything more - would you have any problem with his playing or engaging in any activity more vigorous than tennis?

A. I don't have any evidence that it would be harmful to him.

Q. Such as maybe marathon running or crew?

A. This is a guy that, for other reasons, orthopedic and otherwise, you would probably be careful, in terms of hurting himself. But, as far as his heart goes, as far as his cardiac condition goes, I don't have any evidence that he has to restrict himself.

Q. You say, "Well known data indicates that only

Page 172

vigorous exercise has a significant effect on blood pressure management." Where are you getting that from, sir?

A. Oh, boy. In 1991 or 1992, the Centers for Disease Control published some data that indicated that attempts to control blood pressure by exercise alone were probably not adequate and that the only time anybody really showed any modification of blood pressure was when the exercise reached levels that were above low levels of exercise. So, in other words, more intense exercise is required if you were going to hope to get any benefit directly related to blood pressure lowering.

Q. Was that in patients not taking blood pressure medication?

A. I don't know the answer to that.

Q. Certainly, that would be a difference in this particular patient, because he was taking blood pressure medications, correct?

A. No. The idea being that exercise can modulate blood pressure is certainly not new, whether or not somebody is taking blood pressure pills. The idea is that vigorous exercise can lower the blood pressure even in the face of blood pressure pills. However, it is generally not considered to be enough

Page 173

for most people to substitute for pills on a regular basis. And that's why we use medications.
Q. **Okay.** Let's just focus in on what you have written. The sentence is, "Well known data indicates that only Vigorous exercise has a significant effect on blood pressure management." You wrote that, right?
A. Correct.
Q. Now, the question, then, With regard to Mr. Peacock is, could less than vigorous exercise have had a significant effect on blood pressure management for him, given the fact that he was taking blood pressure medication?
A. Yes. What I have attempted to do here is isolate exercise alone. Medication probably doesn't have much to do with it, in the sense that -- what I am trying to say – what I have attempted to do – in this entire paragraph, not just that sentence -- is to say that moderate physical activity is certainly well known to reduce your risk of cardiovascular disease.
And certainly, where we encourage people to walk or do other mild to moderate activity, this is the generalized exercise prescription we give. If we are interested in lowering blood pressure by

Page 174

giving an exercise prescription, that exercise prescription has to be fairly vigorous.
Q. But, if there is blood pressure medication that is being taken in conjunction with the exercise, then can the exercise be moderate in conjunction with the drug to affect the blood pressure?
A. Yes. If your goal is to lower blood pressure by exercising, it has to be vigorous, whether or not you are taking the pills. You are taking the pills to lower blood pressure, too. So, they are two different avenues of attack on this hypertension.
Q. So, if he is taking blood pressure medication, and let's just say the blood pressure is 120 over 80, and now he has *only* moderate activity, you would not expect the blood pressure to be even lower?
A. I wouldn't expect it to go lower; exactly.
Q. So, in terms of moderate activity, in terms of lowering blood pressure, there is no benefit?
A. None that has redly been scientifically demonstrated. That's right. And that's the data that CDC was trying to say – that it requires vigorous exercise to lower it, In the absence of vigorous exercise –
Q. Might as well not do otherwise --

Page 175

A. Moderate exercise is certainly good for you, but it certainly is not going to lower your blood pressure.
Q. Do you have that study from the Centers for Disease Control?
A. I have a slide from it. I am sure I can find that,
Q. Okay.
A. There may actually be a textbook reference to that, too.
Q. At some point in this -- you say on the first page in the second paragraph, "No significant electrocardiographic changes were noted, other than those associated with left ventricular hypertrophy and repolarization abnormality."
In terms of causing an ischemic event, aren't those changes significant?
A. No.
Q. What if any significance to you was the cardiac cath finding of an 80 percent stenosis in the distal circumflex artery in terms of an ischemic event or an arrhythmia for this man?
MS. CARULAS: I'm just going to object, because I think we discussed all of this two hours ago. I mean, he went through

Page 176

the significance of the cath –
THE WITNESS: clearly, coronary artery disease is a common finding in patients. Autopsy studies from Vietnam vets who are 18 years old have demonstrated significant disease. And yet, clinically it doesn't translate into symptoms, disease, events, so that I would say it is clearly an anatomic finding, something to be noted, something to consider if events take place. On the other hand, in terms of that specifically being a cause of something, I just don't have evidence for it.
BY MS. SPERANDO.
Q. If vigorous exercise such as tennis should have been prohibited, who had the duty within the standard to do it, Doctor Boulware, Doctor Biblo, or both?
MR. HUPP: objection. Hypothetical.
THE WITNESS: If it should have been prohibited – I don't have any basis for prohibiting it at all. But, if a person has a reason for which they shouldn't exercise, I would think all of their doctors would discuss

that with them.
Q. Well, okay. I am talking about specifically with regard to this case. The consult as done by Doctor Biblo, and Doctor Bodware being his primary care physician – if, in fact, he should have been told he could not exercise or given the risks of doing it, who had that responsibility?
MS. CARULAS: objection.
THE WITNESS: Any physician that has a relationship with the patient.
MS. S P E W : That's it, doctor.
---o0o---
Thereupon, the deposition was concluded at 4:15 p.m.
---o0o---

CERTIFICATE

STATE OF OHIO, } SS:
COUNTY OF CUYAHOGA.)

I, Priscilla A. Hefner, a Notary Public within and for the State of Ohio, duly commissioned and qualified, do hereby certify that the foregoing witness was first duly sworn to testify the truth, the whole truth, and nothing but the truth; that the testimony then given by him was reduced to writing by means of Stenotype; *that* said Stenotype notes wen subsequently transcribed in the absence of said witness; that the foregoing is a true and correct transcript of the testimony then given by the witness as aforesaid; that I am not a relative, attorney, or counsel of any party or otherwise interested in the events of this action.

IN WITNESS WHEREOF, I have hereunto set my hand and affixed my Seal of Office in Cleveland, Ohio, this _____ day of _____ 1997.

_____
Priscilla A. Hefner
Registered Professional Reporter
Notary Public in and for
the State of Ohio.

My commission expires: