In The Matter Of:

Karen L. Armour, etc. v.
Patrick A. Rich, D.O., et al.

John P. Conomy, M.D., JD
November 12, 2003

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IN THE COURT OF COMMON PLEAS
SUMMIT COUNTY, OHIO

KAREN L. ARMOUR, etc.,

Plaintiff,

JUDGE COSGROVE
-vs- CASE NO. 2002-07-4063

PATRICK A. RICH, D.O.,
et al.,

Defendants.

Deposition of JOHN P. CONOMY, M.D., JD, taken as if upon cross-examination before M. Sheila Noce, a Registered Professional Reporter and Notary Public within and for the State of Ohio, at the offices of John P. Conomy, M.D., JD, 1730 West 25th Street, Cleveland, Ohio, at 5:00 p.m. on Wednesday, November 12, 2003, pursuant to notice and/or stipulations of counsel, on behalf of the Defendants in this cause.

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Dr. Dean Rich.

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JOHN P. CONOMY, MD, JD, of lawful age, called by the Defendants for the purpose of cross-examination, as provided by the Rules of Civil Procedure, being by me first duly sworn, as hereinafter certified, deposed and said as follows:

CROSS-EXAMINATION OF JOHN P. CONOMY, M.D., JD
BY MR. KURI:

(Thereupon, Defendant's Exhibits A and B were marked for purposes of identification.)

Q: Hi, doctor, my name is Phillip Kuri. We're here today to take your deposition regarding a case you've reviewed. I represent Dr. Patrick Rich in this case. If you don't understand any of my questions at any time, let me know and I'll repeat or rephrase the questions until you do understand, is that fair?

A: Yes.

Q: You've obviously been deposed before. Do you keep a running count of how many times you have been deposed?

A: No, I don't. It's many.

Q: When you say many, are we in the hundreds yet?

A: Probably. How about you, have you done hundreds of these yet?

Q: I have. I have.

A: Well, we're probably near even.

Q: Now, I know you do work, you review cases for the defense and you review cases for plaintiffs. What's your breakdown at this current time?

A: It's surprisingly stable at about 50/50, Mr. Kuri, over the years. Actually, in terms of personal injury and my own patients, clearly it favors defense.

In recent years, something not so odd has happened, and that is that court appearances tend to increasingly favor plaintiffs. It has, I think, a good deal to do with the nature of change in litigation. There's plenty written about it.

One article that stuck in my mind in the ABA Journal was called Disappearing Trial, and it has to do with extrajudicial settlements for matters, particularly medical malpractice.

Q: Okay. You have a juris doctorate, correct?

A: I do.

Q: Do you keep up with that at all?

A: What do you mean by keep up?

Q: Well, let's start with, do you practice?

A: No, I'm not a lawyer. I hope never to be one.

Q: Do you keep up in terms of your education in regards to matters of the legal community?

MR. MISHKIND: I'm going to object to the form of the question, but you can go ahead, doctor, if you understand it.

Are you talking about CLE courses?

Q: That could be one, or do you just keep up on your own, whether it's legal/medical matters or just general legal matters?

MR. MISHKIND: You can answer.

A: I do keep up, largely in the form of writing, teaching, participation in seminars.

I do it because I'm interested in it and enjoy it.

Q: It's probably a good time for this. I'll hand you what has been marked as Defendant's Exhibit B. If you will identify that for the record.

A: Yes, this is my curriculum vitae, and it has a run date of January 2003.

Q: Is that up to date then?

A: As of January 2003, yes.

Q: Is there anything off the top of your head that

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you can think of that has occurred since then?
A: Lots of things have occurred since then. I'm not sure I want to tell you about them.
Q: That you would place on your CV?
A: Committee assignments, publications and lectures, but pretty much the same.
Q: Anything that would be related to the issues in this review?
A: I think not.
Q: While we're talking about your CV, is there any article or periodical that you've authored that would be germane to the issues in this case, whether it be a PE or stroke related to a PE?
A: I think relevancy resides in the eye and mind of the beholder. About 25 percent of the articles, books and book chapters are related one way or another to the issue of stroke. I'm sure that many have at least a tangential relationship to this case, because many of them deal with the relationship between blood pressure and brain activity and brain blood flow.
So do they have some relationship? Yes. Are they particularly and specifically related to a close fit with all of the clinical aspects of the problems faced by Mrs. Speicher? I don't think

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that they're absolutely on point, but related, yes, they are.
Q: Are there any articles that are kind of on point with the facts of this case, a PE which precipitates a stroke?
A: I don't believe so.
Q: Have you ever read any articles where a PE precipitated a stroke?
A: Yes, I have.
Q: What articles are those?
A: Well, there is, and I'm recalling here without specific citations for you, but pulmonary embolus leading to systemic hypotension leading to stroke I think has been addressed by some experts in a publication of one or another learned treatises.
They tend to aggregate in textbooks of stroke. I think that Louis, L-O-U-I-S, Caplan, with a C, has written about this. James Toole, with a T-O-O-L-E has written about it very early on, preceding even your embryonic era, Mr. Kuri. A man at Wayne State University, and I'll think of his name as time goes on here, wrote about this subject.
But again, in any anthology of stroke, that is textbook of stroke, stroke publications

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touching upon this relationship, it's old and well documented.
The man's name in Detroit is John Sterling, by the way, S-T-E-R-L-I-N-G, Meyer, M-E-Y-E-R.
Q: John Sterling-Meyer was his full name?
A: Yes, he was British and preferred to have a hyphenated last name, like Holdsworth.
Q: Other than those names that you just mentioned, can you be more specific with citations or actual names of publications?
A: No, but if you know those names and find your way to a medical library, you'll have the truth revealed unto you.
MR. MISHKIND: Phil, when you refer to citations, are you referring to books or articles?
MR. KURI: Everything.
A: If you find the books, there will be hundreds of references to journal articles. It's not hard to do at all.
Q: Have you done so in this case? Have you done any medical research at all into the issues in this case?
A: It would be medical research for legal purposes were I to have done so, but this is not an arcane

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subject. I think I would rely on what I generally know and use in the course of a workday.
Q: Okay. Other than the names that you just provided me as potential authors of articles or textbooks or some type of periodicals, can you think of any more as we sit here today?
A: No, but if I do, I'll be happy to let you know.
Q: I appreciate that. What percentage of your practice would you say deals with the care and management of stroke patients, CVAs?
A: Roughly a quarter.
Q: Let's just briefly go over the types of strokes or CVAs that exist in general. How many ways can you have a stroke?
A: Well, you can have it one way, and that is the interference of the blood supply, one way or another an artery or vein, in the restricted area of the brain that leads to a permanent destruction of brain tissue.
Q: Is that something that's more focal? Is that what you're discussing?
A: I would discuss whatever you would want to ask me.
Q: I'm asking you what you meant by a restricted

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area.
A: Well, it is focal in that sense. Strokes occur in circumscribed areas of the brain. That's part of the definition of stroke.
Q: That's one way a stroke occurs, how else?
A: Tell me what you mean.
Q: Sure. There are certain types of events which precipitate a stroke, correct?
A: Yes.
Q: What are those things?
A: Well, occlusion of a cerebral vein or artery. I'm going to get rid of the issue of cerebral veins, which are 50 percent, so that will truncate our discussions, but in situ obstructions, that is thrombosis or embolization, that is probably in one place that moves to someplace else.
Now, the source of that embolization can be of the heart, any of its chambers, any appendages or valves and any of the conduits leading from the heart to anywhere in the brain. That is the system of arteries that go there.
Those occlusions can be single or multiple. They tend more often than not to be single, and in their wake, should the occlusion be sufficient

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and the time of occlusion be sufficient and unrelieved, death of brain tissue, either in a bland way without the presence of hemorrhage or with hemorrhage can occur. Those are the common ways stroke occurs.
Arteries themselves can also be the site of primary diseases. Inflammatory conditions, for instance, of the artery, degenerative conditions of the arterial wall are examples of such. Those don't pertain to Mrs. Speicher.
Q: What you're talking about here does not involve a stenotic condition?
A: What I'm talking about here and what we will talk about here eventually, that is her stroke, does involve a stenotic condition in my opinion.
Q: Does what you just described before, the arteries and the diseases of that type being a less common avenue for a stroke, does that involve a stenotic process?
A: No, stenotic processes are common in the production of stroke.
Q: Okay. Are there any other ways to have a stroke? Have we exhausted all of —
A: No, we have hardly exhausted anything yet. I don't want to exhaust you in going on with this.

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She did not dissect her cerebral area, for instance; she was not in a wrestling match; she wasn't in a wreck; she didn't have her hair washed backwards over a sink. She didn't take birth control pills and on and on.
Q: I think you're being more specific than what I'm trying to get at.
A: Tell me.
Q: The overall process in which a stroke can occur, whether it be embolic or whatever, it's my understanding there are probably about three or four general types of ways which a stroke can occur. Am I correct or am I wrong?
A: You may be correct. I don't know. Why don't you continue to instruct me. There are lots of ways a stroke can be generated, but in the end, the death of a restricted portion of brain tissue from the distribution of the cerebral artery of the brain is what happens. There are hundreds of ways of getting to that.
Q: I got you. Can you describe what Health Systems Design, Inc. is?
A: Yeah. It is a corporation of which I am the president, the only effective working member, and which forms a convenience and provides

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orderliness in my life.
It deals with a number of things I do that call upon medicine but are not the practice of medicine, including my testimony here today.
Q: What other types of things are involved?
A: It involves a variety of consultative work with a variety of institutions, medical, organizational, governmental, inside and outside the United States. It involves lecturing, it involves seminar performance and preparation.
Q: Okay.
A: It involves many of the things that I do.
Q: You have prepared a report dated June 14th, 2003, correct?
A: I have.
Q: And I'll hand you what has been marked as Defendant's Exhibit A. Is that a copy of the report you prepared in this case?
A: Yes, it is.
Q: Did you prepare any other reports?
A: No. This is the only report I prepared.
Q: This is probably the best time to quickly go through what it is that you reviewed and what you have in front of you.
A: Um-hum. Well, let me approach it this way if I

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may.
The report that you've showed me is a report that I authored which lists a number of things that were in my possession at the time that report was written. That includes the records of Drs. Patrick and Dean Rich, the records of a Barberton Hospital, the records of the Akron General Hospital, a deposition of Dr. Patrick Rich and subsequently, finally, I did receive a deposition of Dr. Dean Rich. The death certificate was enclosed with the records I received and the brain imaging studies, which I have here with me.
Now, Mr. Kuri, since then, I have received some other things, some of them very recently. I received a report, and by recent I mean within the last few days, a report from Dr. Bruce Ammerman done at your request. I mentioned the deposition of Dr. Dean Rich, a report from Dr. Ronald Bacik addressed to Mr. Howard Mishkind, another to Dr. Howard Mishkind.
MR. MISHKIND: Dr. Howard Mishkind?
A: It ought be. To Mr. Howard Mishkind from Dr. Mark Bibler. And that's —

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MR. MISHKIND: I think you also have two other defense reports in the stack in there.
A: I do. Dr. Lawrence Martin. I'm not sure just when I received that, but I do have that report as well.
MR. MISHKIND: Just in fairness, I think Dr. Herwig's report is in there also.
A: It is.
Q: Could you just list off the dates of the correspondence that you have received from somebody in plaintiff's counsel's office. I don't think all of them are from Mr. Mishkind.
A: Yeah. Again, I may or may not have all of them. I tend not to keep them all. The earliest I have is September 27th, 2002. That's from Mary Ellen Sansbury and from Jean Tosti of the Becker & Mishkind office thanking me for agreeing to review the medical records.
So apparently this was preceded by a phone call. I have no recall of that day. They generally come in the course of the workday, and I don't keep a record of them, but it must have been around that time.
There is additional correspondence, generally

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in the form of sending one or another of the records or depositions that I've just mentioned to you, dated February 13th, April 14th, March 19th, July 24th and this week, 2003.
Q: What, just from looking at the correspondence, can you tell about when you were first contacted to review this case?
A: It must have been shortly before September 27th, 2002. The usual latency between receiving records and agreeing to receive a case is very frequently very short. Not as short as the time it takes a Pakistani cab driver to blow his horn in Manhattan after the light turns yellow.
Q: I got you. The September 2002 date would be approximately when you would have been first contacted or maybe shortly before that?
A: I would say it must be very near September 20th or 25th.
Q: Have you been retained by the law firm of Becker & Mishkind or anybody there prior to this case?
A: Yes, I have.
Q: About how many times?
A: Probably two or three times. I'm not certain. I've never testified for — I don't know if I've

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testified for Mr. Mishkind. I don't recall that I did, but I've probably given a deposition or two for him, and for others in his office, probably the same number of times over the years. Now, over the years is over more than, much more than a decade. I became acquainted with Mr. Mishkind when he was working with the firm of Fred Weisman Associates.
Q: When you say testified, that would have meant deposition or trial?
A: Correct.
Q: Do you know how many times you've reviewed cases for the law firm of Becker & Mishkind?
A: About that many times, probably three or four times in the course of a decade. I don't remember the cases specifically, I'm sorry. One recent case I do, but that's the only one.
Q: Now is probably a good time to ask you, have you ever reviewed a case with similar facts as this?
A: With similar facts as this?
Q: Yes.
A: Hypotension related to stroke in an ill woman with a cardiac arrest, sure, and again, that is a common scenario, and I can't tell you how many times. I would guess a dozen, but there may be

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more than a dozen.
Q: How about the PE precipitating hypotension which results in a stroke?
A: That as well, but I can't be specific. That is not an uncommon situation in ill, hospitalized people.
Q: So what you're telling me is that you believe you have reviewed cases like that?
A: Yes.
Q: Have you ever given testimony in a case like that?
A: I honestly don't recall, Mr. Kuri.
Q: Can you remember any specifics about those cases that you reviewed?
A: Not other than what I've already framed. Pulmonary embolus as a mode of death is extremely common in hospitalized people particularly. Stroke is not uncommon in the elderly, and particularly in elderly persons with cerebrovascular disease such as she.
Q: And just so I understand, specifically where the allegation is, a PE resulting in hypotension, which somehow a mechanism occurs wherein a stroke evolves. In that setting, you believe you've reviewed and possibly testified in cases with

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that fact pattern?
A: I don't recall whether I have or not. The fact pattern is a common one, and certainly in my own patients it's not uncommon.
Q: Whether it's common or not, my question is simply have you reviewed a case like that or have you testified in any case with that fact pattern?
A: I don't recall testifying in a case like that. I recall, but I can't give you the names of people who have had a similar fact pattern of this as a mode of exit from this existence.
Q: When you say similar, are you simply referring to the aspect of hypotension resulting in a stroke, and the PE not being part of the equation?
A: No. The pulmonary embolus, how many times I don't know, and I'm guessing about numbers over 40 years, okay, and I would guess maybe 10, a dozen times now. When it comes to hypotension associated with stroke, that number becomes a very large number.
Q: Okay. In this case, based upon a review of your report, it appears that you were not asked to comment on standard of care opinions in this case, is that fair?
A: That's correct.

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Q: Is it your plan to opine on standard of care in this case?
A: It depends on whether it's planned to be asked of me.
Q: Do you have any opinions in this case regarding the standard of care?
A: I don't mean to be evasive or cute in any way, Mr. Kuri. Talking about causation, it's intertwined somehow with an inference of standard of care, number one.
Q: Let me —
A: Secondly —
Q: I'm sorry. I didn't mean to cut you off.
A: I kind of talk too much, forgive me. Pulmonary embolus is a common disorder. Everybody knows about it. So you know, you don't have to be an expert of the lung to know about this. It is a common situation. So having said that, I'll stop talking.
Q: Do you plan at the trial of this case to testify that either doctor, Dr. Dean Rich or Pat Rich deviated from their standard of care in the care of the patient?
MR. MISHKIND: Let me interject, before you ask him, and certainly you can

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ask him. I'm not necessarily sure you're going to like what he has to say, but I think in his report, I've asked him to comment only on the issue of proximate cause, but I'm not going to prevent you from asking him questions.
MR. KURI: Let's make this easy.
MR. MISHKIND: Sure.
MR. KURI: Are you going to ask him questions regarding opinions of the standard of care of either Dr. Rich in this case?
MR. MISHKIND: I wasn't planning on it. If you're going to ask him questions on an area that his report is pretty clear that he was only asked to comment on issues of proximate cause, I do not as I sit here right now, Phil, in all fairness, and I'm not trying to play games with you, but I asked Dr. Conomy, a neurologist, to look at this case from the standpoint of causation between the PE and the stroke and issues of morbidity as well as life expectancy issues, and those are the primary areas.

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I will tell you that he does have opinions, just in discussing the matter with him, on the issue of standard of care, but I do not intend to ask him, and I think the report does not touch on that. So under those circumstances, I think I probably would be precluded unless you open the door.

MR. KURI: My point was, is there going to be a change in what was in the report? In essence, I don't want to spend another hour here talking about standard of care if we don't have to.

MR. MISHKIND: I do not intend to ask the doctor standard of care questions. I expect to ask questions on proximate cause issues and damage and causation, and we obviously have other experts.

MR. KURI: You said there were opinions potentially regarding life expectancy?

MR. MISHKIND: Yes.

MR. KURI: I did not see that in the report, but you do plan to ask those questions.

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MR. MISHKIND: I do.

Q: Let's get that out of the way. Do you have any opinions regarding Jean Speicher's life expectancy?

A: Yes.

Q: What are those?

A: Well, she's 77, and was robust. She had an illness, when it was localized to her leg, that was eminently treatable. I don't know any reason outside of the evolving nature of her shortly fatal illness that would have kept her from living those years a woman of 77 would ordinarily live, which is another ten years.

Q: All right. Can you be more specific about your basis as to why it would be ten more years?

A: That's the life table projection of a 77-year-old basically healthy person, realizing that nobody 77 is perfectly healthy. That's just an inference based on averaging.

Q: Well, to follow up on the fact that she was basically healthy, what was her previous health condition?

A: Nothing terrible. She had diverticulitis, she suffered obstipation, she had dizziness from time to time, she had a number of benign complaints,

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pain in her joints and ringing in the ears and so forth and so on. I think the occasion of casual sinus trouble —

Q: What about —

A: — but no major disease, cancer or prior stroke or the like that would have been a clear signal to offer you a number diminished from ten years.

Q: Sorry. I apologize for talking over you. Sometimes I feel you're done.

A: Well, when I see you want to talk, I keep talking.

Q: You do that on purpose.

A: Yes.

Q: What about the presence of stenotic cerebrovascular disease, would that reduce her life expectancy at all other than the normal ten years you would expect from a relatively healthy 77-year-old?

A: No, it wouldn't. Her stenotic cerebrovascular disease became symptomatic only under circumstances of severe and sustained systemic hypotension. There's no reason to think that would have happened. And furthermore, that degree of atherosclerotic disease, which it is basically, is not likely to become symptomatic

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until it becomes severe. There is no reason to think that would have happened.

Q: What can you point to in her medical chart or history that would indicate the level of the stenotic cerebrovascular disease that you believe she had?

A: None prior to the development of a very large cerebral infarction in the course of her systemic hypotension. That's how it was found out.

Q: Then how can you opine as to whether it was severe as opposed to relatively benign?

A: Good question. It wasn't severe, because it would not be likely to produce any flow impairment until the degree of stenosis exceeded 70 percent under the circumstances of normal profusion pressure, which she didn't have.

Lesser degrees of stenosis, even so-called mild or benign degrees of stenosis become symptomatic only when profusion pressure falls to a very low level and is sustained there, as hers was.

Q: Is there anything from her records, her testing, on review of the CT scan that shows conclusively that she had cerebrovascular disease?

A: Conclusively is a key word. It would have taken

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an autopsy and microscope to be conclusive. Highly probable based on the distribution of her stroke at the time she was hypotensive. It didn't happen every place, it happened in a specific place, and it happened in a specific place because flow in that specific place was reduced to a critical level in contrast to the rest of the brain.

Now, the usual cause for that, and particularly in an elderly person is focal stenotic disease. That gets back to all of those articles you tweaked me about in your opening barrage.

Q: Let's take a step back and talk really generally about how this process occurred in Jean Speicher.

A: Which process are we talking about?

Q: The ultimate end that a stroke occurred. Let's take it from beginning to end. She comes to Akron General, she's diagnosed with a PE, I'm going to let you take it from there, and I'm asking for a long-winded answer as step-by-step as to how the process worked which ended up with a focal CVA?

A: What I'm going to draw for you, Mr. Kuri, is the left side of her brain, okay, this is a cartoon.

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We'll look at the carotid arteries coming up forming a siphon, and then the middle cerebral artery, and I'm going to draw a straight line here. I know that she had Doppler studies of her extracranial circulation at the Barberton Hospital in January, and that there was no critical stenosis.

Q: January of what year?

A: The year she died, '03, was that it?

MR. MISHKIND: No, I think she died in '01.

A: Well, it was that year.

MR. MISHKIND: I'm not sure if it was '00 or '01 in the Doppler.

Q: Why don't we double-check that.

A: She died in '01, so it would be '00 at the Barberton Hospital. It would be — pardon me. It would be January 25th of '01 or a time around there, and it's in the tabbed portion of the chart called Echo, or at some point.

Q: Got you.

A: You've got it.

Q: Well, my notes indicate a January 25th echo.

A: That's what I'm talking about. She has normal arteries coming up to this point.

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Q: Up to what point, I'm sorry?

A: Up to the point I'm drawing. Stick with me. Leave your yellow pad and join mine.

Now, this is open to this point, and she has no stroke or stroke-like symptoms under conditions where her mean arterial pressures are of an average or normal variety, okay.

Q: Can I stop you?

A: Sure.

Q: While you're answering that, what would be the mean or average variety?

A: Let's say her perfusion pressures in these arteries is something between 70 and 100 millimeters of mercury. Okay. That is taking her high number on the blood pressure and low number and adding them up and dividing by two to get mean pressure, okay.

So between those numbers, she's not having any trouble at all. Now, if you're dealing with a degree of noncritical stenosis with these pressures, there is no announcement clinically that anything goes wrong. But if the mean arterial pressures drop, and hers dropped at one point to nearly nothing, yes.

Q: Let's —

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A: No, not let's. I don't want to be selling you something here.

Q: Okay. Which pressures are you talking about?

A: Mean arterial pressures. I'm now looking at Page 149 of document Number 40, exhibit whatever if you would like it. And here at a time in the course of this mayhem of cardiac arrest and ventricular fibrillation and multiple drugs and on and on, her mean arterial pressures, I don't know if it's unrecordable or nearly unrecordable, but it's less than 20 torr.

Q: At what time?

A: Sometime around 1:30, 2:00 in the morning. Now this is not, this strip isn't dated, but it would be on here. I'm not sure. They have a printout date, but this is 2/6, looks like 2/6 at 2:00. You agree?

Q: 2:00 in the morning?

A: Yes, these are military times.

Q: It's hard for me to see.

A: Okay. Again, I don't want to be —

Q: That's all right.

A: Now, we know that this didn't happen, there's no V shape in here, this is simply a matter of sampling, but we do know that at least at one

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point her pressures are down here 10, 20 and so forth. You would agree that's very low. I don't want to gloss it over.

Q: Okay. A half hour earlier the mean is?

A: It's around 60, 70, maybe a little more.

Q: Okay. Do you have a problem with that mean in terms of being average, below average?

A: No, I don't think her brain had a problem with it at that point. She was sick in many other ways but not with brain blood flow.

Q: So would it be fair to say then in your opinion you believe that the stroke or CVA in this case occurred sometime between 1:30 and 2:00 in the morning?

A: Not necessarily. I think it's probable that it did, but don't depend on one little graph to tell you that.

Q: Could it have happened before that time period, or would it be after if you were going to err on either side?

A: No, and I'll tell you why. The sampling times are far enough apart that anything could have happened in between. You just don't know. These are not continuous. There's a plot of recordings.

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Q: So if you took it at 1:14 where they plotted it and again plotted it at 1:32, at 1:25 it could have had a dip like we're seeing at 2:00?

A: It could have been anything. This is a matter of points. Now, if this was a continuous recording, you're dealing with a machine that is ready to spit out data above and below certain numbers and just keep crunching it out, then you have a better lead, but we know at least at this time that that happened.

Now, when that happens, here's what happens to blood flow in areas of noncritical stenosis. The perfusion pressure of let's say 70 torr, 70 millimeters of mercury is enough to push past this obstruction basically without ever noticing it. Because the force behind it is sufficient to overcome the obstruction, that's a descriptive term. There's mathematics behind it, but assume what I've told you is stated correctly.

Under conditions of lower pressure, that won't happen. At that point a noncritical stenosis becomes critical because perfusion pressure is very low. So that in an adjacent vessel without that degree of stenosis, and let's say at a perfusion pressure of 60 or 40, blood

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will continue to flow normally, but with a stenosis that is even less than 70 percent, call it 50 percent, blood flow becomes reduced and infarction occurs downstream.

Now, downstream here is in the distribution of the middle cerebral artery. This will not be on the echo. It wouldn't be seen in the echo studies. She would need a transcranial Doppler study to show this, and that wasn't done because there's no indication to do that.

But what it does tell you, and it's kind of reasoning, if not backwards in the sense of backward mentality, we know that her brain infarction occurred in the distribution, and that is in the distribution of the left middle cerebral artery. Why there and not in the posterior cerebral artery or in the anterior cerebral artery, and that's because it's very likely under the circumstances that an otherwise noncritical degree of stenosis occurred right at that point.

That's why the infarction occurred there and not everywhere. When there is no critical stenosis anywhere, you don't get brain infarction of the type she suffered, you get something

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other.

Q: Something more global?

A: To be sure, bilateral, so-called watershed. Watershed, which I hope you won't ask me to get into, or global cerebral anoxia, which the whole brain, if you will, is stroked.

Q: I do want to ask you a couple questions.

A: That's why you're here, earning your keep. I can't imagine you not doing that. Let me just color this in with a Cady Reporting black pen. That's the distribution of the left middle cerebral artery. I will sign this with date and time.

Q: Sounds good.

MR. KURI: And we might as well go ahead and mark it as an exhibit and get that out of the way.

(Thereupon, Defendant's Exhibit C was marked for purposes of identification.)

Q: I'll hand you back what has been marked as Defendant's Exhibit C. If you can briefly describe for the court reporter what you have just done and what's on there.

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A: This is a cartoon. It's a lateral view of the brain that represents an impairment, focal impairment of blood flow at the origin of her left middle cerebral artery. It will correlate, but in a different plane, than what we may get to see in her imaging studies.
Q: Would it be a fair statement to say that because she had a focal CVA, that she only had one area of stenosis?
A: First of all, all CVA is focal in the sense that we're talking about. So let's get rid of that part. It doesn't tell us this is the only region of focal stenosis. It's the only one infarcted. She had a distribution.
Now, the problem with that is that this is the main artery, if you will. This is to the brain what Interstate 77 is to Akron in terms of blood flow or traffic flow from Cleveland. It is the main artery that will serve the blood supply of two thirds of one half of the cerebral hemisphere.
Q: Okay. Could you list for me every medical fact that you can pull up from the record or whether reviewing the films that she had stenotic cerebrovascular disease?

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A: The likelihood of stenotic cerebrovascular disease becomes huge when a brain infarction is suffered under the condition of systemic hypotension. And that's what she had. There aren't every single fact. That is the fact.
Q: So based upon the result, you're stating that's why you believe she had to have some type of stenotic process?
MR. MISHKIND: Objection.
Q: You're working backwards in order to assume that she had a stenotic process based upon the result, correct?
MR. MISHKIND: Objection.
A: Not only correct, it's the only way to work.
Q: Well, there are certain studies that would show that if she were to have them beforehand, correct?
A: But she didn't, did she?
Q: I'm just asking.
A: She did not have them.
Q: The only way that you're coming to this conclusion is you're taking the result and working backwards.
A: Sure.
Q: Am I correct?

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A: Yes. It's like, you know, looking at a crater in Bagdad and wondering how it got there.
Q: Are there any other possible avenues, medical conditions, ways in which a stroke could have occurred in this patient?
MR. MISHKIND: Objection to the form of the question in terms of possibilities, calls for speculation, but go ahead.
A: There are literally thousands of possibilities, and again, I don't want to be cute or facetious. She did not get struck in the head by lightning and so on. Under this fact pattern, I think that all of those considerations become very highly improbable.
Just let me mention a couple of things, not that I think it occurred, but that are more probable than this whole universe of possibilities.
Q: Sure.
A: Might she have had a hole in her heart in the wall that divides either the right ventricle from the left or the right atrium from the left? A patent foramen ovale, a defect that had caused a blood clot in her leg to somehow get into her

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brain.
Think of it. It's a paradox, isn't it? Yes, it is. Say yes, it is, if I'm answering the question.
They're called paradoxical emboli because something that should get into the right heart, or into the circulation, lung circulation, winds up in the brain.
Now, it does that under conditions of atrial and ventricular septal defects and is called paradoxical embolization. It's a way of a blood clot say in the vein of the pelvis or femoral vein or somewhere in the leg getting into the brain by finding a secret passage, if you will, and then causing the stroke.
I don't see it very often. I've seen it twice in the last month, but it's probably going to be your next birthday before I see another one of those again.
That didn't happen to her because there is no demonstration of a hole in her heart by reliable studies, again, previously done in a January hospitalization that looked at certain functions of her heart.
Q: Now —

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A: Hold on. So she didn't have that. She had cardiac arrhythmias, a wobbling of the heart, if you will, shimmering, and ventricular fibrillation in the course of this. People whose hearts shimmer and shake develop blood clots in the heart that find their way to the brain.

However, that's very unlikely, because that condition didn't last very long before she died and usually requires some time with an ongoing arrythmia, 48 hours or more, generally much more before the clot will form in the brain.

So those are more reasonable speculations, possibilities than what actually happened to her, but they didn't.

The most highly probable cause of what happened to her is focal stenosis combined with the systemic hypotension that produced the big brain infarct.

Q: All right. This is as good a time as any to ask you the possibility of Dr. Ammerman's belief that the CVA was secondary to a middle cerebral artery thrombosis.

A: I don't wish to speak for Dr. Ammerman. She had thrombotic disease. That's what produces stenosis. We're talking about hardening of the

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arteries, Mr. Kuri.

Do I disagree with that? No, I don't disagree with it, but do I disagree with him in the sense that this artery, perfectly normal, somehow became thrombosed just then. If that is his inference, then I don't agree. Technically this is thrombotic, it's atherothrombotic disease. It's commonplace in an elderly woman.

Q: All right. Is there anything to indicate that she wasn't having some type of thrombotic process or disease going on in the record?

A: Well, she did have thrombotic disease, she had pulmonary thrombotic disease, which is — and clearly these have been going on for some time.

I'll tell you why I think that's the case. She has the dilated right ventricle, she has some degree of tricuspid insufficiency, and she has pulmonary hypertension. Those are all consequences of precedent pulmonary embolization or pulmonary thrombosis, thrombi in pulmonary vessels. But now we're talking about her lungs, not her brain. This is thrombotic disease, yeah, but it's in another place.

Q: Is there anything you can point to in the record to say that it could not have been cerebral

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artery thrombosis?

MR. MISHKIND: Objection to the form of the question. Calls for speculation. Go ahead.

A: No. Again, I want to get back to the point. This condition is the product of cerebral artery thrombotic disease. Now, if by thrombosis you mean complete, sudden and de novo occlusion of the cerebral artery from God knows what cause, that's not my opinion.

Q: Okay. For my own understanding, would this include some type of break off of say plaque or something along those lines?

A: Good thinking. That's an excellent question. I don't think she had artery to artery embolus. That is the piece of junk that breaks off from the known plaque in the carotid artery, for which there was no demonstration by the way, that traveled upstream and got stuck at the point that I've made the narrowed segment here.

I don't think that that happened because of the lack of demonstration of disease in the carotid artery at that point close in time to this event. So while it's a good thought, there's no evidence for it, Number 1. Number 2,

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it's not necessary to invoke it.

Q: What's not necessary to invoke it?

A: That particular mechanism.

Q: You lost me there.

A: Stick with me. This is not medicine, it's just logic, okay. It derives from one William of Occam, a Dominican monk who was a pain in the neck to the fathers of the church and had to hide himself in France to escape inquisition.

He said that once you have an explanation that is both efficient and sufficient to explain the phenomenon, it has an effect related to its cause, that the invocation of other causes to explain the same event is illogical, inefficient and wasteful. So once we have one mechanism in place, to then put another one on top of it and on top of it, layering is illogic.

Q: Okay.

A: It's called Occam's Razor. It should be used on lawyers more often.

Q: Let's continue on the line of cause and effect. In your scenario as to how the cerebrovascular disease played a part in creating the CVA, let's remove the cerebrovascular disease.

A: Remove the stenosis.

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Q: Let's remove the stenosis. Could this have possibly happened?
A: Possibly anything can happen. Is it likely to? No. What is likely to happen under the circumstances that you've raised is watershed infarction on both sides of the brain, or global cerebral ischemia, in which the whole brain becomes anoxic kind of at once. There is no focality to that. These are general conditions of brain injury under circumstances of systemic hypotension. They're the kinds of things in young children who drown, for instance.
Q: Okay. Let's assume the watershed event. Is it the lack of oxygen perfusion in the blood that would cause the watershed event?
A: It can be oxygen, but it's usually general circulatory failure. It's not just oxygen, it's oxygen, glucose and blood flow all at once.
Q: Why in this case then did the watershed event not occur before the focal event?
A: Because the focal event will take precedence because of its lower threshold of achievement. It's more likely to occur before these global situations occur because of the stenosis involved.

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Q: I know I've touched on this with you before, but just so we're certain, can you give me the time period based upon your review of the chart of when the CVA must have occurred? Can you give me the beginning to end as opposed to saying it occurred at this specific time?
MR. MISHKIND: Objection.
A: No, these events were set in motion sometime around 11:00, 11:15 on the night of the 5th, I believe. She was really quite unresponsive through that time. I would say that, you know, it occurred before 3:00 in the morning.
This episode of severe and sustained hypotension in terms of the acquisition of brain infarction becomes probable, but could it have happened before that? Sure, it could have.
We just don't have the kind of observations that would have been more probative. That's not a criticism. There's a lot going on, and her circulation failing, her heart is failing, she's flipping emboli, bad stuff.
Q: Okay. Let's get on paper what you believe, or what the medical community believes is the general range of what you would consider hypotension, and take a patient such as her.

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A: I'm going to purposely subvert your question, but only slightly.
Q: Okay. Let's not use that same piece of paper.
A: Okay. This particular rendering is going to be to Phillip Kuri. One or two Ls in Phillip?
Q: Two.
A: And it's going to be entitled Brain Blood Flow autoregulation. The brain has an extraordinary capacity to protect itself over a wide range of blood pressures, so it gets to be the hog. The brain is the hog in terms of the acquisition of blood flow.
As you sit here listening to me going to medical school this afternoon, one out of every five drops of blood that pumps from your heart goes to your brain. As you go home and watch the Browns' coach explain away again another insulting loss, you still get one out of five drops. You go to sleep tonight, take care of your children, kiss your wife, it still gets one out of five drops of blood flow. If you have little children, theirs gets even more; one out of two, not one out of five, and it will do that whether your blood pressure is raised because of the tasks performed.

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So if you look at what happens in terms of brain blood flow, which is exactly 55 milliliters of blood per 100 grams of brain tissue in your brain; healthy, thoughtful person you are.
Now, as your blood pressure fluctuates, this number will be maintained within a wide range. So that if you look at blood pressures down here around 0, and off the other end, I'm talking about mean arterial pressures up here of 200. So to take the blood pressure, if you would, of 250 over 150 up in this range to produce the mean arterial pressure here, and for 0 it's going to be 0 over 0, that's the only possibility.
Within the mean and two standard deviations of this, brain blood flow will be maintained in about this range, and this is something around 45 and this is around 65 milliliters per 100 grams per minute.
Let's forget about the higher end of the curve because we're not talking about severe hypertension. We're only talking about the lower end. Once mean arterial blood pressure falls below about 40 torr and lower, 40, 20, 10 and 0, it does this. This is global flow.
Now, this is flow measured everywhere. We're

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not talking about stenotic arteries. Stenotic arteries are going to shift this curve to the right, and the flow will fall sooner because of the presence of stenosis.

Why that influence, because the flow is related to the inverse of the 4th power and the radius of those vessels. That's Bernoulli's principle. Here a small reduction under these conditions of pressure is going to have a great influence on the nature of blood flow. So this is the right shift that would occur under the circumstances of stenosis.

This whole business where this is a good range for about 80 percent of available blood pressures is what the brain does. No other organ does this, but other organs, your calf muscles can get away without blood flow for 10 minutes.

If you sit on your leg too long, you can get up, shake your leg, and nothing happens. But don't try that with your brain. It can't.

So shift of focal stenosis is to shift this to the right but only where the stenosis applies, and that is in the distribution of a vessel.

Q: While you are talking about a shift to the right, it gives me the opportunity to ask you whether

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you agree with Dr. Ammerman that there was a shift to the midline structure from the left to the right by approximately eight millimeters.

A: Yeah, you want to see that?

Q: We'll put that up, but do you agree with what he said there?

A: Infarcted brain swells and pushes healthy brain around, that's another one of its bad effects, but that's what he's talking about.

Q: Okay.

A: We're not talking about — this is, I'm looking at a mathematical graph that has nothing to do with an MRI machine.

Q: What I'm about to ask you is really basic, but I would like to know your answer. What's the significance of this being on the left in this patient as opposed to on the right side?

A: Had she lived, then the function of language would have been involved. She would have been aphasic. Had she lived, the right side of her body would have been paralyzed and not the left.

Now, this is in contrast with what happens on the other side of the brain. Had this involved a right hemisphere, her language function would have been preserved but the left side of her body

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paralysed.

Q: Is it more common or maybe not common at all, but is it more common to have a left-sided CVA in the presence of hypotension, or is it more common to have a right-sided CVA?

A: Under these conditions, it's random. It simply depends on where the stenosis is critical or worse. There's a number of other factors, but there's no preference on the part of the brain to where it will infarct.

Q: Is the right side of the brain perfused before the left side of the brain from the cerebral artery?

A: No, it's not.

Q: Is it the reverse?

A: No.

Q: It just happens at the same time?

A: It happens at the same time. Now, there are changes in regional blood flow that have to do with certain brain functions, vision, language.

As you're rubbing your lips with your right index finger, the right motor strip is getting a little more blood than the left. As I'm mouthing off, my language centers and the parts that involve both sides of my brain relative to my

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lips and tongue are going to light up if you have the right tools to measure that. So there are critical differences that depend on function, but in terms of overall global flow, blood flow, no.

Q: You had the opportunity to review Dr. Bacik's report and Dr. Bibler's report, correct?

A: Yes.

Q: Was there anything that you disagreed with in Dr. Bacik's report?

A: Could I look at it?

Q: Sure. You have it there. And we might as well stick to the proximate cause questions as opposed to standard of care issues.

A: No. In terms of the statements he made here, I don't have disagreement.

MR. MISHKIND: I didn't hear you. Your voice has a tendency of trailing off. You're such a soft-spoken person.

A: No, I don't have a fundamental disagreement with what he's stated.

Q: You said fundamental, do you have any disagreement?

A: No, I don't think as stated, no.

Q: Let's move on to Dr. Bibler's report.

MR. MISHKIND: Again, you want him

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to concentrate on proximate cause, not standard of care, because Dr. Bibler has a lot of opinions on standard of care.
Q: So I could take you down to probably Paragraph 5 would probably be the area.
A: No, I don't.
Q: All right. He states in that — why don't you take a look at that paragraph again, 5?
A: Number 5, yes.
Q: It says, As a direct consequence of her massive embolus, she had hemodynamic compromise which resulted in a large hemispheric ischemic stroke.
Do you have any problem with the terms he used, large hemispheric ischemic stroke?
A: No, I don't.
Q: What's he describing there?
A: Simply what we have been talking about for the last umpteen minutes.
Q: When he uses the terms large hemispheric ischemic stroke, then tell me —
A: I agree with that. She had a large stroke without hemorrhagic effect involving her left hemisphere in the course of this. That's what he's saying, isn't it, and I don't disagree with it.

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Q: Does that comport with the focal nature of what occurred to her?
A: Well, yes, it does. Stroke, again, is a focal event.
Q: In general, when you use the term hemispheric then, what are you talking about?
A: Well, he's talking about the cerebral hemisphere, the left cerebral hemisphere.
Q: All right. Continuing on in that paragraph, later on he says, In the absence of the massive pulmonary embolus that she sustained that night, it is very unlikely that any of the other complications would have supervened.
A: Yes.
Q: Do you have any understanding what he means by other complications?
A: I think he's talking about her multiple organ failures and her stroke.
Q: Okay. I believe I asked you, taking the stenotic cerebrovascular disease out of the equation, you still believe she would have had a watershed or global type of stroke in this scenario?
A: Yes.
Q: What would you say her chances were of having a stroke simply because she had stenotic

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cerebrovascular disease?
MR. MISHKIND: Let me object. You mean if the pulmonary emboli was not —
MR. KURI: Yes. Forget about the hospitalization.
MR. MISHKIND: Just remove the pulmonary, and what was her chance of stroke?
Q: It's your opinion that she did have stenotic cerebrovascular disease. Let's take that, what are her chances of having a stroke as the result?
A: It leaves me wondering what's to come second and third, but I'll answer. She's 77, she's a woman, she does not have any severe cardiovascular disease. So her cumulative stroke risk given these circumstances is probably 3 percent per year cumulative, which comports with ten years of life expectancy.
Q: Okay. Based upon your review of the records and what you know in regard to her previous medical condition, are you saying that her most likely timely death would be the result of a stroke?
A: Probably not. Something else. That would be in the third rank. Even given her age, heart disease and cancer are going to be much more

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common causes of death in an otherwise healthy 77-year-old woman than stroke, but it's up there.
Q: Even with the stenotic condition?
A: Sure.
Q: She didn't have any evidence of any heart condition, I think you already mentioned that, correct?
A: No, but she's 77. That's enough. You're not going to live another 50 years when you're 77.
Q: Okay. So you're putting stroke as the third likely cause of death for this lady?
A: Statistically, that's exactly what it is.
Q: Would that be the same whether she had a stenotic condition or not?
A: If she didn't have a stenotic condition, it would be less than that, probably a percent less per year cumulative risk.
Q: Okay. I think when you were kind enough to create this chart for me, my original question was much more of a simple nature, and so I want to go revisit it for a minute.
A: Fine. It may require the collaboration of yet another chart.
Q: I don't think it does. Let's start with this question. I just want to know if a patient's in

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the hospital and you're checking their vitals, what would you consider to be a patient that is hypotensive, what range, just a patient in general?

A: Okay. Anybody who's got a cerebral perfusion pressure of less than 70 torr. Now again, that is hypotension that may or may not mean anything under the circumstances, but is it hypotension? Yeah, it is.

Q: Okay. Can we put that in terms of blood pressures?

A: Well, any number that you would like to add up and divide by two and come up with a number less than 70, it's up to you. It could be 70 over 70.

Q: Okay. So anything less than 70?

A: Yes. In terms of mean arterial pressure.

Q: Okay. Is the body equipped to maintain 65 on a fairly regular basis?

A: You know, you can inch your way along, but again, go back to this chart you didn't like.

Q: I didn't say I didn't like it. It wasn't quite the answer to my question .

A: Your wide range, even in elderly people with pressures that are even below 60 torr, you're not likely to get into great trouble, and some can

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tolerate that even for a while. Is it a good idea to do that? It is not.

Q: But it's your position that regardless of the stenosis, she was likely to get in trouble in this case from the point of the CVA?

MR. MISHKIND: Objection.

A: No, I don't think that's what I said. Would you repeat that. I don't think I understood you.

Q: Sure. I think your testimony previously had been had she not had a stenotic condition, she was likely to have a watershed event?

A: Those are very bad things to have. They kill people too, just like this.

Q: I don't disagree with you, but that's what you were telling me before?

A: She would have been more likely to have global cerebral ischemia of some form, that's what I was saying.

Q: Let's pull your report up real quick. On Page 3 you state in the second paragraph, "She suffered prolonged and sustained hypotension in spite of the administration of levophed and TPA." I need you to give me the beginning of what you believe to be the sustained hypotension and

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the end if possible.

A: If you look at numbers here that hover around 60, that's hypotension. That's her mean arterial pressure. Nearly all of these are 60 or less. Here's a dip down to nothing or nearly nothing. Just what shape this V ought to be in and how prolonged, acute it is you can't tell because the sampling points are widely distributed. Then she sustained here, looks to be about 60, 70, then drops down again to numbers around 40. This goes on at least in this particular recording, and those that follow it are all like that. They're all, Mr. Kuri, they're too little, at least for the most part. There's a blip up here to a mean arterial pressure of around 80 and another to around 100. These are much more normal. Now, she's getting levophed and other drugs to sustain her blood pressure at this point, so she's getting the boost to do this, but nearly everything you see is low. Again, there's an occasional normal recording, but it looks to me, without counting them, that 80 or 90 percent of those recordings are in hypotension.

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Q: Did you check her blood pressures in Barberton?

A: I don't recall low pressures like this from Barberton. I think they're all higher, but I don't have a plot of them. Let me look, okay.

Q: Sure.

A: Now, from Barberton, you're talking about the doctor's office or hospital?

Q: She was in Barberton Citizens Hospital.

A: Okay. I wanted to make sure and not the doctor's office.

Q: Sure.

A: There are a couple places I want to look. I want to look not only at the physical recordings, but also at the EKG and some of the other places. Okay. I'm looking at the notes from the cardiac service. This is the one that demonstrates the stress on the right side of her heart. I'm going to the nurses notes. Her mean arterial pressures here are what, 70, 80, up. Some of them are around 100. They're not low for her, and there are not a lot of recordings either. 100, 100, 110, 110, 80. The low ones are around 80.

Q: Let's go to Number 1 of your Analysis and Opinions. You indicate that you share the

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opinion of her medical caregivers during her terminal hospitalization that the cause of her death was pulmonary embolization.
Where are you picking that up?
A: Death certificate.
Q: That would be one physician, correct?
A: And I think the notes here are redolent of pulmonary embolism treatment; TPA, heparin for pulmonary embolism. It would be hard for me to infer anything else based on the records from Akron.
Q: Let's talk about what you wrote. You said you shared the opinion of her medical caregivers. So you were pulling the opinion from the caregivers as to what the cause of death was, correct?
A: Yeah, they're the ones that stated it, sure, and I agree with them.
Q: Anywhere was it stated, other than on the death certificate, that the cause of death here was pulmonary embolus?
A: Yes, let me go back to her discharge summary.
Q: Sure.
A: Final diagnosis, acute pulmonary embolus. This was signed by Don C. Bradford, D.O., who was not a caregiver.

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Q: Does the final diagnosis mention that she had a stroke?
A: Well, not from what I'm reading. I mean he's looking at one single most important event to him.
Q: Okay.
A: And that's pretty — I agree with that too.
Q: If you were following this patient and you were writing down the final diagnosis, would you have omitted the stroke?
A: Would I have, no, but I'm not criticizing Dr. Bradford for doing it.
Q: I'm not asking you to criticize.
A: And I'm telling you I don't. And under the circumstances, normally how I love the brain, I would have made some comment about it in my remarks. Now, I'm paraphrasing Richard Fricker, but shortness of breath, I think it's hypoxemia secondary to PE, pulmonary embolus, shortness of breath, da-di-da-di-da, swollen lower leg, oxygen, heparin. That's pulmonary embolus talk. It's hard to construe it was anything else, at least for me.
Q: Okay. What time was that at?
A: That's when she came in the hospital.

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Q: She didn't have a stroke yet?
A: Well, that wasn't your question. You asked me about pulmonary embolus.
Q: My question was your statement Number 1, "I share the opinion of her medical caregivers during her terminal hospitalization that the cause of her death was pulmonary embolization."
We've talked about the death certificate, and I'm asking you to show me where that is anywhere else, if you did pick it up from anywhere else.
A: Again, let me look. I don't want to misspeak.
Q: Sure.
A: Well, she's not going to have it listed in the cause of death until she's dead, and that's where I got it from. But I'll tell you, Mr. Kuri, it's hard for me to look at this record and tell you anything that might have been in the mind of anyone for the cause of death other than pulmonary embolus.
Q: Okay. What I was just trying to get to is, you used the word caregivers, and I was trying to follow up with what you meant by who it was other than on the death certificate?
A: Again, not to be obstinate, but I would think that were a poll of her caregivers taken, and

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were you asked why did she die, answers other than pulmonary embolus would be rare indeed.
Q: Let's turn to Number 3 on your Analysis and Opinions. We've touched on this. You're basically saying had she not been hypotensive from the PE, this never would have happened?
A: Not quite. That's not what I'm saying.
Q: All right.
A: And again, I want to be very careful to avoid the standard of care issues when I answer this question.
Q: Okay.
A: There was a time when she had swelling of her left leg, where she was known to have some degree of tricuspid insufficiency, a right ventricle that was dilated and high pulmonary artery pressures. All of these things are pathonomic of what happens in pulmonary embolization. Not at the instant of embolization, but after embolization is repeated and after it's made progress.
There's very high resistance in the lung and in pulmonary blood vessels in a sudden event, say a satellite embolus in the pulmonary emboli, because it branches, and you could have a big

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blood clot that really includes both the right and left branches of the pulmonary artery.

Pulmonary pressure between that embolus and the right ventricle goes very high but the heart isn't dilated. That takes time. What happens to that embolus is it fragments and goes out to blood vessels in the periphery lungs and causes shortness of breath and cough.

And that will over time, because of the work the right ventricle has to do, causes thickening of the walls and dilation of the chambers. That picture takes time to occur; several days, a couple of weeks before you see this picture that was found during her echocardiogram.

So when I say the treatment of pulmonary embolization, it really precedes her appearance at the Akron Hospital. She had plenty of evidence that that indeed was what was wrong with her before she got to Akron, and when she got to Akron and subsequent to that, this unfortunate condition simply continued and led to her death.

Q: At what point in her care before she got to Akron General would some intervention had to have occurred before the PE in order to have kept this result from occurring?

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A: Well, certainly there was opportunity to do it in her hospitalization at Barberton.

Q: If it was done then, it's your opinion that the events that occurred at Akron General would not have happened?

A: That and subsequent events, including our conversation here today. It would have been — she would have been treated and spared all of this, as would we.

Q: Number 4, you say in the last sentence, "In this setting she experienced generalized brain anoxia and stroke which were contributing causes to her demise."

What do you mean by that?

A: She developed brain swelling, herniated, and I think that is something that contributed to her death. She might have died at another instant, but I don't think she would have survived what was in her lungs.

Q: What caused the generalized brain anoxia?

A: I think in addition to focal infarction is this episode stretched on, and the decisions were made to give her up. These were combined decisions, I understand, with the family. Treatment stopped, she became more anoxic and died, her breathing.

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Q: This is more of a result of the decision that she wasn't going to get any better, and so?

A: Yes.

Q: Okay.

A: It was a decision based on the inevitable confrontation with futility.

Q: So the brain anoxia wasn't something that necessarily occurred at the time when the focal CVA had occurred. This is later on, we're not going to treat anymore, this is what happens when you don't treat anymore?

A: These are cumulative events.

Q: Okay. Let's pop these up there real quick.

MR. KURI: Are these copies?

MR. MISHKIND: I'm sure.

A: Copies of these are like dollar bills. They're not copies. These are done on the computer with the same information that made the original. They're like plates at the mint.

Q: The quality wasn't what I was asking about, it was like asking could we circle on it or something.

A: It would depend on one's nature and attitude toward neatness, completeness and the refrain from schmutz.

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You know what schmutz is? The actual word is schmutzig.

MR. KURI: We might as well mark those real quick.

MR. MISHKIND: You can mark them, but I'm obviously keeping them.

(Thereupon, Defendant's Exhibits D, E, F and G were marked for purposes of identification.)

Q: For clerical measures, I'm handing you Exhibit D. Explain what this is.

A: A graph showing brain blood flow autoregulation.

Q: Now, we have films up in the box Exhibits E and F. Would you describe for the court reporter what these are?

A: Computer tomographic scans of the brain. These were done at the Akron General Hospital upon Jean Speicher on February 6, 2001 with an acquisition time at around 9:30 in the evening.

Q: Based upon the report that was generated from these films, it would appear a CVA was identified, correct?

A: A stroke was identified, yes.

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Q: Can you pinpoint, I think you've testified that the stroke itself was focal. Can you pinpoint in any of the frames where you can, where you see that, where you see the stroke?

A: First let me tell you what this is. This is a CT brain scan. It's developed and used based upon the ease by which an x-ray beam can travel through a volume of tissue.

Q: Okay.

A: The brain is fairly soft. X-ray beams go through it relatively easily. Bone is pretty hard, and x-ray beams don't go through it very easily, and the way that the computer attached to these machines is made is that things that impede the travel of x-ray beams are white, like bone, and things that don't impede the travel of x-ray beams, like water, is black, and intermediate densities in terms of the resistance of penetration are gray or shades of gray, and the gray can be scaled, and then it's reconstructed to look like a human brain.

Strokes are not likely to be seen the instant they occur. A few can, and there are some telltale signs we need not get into. In terms of ripening it takes several hours or sometimes days

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to see them.

I'm going to use a blue pen to point to the areas of brain infarction. Now, the cartoon that I drew for you before is looking at the brain at the side. That's not how this is done. We're looking at the brain through slices in about this plane. I'm pointing now with my hand in kind of a slanted fashion above my ear.

The volume of tissue that we're looking at is a few millimeters thick. So they're being sliced in this direction through the brain.

When the slices start, you're looking at the brain underneath it and so forth, and you almost immediately pick up at the top and going down, an area that is abnormal. I'm going to draw through the bone, the beginning of what you see is an abnormal area with this blue pen producing the sign of the blue arrow. Let me get the kids' crayons.

You start to look at something here on the left side. Now, the left side is on your right, and you're kind of looking from the top down with things inverted. That's just the convention here. As you begin to look farther, you see that this area becomes larger and larger. I'm not

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going to go through every single frame.

Q: Sure.

A: Here you see that the whole portion of the hemisphere, and I'm now pointing at Image 32. This is kind of blacker than this. It means it's in water. You can see what also happened. This structure in the middle of the ventricle here in this frame, and again this is frame Image 32 should be exactly in the midline. The midline is here. You see how this is pushed over.

Q: What is pushed over?

A: See how this thing that ought to be straight is kind of pushed from the left to the right side. The ventricle on the left side with the water containing chamber is almost closed. This is swollen and infarcted brain tissue. So this is erasable, and I'm not ruining anybody's x-rays with it, and it doesn't show up very well, but this whole area of the brain is infarcted.

Now, that's focal, and it's also the precise distribution of the middle cerebral artery. That's what perfuses this. There are portions of brain that are not infarcted, areas of the brain far in the back that are preserved.

The other side of the brain is preserved in

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further sections, and I'm looking now at Image 19. The cerebellum, that's not affected, it's in the bed of another circulation and another route, not Interstate 77 at this point.

See, you're looking at infarction all the way as this series of slices comes down here, and it is all in the circulation of the middle cerebral artery on the left side of the brain. Not only is the brain infarcted, the area of infarction has caused swelling so that this damaged brain is now pushing its way into the property, both property lines of otherwise healthy brain.

Q: Can you give me the size of the focal affected area approximately?

A: Make a fist. That size. The size of your fist.

Q: Is there anything that you can see on these CT scans which supports your contention that a stenotic process was involved?

A: Again, the focal nature of this. It's restricted to the distribution of a single known named cerebral vessel, not other vessels. It's not every place, it's not a watershed infarction, it's not global anoxia. It's a stroke in the distribution of single vessels. That happens because there's something wrong with the vessel.

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That happens 100 percent of the time.
Q: My question for you is there anything that you can see on here other than the fact that you know —
A: I see. No. You don't see blood vessels on this particular study. Now, you can get some idea if you use contrast material, which was not done here, there's really no reason to. Again, that's not a criticism. There are other forms of study that could be done to illuminate that problem, but again, there's no reason to do things like MRI or direct angiography. Those would firm things up a little more, but we have to deal with the evidence we have. And we have enough evidence.
Q: Is there a frame that best shows a complete picture of the stroke better than any of the others?
A: No, they all show it. You have to make a composite out of this. If you were to take one favorite picture away from this, oh, let's see, it would probably be Image 32, because not only does it demonstrate infarction, it demonstrates brain swelling in compartmental shift.
Q: Okay. Thank you.

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A: You're welcome.
Q: What is heparin-induced thrombocythemia?
A: In persons with chronic heparin administration that are carried out for long periods of time for a variety of illnesses this lady doesn't have, there can be suppression of platelet production.
Now, in persons with embolic disease, particularly repeated, recurrent massive embolic disease, the platelets get consumed in persons receiving heparin. That's the acute effect of heparin. It is an acute effect of somebody that is a thrombosis factory, as this lady is. She's thrombosing stuff all over the place. Her platelet counts, her absolute platelet number is going to be diminished.
In chronic heparin administration it's not platelets being consumed. They're not being produced. She doesn't have that.
Q: So you noticed in this particular patient her platelets are probably reduced by half?
A: Sure, but every place she makes a blood clot, she's going to have hundreds of thousands of platelets at the same site being consumed in a clot.
Q: Is heparin-induced thrombocythemia a possibility

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in this patient?
A: No. In my opinion, no.
MR. MISHKIND: Objection.
Q: Is it possible that she simply had a stroke unrelated to the events which hospitalized her?
MR. MISHKIND: Objection.
Go ahead.
A: I don't think so. I think there's a much more probable causal relationship between the event of pulmonary embolization ensuing and regional brain infarction. It's not just happenstance.
Q: I'm not asking you if it's more probable or not. Is it impossible medically speaking or a medical possibility?
MR. MISHKIND: Let me object to the form of the question. You know that's not the requisite degree of belief.
Q: I'm not asking —
A: It's like falling in love, anything can happen, but I don't think that happened.
Q: Okay. Have you ever had a patient who had a stroke which resulted from these circumstances, PE hypertension coupled with —
A: Hypotension.
Q: I'm sorry, hypotension, coupled with a stenotic

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process?
A: Yes.
Q: How many times?
A: Not very many. I don't know, 40 years, ten, a dozen.
MR. KURI: I think we're almost done. Let me take a look through here.
A: I hope to God you're right.
MR. HOLDSWORTH: I have a couple of brief questions.

CROSS-EXAMINATION OF JOHN P. CONOMY, M.D., JD
BY MR. HOLDSWORTH:
Q: Doctor, I think you told Mr. Kuri earlier that you believed that Mrs. Speicher's pulmonary embolism could have been treated to prevent this focal stroke sometime during the hospital stay at Barberton Community Hospital, is that correct?
A: Yes.
Q: Do you have an opinion as to whether or not the pulmonary embolism could have been treated at the point of say February 1st, 2001?
A: Tell me, where was she on February 1st. She went to the doctor that day. Yes, I think it could have been.

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I think by the time she presents herself to the Akron Hospital, she's very sick and having massive troubles, not really evident at the moment she was admitted, but shortly thereafter. But I think on the 1st, yes. What was her admission date at the Akron Hospital?

MR. MISHKIND: She was admitted on the 5th, she was discharged from Barberton on January 28th.

A: On the 1st she saw Dr. Dean Rich. I think had she been treated that day, she would have been saved.

Q: Fair enough. And I believe you also talked with Mr. Kuri earlier about if you were removing the pulmonary issues from this case, if Ms. Speicher had not had a pulmonary embolism, that there was, I wrote this down so I would have it right, a cumulative stroke risk of 3 percent per year, is that correct?

A: Yeah. Now, that again, is about more than three times your stroke rate. It basically reflects her age.

Q: Okay. So that assessment of 3 percent per year is based upon her 77 year age?

A: Three percent cumulative.

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Q: Are there any other factors that you base that opinion on?

A: No, because if she were hypertensive or diabetic, the number would be much higher. Three percent is a fairly low number for somebody 77. It means they're 77 but don't have much else wrong with them.

Q: So a 77-year-old without any other health problems or other diagnosed health conditions.

A: 77, 3 percent; 78 is 6 percent. It adds up, it doesn't get smaller, but the numbers that are built up for concurrent heart disease or for the appearance of malignancy are even higher than that in 77-year-olds.

Q: So when Ms. Speicher progressed to 78 years of age, her cumulative risk would increase to 6 percent?

A: It would have accumulated to 6 percent at the end.

Q: Nine percent at 79; 12 percent for 80, so on and so on?

A: Yes, it's cumulative odds. It gets bigger over time. Now, it may not grow at the same numeric rate per year, but in general that's the progression.

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MR. HOLDSWORTH: That's all the questions I have.

MR. KURI: I don't have anymore questions either. Thank you for your time.

MR. MISHKIND: We will read. If you would reflect on the record, as we've done previously, that the doctor, and I'll be happy to make arrangements to get the transcript to the doctor, but since we're close to trial, rather than extending to 28 days on the reading, we've agreed to 14 days rather than seven days —

MR. KURI: I'm ordering it as soon as you can get it to me.

MR. MISHKIND: — for purposes of the doctor reading it over.

MR. KURI: No problem.

JOHN P. CONOMY, M.D., JD

CERTIFICthe T E

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

I, M. Sheila Noce, the Notary Public within and for the State of Ohio, authorized to administer oaths and to take and certify depositions, do hereby certify that the above-named witness was by me, before the giving of their deposition, first duly sworn to testify the truth, the whole truth, and nothing but the truth; that the deposition as above-set forth was reduced to writing by me by means of stenotypy, and was later transcribed into typewriting under my direction; that this is the true record of the testimony given by the witness; that said deposition was taken at the aforementioned time, date and place, pursuant to notice or stipulations of counsel; that I am not the relative or employee or attorney of any of the parties, or the relative or employee of such attorney or financially interested in this action; that I am not, nor is the court reporting firm with which I am affiliated, under the contract as defined in Civil Rule 28(D).

IN WITNESS WHEREOF, I have hereunto set my hand and seal of office, at Cleveland, Ohio, this ____ day of _____, A.D. 20____.

M. Sheila Noce, Notary Public, State of Ohio 1750 Midland Building, Cleveland, Ohio 44115
My commission expires January 22, 2006

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