THE STATE OF NEW HAMPSHIRE

ROCKINGHAM COUNTY, SS. SUPERIOR COURT

RALPH HOFFMAN, Executor of the Estate of Virginia Hoffman, RALPH HOFFMAN as f/n/b/f of Sydney Hoffman and RALPH HOFFMAN, Individually,

Plaintiff,

vs.

EXETER HOSPITAL, INC. and EXETER HEALTH RESOURCES, INC., et al.,

Defendants.

No. 03-C-970
No. 03-C-841
No. 04-C-162
No. 04-C-594

The discovery deposition of JOHN P. KRESS, M.D., taken in the above-entitled cause, before Etta R. Jones, a notary public of Cook County, Illinois, on the 17th day of August, 2005, at the hour of 10:00 o'clock a.m., at 5841 South Maryland Avenue, Chicago, Illinois, pursuant to notice.

REPORTED BY: ETTA R. JONES

LICENSE NO.: 084-003372

APPEARANCES:

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I N D E X

(WHEREUPON, Kress Deposition Exhibits Nos. 1 and 2 were marked for identification.)

MR. LAX: Would you please swear in the witness.

(WHEREUPON, the witness was duly sworn.)

MR. LAX: Good morning, Dr. Kress. My name is Jonathan Lax. I am from the law film of Nelson, Kinder, Mosseau & Saturley. I represent Dr. Deranian.

On the phone we have Elaine Michaud, who represents Exeter Hospital, and there are a couple other defense counsel that will probably be joining us.

JOHN P. KRESS, M.D.,
called as a witness herein, having been first duly sworn, testified upon oral interrogatories as follows:

E X A M I N A T I O N

BY MR. LAX:

Q. Would you please state your full name for the record.

A. John P. Kress.

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Q. What's your business address where we are today?
A. University of Chicago, 5841 South Maryland, Chicago, Illinois 60637.
Q. Dr. Kress, have you ever been deposed before?
A. Yes.
Q. How many times?
A. Between 20 and 30.
Q. In that case, I am sure you know the ground rules, but I will quickly go over them.
I am here to ask you questions today about the work you have done and the opinions you have formed in this case. I would appreciate it if you would give me complete and truthful answers to those questions.
My questions might not make sense, or they may be confusing. If that's the case, please interrupt me and tell me the question doesn't make sense, and I will try to ask a better question.
If you answer a question, I will assume that you understood it. Because there is a stenographer here, only one of us can

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talk at a time. I will try to make sure you are done with an answer before I ask a question, and I would appreciate it if you try to make sure I am done with the question before you start answering.
She can only take down verbal answers, so we both need to keep our voices up and give yes, no answers instead of gestures.
If you need to take a break at any time, you can. Just let us know.
At the end of the deposition, a transcript will be prepared for you to review and make any necessary changes. Then we would ask you to sign it and get it back to us within 30 days.
Do those instructions make sense?
A. Okay.
Q. Great.
Over what period of time have you given the 20 to 30 depositions that you have discussed that you said you have given?
A. Six years.
Q. Have all of those depositions been in

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the role of an expert consultant in malpractice litigation?
A. No.
Q. How many of those depositions have been as a consulting expert?
A. All but two.
Q. Those two cases, were they cases in which you were a party?
A. One of them, yes. The other, I believe it is called a material witness.
Q. Were those two cases medical malpractice cases?
A. Yes.
Q. What was the nature of the allegations in those two cases?
A. The one -- the first one, a patient who suffered from an epidural hematoma and abscess. The second was a patient who suffered from multiorgan systems failure, including respiratory failure and a prolonged hospital course. Ultimately, the patient died.
Q. Were those both death cases?
A. Yes.
Q. Who were the defendants in the second

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case?
A. Schwab Rehabilitation Hospital.
Q. Were you a treating physician?
A. No.
Q. What was your involvement as a material witness?
A. Let me step back. Sorry.
I was involved in the care of the patient but was not named in the suit, so I guess the answer to your question, was I a treating physician, should be yes.
Q. In the first case that you were a party, what was the nature of the allegations against you?
A. That there was a delay in diagnosing an epidural abscess.
Q. Did you retain experts to help in the defense of that case?
A. It is ongoing.
Q. Are there experts that are reviewing the case to assess compliance with the standard of care?
MR. BROWN: I don't know whether he ought to be testifying about a pending case and

the status of it.
BY MR. LAX:
Q. Are there experts that have been disclosed? Do you know?
A. Not to my knowledge.
Q. In the 20 to 30 less two, so 18 to 28, cases in which you have been deposed as an expert, what are the states of origin of the cases that you have consulted on?
A. Illinois, Florida and New Hampshire.
Q. What's the breakdown in numbers?
A. The vast majority are Illinois. The New Hampshire is this one case. Three or four in Florida, I believe.
Q. In those cases, how has your work been divided between consulting on behalf of plaintiffs versus consulting on behalf of defendants?
A. Somewhere, I would say, 40 percent for plaintiffs to 60 percent for defendants, a rough estimate.
Q. Are you associated with any company that markets the services of experts?
A. No.

Q. Have you ever been affiliated with a company that markets your services?
A. No.
Q. How do lawyers who have retained you find information about you? Do you know the method by which they have come across your name?
A. In the vast majority, it is word of mouth.
Q. Is this the first case you have consulted on in New Hampshire?
A. Correct. I am sorry. That's not correct. There are two other cases that I consulted on with the same firm as the current.
MR. BROWN: The status of those cases you need not comment on.
MR. LAX: That's fine.
BY MR. LAX:
Q. Have any of the cases in which you have been retained as a consulting expert been Federal Court cases?
I will tell you why I am asking. It may help you in answering. I am wondering if you have ever had to prepare a Federal Court disclosure describing all the

federal cases you have served as an expert in.
A. I haven't had to do that. There is one case from many years ago that never went to the point where such information was necessary, but it was a case that was being evaluated by the federal government.
Q. Have you kept a list of all the depositions you have given?
A. Only insofar as I can remember them, but I don't have a piece of paper, no.
Q. Did any of the cases in which you have been involved as an expert involve the propriety of giving Lopressor? Have you ever commented as an expert before on Lopressor use?
A. That specific drug?
Q. Yes.
A. I don't believe so.
Q. Why don't I start more broadly.
Can you describe for me the types of cases in which you have been retained as an expert and in which you have been deposed?
A. My practice is pulmonary and critical care medicine, and so the cases that I have been involved in have focused on a wide variety of

issues centered around those particular disciplines.
Q. Are there any subcategories that the cases tend to fall into that you can break them down for me?
A. It would require me to give you a whole long list, which I am happy to do as best I can off the top of my head, but there aren't any that come to mind, broad strokes categories, if that's what you mean.
Q. Have any of them involved the treatment of patients with DIC?
A. Yes.
Q. How many of those cases or about how many times have you testified as an expert in cases involving DIC?
A. I can't remember.
Q. In those cases was assessing DIC as a possible cause of death part of the work you did?
A. In the vast majority of cases that I have been asked to look at, an assessment regarding cause of death is a part of the evaluation, yes.
Q. Have you ever opined that DIC was the

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cause of death in any of the cases you have worked on as an expert?
A. Contributing perhaps.
Q. Do you know what states those cases were in or who the lawyers were that would have taken that deposition?
A. I can't remember.
Q. Do you know when those cases were pending?
A. No.
Q. Within the last six years?
A. Within the last six years.
Q. Have any of the cases in which you have been retained as an expert required you to consider a possible amniotic fluid embolism?
A. Not to my recollection.
Q. Have the cases you have been involved in as an expert called upon you to testify concerning the resuscitation of critically ill patients in the ICU?
A. Yes.
Q. Have you testified about the propriety of the care given in terms of resuscitative efforts?

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A. Yes.
Q. In cases prior to this?
A. Yes.
Q. In how many cases?
A. More than half.
Q. Okay.
A. That's a broad category that you are touching on, so I will answer it with the condition of its breadth. So within those confines, yes.
Q. Okay.
Were those cases Illinois, Florida, both?
A. Both.
Q. This may be slightly more specific.
Have any of the cases you have worked on as an expert required you to testify about the fluid management of patients in the ICU and their volume status?
A. I can't remember the details. I am sorry.
Q. Would that be part of what you would consider in assessing the resuscitative efforts?
A. Yes. Again, if you are asking in a

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broad category of resuscitation of shock, I have opined on several occasions in that capacity.
Q. Okay.
In those cases do you know whether you were working on behalf of the plaintiffs or defendants?
A. Both.
Q. What are you charging for the time you spend in this case?
A. I don't remember. I think it is $300, but I honestly don't remember.
Q. Is that something you do through the hospital, or is it your own -- done on your own time?
A. It is my own time.
Q. Do you have a separate billing corporation?
A. Can we stop for a minute, please.
Q. Sure.
(WHEREUPON, a brief pause was taken.)
(WHEREUPON, Mr. Mallory entered the proceedings.)

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(WHEREUPON, Greg Peters entered the proceedings via telephone.)
MR. LAX: Back on the record.
BY MR. LAX:
Q. I had just asked you about your fees. You said you think you are charging $300 an hour?
A. Correct.
Q. Do you know -- strike that.
Do you know how much time you have spent working on this matter to date?
A. I don't have an exact number, so it would be a guess.
Q. Can you give me a range?
A. Between 10 and 20 hours.
Q. Have you issued any invoices yet --
A. Yes.
Q. -- that describe the work you have performed?
A. Yes.
MR. LAX: After the deposition I will request a copy of that.
MR. BROWN: I have no problem with that. Just send us a letter of what you want.

We will respond.
THE WITNESS: I am sorry. Do you want me to or do you do that?
MR. LAX: I will get it from him.
MR. BROWN: When it is all over, he will send a letter of the documents he wants. If I have the invoices, we will send them along. If not, I will give you a ring and get them from you.
THE WITNESS: Okay.
BY MR. LAX:
Q. How have you spent that 10 or 20 hours generally? What work have you done?
A. The vast majority of it is reviewing the data, reviewing the data of the records, of course the depositions. I can't tell you the breakdown, but those are the times.
Q. Ken indicated that he could tell me what depositions you have reviewed. Is there a list?
MR. BROWN: Well, I know we sent the doctor I think all of the -- Dr. Deranian, Mr. Hoffman, the anesthesiologist, a nurse, Hoffman, McKee, Daley, Deranian, Burns, Carignan, Dershwitz, and your client's answers to

interrogatories.
MR. LAX: Okay.
BY MR. LAX:
Q. You just showed me a binder, Doctor. That includes those seven depositions and Dr. Deranian's answers to interrogatories. There is a number of flags on different pages in that. What do the flags represent?
A. They represent just points during the various depositions where particular matters that are called to my attention, and so I flagged them for future reference.
Q. As you were reviewing those depositions, anything that you wanted to quickly be able to find again the next time you looked at it you put a flag on?
A. That's correct.
Q. Did you take any notes in addition to putting flags on?
A. No.
Q. Have you taken any notes in connection with your review of the materials in this case?
A. No.
Q. Have you written a report?

A. No.
Q. Is that something you were asked not to do?
A. I don't remember. I just, as a general rule, don't.
Q. Are there other documents you brought with you today that represent your file or the materials you reviewed?
A. There is a folder that is titled Medical Records of Virginia Hoffman that has the medical records of Virginia Hoffman.
Q. Are these all the medical records in this case you have reviewed? Are there any other documents that you reviewed -- better question, are there any other documents you reviewed that are not in one of the two binders that you showed me?
A. Those are the medical records, yes.
Q. I think when we take a break I am going to look at these and I will see if there is anything I want to mark as an exhibit. You can put them behind you because the table is kind of small.
On the medical records

themselves, did you take any notes?
A. I don't believe so.
Q. Have you ever testified in New Hampshire before?
A. No.
Q. Have you ever given courtroom testimony as an expert, or has all your testimony been limited to deposition?
A. No, I have done courtroom testimony.
Q. How many times have you given courtroom testimony?
A. Once.
Q. Is that in Illinois?
A. Yes.
Q. Was that in a case on behalf of the plaintiffs or defendants?
A. Plaintiffs.
Q. What was the name of that case? Where was it pending?
A. I don't recall the name, and it isn't pending. It is settled. It was some time ago.
Q. Do you recall who the lawyers involved were?
A. No.

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Q. Have you ever been to Exeter Hospital?
A. No.
Q. Have you ever met Paul Deranian, my client?
A. No.
Q. Do you have any criticism of his background or training or his qualifications?
A. No.
Q. Do you have an understanding of how the ICU at the Exeter Hospital is staffed?
A. Yes.
Q. Can you give me your understanding of how the ICU at Exeter is staffed?
A. Before I do that, I will ask you to expand on your question so I understand specifically what you want me to refer to.
Q. Well, what I am really interested in is comparing the ICU there to the ICU here at the University of Chicago in terms of the makeup of the staff.
Are there attendings, nursing staff? It is important to me, since you are opining about the care provided to Mrs. Hoffman in the ICU there, to get a sense of your

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understanding of how that ICU is staffed.
A. It is staffed by attending physicians and critical care nurses, as I understand it; ancillary staff such as respiratory therapists, as I understand it.
Of course, as I have said, I have not been there, so I am basing this on reading the records. I do not see it as a teaching hospital, so I don't see that there is house staff there.
With regard to your pointed question, in comparison to the University of Chicago, that's probably one of the main differences between the two institutions as best I can tell.
Q. Are there -- are you aware of intensive care units at other hospitals that aren't teaching hospitals like Exeter where there is not 24-hour coverage by attending physicians?
A. Yes.
Q. In your mind, it doesn't violate a standard of care for hospitals not to have attending physicians at ICU's; is that correct?
A. Can you repeat that question, please.

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Q. Is one of the opinions you are going to have in this case -- strike that.
Do you fault the hospital for having an ICU in which there is not 24-hour coverage by an attending physician?
A. Okay. I just want to be sure because that sounds like a different question than what you asked before.
Q. I rephrased it in a better way.
A. Thanks.
So the issue is not are there attending physicians in the intensive care unit who have training in intensive care but rather are they there 24 hours a day.
I don't believe that there is a standard of care that mandates 24-hour coverage currently in this country; 24-hour in-house coverage, meaning physical presence in the building.
Q. So there are places where there are attendings available by phone for consult?
A. Correct.
Q. Is that your understanding of what was occurring in Exeter in May of 2002?

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A. Yes.
Q. Is it your understanding Dr. Deranian was available by phone even after he left the hospital --
A. Yes.
Q. -- on May 22nd?
In your review of the record, did you see anything that indicated Dr. Deranian ignored a request to stay at the hospital?
A. In a deposition that I read by a family member, there was a discussion about asking whether he might be able or willing to stay. Apart from the family request, I saw no other documentation.
Q. So you didn't see any indication that the nursing staff had asked him to stay?
A. I see no indication of the nursing staff requesting him to stay in the hospital.
Q. Or that any other doctors requested him to stay?
A. Not that I saw.
Q. Okay.
Did you also see in the depositions that he lived five minutes from the

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hospital?
A. I did.
Q. In your mind, is that a reasonable distance for someone who may need to respond to medical emergencies at the hospital to live from the hospital?
A. Sure.
Q. Can you describe for me how the ICU at this hospital is staffed and how it is different from the Exeter Hospital?
A. Well, this is a teaching hospital. The intensive care unit is closed. What that means is that patients admitted to the intensive care unit, the medical intensive care unit, which is where I work, are cared for under the intensive care unit team. That is a board certified critical care physician who takes over primary care duties upon admission to the intensive care unit. Additional physicians involved in the patient's care will play a consulting role.
Q. Is there a separate surgical ICU?
A. There is.
Q. How is that staffed?
A. The surgical ICU is an open ICU. That

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is patients transferred to the ICU from other locations are cared for by their respective primary teams who are managing the patients on the medical ward, and there is not a transfer of responsibility to the primary intensive care service.
Q. Are there attending physicians specifically assigned to the surgical ICU?
A. There are attending physicians who are critical care certified who play a consultative role but not a primary care role.
Can I go off just for a second?
Q. Sure.
(WHEREUPON, a discussion was held off the record.)
BY MR. LAX:
Q. In addition to the attending physicians in the surgical ICU who are critical care certified, are there also interns and fellows? What other medical staff work in the surgical ICU?
A. In the surgical ICU there is a similar model of house staff training, which is interns, residents and fellows, yes.

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Q. Are there any attendings, interns, residents or fellows in the surgical ICU that are just board certified in anesthesia but who don't have a critical care certification?
A. Yes.
Q. Okay.
A. Well, not interns and residents and fellows. They, of course, wouldn't be board certified because they haven't finished their training. So there are attending physicians who are board certified in anesthesia.
What was the second part of your question again?
Q. Who are just board certified in anesthesia who don't also have critical care certification.
A. I don't know the answer to that. I don't know. There are certainly several anesthesiologists who work in the intensive care unit. What each individual's board certification status is I just don't know.
Q. In the medical ICU where you work, are there any attendings who are solely board certified in anesthesia but not additionally

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board certified in critical care?
A. No, not to my knowledge. Indeed, there aren't any attendings that are anesthesia trained except one individual and myself, of course, but I don't practice anesthesia.
Q. And there is 24/7 coverage by an attending in the medical ICU?
A. No.
Q. Can you explain how it is staffed in terms of coverage?
A. There is 24/7 coverage by house staff with 24/7 availability by telephone by faculty and fellows, but the faculty do not spend the night in the hospital.
Q. How frequently would you say is it that you get calls from house staff on patients in the ICU when you are on call?
A. Every night.
Q. Do you also get calls from the nursing staff in the ICU?
A. Rarely because they go through the house staff. On occasion it may be that the answer that a house staff person gives doesn't satisfy the nurse, but generally not.

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Q. What types of issues have you been called on with requests for information from the house staff?
A. Anything that covers the discipline of critical care, which would take a lifetime to discuss.
Q. I promise not to keep you here for a lifetime today.
I have had marked as Exhibit 1 a copy of your August 1, 2005 CV. I had previously only had a copy of your CV from 2003. Can you briefly describe for me what's different about this than the CV I had from two years ago.
A. Can I see the old one.
(WHEREUPON, the document was tendered to the witness.)
THE WITNESS: Well, the old CV doesn't include my research grant, although that was an omission. It was there. I just hadn't put it on.
BY MR. LAX:
Q. Which grant is that?
A. It is NIH K-23 grant entitled Strategies

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for Improving Sedation Outcomes in the ICU.
The other major addition just has to do with the fact that I am the co-director of the American College of Chest Physicians' Critical Care Board Review Course, which is an annual course. So I have done that now for three years, the third one being in about three days. So the third one hasn't been completed yet.
Q. All right.
A. In addition to that, the only other major modifications would be any publication that came after 2003, and there are dozens of those. I could go through them all if you would like, but they are --
Q. I will be able to tell which ones those are.
A. Correct.
Q. The one that you printed out today, is there anything brand new that's not included that would need to be added to make it up to date for August 17th instead of August 1st, any brand new chapters or brand new articles?
A. There is a chapter that was just submitted to Martin Tobin's textbook on

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mechanical ventilation which isn't included here.
The other thing would be the newest edition of the critical care textbook Principles of Critical Care that was written by Hall, Schmidt and Wood; the Harrison's Internal Medicine 16th edition chapter on Principles of Critical Care. Those are the major additions.
Q. Are those -- is the newest critical care text by Hall out yet or is that in the works?
A. It is out, yes.
Q. The same with Harrison's, the 16th edition is now out?
A. Correct.
Q. I just quickly want to go over your education.
You got a B.S. from Notre Dame in 1987?
A. Yes.
Q. Got your M.D. at St. Louis University School of Medicine?
A. Correct.
Q. Would you describe the internship and residencies you performed.
A. I initially did a preliminary internship

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in Chicago at St. Joseph Hospital, and then from -- that was for one year, and then from that point forward I have been at the University of Chicago.
So I was a resident in anesthesiology from 1992 to 1995 and finished that training, and then I was a resident in internal medicine, again at the University of Chicago, followed by a fellow in pulmonary and critical care at the University of Chicago from '95 to '96 and '96 to '99, respectively.
Q. Okay.
You are board certified in anesthesiology?
A. Correct.
Q. And you passed that exam the first time you took it?
A. Yes.
Q. What other board certifications do you hold?
A. Board certified in internal medicine, board certified in pulmonary medicine, board certified in critical care medicine.
Q. Are those tests that have to be renewed?

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I mean you have to sit for exams periodically for those?
A. The anesthesiology, no. The other three, yes.
Q. When is the next time that you will have to sit for those exams?
A. They are every ten years.
Q. So you have a while to wait.
You are licensed in Illinois. Do you hold any other medical licenses?
A. No.
Q. What's your current title here at the University of Chicago?
A. Assistant Professor of Medicine.
Q. You have been an assistant professor for how long?
A. Since 2001.
Q. When you were first hired by the University of Chicago, what was your title?
A. In 1999, Instructor of Medicine until 2001.
Q. What are the next steps up the ladder that you would be eligible for?
A. As far as academic appointments?

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Q. Yes.
A. The next would be associate professor.
Q. Have you been up for that or will you be up for that?
A. Not yet. It is a fairly regimented hierarchy, so I have a couple more years for that.
Q. Then after that?
A. Professor.
Q. Do you have any titles that you hold in addition to being an assistant professor?
A. I am the Director of the Pulmonary and Critical Care Procedure Service.
Q. Can you tell me what that entails?
A. Yes.
For about three and a half years now we have had a service that has provided interventions for patients, which run the spectrum of all the various invasive procedures that are done, and so those include central venous catheters of all types, those include lumbar punctures, they include chest tube placement, they include thoracentesis, they include paracentesis. Those are the main ones,

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and then a whole variety of miscellaneous things.
Q. Are those -- those are all procedures that occur in the medical ICU?
A. No. They occur throughout the hospital and also in the outpatient setting.
Q. How many people work under you in that service?
A. I have one nurse who is full time, I have seven faculty members, and then the service is open to medical house staff and students, so that it is a rotation that they can sign up for.
Q. Do medical students rotate through that service for training in those procedures?
A. Correct.
Q. Are you responsible for the clinical teaching of the medical students that rotate through the service?
A. Yes, I am.
Q. How much of your time spent in the hospital is devoted to that service versus the other work you do in the hospital?
A. Well, I rotate on the service myself typically about four times a year, and those are two-week blocks. The administrative duties never

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go away.
Q. Can you describe for me sort of all the activities you do at the hospital how much is devoted to administrative activities, how much is devoted to teaching of students, residents, fellows and how much is devoted to direct patient care?
A. I can't give you a number that adds up to a hundred percent because they all overlap. Of course, when I am doing the procedure service, I am also teaching because it is the nature of what we do, and ditto with patient care.
Indeed, even the research I do there is teaching involved because we have trainees who are learning how to do that, and the research I do is patient-care based. There is a lot of overlap.
The administrative part I generally do not involve trainees because it is not really relevant to their experience.
Q. How much of your time is spent in the medical ICU?
A. On service?
Q. Or teaching.

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A. Well, when I am on service, again, the way the rotations are set up is two-week blocks at a time. Again, I typically have four of those a year. That's where patient care and teaching is occurring extensively.

In addition to that, I will perhaps be doing a procedure and be in the intensive care unit, so there is overlap there. Occasionally if we have a patient on a study, I might be involved with house staff coming up and asking questions. That's done on a more sporadic basis.

Q. Do you have your own client base? When there are patients involved in studies, are you ever the primary responsible physician for patients in the ICU?

A. When I have patients on studies, I am not involved in their care. Those two roles never cross over. So if I am on service and a patient is on one of my studies, another individual who works with me on the study will take responsibility for proceeding with the protocol of the study, and I will disconnect myself from that.

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Q. When you are on service, are there -- are you the primary responsible physician for the care of patients in the medical ICU?

A. Yes.

Q. Are there instances when you have been the primary responsible physician for patients in the medical ICU when you have consulted with anesthesiologists outside of the ICU on issues of the ongoing care of those patients?

A. Absolutely.

Q. On what types of issues?

A. Any issue that would be relevant to the training and expertise that an anesthesiologist would have.

Q. Can you give me examples?

A. Airway management.

Q. Can you think of an instance where you have consulted with an anesthesiologist on fluid levels?

A. Not typically actively. Certainly there are times when we work together. I am open to others' input, by all means.

So, for example, a patient who may need to have an intratracheal tube placed, I may ask an anesthesia person or team to come, and we may have a discussion about the patient's fluid status vis-a-vis the planned intervention, in which case we will discuss back and forth. So it is an open collegial format for these kinds of things.

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Q. In your teaching role, are there classes that you currently teach, classroom lecture, or is it all clinical teaching in the hospital?

A. No. There are many, many classroom lectures.

Q. What classes are you currently teaching?

A. Well, there is a curriculum while house staff are in the intensive care unit on the rotation with a daily hour lecture. That's a didactic lecture.

We have a daily morning report in pulmonary and critical care which I attend and many times run. I am not the one who is in charge of it, but in the event that the person in charge is unavailable, I often take the role of running it. In fact, I just finished the last two weeks while one of my colleagues was on vacation.

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There is an internal medicine morning report which I am invited to speak at probably three or four times a year, and that's another formal didactic session that covers pulmonary and critical care medicine issues.

There is a morbidity and mortality conference which is run by internal medicine that happens weekly that I have been asked to be a discussant at, and typically about three or four times a year for that.

There is a lecture series that is given to internal medicine house staff, in particular, the junior house staff, that is interns or new doctors, and I have in the past been asked to lecture on sepsis, shock, pulmonary embolism, central venous catheter placement. Those are the ones I can remember.

Do you want me to keep going?

Q. If there are more didactic lectures that you routinely give, yes.

A. I give lectures to the medical students as part of the curriculum, and the lectures there, again, are on shock and respiratory failure.

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I give lectures to the anesthesia residents, and those topics are respiratory failure, preoperative pulmonary evaluation.

Those are the ones I can remember.

Q. Do you have a syllabus or prepared materials to the didactic lectures you give or teaching slides that you use for the lectures you give?

A. Yes.

Q. Are those something that if I send a letter through counsel you would be able to provide for me?

MR. BROWN: There may be propriety interest in that, so I am not so sure I am going to agree to it as we sit here, but we can consider that.

THE WITNESS: It is all electronic. These are Power Point slide presentations. So, yes, by all means I have them. The answer to your question is they are available, yes, typically for a lecture that I give.

I wasn't finished, but I am

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happy to quit if you want me to.

MR. LAX: If there are more lectures, I need to hear them.

THE WITNESS: I print out the handout for -- that's the usual routine.

The emergency medicine residents, I just gave a talk to them on sepsis and shock the other day.

You want just the ones that are within the institution; is that right?

BY MR. LAX:

Q. Well, you lecture outside of the institution as well?

A. Yes.

Q. Those are listed in your CV?

A. Yes.

Q. On any of your -- in any of your didactic teaching, do you have reading materials that you put on a syllabus for the students?

A. The way that my lectures and most individual's lectures are formatted is to discuss the principles of the pathophysiology and specifically to allude to references that might be relevant. So, for instance, a person might

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have a handout of the Power Point presentation where a particular article would be referenced, and that individual would be free to go to the library and look it up if they chose to do that.

Q. Are there textbooks that you consider to be reliable sources or authoritative sources for critical care medicine?

A. There are numerous textbooks that are available on the topic of critical care medicine. I couldn't possibly list them all for you.

Q. Which do you find yourself turning to most frequently if you have a question about critical care medicine?

A. You want me to list them? There is a long list.

Q. The top ten. I am interested in what you consider to be the textbooks on critical care medicine. If I want to go get smart on critical care medicine, where should I start?

A. Paul, Schmidt and Wood I just mentioned. I have written in that book. I mentioned Martin Tobin's book on respiratory failure and mechanical ventilation. Of course there is -- Delinger has a textbook on critical care

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medicine. I believe Parillo has a textbook on critical care medicine. Civetta, C-i-v-e-t-t-a, I think has one on critical care medicine. Those are the ones that come to mind off the top of my head.

I am not sure I could label anything as authoritative. The messages tend to be similar across those, but as one might imagine, when you have a large number of resources, there may be slight differences in the way that they are laid out with regard to a particular topic, for instance.

Q. You mentioned that you participate in the morbidity and mortality conference. Can you describe what that conference entails?

A. Sure.

That's a teaching session for -- that is formatted where a resident will present a case from start to finish. More often than not, it is a case where a patient died, so there is autopsy material to discuss, but not always.

Then usually the format is that the resident discusses the case. The chief

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resident who runs the conference will intermittently stop and ask questions to the various faculty members who are invited. Typically there are two or three faculty members from different disciplines.

For example, a case of respiratory failure from pneumonia might include a pulmonary and critical care physician, an infectious diseases physician and perhaps a pathology physician depending on the information, and those various individuals will discuss aspects of the case as it is unfolding.

Q. Have any of the cases that have been discussed since you have been participating in those conferences dealt with patients who had DIC?

A. I am sure they have. I can't recall them off the top of my head.

Please understand that this is something I do virtually every day in one way or another, so it is just impossible for me to keep track of a specific case. But DIC is certainly something that we see in the intensive care unit and is discussed on a fairly regular interval.

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Q. Does it have a high mortality rate associated with it?

A. Depends on what the underlying cause is.

Q. And what are some of the underlying causes?

A. Well, sepsis is one of the most common.

Q. In this case, you are aware that Mrs. Hoffman had a diagnosis of DIC?

A. Correct.

Q. From reviewing the records, what's your understanding of the cause of the DIC in her case?

A. It isn't entirely clear. I suspect, based on the information available, that the DIC was related to the IUFD, intrauterine fetal demise.

Q. What specific records did you look to -- if at any time you need to look at the medical records, please do.

In trying to come up with a cause for Mrs. Hoffman's DIC, what material was relevant?

A. The medical records.

Q. Anything in particular that helps you at

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least come up with a possibility that it was intrauterine fetal demise?

A. Certainly the history, of course, that she had that is compelling. The absence of a plausible alternative explanation is the other thing.

Q. Did you determine whether or not she had underlying sepsis?

A. To the best of my ability, I don't see evidence of that based on the information that's available.

Q. What types of things would you look to in trying to assess whether she had developed sepsis? What portions of her medical records? What tests? What clinical information would be relevant to that information?

A. Sepsis is a syndrome, and it has a concrete definition.

Q. Can you define it for me?

A. Yes. It is the presence of a condition known as Systemic Inflammatory Response Syndrome or SIRS, S-I-R-S, all capitals.

SIRS is present when a person has at least two of the following four findings,

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which are a white blood cell count that is out of the normal range, either high -- too high or too low, a respiratory rate above 20, a heart rate above 90 and a pertubation of temperature away from normal in either direction, either too high or too low; 36 to 38, I believe, outside of that range centigrade.

If a person has two of those or more, they have SIRS. If a person has SIRS and they have a known or suspected source of infection, they have sepsis.

So she had SIRS, but I would say, based on the evidence I see, that the known or suspected source of infection data point is lacking.

Q. What would you expect to see clinically if that was present? How would her test results or her clinical presentation look different if she did have some type of underlying infection?

A. Well, the findings for infection are non-specific and broad, but the things that I would look for would be a history to suggest the presence of infection somewhere in the body. For example -- this would take a long time to go

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through them all. If it is okay, I will just give some examples.
Q. As quick a summary as you can that will make sense.
A. If you are thinking of respiratory infection, is there cough, fever, sputum, purulent-looking secretions, things of that sort, breathlessness? What is the white blood cell count? I mentioned temperature. Is there a temperature? Does the patient have other associated symptoms; chills, things of that sort?
What are the findings on physical exam that would support the presence of an infection wherever in the body that you are trying to identify and then complimentary laboratory data. The white blood cell count, for instance, being one of the more concrete things.
If you are looking at the lungs, a chest x-ray, for instance. Those sorts of things.
Q. Is there anything in Mrs. Hoffman's chart that enables you to rule out the possibility that, in addition to SIRS, she also had an underlying infection which would enable

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you to say she had sepsis as well?
A. All of those examples that I just gave, in her case there were none or very few, and I can't recall, as I sit here now, any that would suggest the presence of infection.
Q. Can massive blood transfusion lead to infection?
A. Massive blood transfusion can lead to SIRS and massive blood transfusion can, in fact, trigger DIC, but massive blood transfusion rarely causes infection. It is conceivable that the blood product is infected, contaminated, but that's extraordinarily rare.
Q. Does being on mechanical ventilation increase the risk of infection?
A. It does, but the time here is too short.
Q. How much longer would she have needed to remain ventilated before she developed the risk of infection?
A. Can you specify what kind of infection?
Q. Anything that would then enable you to say in addition to SIRS she also had sepsis.
A. One of most common causes of infection in the person on mechanical ventilation is

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pneumonia. In fact, it is titled ventilator associated pneumonia. A traditional definition is ventilator associated pneumonia is seen on a patient who has been on mechanical ventilation beyond 48 hours.
Q. So I just want to make sure I understand.
You don't see any evidence that in your mind leads you to believe she did have an infection so that you could say she had sepsis, but am I right there is nothing that enables you to rule it out? There is no clinical marker that says, no, she definitely did not have sepsis?
A. Ruling it out with one hundred percent certainty in a woman who is critically ill is never possible, never. In her case, I am as close to certain as one can be in the context of a critically ill patient with multiorgan failure.
The biggest reason is that the rapid occurrence here did not allow her time to develop a hospital acquired infection. She wasn't in the hospital long enough, and there was nothing upon her presentation from home that would suggest to me that she was infected before

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arrival. So that's the deduction I would make.
Q. You mentioned multiorgan system failure. Is that something that Mrs. Hoffman was experiencing during her stay in the ICU?
A. Yes.
Q. What organs were failing in her case?
A. Well, her circulation, her respiratory system, her brain, transiently at least.
Q. Is there a definition for multiorgan system failure beyond what the plain words mean, that it is more than one organ that's not working?
A. There are various scoring systems that have been developed. There isn't just one, but in plain laymen's terms, what you said is right.
Q. And is it associated with a heightened mortality risk for patients in the ICU?
A. Absolutely.
Q. Is one of the scoring systems that you referenced the Apache II, or is that a different scoring system?
A. The Apache II as a different scoring system. It is a severity of illness scoring system which has three versions, I, II and III.

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The one most typically used is II, and it has been a validated system for 20 years which is used to categorize severity of illness in intensive care patients at an epidemiologic but not case individual level.

Q. Is that a scoring system that you have used in your practice?

A. I use it every day when I enroll patients in research trials. I don't use it as a matter of routine in every individual human that I take care of, no.

Q. Is a person's Apache II score associated with a set risk of mortality in the way that the test is set up?

A. Not in the individual patient, but in the category that you are dealing with, yes. So it isn't a score that you can use to counsel patients or family members about their individual likelihood. You can counsel them insofar as you can say epidemiologically you are in a high risk category of patients, but you can't say in your particular case, Mr. So and So, your Apache II score is this, so your predicted mortality is this.

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Q. In this case, did you attempt to develop an Apache II score for Mrs. Hoffman?

A. It is not possible because the Apache II is a score that was established with a 24-hour ICU interval. So you have to have the worse value within 24 hours. She wasn't in the ICU long enough to have a score if one were to wish.

Now, you could take her worse values for the time she was there, but that would be falling outside of the validation of the test as it was originally published in 1985.

Q. But are there data points in her chart that, if I asked you to assume she was there for 24 hours, you could take those points and put it on Apache II and come up with, at least epidemiologically, a raw number for severity of her illness and then figure out what the associated mortality rate is?

A. Let me say it again because maybe I didn't make it clear.

The answer to your question is no. I can do a number. I can punch a number in, fill in the blanks, so to speak. That's easy to do, but without the 24-hour interval, I am making

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an assessment that was not the way that the test, back in the '80s when it was established and then validated, was performed. So I am falling outside of the guidelines and, strictly speaking, I am using a value that has no concrete merit.

Q. In any of your studies that you have done, have you tried to assess the impact of various treatments on the outcome in ICU patients?

A. Yes.

Q. In those studies, have you identified independent predictors of increased mortality?

A. In my own particular work?

Q. Yes.

A. None of the studies that I have ever done has identified an intervention that we sought to improve mortality, but many of the studies that I have done have identified variables associated with increased mortality.

Q. Is heightened Apache II score something that's increased -- associated with increased mortality?

A. Yes.

That's not my work. That's

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work that has been generated for 20 years. We use the Apache as a measuring stick because it's been so widely validated.

I should just go back, because we are on a little bit of a tangent here, to say that your original question, I believe, was a question about multiorgan system failure. Apache II is not an assessment of multiorgan system failure. It is a different test. I just want to be sure we are clear on that.

Q. What's the test for multiorgan system failure?

A. There are several, but they assess various organs, systems and their function.

Now, Apache does have that as a part of its assessment, but it does not purport to evaluate multiorgan system, per se -- multiorgan system failure, per se. It is a severity of illness score is what it purports to measure.

Q. Even though Mrs. Hoffman didn't remain in the ICU for 24 hours, if you were to take the worse data point for each of the 12 categories for Apache scoring for her situation, would a

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heightened number allow you to say that the severity of her illness is greater than it would be if she received a lower number?
A. I would say that not related to the Apache II score but related to other observations, both in the medical literature and in my own experience.
Q. Okay.
A. But I wouldn't connect it directly to the Apache. I wouldn't use the Apache as a tool here.
Q. That's because of the time she was in the ICU?
A. And for the reasons that I already said, which is it is a good epidemiologic tool, but it is not a good tool for individual patient assessment, which is why, as a matter of practice, I don't routinely use it, and I think most people that practice critical care would say that, that while they can use the tool to look at their overall performance, perhaps, in their ICU, they don't use it as a number to counsel an individual patient or the care of an individual patient.

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Q. I think I understand. Thank you.
MR. LAX: Why don't we take a quick break. I have to stretch my legs, find a men's room, and then we will get back on the record.
(WHEREUPON, a recess was taken.)
MR. LAX: Back on the record.
BY MR. LAX:
Q. Are there specific papers or chapters identified on your CV which in your mind relate to the issues in this case and the opinions that you have developed in this case?
A. It may take me a minute.
Well, I will just start by covering all the teaching that I outlined. As I said to you, the topic of shock is one that I have been asked to speak on in many different capacities within the institution, so I can't count the number of those.
As I said, for the last three years, I am the director or co-director of the American College of Chest Physicians' Critical Care Board Review Course and have lectured on the topic of shock. In fact, in 2002 I lectured on

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the topic of shock at the, at that time, Society of Critical Medicine and American College of Chest Physicians when the two groups had merged. Critical Review Board, of course.
Q. Let me interrupt you for one second.
In this case, it is your opinion that Mrs. Hoffman had shock?
A. Correct.
Q. Starting at what point in her care? When did she develop shock?
A. If I answer that, we may go off the point here, which I am fine to do that.
Q. Remind me, please.
A. I will.
There are several publications which allude to either the management of patients with shock -- because I do critical care medicine, shock is one of the most common reasons that patients come into the intensive care unit. So in some capacity, most of the clinical investigations that I have published have included patients for whom shock was a relevant issue.
Looking at the book chapters,

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in the second edition of Principles of Critical Care, there is acute right heart syndromes, which is a form of shock.
Q. Which page of your CV?
A. I am sorry. Page 9. It is number 2(b).
The Harrison's chapter certainly discusses shock and its management, Harrison's Principles of Internal Medicine 16th edition. That's number 5.
I see a typo here, actually. It is then number 1. I need to fix that typo, but the third edition of Paul, Schmidt and Wood on page 9 covers intravascular devices, which would include their use in the management of shock.
(WHEREUPON, there was a brief interruption.)
THE WITNESS: Under the invited presentations, there is a wide range of topics that would be relevant here. I could go through and try to list them all, but there are many here that would be relevant.
Certainly, as I alluded to, the issue of shock being a topic for presentation at

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the board review courses is probably the one that's the most poignant.
BY MR. LAX:
Q. Are there any others? When I read down the list of invited presentations, I saw central line placement. Is that a presentation that has information in it which you draw on in coming to your opinions in this case?
A. It could perhaps, yes.
Q. What was that presentation on, the method of central line placement or the complications of central line placement? What did it cover?
A. The indications, methods, complications.
Q. What complications did you identify?
A. The common complications of central venous catheter placement include pneumothorax infection, hemorrhage. Those are the major ones.
Q. As part of your work in this case, did you determine whether there was an iatrogenic injury as a result of the central line placement?
A. Yes.
Q. Is that the cause of the thoracic hemorrhage?

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A. As far as I can see, yes.
Q. Any others, going down this list, that you think are specifically relevant to the opinions you have in this case? I see shock on number 33 on page 13. Sepsis on 34.
A. Any topic that covers sepsis would be relevant, and there are numerous topics on sepsis.
Q. Are there written materials that go along with these presentations?
A. There are for some of them. I can't say that there are for all of them.
MR. LAX: I guess what I will do is I will send a letter requesting written materials. You guys can decide if they are proprietary or tell me if we can limit it to the use of this case. If there is a way we can get copies of the ones he has identified as relevant.
BY MR. LAX:
Q. Are there any others looking through it?
A. Again, going now to page 15 and moving on, we talk about the various different lectures on sepsis. There is one here, number 56, which is acute right heart syndromes, which isn't

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specifically relevant to this case but falls under the umbrella of shock.
Q. Can you define acute right heart syndrome for me?
A. Acute means short onset as opposed to chronic. Right heart syndromes means conditions that lead to acute right heart failure.
Q. And --
A. And that is under the broader category of shock.
Q. Now, did Mrs. Hoffman have --
A. She did not.
Q. -- right heart syndrome?
A. Sorry. Are you done?
Q. Yes.
A. She did not.
Q. I see a couple of presentations on central venous catheters. Are those relevant to the work you have done in this case and the opinions you have formed in this case?
A. Yes.
Q. How so?
A. She had a central venous catheter placed in the midst of her shock, so as a part of the

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management of her shock, and suffered an apparent complication related to its attempted placement.
Q. Was that during the initial surgery? When in time?
A. It appears, from the information I have, that it was related to the initial attempts at placement of her central venous catheter, which was during her first surgery for her vaginal bleeding.
Q. Were there any clinical -- peer review clinical investigations that you think are particularly relevant to the work you have done in this case, to the opinions you have formed?
A. Well, specifically, no, but since your previous questions expanded quite broadly, then one might take a broad stroke's view of this and say that if there are any papers here that cover the topic of critical illness and circulatory instability, not directly related to this case, then the answer is yes, there are many, but nothing concretely specifically related to shock in a young woman who is hemorrhaging.
Q. I promise I am going to be off your CV and background and onto this case shortly. I

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have a few more questions I need to ask though.
What are your current research interests?
A. My current research interest is in the management of ICU patients regarding sedation during mechanical ventilation.
Q. Are you still doing research -- I saw on your web page that another clinical research interest was assessing pulmonary edema in the critically ill. Is that something you are currently working on?
A. That paper is being written as we speak.
Q. What are the dangers of pulmonary edema? What dangers does it pose to a patient in the ICU?
A. The biggest danger is compromising respiratory function.
Q. What can that lead to? I mean what complications can arise?
A. Ultimately it can lead to breathlessness, it can lead to agitation, it can lead to decreases in oxygen levels in the blood, it can lead to respiratory failure.
Q. Can it lead to hypoxia (sic)?

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A. That's what decreases in oxygen level in the blood is. That's hypoxemia.
Q. Hypoxemia?
A. Yes.
Q. What causes pulmonary edema to develop in patients in the ICU?
A. There is a very long list of possibilities.
Q. Are there a couple of accepted general things that are known to cause pulmonary edema?
A. The usual approach to pulmonary edema is to first categorize the edema as forming because of a high pressure or a low pressure phenomenon. So the edema -- the fluid in the air sacs in the lung can get there either because the pressure in the vessels is elevated, so-called high pressure edema, or that the vessels are dysfunctional and they lose their integrity so that fluid and protein can leak across the capillaries, the vessels, into the airspaces. That's the first big branch point in the assessment of pulmonary edema.
Under each of those two subcategories, high pressure or low pressure,

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come a variety of additional explanations or causes, which would be myriad.
Q. Can providing a critically ill patient too much fluid lead to the development of pulmonary edema?
A. Yes.
Q. What are the signs and symptoms of pulmonary edema if its cause is overhydration or providing too much fluid?
A. The signs -- the symptoms would be increasing breathlessness, respiratory distress, sometimes coughing.
The signs would be the respiratory rate is increased, the oxygen saturation is falling, the lungs show signs of edema, that is crackles on physical exam, the chest x-ray shows evidence of filling of the airspaces. Those would be the common things.
Q. Can swelling be a sign of the development of pulmonary edema?
A. Swelling where?
Q. The face, the hands.
A. Swelling is not a sign of pulmonary edema. General edema -- I guess is what you mean

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by swelling -- may be associated temporally with pulmonary edema, but one does not necessarily beget the other, and one does not signal or assure the presence of the other.
Q. But is that something that you could look to that might cause you to suspect pulmonary edema or want to try to rule it out?
A. The finding of diffuse whole body swelling in general is a very non-specific sign, so it isn't a particularly useful indicator of pulmonary edema. The various findings that I just outlined for you are much more useful than the finding of swelling, especially in the dependent regions of the body, the extremities.
Q. Are there other physical signs or symptoms that might cause you to suspect pulmonary edema? I mean in addition to the clinical tests you mentioned, are there other clinical factors you would look to that might cause you to suspect the development or heightened risk of development of pulmonary edema?
A. I think we are talking very general categories here because you are talking about a

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common condition syndrome, if you will, that has a very broad differential diagnosis. So within those limits, the presence of pulmonary edema can be discerned best by what I just outlined.

The history is certainly important and a part of the initial evaluation. So a history that would signal to me a risk of pulmonary edema, certain things that are associated with pulmonary edema, congestive heart failure, for instance, would be very important. That's just one example.

Q. In this case, by your review of the medical records, did you see anything that might cause you to be concerned that Mrs. Hoffman was in danger of developing pulmonary edema throughout the course of the night?

A. Well, statistically speaking, a patient who has received massive blood transfusion is at risk for developing lung injury and pulmonary edema. Statistically speaking.

In her particular case, there was no evidence, based upon any of the data that I had available to me, to suggest that she either had or was signaling the development of pulmonary

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edema. Absolutely none.

Q. What parts of her chart would you look to to try to make that determination?

A. The history -- the whole medical records; the history, the physical findings, the nursing notes, which are a part of the history, the laboratory data, the ICU and operating room flow sheets, the chest radiograph reports. All of that.

Q. So do those things enable you to rule it out definitely, or they don't cause you to suspect that it might be occurring?

A. I am going to look here for a minute, if that's all right.

Q. Please do.

(WHEREUPON, a brief pause was taken.)

THE WITNESS: No, I don't see anything.

BY MR. LAX:

Q. You don't see anything that allows you to specifically rule it out?

A. The absence of a significant derangement in oxygenation, that is her oxygen level was

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acceptable, the absence of a report on the chest radiographs of pulmonary edema, the absence of physical findings disclosing the presence of crackles all allow me with a high degree of confidence to rule it out.

Q. You have written some papers on the utility of physical examination compared to invasive monitoring for certain conditions?

A. Yes.

Q. Does that work relate in any way to the opinions you have formed in this case?

A. The only paper that I have published on that refers to a condition called autoPEEP, which specifically is not relevant to this case.

Q. Is autoPEEP -- can you define that for me?

A. AutoPEEP is a condition where the air sacs remain filled at the end of a quiet breath. Normally when we exhale the air sacs empty and they are at the same level of pressure as the pressure in the atmosphere.

AutoPEEP, which is most typically seen in patients with obstructive lung disease, asthma or emphysema, is seen -- strike

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that.

AutoPEEP is a condition where the air sacs are unable to fully empty because of the bronchial narrowing, so that at the end of a breath, the air sacs are still inflated and are still under pressure.

Auto intrinsic is another word that's used, meaning it is within the body, not from a machine outside. PEEP, positive end expiratory pressure, means that those air sacs are still inflated.

Q. Mrs. Hoffman was mechanically ventilated after her return to the ICU in this case?

A. Correct.

Q. Is there a way to assess what her PEEP was? Is that a ventilator setting?

A. The extrinsic PEEP is a ventilator setting, yes, and that's easy to assess. It is a number of a button on the machine.

Q. The fact that she was mechanically ventilated, does that cause any difficulties in assessing her volume status?

A. The assessment of volume status can be complex in patients who are mechanically

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ventilated. It can be complex in any patient. The fact she is mechanically ventilated means that the person making the interpretation, if you are specifically referring to looking at intravascular pressures, must take into account the contribution of the respiratory system to those measurements. But having said that, that can be and should be able to be done by a trained clinician in my view.

Q. In the ICU you work at at the University of Chicago, do you ever have occasion to care for obstetric patients?

A. Yes.

Q. What are some of the routes by which they get to the ICU? What are some of the obstetric complications that tend to land patients in the ICU here?

A. Any obstetric patient at our institution who requires critical care is transferred to the medical ICU and is managed by the medical ICU team. That's the format that we follow. Of course, the obstetrical issues are managed by the obstetricians, but all the other issues are managed by the primary team.

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The most common reasons for admission to the intensive care unit in the obstetrics ward patients are asthma, peripartum complications such as preeclampsia and eclampsia and lung injury, lung air space edema problems, and then the last would be complications related to the gravid uterus, and those would fall under big categories of delivery problems, including hemorrhage, pulmonary embolism and shock.

The obstetrical experience is generally a straightforward and pleasant one, but when obstetrics and critical care cross, those two disciplines often lead to very sick patients.

Q. Are there OB's on the staff here that you have had occasion to work with whose clinical judgment you respect?

A. We have a great obstetrics group here.

Q. Have you ever had a basis to question one of the OB's diagnoses about the cause of an obstetric complication?

A. Not that I can think of.

Q. If one of the OB's here whose judgment you respected felt that a patient had DIC and made a working diagnosis of an amniotic fluid

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embolism, would you second guess that?

A. I can't answer that question. It is a hypothetical question. I don't know how to answer that.

Q. Well, it is a hypothetical question, I know.

A. Yes.

Q. If that's an --

MR. BROWN: You have to give him enough parameters within the hypothetical so he can answer it.

MR. LAX: Let me do this.

BY MR. LAX:

Q. In this case, you are aware that the OB who cared for Mrs. Hoffman suspected an amniotic fluid embolism?

A. I am.

Q. Are there -- have you taken any steps in this case in your review of the medical record to assess that diagnosis?

A. Yes, I have considered that. Absolutely.

Q. In this case, do you agree or disagree with that as a plausible diagnosis?

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A. Amniotic fluid embolism is a diagnosis that is rarely made with certainty. Amniotic fluid embolism is often an inferential diagnosis that is made when in the peripartum period cardiopulmonary compromise occurs, and so in that context, certainly it is a relevant consideration.

The reason -- so it is conceivable in my view that amniotic fluid embolism contributed to the patient's coagulation disturbance around the time of the delivery of the baby. That's a very different situation than 12 hours later when her ultimate clinical deterioration occurred after the uterus had been removed for some time, in which case I think the issue of amniotic fluid embolism is no longer relevant or feasible.

Q. Have you treated patients or have patients been treated in the medical ICU here while you have been here where amniotic fluid embolism was a suspected diagnosis?

A. Yes.

Q. In those cases, what has the mortality rate been?

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A. Well, in the suspected or in the confirmed?
Q. Let's do them both.
A. In the suspected, the mortality rate is high. I don't know what number to give you.
One of the problems with answering that question is that the mortality rate contributes to the confidence in the diagnosis, meaning after the patient has died surprisingly, which is usually the case in an obstetric. Most people don't except an obstetric patient to die. In fact, I would say almost never is that an expected outcome.
So when an obstetric patient dies, there is often a careful consideration of why that may have happened, and when that careful consideration leads to no clear alternative explanation, in the proper clinical context, one may make a deduction that amniotic fluid embolism contributed. That is clearly the most common scenario that we see. So it is after the fact when one says I can't understand why this particular individual died and it occurred at the time or around the time of delivery that one may

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make a deduction.
In my life, I don't know personally of a case where I have ever had utter confirmation. The utter confirmation of amniotic fluid embolism is rarely obtained except at autopsy. It is conceivable that at some time in my life we had an obstetric patient with an autopsy that showed amniotic fluid in the pulmonary circulation, but I can't remember that. So it is an inferential diagnosis in the vast majority of cases.
Q. In your mind, it is conceivable that she did have an amniotic fluid embolism at or shortly after the time of delivery which contributed to the DIC?
A. I believe that's conceivable. I don't have any way to prove that one way or the other, but it is conceivable.
The time delay between that initiation of the bleeding trouble and her ultimate death makes it impossible, in my view, with any reasonable degree of certainty to make a connection between a diagnosis of amniotic fluid embolism and her, the patient's, death. So there

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is an important distinction between those two.
Q. Let me make sure I understand.
She may have had it, and you can't prove one way or the other whether she had it, but in your mind, based on your review, you don't think that's what caused her demise several hours later. Is that a fair summary?
A. That's a fair summary, yes. I will just add to that that the reason I say she may have had it is that her reason for bleeding and developing the coagulation disturbance that she had labeled DIC has no other obvious explanation that I can see.
Q. Does she have other predisposing factors that make it a reasonable thing at least to include on a list of differential diagnoses for the hemorrhage? Are there other risk factors for AFE or clinical signs consistent with AFE that you saw in the record?
A. No. It is often a diagnosis that's established by exclusion.
Q. Are there any factors which have been linked to heightened risk for AFE that you are aware of?

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A. In this case?
Q. Generally, and then in this case did they exist.
A. None that I know of for certain. I am not aware of any literature connecting an increased risk of amniotic fluid embolism and fetal demise. If such literature exists, I just don't know of it.
Q. Okay.
A. Certainly the presence of her fetal demise -- I mean, she has a dead fetus in her uterus -- could contribute to the cascade of DIC in the absence of amniotic fluid embolism. That's the other possible explanation.
I probably should just add that because I think I made a quote earlier where I said I didn't see anything else, and so that's incorrect. That's the other thing. She has a dead fetus in her uterus, which certainly may contribute to the coagulation disturbance without having amniotic fluid embolism.
Q. So there have been OB patients that have expired in the ICU during your tenure at Chicago --

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A. Yes.
Q. Are there patients -- obstetric patients that have died in the ICU after having periods of gains in terms of neurologic symptoms or vital signs but then have gone on to die?
A. Certainly.
Q. I take it they died despite you and your staff's best efforts to provide them with the best care you were capable of providing them?
A. Correct.
Q. Did any of the -- have any of the deaths in the ICU since you have been working in the ICU here result in malpractice claims that you are aware of?
A. In the obstetric realm?
Q. No, more broadly.
A. I think I already told you about the --
Q. The two. Just those two?
A. Those are the only ones I am aware of.
Well, one of them is not -- was not a case here. It was a case at another institution when I was a fellow, which we don't work at anymore. As I said, I wasn't named in the case. I was asked to be a material witness.

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The other one is here.
Q. One other article I want to ask you a question about that you wrote in Chest magazine, Correlates of Prolonged Hospitalization in Inner-city ICU Patients Receiving Noninvasive and Invasive Positive Pressure Ventilation for Status Asthmaticus. That's not an article that I think has any particular relevance to this case, but I do have a question about it.
In that case -- in that article, you indicated that four percent of the patients died during the study. Do you recall the article?
A. I do. You obviously have read it. That sounds right. I can't quote you that exact number off the top of my head, but I have no reason to dispute it. That sounds about right.
Q. Let me ask you, and if you don't know the answer, I will find the article. The article indicated that one of those patients who died had inactive systemic lupus.
A. That sounds right.
Q. In that study, did you make any attempt to determine what role the lupus might have had in contributing to the death of that patient?

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A. The study was a retrospective review of medical charts. So, of course we couldn't make an assessment in real time because it was after the event. I don't recall going into great depth to look at that particular individual case because the goal as a study was to do more of an epidemiological survey.
Q. In this case, did you give any consideration to what role the lupus -- inactive lupus Mrs. Hoffman had might have played in her condition?
A. I certainly made a note of it, but I couldn't see any clear connection between her inactive lupus and her case as it unfolded in the ICU -- in the hospital.
Q. One of the other patients in that study died -- who died had disseminated intravascular coagulopathy, DIC. Do you recall trying to determine what role the DIC might have had in that patient's death?
A. No, I don't.
Q. In this case, you said Mrs. Hoffman had DIC. What role -- well, strike that.

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DIC is associated with heightened mortality?
A. Yes, it may be.
Q. I don't know if it is your critical care textbook or a different one. I have heard it referred to as death is coming. Is that something you have heard residents or fellows use in talking about that diagnosis?
A. Never heard that before. You are the first one I have ever heard use that.
Q. I had marked as Exhibit 2 the letter we received from the plaintiff's counsel dated July 12, 2004. There is a paragraph on the second page that refers to you. I would ask you to take a look at that.
(WHEREUPON, a brief pause was taken.)
THE WITNESS: Okay.
BY MR. LAX:
Q. Have you seen that letter or that description of you and your opinions before today?
A. I don't remember. I can't say I haven't. I can't recall as I sit here today

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explicitly this paragraph in my mind, but it may be that it was given to me and I just don't recall.
Q. In relation to July 12, 2004, when we received this letter, do you have a way by looking at your file to know when you were first contacted by the plaintiff's counsel?
A. I don't. It was in 2003 sometime, but I honestly don't know here as we sit. I suspect I could find it somewhere. I just don't -- keep going, please.
Q. Well, if there is a letter --
A. May 19, 2003.
MR. BROWN: There is probably a second page. Maybe you just kept the first.
MR. LAX: I am going to have that marked as Exhibit 3.
MS. MICHAUD: What was it you are going to have marked as Exhibit 3?
MR. BROWN: Letter of May 19, 2003.
MS. MICHAUD: From whom to whom?
MR. BROWN: From us to the deponent. It is that same letter you have seen in the past. It is the fact pattern letter.

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MS. MICHAUD: Okay.
(WHEREUPON, Kress Deposition Exhibit Number 3 was marked for identification.)
BY MR. LAX:
Q. This letter which we have had marked as Exhibit 3, the context of the letter makes me think you had some discussions with plaintiff's counsel before the date of that letter.
A. That sounds right.
Q. Do you recall any of the details of what you were told this case was about or what your role might be?
A. The details of the case were likely the same as what's outlined in this letter. I don't specifically recall a phone conversation prior to May 19th in any detail.
Q. Can you describe for me what your understanding of the facts of the case was when you first began working on it? What did you think the case involved?
A. Are you asking me to go through each detail or just in a general sense?
Q. How was it described to you in a way

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that made you feel like it was a case that you as an expert might have some useful role?
A. It was described to me as a case involving critical care management of an obstetrics patient who had suffered from an IUFD, subsequent bleeding, both from the uterus and subsequently from the chest as a result of a complication of placement of a vascular device, and the patient's condition ultimately led to her suffering a circulatory arrest in the throes of her management in the intensive care unit.
Q. What did you understand your role to be? What were you asked to do?
A. I was asked to give an opinion about the management of the case as it -- with the relevance or as it was focused on the critical care management of the patient during her time in the hospital.
Q. So there was no limitation on the time frame during which your input was sought in terms of her first trip to the ICU, her thoracotomy, her return to the ICU? Were you asked to comment on her whole course of care in the hospital?
A. Yes.

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Q. Were there any restrictions placed on the scope of your review?
A. I don't recall explicit restrictions. I was asked to give an opinion about the critical care management of the patient, so implicit in that request I suppose would be -- well, let me strike that.
If you are asking me if I remember explicitly the phone conversation, I said already I don't, so forgive me for repeating that, but I am assuming my answer then is under the guise that I don't recall the details of the conversation. I don't recall the conversation at all, but logical extension would allow me to say I am asked to give an opinion about critical care management of the patient, that given that the patient was an obstetrical patient, that I wouldn't be asked to give opinions about the specific obstetrical management since it isn't within my area of expertise.
Likewise, the patient had therapy rendered by a thoracic surgeon. Since I am not a thoracic surgeon, I wouldn't be expected to give an opinion about specifics regarding the

thoracic surgical issues of the case.
Likewise, the patient, during her thoracic, surgical and obstetrical care, was cared for by an anesthesiologist, and I wouldn't be asked to render an opinion about the anesthesiologic management of the case.
Those are the major other individuals or teams that were involved in her care. I suppose you could extend that to nursing, respiratory therapy and every other service that was involved in her care. So outside the area of my expertise, of course I wouldn't be expected to render an opinion about those issues.
Q. After you conducted your initial review, do you recall how you let plaintiff's counsel know that you had come to some preliminary conclusions or how you reported to them what your preliminary conclusions were?
A. I don't recall specifically. My usual practice is after I review all the information that I have been given to have a phone call conversation with a preliminary discussion.
Q. Do you recall that conversation in

relation to July 12, 2004, when it occurred?
A. I don't recall the details. What is described here is consistent, but my recollection is only in broad categories and is similar to what is summarized here.
Q. In other cases you have worked on, have you gained an understanding that at some point in litigation the attorneys exchange summaries of what their experts are likely to testify to?
A. Correct.
Q. What's your understanding of the purpose of that exchange of information?
A. Well, I think the purpose of that exchange -- obviously I am not an attorney, but my understanding of the purpose of that exchange is to disclose to the various parties involved the information and opinions obtained from the various experts so that the case can be directed by the various people involved and the focus can be achieved. Without that, of course, it would have the potential to just wander without a purpose.
Q. You understand it is important for the defendants to understand what the plaintiff's

experts are going to say at the time of trial?
A. Absolutely.
Q. And that without obtaining that information, they would be handicapped in their ability to defend the case?
A. Of course. That would be unfair.
Q. Looking at the document that was marked as Exhibit 2, on July 12, 2004, when I got this letter, if I had asked you then whether the letter fairly summarized your -- all the opinions you had formed in the case, would it be a fair summary of those opinions?
A. It is a summary. Yes, I believe it is a fair summary.
Q. So at that point you would have testified that the care by Defendant Paul Deranian fell below the accepted standard of care when he prescribed the beta-blocker Lopressor in response to her sinus tachycardia, which effectively stopped her heart and caused her death?
A. That's correct.
Q. That's a summary of your opinions as of that time?

A. That's correct.
Q. You would also testify that had Mrs. Hoffman's medical condition been properly managed, she would more likely than not have survived and would have had the opportunity for a better outcome?
A. That's correct.
Q. Were those the only two opinions -- again, recognizing that those are general statements, but are those the two areas of opinion that you were prepared to offer after your review of the medical records?
A. Those are the two major areas, correct.
Q. Is it fair -- would it have been fair for me to assume then that to the extent Dr. Deranian was involved in Mrs. Hoffman's care prior to prescribing the beta-blocker Lopressor, you do not have any opinions that he -- that his care fell below the standard of care?
A. That is correct.
Q. Other defense experts in this case have testified his care up until the prescription of Lopressor was exemplary.
MR. BROWN: Defense expert you said.

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BY MR. LAX:
Q. Other plaintiff experts have characterized his care up to the prescription of Lopressor as exemplary. Would you be in agreement with that?
A. I don't know what exemplary means. I think his care was very good.
Q. Up until what point do you believe his care of Mrs. Hoffman was very good?
A. Up until the point around the time of this tachycardia and prescription of a beta-blocker, Lopressor.
Q. So you don't fault him for any of his orders up until the order for Lopressor?
A. That is a correct statement, yes.
Q. Up until that point, you believe his care complied with the standard of care?
A. Absolutely.
Q. In trying to determine what the standard of care for a critical care doctor was in 2002, what sources did you look to in order to form your opinions in this case?
A. I am going to step back for just a minute, Mr. Lax, just to make sure because I

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don't want to get trapped here.
Q. I am not trying to trap you. I am trying to get a clear record because, as I told you, the purpose of my being here today is to develop an understanding of what it is you are likely to testify at trial.
A. I don't mean to imply you. I mean to trap myself. Forgive me. I didn't mean to be derogatory.
The management of her case up until the point of the administration of the Lopressor was very good. Exemplary -- I think a very, very outstanding strategy of management.
There are things that we may or may not get into here which occurred prior to the administration of Lopressor that could have been done differently that, in connection with the Lopressor, may have led to a decision to use the Lopressor that had they been looked at otherwise would not have; for instance, the choice to not measure her central venous catheter pressure, which others have discussed already in other depositions.
So, as I said, I don't want to

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trap myself by making the statement and then being unable to discuss it intelligently. That is, well, you said that everything up to the moment of the Lopressor was within the standard of care and now you are saying that you didn't measure the CVP, but that happened temporally before the measurement.
I am sure that won't go to that, but I want to make sure I have set myself in a position where I am not going to be unable to have an intelligent discussion.
Q. I will jump ahead. I was going to ask you about whether or not you felt she should have had a CVP pressure taken.
A. Yes. I just use that as an example to set the stage. That's probably the most relevant one. I just want to be sure because we are using artificial marks in the sand here in time. I believe that everything you have outlined is correct.
The decision to give the beta-blocker was the decision that fell below the standard of care, but obviously one can't sever the time before and after with absolute division.

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Q. Sure. For my own sake, because I can't think otherwise, I try to march through this chronologically.
A. We are in complete agreement here. I just want to be sure I have that straight.
Q. Let me just quickly jump ahead because we are there and I am in danger of forgetting.
In your mind, could reasonable doctors, practitioners of critical medicine disagree about whether it was necessary to monitor Mrs. Hoffman's central venous pressure during her ICU stay?
A. Given the importance of knowing what her intravascular volume was, and given that her intravascular volume was changing while she was suffering circulatory instability and ongoing hemorrhage, I would say having information that would educate clinicians to the state of her intravascular volume is critical, and in that category, the CVP is one of, not the only, but one of the most useful tools within the options that I can see that we had for her. So that's a long-winded way of saying I think a CVP should have been measured.

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Q. I understand, but is there a difference between you think it should have been measured and anyone who disagrees with you is an outlier who falls below the standard of care? Do you understand what I need you to understand?
A. I do.
I think that anyone who disagrees with me is outside of the bell-shaped curve in that regard and an outlier, yes.
Q. So someone who didn't feel that it was necessary to monitor the central venous pressure did not comply or would not be in compliance with the standard of care?
A. Yes.
Q. I think I understand.
Let me go back to the question I had, which is when you are trying to decide what is or is not the standard of care for a critical care physician in 2002, what sources of information do you look to?
A. The sources of information I would look to would be the broad spectrum of publications that we have already touched on, expert opinions heard in a variety of different capacities,

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including review courses, various lectures and grand rounds and other didactic continuing medical education formats, consultation with colleagues.
There is a large number of different sources of information. Those are some of the major ones.
Q. Do you also draw on your own experience?
A. Of course.
Q. So there is no one place where the standard of care for critical care physicians in dealing with critically ill patients in the ICU is set forth?
A. That is a true statement.
Q. Do you agree that there are ranges of acceptable interventions and care that can be provided?
A. Yes, I do.
So the standard of care, as you know well, I would imagine, is a potentially elusive goal in terms of its description in some circumstances by virtue of all the points that you yourself have just outlined correctly.
Within that, I believe that the

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information, to get back to your point, that the CVP would have provided would have been useful enough that to not measure it seems -- there seems to be no rational reason, in my view, why one would not measure it. In that context, I think choosing to not measure it would be below what I would view as the standard of care, yes.
Q. But is there any room for disagreement among reasonable critical care practitioners on that particular point?
A. There is room for disagreement, of course, yes. Point of fact, in many different areas practitioners will disagree, but I will grant you there are people who might disagree. I just have the opinion that I just stated.
Q. It is clear from the description of your testimony in the July letter that one of the areas in which you plan to offer testimony that's critical of Dr. Deranian is his decision to prescribe Lopressor.
A. Yes.
Q. Where would you look -- are there any other sources that you haven't already told me about where you would look if you wanted to

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determine the extent to which prescribing Lopressor did or did not comply with the standard of care?
A. I think those are the major ones that I have outlined. I mean, obviously these are broad categories. The textbooks and articles cover a very wide range.
Q. Sure.
Is there any one place that will state that giving 2.5 milligrams of Lopressor to a patient in the condition similar to Mrs. Hoffman's violates the standard of care or thou shalt not do that?
A. I don't believe so.
Q. Do you believe that that's a topic on which reasonable practitioners of critical care medicine could disagree?
A. I will answer your question in a series. The first is that there is virtually nothing in medicine where I have come across a mandate or an executive order that says thou shalt not. There are very few that fall under that. I know that's in quotes, of course, but the logical extrapolation of volumes of data from all of the

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sources that I just covered, textbooks and beyond, all the things that we talked about, would identify the physiologic derangement that she suffered as one where acute beta-blockade would be detrimental and contraindicated.

I don't think that there is any reasonable person who could disagree with that in my opinion. That particular point I think is not one that a reasonable person would offer an alternative opinion.

Q. So in your mind, that's an absolute violation of the standard of care?

A. In my mind, administering a beta-blocker to a patient with the constellation of findings that Mrs. Hoffman's condition had over the course of the hours that she was in the hospital and in the intensive care unit and all of the data available in her records, the administration of Lopressor would be absolutely outside of the standard of care.

Q. And reasonable practitioners couldn't disagree?

A. That's my opinion.

Q. Can you identify for me the things about

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her physiological condition which in your mind make it so clear that Lopressor was contraindicated?

A. Her history of hemorrhagic shock, profound hemorrhagic shock, that was ongoing and resuscitative. She was receiving massive numbers of blood product transfusions.

Her hemoglobin nadir (phonetic) was three grams per deciliter. That's profound hemorrhagic shock from two sources that we have briefly touched on; that is the vaginal bleeding where she had an estimated blood loss, I believe, of five liters and the numbers of blood products transfused that I can't quantify because there were so many.

Q. I promised I wouldn't interrupt you, but let me take it back and break that promise, and I apologize.

Did you make any attempt to try to catalog or categorize or tally the amount of blood transfusions she had at various points in her care?

A. I did.

As far as rendering an opinion

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about this, when one starts to move into the double digits for this, one's ability to utilize those data points to make an assessment starts to flatten out. You reach a flat part of the curve where you essentially say we have given so much blood products that the patient has profound hemorrhagic shock and is at risk for all of the complications associated with it, and the 17th and 18th and 20th or 22nd or whatever number of units of various blood products that are administered do not lend very useful additional information. So that unlike going from the 4th to the 5th to the 6th unit of blood, going from the 19th to 20th to the 21st does not change my view of the likelihood of the complications that we have begun to discuss.

So the answer to your question is, can I sit here right now and tell you how many units of red cells, no, I can't, but the number was massive. In fact, there are descriptions of massive transfusion and its complications. As I say, the number, when you start to get into the numbers that she had received, becomes less important as we get into

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such high numbers.

Q. What does massive transfusion mean vis-a-vis her volume status?

A. Well, the two go hand in hand. Her volume status, by definition, as she is receiving massive numbers of units is falling. Now, you can keep up with it and, in fact, I believe they did a very reasonable job of doing that in spite of how desperately ill she was.

The number of units of red cells with the ongoing blood loss going through more than one blood volume -- I mean, in a human depends on your gender and your size, but as you are pushing through the sixth liter of blood loss, you have lost every drop of blood in your body, the total body blood volume. There is a formula for that. I can't quote it for you here as I sit, but she lost more than her total body volume of blood. I don't think there is any dispute about that.

Q. Is there a time frame during which your statement that you think they did a reasonably good job of keeping up with it coincides with? Is there a point?

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A. Yes, absolutely. From the moment it started. The moment she started to bleed, they took every measure possible to try to keep up, and there were problems along that road, as is often the case in the resuscitation of a patient with massive hemorrhage.

The first major problem being an inability to stop the bleeding. Dr. Ober dealt with that problem. She got another opinion from another colleague. They went through a series of interventions from less to more invasive, ultimately culminating in a hysterectomy. In a young woman, that's not something you would do right away. So they went through a series of steps to try to stop the bleeding.

While the bleeding was ongoing and massive, the anesthesiologists were trying to secure vascular access. That's one of the unfortunate events that led to her -- to a complication, which is they were trying to get a central line in and were unable to initially. Ultimately, one of the other doctors, I believe a surgeon, came in and got a line in,

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and Dr. Deranian ultimately got a second central line in.

That was a struggle, which is not uncommon in a patient with massive hemorrhage, because the veins become constricted, and getting a tube into that vein is quite difficult.

They did everything they possibly could and were able to maintain a reasonable degree of staying, quote, unquote, caught up, although there were some dips along the way, which is the nature of this problem.

Q. Where in the record do you see dips along the way where you believe there were times when they weren't keeping up with her fluid loss?

A. Well, when her hemoglobin was three and when she was hypotensive at various points along her care, those are general indicators that her blood loss and shock -- at the moment in time when those data points are available, that her shock is not being, quote, kept up with, but this is all an ongoing moving target.

Q. At the time that she first came to the ICU, and I understand that's not a time when you

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have particular criticisms of Dr. Deranian -- if you have to respond to a page, let me know. We have been going for a long time. If you want to take a break, let me know.

A. Can we take five minutes?

Q. Sure.

(WHEREUPON, a recess was taken.)

BY MR. LAX:

Q. When Mrs. Hoffman arrived in the ICU following the hysterectomy, how would you describe her condition?

A. Well, she was critically ill. She had just suffered a massive blood loss and resuscitation, and she now had circulatory and respiratory failure being supported.

Q. How was she supported?

A. Her respiratory failure was supported with full mechanical ventilator, her circulatory failure supported with also full mechanical ventilatory support, as well as volume resuscitation in the details that we have already outlined.

I will have to look at my

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records to see whether vasoactive support -- when that was -- if you would like me to go into that detail, I can. I just have to look at the chart.

Q. Please.

(WHEREUPON, a brief pause was taken.)

THE WITNESS: She is receiving Dopamine and Neosynephrine. Unfortunately, the copy I have is cut off here. I don't know if you can help me with this.

BY MR. LAX:

Q. Is that the 24-hour flow sheet?

A. This is the one that starts with a temperature of 93.7. It looks like 17 something, but I can't -- it is cut off.

MR. LAX: Give me a second. I will see if I can find a better copy for you.

(WHEREUPON, a brief pause was taken.)

THE WITNESS: Here it is.

So she goes to the operating room at 1500, and the case goes until, it looks like, 1630 or a little beyond that, 1640, and during the case the volume resuscitation, as we

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have discussed, was substantial, and she was on Neosynephrine infusion at the end of that case, it looks like 1630. Then I am assuming returns to the intensive care unit somewhere around 1700, which is what these flow sheets that I just outlined are. Though they are cut off, I can see that. At that point it appears that she is on Dopamine and Neosynephrine.
BY MR. LAX:
Q. What's the purpose of those drugs?
A. To support the blood pressure.
Q. Do you have any criticism of the use of Dopamine and Neosynephrine in this case?
A. No.
Q. Is that throughout the rest of her care? At some point do you become critical of the Dopamine and Neosynephrine?
A. No, I have no criticism of the use of those vasoactive drugs.
Q. Does low blood pressure despite the use of those vasoactive drugs tell you anything about her underlying condition?
A. Well, that, in conjunction with the history, which is massive ongoing bleeding,

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raises concern for me that she has hypovolemia contributing to her shock.
The fundamental principle of resuscitation of hypovolemia when it is due to hemorrhage is stop the bleeding and replace the volume that's lost. Those are just basic fundamental principles.
We don't typically use vasoactive drugs to support blood pressure in a hemorrhagic case in the absence of stopping bleeding and administering intravascular volume in the form of blood products and Crystalloid. However, as the resuscitation is ongoing, it is reasonable to add, in addition to those supportive measures, vasoactive drugs.
Q. Are there instances though when a case is so emergent that it is appropriate to start those drugs prior to stopping the bleeding or beginning transfusion?
A. Yes.
As a matter of fact, when I give this lecture on shock, I will make that point, which is don't treat hypovolemic shock with vasoactive drugs, but there are extreme

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cases where you may be forced to support blood pressure transiently while you are trying to catch up with the blood loss or volume loss. I think this is one of those cases.
Q. In the operative report you were looking at a minute ago, you said there was massive blood product administration. Is it quantified in that? That's the anesthesia chart you are looking at?
A. These are all the same, just stapled together?
Q. Yes.
A. The one on top is actually the second operation. The one on -- so the first one has an anesthesia start time of 1500, which is your second page there. Do you see that in the upper left-hand corner?
Q. Yes.
A. Down at the middle part of the page written vertically in white with a black outline is the word fluids. If you see that.
Q. Yes.
A. So there we have LR, I think it is, which stands for lactated ringers, and it appears

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that there are two liters of lactated ringers. It looks like FFP, which is fresh frozen plasma, one unit, and then we are counting blood at five units sequentially over the course of that hour and a half to hour and 45-minute interval. It looks like there is also an administration of platelets. I can't read what that says. I am assuming -- that looks like one unit, but I can't actually read it.
Q. That's during the significant hemorrhage you described during the first surgery?
A. Yes.
MR. LAX: Why don't we have that two-page sheet marked as the next exhibit so we will have it in the record. For the record, it is actually -- we will switch the order. It is 2099 and 2100 are the Bates pages. He's been talking about 2099.
(WHEREUPON, Kress Deposition Exhibit Number 4 was marked for identification.)
BY MR. LAX:
Q. So at the conclusion of that surgery when she is returned to the ICU, are there any

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other descriptive terms you would use to describe the condition she was in at that point?
A. Beyond what I already said, critically ill.
Q. Do the nursing notes that describe her as unresponsive, pupils fixed and dilated, doesn't respond to painful stimuli, pale, petechia -- I may be pronouncing that wrong -- noted on face, chest and arms, what does that description correlate with in terms of a diagnosis of her condition?
A. Well, it correlates with a descriptive diagnosis, which is critically ill, as I said, with evidence of multiorgan system failure; in particular, circulatory failure by virtue of the skin findings, by virtue of the lack of neurologic response.
Q. The fact that she is at that point on Neosynephrine and her blood pressure is 98/40, unable to obtain saturation, temp cold, 93.7, BP falling to 80/40 and 70/40 -- those are in the nursing notes -- does that give you any other clinical information that allows you to elaborate on her condition or to describe what condition

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she is in at that point?
A. Yes, it does. It describes to me with a high level of confidence that she has ongoing low output, low cardiac output, state of shock by virtue of the cold extremities, by virtue of the inability to detect a signal on the pulse -- I am sorry, but you will have to give me a second to find the page you are looking at so we are on the same page, so to speak.
Q. Yes. I don't know if yours are Bates stamped the same way as ours. I can give you a copy.
A. Mine aren't Bates stamped at all, as a matter of fact. Great. Thanks. Can I hold on to this?
MR. LAX: Why don't we mark it as Exhibit 5.
THE WITNESS: This is different than the order of mine.
MS. MICHAUD: What is the exact page you are marking?
MR. LAX: It starts with Dr. Ober's note on 05/21, and then on the third page -- it is the nursing progress notes at 1700.

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MS. MICHAUD: That's fine. Okay. Thanks.
MR. LAX: I will have the whole package of progress notes that I have here that goes up to 05/22 at 1330 marked as Exhibit 5.
(WHEREUPON, Kress Deposition Exhibit No. 5 was marked for identification.)
THE WITNESS: So if you will allow me, I will go through this. Would you like me to respond to what you have outlined as this nurse's note?
MR. LAX: Yes.
THE WITNESS: So she was received from the operating room. She is intubated and ventilated. The ventilator settings are full ventilator support, meaning the ventilator is supporting her completely. She is not responding, so neurologically she is not intact. That's seen by her lack of response to pain, the pupillary response.
The petechia are seen in patients with thrombocytopenia and DIC. She is tachycardic. She has an inability to obtain a

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saturation. Now, she is very cold, and she has got a poor peripheral perfusion with a blood pressure falling from 90/48 to 80 to 70.
As the systolic blood pressure falls from 98 to 80 to 70 and the diastolic blood pressure isn't changing, that's a strong indicator that -- that difference between the systolic and diastolic is called the pulse pressure, and that's a strong indicator that the stroke volume, that is how much blood the heart is pumping with each heartbeat, is falling, and that is a very strong indicator of a low cardiac output state, which would be consistent with hemorrhagic hypovolemic shock. That, in my view, is indisputable.
She is getting vasoactive support, but the signal here is that she is still bleeding. Appropriately, at that point a chest tube is placed and, in fact, three liters of blood return, and now the resuscitation is ongoing. As I said, packed red blood cells, platelets, plasma and cryo, replaced blood products is there, meaning this is an ongoing thing. It is an assembly line. They are

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literally pumping blood into her as quickly as they can because she is bleeding out at a concerning rate.
BY MR. LAX:
Q. If the chest tube hadn't been placed at that point to relieve the hemothorax, would she have survived for the next several minutes, half hour?
A. That's difficult to say, but the trajectory of her case leads me to suspect that she may not have.
This is unresuscitated hemorrhage. This is ongoing hemorrhage. It needs to be addressed, and without addressing it by evacuating the thorax -- the evacuation of the thorax served two purposes. Well, more than two.
The two major ones -- first of all, it was diagnostic. It confirmed what I suspect everybody thought, which was she is bleeding into her right chest, but blood coming back -- if what had come back from the thorax was fluid, not blood, but a clear fluid, we would have been addressing a completely different issue here.

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So that was a critical diagnostic intervention, at the same time being a therapeutic intervention. By relieving the blood in the right chest, it allowed her heart to fill more effectively, which allowed her circulation to become a little bit more stable, and I think, if I am not mistaken, in Dr. Deranian's note, if I can jump ahead -- sorry. Let me just find it.
(WHEREUPON, a brief pause was taken.)
THE WITNESS: There is a reference, and I am not able to find it here, that after the chest tube was placed, there was a slight improvement in her hemodynamic condition; that is, heart rate and blood pressure getting better.
BY MR. LAX:
Q. Would you characterize that as a life-saving intervention at that point?
A. Potentially.
Q. Similarly, keeping up with her fluid needs during the resuscitation during her first stay in the ICU was potentially life saving?
A. During her evening in the ICU, there is no doubt in my mind that that was a critical and potentially life-saving intervention.
Q. Okay.
And then arranging the surgery to find and control the source of the bleeding, was that similarly potentially life saving?
A. Yes.
Q. We have marked as Exhibit 4 the anesthesia sheets. During the second surgery, the thoracotomy, can you tell from looking at the anesthesia chart whether they kept up with her fluid needs during that second surgery?
A. It is difficult to know because there are multiple variables that are contributing to this. She is on a vasoactive drug, Neosynephrine, and it looks like Dopamine was added. Those will raise the blood pressure and may do so in the absence of volume resuscitation.
Secondly, the hemoglobin at the time that's marked here in the anesthesia record is 8.6. That would suggest that she's got a better hemoglobin than certainly before when it was 3. She has a low pH, and the low pH with the NIN gap metabolic acidosis that she has from the laboratory records that we have suggests strongly

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that tissue perfusion is being compromised and that she likely has a lactic acidosis, which is seen in circulatory conditions of a low cardiac output state.
Now, a lactate was not measured, but given the information here in the scenario that I am looking at, that is far and away the most likely explanation for the low pH.
Q. I need you to back up.
A. Sorry.
Q. What part of what you just said is the explanation for the low pH?
A. The NIN gap metabolic acidosis.
Q. That's indicative of poor tissue perfusion?
A. Yes.
Q. Did you make any attempt to track that going forward from the surgery through the rest of the evening?
A. Well, of course I did, by virtue of all of the assessments that are made, from the nursing notes and the bedside assessment to the vital signs to the laboratory data, and they all indicate a -- in the early part of her evening

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and into the midnight, and so around the time of her first operation, her postoperative course from her first operation, her preparation for her second operation, her intraoperative course for her second operation, an ongoing blood loss that was being kept up with but only barely.

Q. How does that correlate with her tissue perfusion during that same period?

A. Those would correlate closely in my view.

Q. After midnight, can you tell -- what records would you look at to assess her tissue perfusion from midnight forward?

A. Her tissue perfusion assessment would be based upon the same indicators; the history, the history looking at the fact that she had now, from the second operation -- and this is only the operating room -- received 18 units of packed red blood cells, four units of FFP, six units of platelets and six liters of lactated ringers, and the time in the operating room of the second case, starting at 2000 and finishing at about 2200, is a little bit longer than the first case but not much. Two hours versus one hour and

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about 45 minutes. So the cases were similar in their duration.

The first case received five units of the red cells, packed red blood cells, and the second case received 18. So the bleeding catastrophe was more profound in the second case than it was in the first based upon those parameters.

Q. So in both she got massive transfusions?

A. Correct.

Q. And massive fluid replacement?

A. Yes.

There is another indicator, which is subtle, but certainly is worthy of mention, and that is if you look at the end title C02 on the second case -- and that is a measurement in the middle part right here. So what the anesthesiologist typically does, and we do this in the intensive care unit as well --

MR. LAX: He is on page 2100 so you can keep up.

THE WITNESS: It is in the middle. It is under the monitors, and it is titled capital E -- like Edward -- capital T -- like Tom -- C02,

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and if you look at the values -- so what is the end title C02?

The end title C02 is the measurement of the carbon dioxide concentration in the exhaled gas that comes out the breathing tube. Of course, normally that value is well known. In the blood, the C02 level normally is 40, and in the end title, it is normally a little bit lower than that, but not much. So in the mid 30s. 35 to 37 is the normal end title C02.

When the end title C02 falls in this context, it strongly suggests that cardiac output is not adequate. The reason that it suggests that is that since the blood -- I am sorry -- the heart pumps blood to the lungs and the lungs filter the carbon dioxide from the blood, that if the blood flow to the lungs is reduced, then the amount of carbon dioxide going to the lungs will fall, and the amount of carbon dioxide coming out the mouth or the endotracheal tube will fall. So end title C02 correlates reasonably well with cardiac output.

You see in her case that the end title C02 ranges from 10 to 22, which is

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profoundly reduced and in this context makes me suspect that her cardiac output is compromised. Now, that's not surprising, I think, to anyone because she has bled out and received 18 units of blood. So this is all consistent.

The data points here then are all consistent and in the same direction, and that's useful information because particularly in intensive care patients we sometimes have conflicting data points where one test will say there is a problem in one direction but another test contradicts that, and then we have to come to grips with how to consolidate the information.

In this case, as best I can see, every data point is clearly and decisively in the same direction and supports the diagnosis of hemorrhagic shock. In my view, there is no dispute about that. There is nothing here that I can see that calls that into question.

BY MR. LAX:

Q. In terms of relating that hemorrhagic shock to the tissue perfusion, as the evening progresses, what data points do you look to and what do they tell you about her perfusion as the

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night goes on?
A. Are you referring to out of the second operating case now?
Q. Yes, from midnight forward.
A. Looking at tissue perfusion in a patient in the ICU can be challenging, but it is the definition of shock; that is, inadequate tissue perfusion at a multiple organ level, meaning many organs are not getting adequate blood flow. That's what shock is. So we have to target each organ. So the organs that can be targeted in the body are what the clinician will hone in on.
So what organs in Ms. Hoffman can be targeted? The brain. If the brain is not receiving adequate perfusion, then mental status will be impaired. This is an area that my group has done a lot of research on, so it is an area that I am particularly attentive to. If the patient is not getting blood to the brain, the patient will be unresponsive, and you see that. The patient is not responding to painful stimulus, is with fixed dilated pupils. Her brain is not getting adequate perfusion.
Now, that's a transient event,

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and that's very important here. It is transient, meaning as the evening and early morning wears on, she starts to wake up. Why is she waking up? Because perfusion is being restored. She is awake. She goes from dilated fixed pupils and completely unresponsive, even to painful stimulus, to interactive, able to follow commands, to process complex requests.
I believe there was a note in there that she was able to stick her tongue out upon request. That requires cortical upper level processing. That's not a reflex. That requires a person to be intact neurologically to a good degree. We use that routinely. So that clearly indicates to me that her shock, with regard to brain perfusion, is improving.
More data to support that, her skin temperature. She is cold and she is warming up. The reason the skin is cold in a patient who is in a low cardiac output shock is because the blood vessels to the skin constrict, and the reason they constrict is that as the blood flow is compromised, the body is able to redistribute blood to the more vital organs and away from the

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less vital organs, and the skin is clearly a less vital organ than the brain or the kidneys or the heart.
Other organs that may be followed here, the urine output. So the urine output is a signal of kidney function, and so that would be something that one would look to. That was trickier to follow because she received a diuretic, which will make it harder to use that as an end point. Regardless, that would be another end point that I would follow asking the question of tissue perfusion.
Q. Let me stop you there for one second because we have had some testimony from other experts about her urine input and output.
Is that something that you try to track or that you looked at in your review of the records?
A. I did.
Q. What were you able to -- strike that.
Did you observe that she was given more input than she output during her stay in the ICU?
A. Yes.

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Q. Let me mark this as Exhibit 6 I think we are up to.
(WHEREUPON, Kress Deposition Exhibit Number 6 was marked for identification.)
BY MR. LAX:
Q. I have marked as 6 several pages of the patient care 24-hour flow sheet for 05/22 and then 05/21. Does looking at that document, which has been marked as Exhibit 6, enable you to try to assess how much fluid she was given versus how much she output?
A. Yes. On the way the shift is broken down, it is by day. So at the midnight strike of the clock, the first sheet ends, and that's the 05/21/02 sheet. It looks like we start in the intensive care unit 1600, and as best I can tell here, the total intake is 29,000 milliliters or 29 liters.
The one piece of information that's not entirely clear to me here is -- it says 60 units in summary under the blood products, so I don't know if that means that each unit is accounted for by either a packed red

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blood cell or a platelet or an FFP. I am assuming that's what it is, but it is not clear to me. Then there is five liters of normal saline.

Q. What's the purpose of normal saline in a critically ill patient?

A. Normal saline is a Crystalloid which is intended to give fluid to resuscitate volume depletion.

Q. That was an appropriate thing to give to Mrs. Hoffman?

A. Yes.

Q. During that same period of time, what's her output?

A. Well, it says here the urine output from 1700, 1800 and 1900 is 15 ml's, and then we jump ahead to 2300, and it says 1665. So there is a jump from about 50 ml's or 45 over three hours, which is low. That's what I would refer to as oliguria, or reduced urine output.

Then we frame shift to 2300, and I am assuming that's the time in the operating room, when we are now up to 1665 ml's. Now there is also three liters out in the chest

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tube. So her total output is 4.6 or 4665 ml's, but three of those is bleeding out of her chest. So the majority of her output is actually hemorrhage.

The output from the urine, it looks like 1665. The operating room record reports 400 ml's, and I don't know how that 400 ml's adds into this 1665.

Regardless, the total units for the seven or eight-hour shift that she is here, the evening shift into the midnight hour, is 1.6 liters.

Q. Does that help you at all to assess whether she is volume replete for the time period recorded?

A. It isn't a very useful measurement because I don't have details about the time course here, given the summary of suddenly seeing 1650 and, importantly, the urine output was impacted by the administration of a diuretic.

Q. Is the administration of the diuretic something that you have formed an opinion about in this case?

A. I think the administration of the

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diuretic is something that I personally wouldn't have done. The argument that was proposed, to try to I believe the word tweak the kidneys was used, in my opinion, is not based upon any sound data. So I personally would not have done that, in particular, when the concern about hypovolemic shock was present.

Q. Well, I understand you wouldn't have done it, but is this something you feel falls below the standard of care? Giving the 40 milligram dose of Lasix, is that something you believe was a violation of the standard of care?

A. I believe it falls outside of the standard of care. I believe that that is something that should not have been done.

Q. Is that something you think reasonable practitioners of critical care could disagree about, or do you think it is like your belief about Lopressor, it is just absolutely contraindicated?

A. I don't say it as emphatically as I do about the Lopressor. There is a disagreement, but the medical literature, in my view, would support what I said.

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Q. Which particular literature are you referring to? Where would I go to find a statement that a 40 milligram dose of Lasix would have been contraindicated for a patient like Mrs. Hoffman?

A. You wouldn't go anywhere to find that explicit statement, just as you wouldn't go anywhere to find the 2.5 milligram dose of Lopressor, but the principle behind the pathophysiology, you could go to any number of the large number of textbooks and references that I referred to and find it there.

Q. That principle is --

A. The principle is -- I am sorry for interrupting. Go ahead.

Q. I need to understand what principle is it that you think is violated by giving the diuretic?

A. The principle of administering diuretic therapy to a patient who has oliguria in the throes of hypovolemic shock.

Q. What is the risk you run by giving a diuretic to a patient in that state?

A. The risk you run is worsening perfusion

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and shock. In this case, the other important risk or issue is that it takes away one's ability to utilize the urine output as a measure of perfusion.

Q. For what period of time? What period of time is the period of impact that a 40 milligram dose of Lasix would have on Mrs. Hoffman's natural biological urinary function?

A. The effect of Lasix is dependent on a variety of factors, some of them very relevant to this patient; for instance, the fact that she is in shock, and so her metabolism of drugs may not be normal.

A general rule of thumb is that the effect of Lasix lasts somewhere between six and ten or twelve hours, but there is a wide range. Certainly the duration of her care -- she died in the early morning hours, but for the duration of her care from the moment she received the Lasix to the moment she died that the urine output was not -- was a less useful measurement by virtue of the administration of that Lasix, and the Lasix was still potentially having an effect up to the moment she died.

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Q. By looking at her urine output over the early morning hours, that it goes from 1000 to 200 to 85 to 120, does that help you at all in terms of identifying the impact the Lasix was having on her biological urinary output?

A. It allows me to make a cause and effect correlation, but a more important question is not what is the urine output but what is the renal perfusion, the blood flow to the kidneys, and those two variables are not related tightly when the administration of a diuretic uncouples them.

Q. So I think the short answer is no, the output drop and then increase doesn't really help you assess the extent to which Lasix is impacting her urinary function.

A. That's a fair summary.

Q. Is the duration of the impact of Lasix dose dependent?

A. It is dependent on a number of variables. The dose, I suppose, could be one of them, but the patient's underlying organ system function, livers, kidneys, circulation, are going to be another important variable.

If the perfusion to various end organs is impaired, as it often is in critically ill patients and it certainly was in Mrs. Hoffman, then the reliability of pharmacologic descriptions of metabolism of this drug or, for that matter, of any drug is less concrete.

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Most of the drugs that are studied are studied in healthy volunteers. So the pharmacology that might be applicable to a healthy volunteer is not necessarily relevant here.

Q. Do you use Lasix in your practice?

A. I do.

Q. For what conditions?

A. The most common condition I would use Lasix or a loop diuretic would be for pulmonary edema and in the setting of congestive heart failure.

Q. We have talked about it, but in this case, you didn't suspect that that's occurring in Mrs. Hoffman at the time the diuretic is given?

A. I am virtually certain that it is not occurring. There is no evidence, absolutely none.

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Q. Does Lasix have any vasodilating properties?

A. It does.

Q. Can it assist in the process of oxygenation?

A. By virtue of decreasing pulmonary edema, by all means it may have a major impact on gas exchange across the lungs. As I said, that doesn't strike me as a relevant concern in this patient.

Q. Do you see any clinical indication in Mrs. Hoffman's chart for a loop diuretic?

A. No.

Q. So anybody who believes that there was a need for a diuretic in your mind would be an outlier and would not be complying with the standard of care?

A. Yes.

Q. Other than the intake-output chart, are there other portions of her record which in your mind would be relevant to assessing her volume status during the early morning hours?

A. Well, the intake and output is going to be coupled to the history. As I said, again,

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that's critical.
The foundation of this discussion, the foundation of her assessment -- forgive me if I am repeating the same thing -- must be established with absolute concrete certainty, and that is that it is a patient who has a life-threatening, ongoing hemorrhagic condition coupled to a disturbance in coagulation, but not solely due to that because she has a laceration in her vessels. So that's the background.
It is ongoing with very little evidence that it's been fully addressed; addressed meaning the bleeding is completely stopped and the intravascular volume state is resuscitated with an adequate time period to assure the caregivers that this moving target has stabilized.
That's a critical part of this. I can't overemphasize how important that is; that if she stabilizes but transiently and then bleeds again -- that's what she did. She did that several times in the course of her care, at least the most concrete one being after the first

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operation and up to the second.
So now we have a circumstance where this behavior is manifesting itself now in the face of severe coagulopathy. And so this ongoing repetitive nature to this hemorrhagic hypovolemic shock must be addressed, and in my view, there must be confirmation that the problem is resolved and that it remains resolved. You need time for that. She hadn't had that up to the point that she arrested.
Q. Are there points in time when you feel that she had stabilized? Were there -- what I am specifically asking is Dr. Deranian testified and his orders indicate that he had assessed her before he left the hospital. At that point do you have any criticism of his orders and his entries in the progress chart in terms of his assessment of the patient?
A. No. I think up to the point where she was awakening and following commands and her blood pressure was improving and her skin temperature was warming and she was, as I said, more lucid, that was an indicator to me that her condition had improved, that she had turned the

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corner, so to speak, that she was getting better, and what I would say and I believe what he said was this is good news. She is getting better. She has a real chance of getting through this life-threatening condition.
Q. Was she out of the woods at any point? I mean, at that point would your assessment that she was critically ill have changed?
A. No, but she still remains on a ventilator with massive blood transfusion, with profound thrombocytopenia, with an ongoing metabolic acidosis, with a slightly but importantly increasing creatinine.
Q. First tell me where you get that and then tell me what the import of it is.
A. Okay. I just have to find the labs here.
The creatinine is the most commonly used lab indicator of renal function. Creatinine is a breakdown product of muscle that is excreted by the kidneys, and when the kidneys are dysfunctional, the creatinine will rise, but it happened slowly.
It is said that if you took the

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kidneys out of a person, we would never do that, of course, but if you did, that the creatinine would double each day. So she only had about 12 hours under her belt. So there isn't enough time to draw points on the curve to have the creatinine be a very useful indicator.
Within that limitation, her creatinine is 0.7 at 1715 and 1.3 at 2 in the morning, so it is almost doubled. Now, I will say that with such a narrow time interval that one cannot use that data point with an extraordinary level of confidence or reliability, but as I said before, all of the data points are looking in the same direction. This is consistent.
If the creatinine on admission had been 1.3 and now it was .6, that would be a conflicting data point. That would make me wonder, gee, I wonder why that is. It wouldn't change my opinion, but it would make me wonder. That would be an inconsistent data point, but there aren't any of those here.
Q. Does that show that her volume status is improving during this time?

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A. The fact that her creatinine is rising?
Q. Yes.
A. No. If anything -- as I said, I am very cautious in making a concrete and utterly confident statement about the creatinine because we just don't have enough time, but given that it has risen, that's a suggestion to me that her volume status is worse, that her perfusion is worse, that her kidneys are not getting enough blood flow, and that is all consistent with the fact that the urine output has fallen up until the moment when she received the diuretic.
Q. Other than the intake and output, which we have talked about, the creatinine, the ongoing metabolic acidosis, are there any other indicators of volume status that you looked to in her chart?
A. Well, of course the blood pressure and particularly the pulse pressure, as I indicated, is one. The heart rate. The vital signs of course are going to be indicators of volume status, but they have limitations.
Q. What limitations are there in using blood pressure, pulse pressure and vital signs as

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indicators of volume status?
A. Do you want me to go through each one of them?
Q. Please.
A. The blood pressure can be impacted by catecholamines, vasopressors, both administered through an IV, which she is getting, and also endogenous. The body makes epinephrine. Her epinephrine level produced by her body was likely extraordinary in the midst of this crisis.
Pain, agitation, other drug effects, none of them are obvious in the chart here, but those sorts of things can impact blood pressure as well. Obviously, as I said, the volume status being one.
With regard to the heart rate, well, the heart rate, when it increases, there is a long list of reasons for that. If the increase in heart rate is a sinus rhythm, which it was in her case, hypovolemia is high on the list.
Anxiety, pain, agitation, temperature pertubations, particularly fever, any other type of distress, respiratory distress. The bleeding into her chest and the compression

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of the chest wall and the lung can elicit a tachycardia and, of course, tachycardia, I think I said at the very beginning, may be an indicator of intravascular volume depletion.
Q. At some point do you believe that that manifested itself in the record for Mrs. Hoffman?
A. I do, but that's because I assess the situation without taking those data points in isolation. The reason I outlined for you a long and certainly not complete list of the causes of high blood pressure and tachycardia is because of that long list I just give you, there are many things on that list that have nothing to do with hypovolemia. So to say the heart rate is up so the patient must be hypovolemic is not a rational thing to do, but it is consistent with the other data points.
Q. At some point do you believe there is evidence in the chart that allows you to say confidently that she was hypovolemic?
A. Numerous times in the chart, yes.
Q. At what point do you start to believe she is hypovolemic?
A. From the moment she started to trickle

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blood from her vaginal vault, to the first operation, to the return to the intensive care unit, to the hemorrhage into her chest, to the second operation, and to the return back to the intensive care unit with those hypovolemic occurrences truncated by intermittent improvements based upon her resuscitation.
So it is a moving target, but the line is not straight. The line is up and down. If you asked me to give her a volume score across the course of her intensive care unit stay or hospital stay, I would say that the dots on the line are not horizontal but up and down, but the general trend is most of the time hypovolemic, many times profoundly so, and the only clear-cut times that I see that she is clearly not hypovolemic are in the morning when she walks into the hospital before this event all began up until the time that her induction began. She is ambulating in the hall having contractions before she delivers the fetus. I see no evidence she was hypoglycemic then.
Then there is an indicator that perhaps we are catching up, but I can't say we

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have fully caught up, when after the second operation she is waking up, following commands, the blood pressure is getting better, her skin is warm, and all the things that we already outlined. That signals to me that her volume status is improving transiently.
Q. When does it then drop off again?
A. I believe it drops off again when -- the occurrence for how many times this happened I can't quantify for you, but several times when she had another event of hypotension and tachycardia, which was the time when the nurse at the bedside called the critical care physician to indicate to him the blood pressure was falling and the heart rate was rising.
Q. In response to one of those phone calls, was it appropriate for Dr. Deranian to try to infuse volume by ordering three units of blood?
A. Absolutely.
Q. Would that improve her volume status?
A. It depends.
Q. Is there anything in the chart that enables you to say whether that helped her volume status?

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A. I am going to ask you to let me find the details because I can't remember all the specifics of the vital signs.
Q. There is another document that might help with that.
MR. LAX: I am marking the 24-hour flow sheet from 2330 on as the next exhibit.
(WHEREUPON, Kress Deposition Exhibit Number 7 was marked for identification.)
BY MR. LAX:
Q. I have had marked as Exhibit 7 a two-page document. The copy I have is marked pages 2112A and 2113.
A. Can you just repeat the question so I know.
Q. You said there was a period of improvement in her volume status. I asked about whether the three units of blood was an appropriate intervention to try to improve her volume status, and then I think I asked you do you see anything in the chart which allows you to say whether her volume status improved at all as a result of the three additional units of blood.

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A. Right.
I am going to ask you for one other piece of information just so I have it all in front of me, and that is do we have a narrative from the nurses notes -- I need to find that -- that is timed so I have that to put it next to this? I know we have it. I just have to find it.
(WHEREUPON, a brief pause was taken.)
THE WITNESS: So we see that in the early hours of the morning, say starting at 2:30 a.m., she has a blood pressure of 115 over looks like 50 and a heart rate of about 145, and then a half hour later it is similar. Then as we move to the 4 o'clock or 3:30, we start to see the blood pressure falling and the heart rate rising, so that we are now with a heart rate upwards, based on the chart at least, in the mid 150's and the systolic blood pressure has fallen now below 90, and that's in the face of escalating vasoactive drugs, at least Neosynephrine, which has gone from 120 to 150 at 2:30 in the morning to 200 at 3 o'clock in the morning. So there is

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a titration up.
At 3 o'clock in the morning there is three units of red cells, and after that there are a couple of indicators of transient improvement. After three units of red cells, which were given at 3 o'clock in the morning -- I am sure they took a little bit of time to go in -- it looks like here -- patient's blood pressure remains dependent on Neo and Dopa, as I said, Dopamine -- Neo at 200mcg, Dopamine at 10mcg -- to maintain a systolic blood pressure greater than or equal to 90. Patient tachycardic; max 160, runs on average 140 to 150. Dr. Deranian aware.
BY MR. LAX:
Q. You are aware there is testimony there may have been heart rates higher than that as the morning went on?
A. I am.
Q. And blood pressures lower than are shown on the chart as well?
A. I am.
Neuro at 2300, pupils sluggish but responsive; blinks eyes, squeezes hands.

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Hand grips family; head yes, no, attempts to speak.

This is a summary statement timed at 4:10 but clearly is a summary over several hours since there is clear indication that at least the neuro assessment timing is 2330. So we are getting a summary of several hours of data, which makes it impossible to pinpoint to the minute each of these events.

Regardless --

Q. Just so you keep it in mind, the specific question was what do you see that helps you assess the impact of the three units of blood as it affected her volume status.

A. Yes, I understand. The reason I am going through this in detail is because I am trying to see the trajectory of these changes in order to best answer your question.

Then they talk about the patient more awake, attempting to pull at the endotracheal tube and received morphine and Ativan. Now, that's timed on the nursing note here at 4 o'clock in the morning or 3:30. So we are moving forward in time. These are all

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indications that she is improving.

More awake, sluggish. Then she goes from sluggish to talking or trying to talk -- of course she can't talk with an endotracheal tube in -- to now needing wrist restraints, to now needing morphine and Ativan so she doesn't pull out the endotracheal tube. She is clearly more awake from that perspective.

That data point suggests to me with a reasonable degree of confidence that her circulation is better, improving and has transiently stabilized.

Q. Does that help you assess whether she is less hypovolemic than she was earlier?

A. That helps me to assess, and to answer your question specifically, that the three units of blood likely -- more likely than not were effective in improving her volume state; not correcting to normal necessarily, but moving it in a positive direction.

Q. Then at what point do you believe she again turned the corner or started to become more hypotensive?

A. This again gets back to the point I made

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before. I can't overemphasize how important this is, and that is that, to your pointed question are we out of the woods, the answer is absolutely not because this is clearly a moving target. We are not yet stabilized to the point where I can say the dust has settled, the bleeding has stopped, the circulation is stabilized and now we just need her some time to heal from her surgery and she will be fine.

As I sit here looking at this, I say to myself, at 4 o'clock in the morning when the next event occurs, which is another drop in blood pressure and an increase in heart rate, and whether it is the flow sheet that gives these data points or the deposition of the nurse and the doctor, which seem to differ to some degree, regardless, the trend is in the same direction, and that is that once again she is declaring herself as dropping her blood pressure, increasing her heart rate on a background of recurrent hemorrhage and recurrent clear-cut indication of circulatory compromise and hypovolemic shock.

So that's where we are at. In

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my view, there is absolutely no dispute. You can't possibly, in my opinion, interpret it any other way. You have to take the track record here. You can't ignore that. You have to take the background problem and the direction in the trajectory and factor that into the equation. You can't simply say she is awake now and she's better and everything is fine because the next event that happens, in my view, just emphatically demonstrates what I said, which is there is another event coming. I am not sure why it is happening this time, but it took two major operations to figure out the first two events, and we are still not clear and out of the woods at this point.

Q. When you say there is another event coming, do you believe Mrs. Hoffman could have sustained the falling blood pressure and the increasing tachycardia much beyond 4:50 or do you believe she was heading toward some status change based on the trend you see?

A. Based on the trend I see, I am concerned, more likely than not, that she is going to have another status change.

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Q. At that point, based on her track record up to that point, do you believe it was a potentially fatal status change?
A. In the absence of appropriate intervention, it has that potential, just like every other one where, in fact, appropriate intervention was undertaken as we have already outlined and her potential for arrest was prevented.
Q. What do you think was causing the tachycardia and the falling blood pressure at that point?
A. Hypovolemia and another event of hemorrhage.
Q. What evidence would be useful to assessing whether or not there was some type of hemorrhage going on at that point?
A. Well, a central venous pressure would have been very useful here potentially. Because the bleeding event is happening, if that's what it is -- the hypovolemic event is happening so quickly, there may not be enough time to gather all the data points.
We have gone through fairly

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extensively the assessment of the adequacy of organ perfusion. So the creatinine isn't going to rise because it has happened so quickly. The mental status perhaps could fall, but she has received a sedative, which makes it hard to use that as an end point. Now, I am not criticizing the use of a sedative. She is awake with an endotracheal tube and perhaps needed one, but it does inhibit your ability to use that particular end point.
One could look at the urine output, but that's hindered by the administration of a diuretic. One could look at her tissue perfusion; what does her skin look and feel like, are her extremities cool or are they warm. One could look at the pulse oximeter and one's ability to detect it.
None of these are absolute yes or no, but they are all data points that we have gathered before that had been supportive of this, and the most important data point in my view -- one of the most important data points is the history, and the history is so clear here that one, I believe, must make the assumption, in the

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absence of utterly certain data, that this is happening again.
Q. Does her low serum HC03 at this period tell you anything about her status?
A. Well, it does, but it never was not low, or at least for any reasonable time, and it hadn't changed all that dramatically.
Let me just make sure I am not misstating the evidence. You may have a laboratory value that's later than the one that I saw, which was I think at 0200.
Do we have a chemistry panel that's later than that?
Q. Tell me if this would help.
A. These are the calculated bicarbonates. These aren't the serum HC03's. These are blood gases. We will get to that in a minute.
Q. Would that help you or not?
A. It could, yes. So if you look at the calculated bicarbonate -- are these in order?
MR. LAX: Off the record.
(WHEREUPON, a discussion was held off the record.)

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(WHEREUPON, Kress Deposition Exhibits Nos. 8 and 9 were marked for identification.)
BY MR. LAX:
Q. We have marked as Exhibit 8 the blood gas reports. Mine is Bates stamped 2177 to 2182, and Exhibit 9 is the complete blood panel work. Mine is Bates 2135 to 2150.
I had asked you about the low serum HC03.
A. Yes.
So she has a profound metabolic acidosis with a bicarbonate, or serum HC03, which is between 11 and 13 from 1715 to 0200 on the chem 7, and NIN gap, which leads me to consider inadequate tissue perfusion and lactic acidosis.
That number hasn't really changed for quite some time during this nine-hour interval, and it just reemphasizes the point that she has ongoing shock that's in the midst of resuscitation but has not yet been fully resuscitated.
Indeed, if it had been fully resuscitated, the recovery of the low bicarbonate

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carbon dioxide level, HC03, would lag in time. It isn't an immediate recovery. So unlike the brain where the blood flow back to the brain gets better, you generally see a reasonably quick recovery turnaround. You don't see that degree of metabolic acidosis recovery in the span of minutes. You are usually talking hours or perhaps even longer, especially if there is kidney injury, which there may have been here, although I don't have enough concrete evidence yet to say with absolute certainty, but I suspect it.

The blood gases calculate the bicarbonate level, the HC03. That's not a direct level. It is a calculation, and it is based upon fundamental principles of physics and physical chemistry. If you want, I can go through the equation.

Q. Please don't.

A. Basically it is a calculated number, but it is consistent across the board so that by the time we get into the midnight hour of -- well, the 22nd, the bicarbonate is around 13, and it stays there throughout all the blood gases,

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including one up to 0300 or 0310, which is the latest laboratory point I have where it is still calculated to be 13.8.

So this is all consistent, and because we are at that point of a low bicarb, I am not surprised that fluctuations in her volume status and fluctuations in her hemodynamic status are not reflected by dramatic changes in the HC03.

So I wouldn't -- it is a useful end point, but it is not a useful end point at the last moment in time when we are trying to say has this last intervention made a difference. There are two reasons for that. First of all, the timing lag in the second is -- in fact, we don't have a data point after these last interventions to even answer the question.

Q. During the same time period you have just been talking about, her coagulopathy hasn't totally resolved, has it?

A. No.

Q. That's despite proper interventions with blood product to try to address it?

A. Well, yes, that's correct. The

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fundamental principle behind treating DIC is to treat the underlying cause, which they are doing to the best of their ability, and that is to resuscitate her hemorrhagic shock.

Q. Is she still anemic during this last period before the Lopressor?

A. She is.

Q. What does that tell you about her status or her prognosis at that point?

A. Well, the hemoglobin level is 8.5 at 2 o'clock in the morning, which is the last one I see here, and when you have pushed through 20 some odd units of red cells or more, I don't know the exact number, but 60 units of total products and the substantial portion of those were red cells -- red cell units and the volume that she got and the fluids, it is very difficult to use a hemoglobin level as anything other than a trend indicator.

You can't plant yourself on the foundation of the hemoglobin is now 8 so we are okay because how much is going in and how much is going out and how the tank is changing in size, the tank being the blood volume, all of those

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things are going to contribute.

This is a little bit like having a gaping hole in the side of a bucket and you are pouring water in as fast as you can and the water is pouring out the gaping hole as fast as it can. In the meantime, you are changing the size of the bucket, and then you draw a drop of water out of the bucket and try to measure the concentration of something in it and get a number and say now I know what's going on. It is very difficult to do in the setting of massive resuscitation like this.

Q. Let me show you a letter I got on May 6th that describes the opinions of a doctor that's been retained by the plaintiff named Dr. Dershwitz.

A. Yes.

Q. Is that something that you have seen before?

A. I have seen his deposition. I don't recall reading this letter per se, but his deposition -- some of the major points of the deposition are summarized under these four bullet points on this letter.

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MR. LAX: Off the record for one second.
(WHEREUPON, a discussion was held off the record.)
(WHEREUPON, Kress Deposition Exhibits Nos. 10 and 11 were marked for identification.)
BY MR. LAX:
Q. I had marked while you were out, Doctor, as Exhibit 10 the May 6, 2005 letter summarizing Dr. Dershwitz's opinions, and I asked if you had seen that. You indicated you read his deposition but you hadn't seen the letter; is that correct?
A. Correct.
Q. Then let me show you what I have had marked as Exhibit 11, an August 1, 2005 letter, which says, I have asked all my experts about your defense of amniotic fluid embolism. None of them agree that it is the cause of death. For purposes of their depositions, you should assume these opinions will be elicited at trial. You are welcome to inquire.
Then it says, in addition, though I had asked some of my experts to address

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only the issue of Lopressor use, they all agree with the testimony of Dr. Dershwitz regarding other violations of the standard of care. Though I do not know at this time if I will elicit that testimony at trial, you should assume I will for purposes of their depositions.
Have you seen that letter before?
A. No.
Q. Do you recall any conversation with plaintiff's counsel in the July or August time frame of this year in which you discussed Dr. Dershwitz's deposition or his opinions?
A. Mr. Abramson and I have discussed Dr. Dershwitz's deposition, yes.
Q. I don't want to press you on the issue. I don't want to know the details of that conversation, but why don't we look at the four points raised in the May 6th letter.
A. Okay.
Q. Can you tell me when during your work in this case did you come to -- or did you develop the opinion that the failure to order a maintenance infusion of Crystalloid was a

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violation of the standard of care by my client? I should say how do you come to the conclusion that failure to order a maintenance dose of Crystalloid was a violation of the standard of care?
A. I was going to say I don't recall explicitly making that statement, so you are asking me to comment on the statement that another person has made.
I will start by answering your question in a broad way, which is these four points outline the underlying problem that we have been talking about for quite some time. The first point regarding a maintenance infusion of Crystalloid is a point that I have an opinion on regarding the ongoing administration of volume to correct the hypovolemia.
Now, the way that one chooses to do that, by giving maintenance fluid or by giving intermittent boluses of fluid with intermittent assessments, there is no difference in the clinical implications of that. In my view, they are essentially one in the same. So as a matter of style, I don't typically

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administer maintenance fluid, per se, but rather assess on an ongoing basis and give frequent administration. The end point is identical.
Q. So in this case you do not share Dr. Dershwitz's opinion that the failure by Dr. Deranian to order a maintenance dose of Crystalloid was a breach of the standard of care?
A. I don't disagree with that. I didn't explicitly make that statement at any point in my assessment.
Q. Is that an opinion that you think you will offer at the trial of this matter?
A. I will answer the question, if it is asked, in the manner that I just did. That is, that the administration of a maintenance infusion, per se, is one approach to the ongoing resuscitation of a patient with hypovolemia, and it is certainly within the standard of care.
There are other ways that one could address that. So strictly speaking, if you ask that question in a vacuum, the answer is no, there isn't any clear evidence to me that that is the only way one can address that issue. We are not asking the question in a vacuum. We are

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asking it in the context of treatment.
The spirit of that statement is ongoing volume resuscitation must be assured, and if that is not done, that's a violation of the standard of care, and I agree with that.
Q. In this case, do you think the attempts to maintain and supplement Mrs. Hoffman's volume from midnight on with the three units of blood and any other fluids that were administered fell within the standard of care of trying to address a volume deficit in a patient with hemorrhagic shock?
A. They were able to keep up, as best I can tell, up until the very end with the various deteriorations in that, and one might argue -- I am now interpreting Dr. Dershwitz's opinion here to say that a maintenance infusion may have had a higher level of assurance.
Maintaining intravascular volume with a maintenance infusion is within the standard of care, and the specific question that you asked me, was it evident from the evidence that they were able to maintain adequate volume status, the answer is no. There were a series of

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events that led to periods of time when intravascular volume state was not maintained.
Q. Could different reasonable practitioners of critical care medicine have a difference of opinion on whether the attempts made by Dr. Deranian to keep up with her fluid loss and her possible fluid status complied with the standard of care, or do you think anybody that said the order for three units of blood and the other steps he took was a clear violation of the standard of care?
A. Well, it was more than an order of three units of blood. She got 60 units or whatever.
Q. I am focusing on the early morning hours.
A. Okay.
If that order was not associated with a careful ongoing assessment, then I believe your statement is correct. That's where the problem is. That gets us to point number 3.
These points can't be looked at in isolation. They have to be connected. I am not trying to avert your question. I am just

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telling you that the issue -- I know you want to go through these sequentially, but to answer your question, I am going to have to move ahead to number 3 --
Q. Well --
MR. BROWN: Let him finish his answer.
MR. LAX: I was just going to say why don't you read number 3 so we know what we are talking about and so the record is clear.
THE WITNESS: Number 3 is failure to recognize and treat ongoing hypovolemia, comma, and I will just interject there and say within that is the assumption that treating hypovolemia is giving intravenous fluid, and maintenance infusion of Crystalloid is part and parcel to that. So in that context, the answer is emphatically yes.
Let me finish -- (continuing) and failure to quantitate her volume status by one of the indices available to him that we have discussed fairly extensively, and the one that's the most poignant perhaps is the CVP; failure to stay at night or direct the nurse in detail how to manage the status. So I agree with the or,

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direct the nurse in detail. That's the end of number 3.
Then there is an and to number 4. I have just read to you number 3.
BY MR. LAX:
Q. Taking number 1 and 3 together --
A. Yes.
Q. -- what is your opinion about whether or not Dr. Deranian's attempts to keep up with and assess Mrs. Hoffman's volume status complied or didn't comply with the standard of care?
A. They complied with the standard of care up until the final event, which is the last one that we touched on, although I don't know that we have exhaustively finished it yet, and that is the last report in the 3 o'clock, 4 o'clock hour where the nurse and the doctor to some extent, I guess, disagree about the vital sign changes, but the last event of hypotension and tachycardia where at that point he did not comply.
He did not maintain intravascular volume. He did not keep up with the volume state. That coupled to point number 4 -- inappropriately ordering the Lopressor is

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what point number 4 is -- is critical.
So as I said, these are connected intimately. You can't dissect them out and just look at one in isolation.
Q. With respect to CVP pressure monitoring, do you believe that the standard of care required --
MR. BROWN: We already did that.
MR. LAX: All right.
BY MR. LAX:
Q. What do you think -- strike that.
What is the reason that you think Lopressor was contraindicated when Dr. Deranian got the last phone call from the nurse and gave that order?
A. Because the patient was manifesting a physiologic response to yet another event of hypovolemic shock, with a physiologic response normally in her case being an increase in heart rate in an effort by the circulation to maintain forward flow cardiac output.
Cardiac output is the heart rate times the stroke volume. So as the heart rate increases, that is often the body's response

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to hypovolemia and low output to try to maintain forward oxygen and blood delivery.
Q. Are there other causes of sinus tachycardia which you considered in this case but ruled out other than hypovolemia?
A. We talked about the long list of those. The ones that I would consider here would be agitation, pain, but she had received a sedative and wasn't behaving in that way. The others that I listed aren't really relevant. The temperature changes and all other things that we talked about don't seem relevant here.
Q. Do you use Lopressor in your clinical practice?
A. In this scenario?
Q. Ever.
A. Ever, yes.
Q. For what conditions?
A. Probably the most common condition that we use Lopressor, which is a beta-blocker, is for patients who have acute myocardial ischemia where it's been shown to be protective of the heart.
Q. In what doses do you use Lopressor?
A. Well, the drug can be administered

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either by intravenous or by oral formulation. In the intravenous formulation, it depends on the underlying clinical context. So if the patient has little concern for hemodynamic stability, one might start with two and a half or five milligram intravenous injection.
There is no single dose that one can report to answer your question. There are times when one might use a higher dose. In an oral dose, fifty milligrams or even a hundred milligrams are given. I know there has been discussion in other depositions about using even smaller doses than that, and there may be circumstances when hemodynamic issues are less clear where one might consider a smaller dose.
Q. Do you believe the dose of 2.5 milligrams ordered by Dr. Deranian was excessive in this case?
A. I think the drug was the wrong drug. Let me answer your question another way. If you are asking me had he given .5 or 1 instead of 2.5, would that have been okay, no.
Q. So it is the fact that he used this particular beta-blocker in your mind was

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contraindicated and fell below the standard of care?
A. Yes.
Q. The reason is largely due to her underlying hypovolemia?
A. Correct.
Q. If you were confident, hypothetically, that she was normal -- if she had adequate volume status and she had this falling blood pressure and this increase in heart rate, would that change your view at all of whether Lopressor was contraindicated?
A. No, not in the sinus tachycardia in this particular case.
Q. So in your -- it is your opinion that if a patient has sinus tachycardia, there is never a clinical indication for Lopressor?
A. No, I didn't say that. Let me step back.
It is rarely indicated. The example of myocardial ischemia that I outlined before might be a circumstance. In the cardiac ICU, if a patient comes in with crushing chest pain and ST segment changes with a cardiac enzyme

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leak and has a heart rate of 125 and the rhythm is sinus, I would by all means -- and a blood pressure that is stable, I would by all means give a beta-blocker, assuming there wasn't a contraindication like severe asthma or something like that, as well as other therapies for acute myocardial infection.

Q. Do you think that if Dr. Deranian had just ordered a fluid challenge in response to the tachycardia and hypotension at 4:50 the decompensation that occurred over the ensuing minutes would have been avoided?

A. Yes.

Q. What do you base that on?

A. The fundamentals of the pathophysiology that I have outlined in a patient with hypovolemic shock and the compensatory tachycardia that is seen as a way of maintaining homeostasis that was blocked by the administration of this drug.

I think that's it. Maybe I will ask you to repeat the question to make sure I finished it.

Q. I think you answered it.

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How long a period of time would you expect the 2.5 milligram dose of Lopressor given as a bolus to impact Mrs. Hoffman's vital signs?

A. How long would it last?

Q. Yes. What's the period of its duration?

A. Well, with all the caveats that I gave you for the Lasix, which are many, this is a drug that typically lasts for hours.

Q. Are there drugs that can be used to reverse its effect or blunt its impact?

A. There are. They aren't particularly effective, but one of them, for instance, in overdoses we sometimes use Glucagon. That's the one that comes to mind for me.

Then of course you can give drugs that stimulate the beta receptor to try to overdrive or -- since the drug competitively blocks the beta receptor, so a drug like Isoproterenol, for instance, or Dobutamine or even Dopamine in high doses. Definitely the one that is most specific for the beta receptor that I know of would be Isoproterenol. When a drug like this is given, these drugs may, even when

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given in high doses, potentially be ineffective, and certainly in the case of an overdose that's the issue.

Q. Does the fact that -- strike that.

Is Epinephrine another drug that could be used?

A. Epinephrine is another, norepinephrine. They all have beta activity.

Q. Did you review the code sheet in this case at all?

A. I did.

Q. Do you have any criticism of how the code was run?

A. No.

Q. Does the fact that some of the drugs you just mentioned were given and they did not cause Mrs. Hoffman to change course indicate anything about her underlying condition?

A. No. It would indicate to me that by the time a person reaches circulatory arrest, even when resuscitation occurs quickly, that the prognosis is very poor, and so while epinephrine is given as a part of advanced cardiac life support resuscitation, by that time, if you will

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excuse the analogy, the horse is out of the barn and you may not be successful.

Q. Are there other reasons you believe the resuscitation in this case was unsuccessful other than the fact that Lopressor had been given?

A. Well, I suspect that in the context of hypovolemia that it was more difficult than it would have been had she not been hypovolemic. Certainly that is an important contributor.

(WHEREUPON, a brief pause was taken.)

BY MR. LAX:

Q. Other than the temporal relationship between giving the Lopressor in Mrs. Hoffman's decompensation, are there other factors that you see in the chart that support your opinion that Lopressor was the immediate cause of her death?

A. Well, the temporal relationship is compelling, of course. It is a matter of minutes. That, coupled to the principles of the pathophysiology that I have outlined and the issues of compensatory hemostasis. So those two together.

Q. Would you expect a dose as small as 2.5

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milligrams to have caused this dramatic of decompensation as we see in this case?
A. It is certainly very plausible given how tenuous her condition was, as I said now on multiple occasions. So absolutely I would.
Q. It is plausible I understand, but is it something you think is a certainty or plausible or could happen? Where on that spectrum would you put it?
A. It is near the highest level of certainty that I could have as a clinician without divine insight.
MR. BROWN: That answers your question.
MR. LAX: It does. It doesn't get much stronger than that.
BY MR. LAX:
Q. Is there any way to tease out, as a cause of her tachycardia, the hypovolemia you have been describing versus underlying stress or her SIRS which you talked about?
A. Well, the hypovolemia is one of the variables that contributes to the SIRS. As I said before, the SIRS is tachycardia, tachypnea,

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white blood cell count pertubation and a deviation of the temperature. The temperature was deviated. That's not directly related to the tachycardia, but the tachycardia is related to the tachycardia. That's one for sure. The other is the tachypnea, which was mild, not severe, in the low 20's.
The SIRS criteria were established back in 1992 by a consensus conference vote essentially, and that was before my time. That consensus conference of experts essentially defined a logical, but somewhat arbitrary, set of criteria, and the intent behind that was to cast a wide net, so to speak, so that much of what I see that is SIRS is not necessarily due to infection or sepsis. As I said, that's a different category.
You will see -- most of the patients that come into the intensive care unit have SIRS.
There was another part to your question. I am sorry.
Q. Whether you can distinguish as a cause of tachycardia the SIRS from the hypovolemia.

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A. And you asked me about stress was the other one.
Q. Systemic stress.
A. Systemic stress. So systemic stress, which elicits catecholamine response -- the sympathetic nervous system and the adrenal glands excrete epinephrine, norepinephrine, vasoconstricting drugs that the body makes naturally and are released in the setting of stress of any type.
As I said before, this woman was in the extremes of stress. So every last drop of epinephrine that her adrenal gland could make was being made. So she is at a maximal level of stress, and so it isn't surprising to me that she is tachycardic. That's her baseline, but then when it escalates above and beyond that, it suggests to me that the stress level has increased as a result of the hypovolemia and that the body is trying to squeeze out every last additional drop of the catecholamines that are necessary to maintain hemostatis.
Q. If they had transfused her central venous pressure in the same time frame just prior

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to the Lopressor, that data would need to be interpreted before you could act on it; it doesn't simply say add fluid or remove fluid. I mean, it provides you data that requires interpretation; is that correct?
A. Yes.
Q. That cold be confounded by her mechanical ventilation at that point?
A. There is the potential for that, although, as I said, a seasoned clinician, more often than not, is able to work through that by looking at the measurement.
By convention, we measure it at end expiration. The reason for that is that the respiratory activity of the ventilator pushing air in and out of the chest will change those pressures, but at end expiration there is a transient period of no gas flow into the chest, and that's the period where the measurement should be read.
Q. Are you aware of any studies that demonstrate the use of a reliance on CVP -- transfusing CVP improved outcome in the ICU?
A. I am not, but there aren't any studies

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that have explicitly asked the question in the context of a hemorrhaging patient with all the caveats of this particular patient.

I do clinical investigations, and so I can tell you it would be virtually impossible to design such a study because making these measurements in real time would be difficult.

In 2005 and, for that matter, in 2002 or even in the '90s, it had been established and remains established as a basic fundamental principle of physiology such that it would be hard to design a trial that would prevent people from having this piece of information in a randomized consented trial.

You would have a hard time getting an ethics committee to approve that, I suppose. To say, well, we are not going to use something that's sort of commonly used because we want to see if it makes a difference in how you do, most people would have a problem with that.

Q. The remaining items on this May 6, 2005 letter that we have marked as Kress Exhibit 10, part of 3 says, failure to stay at night or

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direct the nurse in detail how to manage the volume status.

In your review of the records, is that an opinion that you reached? Are you critical of Dr. Deranian's interactions with the nurse?

A. Well, insofar as he gave her a direction to give Lopressor, yes.

Q. Prior to the direction to give Lopressor, are you critical of his not remaining at the bedside?

A. To be fair, the statement says or, not and. It says failure to stay at night or direct. I think it is implicit in the statement that it wasn't absolutely necessary that he physically remain in the building, and I share that view. It would have been one approach, but not the only.

But to give the nurse direction so that she was able to manage the volume status adequately, whether that be standing there at the bedside with her or over the telephone five minutes away, either one is acceptable in my view.

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Q. Are there -- other than Lopressor, are there any other causes of death which you considered in this case but ruled out?

A. Well, we have discussed a number of other issues here. The immediate cause of death in my view is the Lopressor. It isn't a cause of death in a vacuum.

Clearly, if she had received Lopressor upon arrival at 7 a.m., she wouldn't have died, but she received Lopressor on a background of profound unresuscitated hypovolemic shock. That in my view is the obvious immediate cause of death.

Q. Any others that were on your list of differential etiologies which you have eliminated?

A. Well, we have talked about some of them. You mentioned amniotic fluid embolism. I won't repeat what I have already said. That's been discussed. I didn't consider it very strongly until I read others in the records who had considered that possibility, but for the reasons I already said, I was able to quickly dismiss that.

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Of course, ongoing hemorrhage is contributing to this for sure, but that is not the cause of death. That is the handicap that sets her up for the cause of death, which is the administration of the Lopressor.

Q. Do you think you have described for me all of the opinions you have formulated in this case?

A. I believe I have. I can't think of anything of any substance that we haven't covered.

Q. Are there -- strike that.

Do you think you have described for me the central records you have relied on? Have you talked about the records that were central to your forming your opinions in this case?

A. Yes.

Q. Did you do any literature search in connection with your work in this case?

A. Not explicitly in connection with this case, but as this is ongoing and I am asked to give lectures and things, including the one I am giving on Friday on shock, I have certainly

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updated my lecture on shock, not for the purpose of this case, but for the purpose of the other things. So I suppose you could label it as a convenience that I so happen to have to review this area to keep up on what I am asked to do as a part of my job, and certainly I use that when I formulate opinions.
Q. Do you anticipate doing any further work in connection with this case at this point?
A. With regard to literature review?
Q. With regard to anything.
A. I guess I will wait to hear from you all on that.
Q. If you --
A. Potentially, sure. Obviously the case isn't done yet as far as I can tell. I certainly -- if you are asking me do I plan on washing my hands today and never coming back, no.
Q. If you change your opinions or something you review changes your opinions, I trust you will let counsel know so they will let us know.
A. Of course.
Q. Anything we have talked about that you feel like you need to clarify or explain so that

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I have a good idea of what your testimony will be?
A. I don't think so.
MR. LAX: In that case, I want to thank you for your time. I don't think I have anything further. I don't know if there are topics I have omitted that anybody else wants to ask about.
MR. MALLORY: I am Mark Mallory, and I represent Seacoast Anesthesia. I hope to restrict this to very few questions.

E X A M I N A T I O N

BY MR. MALLORY:
Q. When you received the file from Abramson, Brown & Dugan, were you specifically asked to not review the anesthetic care of the patient during her admission to Exeter Hospital?
A. I don't remember it as an explicit negative. I remember it as more of an explicit positive in the category that I outlined already.
Q. Meaning that you were asked to review the intensive care issues?
A. I was asked -- well, my colleague who has worked with Abramson, Dugan & Brown has

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connections to this and said I know of someone who might be able to help. I think it was Mark Abramson who approached me. I know it was Mark Abramson who approached me and asked me if I had any ability to render an opinion on a case of a patient in the intensive care unit who had suffered after an obstetrical catastrophe and such. I said of course.
He said we are looking for an opinion regarding the patient's care in the intensive care unit and what my credentials are, and I told him what they were. He said we would like your opinion in that regard. I said fine.
Q. You are both a board certified internal care doctor with a specialty in critical care medicine as well as a board certified anesthesiologist; is that correct?
A. Yes.
Q. You were provided the entire record of the patient's care, correct?
A. Yes.
Q. If you had, in the course of your review of the records, identified any areas that you thought indicated anesthetic misconduct or

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inappropriate care, would you have called those to someone's attention?
A. Well, I wasn't asked to give an opinion in that regard, so my focus -- as I have said on numerous occasions here, you can't interpret these events in a vacuum. You can't disconnect everything.
Certainty if I saw something related, I would make a note of it, but only insofar as I am being asked to give an opinion regarding the critical care. I think I have alluded to that.
I think that there was a bleeding event related to a line placement that was critically important to the management that she received in the intensive care unit under the direction of the critical care physician. So to say, well, that's irrelevant, I am not going to pay any attention to that obviously would be shortsighted.
On the other hand, to ask me specifics about the anesthetic management and the placement of the catheter and all that, no. I was focusing my attention on the critical care

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management. I limited the evaluation in that regard. I hope that answers the question.
Q. I think it does. What it tells me is you don't have any opinions that there was any departure from the standard of care in regard to the anesthetic.
MR. BROWN: Objection. That's not what he said. He said he didn't render an opinion. He wasn't asked to render an opinion in that regard.
BY MR. MALLORY:
Q. You don't intend to express any opinion at trial in regard to the anesthetic care of Mrs. Hoffman?
A. Insofar as I haven't been asked to do that and I haven't focused my attention on any of those issues, it seems irresponsible for me to suddenly render an opinion in an area that I haven't really been asked to give one.
Q. Fair enough.
MR. BROWN: We don't intend to at trial, Mark.
MR. MALLORY: I understand.
MR. BROWN: You have your own experts.

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MR. MALLORY: I understand.
MR. BROWN: I just wanted to remind you of that.
BY MR. MALLORY:
Q. In your direct examination with Mr. Lax, you made reference to the attempts of the anesthetic team to place some central lines and the difficulties they had experienced. I just wanted to know whether or not, as a baseline matter, you felt that it was appropriate under the circumstances of Mrs. Hoffman's condition to be making the attempts to place the central lines when they did.
A. From the perspective of a person who will be taking care of the patient both before and after the operation, I believe it is very important and appropriate to establish venous access in a patient who is bleeding to death. So having her return under my care if I am the intensive care physician, by all means I would expect that those things would be done. If they weren't done, then it would be my responsibility to --
Q. Get them done.

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A. Right. Certainly that would be within the realm of what I would expect.
MR. MALLORY: That's all I have.
MR. LAX: Can I do one thing on the record? I don't think I have any more questions. Can I just see your binder of the depositions.
MS. MICHAUD: I do have a couple questions.
MR. LAX: Go ahead. I am going to take a look at this while you are doing that.
MS. MICHAUD: Doctor, my name is Elaine Mishaud. I represent Exeter Hospital. I will make this very, very brief.
Ken, Dr. Kress is not going to be identified as an expert providing opinions regarding the nursing care and the conduct of the nurses in this case; is that correct?
MR. BROWN: That's correct.
MS. MISHAUD: So I will not ask you, Doctor, about any concerns or questions you may have with respect to the nursing care. I do have a couple questions with respect to your testimony earlier regarding the potential causes of Mrs. Hoffman's DIC.

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E X A M I N A T I O N
BY MS. MICHAUD:
Q. In particular, am I correct that based on your review of the records, you identified two plausible explanations for her DIC, one being the intrauterine fetal demise, the other being potentially an amniotic fluid embolus? Is that correct?
A. Those seem to me -- I am not a hematologist, so I am not an expert in DIC. Certainly I see DIC with a regular enough frequency that I can render an opinion, and those explanations are the ones that I would put highest on the list. I don't mean it to be exhaustive, but yes. The answer to your question is yes.
Q. In your experience in dealing with patients with DIC, have you had direct experience with DIC which has been confirmed as being triggered by an intrauterine fetal demise?
A. I can't tell you as I sit here today that I recall an explicit incident. However, that is certainly something that one would consider.

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Q. Doctor, in your experience, and perhaps not direct experience but simply the knowledge that you have related to intrauterine fetal death triggering DIC, do you have an understanding as to the typical length of time the fetus should remain in the uterus before DIC is triggered?
A. Only in very broad terms. I will say that my understanding is that the longer that it remains -- that the expired fetus remains in utero, the more likely there are to be problems in that regard, but I can't quote you a specific time reference or line to that effect.
Q. In this instance, do you have an understanding that, based on Mr. Hoffman's testimony and also information in the record, that, at most, the Hoffman intrauterine death occurred less than 48 hours before Mrs. Hoffman was induced for delivery?
A. I do.
Q. Based on the fact that it is a brief period of time, the 48 hours, would that make it less likely that the intrauterine fetal death would have been the trigger for the DIC?
A. I believe so.

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Q. When you compare your two plausible alternatives, the intrauterine fetal death and the amniotic fluid embolus, would that steer you more towards the amniotic fluid embolus as a trigger for the DIC?
A. Both of those, in the context that you have painted them, are rare events. Amniotic fluid embolus is a rare event. So to invoke amniotic fluid embolus, as I said earlier, is a diagnosis of exclusion. So I don't have a strong obvious reason for the DIC.
Your point is correct. The period that the fetus had been in utero after it had died, to the best of one's ability to answer that, and that's based upon the mother noting that the baby wasn't moving anymore and then the subsequent visit to the obstetrician, the ultrasound that disclosed no fetal heart activity and such, the time frame was short.
So neither of these are obvious glaring explanations where one would say it is crystal clear to me why this woman developed DIC. In fact, if it had been crystal clear, I think everyone wouldn't have been so surprised on the

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early part of her care, the obstetrician, the obstetrical nurses, the patient and the family, for that matter. I am quite sure they didn't expect to come into the hospital and have these events unfold, nor the operating staff, the surgeons, the obstetricians, everyone involved, the anesthesiologist.
So it occurred unexpectedly, and often that's the case when there isn't a crystal clear reason. If she came in in septic shock and started to manifest DIC, everyone would say this is not surprising, but this was surprising.
So either of those explanations that I have outlined are not high likelihood in and of themselves. There are certainly times in this condition, as in many conditions, where the reason we don't -- the reason for the problem we don't discover, the so-called idiopathic. So one might put that into the bin of possible explanations as well. It is unsettling to have to give it that, but there are many times where we simply say I don't know why this happened.
Q. Recognizing that both amniotic fluid

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embolus and intrauterine fetal death in triggering DIC are rare events, can you in this particular instance rank one over the other as more plausible?
A. No, I don't think I can. They are both unlikely given the scenario that we have outlined. They are near the bottom of the list in terms of statistical likelihood. It would be, I think, foolish for me to try to put one higher than the other. They are both unlikely.
Q. Just so I am clear, when you say both are unlikely, Doctor, are you stating that there are other possible more likely causes of her DIC?
A. No. Let me make it clear. You made a good point.
What I am saying is that, statistically speaking, in all patients presenting with DIC, those two explanations are both quite uncommon. In her case, I think one of the two is probably what happened, but that's because I don't have a more common -- and the example I gave was sepsis because it is one of the most common that we see -- a more common explanation to substitute for the rare ones that

I have.
So is it possible that it is one of those two or a third or fourth that I can't uncover? Yes. In her particular case, intrauterine fetal demise or amniotic fluid embolism have to be higher than all comers because we don't have another explanation, but having said that, they are both in and of themselves rare events.
Q. Again, in this particular instance, you would consider them equally plausible but not one over the other?
A. I would hesitate to use the term equal because the implication there means that I know with certainty that one is the same as the other. I would rather state it to say neither of them clearly overshadows the other in such a way that I can confidently rank order them.
MS. MICHAUD: Thank you, Doctor. That's all I have.
MR. BROWN: Greg.
MR. PETERS: I just want to confirm you don't plan to use or offer any testimony from Dr. Kress against Core at trial.

MR. BROWN: Correct.
MR. PETERS: Based on that, I have no questions.
MR. LAX: In that case, Doctor, I will thank you for your time. Like I said at the beginning, a copy of your transcript will be provided to plaintiff's counsel. I would ask you to take a look at that and make any corrections you think are necessary to make it a complete and accurate transcript.
If you change the light was green to the light was red, we will probably comment on that and how it might impact your credibility, but we do want it to be accurate, so please take your time and make the corrections and get it back to us. If you don't, we will assume it was perfect the way it reads when you get it.
MR. BROWN: We will thank the stenographer for putting up with all of us today.
(FURTHER DEPONENT SAITH NAUGHT.)

STATE OF ILLINOIS)
) SS:
COUNTY OF C O O K)
I, ETTA R. JONES, a notary public within and for the County of Cook and State of Illinois, do hereby certify that heretofore, to-wit, on the 17th day of August, 2005, personally appeared before me, at 5841 South Maryland, Chicago, Illinois, JOHN P. KRESS, M.D., in a cause now pending and undetermined in the Circuit Court of Cook County, Illinois, wherein RALPH HOFFMAN is the Plaintiff and EXETER HOSPITAL, INC., et al., are the Defendants.
I further certify that the said witness was first duly sworn to testify the truth, the whole truth and nothing but the truth in the cause aforesaid; that the testimony then given by said witness was reported stenographically by me in the presence of the said witness and afterwards reduced to typewriting by Computer-Aided Transcription, and the foregoing is a true and correct transcript of the testimony so given by said witness as aforesaid.

I further certify that the signature to the foregoing deposition was not waived by counsel for the respective parties.
I further certify that the taking of this deposition was pursuant to Notice and that there were present at the deposition the attorneys hereinbefore mentioned.
I further certify that I am not counsel for nor in any way related to the parties to this suit, nor am I in any way interested in the outcome thereof.
IN TESTIMONY WHEREOF: I have hereunto set my hand and affixed my notarial seal this _____ day of _____, 2005.

_____
Notary Public, Cook County, Illinois