

1 you are at a remote location, the oath administered by
2 Ms. Garcia will be the same as if you were here present
3 in the courtroom. It requires you to tell the truth
4 and subjects you to penalties of perjury if you do not
5 answer those questions truthfully.

6 Do you understand that?

7 THE WITNESS: Yes, I do.

8 THE COURT: All right. With that, I'll ask
9 Ms. Garcia to place you under oath.

10 ERIN DAVID BIGLER,
11 called on behalf of the Petitioner, having been first
12 duly sworn upon oath, was examined and testified as
13 follows:

14 THE CLERK: Please state your name, and spell
15 your last name for the record.

16 THE WITNESS: My name is Erin, E-r-i-n, David
17 Bigler, B-i-g-l-e-r.

18 THE CLERK: Thank you.

19 THE COURT: Mr. Nevin, you may inquire of the
20 witness.

21 MR. NEVIN: Thank you, Your Honor.

22 DIRECT EXAMINATION

23 QUESTIONS BY MR. NEVIN:

24 Q. Dr. Bigler, will you tell the Court where you
25 live and what it is you do for a living, please?

1 A. Yes. I live in Provo, Utah, and I'm a
2 professor at Brigham Young University and the
3 University of Utah.

4 Q. And what's the area in which you teach,
5 Dr. Bigler?

6 A. I teach in the area of clinical
7 neuropsychology and neuroscience.

8 Q. Dr. Bigler, would you please explain briefly
9 what your education has been and what your employment
10 has been since you've been in the field that you're in
11 now?

12 A. Yes. I completed an undergraduate degree in
13 psychology at Brigham Young University in 1971, and I
14 stayed at BYU to do my doctoral work, which I completed
15 in 1974.

16 Upon completion of my doctoral studies, I was
17 the recipient of a National Institute of Health, plus
18 doctorate fellowship that I took at the Barrow, that's
19 B-a-r-r-o-w, Barrow Neurological Institute at
20 St. Joseph's Hospital and Medical Center in Phoenix,
21 Arizona, and I was there until 1977.

22 And upon completing my post-doctoral
23 fellowship, I joined the faculty at the University of
24 Texas to develop their clinical neuropsychology
25 training program in their Ph.D. program in clinical

1 psychology. And I led and directed that program from
2 1977 until 1990 at the University of Texas where I went
3 through the academic ranks from assistant to full
4 professor.

5 In 1990, I returned to Utah and joined the
6 faculty at BYU where I am a professor of psychology and
7 neuroscience and where I direct the clinical
8 neuropsychology subspecialty training in the clinical
9 psychology Ph.D. program here at BYU.

10 I am also an adjunct professor of psychiatry
11 at the University of Utah, where I'm involved in
12 several large research projects. And currently I have
13 research that is funded by the National Institute of
14 Health on traumatic brain injury, and also on
15 developmental disorders such as autism.

16 Q. Are you also an adjunct professor of
17 radiology?

18 A. I have held that appointment as an adjunct
19 professor of radiology until 2006. In consolidating my
20 time and effort and focusing, I am no longer an adjunct
21 professor of radiology in the radiology department at
22 the University of Utah.

23 Q. Now, those appointments as adjunct professor
24 at the University of Utah, were those at the medical
25 school or --

1 A. Yes.

2 Q. They are.

3 A. At the university of -- yes, they are at the
4 University of Utah Medical School.

5 Q. Do you have any particular emphasis in the
6 area of brain imaging?

7 A. Yes. That has been my specialty throughout my
8 professional career. While I was doing my
9 post-doctoral training at St. Joseph's Hospital, they
10 were actually the first hospital in the southwest to
11 receive a computerized tomogram. And that
12 revolutionized how we could image the brain and view
13 the brain in a living individual. And that came online
14 in late 1975.

15 And since that period of time, I have been
16 studying the relationship between various brain
17 abnormalities that can be seen on brain scans and how
18 that relates to behavior, and particularly the behavior
19 that we can examine using neuropsychological
20 techniques.

21 Q. Are you part of an institute or a laboratory
22 at Brigham Young that focuses on brain imaging?

23 A. Yes. I actually developed this laboratory
24 while I was at the University of Texas. It's called
25 the Brain Imaging and Behavior Laboratory. And what it

1 focuses on is studying the findings on brain scans and
2 relating that to the behavior of the individual. And
3 we've been doing this type of research, as I mentioned,
4 since the mid 1970s.

5 Q. Dr. Bigler, are you board certified in any
6 fields?

7 A. Yes. I'm a licensed psychologist and I'm
8 board certified in the field of clinical
9 neuropsychology by the American Board of Professional
10 Psychology.

11 Q. Could you say just briefly what the difference
12 between neuropsychology and psychology is?

13 A. Certainly. Neuropsychology is the study of
14 the interaction between the human brain and behavior of
15 the individual. Clinical psychology and psychology in
16 general is typically looking at the broader view of
17 behavior without necessarily considering how the brain
18 is regulating behavior.

19 In neuropsychology, the basic premise is that
20 the brain is the master organ of behavior, and that by
21 studying how the brain functions we can relate that to
22 behavior. But we can also do the inverse.

23 So we can look at behavior and make inferences
24 about how well or how dysfunctional the brain may be in
25 a given individual.

1 Q. Well, as long as we're on this subject, could
2 you just distinguish briefly for us the fields of
3 neurology and neurosurgery and how they relate to your
4 field?

5 A. Certainly. Neurology and neurosurgery are
6 medical specialists. And, obviously, they have to
7 first have a medical degree and then go through their
8 respective residency programs to certify in the areas
9 of neurology and neurosurgery.

10 The compliment between neurology,
11 neurosurgery, and neuropsychology is that going back to
12 the brain being the master organ of behavior and
13 psychologists being interested in behavior, we're the
14 discipline that looks at the overall behavior of the
15 individual.

16 The neurologist looks more at the medical
17 aspects of what has happened or has been dysfunctional
18 with a brain. The neurosurgeon looks at it from the
19 standpoint of what's a surgical treatment problem and
20 how that may be carried out and how that may be
21 medically managed.

22 Q. Thank you. Do you have publications that you
23 have made, completed during the course of your career?

24 A. Yes.

25 Q. And let's talk just first about books. Have

1 you published books that --

XDp 2 MR. NEVIN: You know, Your Honor, I might say,
3 I have marked as an exhibit, Exhibit 1061. 1061-2.
4 And I believe the parties will stipulate that this is a
5 true and accurate copy of Dr. Bigler's vitae. I move
6 it into evidence at this time.

7 THE COURT: 1061-2?

8 MR. NEVIN: Yes, sir.

9 THE COURT: Okay. I --

10 MR. NEVIN: Yeah, I think it's actually
11 supposed to be 1061-3.

12 THE COURT: Thank you. That's what I have it
13 marked as. Is it 1061-3, then, that you're offering?

14 MR. NEVIN: Could I inquire of the clerk, Your
15 Honor --

16 THE COURT: Yes, you may.

17 MR. NEVIN: -- whether the original of the
18 vitae is actually marked as 1061-2 or 3? This would
19 be -- it's supposed to be a document about 65 pages in
20 length.

21 THE COURT: Well, just so we're clear --

22 MR. NEVIN: It's 3? Okay.

23 Yes, Your Honor. I wanted to make sure the
24 correct exhibit sticker was on. It is 1061-3. I move
25 that into evidence.

1 THE COURT: Any objection?

2 MR. ROSENTHAL: No objection, Your Honor.

3 THE COURT: 1061-3 will be admitted.

4 (Whereupon, Exhibit 1061-3 was admitted.)

5 BY MR. NEVIN:

6 Q. And Dr. Bigler, your vitae dated October of
7 2006 is in evidence now. And would it be correct that
8 this vitae lists your publications?

9 A. That is correct.

10 Q. Okay. And just briefly, if you would, have
11 you authored books that deal with the issues of
12 neuroimaging?

13 A. Yes, I have.

14 Q. Would you just describe those for us briefly?

15 A. Well, when I was still a professor at the
16 University of Texas it was obvious to me that a
17 textbook needed to be directed integrating what we knew
18 about neuropsychology then and brain imaging.

19 And so in 1984 I published a textbook by the
20 University of Texas Press titled "Diagnostic Clinical
21 Neuropsychology," and that textbook focused primarily
22 on computerized tomography because that's all that we
23 really had then, and how various brain lesions related
24 to the behavior of the individual and how that related
25 to their neuropsychological performance on tests.

1 That was followed up by another textbook in
2 1989 called "Brain Imaging and Neuropsychological
3 Function." And this was actually the first edited
4 textbook that was directed to this emerging field of
5 integrating imaging with behavior.

6 And then following that, in the 1990s, there
7 was an update of the "Diagnostic Clinical
8 Neuropsychology" textbook by the University of Texas
9 Press. And then a two-volume textbook series by Plenum
10 Press dealing with neuroimaging. That's the title of
11 those books. It's "Neuroimaging One" and "Neuroimaging
12 Two."

13 Q. The neuroimaging series, where would those be
14 used as text -- textbooks? In what kind of an
15 educational program?

16 A. Well, those have been used in clinical
17 neuropsychology training programs, in neuroscience
18 programs, and also in medical schools in the specialty
19 areas of neurology, psychiatry, and physical medicine
20 and rehabilitation.

21 Q. Dr. Bigler, have you also published articles
22 in scholarly journals?

23 A. Yes.

24 Q. And roughly how many such articles have you
25 published during your career?

1 A. I believe it's around 250.

2 Q. And of those 250, can you say roughly how many
3 of them deal with this area of neuroimaging and
4 behavior?

5 A. Probably two-thirds of them have some
6 component of brain imaging. That's the basis of the
7 article.

8 Q. Do you consider yourself to be a leader or to
9 be somewhat prominent in this field of neuroimaging and
10 its relationship to behavior?

11 A. Yes.

12 Q. Are there others who have also published in
13 this area whose contribution to the literature and the
14 research is similar to yours?

15 A. Yes. This is a major area of research and
16 emphasis throughout the world in neuroscience centers.
17 We have marvelous imaging technology now. And this is
18 where the big search is, to look at the relationships
19 between imaging findings and behavior.

20 Q. Doctor, when did you first have some contact
21 with anyone about this case we're here on today
22 involving Richard Leavitt?

23 A. I just have to get my notes. For some reason
24 the screen is now showing my computer and not you. Did
25 something just change?

1 THE COURT: Not that I know of from our end.

2 MR. NEVIN: Not from our end.

3 THE WITNESS: That's okay. You're in a little
4 screen I see in the bottom.

5 But with regards to your question, I received
6 materials on October 9, 2006, from you, and I believe
7 that you had contacted me by phone sometime prior to
8 that.

9 BY MR. NEVIN:

10 Q. Doctor, I'll say let's continue with the
11 questioning. We are seeing your computer on our screen
12 here instead of you.

13 THE COURT: Counsel, I'm going to summon
14 someone from our automation -- even though I think it's
15 probably something on the other end, at least they may
16 be able to tell us what Dr. Bigler can do to correct
17 this.

18 BY MR. NEVIN:

19 Q. I was just going to say, Dr. Bigler, if you
20 have the ability to -- if something has changed from
21 your end and if it can be changed back so that we have
22 your picture in the presentation system again, that
23 would be useful.

24 A. Well, we didn't touch anything here. So I'm
25 not sure that it was initiated on our side, but maybe

1 there's something that -- on the computer --

2 THE COURT: There. We're back.

3 THE WITNESS: There we go. Okay.

4 THE COURT: I don't know what caused that,
5 since no one is pushing any buttons over here that I
6 can see. Anyway, let's go ahead.

7 BY MR. NEVIN:

8 Q. There is nothing in your training, I take it,
9 that permits you to answer these questions, Doctor?

10 A. No. That's why I have staff around me,
11 because I blame them for everything that goes wrong.

12 Q. Excellent. That's a great strategy.

13 Doctor, you were saying that I contacted you.
14 And if you could just, you know, quickly tell us what I
15 asked you to do and if you could describe briefly what
16 you have done in the case since being involved in it.

17 A. Yes. Well, as I mentioned, I believe that you
18 contacted me first by phone, probably in late September
19 of 2006, and you indicated that you had a client and
20 that there were some imaging studies and that there
21 were also neuropsychological tests, psychological
22 tests, and psychiatric evaluations that had been done.
23 And there was a question about how findings in an
24 earlier CT scan, in a 1996 MRI scan and a 2006 MRI scan
25 and what those findings might mean in terms of an

1 individual's neuropsychological status.

2 Q. And let's get one thing out of the way,
3 Doctor. Are you being paid for the consultation that
4 you're providing in this case?

5 A. Well, I am not being paid directly. The brain
6 imaging and behavior lab at the university does bill
7 for any services like this. But none of that comes to
8 me directly. It goes to a research account here at the
9 university.

10 Q. And do you know what the rate is that we're
11 paying you?

12 A. Yes. The rate for the lab, I believe, is 200
13 an hour.

14 Q. Okay. Thanks, Doctor.

15 Well, tell us briefly what you have reviewed
16 prior to coming to -- well, I was going to say coming
17 to court -- prior to appearing in court today.

18 A. I've reviewed those scans that I already gave
19 dates on. And then I have reviewed this stack of
20 materials. Do you want me to go through this quickly
21 then?

22 Q. Well, given that we may have some time
23 limitations here and that we'd like to move along, let
24 me ask it this way. Is it correct that you've reviewed
25 these scans and that you have also reviewed the reports

1 of Drs. Beaver, Missett, Engle and Martell?

2 A. That is correct.

3 Q. And are there additional materials beyond
4 those which you've reviewed?

5 A. Dr. Andersen's report. The radiology -- wait
6 a minute. Dr. Jackson -- no, excuse me. The
7 deposition of Bruce Andersen.

8 Q. And I think it's correct that you've also
9 reviewed reports that were prepared by radiologists
10 regarding the scanned MRIs in '96 and in 2006; is that
11 right?

12 A. That's correct.

13 Q. Okay. And have you also had a report from a
14 radiologist just recently, a Dr. Mack, who comments on
15 the 1996 MRI?

16 A. Um...

17 Q. And I think that would be in the form of a
18 brief e-mail report that was sent to Mr. Parnes and
19 then forwarded on to you.

20 A. Yes, I'm just trying to find that here.

21 I apologize. I don't seem to have that in my
22 file right at this moment, but I do remember that.

23 Q. Okay. Well, I -- it may not be necessary to
24 have it for the purposes of the testimony here today,
25 Doctor.

1 So let me ask you, and let's begin -- well,
2 we'll talk about this in more detail prior to
3 concluding your examination, but if we could, could you
4 just say in overview what -- could you just say an
5 overview what you observed about -- if anything, about
6 Mr. Leavitt's 1996 and 2006 MRIs? And then
7 anticipating that you're going to refer to some aspects
8 of those screens, I think I'd like to go ahead and try
9 to display those for the Court so that we can see
10 those. And then we will proceed from there to some
11 discussion of what those features of those scans mean
12 in your opinion.

13 So let's start with just an overview of what
14 you saw there.

15 A. Yes. In the 2006 report, I think that's
16 probably the place to start because that's the most
17 recent scan and it has probably the best technology
18 going with it that's most contemporary with the kinds
19 of imaging that's done today.

20 And looking at that report, that report
21 indicates that there are white matter, what are
22 referred to as signal changes in the brain of this
23 individual. And it outlines where those abnormalities
24 are and it pinpoints those abnormalities and then it
25 has the conclusion that these are possibly related to

1 ischemic change, old trauma, or some other etiology
2 affecting the white matter of the brain.

3 The white matter of the brain is the part of
4 the brain that connects brain cells, and that's kind of
5 like the telephone lines that allow us to talk to one
6 another. The white matter reflects the connections in
7 the brain that allow different areas of the brain to
8 talk to one another, so to speak.

9 The location of white matter abnormalities has
10 some bearing on a patient's neuropsychologic,
11 neuropsychiatric status and tells us something about
12 what may relate to behavior in that individual.

13 Some of the abnormalities are located in the
14 frontal region of this individual's brain and they are
15 distinctly seen on the scan.

16 If you go back to the 1996 scan, a different
17 era, somewhat different technology, certainly not as
18 good as the technology that we have today. And even
19 though it's not reported in the scan radiologic report,
20 you can actually see that these white matter changes
21 and differences were there in '96.

22 And then if you go back to the old CT scan
23 that was done back in 1983, it was also questioning
24 whether there were white matter changes and whether
25 there were white matter changes happening in the brain

1 at that point in time that might be, as I'm reading
2 from the report, an indication of a demyelinating
3 disease.

4 So what you have, in my view, here is a series
5 of scans that have actually pointed to some potential
6 problems in this individual's brain, and these can be
7 visualized, they can be identified. They do have some
8 relevance in evaluating an individual such as this.

9 Q. Okay. And, Doctor, let's do -- let's first do
10 one thing. And I think you were probably reading from
11 the report of the CT scan, and I'll represent to you
12 that that's actually dated in 1985, 198- -- not 1983,
13 although that copy is so bad that you're probably lucky
14 to have even gotten in the right decade on that.

15 THE COURT: Counsel, could you identify the
16 scan by the doctor or the name or your exhibit number?
17 I assume those are all going to be stipulated in? It
18 might be easier to do it as we go along.

19 MR. NEVIN: I actually will get that done as
20 we move through the examination.

21 THE COURT: All right.

22 BY MR. NEVIN:

23 Q. Well, Doctor, let's now have a run at
24 displaying first the 2006 MRI. And I can --

25 MR. NEVIN: Your Honor, I will say I can put

1 that up on the screen.

2 BY MR. NEVIN:

3 Q. Doctor, do you -- is your understanding of
4 where you are right now that you're able to put the
5 particular scans from the 2006 MRI up on the screen
6 from your end?

7 A. I have the 2006 on the screen right now. We
8 have the 1996 on a flash drive, so we'll have to take a
9 quick break for a moment. But if we -- if we go to my
10 screen, which -- can you go to my screen?

11 Q. No, I think you just to send us -- we have to
12 change from -- so what you are sending us is from -- is
13 your computer.

14 THE WITNESS: Where did she go? Yeah, that's
15 what we need.

16 Okay.

17 THE COURT: Now, Counsel, what is the exhibit
18 number here?

19 MR. NEVIN: This would be, Your Honor,
20 Exhibit -- we are looking at Exhibit 1072-1. This is a
21 DVD which contains the images from the 2006 MRI, and I
22 move it into evidence.

23 THE COURT: Any objection?

24 MR. ROSENTHAL: No objection, Your Honor.

25 THE COURT: Exhibit 1072-1 is admitted.

1 You may proceed.

2 (Whereupon, Exhibit 1072-1 was admitted.)

3 MR. NEVIN: Thank you, Your Honor.

4 BY MR. NEVIN:

5 Q. Doctor, are you still -- are you able to hear
6 me at this point?

7 A. Yes.

8 Q. Okay. And you have put an image from the 2006
9 MRI up on the screen right now; is that correct?

10 A. That is correct.

11 Q. Okay. Now, is there something meaningful in
12 the imaging that you're displaying on the screen now,
13 and if so, could you describe what that is?

14 A. Yes. They are already highlighted. And here
15 is my cursor that I'm controlling. And so there is a
16 little bit of a delay, but I'm going to move it over
17 and you can see the arrow right here. That arrow is
18 pointing to that white blotch that appears right here
19 that I'm pointing to, that's what's called a
20 hyperintense signal.

21 What is a very nice circumstance in the normal
22 brain is that the normal brain is symmetric. And so
23 looking at one side of the brain is a reference for
24 what the other side of the brain should look like.

25 So if you go to this hyperintense signal

1 that's right here and you come directly over to this
2 region here in the brain, that region in the brain
3 doesn't show that signal intensity. And that is why
4 this is called by the radiologists an abnormal focus in
5 the MRI on that 2006 date.

6 This is in the frontal area of the patient's
7 brain. And if you move down here, there's another
8 arrow and --

9 Q. Doctor, excuse me. You're now moving down to
10 the lower left-hand corner?

11 A. Yes. I'm moving down to the lower left-hand
12 corner.

13 Q. And I might ask as well, this is an area -- on
14 the DVD of this 2006 MRI, the portion of the DVD that
15 we're looking at is the screen that's entitled montage;
16 is that correct?

17 A. You know, I don't -- oh, yes, yes. Yes, it is
18 on the lower left. Yes, that's correct.

19 Q. And this is a series of images that have been
20 brought here to this screen by the radiologist to
21 illustrate the radiologist's findings; isn't that
22 correct?

23 A. Yes. It's my understanding this is done by
24 Dr. Jackson. This is not done by me.

25 Q. Okay. Go ahead, sir.

1 A. So here is another area of what is referred to
2 as a hyperintense signal that I'm pointing to right
3 here and the radiologist identified with this arrow.
4 This again is in the white matter of the brain. It's
5 in the frontal area of the brain. And you see that
6 there is not a corresponding signal in the other
7 hemisphere.

8 And then moving to the lower right-hand
9 corner, we see again a signal difference in the
10 inferior frontal area. This is just above the eye now.
11 We're very low in the frontal lobe. And I'm circling
12 it here. And that shows, again, the hyperintense
13 signal difference in the frontal area that's not seen
14 in the other frontal lobe.

15 Q. Okay. Doctor, if I could interrupt for just
16 one minute and ask you a couple of quick orienting
17 questions.

18 These images that you have pointed to, they
19 are showing an image of the head looking down from the
20 top; is that correct?

21 A. It's actually more like you're looking up from
22 the bottom because this is the left and this is the
23 right. So it's as if you're looking or facing the
24 patient. So in radiologic perspective, it's as if
25 you're looking at the patient. So what's on your left

1 is actually the patient's right and what's on your
2 right is actually the patient's left. And you'll see
3 right here, I am circling it, the L stands for left,
4 the R stands for right.

5 Q. Okay. And the top of these three images that
6 you have pointed to so far, the top of them would be
7 the front of the person's head, the bottom of them
8 would be the back of the person's head; is that right?

9 A. That's correct. This is -- where I'm wiggling
10 the cursor now, the nose would be just beneath here and
11 this is the back of the head. If you go to the upper
12 left-hand image here, this is more recognizable to
13 people that this is the brain.

14 And here is the frontal lobe. And this
15 abnormality that's in the lower right-hand corner would
16 be in this region here, in the inferior frontal area.
17 In the lower left-hand corner, this lesion that's here
18 where the arrow is pointing is up in this mid frontal
19 region of the brain, that I'm pointing to here. And
20 the signal change or difference that's seen in the
21 upper right-hand corner is more posterior in the
22 frontal region at around this level here. And --

23 Q. Okay.

24 A. -- so this is the front of the head. You can
25 see where the nose is starting to emerge here. This is

1 the back of the head, and here is the spinal cord here.
2 This is the cerebellum, and this is the cerebrum of the
3 brain.

4 Q. Now, Doctor, we -- I think it's correct, we do
5 not have a way of recording all of the things that you
6 have just indicated and correlating them to an actual
7 exhibit. So I'm going to say back, for purposes of the
8 record, what you just said and listen and tell me if
9 you think this is correct.

10 We might refer to the image in the upper left
11 as -- the image in the upper left shows the brain
12 looking at the side of the head with the person's --
13 this person's nose to the left and the back of their
14 head to the right; is that correct?

15 A. This is what is referred to as a mid-sagittal
16 view. It's like you split the head right down the
17 middle. So if you came right down the middle of the
18 face between the eyes and you removed one-half of the
19 head, it's like you're looking at the inside remainder
20 of the head.

21 Q. So the image in the upper right and the
22 abnormality that's indicated there or the
23 hyperintensity that's indicated there, when you go over
24 to the image in the upper left, that hyperintensity
25 appears pretty much right in the middle of the brain

1 area in the upper left-hand scan; correct?

2 A. Yes. I'm going to do one more thing here just
3 as a visual aid. This is what's called an axial view.
4 It's like you're cutting in the horizontal plane. And
5 so if we come over here, what we're doing is we're
6 cutting through at about this level right here that I'm
7 showing. And this abnormality here, when you flip the
8 image and now looking at it in the horizontal plane, is
9 showing a change in the frontal lobe around this
10 region.

11 Q. Okay. And again you're indicating, for
12 purposes of the record, you're indicating a spot pretty
13 much right at the bottom of the brain area right
14 centered from back to front in the upper left-hand
15 image; is that correct?

16 A. That's correct.

17 Q. All right. And now with respect to the image
18 in the lower left, that is -- the hyperintensity
19 indicated in that lower left image is one that's
20 located toward the front of the brain and fairly high
21 up in the brain there in that upper left-hand image.
22 Is that correct?

23 A. Not as high up as what's in the upper right
24 hand, but it's definitely in the frontal area. And
25 this little curvature right here that I'm pointing to,

1 this is called the ventricle. And if we move up to
2 this image here, this white area right here is the
3 ventricle.

4 And so this is just lateral to the ventricle.
5 So it's in the white matter in and around this region
6 of the brain that I'm showing right here. It clearly
7 is in the frontal lobe.

8 Q. Okay. And now with respect to the lower right
9 image, again similar in terms of back to front and side
10 to side to the lower left, but lower in terms of top
11 and bottom?

12 A. That's correct.

13 Q. Okay. Doctor, thank you. And let's now just
14 see if we can talk and have a similar discussion about
15 the 1996 MRI.

16 A. Okay. We are going to have to insert a flash
17 drive here and insert a few things real quick. I don't
18 know if you want to take a momentary break or --

19 Q. Doctor, another option would be for me to
20 display these 1996 images from the MRI here on our
21 system and you could point me to the particular image
22 numbers that you wanted to have placed on the screen.

23 THE COURT: Counsel, you would need to turn
24 off, then, the video feed or switch the input source.
25 Is that already arranged?

1 MR. NEVIN: We have done that previously. So
2 we would just have audio from Dr. Bigler and video from
3 me.

4 THE COURT: That's fine.

5 BY MR. NEVIN:

6 Q. Should we proceed that way, Dr. Bigler?

7 A. Well, it might be better if we do it on this
8 end just because I think what you would call up would
9 be all of the images and then we'll have to search the
10 ones for the right sequence, because I think we have --
11 we've got the right images right here. So since we are
12 already linked, can we take just a moment?

13 THE COURT: Yes. Go ahead. We will just stay
14 in session while you're doing that.

15 THE WITNESS: Are there any other verbal
16 questions that you can ask while -- I have my lab
17 director here who is --

18 THE COURT: Go ahead.

19 THE WITNESS: He's setting this up.

20 THE COURT: Yes. That's a good idea. And
21 while we are at it, I assume this is 1071-1 and 2?

22 MR. NEVIN: Yes, sir, I so move.

23 THE COURT: Is there any objection?

24 MR. ROSENTHAL: No objection, Your Honor.

25 THE COURT: All right. 1071-1 and -2 are both

1 admitted.

2 (Whereupon, Exhibits 1071-1 and 1071-2 were
3 admitted.)

4 THE COURT: Did you intend to offer 1072-2,
5 which I think is the report?

6 MR. NEVIN: Yes, Your Honor.

7 THE COURT: Is there any objection?

8 MR. ROSENTHAL: No objection, Your Honor.

9 THE COURT: All right. That exhibit is
10 admitted as well.

11 (Whereupon, Exhibit 1072-2 was admitted.)

12 BY MR. NEVIN:

13 Q. Dr. Bigler, while that's taking place, we keep
14 using this term "white matter hyperintensity." One of
15 the things I need to ask you, of course, is what is a
16 white matter hyperintensity?

17 A. Well, the brain, from a gross imaging
18 standpoint, has three unique characteristics, and that
19 has to do with the tissue composition.

20 So there's gray matter, and that's where cell
21 bodies reside. There's white matter; those are the
22 pathways in the brain. And then there is cerebral
23 spinal fluid. And the cerebral spinal fluid is
24 contained within ventricles which are internal cavities
25 of the brain and on the surface of the brain.

1 All of those different tissue types give us a
2 very characteristic and clean appearance on magnetic
3 resonance that is reflected in what is referred to as
4 the signal that is being emitted by that tissue.

5 And so you look for uniform signals. So
6 normal gray matter has a uniform appearance. Normal
7 white matter has a uniform appearance. And as I
8 mentioned earlier, the appearance is -- in the normal
9 brain is symmetric.

10 So what you see in the gray matter on one side
11 of the brain is what you expect to see in the gray
12 matter on the other side of the brain. In other words,
13 they should be a mirror image of one another.

14 Also, in white matter you should see a very
15 nice margin in the white matter as it connects to gray
16 matter. And you look for those sort of clean
17 boundaries because that helps you identify where
18 different kinds of problems and difficulties and
19 abnormalities may be in a particular brain.

20 And the other thing that one does, then, is
21 look at the location. There are areas where there are
22 higher frequencies for these changes that occur in the
23 brain and how that relates to behavior.

24 So it looks like we have images that are up on
25 the screen at this time.

1 Q. Right. Why don't you go ahead and -- excuse
2 me. Why don't you ahead and -- and these would be
3 images now from the 1996 MRIs; is that correct?

4 A. That's correct.

5 Q. Well, why don't you just go ahead and point to
6 us, show us what parts of that, if any, you consider to
7 be significant.

8 A. Well, here again, the image quality is
9 different, but these scans do show that there are white
10 matter changes.

11 Now, can you see my -- let me just ask my lab
12 director, is there a way to make the cursor more
13 distinct? See, it turns gray when I move in.

14 Just one moment.

15 (Pause in the proceedings.)

16 Okay. Can you see what we're outlining?

17 Q. I can't.

18 A. Okay.

19 Just one moment. Just bear with us.

20 (Pause in the proceedings.)

21 Okay. There we go. You can see here is the
22 cursor. I'm going to move it over.

23 Can you follow the cursor now?

24 So here is the cursor. I'm going to slowly
25 move it over and I'm going to put it right on top of a

1 white matter hyperintensity that is just off to the
2 side of the ventricle. It's in the same position that
3 is seen in the 2006 scan. And now I'm going to circle
4 it in yellow.

5 So I'm going to click and draw. And right in
6 the middle there is a white matter hyperintensity that
7 I put the cursor right on top of and it's in the same
8 position that is seen and viewed in the 2006 scan. You
9 can see it over in this image that's right here as
10 well. It's that white matter right there in the
11 center. Again, it's in the position that is seen more
12 clearly and more distinctly in the 2006 scan.

13 Q. Doctor, could you just state for the record
14 which images you were presenting on the screen now?

15 A. Yes. If you look right here, that says,
16 "Study 3, Image 56." And the first one I did also says
17 "Study 3, Image 43. And both of those are from the
18 February 22, 1996 scans.

19 Q. Are there other images from the 1996 MRI that
20 you think should be presented here to illustrate this?

21 A. These are the ones that we focused on for this
22 moment with our bit of imaging technology problem.
23 There is another image. That will take us a little bit
24 of time to find.

25 Q. Well --

1 A. But I think --

2 Q. If you could just state for the record,
3 Doctor, the number of the other image. And I think we
4 don't need to go through the process of actually
5 illustrating that at this point.

6 A. There is another image that, in my opinion,
7 also shows the white matter change. You have to
8 remember that the technology is a bit different and the
9 characteristics of the slices and the angles and the
10 width and those features are different. But there is
11 another image that also shows the white matter signal
12 change that we talked about in the 2006 image that can
13 be seen in the 1996.

14 Q. And, Doctor, without going through the steps
15 of presenting it on the screen, can you just identify
16 the image number of that third image, the one that's
17 not being presented?

18 A. The Image 54 --

19 Q. Image 54.

20 A. -- Study 3.

21 Q. Okay. Thank you.

22 And, Doctor, thank you very much for making
23 the effort to get those on the screen. Can we return
24 to having you in the presenter again, please?

25 Great. Thank you.

1 Now, Doctor, I asked you when we had a moment
2 while we were waiting there for the technology, I asked
3 you to explain what a white matter hyperintensity was,
4 and you had begun that process. And could you just
5 complete those remarks, please?

6 A. Well, it just is an indication that something
7 is not correct with the white matter in that region of
8 the brain, which implies that the connections in and
9 out of that region of the brain are not normal. And it
10 often can be related to the behavior of the individual.

11 Q. When you say the connections are not normal or
12 that there is something that has happened to the
13 connections, what kind of connections are you talking
14 about?

15 A. Well, the white matter is formed by the axons
16 of the brain. And the axons are coated with a fatty
17 substance called myelin, and that's why it's called
18 white matter because it has high fat content. And
19 these are the connections.

20 So you have a cell body and then you have a
21 connection that goes to another cell, and that's how
22 the brain communicates. When I teach this, I'll
23 actually hold my hand up like this and I'll, for the
24 class I'll say, this is the nucleus of the cell. So
25 here is the cell body.

1 Q. Pointing to your palm now?

2 A. Pointing to my palm.

3 And so this is where the machinery is that
4 keeps the cell alive and all of the metabolic function
5 of the cell is taking place here. But the input to the
6 cell comes from the dendrites. That's a tree-like
7 structure that surrounds the cell body. But coming out
8 of the cell body, there is just a single filament that
9 projects out, and that's the axon.

10 Coated around the axon is this fatty sheath
11 called myelin, and this is how cells connect one with
12 another.

13 So you and I are talking right now, so we have
14 a part in our brain that's listening and connecting to
15 the speech centers. All of those connections are
16 happening by this part here, the axons, the
17 connections. If you have a white matter signal change
18 or difference, that means that the connection in this
19 part of the brain is not connecting properly with the
20 other parts of the brain that it normally would connect
21 to.

22 Q. And does that mean that the connections are
23 being broken?

24 A. It could, yes.

25 Q. Are there other effects that the white matter

1 change could cause besides breaking the connection all
2 together?

3 A. Well, what can also happen is that the brain
4 will have to reroute around this abnormality. In other
5 words, adapt to this brain abnormality. And instead of
6 sending the signal directly through, you have to send
7 it around.

8 I believe I may have mentioned this in the
9 deposition that I gave, that in Utah we have only one
10 north-south highway or freeway, and that's Interstate
11 15, which, of course, goes through Idaho, as well. If
12 Interstate 15 is blocked, you can still get around, you
13 may still be able to get to your destination. It takes
14 longer. It's not as effective. It's not as efficient.
15 And so you have to reroute.

16 The brain does that, as well. That's how it
17 deals with these kinds of abnormalities.

18 Q. Does that have a particular effect, having to
19 take longer for the signal to arrive or having to go
20 around another route?

21 A. Yes, it can affect how the brain regulates
22 behavior. It may make things less efficient. It may
23 make responses slower. Speed of processing can be
24 affected. Those variables are things that are known to
25 be related to white matter abnormalities that are seen

1 on the brain.

2 Q. Okay. With this kind of basic understanding
3 of what a white matter signal change or a white matter
4 hyperintensity is, let me ask you now, you've pointed
5 out on the 2006 and the 1996 MRIs that there are these
6 white matter abnormalities. Are those in essentially
7 the same places -- seen in the same places on those two
8 scans?

9 A. That's correct.

10 Q. Now, with respect to the 1985 CT scan, and
11 recognizing that we don't actually have those films
12 with us anymore, we just have the report, what can you
13 say, if anything, based on that report about whether
14 abnormalities were present also in 1985 and whether
15 they may have been in the same areas that abnormalities
16 are seen in 1996 and in 2006?

17 A. Well, as I pointed out when I started off this
18 discussion, the radiologist on this scan points to the
19 centrum semiovale. That's a deep white matter area of
20 the brain. And it's just beneath that area where one
21 of the abnormalities is noted in the 2006 and that you
22 can also reference in the 1996. They also raise the
23 question of demyelinating disease. Demyelinating
24 disease is a disorder where the white matter is
25 affected. And then the radiologist also indicated, and

1 I quote, "very slight cerebral cortical atrophy,"
2 meaning that to this radiologist's eye, the brain was
3 somewhat shrunken in 1985 when this was first read.

4 Now, I do want to point out on this that the
5 findings that we've been talking about today are in the
6 right hemisphere, and this radiologist was pointing to
7 the left hemisphere where the centrum semiovale
8 differences were noted.

9 As far as the atrophy, the radiologist was
10 indicating the atrophic appearance to be more in the
11 frontal part of the brain. And as I indicated earlier
12 in the testimony where these white matter abnormalities
13 are that had been pointed out, they are in the frontal
14 region of the brain.

15 Q. Dr. Bigler, can you infer anything from the
16 fact that the abnormalities are referred to in the 1985
17 CT scan, also in the '96 MRI, also in the 2006 MRI?
18 Can you infer anything about the fact that when these
19 abnormalities arose or became present in Mr. Leavitt's
20 brain?

21 A. I don't believe so, because there are no
22 specific markers in any of these abnormalities as to
23 their precise etiology or when they occurred. And, of
24 course, at any point in time when you are doing a scan
25 like this, you're only scanning the patient at a

1 precise moment in time, not over time. And you're only
2 getting a snapshot of the brain at that moment, which
3 is really the sum of that individual's genetic and
4 environmental history up to that point. And there's
5 nothing here that I see that would help us pinpoint
6 when these abnormalities occurred or what their precise
7 etiology may be.

8 Q. Knowing that they are present in '96 and
9 knowing that they are present in 2006 and knowing that
10 there is a reference to abnormality in 1985, does that
11 make it likely that they would have been there also in
12 1989 or 1990?

13 A. Correct.

14 Q. All right. Well, Doctor, I think I'd better
15 ask you to explain, if you would, please, what can
16 potentially cause white matter hyperintensities like
17 this to be present in a person's brain?

18 A. There are a number of potential factors.
19 There can be developmental errors in the way the brain
20 first develops that can be associated with white matter
21 hyperintensities. We see a greater number of them in
22 developmental disorders in the brain, including things
23 like learning disability and disorders like attention
24 deficit hyperactivity disorder.

25 There are other factors that may be associated

1 with this such as head injury. Traumatic brain injury
2 increases the likelihood of having white matter
3 hyperintensities. A variety of medical factors may
4 also relate to this. Hypotension relates to presence
5 of white matter hyperintensities. Obesity and diabetes
6 relate to hyperintense abnormalities in the white
7 matter of the brain. They do increase with the aging
8 and they are also associated with a broad spectrum of
9 degenerative disorders of the brain. However, in cases
10 of degenerative disorders you see a great accumulation
11 of these as the person ages. And we don't really see a
12 significant difference between the '96 imaging and the
13 2006 imaging in this patient. So I don't think this
14 represents degenerative disease.

15 Q. Doctor, I will represent to you that we have
16 put Mr. Leavitt's birth certificate into evidence and
17 that he was born in 1958. Therefore, the 2006 scan was
18 performed when Mr. Leavitt was 48 years of age, the '96
19 when he was 38, and the 1985 CT scan when he was 27.

20 Does age say anything to you about the cause
21 of the development of white matter change?

22 A. Yes. I indicated earlier that increasing age
23 is associated with developing white matter changes in
24 the brain. But we, as well as others, have published
25 on this very topic, and you typically don't see

1 increase in white matter changes until after about age
2 50.

3 And so showing white matter changes in the 30s
4 is uncommon. I'll put it that way. It does happen in
5 normal individuals that are screened for neurologic and
6 psychiatric disorder. But the norm is for there not to
7 be white matter signal differences in the MRI in your
8 30s.

9 Q. But is it correct that after about age 50, we
10 start to see white matter hyperintensities showing up
11 with a higher frequency in the brains of the normal
12 population?

13 A. That is correct.

14 Q. First, just to return to this question, and I
15 think you spoke to this previously to a certain degree,
16 but I want to ask you it so that I'm clear, can you
17 tell by looking at these white matter hyperintensities
18 on these MRIs, can you say conclusively to a reasonable
19 medical certainty what caused them?

20 A. Well, no, you cannot -- from my standpoint,
21 you cannot say what caused them. I'm not a
22 radiologist, so I want to make sure that I'm not giving
23 medical testimony here. I'm doing this from the
24 standpoint of a neuroscientist and a neuropsychologist
25 with an imaging background.

1 Q. I appreciate that.

2 Sir, could trauma associated with a premature
3 birth cause white matter hyperintensities to form in a
4 person's brain?

5 A. Yes, that, in fact, is a well-established
6 relationship. And that's what I was referring to
7 earlier in terms of developmental disorders, one of the
8 causes, one of the factors.

9 Q. Can closed-head injury cause white matter
10 hyperintensities to form?

11 A. Yes. Closed-head injury is analogous to
12 traumatic brain injury. I believe I indicated that
13 traumatic brain injury is one of the factors associated
14 with increased the likelihood of white matter changes
15 in the brain.

16 Q. Now, do you see in the scans that are present,
17 in the white matter hyperintensities that are present
18 in Mr. Leavitt's scans, do you see anything or the
19 absence of anything that bears on whether they may have
20 been caused by trauma?

21 A. Yes. There is a clear and distinct finding
22 that is definitive when present for trauma, and that's
23 what's referred to as hemiacidrin. That's a blood
24 by-product that is left in the brain that's a marker
25 for sheering. And sheering is the tearing or twisting

1 and pulling apart of brain tissue. And it not only
2 pulls apart brain tissue, but it pulls apart the blood
3 vessels. And so the blood vessels leak blood into the
4 brain. And that's a very distinct and different signal
5 on the MRI, and that's not present here.

6 So you can still get white matter
7 hyperintensities that are from trauma. But when you
8 don't have hemiacidrin deposits in the brain that are
9 associated with the white matter signal difference,
10 then that means that you haven't proven that it's head
11 injury.

12 Q. Okay. So to say that another way, this
13 could -- these white matter hyperintensities could be
14 from trauma. It's just that you can't conclusively say
15 that?

16 A. That's correct. As I said, I don't think you
17 can, in this individual, say why these things are here.
18 You can only raise historical medical facts that have
19 increased likelihood of being associated with the white
20 matter abnormalities.

21 Q. Is it possible for the inhalation of paint
22 fumes to give rise to white matter hyperintensities?

23 A. Yes.

24 Q. And do you see -- among children who suffer
25 from hyperactivity, do you see a higher incidence of

1 white matter hyperintensities in their brains upon
2 scanning?

3 A. That has been reported, along with learning
4 disabilities, correct.

5 Q. Well, Doctor, we've talked about what may
6 cause white matter hyperintensities. You've pointed
7 out that what -- physically what the white matter
8 hyperintensities do is alter the flow of electrical
9 impulses within the brain through the axons and require
10 rerouting and so on and disrupt the signal.

11 Now I'd just like to ask you to explain what
12 the effect in terms of behavior is of white matter
13 hyperintensities being present in the brain.

14 A. Well, this is an area of very intense
15 investigation. Many researchers are working on this
16 very issue because we see increased likelihood of white
17 matter hyperintensities in most of the major
18 neuropsychiatric disorders, including depression,
19 anxiety where cognitive disorders are present.

20 We have already talked about learning
21 disabilities, attention deficit hyperactivity disorder,
22 other disorders of impulse control. These studies have
23 shown that something amiss in the white matter creates
24 a greater likelihood that an individual may have
25 neuropsychiatric symptoms or problems.

1 Q. Would that include personality features that
2 included explosive episodes or a tendency to explode
3 and then recede into a calm state?

4 A. Yes. These studies have looked at problems
5 with impulse control, regulation within the frontal
6 lobe. The frontal lobe is the major part of the brain
7 that's involved in regulating our emotions.

8 So, yes, those studies have been done. And
9 there is documentation that white matter changes may be
10 associated with these type of psychiatric features.

11 Q. Sir, do you make a distinction between
12 cognitive psychological problems and emotional
13 psychological problems?

14 A. Yes, I do.

15 Q. Could you explain them, please?

16 A. Well, some of the disorders that may affect an
17 individual may primarily affect memory, language, or
18 what we call executive function, complex learning,
19 problem solving, but not necessarily impact their basic
20 emotional state.

21 And quite the opposite may also occur where
22 the individual may have very intact cognitive ability,
23 normal intellectual and executive functioning, but yet
24 be very dysfunctional with regards to their emotions
25 and how emotions are controlled.

1 To a certain extent, although there is also a
2 great amount of overlap, but to a certain extent the
3 emotional circuitry in the brain is a bit different
4 than the cognitive circuitry. Now, those two have to
5 communicate and work one with the other, but the
6 cognitive aspect of mental function is somewhat
7 different than the emotional aspect of mental function.

8 Q. Doctor, is -- does everyone who has white
9 matter hyperintensities in their brain, let's say,
10 every 27-year-old or every 37-year-old or every
11 47-year-old, does everyone who has white matter
12 hyperintensities in their brain also have
13 neuropsychological disorders?

14 A. No.

15 Q. Let's be clear about what you're saying in
16 this respect. Is there a direct causal relationship
17 between white matter hyperintensities and behavior?

18 A. We believe that there is a relationship. How
19 directly causal that is is, of course, what we continue
20 to research and try to better understand.

21 Q. But what you said previously was that there is
22 a higher frequency or likelihood of white matter
23 hyperintensities being present among persons with
24 neuropsychiatric disorders; is that correct?

25 A. That is correct.

1 Q. Well -- sorry.

2 A. No. I didn't say anything.

3 Q. Okay. I'm sorry. I thought I had interrupted
4 you.

5 You spoke, when we took your deposition, of a
6 two-way experiment. And the first part of this was
7 that if you take two groups of people who are as close
8 demographically as possible, are the same age, are the
9 same sex, the same race, but one group has a
10 neuropsychological or neuropsychiatric disorder and the
11 other group doesn't, you see a difference between those
12 two groups in terms of the number of white matter
13 hyperintensities; is that correct?

14 A. That's correct.

15 Q. And could you explain what that difference is?

16 A. Well, there's a higher frequency of white
17 matter abnormalities on the MRI in those who are in the
18 psychiatric group. And it doesn't mean that we don't
19 see white matter abnormalities in the control group.
20 We often do. In fact, one of the studies we published
21 in the appearance of white matter hyperintensities in
22 the normal population still found a few hyperintense
23 signal abnormalities in the control subjects who were
24 screened not to have psychiatric disorder, not to have
25 active neurologic disorder or a history of neurologic

1 disorder.

2 So it does occur in the normal population and
3 in the control sample. But as soon as you move into
4 any one of these clinical populations, you see a higher
5 frequency of white matter abnormalities. And what that
6 tells us is that something amiss with the white matter
7 must relate in some fashion to having an increased
8 likelihood for psychiatric disorder.

9 And we also believe that where those white
10 matter hyperintensities are probably leads to different
11 psychiatric manifestations of the disorder.

12 Q. Doctor, the other side of your two-way
13 experiment was that if you encountered someone who has
14 white matter signal changes but you don't know which
15 group they fit in, either the control group or the
16 group that's known to be suffering from a disorder, so
17 if you're dealing with this person who simply has an
18 increase in white matter hyperintensities, you then can
19 make a prediction about which group they fit into; is
20 that correct?

21 A. That's correct.

22 Q. And what is your prediction then?

23 A. You would predict they would be in one of the
24 psychiatric disorders.

25 Q. All right. Is the location of white matter

1 hyperintensities important in predicting the likelihood
2 that they will have an affect on behavior?

3 A. Correct.

4 Q. And could you explain that, please?

5 A. Well, abnormalities that show up in the
6 frontal and temporal lobes of a patient's brain
7 typically have different characteristics than what are
8 seen when the white matter changes are in the parietal
9 or occipital areas of the brain. And the frontal part
10 of the brain is involved in emotional regulation and
11 executive function.

12 Typically, when abnormalities are in the lower
13 part of the frontal lobe, those may be more associated
14 with emotional regulation problems, impulse control
15 problems. When the abnormalities are higher in the
16 frontal lobe, those tend to be associated more with
17 cognitive problems.

18 When the frequency is looked at, then the
19 overall number of white matter hyperintensities also
20 seems to have a bearing on both of those, that the
21 greater the number of white matter hyperintensities,
22 the greater the likelihood for emotional dysfunction
23 and cognitive dysfunction.

24 Q. Now, I believe you said previously that
25 Mr. Leavitt does have white matter hyperintensities in

1 the inferior frontal region of his brain?

2 A. That is correct.

3 Q. Doctor, if Mr. Leavitt had been subjected to
4 an MRI in 1989 or 1990, could these abnormalities of
5 the sort you're describing here have been detected?

6 A. Yes.

7 Q. And the work that you have been doing, your
8 research began long before 1990, and, of course, has
9 continued long after it as well; is that correct?

10 A. That's correct.

11 MR. NEVIN: Could I have just a moment, Your
12 Honor?

13 THE COURT: Yes, certainly.

14 (Pause in the proceedings.)

15 MR. NEVIN: Dr. Bigler, thank you. I don't
16 have further questions.

17 THE COURT: You may inquire of the witness.

18 MR. ROSENTHAL: Thank you.

19 CROSS-EXAMINATION

20 QUESTIONS BY MR. ROSENTHAL:

21 Q. Good morning, Dr. Bigler. We have met before,
22 have we not?

23 A. Yes, we did. Good morning.

24 Q. You commented, in response to Mr. Nevin's
25 question, that in '89 or '90 those white matter

1 hyperintensities could have been found if an MRI was
2 done; did you not?

3 A. I did.

4 Q. In fact, in the 1996 MRI that was performed, I
5 believe at the request of Mr. Nevin, those white matter
6 hyperintensities were not found until literally 2006,
7 after the 2006 MRI was done?

8 A. In retrospect, going back, you can see them in
9 the 1996 scan. They were not reported in 1996. They
10 were there, but they were not reported.

11 Q. Not recorded -- or reported by the radiologist
12 or the neurologist; is that correct?

13 A. That's correct. I'm not sure -- something
14 just happened again. The images went back on the
15 screen rather than you. Oh, there we go.

16 Q. Thank you.

17 Also, you've indicated that the number of
18 white matter hyperintensities in one's brain has an
19 impact. In fact, Mr. Leavitt has three white matter
20 hyperintensities; does he not?

21 A. Based on the most recent imaging, those are
22 the three that are readily identifiable.

23 Q. And they are very small; are they not?

24 A. They are small. That's why they're called
25 what they are called, foci. I think the term was used

1 in the radiologist report. That means a focal area of
2 abnormality, a small area of abnormality.

3 Q. And the smallness of them has an impact on
4 their potential seriousness in terms of neurological
5 functioning; do they not?

6 A. Yes. The size of a lesion has a relationship
7 to their impact on how the brain functions.

8 Q. And you used the word "lesion." In fact,
9 these are not lesions. They are signal abnormalities;
10 are they not?

11 A. Well, those two terms are often used
12 interchangeably and they are considered an abnormality.
13 And when postmortem studies are done, there is
14 typically something that has degenerated or a change in
15 that particular region of the brain that would count as
16 a lesion or an abnormality.

17 Q. And so, basically, what you're saying is the
18 signal is not uniform across that hyperintensity or
19 abnormality?

20 A. That's correct.

21 Q. And you've already stated, have you not, that
22 the brain allows for rerouting of signals around those
23 abnormalities; do they not?

24 A. Yes, I have stated that.

25 Q. And if these white matter hyperintensities are

1 caused by a head injury, they never go away; do they?

2 A. No.

3 Q. You see white matter hyperintensities with
4 people who have cardiovascular problems?

5 A. That is correct.

6 Q. As well as diabetic problems?

7 A. Correct.

8 Q. And they do not necessarily indicate psychosis
9 of any kind; do they?

10 A. That is correct.

11 Q. And in a normal healthy population, haven't
12 your studies and those of your colleagues indicated
13 that between five and ten percent of a normal healthy
14 population have white matter hyperintensities?

15 A. Well, I think in my deposition I was asked a
16 similar question. It's a little more complex to answer
17 than yes or no with that because it depends on the age
18 and what you're screening for or not screening for.

19 If you have a very healthy population and
20 you're sampling individuals under age 50, it's probably
21 down in the range of two to three percent of
22 individuals that would be considered controls or in the
23 normal population that would have white matter changes.
24 If you broaden the medical category and the age range,
25 then, as you stated the question, I would agree with.

1 Q. And I believe you've already stated, you
2 cannot tell why Mr. Leavitt has those three white
3 matter hyperintensities; can you?

4 A. No.

5 Q. Can you tell whether they were all caused by
6 the same problem?

7 A. No.

8 Q. And just to make sure I've asked this in the
9 correct way, those hyperintensities, those different
10 signal characteristics are not necessarily indicative
11 of an abnormality?

12 A. Short of a postmortem analysis of the actual
13 tissue, that is correct.

14 Q. Use of drugs can cause white matter
15 hyperintensities?

16 A. They can.

17 Q. Alcohol?

18 A. Very prolonged alcohol use and abuse can be
19 associated with white matter hyperintensities.

20 Q. And smoking can also cause white matter
21 hyperintensities; can it not, Dr. Bigler?

22 A. It can. That's associated with the
23 cardiovascular ischemia problem with chronic smoking.

24 Q. You mentioned cerebral atrophy. That, like
25 white matter hyperintensities, is just a generic

1 statement of the appearance of the brain without
2 specifying how either the atrophy or hyperintensities
3 were caused or what caused them?

4 A. That's correct.

5 Q. Haven't your studies found that some white
6 matter hyperintensities represent large benign
7 perivascular spaces and don't influence any
8 neurological function?

9 A. That's similar to your earlier question.
10 That's why I was responding with the statement of the
11 postmortem. Unless you actually have postmortem of the
12 tissue, it may be what's called a perivascular space,
13 which is just where the blood vessels are penetrating
14 the white matter.

15 Q. And many writings and studies before 2002 and
16 2003 found that there was a failure to demonstrate a
17 systemic relationship of cognitive function and white
18 matter hyperintensities; isn't that correct?

19 A. That is correct. I believe that's a
20 technology problem reflective of the earlier studies
21 rather than what we're finding now.

22 Q. Now, the white matter hyperintensities in
23 Mr. Leavitt's temporal lobe are in the non-dominant
24 hemisphere; are they not?

25 A. That's correct.

1 Q. And what does that hemisphere of the brain
2 deal with, sir?

3 A. The right hemisphere tends to deal more with
4 non-verbal visual spatial, non-language functions of
5 the brain.

6 Q. And you've already testified that
7 hyperintensities in the frontal and temporal lobes are
8 more likely to be associated with memory and executive
9 dysfunction. Is that accurate?

10 A. I believe I made a distinction between and
11 separated out cognitive versus emotional, that you can
12 have just the changes in emotional functioning without
13 affecting cognition, and you can have the white matter
14 changes affecting cognition without affecting emotion,
15 depending on where the lesions are, where the
16 abnormalities are and what they are doing in affecting
17 the brain and its functioning.

18 Q. And, again, the lack of hemiacidrin staining
19 is indicative of no head injury based on your study of
20 the MRI scans; is that correct?

21 A. I think I answered that earlier, that it
22 doesn't rule it out. But without the hemiacidrin, then
23 you can't say that this was sheer injury to the brain
24 or some other vascular reason that would result in a
25 blood vessel hemorrhaging and leaving that deposit.

1 There is not hemiacidrin staining, as pointed out by
2 the radiologist, and you can't see hemiacidrin in any
3 of these scans that I have looked at.

4 Q. And based on the three white matter
5 hyperintensities that you observed in the scan, would
6 you expect Mr. Leavitt to have difficulties with
7 executive functioning?

8 A. It certainly could be the case. One of the
9 lesions is in the superior, more superior frontal area
10 where that association has been made.

11 Q. What about memory, sir?

12 A. I would say the same thing.

13 Q. And as well as emotional control and
14 regulation?

15 A. Well, two of the lesions are more in the
16 inferior frontal area, close to and in the regions of
17 the emotional circuitry of the brain. And so I
18 think --

19 Q. Go ahead, please.

20 A. I was just going to say, and so I think, all
21 things equal, the emotional aspect would probably be
22 more likely than the cognitive.

23 Q. And if those scans indicate a problem with
24 emotional functioning, you would expect that to be
25 something that displayed itself in his normal

1 day-to-day activity; would you not?

2 A. Let me just clarify one thing. The scans
3 don't show anything about emotion. So this is left up
4 to the clinicians that have actually examined the
5 patient and seen the patient or the individual. I have
6 not done that. I have not examined this individual.

7 So all that I'm referring to is the location
8 of these abnormalities, where they are in the brain and
9 how they may relate to neuropsychological,
10 neuropsychiatric features of this individual.

11 Q. And one of those neuropsychiatric features,
12 Dr. Bigler, would be emotional function, would it not,
13 sir?

14 A. That's correct.

15 Q. And if he had a difficult -- had difficulties
16 with emotional function, would you expect that to be
17 displayed in his daily, weekly, and monthly conduct?

18 A. Yes.

19 Q. You are aware, are you not, that Mr. Leavitt
20 has heart disease?

21 A. Yes.

22 Q. And that's one of the causes of white matter
23 hyperintensities?

24 A. Correct.

25 Q. You're also aware that he suffers from

1 diabetes; are you not?

2 A. Yes.

3 Q. And that is also one of the causes of white
4 matter hyperintensities?

5 A. Correct.

6 Q. And I think you've already testified that
7 white matter hyperintensities can be caused by dilated
8 perivascular spaces or show up in that manner?

9 A. Correct.

10 Q. And that is shown by Mr. Leavitt's MRI?

11 A. He has white matter signal changes. You can't
12 take it any further. There isn't a way to say that
13 this is a dilated perivascular space or it's actual
14 degradation of the myelin in that area, whether it's an
15 old sheer lesion, whether it's a little stroke that
16 could have been caused --

17 MR. ROSENTHAL: What happened? Doctor, can
18 you hear me?

19 THE CLERK: I don't think we have a
20 connection.

21 THE COURT: We are obviously not getting a
22 signal from them.

23 MR. NEVIN: Looks like the signal failed from
24 down there, but I bet -- I'll bet if we get Mr. Hansen,
25 he could...

1 THE COURT: Ms. Garcia will send an instant
2 message, see if we can get him up here.

3 (Pause in the proceedings.)

4 THE COURT: Counsel, let's take a very short
5 break. I would like to wrap this up before lunch.
6 Let's take a short recess.

7 We will be in recess until further call.

8 (Whereupon, the Court recessed.)

9 THE COURT: Doctor, I will remind you, you are
10 still under oath.

11 Mr. Rosenthal, you may resume your
12 examination.

13 MR. ROSENTHAL: Thank you, Your Honor.

14 BY MR. ROSENTHAL:

15 Q. Dr. Bigler, you had commented earlier about
16 the 1985 CT scan that indicated problems in the left
17 hemisphere; is that correct?

18 A. Yes.

19 Q. But in the '96 and 2006 scans, it indicates
20 that the difficulties are in the right hemisphere; is
21 that correct?

22 A. That's correct.

23 Q. Can you opine on whether the '85 is talking
24 about something different or that the '85 scan was read
25 incorrectly?

1 A. Well, I don't think anyone can comment about
2 it being read incorrectly because it's not available to
3 review. I believe what I was trying to make a point on
4 is that it was talking about white matter. That's what
5 the centrum semiovale is. It was talking about white
6 matter in this individual and that they were seeing
7 differences in the white matter on the CT.

8 Now, computerized tomography is a different
9 technology and it doesn't have the capability that
10 magnetic resonance imaging has in looking at the
11 details of white matter differences.

12 So the only point that I was trying to make is
13 that, in 2006, we're talking about white matter. In
14 1985, the radiologist was also talking about white
15 matter. It just so happens that what was being viewed
16 then was in the left hemisphere rather than the right
17 hemisphere.

18 Q. And don't your studies, Doctor, show that
19 white matter hyperintensities in the centrum semiovale
20 are generally associated with cognitive impairments?

21 A. Yes, we have demonstrated that. But in an
22 aging and older population, not a younger population.

23 Q. But if one has a white matter hyperintensity
24 in the centrum semiovale, you would expect some
25 cognitive impairments; would you not?

1 A. Well, again, it goes back to this relationship
2 between how large are the abnormalities, how many of
3 them are there, and their location in the centrum
4 semiovale. That region of the brain goes from the
5 front of the brain to the back of the brain. And as
6 those are more associated with abnormalities towards
7 the front, that's when they have more of the cognitive
8 effect.

9 Q. And in Mr. Leavitt's case, they are very small
10 white matter hyperintensities?

11 A. The white matter hyperintensities that he has
12 are small, that is correct.

13 Q. All right. Is there any significance to the
14 fact that Mr. Leavitt's 1985 EEG was normal, according
15 to the physician that rendered it?

16 A. Well, EEG is a very different technology.
17 It's a physiologic measure of brain activity based on
18 electrodes placed on the scalp. I believe one of the
19 reasons for doing the EEG was to rule out something
20 along the lines of a seizure disorder, something in the
21 spectrum of epilepsy. And that's what the EEG can do.

22 Q. But nevertheless, an EEG in 1985 would have
23 picked up difficulties in this signal connection; would
24 they not, Doctor?

25 A. No. You can have a perfectly normal EEG and

1 have a very abnormal brain scan with regards to white
2 matter. And what the standard EEG is doing is it's
3 only assessing resting brain activity, not activity of
4 the brain actually engaged in some cognitive task.

5 Q. Doctor, in the packet of literature and
6 reports that you received from Mr. Nevin and
7 Mr. Parnes, did you receive the reports of Dr. Groberg?

8 A. I don't believe I have a report on
9 Dr. Groberg.

10 Q. What about Dr. Gordon, sir?

11 A. I don't believe I have that.

12 Q. Thank you.

13 Doctor, Mr. Nevin asked you about being
14 exposed, an individual being exposed to paint fumes and
15 whether that could cause white matter hyperintensities.
16 You recall that; do you not?

17 A. Yes, I do.

18 Q. And being exposed to paint fumes is another
19 way of saying hypoxia; is it not? The lack of oxygen
20 to the brain?

21 A. Well, it depends on how it's occurring. If
22 it's occurring as part of abuse, like spray paint
23 inhalation, there can also be toxic elements of what is
24 actually inhaled in the paint that may also affect the
25 brain. But if one is overcome by fumes, then that may

1 be the basis for hypoxia. And hypoxia is associated
2 with white matter hyperintensities.

3 Q. And hypoxia shows up on the MRI scan very much
4 like a stroke does; does it not?

5 A. Correct.

6 Q. And you saw nothing to indicate that type of
7 defect in the MRI of Mr. Leavitt?

8 A. Well, it goes back to what we talked about
9 earlier, that there isn't anything in the imaging that
10 specifies etiology, meaning the reason why this is
11 here. There's another characteristic feature that is
12 associated with moderate to severe hypoxia, and that's
13 what's called bilateral basoganglia and bilateral
14 hippocampal lesions. That is not evident in the
15 imaging here.

16 When you see bilateral basoganglia and
17 bilateral hippocampal lesions in someone with a history
18 of hypoxia, that's almost, by definition, proving that
19 those abnormalities were caused by hypoxia.

20 Q. And, in fact, you did a study of something
21 very similar concerning carbon monoxide poisoning; did
22 you not?

23 A. That's correct.

24 Q. And in that study, approximately 30 percent
25 had cognitive impairments?

1 A. Correct.

2 Q. Which meant problems with executive
3 functioning, slow mental processing, impaired
4 visual/spatial ability?

5 A. Yes. Visual/spatial.

6 Q. Spatial. I'm sorry.

7 A. And that's correct. That's correct.

8 MR. ROSENTHAL: One moment, please.

9 Thank you, Dr. Bigler.

10 THE WITNESS: Thank you.

11 THE COURT: Mr. Nevin.

12 REDIRECT EXAMINATION

13 BY MR. NEVIN:

14 Q. Doctor, on that last point, I want to make
15 sure your testimony is clear. Have you changed -- is
16 there any change in what I understood you to say
17 previously, that we cannot say, simply based on looking
18 at these white matter hyperintensities, what caused
19 them?

20 A. That is correct. We cannot tell what caused
21 them.

22 Q. And you've spoken a couple of times about the
23 issue of hemiacidrin staining, and you have said that
24 there is not hemiacidrin staining present in these
25 scans; is that correct?

1 A. That can be visualized with the resolution
2 that we have. I need to -- I didn't say earlier, but
3 there can be hemiacidrin below the threshold of
4 detection by MRI that is seen at postmortem.

5 What this is just telling us is that with the
6 resolution of the MRI as it is today, we cannot see
7 hemiacidrin in these images.

8 Q. Right. And that's why I understood you
9 previously to say that the absence of hemiacidrin
10 staining does not mean that these white matter
11 hyperintensities were not caused by trauma --

12 A. Correct.

13 Q. -- but rather, if the hemiacidrin staining
14 were present, that would allow you to conclusively say
15 that they were?

16 A. Correct.

17 Q. It remains possible that they were caused by
18 trauma?

19 A. Correct.

20 Q. Now, based on looking --

21 A. Just as -- well, I was just going to say the
22 radiologist says in his report, "old trauma." So
23 that -- that's one of the factors that could cause this
24 type of abnormality on an MRI.

25 Q. The radiologist says in his report that an old

1 trauma cannot be ruled out; correct?

2 A. Correct.

3 Q. Okay. Now, the reference has been made
4 several times to these white matter hyperintensities
5 being small, and being very small, and so on.

6 First, just exactly how big are they?

7 A. Well, they're outlined in the radiologist's
8 report. In terms of their millimeter, 3 millimeters, 4.
9 to 5 millimeters, 2 to 3 millimeters. And the
10 smallness of them needs to be looked at in the context
11 of how complex the brain is. And in one cubic
12 millimeter, which is just, you know, enough to see
13 light between your two fingers, in one cubic millimeter
14 you can have millions of pathways. And in a cubic
15 millimeter of cortical tissue, you have tens of
16 billions of synapses.

17 So small lesions, even though they appear
18 small, they can be very significant. That's why we are
19 paying attention to these problems in neuropsychiatric
20 disorders and not overlooking, but, in quotes, the
21 smallness of them.

22 Q. Thank you, Doctor.

23 You were asked about whether a person who had
24 a neurologic disorder, one that was caused by or
25 related to white matter hyperintensities would behave

1 in a particular way all the time, questions to that
2 effect. Do you remember those?

3 A. Yes.

4 Q. I just wanted to ask you, would you expect to
5 see a person with a neurologic disorder functioning
6 differently if they were in a highly structured
7 environment?

8 A. That is typically the environment that someone
9 with a neurologic problem will function the best in,
10 where things are highly structured, highly predictable,
11 and behavior can flow in that type of structured
12 environment that may appear entirely normal. It's
13 typically in other environments where the problem
14 behaviors may be more greatly expressed.

15 Q. Doctor, you've been asked about several things
16 that can cause white matter hyperintensities, heart
17 disease, diabetes, these things of things.

18 In your research, does it matter -- in terms
19 of the effect that white matter hyperintensities have,
20 does it matter what causes them?

21 A. Well, to a certain extent it does because we
22 also looking at using that information. In other
23 words, you wouldn't want to ignore whether this is
24 caused more by hypertension versus head injury versus
25 aging, in how it impacts the psychiatric status of the

1 patient.

2 On the other hand, at this point in time we
3 honestly don't know the cause of white matter
4 hyperintensities in many of these subjects.

5 And so when we see it in someone who has a
6 history of depression and no other risk factor, that
7 becomes significant from a clinical standpoint, from a
8 management standpoint. It tells us more that there is
9 a biological marker in that individual's brain that
10 something probably isn't right, either with its
11 chemistry or its structure, that's predisposing that
12 individual for the psychiatric symptoms that they're
13 experiencing.

14 And that's how we view it right now.

15 Q. In terms of the effect that it's having, if
16 it's caused by diabetes, if it's caused by trauma, that
17 might bear on what you -- what other treatments you
18 provided to the person. But any of the white matter
19 hyperintensities could have an effect on behavior; is
20 that correct?

21 A. That is correct.

22 Q. And you were asked questions about the
23 incidents of white matter hyperintensities in a normal,
24 healthy population, and you made the remark that if you
25 were dealing with people under the age of 50, you would

1 expect to see those occurring at around two to
2 three percent of the normal healthy population.

3 A. Correct.

4 Q. What about in persons who were 27 years of
5 age, that young? Do you know what percentage of the
6 normal healthy population at age 27 would have white
7 matter changes?

8 A. That particular study has not been done. We
9 have done a sub-sample in that particular age range. I
10 don't have that publication in front of me right at
11 this point in time, but there were few if -- and
12 including none in some of our age categories under 50
13 who had white matter hyperintensities.

14 Q. Doctor, you were asked about the possibility
15 that this was a dilated perivascular space as opposed
16 to an abnormality. Is that what's also known as a
17 Virchow-Robin space?

18 A. That's correct.

19 Q. Is there any way to predict the likelihood
20 that these are or are not Virchow-Robin spaces?

21 A. Well, the radiologist points this out in his
22 report. The radiologist at the end of his report
23 actually says: "There are scattered" -- and I'm
24 reading this -- "2 to 3-millimeter hyperintensities in
25 the Corona radiata of the frontal and parietal lobes

1 which are probably just perivascular space" --
2 "prominent perivascular spaces."

3 So the radiologist who's looking at this is
4 saying, hey, there are signal differences that are in
5 this brain. They are in the frontal and parietal area.
6 The Corona radiata is the radiating crown, that's Latin
7 for radiating crown, of the white matter with the brain
8 and there are scattered findings in that region.

9 But I think those are perivascular spaces.
10 These others are not perivascular spaces. I believe
11 that's what the radiologist is communicating in this
12 report.

13 And so the hyperintense signals that we
14 pointed out earlier in testimony today, those are, in
15 my opinion, not perivascular spaces. Those are white
16 matter abnormalities in the white matter tissue of this
17 individual's brain.

18 MR. NEVIN: Doctor, thank you very much.

19 No further questions, Your Honor.

20 THE COURT: Mr. Rosenthal, anything else?

21 MR. ROSENTHAL: Nothing on that, Your Honor.

22 THE COURT: All right. Thank you very much,
23 Doctor, for participating by video conference. We'll
24 terminate the connection at this time.

25 Counsel, we will be in recess -- let's try to

1 reconvene at 1:30. I assume we are on task, on target?

2 MR. NEVIN: Yes, sir, I think we are.

3 MR. ANDERSON: I believe we are, Your Honor,
4 yes.

5 THE COURT: All right. Then we will be in
6 recess until 1:30 this afternoon.

7 (Whereupon, the Court recessed.)

8 THE COURT: Counsel, before we start, I
9 understood that counsel had thought it was perhaps a
10 good idea or it might be helpful to have a couple of
11 lay witnesses from the Blackfoot area testify in
12 Pocatello on Tuesday while I'm there. I have no
13 problem with that. I guess Ms. Heinz, we need to make
14 sure she is available. I assume she can travel to
15 Blackfoot on that day as well. So that's no problem.
16 If that's your preference, we can certainly proceed in
17 that fashion.

18 MR. PARNES: I would appreciate it, Your
19 Honor. The witnesses have been here and we're not
20 going to get to them today, given the schedule. I'd
21 rather have them released and not have to have them
22 come back.

23 THE COURT: Okay. I assume we will have
24 counsel present here or in --

25 MR. ROSENTHAL: Both, Your Honor.

1 MR. PARNES: Both. What we were thinking was
2 that the only other witness is Dr. Andersen on Tuesday
3 morning, and Mr. Nevin and Mr. Rosenthal would do them
4 here, and Mr. Anderson and I would do...

5 THE COURT: Okay.

6 MR. PARNES: We thought that might be easier.

7 THE COURT: All right. I would just make sure
8 you think through the logistics of that. I guess,
9 actually, after we get through with Dr. -- is it
10 Anderson?

11 MR. NEVIN: Yes, on Tuesday.

12 THE COURT: On Tuesday, we'll just shift
13 everything pretty much to Pocatello because we will
14 have two attorneys there and the witnesses there;
15 correct? We won't do any video conference hookup?

16 MR. ANDERSON: Correct.

17 MR. ROSENTHAL: Right.

18 THE COURT: Okay.

19 MR. PARNES: We could do that and waive.

20 THE COURT: Well, we could, but it would be
21 more just so you could observe rather than actually
22 participate in the --

23 MR. PARNES: Right.

24 MR. NEVIN: I was thinking as far as, I don't
25 know if Madam Court Reporter wants to travel or not,

1 but if we were -- if she were recording the proceedings
2 here, if it stayed on the video conference, presumably
3 she could just record --

4 THE COURT: What may seem to be easier from a
5 layman's point of view, it may not be true if you're a
6 court reporter, because it's a tough job. So it might
7 be better if she's there.

8 MR. PARNES: Thank you.

9 THE COURT: Okay. We're going to take up the
10 defendant's -- or the respondent's expert; correct?

11 MR. ROSENTHAL: One of them. Yes, Your Honor.

12 THE COURT: Okay. Go ahead and call your next
13 witness, Mr. Rosenthal.

14 MR. ROSENTHAL: Dr. Robert Engle.

15 THE COURT: Sir, would you step before the
16 clerk and be sworn.

17 ROBERT ENGLE,
18 called on behalf of the Respondent, having been first
19 duly sworn upon oath, was examined and testified as
20 follows:

21 THE CLERK: Thank you. Please have a seat in
22 the witness stand, and please state your name and spell
23 your last name for the record.

24 THE WITNESS: Robert K. Engle, E-n-g-l-e.

25 THE COURT: You may inquire of the witness,