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November 22, 2006

David Z. Nevin
303 West Bannock
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Re: Richard Leavitt

Dear Mr. Nevin:

You have asked me to provide some additional commentary to my prior e-mail response reviewing the imaging studies in terms of neuropsychological issues in this case. You had previously sent a copy of the deposition of Bruce Anderson, M.D., and all of the associated documents that were in his file. Additionally, I have recently received the neuropsychological examination by Craig W. Beaver, Ph.D., dated April 10, 2006, and then Robert Engle, Ph.D., September 11, 2006, Daniel A. Martell, Ph.D., September 29, 2006, and James R. Missett, M.D., Ph.D. One of the issues you have asked me to address goes back to the CT findings that were obtained on this individual back in 1985. It is my understanding that those films are no longer available for review, we only have the report, and the report is difficult to read, but it does apparently indicate "very slight cortical cerebral atrophy," and also a question of abnormality in the white matter "suggesting the possibility of demyelinating disease." There is an issue of head injury. This is actually elaborated on in the various reports by Drs. Beaver, Martell, Engle, and Missett. As outlined in Dr. Martell's report, there is a sledding accident when Mr. Leavitt was a young man, and apparently Mr. Leavitt has also stated that he "thinks" he lost consciousness for perhaps 30 seconds in some, but not all of the clinical interviews that he has done in the past to questions asked of various mental health providers. There was also another incident in 1985 where he attempted to take his life with an overdose. As indicated in Dr. Martell's report, this individual "claimed no memory for two-to-three days surrounding the incident." He also claims to have had an altercation with prison guards in 1988 and that he was fading "in and out" of consciousness.

Presence of cerebral atrophy can be associated with a variety of etiologies, including idiopathic, head injury, hypoxia, and substance use/abuse. Without the opportunity to directly examine the 1985 CT scan findings, and in the absence of other neuropsychological studies and tests that could have been done at that point in time, it is very difficult to comment further on the clinical significance of the 1985 scan findings. However, we do know that the patient's recent MRI has

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shown and been interpreted as indicating presence of white matter hyperintensities. There is an earlier scan that was done on February 22, 1996, and white matter hyperintensities were not interpreted in the findings, yet they are evident on some of the sequences that were performed, and at least one of them is in the same position as noted on the FLAIR sequence in the most recent imaging. This has been confirmed by Dr. Alexander Mark, a neuroradiologist, who has reviewed the 1996 imaging. His e-mail response to Mr. Andrew Parnes confirms this observation, that the signal changes seen in the recent scan, were actually present in the 1996 scan, although not reported.

The issue of the degree of cerebral atrophy that may be present in a scan can be addressed with what is referred to as quantitative neuroimaging analysis. Obviously, to do that, one would need the 1985 CT scan. My research, in fact, demonstrated in the early 1980's methods for image quantification to establish the degree of cerebral atrophy. This is also technology that could be applied to the 1996 and the 2006 scans. Presence of cerebral atrophy is something that also relates to neuropsychiatric disorders, particularly if the changes are in the frontal and temporal lobes.

Presence of white matter hyperintensities has significance in understanding clinical symptomology. Presence of white matter hyperintensities are more likely seen in patients that have neurologic and/or neuropsychiatric history than in the normal population. White matter hyperintensities have been associated with a variety of neurodevelopmental disorders as well as acquired disorders such as head injury, hypoxia, and substance use/abuse. When there is concomitant hemorrhaging of the magnitude that leaves a detectable hemosiderin deposit (i.e residual blood by-product), that is often diagnostic of traumatic brain injury when the hyperintensity is concomitant with the hemosiderin deposit and there is positive history of head injury. Hemosiderin is not detected in this case, and, therefore, it could not be concluded unequivocally that these findings are associated with his history of head injury and possible hypoxia. There is also an increased likelihood in patients who have histories of depression and impulse regulation problems to have abnormal scan findings, including white matter signal abnormalities.

The location of abnormalities also has some bearing on the patient's clinical status as well. The more anterior or frontal the abnormalities, the greater the likelihood that there will be some disruption in emotional function, impulse control, temperament as well as executive ability. There are several studies that have related the number and location of white matter hyperintensities to neuropsychiatric conditions.

There are also, other factors that can be associated with presence of white matter abnormalities, including cardiovascular disease and hypertension. It is my understanding that Mr. Leavitt has a history of heart disease and had stent surgery in 2004. Accordingly, it is possible that the white matter hyperintensities are related to his history of heart disease.

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In reviewing these records, I do not see other additional risk factors in this individual for the presence of white matter hyperintensities. These findings are objective findings and do have implications of subtle underlying pathology of the cerebral white matter that could affect an individual's neuropsychological status.

Lastly, you have also asked me to address the timing and availability of technology. CT imaging began to be used in a clinical setting in the early to mid-1970's. Clinical use of MRI technology became widely available in the United States in the mid-1980's, but the early scanners used lower magnetic field strength in the range of .3 to .5 Tesla. By the early 1990's most scanners were being upgraded to 1.0 to 1.5 Tesla and now some centers have 3 Tesla magnets. The software improvements for data processing have greatly changed since MRI was first introduced and all of these factors relate to what kinds of pathologies can be identified and how abnormalities can be viewed. As to a more fine-grained image analysis, MRI is generally superior to CT, even at lower field strength. With higher field strength smaller lesions or abnormalities can be identified.

I hope these points have covered the basic issues with regards to imaging in this individual. As with my previous e-mail, there is considerable elaboration I can offer on all of these different points.

Sincerely,



Erin D. Bigler, Ph.D.

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