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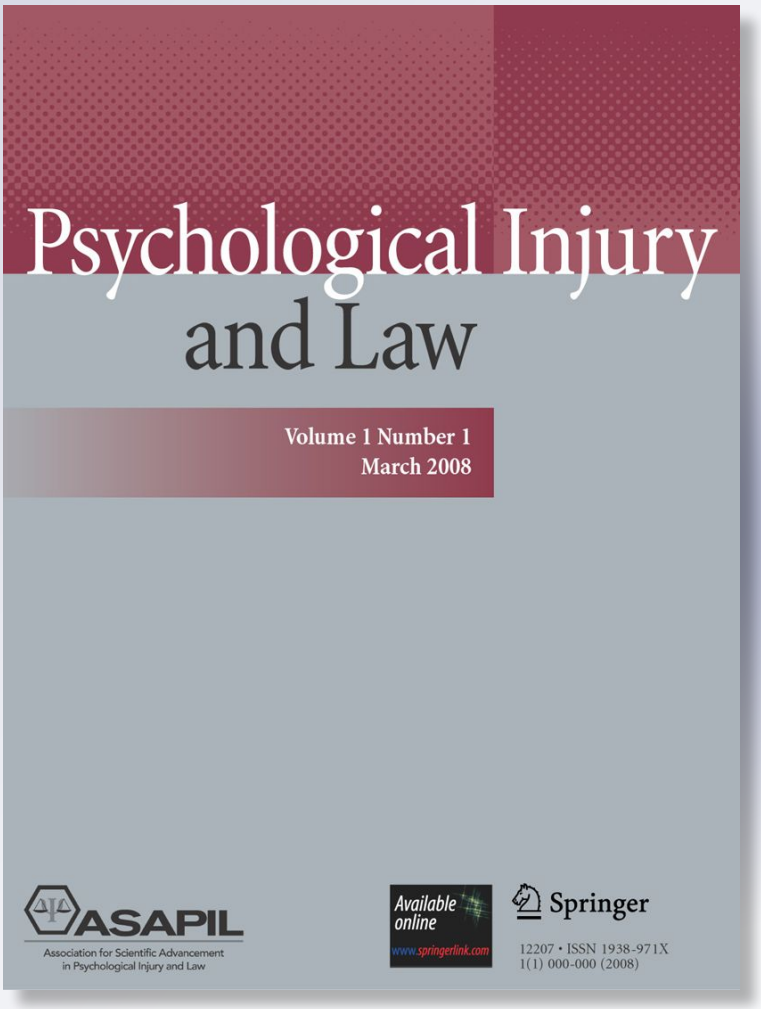
Psychological Injury and Law

ISSN 1938-971X

Psychol. Inj. and Law
DOI 10.1007/s12207-011-9105-4



Springer

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Traumatic Brain Injury Alters Word Memory Test Performance by Slowing Response Time and Increasing Cortical Activation: An fMRI Study of a Symptom Validity Test

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Received: 5 October 2010 / Accepted: 28 July 2011

Abstract The Word Memory Test (WMT) is an established symptom validity test that relies on verbal memory performance to make inferences about "effort." Previous studies, using a functional MRI (fMRI) adaptation of the WMT with healthy controls, have shown that successful completion of the WMT relies on a widespread network of neural systems associated with high cognitive effort. Additional studies using the same fMRI paradigm with patients with severe traumatic brain injury (TBI) suggest that increased activation of cortical regions associated with cognitive load are recruited to meet the cognitive challenges that the WMT places on a compromised neural system. This study builds on previous findings as a result of highly uncommon circumstances in which fMRI data on the WMT task were made available from the very same individual both 1 year before and 1 year after sustaining a TBI. Interestingly, the effect of TBI did not appear to impair performance on the WMT in terms of standard accuracy measurements, though response times were notably slower. The main fMRI finding was a significantly stronger and more widespread pattern of activation post-injury, particularly in the frontal and parietal brain regions, suggesting that stronger engagement of these networks was necessary to sustain accurate WMT performance compared to pre-injury testing. This unique source of data, together with previous findings, suggests a more complex relationship between effort and performance levels on the WMT than what is commonly assumed.

Keywords fMRI · Word Memory Test · Neuropsychological assessment · Symptom validity testing · Effort testing

M. D. Allen (✉) · T. C. Wu · E. D. Bigler
Department of Psychology, Brigham Young University,
1022 SWKT,
Provo, UT 84602, USA
e-mail: m_allen@byu.edu

T. C. Wu
e-mail: trevorwu2002@yahoo.com

E. D. Bigler
e-mail: erin_bigler@byu.edu

M. D. Allen · T. C. Wu · E. D. Bigler
Neuroscience Center, Brigham Young University,
Provo, UT, USA

E. D. Bigler
The Brain Institute of Utah, University of Utah,
Salt Lake City, UT, USA

E. D. Bigler
Department of Psychiatry, University of Utah,
Salt Lake City, UT, USA

M. D. Allen
Functional Imaging Unit, Riverwoods Imaging Center,
Provo, UT, USA

Introduction

Symptom validity tests (SVTs) are commonly administered when evaluating the cognitive effects of traumatic brain injury (TBI) and are intended to assess "effort" on the part of the patient (Millis 2009). One well-established and commonly used SVT is the Word Memory Test (WMT, Green 2003). As a typical SVT, the WMT employs a cognitive task, in this case a series of verbal memory tests, which most individuals can perform without error. The construct that a simple SVT assesses "effort" centers on the fact that passing the SVT shows that a minimal level of effort has been exerted to pass a task that is commonly

Published online: 19 August 2011

passed by most individuals, even those with neurological impairment. If an examinee cannot pass even an easy SVT measure, it is assumed that minimal effort is not being displayed, not only on the SVT at hand, but for other neuropsychological measures as well. Specific to the WMT, Flaro et al. (2007) have stated "the effort components of the WMT were designed to avoid confusing actual impairment with deliberate exaggeration… "where WMT performance is "….virtually insensitive to all but the most extreme forms of impairment of learning and memory and the range of genuine scores is very narrow."(p. 374). Despite its apparent ease, however, the WMT does require neurocognitive resources and is thus potentially susceptible to failure due to underlying neural dysfunction. For example, individuals with memory disorders associated with some types of degenerative disease may "fail" the WMT (i.e., perform below established cut-score levels; see Merten et al. 2007) as do some patients with TBI (Flaro et al. 2007). Likewise, patients with major neuropsychiatric disorders like schizophrenia also exhibit a high WMT failure rate (Gorissen et al. 2005).

Clearly, brain injury and associated neurocognitive deficits can affect SVT performance. However, a major obstacle to SVT interpretation in the TBI patient is that only a few studies have attempted to examine how neuroimaging-identified abnormalities relate to SVT performance. Using a functional magnetic resonance imaging (fMRI) activation paradigm, Allen et al. (2007) and Larsen et al. (2010) examined the neural substrates required for performing the WMT in healthy controls. These studies demonstrated that WMT performance relies on an extended network involving long neural tracts that engage memory and attentional systems throughout the brain but left open the question of how damage to these networks would affect WMT performance. However, in a novel fMRI study of the WMT, Wu et al. (2010) demonstrated that two severe TBI patients who performed above cut-score levels on the WMT showed two divergent patterns of activation. Because patient activation patterns differed both from one another and from controls, these results suggest that brain injury had altered the basic cognitive network for normal WMT performance in these patients, who instead relied on adaptive and/or compensatory systems to perform well. Importantly, if brain injury alters the network necessary for normal WMT performance, it would suggest that the more this network is perturbed by injury, the more likely WMT performance would suffer.

While the findings of Wu et al. (2010) suggest that patients with severe TBI might rely on atypical brain systems in order to meet the cognitive demands required of the WMT, patients with mild TBI might be expected to show a different pattern. Based on results of previous fMRI studies using working memory and other cognitively demanding testing paradigms (McAllister et al. 2001, 2006), we might instead expect increased activation of the systems typically associated with WMT performance, particularly in prefrontal and parietal cortices. Following a theoretical development based on "cerebral challenge" as articulated in Hillary et al. (2006), for example, in brain injury, the neural system may respond to processing demands which are normally relatively unchallenging, in a way that resembles the neural system of healthy controls presented with relatively more challenging demands. We further suggest that this increase in activation of the typical system, as opposed to reliance on atypical systems, would more likely be observed in mild TBI, where brain injury results in tissue which is largely structurally intact, yet functionally compromised. Furthermore, as summarized in Hillary et al. (2006), within the dorsolateral prefrontal cortex (DLPFC), fMRI responses to increased cognitive load tend to appear more strongly in the right DLPFC compared to the left DLPFC, both in healthy controls as well as in clinical samples.

In Larsen et al. (2010), a sample of healthy young adults were recruited to examine functional activation associated with the WMT. Approximately 1 year after participating in the study, a female subject in her 20s sustained a TBI in an auto-pedestrian accident. Though tragic, this circumstance provided the patient with an opportunity to repeat the fMRI WMT to determine if activation patterns differed from pre- to post-injury. Given the role of frontal networks in memory performance and the fact that this patient has multiple right frontal white matter signal abnormalities, this within-subject design provides a format to explore how brain injury may change neural activation patterns associated with performing an SVT like the WMT.

Materials and Methods

Subjects

TBI Patient As mentioned above, the patient described in this study had volunteered as a control subject in a previous fMRI study of the WMT and approximately 1 year later sustained a TBI in an auto-pedestrian accident. On impact, she was thrown 15–20 ft with a second head impact on a cement curb. A brief alteration of consciousness was documented by a witness at the scene, and she remained confused according to eyewitnesses until emergency medical personnel arrived a few minutes after impact. Her pre-resuscitation Glasgow Coma Scale (GCS) by emergency personnel was 11 but 13 thereafter upon emergency room assessment. Initial CT imaging was negative with no skull fractures or intracranial hemorrhages. For approximately 8 h post-injury, her GCS fluctuated between 14 and 15. She was discharged after 24 h with a

diagnosis of "concussion." At 1 month post-injury, the patient reported persistent headaches, photophobia, dizziness, and problems with balance along with depressed mood, poor sleep, and cognitive dysfunction. At that time, a 3 T MRI scan revealed distinct right frontal hemorrhagic shear injuries and white matter signal abnormalities, as shown in Fig. 1a.

Because the patient was a control subject for Larsen et al. (2010), a few details about her pre-injury physical and cognitive status are known. Specifically, she was screened for psychological and neurological disorders as well as current or past psychotropic medication use. She did not have prior history of head injury or psychiatric disorder and met all requirements for participation in the Larson et al. investigation as a control subject. As for pre-injury cognitive functioning, the patient was a graduate student in a program with high admission and continuance standards. During the year post-injury, she was in active treatment in an outpatient neurorehabilitation program receiving cognitive rehabilitation, supportive psychotherapy, and pharmacotherapy. At the time of post-injury fMRI scanning, the patient reported that her subjective symptoms of brain injury—including headache, dysthymia, dizziness, and fatigue—were relatively low.

Procedures

For full details on the methods associated with the fMRI adaptation of the WMT, see Larsen et al. (2010). Briefly, the patient first memorized a set of 20 semantically associated word pairs (e.g., pants–belt). Next she was given an immediate recognition test in which each a word from the study list was paired with a new semantically related "foil" word (e.g., belt–buckle) and was required to indicate which of the two words had appeared earlier, by pressing either a right or left button on a response pad. After 30 min, the patient was given a second, delayed recognition test in which each word from the study list was paired with a new "foil" word (e.g., belt–loop). Functional images were acquired during this latter phase, which included both test and control conditions. During the test condition, stimuli were presented in blocks of eight sequential test trials. After each test block, a control block was presented in which two boxes were displayed on the screen in the same positions as the word stimuli had been, with one of the boxes filled in. Subjects were simply asked to indicate which box was filled in using the response pad. Each control block included eight stimulus items. This control task was selected because it imitated the motor activity of the test blocks but clearly required fewer cognitive demands and included no memory component. A total of five test–control block cycles were presented. Identical procedures were used for both pre- and post-injury testing, which occurred approximately 1 year before and 1 year after injury, respectively.

Functional images were acquired at 23 contiguous axial locations (5-mm slice thickness, 3.75×3.75 in-plane resolution) using an EPI–BOLD sequence with the critical parameters TR=2,000; TE=40 ms; flip angle=90. Preprocessing procedures included acquisition delay correction, motion correction, and spatial smoothing. The first image of the post-injury series was corregistered to the mean of the pre-injury images prior to motion correction. Mean images from both sessions were analyzed for pixel overlap to verify successful corregistration. A statistical analysis was performed using a time-series analysis of covariance (ANCOVA) implemented in SPM5 to test each voxel, for each condition (test and control) against the null hypothesis that changes in BOLD signal in that voxel, over the duration of the experiment, did not significantly correlate with the temporal sequencing of the cognitive task of interest. A boxcar waveform convolved with a synthetic hemodynamic response function with a 4-s lag-to-peak was used to model task-related activation. The data were high-passed-filtered in time using a set of discrete cosine basis functions with a cutoff period of 128 s and were conditioned for temporal autocorrelations using AR1 correction. Using the contrast weights from the ANCOVA analysis, t tests were performed for the contrast test–control. Further t tests were performed comparing this contrast (test–control) pre-injury versus post-injury. All resulting images are displayed with a family wise error-corrected threshold of $p<0.05$ and voxel extent threshold of 12.

Results

Significant regions of activation observed both in pre- and post-injury exams were generally consistent for what has previously been observed both at the group level (Allen et al. 2007; Larsen et al. 2010) and at the individual level (Wu et al. 2010). Significant regions of activation included the ventral visual processing stream, motor cortex, and frontal–parietal attentional systems (see Table 1 and Fig. 1b) with highly similar loci of activation peaks in pre- and post-injury exams. However, pre-injury activation included the hippocampus, which was absent post-injury, whereas post-injury activation included additional peaks in the dorsolateral prefrontal cortex compared to pre-injury activation. The most obvious difference between the scanning sessions, however, is the fact that activation is significantly stronger, with a greater extent of suprathreshold peaks, in the post-injury results, particularly in the frontal and parietal cortex. Specifically, out of 12 activation peaks observed for this patient, eight had significantly higher activation post-injury compared to pre-injury.

In terms of behavioral performance, the patient's responses were highly accurate both pre- and post-injury

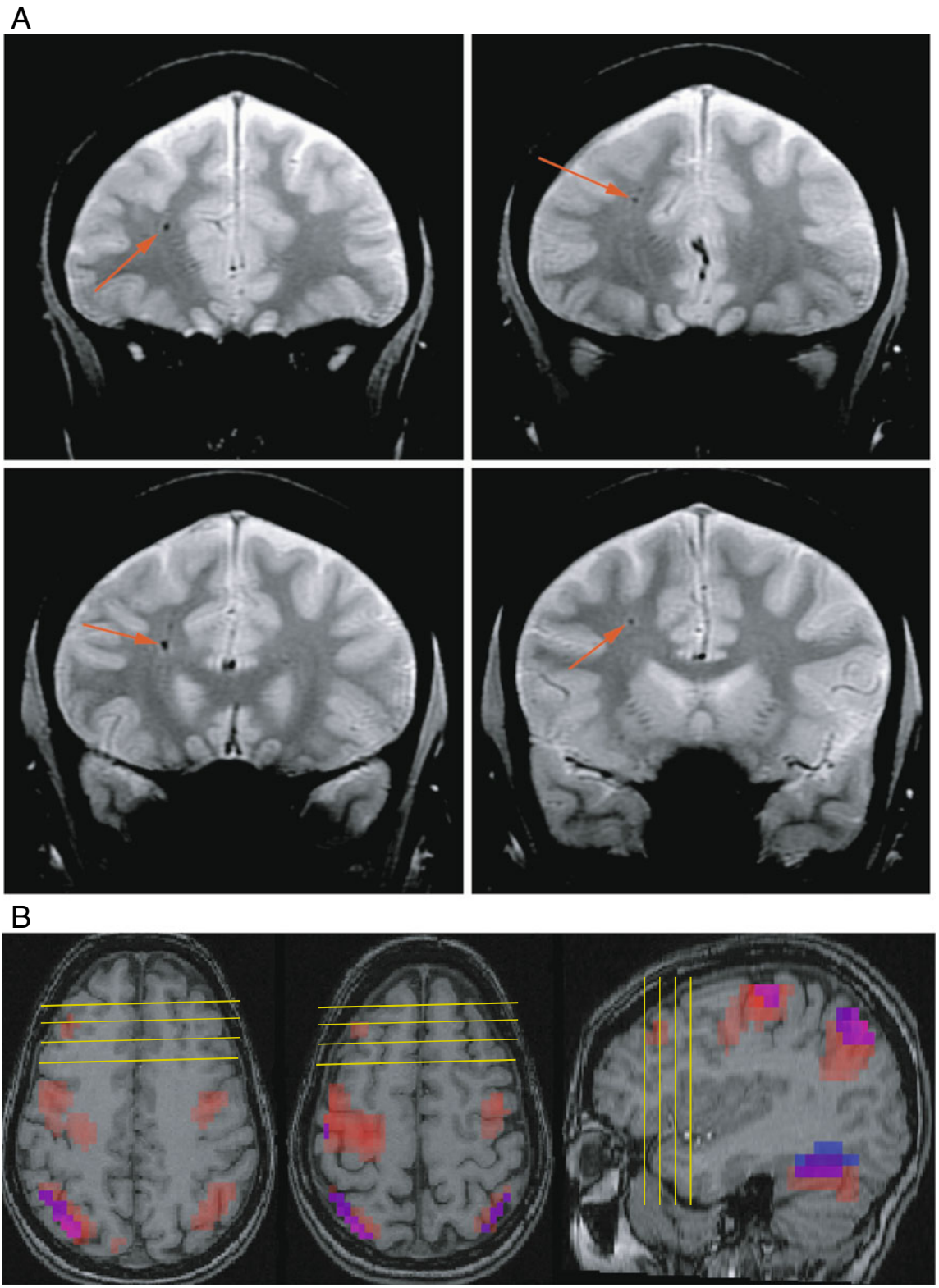

Fig. 1 **a** Four sequential gradient recalled echo sequences starting anteriorly (*upper left image*) and moving posteriorly by approximately 7 mm (*upper right*, to *lower left*, to *lower right*), showing multiple hemosiderin deposits in the right frontal regions. These corresponded with white matter signal changes on the fluid attenuated inversion recovery sequences (not shown) but reflective of white matter damage in addition to the location of the hemosiderin deposit. All images shown in radiological convention (left=right). **b** Axial (*left* and *middle*) and sagittal (*right*) overlays of significant activation on the DR subtest of the WMT. *Blue*=pre-injury; *red*=post-injury; *purple*=overlap. *Yellow lines* indicate placement of coronal cross-sections above

Table 1 Areas of significant activation for the contrast test–control on the delayed recognition subtest of the WMT for TBI patient pre-injury, post-injury, and control group

Anatomical region	Control group[a] Mean *t* score[b]	Pre-injury *t* score $p<0.05$[c]	Post-injury *t* score $p<0.05$[c]
Right middle frontal gyrus/premotor area	5.11	4.02	8.65[d]
Left middle frontal gyrus/premotor area	3.67	2.81	4.85[d]
Medial supplementary motor area	3.09	3.21	3.40
Right superior parietal lobe	4.89	4.36	7.14[d]
Left superior parietal lobe	3.44	3.64	4.12[d]
Right fusiform gyrus	4.58	4.19	4.27
Right lingual gyrus	3.05	3.47	4.74[d]
Right inferior occipital gyrus	3.57	3.19	5.15[d]
Left fusiform gyrus	2.50	2.87	4.24[d]
Left lingual gyrus	4.28	3.43	5.16[d]
Left inferior occipital gyrus	3.82	3.12	3.50
Bilateral anterior insula	2.32	–	–
Right hippocampus/parahippocampal gyrus	–	2.61[e]	–

[a] Based on reanalysis of control group reported in Larsen et al. (2010), excluding the TBI patient of this study (i.e., pre-injury)

[b] Group means were derived using the region of interest smoothed peak extraction procedure described in Allen and Fong (2008)

[c] Familywise error-corrected

[d] Post-injury>pre-injury ($p<0.05$)

[e] Pre-injury>post-injury ($p<0.05$)

(100% and 97.5% correct, respectively), with no significant difference. The pre-injury mean response latency was 706 ms (S.D.=155; range, 364–1,045), whereas the post-injury mean was 841 ms (S.D.=246; range, 449–1,435). Using the modified *t* test of Crawford and Howell (1998) to compare latencies with those from the remaining cohort of healthy control subjects from which the patient was drawn (control group mean=630; S.D.=104), the patient was significantly slower than the controls post-injury ($t_{[10]}=2.03$; $p<0.05$), but not pre-injury ($t_{[10]}=0.65$).

The above findings suggest an association between fMRI activation strength and reaction time, where both relatively more activation and longer reaction times are present post-injury compared to pre-injury. In order to examine this association further, a post hoc correlational analysis was performed in which the mean fMRI activation across the entire cortex was paired with the mean reaction time for each of the ten test blocks across both scanning sessions. To do this, the patient's cortex was custom parcellated into 45 functional regions per hemisphere using anatomical landmark definitions provided by Tzourio-Mazoyer et al. (2002). For each region, the mean *t* score was extracted to compute a global activation mean for the whole brain across all regions. The correlational analysis then paired mean activation with mean response time for each task block, where reaction time is assumed to be orthogonal to the original factor (test–control task difference) that was used to compute the activation *t* values. This analysis revealed a significant correlation ($r=0.69$. $p<0.01$).

As shown in Fig. 1a, scattered visible hemosiderin deposits were detectable within the right superior frontal region, an indication of traumatic shear lesions (Parizel et al. 1998). A fluid attenuated inversion recovery sequence (not shown) also demonstrated white matter signal changes in similar regions. Such lesions are likely to disrupt the connectivity of this region of the frontal lobe with the rest of the brain, providing a possible explanation for the patient's slower (yet accurate) performance post-injury.

Discussion

For this patient, we found that the loci of brain activation after injury were consistent with those observed prior to injury. However, the strength and spatial extent of the activation foci were notably increased post-injury. One possible explanation for these findings is a "cognitive load" account (Hillary et al. 2006). This explanation is a logical extension of fMRI findings in unimpaired subjects, where a greater extent of activation is typically associated with higher cognitive demands (Braver et al. 1997). Previously, explanations based on cognitive load have been applied to fMRI findings in patients with mild TBI. For example, McAllister et al. (2001, 2006), report that patients with mild

TBI show increased cortical activation while performing cognitive tasks at a level comparable to controls (at least in terms of task accuracy). It is possible that the current findings might be explained in a similar way to those of McAllister et al., given that she performed at equally high levels both pre- and post-injury but showed greater activation post-injury. Moreover, the fact that post-injury response times were both slower and more variable is consistent with the observation of wider functional activation extent, with more widespread activation peaks and fits with the assumption that wider activation extent reflects that the very same task (the WMT) placed greater cognitive load demands on the patient pre-injury compared to post-injury. It is important to note that this patient's activation profile is consistent with that noted in Hillary et al. (2006), in that increased activation was much more prominent in the right DLPFC than in the left DLPFC (see Fig. 1b). For this patient, these functional changes are assumed to be a consequence of reduced cognitive resources due to neurological damage. Her 1-month post-injury clinical MRI shows white matter and hemorrhagic lesions that would be disruptive of frontal brain networks. However, because she reported persistent symptoms of depression, headache, and fatigue, one might suspect moderating/mediating effects of these factors as well, though at the time of testing, all such symptoms were minimal. Regardless of any additional moderating variables, however, the observation of increased cognitive demand with mild brain injury presents a serious challenge to the assumption that the WMT is "virtually insensitive to all but the most extreme forms of impairment of learning and memory" (Flaro et al. 2007, p.374).

Although the current patient appears to fit in with the mild TBI findings of McAllister et al. (2001, 2006), other fMRI studies using patients with moderate to severe TBI raise the prospect of a more complex relationship between cortical damage and fMRI activation strength. For example, some recent fMRI studies of moderate and severe TBI patients employing working memory paradigms have found increased cortical activation (Newsome et al. 2007; Scheibel et al. 2003; Turner and Levine 2008), while other studies report decreased activation Sánchez-Carrión et al. (2008) as well as both increases and decreases (Strangman et al. 2008), depending on such factors as individual patient performance and brain regions of interest. Therefore, while there may be no simple generalization that predicts a precise course of functional adaptation for all brain injuries, the account given here, which posits increased activation as a result of reduced cognitive resources, appears to provide a reasonable explanation for the case at hand.

By using fMRI, response latency measures, and having had the unusual benefit of pre- and post-injury comparisons, we have discovered important details about this patient. Of course there are many limitations to an $N=1$ study such as this. If it were possible, it would have been ideal to have re-scanned all of the other subjects in the control group as a comparison for longitudinal change. Nonetheless, these patient-specific findings add to a growing body of evidence which challenges assumptions that rely on simple binary decisions of "good" versus "poor" effort on SVT measures (see McGrath et al. 2010). For example, in patients with mild TBI diffusion tensor imaging (which was not done on this patient) has shown damage to long white matter tracts of the brain (Messe et al. 2011). The more these tracts are damaged the more the attentional and working memory networks could be damaged that could slow response rate and alter SVT performance. Likewise, damage to the inferior frontal, frontotemporal, and limbic areas of the brain are commonplace in TBI, including mild TBI (Levine et al. 2008), and damage to these systems may affect motivation and drive (Pardini et al. 2010). All of these issues have simply not been systemically investigated with SVT measures and represent another limitation to SVT interpretation.

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