Psychiatry Research: Neuroimaging 233 (2015) 367–372

Contents lists available at ScienceDirect

Psychiatry Research: Neuroimaging

journal homepage: www.elsevier.com/locate/psychresns

Neural correlates of malingering in mild traumatic brain injury: A positron emission tomography study

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ARTICLE INFO

Article history:
Received 15 October 2014
Received in revised form
5 May 2015
Accepted 24 June 2015
Available online 27 June 2015

Keywords:
PET
Test of memory malingering
Ventromedial prefrontal cortex
Mild traumatic brain injury
Effort
Combat trauma

ABSTRACT

The detection of malingering in cognitive performance is a challenge in clinical and legal environments. Neuroimaging may provide an objective method to determine the source of failure on tests of symptom validity. Participants comprised 45 combat veterans, 31 with mild traumatic brain injury (mTBI), not seeking medical or legal compensation, who completed the Tombaugh Test of Memory Malingering (TOMM) and a positron emission tomography (PET) scan. Based on TOMM performance (i.e., less than 45 of 50 total correct, suggesting suboptimal effort or malingering), subjects were separated into poor TOMM score (PT; $n=10$) and good TOMM score (GT; $n=35$) groups. Voxel-based multiple regression analysis with Group (GT/PT) predicting uptake of fluorodeoxyglucose revealed decreased brain metabolism in the ventromedial prefrontal cortex of poor performers. The current findings may suggest that poor TOMM performance in those with combat trauma and mTBI may be related to ventromedial prefrontal cortical dysfunction. These findings have important implications for the disentanglement of feigned versus actual memory impairment, where the latter may be secondary to neural mechanisms not consistent with forgetting or deception.

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1. Introduction

Performance on measures of cognitive effort provides the context for the interpretation of neuropsychological test results. Individuals who fail effort testing may be categorized as severely impaired or malingering, and misassignment may result in failure to receive appropriate services (Lange et al., 2012). Mild traumatic brain injury (mTBI) has been widely linked to higher rates of failure on effort testing (Binder and Rohling, 1996). However, the prevalence of malingering in mTBI is difficult to determine for a number of reasons. Poor performance may be associated with brain injury (Willis et al., 2011), feigning impairment for external incentives (Bianchini et al., 2006) or both (Rogers and Vitacco, 2002). Further complicating matters, aside from autopsy for histopathological change directly following traumatic brain injury, a gold standard for diagnosis of a mild head injury is not clearly identified. Finally, successful malingerers, by definition, are not detected and thus cannot be included (Hurley and Deal, 2006). Therefore, there is a great need for improved assessment of cognitive effort in head injury patients.

Forced-choice neuropsychological testing, or symptom validity testing, is traditionally used to detect poor cognitive effort (Pankratz and Binder, 1997). This approach often invokes the illusion of task difficulty to increase test specificity (Hiscock and Hiscock, 1989). The Test of Memory Malingering (TOMM) is one of the most researched symptom validity tasks and uses a visual memory recognition procedure during which the assessor asks the participant to decide which of two pictures of common objects was shown previously (Tombaugh, 1996). A score significantly below the mean for individuals with confirmed cognitive impairment (a criterion of 90%, or 45/50, on the second recognition trial) suggests the possibility of less than optimal effort consistent with malingering (Tombaugh, 1996). While this cutoff score yields excellent sensitivity and specificity (100%) in student simulators and controls (Tombaugh, 1997), in samples of clinical populations some patients score in the simulator range. Furthermore, the sensitivity and specificity for patients with a complaint of traumatic brain injury is uncertain since there may be no independent validation of the injury (e.g., histopathology). Therefore, while neuropsychological measures of suboptimal effort can be highly effective, there is concern that some individuals may be missed, and others may be coached to avoid detection. For these reasons, objective means to detect effortful deception and simulated low effort performance are needed.

The majority of studies on the neural correlates of effortful

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http://dx.doi.org/10.1016/j.pscychresns.2015.06.007
0925-4927/Published by Elsevier Ireland Ltd.

failure have been conducted in healthy controls asked to simulate failure during functional neuroimaging. Findings indicate that the same prefrontal neural networks that support simulated task failure also underpin tasks that demand high cognitive effort (Larsen et al., 2010). Therefore, there may be substantial similarity in the neural correlates of "trying to fail" and "trying to succeed." A few studies have examined neural processes supporting *unintentional* memory failure. A recent meta-analysis revealed that unintentional false recognition responses (e.g., false alarms) were associated with significant activation of the medial, middle, and ventromedial prefrontal cortex, with the strongest effect in the ventral anterior cingulate cortex (vACC) (Browndyke, 2012). These findings suggest that one potential mechanism of symptom validity test failure, with special regard to false alarms, may be associated with dysfunction of the ventromedial prefrontal cortex.

As effortful failure results in a similar pattern of neural engagement as does effortful success (Larsen et al., 2010), research on instructed malingering may not provide insight into dissociating deception from other mechanisms of symptom validity test failure, such as intentional lack of effort or brain pathology. Furthermore, while conventional T1- and T2-weighted imaging techniques are relatively insensitive to the detection of mild neurotrauma, position emission tomography (PET) has been used to identify suboptimal brain function in the absence of structural lesions (Ruff et al., 1989, 1994; Chen et al., 2003; Belanger et al., 2007; Kato et al., 2007; Zhang et al., 2009; Kim et al., 2010; Provenzano et al., 2010; Peskind et al., 2011; Byrnes et al., 2014; Buchsbaum et al., 2015). Thus, we examined patterns of fluorodeoxyglucose-F18 (FDG) uptake in PET studies carried out in veterans with spontaneous failure on the TOMM who participated in a research protocol, were assured of the confidentiality of their test scores, and therefore had no benefit eligibility or financial incentive for poor performance. We hypothesized that poor TOMM performance might be linked to abnormal FDG uptake in the ventromedial prefrontal cortex, consistent with this region's role in compensatory response during false alarms (Browndyke, 2012) and memory retrieval processes (Moscovitch and McAndrews, 2002; Gilboa et al., 2006).

2. Methods

2.1. Subjects

Forty-five Operation Iraqi Freedom (OIF) and Operation Enduring Freedom (OEF) Veteran volunteers completed a computerized version of the Tombaugh Test of Memory Malingering (TOMM) and a positron emission tomography (PET) scan. Based on TOMM performance (i.e., less than 45 of 50 suggesting malingering) subjects were separated into poor TOMM performance (PT; $n=10$) and good TOMM performance (GT; $n=35$). All subjects were male, approximately 30 years old, and on average completed 14 years of education. Groups did not differ significantly on age, education or ethnicity (see Table 1). Of the 45 Veterans, 31 reported a history of mild traumatic brain injury (mTBI). Those Veterans with mTBI were analyzed as part of the whole cohort and separately. A whole brain analysis of the effects of mTBI on this sample has been reported elsewhere (Buchsbaum et al., 2015). The study was approved by the University of California San Diego (UCSD) Institutional Review Board and the San Diego Veterans Administration Research and Development review. A signed informed consent was obtained from each subject. Subjects were informed that the results would be kept confidential within our research group and thus would not affect any VA benefits.

The acquisition of PET imaging lasted about 40 min. Before coming in for a morning PET scan, subjects were instructed not to use caffeine or nicotine, or eat any foods with a high glucose concentration (after midnight) the night before. If subjects were to be scanned in the afternoon, they were asked to omit caffeine, nicotine, food and fluids (except water) for at least 4 h before the imaging session. All subjects were asked to abstain from alcohol for 48 h before their appointments. These precautions were taken to keep plasma glucose levels consistent and within a normal physiological range to reduce potential variability in the glucose metabolic measures.

Table 1
Characteristics of participants ($n=45$).

	Good TOMM	Poor TOMM	*t/chi*	*p*
Gender			0.000	1.000
Female	0	0		
Male	35	10		
Level of education			1.580	0.664
High school graduate	3	0		
Partial college	27	8		
Bachelor's degree	4	2		
Master's degree	1	0		
Ethnicity			2.370	0.668
African American	3	0		
Asian/Pacific Islander	3	0		
Caucasian	17	6		
Hispanic/Latino American	11	4		
Other	1	0		
Age (years)	28.20 (5.03)	32.80 (6.09)	2.185	0.034
Education (years)	14.06 (1.28)	14.70 (0.82)	1.895	0.065

Note Age and education are presented as mean (standard deviation). TOMM=Test of Memory Malingering; Good TOMM ($n=35$); Poor TOMM ($n=10$).

Subjects were assessed for psychiatric comorbidities using the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID) (First et al., 2002). A posttraumatic stress disorder (PTSD) diagnosis was determined by the Clinician-Administered PTSD Scale (CAPS) (Blake et al., 1995) (a score of at least 65). Combat-related mTBI, common among veterans involved in the Iraq and Afghanistan conflicts (Hoge et al., 2008), was defined by the following two criteria proposed by the American Congress of Rehabilitation Medicine: (1) A traumatically induced physiological disruption of brain function as indicated by at least one of the following: any period of loss of consciousness, any loss of memory for events immediately before or after the accident, any alteration in mental state at the time of the accident, and focal neurological deficits that may or may not be transient. (2) Severity of the injury does not exceed loss of consciousness of 30 min, Glascow Coma Scale score of less than 13 after 30 min, and posttraumatic amnesia of 24 h. Subjects were excluded if they had a history of bipolar disorder type I or any psychotic disorder, initiated psychotropic medication within the last 4 weeks or fluoxetine within the last 6 weeks, current use of mood stabilizers, current daytime dosing of benzodiazepines, multiple concurrent psychotropic medications, a history of more than 2 years of alcohol abuse or metal in their body.

2.2. Measurements

The TOMM is a 50-item recognition test designed for adults to discriminate between true memory-impaired patients and malingerers. Subjects are shown 50 simple line drawings for 3 s each, at 1 s intervals. Immediately afterwards, they are given 50 recognition panels, with 2 pictures per slide, each panel containing the previously shown picture and a new picture. The subject is asked to indicate which picture was previously viewed. Feedback regarding the correctness of the subject's response is given right away. Two learning trials of all 50 items are administered one after the other. Scores range from 0 to 50 for each trial. A score lower