Brain and Cognition 111 (2017) 112–126

Contents lists available at ScienceDirect

Brain and Cognition

journal homepage: www.elsevier.com/locate/b&c

Autobiographical and episodic memory deficits in mild traumatic brain injury

Jeffrey D. Wammes [a,*], Tyler J. Good [b,c], Myra A. Fernandes [a]

[a] Department of Psychology, University of Waterloo, N2L 3G1 Waterloo, Ontario, Canada
[b] Department of Psychology, University of Toronto, M5S 1A1 Toronto, Ontario, Canada
[c] Rotman Research Institute, Baycrest Centre, M6A 2E1 Toronto, Ontario, Canada

ARTICLE INFO

Article history:
Received 7 January 2016
Revised 11 November 2016
Accepted 14 November 2016

Keywords:
Mild traumatic brain injury
Concussion
Aging
Episodic memory
Semantic memory
Autobiographical memory

ABSTRACT

Those who have suffered a concussion, otherwise known as a mild traumatic brain injury (mTBI), often complain of lingering memory problems. However, there is little evidence in the behavioral literature reliably demonstrating memory deficits. Thus, in the present study, cognitive profiles including measures of general executive functioning and processing speed, as well as episodic and semantic memory were collected in younger and older adult participants with or without a remote (>1 year prior to testing) mTBI. We first investigated whether there were observable episodic and autobiographical memory impairments associated with mTBI within an otherwise healthy young group. Next, because previous work had demonstrated some overlap in patterns of behavioral impairment in normally aging adults and younger adults with a history of mTBI (e.g. Ozen, Fernandes, Clark, & Roy, 2015), we sought to determine whether these groups displayed similar cognitive profiles. Lastly, we conducted an exploratory analysis to test whether having suffered an mTBI might exacerbate age-related cognitive decline. Results showed the expected age-related decline in episodic memory performance, coupled with a relative preservation of semantic memory in older adults. Importantly, this pattern was also present in younger adults with a history of remote mTBI. No differences were observed across older adult groups based on mTBI status. Logistic regression analyses, using each measure in our battery as a predictor, successfully classified mTBI status in younger participants with a high degree of specificity (79.5%). These results indicate that those who have had an mTBI demonstrate a distinct cognitive signature, characterized by impairment in episodic and autobiographical memory, coupled with a relative preservation of semantic memory.

1. Introduction

In the current study, we tested whether having suffered a remote mild traumatic brain injury would be associated with lingering memory issues, particularly in episodic, and personal autobiographical memory (AM). The detrimental consequences of having suffered a mild traumatic brain injury (mTBI) are becoming more apparent in a number of different cognitive domains, promoting a shift in attitude from the casual nature with which these injuries have been discussed in the past. One need not look back too far in history however, to encounter alarming quotes such as this one from the former chair of the NFL's mTBI committee in 1994: "Veterans clear more quickly than rookies ... They can unscramble their brains a little faster, maybe because they're not afraid after being dinged" (Farber, 1994). This quote is just one example, but is demonstrative of a more general phenomenon that existed in the past. This casual attitude toward concussion and related consequences is not unusual in the sporting world, or within the broader public. However, the slow return to play of superstar athletes, and potentially reduced prowess upon return, has gradually begun to reverse this trend, and draw public attention to a broader impairment resulting from these instances of getting one's 'bell rung'. Cognitive research has been instrumental in delineating the veridical severity of these 'mild' injuries. In the present experiment, we explored whether mTBI might lead to observable and lingering deficits in memory performance.

For the purposes of the current work we define mTBI, or concussion, as a closed head injury that results from the head being hit, striking an object, or enduring any acceleration or deceleration

* Corresponding author at: Department of Psychology, University of Waterloo, 200 University Ave W, Waterloo, ON N2L 3G1, Canada.
E-mail address: jwammes@uwaterloo.ca (J.D. Wammes).

http://dx.doi.org/10.1016/j.bandc.2016.11.004
0278-2626/

force (Kay et al., 1993). Those who have suffered an mTBI often report more confusion, difficulty concentrating, and memory failures than age-matched controls (Vanderploeg, Belanger, & Curtiss, 2009; Vanderploeg, Curtiss, Luis, & Salazar, 2007). While notable, these observations are made primarily within the acute stage (<3 months after) of injury. Despite the prevalence of these complaints, findings from neuropsychological measures and the results from cognitive task performance have been mixed (Binder, Rohling, & Larrabee, 1997; Schretlen & Shapiro, 2003; Zakzanis, Leach, & Kaplan, 1999). However, research has shown some evidence for prolonged deficits, primarily in the domains of attention (Binder et al., 1997; Chan, 2002; Solbakk, Reinvang, Nielsen, & Sundet, 1999; Vanderploeg, Curtiss, & Belanger, 2005) and processing speed (Bernstein, 2002; Johansson, Berglund, & Rönnbäck, 2009; Potter, Jory, Bassett, Barrett, & Mychalkiw, 2002). Still, in the majority of domains and tasks, it appears that performance reductions are limited to the acute stage for most people, with little evidence for more chronic deficits (Schretlen & Shapiro, 2003).

1.1. Brain basis of mTBI

In concert with experiments focusing on finding reliable behavioral indicators of mTBI, there has also been an ongoing search for objective physiological markers, often employing diffusion tensor imaging (DTI). Physical evidence of mTBI however is elusive, as many people do not seek medical attention afterward (Jeter et al., 2013), and conventional imaging techniques usually fail to detect abnormalities (Bazarian et al., 2005; Belanger, Vanderploeg, Curtiss, & Warden, 2007; Cohen et al., 2007), or the observed differences are resolved within 45 days post-injury (Maugans, Farley, Altaye, Leach, & Cecil, 2012; McCrea, Prichep, Powell, Chabot, & Barr, 2010). In fact, even when those with a history of mTBI were each scanned using three different imaging techniques (MRI, MEG, SPECT), only 73% of the participants with a history of mTBI showed evidence of abnormality in *any* scan, and only 13% showed abnormalities in all three (Lewine et al., 2007).

DTI seems to be the most reliable technique with which mTBI-related pathology has been detected. Low fractional anisotropy (FA) measures derived from this technique indicate that water diffusion is occurring relatively uniformly in all directions, and are indicative of pathological white matter tracts (i.e. a lack of normal directional diffusion). This is an especially relevant measure for mTBI, which results in the cutting or shearing of axons (Biasca & Maxwell, 2007; Bigler & Maxwell, 2012; Büki & Povlishock, 2006; Johnson, Stewart, & Smith, 2012; Tang-Schomer, Patel, Baas, & Smith, 2010). Moreover, the initial injury seems to trigger a cascade of cellular activity that culminates in more widespread axonal damage and pathology over the course of a few months post-injury (Bigler & Maxwell, 2012; Johnson et al., 2012; Povlishock, 1992; Stirling & Stys, 2010; Sulaiman et al., 2011).

Accordingly, DTI has provided evidence for axonal pathology in mTBI, showing abnormalities in FA in this group (Bazarian et al., 2007; Lipton et al., 2012). Findings show primarily low FA values in the acute and chronic stages of recovery, suggesting that white matter integrity has been compromised (Arfanakis et al., 2002; Jorge et al., 2012; Little et al., 2010; Niogi et al., 2008; Shenton et al., 2012, but see Lipton et al., 2012). Critically, these differences in FA appear to be associated with performance on cognitive tasks in mTBI groups (e.g. Jeter et al., 2013). Moreover, research has shown that there can be domain-specific cognitive impairment, and the pattern of these impairments tends to logically map on to the locus of white matter damage in mTBI (Wilde et al., 2010; Yallampalli et al., 2013). Thus this injury to axons is likely the neural substrate underlying any cognitive and neuropsychological deficits that might occur as a result of mTBI (Jeter et al., 2013; Meythaler, Peduzzi, Eleftheriou, & Novack, 2001; Sharp & Ham, 2011).

1.2. Evidence for memory dysfunction in mTBI

The focus of the current work is in the domain of memory, with a particular focus on exploring deficits that might linger beyond the acute stage post-injury. As mentioned in the foregoing, there is evidence to suggest some specificity in the cognitive impairment experienced by those who have had an mTBI, which is related to the regions showing the greatest reductions in FA. These observations are often made with respect to performance on memory tasks, which then maps on to white matter pathology in tracts commonly associated with memory (Jeter et al., 2013). Most importantly, DTI research suggests that the medial temporal lobe and the fornices (which have projections to and from bilateral hippocampi) are among the areas in which white matter integrity is most compromised in mTBI (Niogi & Mukherjee, 2010), a finding that has been confirmed post-mortem (Blumbergs et al., 1994). Interestingly, such differences in FA in the fornix and hippocampal formation correlate significantly with performance on tasks of associative and verbal episodic memory (Jeter et al., 2013; Kinnunen et al., 2010; Yallampalli et al., 2013). Other work that mTBI-related deficits can be specific to episodic memory using alternate measures of white matter integrity (Geary, Kraus, Pliskin, & Little, 2010; Kraus et al., 2007; Levin et al., 2010), general brain activity (MEG: Lewine et al., 2007; fMRI: Monti et al., 2013), and FA measures in other white matter tracts (e.g. the uncinate fasciculus, which has connections with amygdala and hippocampus) (Niogi et al., 2008). Taken together, the conclusions from the foregoing neuroimaging research lead logically to the prediction that even in chronic stages of recovery, memory deficits would likely be prevalent in those who have suffered mTBI.

Results from behavioral studies exploring deficits in memory performance that endure beyond the acute stage of injury however, have been quite mixed. One early meta-analysis explored exclusively mild TBI (excluding moderate or severe injuries), including only samples collected beyond the acute stage (greater than 3 months) of recovery. The authors tested for emergent effects within a number of cognitive domains, including memory acquisition and delayed memory performance, and found no reliable evidence for memory costs (Binder et al., 1997). The majority of the included studies showed no memory deficits (Alterman, Goldstein, Shelly, Bober, & Tarter, 1985; Bornstein et al., 1993), and those that did either tested participants under oxidative stress (Ewing, McCarthy, Gronwall, & Wrightson, 1980) or included a broad spectrum of injury severities, rather than being selective of those with mild concussion (Dikmen, Machamer, Winn, & Temkin, 1995). As such, despite having found memory deficits as a result of head injury, their findings were likely driven by participants with injuries of greater severity, rather than mTBI. Consistent with this idea, performance of those within their mildest severity group was found to be comparable to that of controls (Dikmen et al., 1995). Thus, this study provided no evidence of *enduring* memory deficits as a result of *mild* TBI, or concussion. A subsequent re-analysis of two meta-analytic datasets (Binder et al., 1997; Frencham, Fox, & Maybery, 2005) including many of the foregoing studies, demonstrated that memory deficits may exist in specific tasks (e.g. verbal paired memory), but were obscured by collapsing across memory tasks (Pertab, James, & Bigler, 2009). Perhaps the most relevant study analyzed by that work reported a deficit in difficult paired associates problems during the chronic stage of