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Note

Autobiographical memory and structural brain changes in chronic phase TBI

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ABSTRACT

Traumatic brain injury (TBI) is associated with a range of neuropsychological deficits, including attention, memory, and executive functioning attributable to diffuse axonal injury (DAI) with accompanying focal frontal and temporal damage. Although the memory deficit of TBI has been well characterized with laboratory tests, comparatively little research has examined retrograde autobiographical memory (AM) at the chronic phase of TBI, with no prior studies of unselected patients drawn directly from hospital admissions for trauma. Moreover, little is known about the effects of TBI on canonical episodic and non-episodic (e.g., semantic) AM processes. In the present study, we assessed the effects of chronic-phase TBI on AM in patients with focal and DAI spanning the range of TBI severity. Patients and socioeconomic- and age-matched controls were administered the Autobiographical Interview (AI) (Levine, Svoboda, Hay, Winocur, & Moscovitch, 2002) a widely used method for dissociating episodic and semantic elements of AM, along with tests of neuropsychological and functional outcome. Measures of episodic and non-episodic AM were compared with regional brain volumes derived from high-resolution structural magnetic resonance imaging (MRI). Severe TBI (but not mild or moderate TBI) was associated with reduced recall of episodic autobiographical details and increased recall of non-episodic details relative to healthy comparison participants. There were no significant associations between AM performance and neuropsychological or functional outcome measures. Within the full TBI sample, autobiographical episodic memory was associated with reduced volume distributed across temporal, parietal, and prefrontal regions considered to be part of the brain's AM network. These results suggest that TBI-related distributed volume loss affects episodic autobiographical recollection.

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1. Introduction

Autobiographical memory (AM) tasks draw upon multiple cognitive operations that bring to consciousness details about both personal past episodes (i.e., episodic AM) or personal factual information (i.e., personal semantic memory; Conway, 2001; Levine et al., 2002; Renoult, Davidson, Palombo, Moscovitch, & Levine, 2012). Past neuroimaging work has shown that AM is supported by a network of regions, including the medial and lateral prefrontal, posterior cingulate, and medial and lateral temporal cortices, and the medial temporal lobes (Cabeza & St Jacques, 2007; Svoboda, McKinnon, & Levine, 2006). Alterations to regions within this network, particularly in the medial temporal lobes, leads to deficits in episodic AM (for review, see Moscovitch, Cabeza, Winocur, & Nadel, 2016; Sheldon, Farb, Palombo, & Levine, 2016; Winocur & Moscovitch, 2011). Less is known about the effects of damage to other parts of the AM network (for exceptions, see Berryhill, Phuong, Picasso, Cabeza, & Olson, 2007; Bright et al., 2006; Davidson et al., 2008; Kopelman, Stanhope, & Kingsley, 1999; McKinnon et al., 2008).

Traumatic brain injury (TBI) is characterized by diffuse axonal injury (DAI; Povlishock & Katz, 2005) causing volume loss across the cortical mantle (Levine et al., 2008) as well as focal cortical contusions in ventral frontal and anterior temporal regions (Gentry, Godersky, & Thompson, 1988). Given the distributed nature of the functional neuroanatomy of AM, TBI provides a unique lesion model for the understanding brain mechanisms underlying AM. Moreover, it is important for clinical reasons to clarify the effects of TBI on AM, as memory complaints in general are a cardinal sign of post-TBI cognitive changes (Stuss & Gow, 1992) and the most common cognitive complaint following TBI (Mateer, Sohlberg, & Crinean, 1987).

Although TBI is associated with retrograde amnesia and confabulation in the acute phase (for review, see Schacter & Crovitz, 1977), few studies have assessed AM in chronic phase TBI patients. Severe TBI is associated with impaired episodic AM (Carlesimo et al., 1998; Coste et al., 2011, 2015; Knight & O'Hagan, 2009; Levin et al., 1985; Piolino et al., 2007; Rasmussen & Berntsen, 2014) whereas results in mild to moderate TBI have been mixed, with case study evidence of impairment (e.g., Starkstein, Sabe, & Dorrego, 1997) and impairment in university students with concussion history (Barry & Tomes, 2015) but not in veterans with mild blast-related TBI (Palombo et al., 2015). All previous studies of TBI have used small samples recruited post-acute hospitalization, excluding patients with good recovery who did not present for treatment following hospitalization.

We assessed AM in an unselected sample of 70 patients recruited from initial hospital admission spanning the full spectrum of TBI, along with socioeconomic-matched comparison subjects, using the Autobiographical Interview (AI; Levine et al., 2002). In this technique, naturalistic autobiographical protocols are transcribed and segmented into internal (episodic) or external (non-episodic) details. The AI allows for the parametric quantification of independent measures of episodic and non-episodic memory from within a single narrative, whereas other measures probe these memory processes through separate interviews (e.g., Kopelman, Wilson, & Baddeley, 1989) or focus only on episodic AM (e.g., Piolino, Desgranges, Benali, & Eustache, 2002). The AI assesses memory for events selected by participants prior to testing, isolating elaboration of event details from generation and selection of events that are highly dependent on executive processes. Finally, we probed five lifetime periods to assess age-of-memory (i.e., temporal gradient) effects with the prediction that episodic AM impairment in TBI would show a flat temporal gradient, as expected with diffuse damage (Carlesimo et al., 1998; Piolino et al., 2007).

The AI has been applied widely in samples of patients with brain disease (e.g., Addis, Moscovitch, & McAndrews, 2007; Irish et al., 2011; McKinnon et al., 2008; Murphy, Troyer, Levine, & Moscovitch, 2008; Rosenbaum et al., 2008), aging (Addis, Wong, & Schacter, 2008; Levine et al., 2002), and psychiatric conditions (McKinnon et al., 2015; Söderlund et al., 2014). It has been applied in one study of nine individuals with moderate-severe TBI (Rasmussen & Berntsen, 2014) and one study of veterans with mild TBI due to blast exposure (Palombo et al., 2015). We predicted that patients with severe TBI would have reduced episodic AM relative to comparison participants. Given the conflicting findings in the literature regarding mild-moderate TBI (Barry & Tomes, 2015; Palombo et al., 2015), we had no specific prediction for these groups. We also assessed the relationship of AM as assessed by the AI to performance on an extensive battery of neuropsychological tests, including tests of executive function that have been previously related to AM performance using other measures in TBI (Coste et al., 2011, 2015; Piolino et al., 2007).

To our knowledge, there are no published studies assessing the relationship of AM to structural brain changes in TBI. We assessed the relationship of AM performance to regional brain volumes quantified from high resolution structural magnetic resonance imaging (MRI) scans using a multivariate statistical technique, partial least squares (PLS). We predicted that the recall of internal details would be associated with reduced brain volume in the frontal, temporal, and medial parietal regions known to be associated with AM (Cabeza & St Jacques, 2007; Svoboda et al., 2006). Furthermore, given that the AM network is functionally distributed, we predicted that distributed volume loss would be associated with reduced recollection of episodic AM.

2. Materials and methods

2.1. Participants

Patients were recruited from consecutive admissions to Sunnybrook Health Sciences Centre at approximately one year post-injury as part of the Toronto Traumatic Brain Injury Study (see Fujiwara, Schwartz, Gao, Black, & Levine, 2008; Guild & Levine, 2015; Levine et al., 2005, 2008, 2013). Injury severity was determined by the Glasgow Coma Scale (GCS), as documented by the trauma team leader's score at the time of discharge from the Trauma Unit. For most patients, this score corresponded to the recommended 6-h GCS score (Teasdale & Jennett, 1974). Severity classification (mild/moderate/severe) was upgraded in 10 cases, where extended loss of consciousness (>2 h), post-traumatic amnesia (>48 h), or focal lesions