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Distinction in EEG slow oscillations between chronic mild traumatic brain injury and PTSD

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ABSTRACT

Spectral information from resting state EEG is altered in acute mild traumatic brain injury (mTBI) and in disorders of consciousness, but there is disagreement about whether mTBI can elicit long term changes in the spectral profile. Even when identified, any long-term changes attributed to TBI can be confounded by psychiatric comorbidities such as PTSD, particularly for combat-related mTBI where postdeployment distress is commonplace. To address this question, we measured spectral power during the resting state in a large sample of service members and Veterans varying in mTBI history and active PTSD diagnosis but matched for having had combat blast exposure. We found that PTSD was associated with decreases in low frequency power, especially in the right temporoparietal region, while conversely, blast-related mTBI was associated with increases in low frequency power, especially in prefrontal and right temporal areas. Results support the idea that long-term neurophysiological effects of mTBI share some features with states of reduced arousal and cognitive dysfunction, suggesting a role for EEG in tracking the trajectory of recovery and persisting vulnerabilities to injury. Additionally, results suggest that EEG power reflects distinct pathophysiologies for current PTSD and chronic mTBI.

1. Introduction

While acute effects of mild traumatic brain injury (mTBI) are well established, whether an mTBI has long term neurological effects is extremely controversial. It is well known that many individuals experience persisting symptoms; there is also growing concern that even asymptomatic individuals may have some neurologic compromise that increases the risk for poor outcome if they sustain additional brain insult of any type. However, long-term neurological changes that are specifically linked to mTBI and that may underlie persistent symptoms or prolonged vulnerability to injury have remained elusive.

Recent neuroimaging studies after mTBI suggest the injury may progressively affect brain volume and white matter (Zhou et al., 2013) as well as resting state activation (Messé et al., 2013). Similarly, resting state EEG may show a similar sensitivity to chronic effects of mTBI, with the additional advantages of temporal resolution and ease of measurement. In the acute phase of mTBI, visible abnormalities in EEG, including diffuse slowing, are related to the duration of loss of consciousness (LOC) and post-traumatic amnesia (PTA), and these abnormalities may persist for up to a year (Haneef et al., 2013; Nuwer et al., 2005). In a study of EEG and other metrics collected immediately after an observed sports concussion, it was found that resting EEG measures predicted time until return to play better than neuropsychological, balance, or symptom measures (Prichep et al., 2013). Specifically, an EEG index that included power information discriminated between severities of mTBI and was sensitive to injury effects much longer than other indicators (45 days after injury for EEG vs. at time of injury for other measures). A small number of studies have looked at quantitative resting EEG features in the chronic phase of mTBI to assess for long lasting effects. One study examined EEG from symptomatic patients on average six months after their mTBI, and showed that the increase in slower frequency activity seen acutely both persisted and mapped onto abnormalities in the blood brain barrier (Korn et al., 2005). Another study reported decreased frontal phase synchrony in all EEG bands except alpha in a sample of blast-injured Veterans an average of 2.5 years after injury (Sponheim et al., 2011). Notably, EEG phase synchrony measures were more sensitive in discriminating the blast-injured group from healthy controls than were measures of frontal white matter integrity, to which they were also correlated.

While these findings support a role for resting EEG in evaluating the effects of mTBI, the almost infinite number of possible quantitative index measures poses a challenge. EEG spectral profiles (the amount of power by frequency component of the signal) have known physiological and clinical significance and therefore have potential for meaningfully evaluating mTBI. For instance, different states of consciousness,

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e.g. the vegetative vs. minimally conscious states after severe TBI (Schiff et al., 2014), or responsiveness under anesthesia (Dressler et al., 2004), have different signatures in the spectral profile. Considering these findings and the fact that mTBI by definition involves some acute impairment or alteration of consciousness, it is possible, but untested, that mTBI may be marked by lasting distortion in the power profile. Further, it is hypothesized that the power profile of mTBI will be consistent with a state of mildly reduced arousal, consistent with deficits of attention in this group (Cicerone, 1996). A handful of studies have examined spectral changes in chronic mTBI, but group confounds and inconsistencies in mTBI definitions have made it difficult to infer any specific effect of the injury (Rapp et al., 2015).

Of many potential confounds, psychiatric comorbidity may be among the most important, particularly for combat-related mTBI. In military health care settings, PTSD is especially common and questions remain about the relative contribution of such disorders to neurophysiologic measures, especially in the chronic phase of mTBI. EEG is likely to be affected by pathologic vigilance states like PTSD given that EEG power is known to be sensitive to vigilance states in the uninjured person (Paus et al., 1997). However, the effects of mTBI on EEG power profiles have not been compared to those of PTSD, and so it is not clear how each condition might differentially contribute to EEG power. The goal of the present research is to compare the spectral profiles, with the hypothesis that PTSD and chronic mTBI will both affect the spectral profile, but in patterns consistent with increased vigilance (PTSD) versus reduced arousal (mTBI). Arousal is not a unitary construct but may be defined as including general alertness (vigilance) and a related but higher order process of controlled attention (Coull, 1998) either or both of which could contribute to performance on neurocognitive tests. We also aim to compare spectral profiles with measures of cognitive functioning in order to describe the potential behavioral and functional significance of spectral distortions.

2. Material and methods

2.1. Participants

Participants were active duty service members or Veterans, all of whom had experienced blast exposure during combat deployment in Iraq or Afghanistan within the two years prior to consent. Participants were recruited between 2008 and 2013 from the McGuire Veterans Affairs Medical Center (VAMC) in Richmond, Virginia, and from several nearby military bases. In order to be eligible, one of the following symptoms or events immediately after the blast/explosion was required: dazed, confused, saw stars, headache, dizziness, irritability, memory gap (not remembering injury or injury period), hearing loss, abdominal pain, shortness of breath, struck by debris, knocked over or down, knocked into or against something, helmet damaged, or evacuated. Thus, witnessing a blast from afar was not considered exposure. Participants were excluded if they had ever experienced a moderate or severe TBI (> 30 min in coma, brain bleeding or blood clot (traumatic abnormality on brain CT scan), or post-traumatic amnesia (PTA) duration >24 h). All study procedures were conducted with approval of the McGuire Institutional Review Board.

All participants completed demographic and medical history questionnaires as well as an inventory of current risky alcohol consumption, the AUDIT-C (Bush et al., 1998) and a measure of current neurobehavioral symptoms relative to the period pre-blast exposure, the Rivermead Post-concussion Questionnaire (RPQ; (King et al., 1995). Descriptive information can be found in Table 1. Participants were not excluded based on history of psychotropic medication use, however participants were asked if they had ever been prescribed a medication for behavioral or thought disorder, and if so to provide the medication name(s). Responses were used to estimate potential psychotropic medication use at testing according to the following categories: none/never prescribed, selective serotonin reuptake inhibitor (SSRI), serotonin-norepinephrine reuptake inhibitor (SNRI), other antidepressant, stimulant, atypical antipsychotic, antiepileptic, or benzodiazepine. In six cases, the participant knew they had taken medication for depression but did not know the medication name; these cases were coded as SSRI, the most commonly prescribed antidepressant. Participants were not asked to differentiate between past or current use; however, if participants volunteered that their use was only in the past, this was coded as "none." Medication information is summarized in Table 2.

2.2. Procedures

All data collection took place on the same day in private testing spaces either at the McGuire VAMC or on base for active duty participants.

Table 1
Descriptive statistics. Subscores of the PCL are given for the symptom structure described by Yufik and Simms (Yufik and Simms, 2010).

Characteristic	Level	Full sample	No mTBI	mTBI no PTA	mTBI with PTA	No PTSD	PTSD
N		147	32	44	71	107	40
Age mean (SD)		27.8 (7.9)	30.7 (10.2)	27.3 (6.4)	26.7 (7.3)	26.9 (7.2)	30.1 (7.9)[a]
Education n (%)	Non-high school	1 (<1%)	0	0	1 (1%)	1 (1%)	0
	High school graduate	74 (50%)	11 (34%)	25 (57%)	38 (54%)	52 (49%)	22 (55%)
	Some college	52 (35%)	13 (40%)	19 (43%)	20 (28%)	39 (36%)	13 (33%)
	College graduate	17 (12%)	7 (22%)	0	10 (14%)	13 (12%)	4 (10%)
	Post-graduate degree	3 (2%)	1 (3%)	0	2 (3%)	2 (2%)	1 (3%)
Sex n (%)	Male	141 (96%)	28 (88%)	44 (100%)	69 (97%)	102 (95%)	39 (98%)
	Female	6 (4%)	4 (12%)	0	2 (3%)[b]	5 (5%)	1 (2%)
Months since worst blast median [IQR]		10.9 [13.2]	12.2 [11.7]	14.5 [15.6]	8.5 [9.7]	10.1 [10.3]	14.6 [18.1][c]
	No	100 (68%)	21 (66%)	25 (57%)	54 (76%)	80 (75%)	20 (50%)[d]
AUDIT-C mean (SD)		5.2 (2.8)	4.9 (2.9)	5.1 (2.6)	5.6 (3.0)	4.7 (2.7)	6.8 (2.8)[a]
RPQ		29.9 (12.7)	24.7 (12.7)	30.7 (12.6)	31.8 (12.2)[a]	26.5 (11.3)	39.1 (11.8)
PCL		47.6 (15.5)	43.5 (16.5)	50.0 (16.0)	48.3 (14.5)	41.8 (12.9)	63.3 (9.8)[a]
Hyperarousal		6.3 (2.4)	5.8 (2.5)	6.6 (2.4)	6.4 (2.4)	5.7 (2.4)	8.0 (1.7)[a]
Intrusion		13.8 (4.9)	12.0 (5.1)	14.0 (4.8)	14.5 (4.8)	12.3 (4.6)	17.7 (3.6)[a]
Avoidance		5.2 (2.3)	4.8 (2.6)	5.5 (2.2)	5.1 (2.3)	4.4 (1.9)	7.4 (1.9)[a]
Dysphoria		22.3 (7.8)	20.9 (9.0)	23.4 (8.2)	22.2 (7.0)	19.3 (6.3)	30.3 (5.6)[a]

Tests of group differences conducted between levels of a factor, e.g. PTSD vs no PTSD.
[a] Significant at 0.05, ANOVA.
[b] Significant at 0.05, Fisher's Exact test.
[c] Significant at 0.05, medians test.
[d] Significant at 0.05, chi-square test.