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Neurophysiological correlates of dysregulated emotional arousal in severe traumatic brain injury

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HIGHLIGHTS

- EEG alpha power provides a novel index of arousal disturbances in traumatic brain injury (TBI).
- Diminished alpha suppression and skin conductance levels are in line with diminished arousal.
- Insula and amygdala volume loss may contribute to arousal dysregulation.

ABSTRACT

Objective: This study aimed to elucidate relationships between dysregulated emotional arousal after severe traumatic brain injury (TBI), alpha power and skin conductance levels (SCL), and brain atrophy.
Methods: Nineteen adults with severe TBI and 19 age-, education-, and gender-matched controls (all p's > 0.05) participated. Magnetic resonance imaging (MRI) scan established bilateral insulae and amygdale volumes. Mean EEG alpha power and SCLs were recorded simultaneously across four, 2 min conditions: eyes-closed pre-task baseline, view neutral face, happy face and angry face.
Results: Scalp-wide alpha suppression occurred from pre-task baseline to the face-viewing conditions (p < .001), but was diminished in TBI (p = .04). TBI participants exhibited marginally significantly lower SCL (p = .051), and elevated alpha power hemispherically, contrasting with controls' midline dominance (p < .01). Significant atrophy was observed in most structures in TBI participants (p's = .004–0.04). Larger left insula, left amygdala and right amygdala correlated positively with alpha power and alpha suppression, and SCLs; all structures uniquely contributed to variance in arousal.
Conclusions: Findings suggest that alpha power provides a sensitive measure of dysregulated emotional arousal post-TBI. Atrophy in pertinent brain structures may contribute to these disturbances.
Significance: These findings have potential implications for the assessment and remediation of TBI-related arousal deficits, by directing more targeted remediation, and better assessing post-TBI recovery.

1. Introduction

Deficits in arousal and responsivity to emotional stimuli are well documented in adults following severe traumatic brain injury (TBI). Such deficits are pervasive, being evident in response to different stimulus modality presentations (emotional faces and film clips), and across different types of measures (physiological and self-report ratings). Specifically, the physiological evidence from TBI studies points to muted skin conductance, reduced eye-blink startle and facial EMG (electromyography) responses (Blair and Cipolotti, 2000; de Sousa et al., 2012, 2011, 2010; Hopkins et al., 2002; McDonald et al., 2011; Sanchez-Navarro et al., 2005; Saunders et al., 2006) to aversive stimuli in particular, including angry facial expressions, distressing film clips, and unpleasant pictures (e.g., IAPS; Sanchez-Navarro et al., 2005). Deficits in psychophysiological responding to aversive stimuli in severe TBI are reflected in lower self-reported levels of arousal (e.g., Saunders et al., 2006) and valence (de Sousa et al., 2010). TBI adults who self-report low empathy levels also exhibit reduced facial muscle

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responding to both pleasant and unpleasant stimuli, as well as lower autonomic arousal (i.e., skin conductance, de Sousa et al., 2012). Hypoarousal may therefore contribute to a reduced capacity to understand, and share others' feelings, thoughts and intentions, abilities which underlie successful prosocial communication and behaviour. This postulated link between lowered emotional arousal and impaired empathy levels may in turn account for poor psychosocial outcomes in the adult TBI population.

Anosognosia, or impaired awareness of deficits, is commonly observed in this population. This impairment can result in underreporting difficulties with interpersonal interactions and emotional control post-injury (Prigatano, 2005), highlighting the need for a sensitive and objective measure of TBI-related deficits. Arousal plays a major role in the majority of theories of emotion (James, 1884; Lange, 1887), since it is thought to direct one's own emotional expression, as well as the evaluation of, and behavioural response to emotionally-salient events in the environment. A lack of consensus in the empirical literature, however, exists, as to what constitutes an accurate measure of emotionally produced physiological arousal (Critchley, 2009; Bradley et al., 2008; Vianna and Tranel, 2006). Inconsistent findings on putative indices of arousal (e.g., skin conductance, heart rate, respiration, and pupil dilation) is thought to arise from poor definition of arousal as a construct (Oken et al., 2006). In distinguishing arousal from phasic task-related "activation", Barry et al. (2005) maintain that skin conductance level (SCL) provides an objective 'gold-standard' measure of the relatively slow, longer-lasting state changes that are commonly associated with levels of arousal (Barry et al., 2005; Barry and Sokolov, 1993; Malmo, 1959; Nagai et al., 2004; Raine et al., 2000; Rushby and Barry, 2007), as well as valence effects over time (enhanced for angry versus happy face viewing, McDonald et al., 2011).

Moreover, research by Barry et al. (e.g., Barry et al., 2005, 2007, 2008) has consistently demonstrated an inverse relationship between SCL and alpha wave band activity, specifically mean alpha power, recorded via electroencephalogram (EEG). Based on this research, EEG alpha power shows promise as an electrophysiological index of arousal disturbance following TBI, which may be useful in measuring not only 'global' arousal levels (as is possible with SCL), but also map out topographical or region-specific disturbances in arousal, useful in highlighting patterns of neuropathology in TBI. In a related study, we examined SCL and alpha power, using an eyes-closed/eyes-open paradigm in a group of adults with TBI (Rushby et al., 2013). TBI participants had reduced SCL overall compared with matched controls, and significantly reduced scalp-wide alpha suppression when shifting from the eyes-closed to the eyes-open condition, as well as excess alpha hemispherically compared to controls' midline dominance. These findings were taken to indicate diminished arousal changes, and hypoarousal in the hemispheric extremes, and are consistent with the global and focalised neuropathology of TBI (Singh and O'Dell, 2006). Our previous findings (Rushby et al., 2013) established that patterns of alpha power and SCL were able to differentiate between TBI participants and matched controls during resting states. Based on these findings, the present study aimed to determine whether alpha power may also be used to index autonomic arousal disturbances in a novel context, that is, in response to neutral and emotional facial expressions. This represents an important extension to our previous study (Rushby et al., 2013) since it aims to assess the utility of alpha power by shedding light on the nature of dysregulated arousal mechanisms not only during basic resting states (as per Rushby et al., 2013) but also in more complex affective contexts involving stimuli (emotional face viewing) that are more likely to evoke the kind of dysregulated responding that can lead to problems in social functioning after TBI.

This study also aimed to establish the relations between TBI-related brain atrophy and changes in emotional arousal regulation post-injury. While evidence supports mean alpha power and SCL as electrophysiological indices of arousal, neuropsychological models of emotion posit that ventral system structures, including the insula and amygdala bilaterally, mediate the perception of emotional stimuli, as well as the production and awareness of affective and autonomic responses to those stimuli. Both these structures are vulnerable to damage in TBI (Shamay-Tsoory and Aharon-Peretz, 2007). The insula has an established role in the representation and regulation of emotional arousal and feelings (Singer et al., 2009), by acting as a neural hub for the integration of information about internal bodily states into the subjective experience or awareness of emotion and feelings (Craig, 2002). Supporting this assertion, Bernston et al., (2011) found that patients with lesions to the insula self-reported attenuated arousal and valence ratings of unpleasant and pleasant picture stimuli (IAPS), compared to a control group of lesion patients. Similarly, evidence from neuroimaging studies suggests that enhanced activity in the insular cortex is associated with physiological arousal levels, both during eyes-closed and eyes-open resting states (Goldman et al., 2002; Sadato et al., 1998), and while engaged in a decision-making task (Critichley et al., 2000).

The amygdala, on the other hand, is traditionally implicated in (negative) emotion perception and expression, specifically fear (Lindquist et al., 2012); however, other literature proposes that it has an alternative functional role: processing an emotion's intensity or arousal (Hamann et al., 2002; Kensinger and Schacter, 2006), or salience (Adolphs, 2010), irrespective of valence. In line with this latter view, a recent review of the empirical literature (Lindquist et al., 2012) summarises findings demonstrating that increased amygdala activation accompanied the perception or experience of *any* highly arousing or intense emotion (angry, disgust, fear). Further, when positive and negative stimuli are matched for arousal levels, the amygdala does not show preferential activation to negative valence, thus reinforcing the notion that activation indexes emotional arousal and is not valence-specific (Kensinger and Schacter, 2006). Taken together, it follows that acquired damage to the insula and amygdala, including injury-related volume loss, is likely to be accompanied by abnormalities in arousal and responsivity to emotional stimuli (Bernston et al., 2011), of the kind seen in TBI (de Sousa et al., 2010).

In sum, the present study aimed to investigate whether EEG alpha power and SCL index dysregulated emotional arousal after TBI, as well as examine the contributions of brain volume loss (atrophy) in related structures. It was hypothesised that:

(i) SCL and alpha power are sensitive to arousal changes from a resting baseline to an emotionally-aroused state (EC vs. happy/angry). These changes are reflected in increases on SCL and decreases on alpha power.
(ii) SCL and alpha power are sensitive to general emotion effects (i.e., neutral vs. happy/angry faces) and specific emotion effects (i.e., happy vs. angry), with increases on SCL and decreases on alpha power to emotional faces (happy/angry) in general, and to angry faces versus happy (anger being not only negative but highly arousing in contrast to happy).
(iii) People with TBI demonstrate reduced arousal in response to emotional material as indexed by both lower SCL and heightened alpha power. Consistent with prior research this is expected to be especially the case for angry expressions compared to happy.
(iv) Reduced brain volume in the bilateral amygdalae and insular cortices after TBI shows relationships with the arousal variables at baseline (SCL and alpha power during eyes-closed condition), and in response to emotionally-salient stimuli (SCL and EEG alpha power, while viewing neutral, happy, and angry facial expressions). However, no such relationships emerge with non-pertinent brain structures, namely the bilateral hippocampi.