Overview

Multidisciplinary research is increasingly focused on identifying potential biological markers of posttraumatic stress disorder (PTSD). Convergent evidence leads to theoretical models of a dysregulation in medial prefrontal inhibitory and limbic arousal networks in PTSD. This symposium provides insight into medial prefrontal and limbic function by examining cognitive, affective and clinical processes in PTSD using event-related potential and functional magnetic resonance imaging methodologies. Working memory and inhibitory processing will be explored in cognitive paradigms, fear processing will be examined in an affective paradigm and neural correlates of treatment response and clinical subtypes of PTSD (dissociation) will showcase clinical applications of this research.

04-01

Abnormal recruitment of brain networks during trauma-neutral verbal working memory processing in PTSD

KA Moores¹, CR Clark¹, AC McFarlane², GC Brown³, A Puce⁴, DJ Taylor³

¹Cognitive Neuroscience Laboratory, School of Psychology, Flinders University, Adelaide, South Australia, Australia; ²The Centre of Military and Veterans' Health and Department of Psychiatry, The University of Adelaide, Adelaide, South Australia, Australia; ³MRI Suite, Department of Radiology, Royal Adelaide Hospital, Adelaide, South Australia; Australia; and ⁴Center for Advanced Imaging, Departments of Radiology, Neurobiology & Anatomy, West Virginia University School of Medicine, Morgantown, West Virginia, USA

Post-traumatic stress disorder (PTSD) is characterized by disturbances in concentration and memory, symptoms that are a source of further distress for patients. Abnormalities in working memory (WM) updating have been identified in PTSD (Clark et al. 2003), indicating dysfunction in left hemisphere brain regions critically involved in WM updating. However, it remains unclear whether this finding is because of underlying abnormalities in WM systems in PTSD. Functional magnetic resonance imaging was done for 13 patients with severe PTSD and matched nontraumatized controls, during WM performance where participants either maintained or continually updated verbal stimulus material in separate conditions. The PTSD group failed to show differential activation during WM updating, instead showing abnormal recruitment of WM updating network regions during WM maintenance. These regions included bilateral dorsolateral prefrontal cortex and inferior parietal lobe. Several

other regions were abnormally decreased during WM updating in PTSD including the hippocampus, anterior cingulate and brainstem pons. These results suggest compensatory recruitment of WM networks normally only deployed during updating, which may be linked to the abnormally decreased activity in PTSD during WM updating in other key regions, regions that have been consistently implicated in the neurobiology of PTSD. These abnormalities reflect the difficulty patients with PTSD have engaging with their day-to-day environment.

04-02

Topography of event-related potentials to visuoverbal working memory updating and target detection in PTSD

MD Veltmeyer¹, CR Clark¹, AC McFarlane², RA Bryant³, E Gordon⁴

¹Cognitive Neuroscience Laboratory and School of Psychology, Flinders University of South Australia, Adelaide, Australia; ²Department of Psychiatry, The University of Adelaide, and The Centre of Military and Veterans' Health, Adelaide, South Australia, Australia; ³School of Psychology, University of New South Wales, Sydney, New South Wales, Australia, and The Brain Dynamics Centre; and ⁴The Brain Resource International Database and Brain Resource Company, Ultimo, Australia, and The Brain Dynamics Centre, Westmead Millennium Institute, Westmead Hospital and Western Clinical School, University of Sydney, Australia

This study examined the topography of event-related potentials (ERPs) during working memory updating in medicated (n = 14) and unmedicated (n = 20)groups of patients with PTSD and age- and gendermatched controls. ERPs were recorded from 26 scalp sites during a working memory paradigm that involved identifying when a letter appeared twice in a row in a series of letters. A large positive component at around 400 ms (P3wm) following presentation of nontarget stimuli was considered an index of working memory updating. Group differences were found in the amplitude of this component and also in P3 amplitude and latency following target stimuli. Contrary to expectations, these effects were most apparent in the medicated subgroup. Both groups of patients with PTSD exhibited delayed reaction time, but only the medicated participants were impaired in target detection accuracy. Neither ERP nor behavioural abnormalities were related to CAPS symptom scores. These results are consistent with research that suggests SSRI medication may alter working memory performance, but the results may be due to some other characteristic of the medicated participants such as differing symptom profiles.