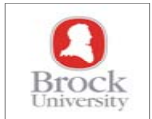




MEMORY PERFORMANCE AS A FUNCTION OF ANXIETY FOR INDIVIDUALS WITH MILD HEAD INJURY



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BACKGROUND

Little research has been conducted to examine the chronicity of cognitive and emotional sequelae following mild head injury (MHI) which can be much less subtle and involve alterations in consciousness without extensive neural loss¹.

Typically it has been suggested that post-concussive symptoms² (i.e., cognitive difficulties, altered affect, and physical complaints) subside after a three month period and functioning is assumed to return to previous abilities^{2,3}. Yet, for some, 15%⁴, these difficulties may not be transient^{5,6,7}.

Furthermore, it has been suggested that individuals with moderate to severe neurological compromise are particularly vulnerable to the adverse effects of stress and flattened affect/arousal¹⁰. Yet with respect to subtle head injuries (i.e. MHI), the findings have been, at best, variable¹¹, at worst, inconclusive, and otherwise silent with respect to arousal levels.

GENERAL RESEARCH QUESTIONS

• Do individuals with MHI have differential arousal from individuals without MHI?

• Do individuals with self-reported MHI have differential cognitive performance that interacts with arousal state (via self-report or manipulated stress or relaxation)?

METHOD

STUDY 1

Participants

University students ($N = 50$); 30% reported history of MHI

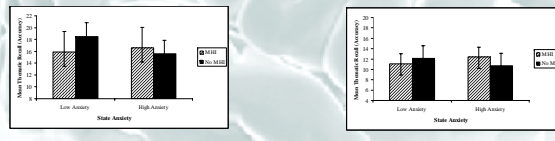
Measures and Procedure

• Standardized neuropsychological tests (memory^{12,13}), questionnaires¹⁴ and self-reported anxiety¹⁵, and a structured interview were administered.

RESULTS: STUDY 1



Immediate, $F(1, 48) = 4.02, p = .050$, and delayed, $F(1, 48) = 6.61, p = .013$, narrative recall performance of individuals with and without MHI differed as a function of state anxiety.



Similarly, recall ability for thematic material for individuals with MHI and without MHI varied as a function of state anxiety for both immediate, $F(1, 48) = 3.85, p = .056$, and delayed recall, $F(1, 48) = 3.93, p = .054$.



Time required for completion of the delayed reproduction (after a minimum 30 minute delay), but not immediate reproduction, of the RCF did vary significantly between MHI and no MHI groups as a function of state anxiety, $F(1, 48) = 4.47, p = .040$.

Most interestingly, individuals with MHI reported significantly less anxiety than individuals without, $F(1, 48) = 4.23, p = .045$.

METHOD: STUDY 2

Participants

University students ($N = 56$); 55% self-reported MHI history

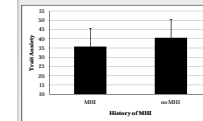
Measures and procedure

• Neuropsychological measures (memory^{12,16}; attention¹⁷; planning¹⁷; abstract reasoning^{16,17,18}; and standard intelligence¹⁶); indices of arousal/anxiety¹⁵; manipulated arousal (via psychosocial stressor¹⁹ or relaxation)

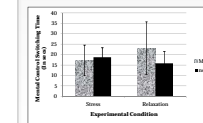
Indices of Anxiety

• physiological recordings of heart rate, respiration, and electrodermal responsivity²⁰; STAI¹⁵; self-reported arousal levels; Everyday life stress¹⁴

PRELIMINARY RESULTS: STUDY 2



Similar to Study 1, persons with self-reported MHI reported less anxiety, $F(1, 54) = 5.228, p = .026$, (i.e. underaroused).



As expected, persons without MHI take longer to complete the task when stressed than when relaxed. In contrast, persons with MHI are faster when stressed than when relaxed, $F = 4.025, p = .05$.

With respect to physiological measures of arousal²⁰, although the MHI group and no-MHI group respond similarly to the psychosocial stressor or relaxation task; anecdotally, individuals with MHI do not appear to maintain the manipulated arousal state for as long as individuals without MHI.

CONCLUSIONS

These findings indicate the potential influence of underarousal that has found to be associated with orbitofrontal disruption and may be implicated in MHI generally. Further, the neurological and emotional sequelae following MHI may not be transient despite both the subtle nature of the head trauma and the competency of the individuals involved (e.g. university students).

The results demonstrate that sustaining a MHI is predictive of persistent deficits in cognitive functioning which is differentially influenced by reported and measured levels of arousal/anxiety.

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