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Acute motor, neurocognitive and neurophysiological change following concussion injury in Australian amateur football. A prospective multimodal investigation

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ABSTRACT

Objectives: This multimodal study investigated the motor, neurocognitive and neurophysiological responses following a sports related concussion injury in the acute-phase (up to 10 days) in sub-elite Australian football players.
Design: Between-group, repeated measures.
Methods: Over the course of one season (six months), 43 male players from one football club (25.1 ± 4.5 years) were assessed for fine motor dexterity, visuomotor reaction time, implicit learning and attention. Motor cortex excitability and inhibition were assessed using transcranial magnetic stimulation.
Results: Of the 43 players, eight suffered concussion injuries, and were compared to 15 non-concussed players (active control) who returned for follow up testing. Post-concussion assessments using the aforementioned tests were carried out at 48 and 96 h, and 10 days. Compared to the non-concussed players, those who suffered concussion showed slowed fine dexterity ($P = 0.02$), response ($P = 0.02$) and movement times ($P = 0.01$) 48 h post-concussion. Similarly, attentional performance was reduced in the concussed group at all time points (48 h: $P < 0.01$; 96 h: $P < 0.01$; and 10 days: $P = 0.02$) post-concussion. TMS revealed significantly increased corticospinal inhibition at 48 ($P = 0.04$) and 96 h post concussion ($P = 0.02$) with significant correlations between increased corticospinal inhibition and response ($r = 0.48$; $P < 0.01$), movement time ($r = 0.42$; $P = 0.02$), and attention performance ($r = 0.44$; $P = 0.01$).
Conclusions: This study has demonstrated that acutely concussed Australian football players show abnormalities in motor, cognitive and neurophysiological measures with variable rates of recovery. These findings suggest that measuring the recovery of concussed athletes should incorporate a range of testing modalities rather than relying on one area of measurement in determining return to play.

1. Introduction

Concussion is the most common type of traumatic brain injury (TBI).[1] Frequently reported in contact sports, sports-related concussion is a biomechanically induced neurological injury; resulting in, but not limited to, headaches, nausea, visual disturbances, fatigue, and transient alterations in mental status (for example disorientation or amnesia), and motor control (loss of balance and unsteadiness) that may or may not involve loss of consciousness, which is reported in between 10% and 20% of cases.[2]

Investigating the progress of neurologic dysfunction and recovery of sports-related concussion in the acute phase following injury (i.e. up to 10 days), is important in determining when an athlete (of any standard of play) can return to training and competition. Recovery following sports concussion has traditionally been assessed via symptom observation, balance performance and cognitive testing.[3] Questions, however, remain regarding the underlying neurophysiological responses following a concussion injury, particularly when athletes may not or no longer show clinical signs or cognitive impairment. Shaw proposed that concussion causes an acute physiological disturbance in the apparent absence of any neuropathology,[1] however more recent neuroimaging evidence has presented acute diffusivity changes in white matter located in corpus callosum and corticospinal tracts.[4] Therefore, electrophysiological methods have the potential to be an appropriate means

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to gain insight into the acute pathophysiological processes of concussion. However, neurophysiological studies in humans, using electrophysiological methods such as electroencephalography and evoked potentials, are currently limited.

Neurophysiological studies using event-related potentials, via electroencephalography (EEG), have demonstrated subclinical cognitive attention and working-memory performance deficits in otherwise asymptomatic long-term concussed athletes.[5,6] Despite the ability of EEG to provide electrophysiological data on brain activity, the technique has some limitations for application to the sporting club environment; for example, extensive pre-testing preparation time and low signal-to-noise ratios limit the opportunity to test outside of the clinical laboratory.[7,8] Alternatively, the motor system, which is more amenable to such testing, could be similarly affected following a concussive injury.[9] Transcranial magnetic stimulation (TMS) with electromyography (EMG) allows for measures of corticospinal excitability and intracortical inhibition.[10] Corticospinal excitation is measured by the amplitude of the motor evoked potential (MEP), whilst intracortical inhibition is measured by the cortical silent period (cSP) duration, reflecting γ-aminobutyric acid ($GABA_B$) receptor activity, from single pulse TMS. In addition paired-pulse TMS measures, such as short-interval intracortical inhibition (SICI), can investigate $GABA_A$ intracortical inhibition activity. TMS has been used extensively in the investigation of neurological disorders such as stroke, Parkinson's disease and multiple sclerosis[10], as well as those with long-term cognitive and motor impairments with concussion sustained in early adulthood.[11,12] Specifically brain injury, Christyakov et al.[13] and Miller et al.[14] showed increased intracortical inhibition in mild to severe TBI patients two weeks following injury. Similarly, De Beaumont et al.[9] showed increased intracortical inhibition in previously concussed collegiate athletes with repeated concussions. More recently Livingston et al., demonstrated reductions in corticospinal excitability and a slowing in central motor conduction time, along with reduction in processing speed in neurocognitive tests, following a sports-related concussion in North American collegiate athletes up to 10 days post-injury.[15] Collectively, these studies[9,15] demonstrate that TMS may be sensitive to physiological disturbances following TBIs including concussion.

To date there are no multi-modal acute time-course studies that have incorporated cognitive, motor, and evoked potential measures following a sports concussion. This preliminary investigation presents fine dexterity and reaction and movement time (motor) and implicit learning and attention (cognitive) as well as corticomotor excitation and inhibition (TMS evoked potentials) in Australian football players at 48 and 96 h, and 10 days following acute injury.

2. Methods

Forty-three males (mean age 25.1 ± 4.5 years; mean height 183.7 ± 7.9 cm; mean weight 86.9 ± 9.9 kg) who regularly compete in a divisional amateur Australian Football league were recruited for the study. All participants were from the one club with players participating across three grades within their division. Consent from individual participants and the club was obtained prior to data collection, with all methods approved by the University Human Research Ethics Committee.

Participants completed pre-screening for prior concussion history and TMS suitability. Those who had sustained a concussion injury within 12 months prior, were diagnosed with a neurological or psychiatric condition, were currently on prescribed medications for a neurological or psychiatric condition, or had cranial metal implants or a neuro-stimulator were excluded from participation.

All testing procedures (including pre-screening, concussion history, neuropsychological and fine motor control testing, and TMS) were completed in one 60 min laboratory visit. If players sustained a concussion they were brought in for 3 visits at 48 h, 96 h and 10 days post injury. Players who did not sustain a concussion (active controls) visited the laboratory three times within a two-week period, completing the same tests, at the end of the season.

Fine motor control was tested using the O'Connor Finger Dexterity test (Lafayette Instrument, USA). This well-established test,[16] with good-to-excellent predictive validity,[16,17] requires the manipulation and placement of three small pins into each hole. We used a modified form of the O'Connor Test by measuring the time it takes to place three pins into one hole on three rows of the board (30 holes in total).[18] Due to learning effects,[16,18] participants familiarised themselves prior to actual assessments.

Visuomotor reaction time assessed the participant's ability to respond and move to a stimulus displayed on the computer screen (Cambridge Cognition, UK). Upon seeing the stimulus appear on the screen participants were instructed to release the press-pad key and touch the stimulus on the screen as quickly as possible. The outcome measured was a response time (stimulus presentation to release of keypad) and movement time (release of keypad to screen touch).[19]

Participants were assessed for cognitive functioning and attention via paired-associative learning (PAL), and intra-extra dimensional (IED) set shift, using standardised tasks from the Cambridge Neuropsychological Test Automated Battery (Cambridge Cognition, UK).[19] Tests were computerised, non-linguistic, culturally neutral and previously validated in a number of population groups, including concussion.[19] PAL required participants to learn the location of coloured-patterned shapes hidden behind white boxes revealed in randomised order.[19] IED tests visual discrimination and shifting and flexibility of attention by displaying two simple colour-filled shapes, with participants learning which one is correct and maintaining the correct response for six consecutive trials.[19]

Using well-established methods[20–22] TMS was applied over the contralateral motor cortex area projecting to the participants first dorsal interosseous (FDI) muscle. Following the surface EMG for non-invasive assessment of muscles (SENIAM) skin preparation and electrode placement recommendations,[23] the skin was shaved (if necessary) and alcohol swabs were used to clean the area of the skin, prior to electrode placement. Surface EMG (bipolar Ag-AgCl) electrodes with an inter-electrode distance of 20 mm were placed over the FDI muscle.[23] EMG signals were amplified (1000×), filtered (10–1000 Hz), and sampled at 2 kHz[23], recording 500 ms responses of the participant's dominant hand (PowerLab 4/35, ADInstruments, Australia). All TMS procedures adhered to the TMS checklist for methodological quality.[24]

Active and resting motor evoked potentials (MEPs) were obtained using a Magstim 200^2 stimulator (Magstim Co, UK) with a figure-of-eight D70^2 coil (external loop Ø105 mm). For reliability of coil placement participants wore a snugly fitted cap (EasyCap, Germany), positioned with reference to the nasion–inion and interaural lines. The cap was marked with sites at 1 cm spacing in a latitude–longitude matrix to ensure reliable coil position throughout the testing protocol.[20,21]

At the location of the "optimal site", where the largest MEP could be observed, the active motor threshold (aMT) was quantified, during a controlled, low-level voluntary contraction of the FDI muscle at 10% of Maximal Voluntary Contraction (MVC); or resting motor threshold (rMT) was determined in a relaxed muscle. Motor threshold was identified by delivering TMS stimuli from a level below the estimated participant's threshold, at increasing intensities of 5%[25] then 1% steps of stimulator output, until an MEP of at least 200 μV and associated cSP for aMT, and at least 50 μV in rMT, could be measured in at least five of the 10 stimuli. For main studies 15 sweeps were taken at 125% of aMT or rMT (30 pulses in total), and stimuli