Traumatic brain injury – A time to reappraise – a wake-up call

Roy G. Beran

Abstract

Mild traumatic brain injury (mTBI) is becoming topical with potential association with post-traumatic stress disorder (PTSD) and dementia and questions are being raised for the Defence Forces. Traumatic brain injury results from head trauma causing altered consciousness or post-trauma amnesia and may be categorised as mild, moderate or severe. It has been recognised since World War I and has re-emerged as a source of concern in sports men and women with potential litigation becoming a reality. Its association with PTSD, cognitive dysfunction, depression and other neurological symptoms is being increasingly recognised with new imaging techniques demonstrating pathology not identified in standard CT and MRI. In June 2012, a U.S. class action for 2.500 football players was initiated and has the potential to be the forerunner of similar actions within the Defence Forces.

Introduction

Issues related to mild traumatic brain injury (mTBI) barely raised a ripple on the surface a little more than a decade ago. Since then there has been a groundswell of interest and investigation which has the potential to become a veritable tidal wave, which may cause a tsunami, the impact of which will be felt for many decades to come.

The connection between mTBI and such conditions as post-traumatic stress disorder (PTSD) or Alzheimer's Disease (AD) are yet to be fully appreciated.¹⁻³ The legal medicine ramifications for organisations which hold vicarious liability for those working within them, or for them, has not yet fully bubbled to the surface.⁴ When it does, it will create waves that most people in authority have chosen to ignore. There was a preliminary approach, by Joint Health Command (JHC), for the Australian Defence Forces (ADF), to create both a centre of excellence for the study of mTBI and to establish a legal medicine service operating within JHC but neither eventuated. The question arises, "Should they happen now or is it already too late?".

What follows is a very brief overview of some of the ramifications and consequences of mTBI which should act as a wake-up call, leaving it to the reader to decide the answers to the above questions.

Definition

Traumatic brain injury (TBI) is defined as head trauma resulting in altered or lost consciousness or the experience of post-traumatic amnesia.⁵ Military personnel experience TBI with falls, motor vehicle accidents and blasts.¹ Blast-related TBI may be

associated with barotrauma, consequent to dynamic changes in atmospheric pressure, or blast forces causing direct impact as a result of provoking the head to hit, or be hit by, foreign blunt or penetrating objects.⁶ Blunt force TBI is classified by severity into mild, moderate or severe with mTBI being the most frequently encountered,¹ especially in serving military people.⁷

Some Historical Perspectives

Potential consequences of mTBI are not a new realisation, even within the modern era of medicine, with preliminary commentary dating back almost a century,⁸ to World War I. The newer realisation that blast injuries are associated with serious consequences re-emerged after World War II⁹⁻¹¹ with a better appreciation of the neuronal consequences following rapidly from that knowledge.¹²⁻¹⁴

There has been a significant Australian connection providing a better delineation of the neuropathology associated with mTBI, through the contribution of Blumbergs and colleagues.¹⁵⁻¹⁷⁾ His focus on amyloid precursor protein should have sent warning bells ringing for the later understanding of the degenerative consequences that are provoked by mTBI, as has emerged in sports men and women.¹⁸⁻²² Again this has had a significant Australian connection, looking particularly at Australian football players and the consequences of concussive head injuries.²³⁻³⁰ McCrory also examined the American Association of Neurologists (AAN) guideline,21 related to head injuries and sport, and concluded there remains a paucity of scientifically evidence-based guidelines for sports people,³⁰ which bodes poorly for military personnel.

While military conflict is not the favoured weekend spectator activity, sport is! The realised serious consequences of mTBI in sport have triggered legal medicine ramifications with a class action against the National Football League (NFL) and the helmet manufacturers in the US.⁴ The consequences of this, for the ADF, are yet to emerge.

In a critical review of the prognosis of mTBI, a decade ago, the authors wrote, "... For adults, cognitive deficits and symptoms are common in the acute stage, and the majority of studies report recovery for most within 3 - 12 months. Where symptoms persist, compensation/litigation is a factor ...".³¹

It is argued that a decade later there continues to emerge epidemiological data attesting to the serious consequences of mTBI. There is evidence that mTBI independently increases the risk of ischaemic stroke by more than 30%, according to a study of over 1 million trauma patients.³² With this type of evidence emerging, the time for cavalier disregard for the consequences of mTBI would seem quite inappropriate.

The same group who raised the spectre of "compensation/litigation", a decade ago,³¹ have also examined the inherent costs, at that time, without definitive findings and concluded, "... The sparse scientific literature in these areas reflects both conceptual confusion and limited knowledge of the natural history of mild traumatic brain injury ..."³³ While this may have been true a decade ago, the emerging data suggest one can no longer ignore what seem to be established sequelae of mTBI and what also appear to be unequivocal foreseeable risks, with definite implications for duty of care.

Overview of mTBI

As stated earlier, much of the current understanding of mTBI has emerged from investigation of sportsrelated concussion. Investigation of almost 3,000 US College footballers, between 1999 and 2001, covering ~ 4250 play-seasons, revealed 184 (6.3%) had concussion, of whom 12 (6.5%) had repeated concussion in the same season.¹⁸ An association was found between reported number of previous concussions and the likelihood of the incident concussion, suggesting a subsequent predisposition.¹⁸

Players, with at least 3 previous concussive episodes, were three times more likely to have the incident concussion, provoking inclusion within the study.¹⁸ Headache was the most common symptom with mean duration being 82 hours, with slow recovery following multiple concussions (30% who experienced at least 3 concussive episodes took more than one week).¹⁸

Major depression has been recognised in association with mTBI and may influence prognosis.³⁴ Seventyfour patients with TBI were assessed, of whom 21 (28%) met criteria for major depression, being older than those without (41 v 32 years; p < 0.01) and more likely to have a past history of depression (24% v 6%; p < 0.05).³⁴ There was no other difference regarding gender, educational status, marital status, employment, previous TBI, premorbid intelligence, family psychiatric history or past substance or alcohol use.³¹ Mechanism of injury, other injuries, CT scanning, Glasgow Coma Scale scores and duration of post-traumatic amnesia were also similar between both groups.³⁴

At six months post TBI, major depression was associated with worse performance on tests for attention, memory and executive function. While > 50% of those without depression had no impairment of cognition, this was only true of 15% of patients with major depression.³⁴

There is further evidence for the association of mTBI and PTSD in ~30-40% of those with mTBI. Of more than 2,500 US troops returning from Iraq, ~ 5% reported loss of consciousness, of whom ~ 44% met criteria for PTSD.³⁵ Soldiers with mTBI, especially with loss of consciousness, had greater risk for poor general health, missed days from work, medical consultations and increased somatic and post-concussive symptoms, although adjusting for PTSD and depression resulted in removing these as significant factors³⁵ other than the presence of headaches.³⁵

Taber et al³⁶ examined blast related TBI and noted the common occurrence of soldiers becoming dazed or unconscious following an explosion, which caused no external evidence of injury but provoked associated prograde or retrograde amnesia. Almost 60% of the "at risk" group of injured soldiers, returning from Iraq or Afghanistan, to the Walter Reid Centre (2003 - 4) had at least mTBI while in combat with TBI consequent to closed head injury occurring in ~ 90%.³⁶ More than 50% (665/1303) with only lower limb explosive injuries had neurological symptoms, including headache, insomnia, psychomotor agitation and vertigo, consistent with TBI.36 Of these, a third (36%) showed electroencephalographic abnormalities with ongoing symptoms.36 The TBI is thought to result from secondary and tertiary blast injuries.³⁶ Comparing blast and non-blast mTBI subjects, concussive symptoms, psychological symptoms and neurocognitive performance within 72 hours of exposure, showed no real differences.³⁴ Both groups showed clinically significant impairment of cognitive reaction time, thought to relate more to duration of loss of consciousness rather than injury

mechanism.37

Among surviving soldiers, wounded in combat in Iraq and Afghanistan, TBI accounts for a greater proportion of casualties than was the case in previous conflicts.³⁸ While there appeared to be some remarkable recoveries in the relatively short term,³⁸ as reflected by return of function, the longterm consequences are extremely guarded when interpreted in the light of the evolving data related to mTBI in sports men and women.

Nature of Injury and Investigations

The recognition of axonal damage following head injury is not new¹² with techniques using magnetic resonance imaging (MRI) employing diffusion tensor imaging (DTI), are providing additional confirmation consistent with traumatic axonal injury.³⁹

Not all those with mTBI show abnormality on DTI, thus confirming mTBI as a clinical diagnosis.39 Bazarian et al⁴⁰ have explored the numerous investigative tools used to examine brain injury following concussion. These include sophisticated MRI with functional imaging or DTI (as cited above), single photon emission computer tomographic scanning, serum biomarkers, formal cognitive and balance tests and positron emission tomography.⁴⁰ Micro-structural white matter lesions are detectable by DTI and correlate with cognitive deficits identified in mTBI, when other techniques fail to do so.⁴¹ A recent case, presented to the Australian Defence Force Reserves NSW Health Triumvirate⁴² demonstrated convincing evidence of a soldier presenting with mTBI, complaining of headache, with normal CT scan of the brain and 1.5 Tesla MRI of the brain, who had clearly demonstrable evidence of an intracranial bleed using 3 Tesla MRI, with the addition of susceptibility weighted imaging.

It follows that the evolution of greater technology and the capacity for better imaging techniques must be considered when the clinical picture suggests mTBI. Without such an approach, it could be argued patients were provided suboptimal care within the context of current knowledge.

Legal Medicine Considerations

In June 2012, a class action for more than 2,500 football players was initiated against the US NFL and helmet manufacturer Riddell Inc. in the US District Court for the Eastern District of Pennsylvania.⁴ Causes for damage included: wrongful death; several varieties of fraud; negligence – referenced over different periods (pre-1968, post-1968, 1987-93 and post-1994); plus negligent hiring and retention.⁴ In addition, the plaintiffs alleged several strict product

liability claims, including design and manufacturing defects, failure to warn and general negligence regarding Riddell Inc.⁴ The plaintiffs also alleged a civil conspiracy jointly against both defendants.⁴

An underlying issue, in the NFL concussion litigation, is the notion that the NFL did not provide sufficient resources to accommodate the long-term health needs of its retired players.⁴ In 2002, Bennet Omalu, a forensic pathologist, conducted autopsies on former NFL players and found evidence of chronic traumatic encephalopathy similar to that found in former boxers⁴ and claimed that a single episode of mTBI could take months, rather than hours or days, to recover.⁴ The role of amyloid precursors, in mTBI damage, has been reported almost 20 years ago¹⁴ and these same precursors are recognised to be involved in so-called tau pathology thought to be a consequence of chronic traumatic encephalopathy.⁴ There has been evidence, since 1980 that these proteins remain in the brain for at least 3 months, thereby suggesting that the paucity of symptoms, associated with post-concussive status, need not equate to the brain having recovered from a concussive episode.4

It is important, within the ADF, to appreciate that there are many more potential litigants re mTBI than might have been involved in the NFL, resulting in a class action. There is far more potential for serious and repeatedly significant exposure to blast and concussive head injury.

Conclusions

Appreciation of the consequences of mTBI is gaining momentum with sports people leading the charge. There is evolving litigation, especially in the U.S., and sporting clubs are considering the consequences of allowing those so exposed to continue participating within the sporting activities. This raises questions regarding a need to provide better 'duty of care'.

The history of mTBI is not new, within the military context, with very important publications dating back 100 years to World War I and re-emerging after World War II. It follows that no one could claim ignorance of what is gaining greater recognition.

Our understanding of mTBI is growing on a daily basis but still requires a real focus to better delineate what might be expected and what kind of services and intervention may be required to adequately meet the expectations of serving personnel, both within the present climate and into the future.

It is argued that the litigation being undertaken against the NFL and helmet manufacturers is only the tip of an iceberg, and is reflective of U.S. experience which may not directly translate to Australia. Despite this disclaimer, such litigation may have far greater ramifications for the military environment than it does in sport. This realisation should cause those in authority to reflect upon what is happening and what may happen into the future. Author's affiliation: Professor, School of Medicine, Griffith University, Conjoint Associate Professor University of New South Wales, Principal: Strategic Health Evaluators Sole and Corresponding author: CMDR Roy G Beran RANR, Suite 5, Level 6, 12 Thomas Street, Chatswood, New South Wales, AUSTRALIA, 2067 Email: roy@royberan.com

References:

- 1. Carlson KF, Kehle SM, Meis LA, et al. Prevalence assessment and treatment of mild traumatic brain injury and post-traumatic stress disorder: A systematic review of the evidence. Journal of Head Trauma Rehabilitation 2011; 26(2): 103-115.
- 2. Bryant RA, Harvey AG. Relationship between acute stress disorder and post-traumatic stress disorder following mild traumatic brain injury. The American Journal of Psychiatry 1998; 155: 625 629.
- 3. Mehta KM, Ott A, Kalmijn S, et al. Head trauma and risk of dementia and Alzheimer's disease: The Rotterdam Study. Neurology 1999; 53: 1959 1962.
- 4. Goldberg DS. Mild traumatic brain injury, the National Football League, and the manufacture of doubt: An ethical, legal and historical analysis. The Journal of Legal Medicine 2013; 34: 157 191.
- 5. Atlanta GA. Traumatic Brain Injury in the United States: A Report to Congress. Centres for Disease Control and Prevention, 1999.
- 6. Warden D. Military TBI during the Iraq and Afghanistan wars. Journal of Head Trauma Rehabilitation 2006; 21(5): 398 402.
- 7. Terrio H, Brenner LA, Ivins BJ et al. Traumatic brain injury screening: preliminary findings in a US Army Brigade combat team. Journal of Head Trauma Rehabilitation 2009; 24(1): 14 23.
- 8. Mott FW. The microscopic examination of the brains of two men dead of commotio cerebri (shell shock) without visible external injury. Journal of the Royal Army Medical Corps 1917; 29: 662 677.
- 9. Fabing HD. Cerebral blast syndrome in combat soldiers. Archives of Neurology & Psychiatry 1947; 57: 14 57.
- 10. Cramer F, Poster S, Stephenson C. Cerebral injuries due to explosion waves cerebral blast concussion. Archives of Neurology & Psychiatry 1949; 61: 1 20.
- 11. Strich SJ. Diffuse degeneration of the cerebral white matter in severe dementia following head injury. Journal of Neurology, Neurosurgery & Psychiatry 1956; 19: 163 185.
- 12. Strich SJ. Shearing of neurofibres as a cause of brain damage due to head injury. Lancet 1961; ii: 443 448.
- 13. Casson IR, Sham R, Campbell EA et al. Neurological and CT evaluation of knocked-out boxers. Journal of Neurology, Neurosurgery & Psychiatry 1982; 45: 170 174.
- 14. Stuss DT, Ely P, Hugenholtz H, et al. Subtle neuropsychological deficits in patients with good recovery after closed head injury. Neurosurgery 1985; 17: 41 47.
- 15. Blumbergs PC, Jones NR, North JB. Diffuse axonal injury in head trauma.

Journal of Neurology, Neurosurgery & Psychiatry 1989; 52: 838 – 841.

- 16. Blumbergs PC, Scott G, Mancuris J et al. Staining of amyloid precursor protein to study axonal damage in mild head injury. Lancet 1994; 344: 1055 1056.
- 17. Blumbergs PC, Scott G, Mancuris Jet al. Topography of axonal injury as defined by amyloid precursor protein and the sector scoring method in mild and severe closed head injury. Journal of Neurotrauma 1995; 12: 565 572.
- 18. Guskiewicz KM, McCrea M, Marshall SW et al. Cumulative effect associated with recurrent concussion in collegiate football players. The Journal of the American Medical Association 2003; 290(19): 2549 2555.
- McCrea M, Guskiewicz KM, Marshall SW et al. Acute effects and recovery time following concussion in collegiate football players: The NCAA Concussion Study. The Journal of the American Medical Association 2003; 290(19): 2556 – 2563.
- 20. McAllister TW, Flashman LA, Maerlender A, et al. Cognitive effects of one season of head impacts in a cohort of collegiate contact sport athletes. Neurology 2012; 78: 1777 1778.

- 21. Gizza CC, Kutcher JS, Ashwal S et al. Summary of evidence-based guideline update: Evaluation and management of concussion in sports. Neurology 2013; 80: 2250 2257.
- 22. Strain J, Didehbani N, Cullum CM et al. Depressive symptoms and white matter dysfunction in retired NFL players with concussion history. Neurology 2013; 81: 25 32.
- 23. McCrory PR, Bladin PF, Berkovic SF. Retrospective study of concussive convulsions in elite Australian Rules and Rugby League footballers: phenomenology, aetiology and outcome. British Medical Journal 1997; 314: 171 174.
- 24. McCrory PR. Were you knocked out? A team physician's approach to initial concussion management. Medicine & Science in Sports & Exercise 1997; 29(Suppl 7): S207 S212.
- 25. McCrory PR, Ariens T, Berkovic SF. The nature and duration of acute concussive symptoms in Australian football. Clinical Journal of Sport Medicine 2000; 10: 235 238.
- 26. McCrory PR, Johnston KM, Mohtadi NG et al. Evidence-based review of sport-related concussion: basic science. Clinical Journal of Sport Medicine 2001; 11: 160 165.
- 27. McCrory PR, Meeuwisse WH, Johnston K et al. Consensus statement on concussion in sport: The 3rd International Conference on Concussion in Sport, held in Zurich, November 2008. Journal of Clinical Neuroscience 2009; 16: 755 763.
- 28. McCrory PR. Sports concussion and the risk of chronic neurological impairment. Clinical Journal of Sport Medicine 2011; 21: 6 12.
- 29. McCrory PR, Meeuwisse WH, Aubry M et al.. Consensus statement on concussion in sport: The 4th International Conference on Concussion in Sport, held in Zurich, November 2012. British Medical Journal 2013; 47: 250 258.
- McCrory PR. Revisiting the AAN Guidelines on sport-related concussion. Nature Reviews Neurology 2013;
 9: 361 362.
- Carroll LJ, Cassidy JD, Peloso PM et al. Prognosis for mild traumatic brain injury: Results of the WHO collaborating centre taskforce on mild traumatic brain injury. Journal of Rehabilitation Medicine 2004; Suppl 43; 84 105.
- 32. Burke JF, Stulc JL, Skolarus LE et al. Traumatic brain injury may be an independent risk factor for stroke. Neurology 2013; 81(1): 33 – 39.
- 33. Borg J, Holm L, Peloso PM et al. Non-surgical intervention and cost for mild traumatic brain injury: Results of the WHO collaboration centre taskforce on mild traumatic brain injury. Journal of Rehabilitation Medicine 2004; Suppl 43: 76 83.
- 34. Rapoport MJ, McCullagh S, Shamnui P et al. Cognitive impairment associated with major depression following mild and moderate traumatic brain injury. Journal of Neuropsychiatry and Clinical Neurosciences 2005; 17: 61 65.
- 35. Hoge CW, McGurk D, Thomas JL et al. Mild traumatic brain injury in US soldiers returning from Iraq. New England Journal of Medicine 2008; 358: 453 463.
- 36. Taber KH, Warden DL, Hurley RA. Blast-related traumatic brain injury: What is known. Journal of Neuropsychiatry and Clinical Neurosciences 2006; 18(2): 141 145.
- 37. Leuthcke CA, Bryan CJ, Morrow CE et al. Comparison of concussive symptoms, cognitive performance and psychological symptoms between acute blast versus non-blast induced mild traumatic brain injury. Journal of the International Neuropsychological Society 2011; 17: 36 45.
- 38. Okie S.Traumatic brain injury in the war zone. New England Journal of Medicine 2005; 352(20): 2043 2047.
- 39. MacDonald CL, Johnson AM, Cooper D et al. Detection of blast-related traumatic brain injury in US military personnel. New England Journal of Medicine 2011; 364: 2091 2100.
- 40. Bazarian JJ, Blyth B, Cimpell L. Bench to bedside: Evidence for brain injury after concussion looking beyond the computed tomographic scan. Academic Emergency Medicine 2006; 13(2): 199 214.
- 41. Niogi SN, Mukherjee P, Ghajar J et al. Extent of microstructural white matter injury in post-concussive syndrome correlates with impaired cognitive reaction time: A 3T diffusion tensor imaging study of mild traumatic brain injury. American Journal of Neuroradiology 2008; 29: 967 973.
- 42. Beran RG. "Traumatic Brain Injury". Australian Defence Force Reserves New South Wales Health Triumvirate, 2013.