Chronic Traumatic Encephalopathy/Traumatic Encephalopathy Syndrome in Military Personnel

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Introduction

Australian Defence Force (ADF) service members' occupational history, consisting of repeated minor traumatic brain injury (TBI) with accompanying cognitive, behavioural, mood and movement disorders, are cardinal symptoms suggestive of the diagnosis of traumatic encephalopathy syndrome (TES) and chronic traumatic encephalopathy (CTE).

In 2015, Beran[1] published an opinion piece alerting the medical and legal profession that litigation, relating to TBI/CTE in the sporting profession, is on the rise in the US and that litigation initiated by service members injured with TBI is likely to follow that trend. In 2018, Beran and Bhaskar[2] published case reports of two ADF service members exposed to blast injuries and subsequently suffered from learning and memory difficulties and Parkinsonism. These cases were indicative of TES. In 2017, Beran's prediction was realised when the *Kennedy v McCarthy*[3] hearing took place in the US District Court for the District of Connecticut.

Kennedy v McCarthy[3] was a nationwide class action lawsuit initiated by two veterans against McCarthy, the Acting Secretary of the US Army, commenced on behalf of approximately 50 000 war veterans who were discharged dishonourably due to bad tempers amounting to misconduct. These veterans argued that their bad tempers were a consequence of TBI induced mental health injury, such as a post-traumatic stress disorder (PTSD), and that their behaviours may have been caused by a psychiatric condition or brain damage resulting from injuries they acquired during their course of employment as a soldier. The veterans requested their misconduct be re-determined and to set aside the determination to discharge them dishonourably. McCarthy denied these allegations.

In November 2021, the US District Court for the District of Connecticut[4] ordered McCarthy to automatically reconsider the decisions, in certain circumstances, involving cases where there were demonstrable PTSD and/or TBI. As a result, thousands of unfavourable decisions were reviewed and the status of more than 51 400 dishonourably discharged veterans was reversed.[5]

CTE is a topic of significant interest for legal medicine practitioners as, according to the ADF statistics, there are 59 095 service members employed by the ADF, of which 28 878 were full-time and 17 454 part-time.[6] A further 1227 defence personnel were deployed to active operations.[7] Exposure to TBI within the military is not restricted to the battlefield. It may be encountered during training, sporting activities, in various non-combat activities and in the Army Reserve setting. *Kennedy v McCarthy* demonstrated the potential for a significant scale class action lawsuit in Australia, initiated by injured service members, against the ADF.

This paper reviews the medical and the tort of negligence aspects of TES and CTE as may be pertinent to the ADF.

Clinico-pathogenesis of CTE

TBI delivers shearing forces to the brain. The force is predominantly focused at the base of the sulci, at the grey and white matter interface, causing a direct injury to the neural and the glial cells, and damaging the blood–brain barrier.[8] In response to the injury, inflammation ensues and allows the macrophages to enter the brain through the permeable blood–brain barrier8 The activated macrophages and the microglial cells release interferon-gamma, inducing a marked increase in HLA Class 1 protein expression8. This signals phosphorylation of tau proteins (hyperphosphorylation), resulting in the accumulation of the hyperphosphorylated tau (p-tau).[9]

The p-tau is a microtubule-associated protein[10]. It maintains the tubules' stability and structure and

transports macromolecules from the cell body to the distal part of the neuron.[10] Excessive p-tau proteins tangle to form neurofibrillary tangles (NFT).[10] Accumulation of NFTs destabilises the tubules, causing progressive cell dysfunction and death, analogous to lipid storage disease, such as Tay-Sachs disease.[8]

Grossly identifiable changes in the brain are more apparent in advanced CTE but unusual in mild CTE (acknowledging that CTE remains a postmortem diagnosis).[12] Characteristic macroscopic alterations include generalised cerebral atrophy with disproportionate atrophy of the medial temporal lobe, mammillary body atrophy, thinning of the hypothalamic floor, marked dilation of the lateral and third ventricles, cavum septum pellucidum with fenestrations and pallor of the substantia nigra.[11]

In 2017, McKee's[12] work was endorsed by the National Institute of Neurological Disorders and Stroke and the National Institute of Biomedical Imaging and Bioengineering (NINDS/NIBIB) as requisite for a pathological diagnosis of CTE. CTE can be diagnosed by identifying *'the pathognomonic lesion (which) consists of p-tau aggregates in neurons, astrocytes and cell processes around small vessels in an irregular pattern at the depths of the cortical sulci'*. The extensiveness of the distribution of p-tau NFT in the brain can be used to stage CTE.[13]

Clinical presentation

McKee et al.[13] conducted a retrospective review of the 85 patients posthumously diagnosed with CTE and confirmed that patients with CTE might be asymptomatic. They may also present with symptoms of headaches, mood changes (depression, mood swings, apathy, anxiety, agitation), changes in behaviour (impulsivity, aggressive behaviour), cognitive function changes (loss of attention and concentration, short-term memory loss, explosivity, poor judgement and decision-making, language difficulties), suicidality, symptoms of motor neurone diseases (MND), Parkinsonism or PTSD.[14] Like other neurodegenerative conditions, CTE can only be diagnosed with a postmortem examination of brain tissue prepared with tau immunochemical stain.[14]

In 2021, the NINDS Consensus Group endorsed the clinical and research diagnostic criteria for TES.[14] Clinical features, symptoms and signs of CTE individuals during their lifetime were analysed by comprehensively reviewing the literature.[15] The NINDS concluded that CTE diagnosed individuals suffered from a similar cluster of clinical features during their lifetime and called this TES. The TES diagnostic criteria allowed the diagnosis of CTE-like illnesses in vivo and allowed TES individuals to be characterised into four categories: suggestive of CTE; possible CTE; probable CTE; and definite CTE.[15] Each of these categories has different sensitivity and specificity in their ability to predict the CTE diagnosis[15]. Research on TES individuals would provide helpful information and insight into CTE, especially if the TES individual's brains can be collected postmortem to validate the correlation between the TES and CTE.

CTE investigations

Most investigations help diagnose acute TBI but are not valuable for diagnosing CTE.[15] Effective diagnostic biomarkers provide the potential to diagnose CTE antemortem. The investigations, using biomarkers aiming to detect the tau pathology, are promising as tauopathy is the underlying pathology of CTE.[15] These investigations are still in an experimental stage, and their results are inconclusive as the outcomes of these tests are limited by small sample sizes, absence of control groups and, most importantly, the lack of postmortem validation.[15]

The following are the biomarkers of interest:

- tau-PET imaging offers encouraging predictive value. 2-(1-{6-[(2-[fluorine-18]fluoroethyl) (methyl)amino]-2-naphthyl}-ethylidene) malononitrile (FDDNP) is a radiotracer that binds specifically to β-sheet pleated sheets, which are present in tau and amyloid NFTs. When FDDNP binds to the NFTs, it allows the positron emission tomography (PET) scan to detect the neurofibrillary tangles.[16] It cannot differentiate different types of NFTs, such as amyloid in Alzheimer's disease and p-tau in CTE, but the pattern of distribution of the NFTs allows one to differentiate between Alzheimer's disease and CTE.[17] Chen et al.[18] demonstrated that the FDDNP-PET scanning of the brains of service members with histories of repeated TBI with suspected CTE showed binding patterns similar to those of retired football players. The illuminated binding pattern was distinct from that seen in Alzheimer's disease and normal aging. Chen's result suggested a potential value of FDDNP-PET for early detection, monitoring and treatment of CTE in vivo in the CTE suspected population.
- Diffusion tensor imaging (DTI) is a magnetic resonance imaging (MRI) technique used to investigate the white matter integrity that is not visible with standard cerebral computer tomography (CT) or standard MRI. It relies on the property of thermal Brownian motion of water molecules and measures the flow of the

water molecules along the axons (white matter).[19] A damaged axon will impede the flow of the water molecules along its axon and represents a breach in the white matter integrity.[20] McCunn et al.[21] used DTI to compare the white matter integrity of healthy civilian subjects and healthy service members exposed to TBI. Compared to the healthy civilian control, they found that service members exposed to TBI had increased white matter irregularities. Although this finding does not diagnose CTE, prospective monitoring of these individuals may reveal an interesting association between TBI and CTE.

- Functional MRI (fMRI) can also evaluate structural and functional changes in the brain after a TBI.[22]

Numerous other tests have the potential to predict an in-vivo diagnosis of CTE. Further research on individual, or a combination of, biomarkers may improve the accuracy of diagnosis of CTE pathology in individuals diagnosed with TES.[15]

CTE cases in the military setting

The relationship between the type and severity of repetitive TBI and CTE in military settings varies. The accurate prevalence of repetitive TBI in the ADF is not measurable as many service members do not report their TBI experience(s).[2] Although exposure to a single TBI[23] was associated with the development of CTE, the consensus is that the CTE arises from repeated TBIs reaching a cumulative dose, in a dose-dependent manner[24], to develop permanent brain damage.

Beran and Bhaska[25] reported two cases of veterans of the ADF who presented with TBI.

- The first case was a 39-year-old Caucasian male soldier who presented with 5 years of deteriorating memory, failing to recognise close acquaintances, difficulty recalling and difficulty retaining specific information. He was exposed to at least 10 blast injuries within 5–50 metres from explosions during deployment. There was no loss of consciousness (LOC) from any of the blast exposures. In 2011, he was within 5–10 metres of a controlled detonation when he felt '*shockwaves through his body when exposed to the explosion*'. In 2009, he reported firing a 66-mm rocket launcher 60 times within a day. He described the rocket launcher as a *'shoulder-fired concussion weapon*'. He also had five episodes of concussions while playing rugby. Clinical tools used to evaluate the soldier's cognition function did not confirm his subjective complaints. Considering that the soldier was exposed to a significant history of repeated TBIs, this is a case that would have benefited by monitoring him to assess whether the symptoms eventuate into TES/CTE.
- The second case was a 39-year-old male soldier who complained of difficulty with anger control, episodes of altered consciousness—which he claimed to be epileptic seizures, gait disturbance with bradykinesia, freezing and bizarre movements, impaired cognition, sleep apnoea requiring continuous positive airway pressure, and various tics and tremors. There was a robotic gait, slow movements, pill-rolling tremor, stuttering and freezing on clinical examination. His memory was also impaired. An MRI of his brain, 48-hour sleep-deprived EEG and DTI MRI were all unremarkable. He initially responded satisfactorily to antiparkinsonian medications. An application of TES criteria on Case #2's clinical features classified him as having 'probable CTE'. Close monitoring of Case #2 was a missed opportunity to gain an insight into the pathogenesis of CTE.

Goldstein et al.[21] performed comprehensive neuropathological analyses of postmortem brains obtained from a case series of four military veterans with known blast exposure, with and without concussive injury. A comparison of their pathological findings against the CTE brains of young American football players demonstrated a remarkable similarity. This suggests that CTE from various professions may have common underlying causation.

McKee et al.[26] reported a case study of five US Military veterans as follows:

1. The first case was a 28-years-old veteran with two combat deployments. He had four episodes of concussion—two before enlisting and two during the combat deployments. He had a concussion without LOC on the first deployment, which was not reported. On the second deployment, there was a concussion with transient LOC following a motor vehicle–bicycle collision. He was subsequently diagnosed with PTSD and committed suicide 2 years later.
2. The second case was a 28-years-old male veteran who experienced several blast exposures during multiple deployments. He was diagnosed with PTSD at age 25. He was shot and killed by the police at the age of 27 when he fired on police and other civilians.
3. The third case concerned a 45-year-old male who became disoriented for 30 minutes after the blast exposure from a single close-range improvised explosive device (IED). Prior to

enlistment, he had a concussion at age 8 following a motor vehicle accident. He was subsequently diagnosed with anxiety and depression and died 2 years later from a ruptured giant basilar aneurism.

4. The fourth case was a 34-year-old male who had two concussions with LOC when exposed to two IED blast exposures, 1 and 6 years before his death. LOC was noted on both occasions, and he subsequently developed depression, anxiety and executive dysfunction. He died from unrelated aspiration pneumonia.
5. The fifth case involved a 22-year-old male who had a concussion without LOC when exposed to a single close-range IED blast 2 years before his death. He was subsequently diagnosed with PTSD 3 months before dying from an intracerebral haemorrhage. He had a history of numerous concussions before enlisting, arising from 2 years of high school football and multiple physical altercations.

In this study, four of the five veterans died from cerebrovascular events years after blast exposures. McKee et al.[26] speculated a possible association between the blast injuries and damage to the vascular integrity as the likely causative factor for these deaths.

Omalu et al.[27] described CTE in a 27-year-old US Marine Corps veteran who was deployed twice and was exposed to mortar and IED blasts, one of which occurred within less than 50 m. He was subsequently diagnosed with PTSD and died by suicide.

McKee et al.[28] reported a postmortem examination of 85 CTE brains from various professions, including 21 veterans. The review of these veteran's medical records showed:

- sixteen were also athletes
- nine veterans were deployed to combat zones
- three veterans had moderate-to-severe TBI while in service (one case from contusion, one case from poorly controlled post-traumatic epilepsy and one case with a traumatic spinal cord injury)
- three veterans were exposed to blasts from the IED and explosive ammunition
- three veterans were diagnosed with PTSD, of whom two were exposed to blasts from IEDs, and one experienced repetitive concussions during combat and in civilian life.

Service members with CTE had TBIs from various sources. Some TBIs occurred before enlisting and others after the enlistment, from various circumstances: sporting activities related and unrelated to their duties; military exercises such as training activities; combats; accidents; and personal activities unrelated to their duty.

Bieniek et al.[29] reported a retrospective review of available records of 1721 patients from their brain bank and identified 66 brains of former athletes with a history of exposure to contact sports. Of the 66 brains of former athletes, 22 had CTE, 35 served in US Armed Forces, six were in combat and three were exposed to blast injuries. They found that CTE was not more prevalent in veterans than athletes and that there was no significant statistical relationship between CTE and the age at symptom onset, symptom duration, age at death, height, education, substance abuse, alcohol or tobacco usage and military service. This study raised the possibility that CTE occurrence may not be related to TBI or military service. It may be a mere coincidence.

Analysis of military CTE cases

The literature review revealed less than 100 reported CTE cases in service members. Except for two cases presented by Beran and Bhaskar, most of the cases reported insufficient clinical details to fully explore the association between the TBI and TES/CTE. There is a common thread of issues arising from these cases:

- CTE was identified in service members of the US Military or ADF.
- CTE was identified in service members exposed to TBIs. Not all individuals had LOC and some did not report the incidence.
- The exposure to TBI occurred in various settings: during a military deployment; during battle; during military training; during exercise; due to a motor vehicle accident; while playing sports; accidental falls; and as the result of pre-existing illnesses, such as an epileptic seizure. Some TBIs pre-existed their enlistment and occurred outside their military duties.
- Blast injuries were the most common form of reported TBI.
- Most service members diagnosed with CTE had the clinical features of the TES during their lives.
- Most CTE-like illnesses began many years after exposure to repeated TBI.

TBI was a ubiquitous clinical feature, connecting the service members to CTE. Not all individuals who were diagnosed with postmortem CTE had clinical TES. Although TBI may have caused a permanent brain

injury that altered one's personality to compel them to become mass murderers and attempt suicide, there is no evidence to support that CTE was the cause of these acts. Despite CTE in military settings, especially concerning the blast injuries, being a mass media sensation, the epidemiological basis linking the TBI causing CTE, at this stage, appears to be not supported by solid evidence. More research is urgently needed to gain further insight into the cause-and-effect relationship between TBI and CTE to safeguard both service members and the ADF's medical and legal interests.

Epidemiology of military TBI, TES and CTE

Military health policy needs to provide evidence-based practice to their service members. Although the knowledge of CTE is evolving, many factors are unknown. The following are the epidemiological inferences that can be drawn from the literature review:

- CTE is a distinct disease entity that can be diagnosed only postmortem, using the NINDS criteria.
- The history of CTE patients demonstrates that they suffered from a cluster of clinical features collected and called TES.
- TES can be diagnosed using NINDS' TES criteria which will help to characterise the likelihood of CTE in a TES diagnosed individual.
- No test can reliably diagnose TES/CTE in vivo, but the investigations that aim to detect the underlying tau pathology in the brain are promising.
- Exposure to repetitive TBIs and subsequent development of TES/CTE is strongly associated, but the cause-and-effect relationship is unproven.
- Service members exposed to repetitive blast injuries have been diagnosed with CTE.
- The true prevalence and incidence of CTE, both in service members and the general population, is unknown.
- An individual's risk factors and predisposing factors for developing CTE are unknown.
- The threshold of cumulative TBIs and the effect of various types of TBI required to develop CTE is unknown.
- TBIs are strongly associated with adverse health and mental consequences.
- Research commitments into understanding CTE are becoming more available but limited, especially in military settings.

The outcome of the International State-of-the-Science Meeting, exploring the potential relationship between blast-related trauma and CTE development,[30] is helpful for the ADF. Although the scientific evidence is limited, the panel acknowledged an association between TBI and CTE. The panel recommended the following:

- A coordinated brain bank is established within the Department of Defence to accept the brains of the deceased veterans to study the prevalence of CTE.
- There is a need for access to blast-exposed tissue samples to explore potential associations between blast exposure and CTE development, including non-blast risk factors, such as genetic susceptibility, age, gender and co-morbidities (i.e. drug and alcohol abuse, and cardiovascular disease).
- Close monitoring of validated diagnostic and screening tools is needed to facilitate antemortem diagnosis of CTE.
- Establish an independent panel to determine whether ongoing longitudinal studies would help determine CTE development and candidate risk factors.
- A prospective longitudinal clinical study to recruit high-risk service members with a history of exposure to TBIs to determine the relationship between the TBI and CTE and help establish a dose-response relationship between TBI and CTE.
- Prevention and mitigation strategies are formulated and implemented to prevent blast-related TBI, including modifying training protocols to reduce blast exposure, improve education and personal protective equipment, and return-to-duty guidelines after blast exposures.
- Westphalen[31] identified that the ADF regularly conducts a periodic health assessment before deployment and review of service members after they are discharged. Health promotion activities/programs are also available to promote and enhance physical resilience. There is a system in place for prospective researchers to take an opportunity to collect data on the at-high-risk group of service members prospectively. In his review of veteran health claim statistics in the ADF, 30–40% of all health claims were from musculoskeletal injury and fractures, which may be associated with significant TBIs. The author concluded that there is room to improve the management of occupational and environmental health hazards, with more emphasis on

prevention than treatment. TES/CTE may also be preventable and could be integrated into such health promotion programs and periodic health assessments.

Ongoing CTE research

The cause-and-effect of TBI and CTE is speculative. The potential impact of recurrent TBI in causing a permanent brain injury and TES/CTE is unknown and has a low probability. Should TES/CTE eventuate, in the context of TBI, it is a debilitating and fatal condition for service members. Acknowledging the social and health significance of a high-risk impact on the probability of an event, generous funding of research into this field is noted, especially by the US Military.

In 2016, the US Military established a neurotrauma consortium (CENC) and initiated a multicentre observational study[32]. It is a longitudinal, prospective study to establish and comprehensively evaluate a cohort of at least 1000 US service members who served in recent military conflicts, to better understand the possible chronic and late-life effects of TBI. The participants will be interviewed every 5 years to monitor the development of TES clinical features. The significance of this study is to identify the link between TBI and TES/CTE. The data collected from this study will give insightful epidemiological information to establish causation and prevent and manage TES/CTE.

In 2019, a 4-year prospective study recruited 240 males participating in high-risk professional American football meeting the TES diagnostic criteria for a prospective 3-year follow-up study designed to detect and diagnose CTE during the life of former footballers and to gain insight into the risk factors, pathogenesis, epidemiology, treatment and prevention of CTE.[33] A similar study in the ADF would be helpful to gain an insight into the TES/CTE in the setting of its services' members. A routine application of the NINDS' TES diagnostic criteria15 would also be helpful in diagnosing TES early in their members' careers.

In 2019, multinational prospective research of former National Rugby League (NRL) players, Retired Professional Rugby League Players Brain Health Study, began collaborating with the National Rugby League, the University of Newcastle, Harvard Medical School and the University of Sydney's Sydney Brain Bank.[34] The research aims to collect brain donations from deceased former NRL players to diagnose a possible CTE and to prospectively document their detailed medical history. A similar study could be devised for the service members of the ADF and would be helpful to gain further insight into the CTE, and possibly TES, in the ADF setting.

In 2009, a TBI and long-term quality of life study of service members was drawn from the Royal Melbourne Hospital trauma registry.[35] The findings suggested that TBI has long-term consequences across all aspects of service members' lives. They experienced worse general health, elevated probability of depression, social isolation and worse labour force participation rates. In 2017, a study investigated the impact of potentially traumatic events on mental health outcomes of service members of ADF. [36] The members who were deployed to combat, packer or relief work in a war zone or region of terror, were more likely to have mental health adversities, such as depression, PTSD and alcohol use disorder. Thomas[37] showed that the prevalence of mental health problems and functional impairment among active component and National Guard soldiers, 3 and 12 months following combat(s) in Iraq was significantly higher than the non-deployed soldiers. Their results concurred with a growing body of literature demonstrating the association of combat in Iraq and Afghanistan with post-deployment mental health problems, particularly PTSD and depression[35]. Further studies are required to determine the true prevalence of mental illnesses in ADF service members who participated in combat deployments. Some of these service members may have experienced unreported TBIs. A TBI registry appears already to be in existence, and it presents another opportunity for the ADF to gain insight into the long-term effect of TBI in service members.

Legal medicine

For an individual to be successful in the tort of negligence, they must satisfy the three elements of the duty of care: establish the duty of care; establish that there was a breach of the care; and establish that there is legal causation that is not too remote.

The ADF owes two paramount duties to their service members. Firstly, the duty to warn of the TES/CTE. Secondly, the duty to diagnose and treat the TBI to avoid developing TES/CTE. Although the cause-and-effect relationship between TBI and CTE is not established convincingly, its association is widely accepted. The Court is unlikely to be sympathetic to a plea of 'we did not know'. The ADF must uphold these duties.

A duty to warn of the risk involves seeking service members to undertake their service to the ADF, knowing that they are at risk of developing TES/CTE, appears fundamental. Service members must understand that this material risk may eventuate,

and they must voluntarily agree to undertake their services within the ADF with the full knowledge of such risk. If the ADF was found to have breached its duty to warn of the TES/CTE risks, the Court may impose legal liability on the ADF for the tort of negligence should the Court believe that the members were not warned and would have acted differently had they been adequately warned.

A duty to diagnose and treat involves the ADF actively seeking to accurately and timely diagnose the suspected TBIs, so that service members can receive appropriate and timely treatment to avoid developing TES/CTE. The ADF needs to warn and educate its members about the material risks of TES/CTE, potentially arising from repeated TBIs. It also needs to have a system in place to diagnose and manage TBIs. A clear and detailed contemporaneous documentation of TBI incidences, treatment records, updated protocols and education programs would assist in documenting the requisite standard of care. It may need to expand the size of the team to deal with administering and coordinating the task. The scientific and medical knowledge about TES/CTE will need continuous monitoring and updating. It is a relentless task but is important because if a service member fails to receive the appropriate treatment for TBI due to inadequate systematic measures being in place, the potentially neglectful conduct of the ADF may be inferred as arising from a breach of the duty of care relevant to diagnosis and treatment, which may be difficult to defend, legally, in the tort of negligence.

A duty of maintaining ongoing research is a subset of the duty to diagnose and treat within this context and may represent an applicable duty to the ADF. In *South Eastern Sydney Area Health Service & Anor v King*[38], a doctor administered chemotherapy to a child to treat a rare brain cancer. The cocktail of medications was administered to the child in a prescribed dosage based on a protocol established by a US-based study group. The US group amended their protocol to reduce the dosage of one of the chemotherapy medications to avoid causing paraplegia. The doctor administering the chemotherapy was unaware of this amended protocol and relied on the superseded protocol. The child subsequently developed paraplegia. Hunt AJA found that:

- the doctor '*has a duty to exercise reasonable care and skill in the provision of professional advice and treatment, and that the standard expected of a medical practitioner is that of an ordinary skilled person exercising and professing to have that special skill*'[39]
- the doctor had '*an obligation to remain informed of any relevant changes occurring in the treatments being administered as within that duty*'[40]
- the doctor '*by proceeding with the treatment of the respondent without ensuring that he had been placed in as good a position as if he had conducted a literature search and any necessary follow-up inquiries himself, he failed in his duty to the respondent in the particular circumstances of this case to be in possession of all the necessary up-to-date information in relation to that treatment*'.[41]

This case is relevant to TBI/TES/CTE management in the military context as the ADF must remain informed of any relevant development(s) in the scientific and medical knowledge relating to TBI/TES/CTE. The duty to possess all the necessary up-to-date information concerning CTE is not a passive expectation but represents an active obligation. The duty is to actively pursue the possession of all the necessary up-to-date information on TBI/TES/CTE for the interests of service members of the ADF.

Conclusion

More things are unknown than are known about TES/CTE in the military setting. Whether in isolation or following multiple mild impacts, TBIs appear to be the requisite precondition for the development of TES-CTE and deserve greater attention.

The ADF owes a legal duty of care to its service members. The duty to warn of the CTE and diagnose and treat TBI/TES/CTE are essential obligations that the Court may place upon the ADF should litigation ensue. The ADF's policy should align with these duties to escape legal liabilities from the tort of negligence. The ADF has the duty and an active obligation to be self-informed about the most up-to-date information about CTE. Despite the current understanding of CTE growing daily, there remains a *'dearth of ongoing commitment to research in this field by people with specialised and developed skills'*,1 especially within the ADF. In contrast to the US Military services, the literature on the impact of TBI/TES/CTE on Australian service members is scarce. More research is needed to understand the pathogenesis of CTE and its risk factors, to safeguard thousands of service members who put their lives on the line to protect the freedoms that we enjoy.

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