*Open Journal of Modern Neurosurgery*, **2013, 3, 23-28**
http://dx.doi.org/10.4236/ojmn.2013.32005 Published Online April 2013 (http://www.scirp.org/journal/ojmn)

# Traumatic Brain Injury in the Military

**Aden McLaughlin**
Intensive Care Department, Gold Coast Hospital, Gold Coast, Australia
Email: aden_mclaughlin@health.qld.gov.au

Received May 22, 2012; revised June 25, 2012; accepted July 15, 2012



## ABSTRACT

Traumatic brain injury (TBI) is a devastating and extraordinarily expensive entity. It is becoming increasingly burdensome in the military setting with societal costs of managing the sequelae of TBI running into the billions of dollars (US$) each year. Increasing awareness among non-neurosurgical medical personnel of the pathophysiology of TBI and rapid and appropriate assessment, triage and treatment will increase the likelihood of a better outcome in any given head injured patient. Careful attention to prevention of secondary injury is vital if further decline following the initial insult is to be achieved. Early and repeated neurological assessment and aggressive management of intracranial hypertension and disorders affecting airway and cardiorespiratory systems are the mainstay of managing moderate to severe TBI. This management may involve medical and surgical options and often requires battlefield assessment prior to aeromedical evacuation. The unique profile and epidemiology of TBI in the military, necessitates ongoing research into primary prevention and appropriate, cost-effective means of assessing and treating these often debilitating injuries. Improvements in the prevention and care of these individuals will lead to enormous individual and societal gains.

**Keywords:** Traumatic; Brain; Injury; Military; Primary Injury; Secondary Injury; Triage; Assessment

## 1. Introduction

In both civilian and military settings, head injury, also referred to as Traumatic Brain Injury (TBI) can be a devastating, highly debilitating and extraordinarily expensive injury. The burden of TBI on military personnel is huge. Head and neck injuries including severe TBI have been reported in 25% of US service members evacuated from Iraq and Afghanistan [1,2]. Up to 40% of military mortality is attributed to TBI [3,4]. Researchers have suggested over 300,000 US veterans of the wars in Iraq and Afghanistan have sustained a mild TBI [4]. Official statistics based on electronic medical records within the US military released by the Department of Defense report 195,547 TBIs were diagnosed in military personnel between 2000-2010. Of this number 2038 were classified as severe; 33,020 were classified as moderate and 150,222 were classified as mild [1]. The societal cost associated with managing TBI and its sequelae in US personnel returned from deployment runs into billions of dollars (US$) annually [5].

The epidemiology of TBI in the military is difficult to assess accurately. With improvements in delivery of medical care and protective equipment, soldiers are now surviving injuries they previously would not have survived [5]. Greater efficacy of treating neurological injury has been facilitated by advanced training of non-neurosurgical medical specialists, and by availability of rapid aeromedical evacuation to definitive care [6]. Prompt and rational treatment of the acutely head-injured individual can make a significant difference to the individual's subsequent outcomes. Understanding the appropriate assessment, triage and treatment of personnel suffering a TBI is essential to achieve best possible outcomes for the individual and the unit. The pathophysiology and acute care of head injury are the main focus of this article.

For a detailed discussion of assessment, triage, management and transport of head injured military personnel, the authors refer you to the Guidelines for field management of combat-related head trauma, Brain Trauma Foundation.

## 2. Primary Brain Injury

The injury sustained by the brain at the time of trauma is referred to as the primary brain injury. It results directly from the trauma and by definition it is irreversible [7] with prevention being the only possible intervention. Classification of primary brain injury based on mechanism of injury identifies three types of TBI; Penetrating; Closed; and Blast [4,8,9].

In penetrating TBI (pTBI) a foreign object penetrates the skull and travels through the brain parenchyma, damaging neurons, glia and fibre tracts [8]. In closed TBI (cTBI) disruption of brain function can occur from brain motion and deformation within the cranial vault, resulting in damage to brain parenchyma, blood vessels and fibre tracts [8]. Blast TBI (bTBI) is currently the predominant cause of TBI in the military [8]. An explosive blast wave induces localized particle motion in the brain parenchyma causing extensive neuronal damage [8].

There may also be secondary, tertiary and quaternary blast effects that directly influence the brain injury. These include impact of material thrown by the blast (secondary), the patient being thrown by the blast (tertiary), and factors not already specified such as burns (quaternary) [8,10].

Primary brain injury can be further stratified by: severity, based on the patients Glasgow Coma Scale score, physical signs and symptomatology; and morphology, which is based on the presence or absence of skull fractures and intracranial lesions [3].

## 3. Secondary Brain Injury

At the time of injury a cascade of events is set in motion by the initial insult. This cascade includes, but is not limited to; perfusion abnormalities, haemorrhage, metabolic derangements, multifactorial oedema; intracranial mass lesions and increased intracranial pressure of several aetiologies [7,9,11-13]. This cascade amplifies the initial traumatic injury and significantly worsens outcomes.

Therapeutic strategies are initiated as early as possible after the primary insult to prevent secondary brain injury and potential subsequent worsening of outcome [3,9, 11,12]. Adequate oxygenation and maintenance of sufficient blood pressure to ensure cerebral perfusion are of paramount importance. A single episode of hypotension in patients with severe TBI is associated with a doubling in mortality (60% vs 27%) [14]. The presence of hypoxia in addition to hypotension is associated with a mortality of approximately 75% [14].

After initial resuscitation, the majority of medical and surgical effort goes into diagnosing and alleviating increased intracranial pressure. Once the point of decompensation on the intracranial volume—pressure curve is reached, salvaging the patient becomes exponentially more difficult and neurological decline and death is imminent.

For a more detailed description of the physiology of brain injury the authors refer you to Guyton and Hall, Textbook of medical physiology 12th Ed.

## 4. Assessment

Initial assessment of trauma casualties in the military setting can be challenging for medical professionals, not only because of the unique austere environment in which assessment must take place, but also because the incidence of multiple severely injured personnel is often high. A typical ratio of dead to injured following a blast is 4:35 [15] making rapid assessment and treatment challenging for first responders. Unlike the civilian adage of the most sick receiving the most care, in field situations, injured personnel must rapidly be triaged into those likely to return to duty, those requiring evacuation and those unlikely to survive [7]. This utilitarian approach ensures appropriate deployment of medical expertise to provide essential care to those who will benefit from it the most and attain the best results for individuals and the unit in the given tactical conditions [7].

Similar to civilian settings initial assessment should follow Advanced Trauma Life Support (ATLS) protocol consisting of repeated primary and secondary surveys maintaining C-spine protection at all times [7]. Although there is limited class 1 evidence on the usefulness of GCS and pupillary examination in the battlefield, it is accepted and indeed recommended as the means of assessing neurological status and by proxy, severity of head injury [7]. The absolute GCS and baseline pupillary size, symmetry and response to light should be recorded. Serial examination of these parameters will identify neurological decline which dictates subsequent treatment and triage [7].

It is important to note that many non-CNS factors can confound a neurological examination. For example a hypotensive patients' neurological examination is unreliable and may return to near-normal following resuscitation to euvolaemia [16]. Blast injury to the globe can cause a traumatic iridoplegia that will leave the patient with fixed dilated pupils that in this case are not necessarily a sign of severe TBI [7].

## 5. Management of Mild TBI

Mild TBI, often referred to as concussion, is defined as a GCS of 14 - 15. Signs and symptomatology can be subtle and diagnosis often requires specialised assessment tools. A patient suffering a mild TBI may exhibit confusion, amnesia or a brief loss of consciousness [1,4,8]. Despite of the initially minor symptomatology, the intracranial injury may evolve and patients may deteriorate and require surgery. Following the injurious event, typically a blast, disruption of cerebral auto-regulation can lead to cerebral hyperaemia. At this stage a second insult to the brain can cause malignant cerebral oedema. This scenario is often referred to as the second impact syndrome, and although rare, it carries a significant mortality if not promptly treated.

It is important to remove patients with suspected mild TBI from duty for observation and reassessment [8]. Tactical conditions may not allow this of course. The

 

majority of patients with acute symptomatology secondary to a mild TBI will not require specific medical treatment per se. The decision to return to duty will be based on factors such as the likely threat the injury poses to the patient and also the patient's ability to continue to make life-and-death decisions [7]. Serial neurological examinations are invaluable in monitoring patients' condition and also to aid decision making. If the patient's GCS deteriorates, the decision to remove from the field becomes easy. If they rapidly return to normal and are oriented to time, place, person and situation, a case may be made for return to duty [7].

Concussion in soldiers is strongly associated with post-traumatic stress disorder and non-specific physical complaints 3 - 4 months after returning home [4,5]. Multiple mild TBIs may be cumulative in their effects and increase morbidity in those exposed [5]. Pre and post deployment screening of all personnel is essential to identify those at greatest risk of developing morbidity upon their return from deployment. Those personnel exposed to a blast should be subject to more detailed assessment [5]. Both the Australian Defence Force and the U.S. Department of Defence have specialised screening tools [2,5,17]. Identification of those at risk, education and early intervention programs are effective at reducing sequelae of mild TBI for individuals [5] and by extension society.

## 6. Management of Moderate and Severe TBI

The approach to the management of moderate and severe TBI is best considered together as the principles are the same, as is the aggressive nature with which they must be applied. A moderate TBI renders the casualty GCS 9 - 13. These patients are prone to neurological decline and without appropriate management can deteriorate to a severe TBI (GCS 3 - 8). Hypotension and hypoxia are the principal causes of deterioration in head-injured patients [3,9,18]. Hypotension is not usually due to the brain injury itself, except in the terminal stages when medullary failure supervenes. Hypotension is more often due to hypovolaemia from blood loss, which may be occult. 50% of battlefield casualties are attributed to exsanguination [19]. Mortality due to severe TBI is decreasing compared with previous conflict, but remains high at up to 25% [20].

A patent airway must be ensured in all patients with moderate - severe TBI. Any individual with a GCS < 9 or any other factor threatening airway security, for example impending obstruction, should be managed with endotracheal intubation [3,7]. These patients should receive supplemental oxygenation and be ventilated to normocarbia [7,21]. Prophylactic hyperventilation has been shown to be harmful and should be avoided [22,23]. Monitoring of oxygen saturations, (ensuring levels greater than 90%), and $pCO_2$/end tidal $CO_2$ are essential [7]. Concomitant pulmonary blast injury is common and should be anticipated [10].

The most important intervention in stabilising circulation in trauma patients is to identify and correct ongoing haemorrhage if possible [3]. Intravascular resuscitation is also essential to maintain intravascular volume and critical perfusion pressure in those that are hypotensive. There is no class 1 evidence dictating ideal BP end points, however a systolic BP of 90 mmHg is suggested as the minimum. Isotonic saline is an appropriate resuscitation fluid [7].

Brain targeted therapy can be instituted at any time if an individual begins to demonstrate signs of cerebral herniation such as uni/bilateral fixed and dilated pupils, asymmetric motor posturing or declining mental status [7]. Hyperventilation and hyperosmolar therapy with mannitol or hypertonic saline are the therapies of choice. Hyperventilation will reduce ICP by causing cerebral vasoconstriction and hence decrease cerebral volume. There is evidence that suggests patients subjected to extended periods of hyperventilation have poorer outcomes at 3 and 6 months and so this should only be employed as a temporising measure prior to more definitive care [7]. Hyperosmolar therapy reduces ICP by favouring movement of water out of the brain and also by improving flow dynamics of blood [7]. Hypertonic saline has a logistical advantage over mannitol in that smaller volumes can be used. However, the evidence for efficacy of hyperosmolar agents mostly pertains to mannitol [7].

Following stabilisation many of these patients will require evacuation to facilities equipped to offer neurosurgical care prior to transfer to definitive care. The timing, mode and indeed the decision to evacuate will depend not only on medical priorities but tactical and logistic issues as dictated by the field situation [7]. Patients with a GCS of 3 - 8 should immediately be evacuated to a facility offering 24-hour CT scanning and 24-hour operating room facilities staffed to deliver prompt neurosurgical care [7]. Removal from the field of those that are GCS 9 - 13 is not as emergent however should be achieved in as timely a fashion as possible.

## 7. Further Medical Management

### 7.1. Monitoring

Monitoring of vital functions is essential in all personnel with TBI. Blood oxygen saturations and end tidal $CO_2$ should be mandatory. Arterial lines should be employed to provide accurate BP monitoring where available. Jugular venous $O_2$ saturations should be monitored if aggressive hyperventilation is being considered. All patients with severe TBI deemed potentially salvageable should have intracranial pressure monitoring [7,21,24].

 

### 7.2. ICP Assessment and Control of Intracranial Hypertension (IC-HTN)

After initial resuscitation, the majority of medical and surgical effort goes into diagnosing and alleviating IC-HTN. Once the point of decompensation on the intracranial volume—pressure curve is reached, salvaging the patient becomes exponentially more difficult and neurological decline and death is imminent.

Clinical signs of IC-HTN include; pupillary dilatation (unilateral or bilateral); asymmetric pupillary reaction to light; decorticate or decerebrate posturing or progressive deterioration of the neurological exam not attributable to identifiable extra-cranial factors [3]. Cushing's triad (of hypertension, bradycardia, respiratory abnormalities) is a late sign, present in ~33% of cases of IC-HTN [25,26].

Intracranial pressure is measured using an intraventricular catheter (IVC) or probe [3]. Insertion is an invasive procedure, requiring excellent sterility but minimal equipment. Potential complications include haemorrhage, which is clinically significant in 2% of cases, and more likely in a multitrauma or coagulopathic patient. A ventricular catheter can also be used to rapidly reduce CSF pressure in acute hydrocephalus. ICP should be kept < 20 mmHg in all cases [25].

Additional measures to control ICP include:

- Elevate head of bed to 30 - 45 degrees to decrease ICP by enhancing venous outflow;
- Avoid hypertension as this will contribute to hyperaemia and increase ICP;
- Ventilate to normocarbia as reduced $CO_2$ will cause cerebral vasoconstriction and reduce cerebral blood flow thereby decreasing intracranial volume. This should be used with care;
- Sedation reduces elevated sympathetic tone and hypertension induced by movement.

If the general measures listed above fail to adequately control IC-HTN, the management can include heavy sedation, hypertonic saline, drainage of CSF, aggressive hyperventilation and mannitol. Hypothermia is not proven as an effective intervention. Surgical decompression is an excellent option if available.

Mannitol and hypocarbia are useful as short term measures if definitive surgical care is anticipated [22]. Their main use is as a temporizing measure in a patient with uncal herniation. Hyperventilation can reduce the ICP 25% - 30% with a $PaCO_2$ of 29 mmHg, but can cause a worsening of the cerebral blood flow and an exacerbation of injury [22,23]. They should be used cautiously, and avoided in volume depleted patients or those with chest injuries [21]. Corticosteroids have no role in the acute management of head trauma [27].

### 7.3. Prophylactic Anti-Epileptic Drugs

The incidence of early post traumatic seizures (PTS) in patients with severe TBI is ~30%; and that of late PTS in the same patient group is ~10% - 13% [8]. Prophylactic use of anti-epileptic drugs (AEDs) is indicated in patients with severe TBI to reduce the incidence of early PTS and avoid adverse effects associated with seizures such as increased ICP, increased BP, metabolic dysfunction and increased oxygen demand [28]. Patients with acute intracranial mass lesions; open-depressed skull fractures with parenchymal injury; penetrating injury and seizures within the first 24 hours are all high-risk for early PTS [28]. Continuation of AEDs longer than one week is generally unhelpful except in those with penetrating injuries. Prophylactic AED use does not reduce the incidence of late PTS [29].

### 7.4. Surgical Management

Surgical management of the head-injured patient may be used in the treatment of skull fractures, IC-HTN, penetrating skull injuries, scalp wounds and the evacuation/ decompression of intracranial mass lesions.

Closed depressed skull fractures may be managed surgically or medically. There is no difference in outcomes in terms of seizure incidence, neurologic dysfunction or cosmetic appearance [30]. Open depressed fractures tend to be managed surgically if the fragment is depressed more than the thickness of the calvaria (or arbitrarily > 1 cm) and if there is evidence of dural penetration, significant intracranial haematoma, frontal sinus involvement, contamination or gross deformity [30]. Extradural haematomas (EDH) occur in up to 8% of TBI [20]. Patients with EDH and a GCS < 9, or an estimated volume > 30 ml should undergo urgent surgical evacuation. Acute subdural haematomas (SDH) are equally common as EDH [20], and may be more insidious in their development. The optimal management is usually surgical evacuation, recommended within 4 hours.

Current principles in management of severe TBI rely on ICP monitoring, aggressive early decompressive craniectomy (DECRA), removal of fragments where symptomatic or accessible, watertight dural closure and a delayed cranioplasty with a synthetic plate [31]. A high index of suspicion needs to be maintained for intracranial vascular damage, including traumatic aneurysms and vasospasm, with a preparedness to treat at the time of DECRA [32].

Frontotemporoparietal DECRA is recommended for unilateral injuries and swelling, and bifrontal DECRA for bilateral frontal lobe and/or temporal lobe swelling as evident on CT [33]. The craniotomy should be at least 12 cm diameter for effective decompression and prevention of ischaemia of the brain at the wound margins. Dural substitute onlay is preferred to harvesting pericranial grafts, in order to minimize operating time [33]. Indication for surgery include recalcitrant ICP's, or a severe

 

TBI awaiting aeromedical transport.

Operation can reduce ICP to < 20 mmHg in 85% of patients [11]. Approximately 30% of this pressure drop is due to removal of the bone flap and the remaining 70% to the durotomy [11]. Some authors suggest the procedure needs to occur within 6 hours of the TBI due to the rapidity with which life-threatening cerebral oedema can occur (11).

### 7.5. Evacuation

Aeromedical evacuation of TBI after initial neurosurgical stabilisation is a very dangerous period for these patients and carries a mortality rate of approximately 4.4% [34]. Physiological stressors in flight include but are not limited to hypoxia, barotrauma, arterial gas embolism, and temperature shifts [35]. All available physiological monitoring should be employed to ensure secondary brain injury and subsequent neurological decline is avoided. Monitoring of intracranial pressure during aeromedical transport is mandatory for any casualty suffering a severe TBI.

## 8. Summary

The significance of TBI in military and civilian settings cannot be over-stated. It is a common and devastating injury with the potential for long-lasting, far-reaching effects. The financial implications alone are astronomical, not to mention the more occult and pervasive effects on individuals and society. For these reasons, the investigation into the epidemiology of TBI in military personnel is continuing. The current best practice evidence-based guidelines for management of combat-related head trauma must continue to be augmented by further research into the utility of current principles specifically in battlefield scenarios [7].

The profile of TBI in the military environment is somewhat different to that of the civilian setting. Patients are of a younger and more resilient demographic, with a higher incidence of blast-related head trauma [36]. Initial assessment and management of TBI in the field mirrors civilian protocols whereby expeditious stabilisation of cardiopulmonary status is required to avoid secondary insults. Attainment of best possible outcomes for these patients subsequently depends greatly on aggressive management of intracranial hypertension, damage control neurosurgery and evacuation through vehicles of opportunity and critical care aeromedical teams [35] to definitive care.

## REFERENCES

[1] Military Health System [Homepage on the Internet], 2011. http://www.health.mil/Research/TBI_Numbers.aspx

[2] Deployment Health Clinical Centre [Homepage on the Internet], 2011. http://www.pdhealth.mil/TBI.asp

[3] J. Ali, C. Aprahamian, R. Bell, C. D. Boyle, D. Feliciano, *et al.*, "Advanced Trauma Life Support for Doctors," 6th Edition, American College of Surgeons, Chicago, 1997.

[4] C. Hoge, D. Mcgurk, J. Thomas, *et al.*, "Mild Traumatic Brain Injury in US Soldiers Returning from Iraq," *The New England Journal of Medicine*, Vol. 358, No. 5, 2008, pp. 453-463. doi:10.1056/NEJMoa072972

[5] N. Ford and J. Rosenfeld, "Mild Traumatic Brain Injury and Bomb Blast: Stress, Injury or Both?" *ADF Health*, Vol. 9, No. 2, 2008, pp. 68-73.

[6] R. Bell, C. Mossop, *et al.*, "Early Decompressive Craniectomy for Severe Penetrating and Closed Head Injury during Wartime," *Neurosurg Focus*, Vol. 28, No. 5, 2010, p. E1. doi:10.3171/2010.2.FOCUS1022

[7] The Brain Trauma Foundation [Homepage on the Internet], 2011. http://www.braintrauma.org/pdf/protected/btf_field_management_guidelines.pdf

[8] G. Ling, *et al.*, "Explosive Blast Neurotrauma," *Journal of Neurotrauma*, Vol. 26, No. 8, 2009, pp. 815-825. doi:10.1089/neu.2007.0484

[9] S. T. Dawodu, "Traumatic Brain Injury (TBI)—Definition, Epidemiology, Pathophysiology," eMedicine, 2011. http://emedicine.medscape.com/article/326510-overview

[10] A. Mayo and Y. Kluger, "Blast Induced Injury of Air-Containing Organs," *ADF Health*, Vol. 7, 2006, pp. 40-44.

[11] K. I. Mathai, *et al.*, "Decompressive Craniectomy in Traumatic Brain Injury," *Indian Journal of Neurotrauma*, Vol. 7, No. 1, 2010, pp. 9-12.

[12] N. J. Cowley and E. J. da Silva, "Prevention of Secondary Brain Injury Following Head Trauma," *Trauma*, Vol. 10, No. 1, 2008, pp. 35-42. doi:10.1177/1460408608089322

[13] A. Unterberg, K. Kiening, P. Schmiedek, *et al.*, "Long-Term Observations of Intracranial Pressure after severe Head Injury. The phenomenon of Secondary Rise of Intracranial Pressure," *Neurosurgery*, Vol. 32, No. 1, 1993, pp. 17-24.

[14] R. M. Chesnut, L. F. Marshall, M. R. Klauber, *et al.*, "The Role of Secondary Brain Injury in Determining Outcome from Severe Head Injury," *The Journal of Trauma*, Vol. 34, No. 2, 199, pp. 216-222. doi:10.1097/00005373-199302000-00006

[15] C. Stevenson, "Evolving Mechanisms and Patterns of Blast Injury and the Challenges for Military First Responders," *Journal of Military and Veterans' Health*, Vol. 17, No. 4, 2009, pp. 17-21.

[16] F. Plum and J. Posner, "The Diagnosis of Stupor and Coma," 3rd Edition, F. A. Davis, Philadelphia, 1982.

[17] W. P. Jones, S. A. Loe, S. K. Krach, R. Y. Rager and H. M. Jones, "Automated Neuropsychological Assessment Metrics (ANAM) and Woodcock Johnson III Tests of Cognitive Ability: A Concurrent Validity Study," *Clinical Neuropsychology*, Vol. 22, No. 2, 2008, pp. 305-320. doi:10.1080/13854040701281483

[18] S. L. Bratton, *et al.*, "Brain Trauma Foundation: Blood

 

Pressure and Oxygenation," *Journal of Neurotrauma*, Vol. 24, Suppl. 1, 2007, pp. S7-S13.

[19] J. Kragh, D. Baer and T. Walters, "Extended (16 Hour) Tourniquet Application after Combat Wounds. A Case Report and Review of the Current Literature," *Journal of Orthopaedic Trauma*, Vol. 21, No. 4, 2007, pp. 274-278. doi:10.1097/BOT.0b013e3180437dd9

[20] M. Eisenburg, M. Christie and P. Mathew, "Battlefield Neurosurgical Care in the Current Conflict in Southern Afghanistan," *Neurosurg Focus*, Vol. 28, No. 5, 2010, p. E7. doi:10.3171/2010.2.FOCUS108

[21] S. I. Stiver and G. T. Manley, "Prehospital Management of Traumatic Brain Injury: Prevention of Secondary Injury in the Prehospital and Emergency Setting," eMedicine, 2011. http://www.medscape.com/viewarticle/585165_3

[22] M. R. Bullock and J. T. Povlishock, "Guidelines for the Management of Severe Traumatic Brain Injury," *Journal of Neurotrauma*, Vol. 24, 2007, pp. 87-91.

[23] J. A. Doyle, D. P. Davis and D. B. Hoyt, "The Use of Hypertonic Saline in the Treatment of Traumatic Brain Injuries," *The Journal of Trauma*, Vol. 50, No. 2, 2001, pp. 367-383. doi:10.1097/00005373-200102000-00030

[24] C. Hoge, H. M. Goldberg and C. Castro, "Care of War Veterans with Mild Traumatic Brain Injury—Flawed Perspectives," *The New England Journal of Medicine*, Vol. 360, No. 16, 2009, pp. 1588-1591. doi:10.1056/NEJMp0810606

[25] N. Juul, G. F. Morris and S. B. Marshall, *et al.*, "Intracranial Hypertension and Cerebral Perfusion Pressure: Influence on Neurological Deterioration and Outcome in Severe Head Injury," *Journal of Neurosurgery*, Vol. 92, No. 1, 2000, pp. 1-6. doi:10.3171/jns.2000.92.1.0001

[26] A. H. Ropper, "Raised Intracranial Pressure in Neurological Disease," *Seminars in Neurology*, Vol. 4, No. 4, 1984, pp. 397-407.

[27] I. Roberts, D. Yates, *et al.*, "Effect of Intravenous Corticosteroids on Death within 14 Days in 10,008 Adults with Clinically Significant Head Injury (MRC CRASH Trial)," *Lancet*, Vol. 364, No. 9442, 2004, pp. 1321-1328. doi:10.1016/S0140-6736(04)17188-2

[28] R. Bullock, R. M. Chestnut, G. Clifton, *et al.*, "Guidelines for the Management of Severe Head Injury," The Brain Trauma Foundation, The American Association of Neurological Surgeons, and The Joint Section of Neurotrauma and Critical Care, New York, Park Ridge, 1995.

[29] N. R. Temkin, S. S. Dikmen, A. J. Wilensky, *et al.*, "A Randomised, Double-Blind Study of Phenytoin for the Prevention of Post-Traumatic Seizures," *The New England Journal of Medicine*, Vol. 323, 1990, pp. 497-502. doi:10.1056/NEJM199008233230801

[30] R. Bullock, R. M. Chestnut, J. Ghajar, *et al.*, "Surgical Management of Depressed Cranial Fractures," *Neurosurgery*, Vol. 58, No. 3, 2006, pp. S56-S60. doi:10.1227/01.NEU.0000210367.14043.0E

[31] P. Agarwalla, G. Dunn and E. Laws, "An Historical Context of Modern Principles in the Management of Intracranial Injury from Projectiles," *Neurosurg Focus*, Vol. 28, No. 5, 2010, p. E23. doi:10.3171/2010.2.FOCUS1038

[32] R. Bell, R. Ecker, *et al.*, "The Evolution of the Treatment of Traumatic Cerebrovascular Injury during Wartime," *Neurosurg Focus*, Vol. 28, No. 5, 2010, p. E5. doi:10.3171/2010.2.FOCUS1025

[33] B. Ragel, P. Klimo, *et al.*, "Wartime Decompressive Craniectomy: Technique and Lessons Learned," *Neurosurg Focus*, Vol. 28, No. 5, 2010, p. E2. doi:10.3171/2010.3.FOCUS1028

[34] R. Fang, G. Dorlac, P. Allan and W. Dorlac, "Intercontinental Aeromedical Evacuation of Patients with Traumatic Brain Injuries during Operations Iraqi Freedom and Enduring Freedom," *Neurosurg Focus*, Vol. 28, No. 5, 2010, p. E1.

[35] J. Reno, "Military Aeromedical Evacuation with Special Emphasis on Craniospinal Trauma," *Neurosurg Focus*, Vol. 28, No. 5, 2010, p. E12. doi:10.3171/2010.2.FOCUS1023

[36] F. Stephens, *et al.*, "Cranioplasty Complications Following Wartime Decompressive Craniectomy," *Neurosurg Focus*, Vol. 28, No. 5, 2010, p. E3. doi:10.3171/2010.2.FOCUS1026

 