

NEUROINTENSIVE CARE

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Abstract

The majority of neurological admissions to military Intensive Care will be for Traumatic Brain Injury (TBI). These injuries will be either penetrating from fragmentation or missiles or blunt due to blast or impact. Intensive Care management of TBI is focused on the prevention of secondary brain injury due to insults such as hypoxia, hypotension and low Cerebral Perfusion Pressure. This management is based largely on comprehensive evidence based guidelines produced by the Brain Trauma Foundation. The most significant dilemma faced by UK military intensivists is whether we should be measuring Intracranial Pressure in patient with severe TBI in the deployed settings; and if so what technique should be used.

Introduction

A wide variety of neurological conditions may require management in intensive care. In the military environment, however, the majority of admissions will be due to Traumatic Brain Injury (TBI). During combat operations these will be a mixture of penetrating injuries due to fragments or bullet, and closed head injury due to blast and impact. Blunt injury, commonly due to road traffic accidents, will predominate during non-combat operations.

The data on combat injuries is notoriously difficult to interpret, but historically, 15-20% of injuries occur above the clavicles (Table 1) [1]. A recent paper indicated that 42% of British casualties killed by hostile action in Afghanistan and Iraq between April 2006 and March 2007 had non-survivable brain injury [2] whilst in Somalia 43% of fatal penetrating injuries occurred to the head or neck [3]. During Gulf War I, however, only one of 24 head injuries required craniotomy [4]. In a series of 224 cases from Afghanistan, 17% involved the head and neck but only 2 craniotomies and 2 neck explorations were required [5]. This data suggests that head injury is frequently the fatal cause of death in military trauma but among survivors the rate of significant head injury requiring surgical intervention is quite low.

	Head & Neck (%)	Thorax (%)	Abdomen (%)	Limb (%)	Other (%)
WW1	17	4	2	70	7
WW2	4	8	4	75	9
Korean War	17	8	7	67	2
Vietnam War	14	7	5	74	
Borneo	12	12	20	56	
Northern Ireland	20	15	15	50	
Falklands	16	15	10	59	
Gulf War (UK)	6	12	11	71	32*
Gulf War (US)	11	8	7	56	18*
Afghanistan	16	12	11	61	
Chechnya	24	9	4	63	
Somalia	20	8	5	65	2*

+ Multiple wounds. * 80% caused by fragments; range of hits 1-45; mean, 9.

** Buttock and back wounds, all multiple fragment injuries as a separate figure.

Table 1. Anatomical distribution of penetrating wounds of the "casualty template". [1].

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Modern helmet design is increasing the protection provided to fragmentation and missiles but appears to be revealing a phenomenon analogous to Behind Armour Blunt Trauma (BABT) where the ballistic energy produces helmet deformity with potential skull fracture and intracranial haemorrhage which may require surgical intervention [6].

Blunt head injuries consist of focal injuries including contusions and haematomas and diffuse injuries such as concussion and Diffuse Axonal Injury (DAI). Neurosurgical interventions may be required to evacuate haematomas or remove contusions, elevate skull fractures or relieve raised Intracranial Pressure (ICP) by inserting an External Ventricular Drain (EVD) or craniectomy.

There is a high incidence of cervical spine injury associated with blunt head injury and so the cervical spine should be considered unstable until cleared [7].

Head injuries are relatively common in the military population and although most will not require surgical intervention they may well require Intensive Care Management.

Intensive Care Management

Intensive care management of the head injured patient depends on the application of good quality general ICU management, including ventilation and sedation, as well as attention to the factors that contribute to secondary brain injury (Table 2). Secondary brain injury occurs when the primary insult to the brain is exacerbated by conditions that are common in the traumatised patient. The final common pathway is ischaemia to the watershed areas of the injured brain extending the damage.

- Hypotension – Systolic < 90 mmHg [8].
- Reduced CPP - < 60 mmHg [8].
- Raised ICP - > 20-25 mmHg [8].
- Hypoxaemia – PaO₂ < 60 mmHg [8].
- Pyrexia – 10% rise in Cerebral Metabolic rate per 1°C rise.
- Hyperglycaemia – excess glucose metabolised anaerobically.
- Hypoglycaemia.

Table 2. Causes of secondary brain injury.

Standard invasive and non-invasive ITU monitoring is complemented by the use of specific additional neurological monitoring modalities and imaging.

Comprehensive evidence based Guidelines for the Management of Severe Traumatic Brain Injury have been produced by the Brain Trauma Foundation [8] and form the main source for the bulk of this review of management.

Blood Pressure and Oxygenation

Both pre-hospital and intra-hospital hypotension have been shown to have a poor influence on outcome from severe traumatic Brain Injury (TBI). In the Traumatic Coma Data Bank (TCDB) [9] a single episode of pre-hospital hypotension (systolic blood pressure (SBP) < 90 mmHg) was among the most powerful predictors of outcome. Similarly hypoxaemia occurred in 22.4 % of severe TBI patients and was significantly associated with increased mortality and morbidity. Every effort must be made to maintain SBP > 90 mmHg and PaO₂ > 60 mmHg by prompt and efficient resuscitation including the use of fluids, inotropes, intubation and ventilatory strategies. However there is some evidence that aggressively treating with inotropes may contribute to an increase in the risk of Acute Respiratory Distress Syndrome (ARDS) [10].

Anaesthesia, Analgesia and Sedatives

Pharmacological agents for analgesia and sedation are frequently used in the head injured patient for sound clinical as well as humanitarian reasons. Minimising pain and agitation will limit potentially deleterious rises in ICP, blood pressure and resistance to ventilation. However some clinicians are concerned about the masking of neurological signs and symptoms and potential adverse haemodynamic effects.

The only study directly comparing sedative agents in neurointensive care suggested some advantages of Propofol infusion over morphine infusion [11], however most of the benefit occurred in the high dose Propofol group and concerns have since arisen about Propofol infusion syndrome with high dose infusions. In the UK it is common practice to use Propofol combined with an opioid for sedation and Morphine and Midazolam infusions for longer term sedation.

High dose Barbiturate (thiopentone) therapy can result in control of ICP when all other medical and surgical options have failed. However it has shown no clear benefit in improving outcome. The Cochrane injuries group have reviewed Randomised Controlled Trials of the use of Barbiturates [12] and concluded; *“There is no evidence that Barbiturate therapy in patients with acute severe head injury improves outcome. Barbiturate therapy results in a fall in blood pressure in one in four patients. The hypotensive effect of Barbiturate therapy will offset any ICP lowering effect on Cerebral Perfusion Pressure”*.

If thiopentone is used, ideally it should be combined with continuous Electroencephalographic monitoring to allow titration to burst suppression. This clearly limits its utility in the military setting.

Cerebral Perfusion Pressure (CPP)

Cerebral Perfusion Pressure is used as an indicator of the driving pressure affecting cerebral blood flow and hence perfusion.

$$\text{Cerebral Perfusion Pressure (CPP)} = \text{Mean Arterial Pressure (MAP)} - \text{Intracranial Pressure (ICP)}$$

Low CPP has frequently been found to correlate with poor outcome. The original work by Rossner stressing the maintenance of CPP greater than 70mmHg showed superior outcome compared to the TCDB [13]. More recent studies suggest there is a critical threshold for CPP that appears to be between 50 to 60 mmHg [8]. Meanwhile other evidence suggests that there are serious deleterious effects of aggressively raising CPP. In a randomised trial comparing CPP targeted with ICP targeted therapy there was a five fold greater risk of ARDS associated with more inotrope and vasopressor use in the CPP targeted group [10]. Pragmatically it would seem that a target CPP of 60 mmHg should be fine tuned in individual patients by monitoring of cerebral oxygenation, metabolism and status of autoregulation.

Intracranial Pressure (ICP)

Intracranial Pressure monitoring is the accepted standard of care for traumatic brain injury (Table 3) as a tool to maintain CPP and hence cerebral oxygenation and avoid secondary injury whilst the traumatised brain recovers. Current data support 20-25 mmHg as an upper threshold above which treatment to lower ICP should generally be initiated. ICP can not be predicted by CT alone and not monitoring ICP while treating for elevated ICP can be deleterious and produce poor outcome. Although there is no convincing evidence that actively treating raised ICP produces improved outcome, morbidity is improved if head injured patients are treated on a neurointensive care unit. This is presumably due to strict adherence to CPP/ICP based protocols.

- All salvageable patients with severe TBI (GCS 3-8) & abnormal CT scan
- Severe TBI with normal CT & 2 or more of following
 - Age > 40 years
 - Motor posturing
 - SBP < 90 mmHg

Table 3. Indications for ICP monitoring [8].

The two devices most commonly used to measure ICP are the external ventricular drain (EVD) and the intraparenchymal monitor or ICP bolt. The EVD is considered the gold standard as the most accurate and reliable method of monitoring ICP and is low cost and the perceived risks of infection and haemorrhage appear rare. Unfortunately for the deployed medical setting, it requires neurosurgical training to insert an EVD; however, it would then be possible to monitor ICP continuously, including during aeromedical evacuation, and actively treat raised ICP. The ICP bolt can be safely placed by intensivists in regional DGHs and presumably deployed medical units. ICP bolts can not be recalibrated once inserted and would not be suitable for use during aeromedical evacuation.

Brain Oxygenation monitoring and thresholds

Although ICP monitoring is routinely used for patients with severe TBI it only gives limited information regarding factors known to be important to the pathophysiology and outcome from TBI such as CBF and metabolism. Therapy is directed at preventing secondary brain injury and is mainly dependant on adequate oxygen and substrate delivery to the brain. Oxygen delivery is dependent on oxygen content and Cerebral Blood Flow.

Systems have been developed which can measure CBF indirectly e.g. Transcranial Doppler (TCD), Oxygen delivery e.g. jugular venous bulb saturation (SjO₂) and brain tissue monitoring or to assess the metabolic state e.g. cerebral microdialysis. To date only jugular venous bulb saturation (SjO₂) and brain tissue oxygen monitoring have sufficient clinical experience to relate data to outcome in TBI. Jugular monitoring provides a global measure of cerebral oxygenation and brain tissue monitoring a focal measure but both potentially allow individual optimisation of CPP targets.

Jugular bulb oxygenation (SjO₂)

Mixed venous jugular bulb oxygenation can be measured by the retrograde placement of an oximetry catheter within the Internal Jugular Vein so that the tip lies at the jugular bulb and samples the average saturation of all cerebral venous blood flow. Evidence suggests that episodes of desaturation (SjO₂ <50-55%) are associated with worse outcomes. If SaO₂ is normal, then a decrease in SjO₂ must be due to either a decreased Cerebral Blood Flow (CBF) or increased cerebral metabolic requirement for Oxygen (CMRO₂). If CPP is maintained then a decreased CBF

must be due to an increase in Cerebral Vascular Resistance. Cerebral desaturation less than 50% is associated with neuronal damage and must be actively treated.

Brain tissue oxygen monitoring

Brain tissue oxygenation can be measured by the placement of an intraparenchymal catheter/probe with a polarographic (Clarke type) cell at its tip. Studies looking at the relationship between outcome and brain tissue oxygen tension ($P_{br}O_2$) have found worsening outcomes with increasing duration of $P_{br}O_2$ less than 15mmHg.

Hyperosmolar therapy

There are currently two hyperosmolar agents in use in TBI: mannitol and hypertonic saline (HS).

Mannitol

Mannitol is widely used in the control of intracranial hypertension following TBI. It is believed to work initially by plasma expansion producing a reduction in viscosity and hence increased Cerebral Blood Flow. The osmotic effect occurs after 15 to 30 minutes and may persist for 90 minutes to 6 or more hours. A single bolus of 0.25 to 1gm per Kg body weight can have short term benefit whilst diagnostic procedures and interventions are accomplished. There is however a lack of evidence for repeated regular administration over a prolonged period.

Hypertonic Saline (HS)

Current therapies used for ICP control such as mannitol have the risk of reducing brain perfusion if care is not taken to maintain intravascular volume and Mean Arterial Pressure. Ideally a therapeutic intervention should decrease ICP whilst preserving or improving CPP. Studies of HS in polytrauma showed the greatest survival benefit and improvement in haemodynamics in the subgroup of patients with associated TBI. HS is believed to exert its effect by decreasing cerebral water content, an osmotic effect, and possibly by effects on the microcirculation.

Prophylactic hypothermia

Hypothermia is often used on admission and to treat raised intracranial pressure in many ICUs however the literature has failed to consistently support any effect on Mortality and morbidity. A further metanalysis conducted by the Brain Trauma Foundation [8] has shown that all cause mortality is not significantly altered by prophylactic hypothermia. However hypothermia was associated with an increased chance of good outcome, or reduced morbidity. There was a significantly lower risk of death if the hypothermia was maintained for longer than 48 hours and better outcomes occurred with the temperature ranges 32-33°C and 33-35°C. Recent evidence suggests that a target temperature of 35°C is as effective at reducing intracranial pressure but with a lower mortality [14].

This data is only applicable to the isolated head injury and we already know that hypothermia is associated with poorer outcomes in polytrauma so its applicability to the deployed military setting is limited [15].

Other measures

Infection prophylaxis

Good clinical practice dictates that ventricular catheters and other ICP monitoring devices should be placed under sterile conditions, use closed systems and minimise manipulations and flushing. There is no evidence for routine catheter exchange or prophylactic antibiotic use. Prolonged use of antibiotics increases the risk of selecting resistant microorganisms but a short course at intubation may reduce the incidence of pneumonia although it has no effect on length of stay or mortality. Similarly early tracheostomy may

reduce ventilator days but has no effect on mortality or incidence of pneumonia.

Deep Vein Thrombosis (DVT) prophylaxis

Patients with severe TBI are at significant risk of venous thromboembolic events (VTE). An incidence of DVT in 20% of severe TBI patients not receiving prophylaxis has been reported. Mechanical means of DVT prophylaxis, such as graduated compression stockings or intermittent pneumatic compression stockings, have demonstrated efficacy in multiple populations and should be used until the patient is mobile unless lower extremity pathology prevents their use. Studies also show that pharmacological means of DVT prophylaxis augment mechanical means and reduce the incidence of VTE in TBI but there is a trend towards increased intracranial haemorrhage. A number of case studies suggest that pharmacological agents should not be started perioperatively. There is not enough evidence to support specific recommendations about choice of agent, dose or timing, although there appears to be a consensus to withhold pharmacological agents until 24 hours after admission or operation whichever is the latter.

Anti-seizure prophylaxis

There is a relatively high incidence of Post Traumatic Seizures (PTS) in TBI and there are potential benefits to preventing seizures following TBI such as preventing episodes of raised ICP and haemodynamic instability or accidental injury. There was also a belief that preventing early seizures might prevent the development of chronic epilepsy. Studies have shown, however, that although phenytoin reduces early PTS it has no significant effect on late PTS and the prevention of PTS has no effect on outcome. Routine anti-seizure prophylaxis is not recommended past seven days. If late seizures occur they should be managed using routine protocols.

Hyperventilation

Raised ICP occurs frequently in severe TBI and is one of the commonest causes of death and poor outcome. Aggressive hyperventilation ($PaCO_2 \leq 3.5$ KPa) was previously a cornerstone of management for severe TBI because it can cause a rapid reduction in raised ICP. More recently it has become clear that persistent hyperventilation can actually worsen outcome. Hyperventilation causes its reduction in ICP by causing cerebral vasoconstriction and a consequent reduction in vascular volume. The increase in cerebral vascular resistance will therefore reduce Cerebral Blood Flow (CBF); often when it is already critically reduced. There is therefore a very real risk of causing significant cerebral ischaemia. A randomised study has shown significantly poorer outcomes when prophylactic hyperventilation is used [16].

Moderate hyperventilation may be of use as a temporising measure for ICP reduction (e.g. whilst awaiting imminent surgical decompression in the presence of a recent increase in ICP or dilated pupil). It is not recommended for prophylactic or prolonged use and ideally should be avoided in the first 24 hours post injury when CBF is often critically reduced. If hyperventilation is used it is safer to utilise a monitor of cerebral oxygenation such as SjO_2 or $P_{br}O_2$. Without monitoring, it may be wise to electively increase MAP to compensate for the increased cerebral vascular resistance and thus hopefully maintain CBF.

Steroids

A number of trials, notably the Corticosteroids Randomisation After Significant Head injury study (CRASH) [17], have failed to demonstrate any beneficial effect of steroids on outcome in TBI. In fact the CRASH study was stopped after an interim analysis showed a worse outcome in those patients treated with methylprednisolone.

Nutrition

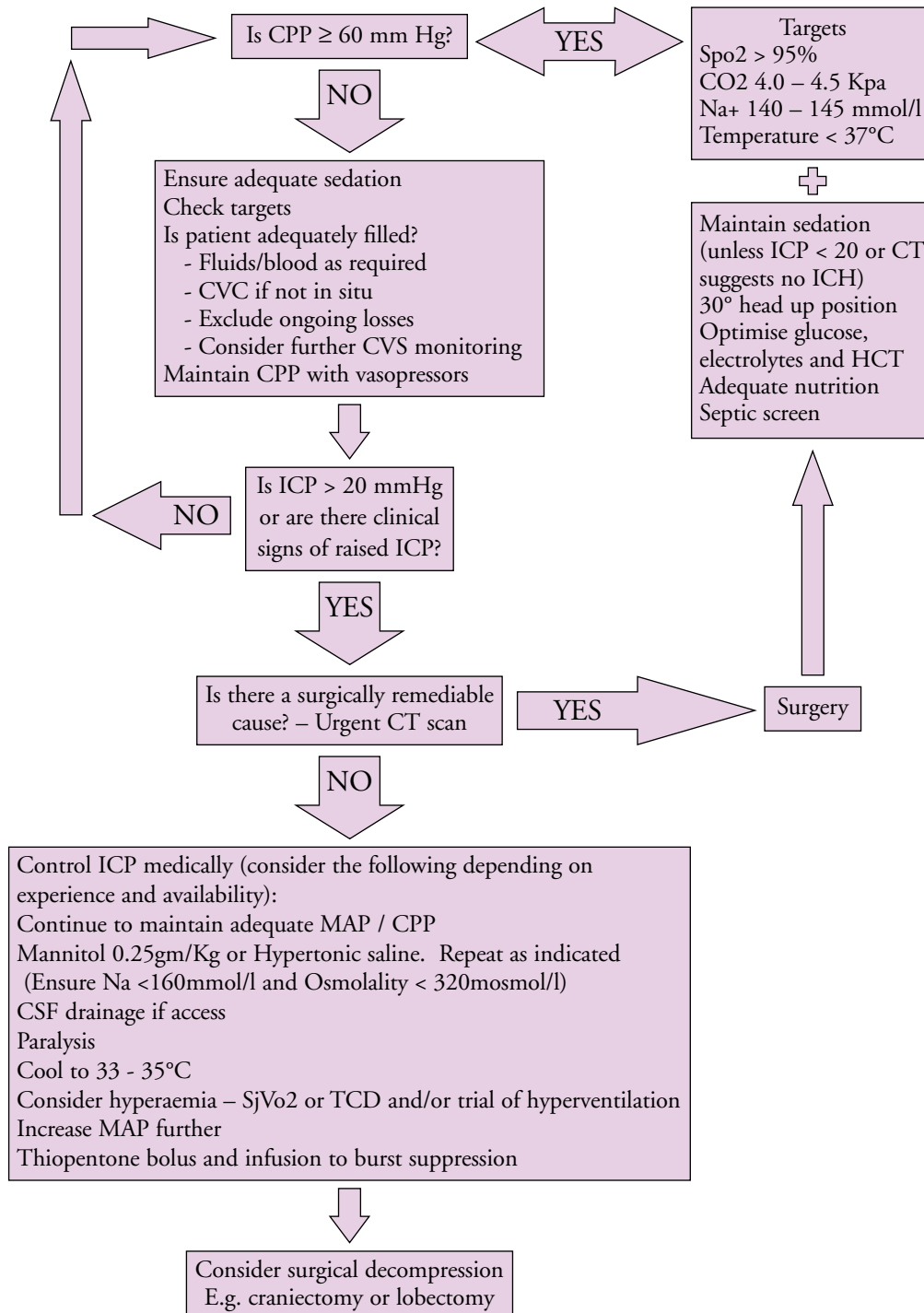
TBI patients lose sufficient nitrogen to lose up to 15% of body weight per week. Evidence from non-TBI patients has shown that a 30% weight loss is associated with worse outcome. Nutritional replacement with 100-140% of resting metabolic expenditure with 15-20% nitrogen calories reduces nitrogen loss. The data supports achieving full replacement by the end of the first week, which would usually entail starting replacement within 72 hours. There is no evidence to currently favour any particular feed or feeding regime.

Glycaemic control is also important; hyperglycaemia has been shown to aggravate hypoxic ischaemic brain injury in animal studies and is associated with worse outcome in human studies. Hypoglycaemia is also detrimental, so an aim to maintain blood glucose at less than 10 mmol/l is probably appropriate.

Summary

The vast majority of neurological conditions requiring critical care support within the deployed military setting will be trauma related. These patients will often have sustained polytrauma and a standard holistic critical care approach to the patient concentrating on the <C>ABC paradigm is important. Specifically for head injured patients, maintenance of systolic blood pressure and oxygenation is of the utmost importance, along with prompt surgical intervention if required. Following initial resuscitation, management is directed towards the prevention of secondary brain injury by the maintenance of Cerebral Perfusion Pressure and brain oxygenation (Figure 1).

Many standard Intensive Care interventions such as DVT prophylaxis and nutrition are appropriate. Prophylactic hypothermia may be possible in the deployed setting but is not



¹ In the absence of ICP monitoring aim for MAP ³ 90 mm Hg and have a low threshold for repeat CT scan.

Figure 1. Intensive care treatment protocol for severe head injury (GCS < 9) ¹

appropriate in the polytrauma patient. It may also be difficult to maintain during transfer and repatriation to the UK. The main dilemma for the military intensivist is whether we should be measuring ICP in this patient group, and if so, how. External Ventricular Drains are the gold standard and are easy to monitor with current ITU monitors and in-flight but require neurosurgical intervention to insert. An ICP bolt can be inserted, after training, but the technology is temperamental, prone to drift and not appropriate for use in flight.

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CRITICAL CARE OF MILITARY BURN CASUALTIES AT ROLE 3 FACILITIES

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Abstract

Burn casualties will inevitably occur in the military environment during both conflict and peacetime. The number and type of casualties will vary on the nature of warfare and the type of troops deployed. New preventative measures have decreased the number and severity of burns found on the battlefield however with new weapon systems casualties suffering from thermal injuries are still to be expected in modern warfare. Over the last 4 decades great advances have been made in the treatment of thermal injuries. These advances are reviewed here with emphasis on those that can be accomplished in the Role 3 facility by non-specialist clinicians. It is beyond the scope of this review to produce didactic treatment protocols but it is hoped that in the near future Clinical Guidelines for Operations will soon reflect these. Where advances have occurred that can not be mirrored in the field hospital early evacuation to specialist facilities back at Role 4 facilities should be a priority.

Introduction

During armed conflict military personnel are at high risk of thermal injury from both battle and accidental injury. The increase in risk is dependant on both the weapon systems deployed and the type of combat engaged in. In conflicts over the last six decades the incident of thermal injury has ranged from 2.3% to as high as 85% (Table 1).

The detonation of a nuclear weapon at Hiroshima in 1945 produced an estimated 57,700 burn victims. This equated to 85% of the total casualties [1]. In contrast during the Panama police

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Conflict	Number of burn casualties (% of total injured)	
World War II Hiroshima 1945	45,500 – 59,500	(65-85)
Vietnam Conflict 1965-1973	13,047	(4.6)
Israeli Six Day War 1967		(4.6)
Yom Kippur War 1973		(10.5)
Falklands War 1982		
- UK Casualties	140	(18.0)
- Argentine Casualties	34/194	(17.5)
Lebanon War 1982		(8.6)
Panama Police action 1989	6/259	(2.3)
Operation Desert Shield/Storm	36/458	(7.9)

Table 1. The Incidence of Burn Injury in Armed Conflict.