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## Isolated traumatic brain injury results in significant pre-hospital derangement of cardiovascular physiology

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### ABSTRACT

**Introduction:** Major trauma can result in both life-threatening haemorrhage and traumatic brain injury (TBI). The pre-hospital management of these conditions, particularly in relation to the cardiovascular system, is very different. TBI can result in cardiovascular instability but the exact incidence remains poorly described. This study explores the incidence of cardiovascular instability in patients undergoing pre-hospital anaesthesia for suspected TBI.

**Methods:** Retrospective case series of all pre-hospital trauma patients attended by Kent, Surrey & Sussex Air Ambulance Trust (United Kingdom) trauma team during the period 1 January 2015–31 December 2016. Patients were included if they showed clinical signs of TBI, underwent pre-hospital anaesthesia and hospital computed tomography scanning subsequently confirmed an isolated TBI.

**Results:** Out of 121 patients with confirmed isolated TBI, 68 were cardiovascularly stable throughout the pre-anaesthesia phase, whilst 53 (44%) showed signs of instability (HR > 100bpm and/or SBP < 100 mmHg pre-anaesthesia). Hypotension (SBP < 100) with or without tachycardia was present in 14 (12%) patients. 10 (8%) patients with isolated TBI received pre-hospital blood product transfusion.

**Conclusion:** Increased awareness that traumatic brain injury can cause significant derangement to heart rate and blood pressure, even in the absence of major haemorrhage, would allow the pre-hospital clinician to treat cardiovascular instability with the most appropriate means, such as crystalloid and vasopressors, to limit secondary brain injury.

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### Introduction

Major trauma is a significant cause of serious morbidity and mortality, particularly in the young [1]. As haemorrhage is the leading cause of death in trauma, haemodynamic instability in the pre-hospital phase of care is often assumed to be the result of ongoing bleeding. A rise in patients' heart rate or fall in their blood pressure is commonly associated with hypovolemic or haemorrhagic shock, but also with obstructive and neurogenic shock. Traditional medical teaching, such as Advanced Trauma Life Support (ATLS) [2], states that isolated traumatic brain injury does not cause shock and that other causes of shock need to be actively sought.

*Abbreviations:* CT, computed tomography; HEMS, helicopter emergency medical service; HR, heart rate; KSSAAT, Kent, Surrey, Sussex Air Ambulance Trust; PLE, pronounced life extinct; RSI, rapid-sequence induction; SBP, systolic blood pressure; SD, standard deviation; TBI, traumatic brain injury.

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Whilst cardiovascular instability following spinal cord injury is well recognised, the same in the setting of blunt traumatic brain injury (TBI) is less well described [3]. The cardiovascular instability following neurotrauma is poorly understood and felt to be multifactorial. Blunt trauma to the brain, particularly to the insular cortex, can result in catecholamine release and cause neuron-mediated cardiac arrhythmias. Catecholamine release can result in mitochondrial dysfunction, myocyte death and cardiac dysfunction, resulting in hypotension and cardiogenic shock.

Partrick et al. [4], found the incidence of hypotension in paediatric patients with isolated traumatic brain injury to range from 33% (age > 12) to 61% (0–5 year old). Mahoney et al. [5] found that isolated traumatic brain injury accounts for 13% of hypotensive episodes after blunt trauma in adult patients.

Acknowledging that isolated TBI may cause cardiovascular instability (tachycardia and/or hypotension) is crucial for delivering the best trauma care to the patient, both in the pre-hospital and hospital phases. Accurately establishing the cause of cardiovascular instability in the pre-hospital phase of trauma care is important, as the clinical interventions are likely to be very different. Patients with haemorrhagic shock may benefit from volume resuscitation

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with blood products, whereas those with neurotrauma are likely to need early anaesthesia and vasopressors. Recently, the occurrence of impact brain apnoea and how this can adversely cause cardiovascular instability has been highlighted [6].

Pre-hospital permissive hypotension is often used during the care of a major trauma patient [7], but this would not be the optimal treatment for patients with isolated TBI. It has been shown that isolated episodes of hypotension increases mortality in TBI patients [8]. Pre-hospital emergency medical care aims to prevent secondary brain injury through optimising cerebral perfusion pressure, oxygenation and ventilation. Accurately identifying isolated TBI as a cause of cardiovascular instability could avoid unnecessary blood product transfusion in the pre-hospital setting and allow patient care to be optimised. TBI patients can require pre-hospital anaesthesia, which can significantly affect patient haemodynamics. Having an understanding of the cardiovascular status and the impact TBI may have on this, is therefore important.

In this single centre, retrospective, observational study we sought to evaluate the frequency of cardiovascular instability (defined as heart rate (HR) >100 bpm and/or systolic blood pressure (SBP) <100 mmHg) in major trauma patients with confirmed isolated traumatic brain injury, requiring pre-hospital anaesthesia.

## Methods

Kent Surrey and Sussex Air Ambulance Trust (KSSAAT) is a Helicopter Emergency Medical Service (HEMS) covering three counties in the southeast England with a resident population of 4.5 million and transient population of up to 8 million. Two doctor/paramedic teams respond in either a helicopter or response car from two separate bases. The service attends approximately 2000 patients per year. KSSAAT uses a bespoke electronic patient clinical record system (HEMSbase, Medic One Systems Ltd, UK), which includes automated downloading of all pre-hospital physiology data. GCS is assessed by the attending HEMS team and recorded in component parts.

HEMSbase was interrogated for the time period 1 January 2015 to 31 December 2016. Two researchers (MG, MEZ) independently extracted the data and found no significant difference between the datasets. Inclusion criteria were adult ( $\geq 17$  year old) patients, who underwent pre-hospital rapid sequence induction (RSI) of anaesthesia, had pre-hospital signs of blunt neurotrauma, with isolated TBI subsequently confirmed on hospital computerised tomography (CT) scan. TBI on CT scan was defined as any formally reported radiological abnormality of suspected traumatic origin, other than isolated skull fracture. Paediatric patients were excluded owing to their varying normal cardiovascular physiology ranges and rare incidence of pre-hospital RSI.

We excluded patients who had a suspected medical event leading to traumatic injury, burns, hanging, patients who were pronounced life extinct at the scene and patients whose pre-hospital physiological data were missing. All pre-hospital RSI cases were reviewed. Individual medical records were reviewed to identify patients with pre-hospital cardiovascular instability using heart rate (HR) and systolic blood pressure (SBP). Cardiovascular instability was pragmatically defined as a single pre-RSI episode of either a HR >100bpm and/or SBP <100 mmHg. Patient records were also interrogated for Injury Severity Score (ISS) and mechanism of injury (MOI).

Patients whose CT results were missing or who died in hospital before having CT were allocated to a 'no follow up' group and were excluded from further analysis. Patients with normal CT scan results were allocated to a 'normal CT group' and also excluded from further analysis.

Patients with base of skull fractures were part of the Isolated TBI group, but were excluded from further analysis if CT showed no

intracranial pathology other than the fracture. Isolated TBI group was then subdivided into haemodynamically stable patients and those with signs of instability based on heart rate and systolic blood pressure.

This project met National Institute for Healthcare Research (NIHR, UK) criteria for service evaluation and formal ethical approval was therefore not required. The project was approved by the KSSAAT Research & Development Committee and registered as a service evaluation with the University of Surrey.

## Results

During this study period, KSSAAT undertook 3873 missions and treated 3485 patients. Patient inclusion is shown in Fig. 1.

Of the 3485 patients, 361 were adult patients who sustained blunt trauma, underwent RSI and pre-hospital signs of blunt neurotrauma. Patients with a medical event leading to minor head injury (GCS14/15 and not deemed to require pre-hospital anaesthesia,  $n = 25$ ), burns patient ( $n = 1$ ), hanging ( $n = 7$ ), patients who were pronounced life extinct (PLE) at the scene ( $n = 6$ ) and patients with missing physiological data ( $n = 5$ ) were excluded. This left 317 patients for further analysis.

Of the 317 undergoing pre-hospital anaesthesia, 39 had no follow up data and 22 had a normal CT scan, leaving 256 patients for further analysis. There were 123 patients with confirmed isolated TBI on CT, of which 2 had base of skull fractures with no intracranial pathology, leaving 121 (47.6%) patients in the isolated TBI group and 133 (52.4%) patients with polytrauma patients, with or without TBI.

In the Isolated TBI group, mean age was 54.9 years ( $SD \pm 20.7$ ) and median age was 58. In the polytrauma group mean age was 45.1 years ( $SD \pm 21.3$ ) and median was 40. The proportion of each gender was comparable in both groups. Patient demographics are shown in Table 1.

Out of 121 patients with confirmed isolated TBI, 68 were cardiovascularly stable throughout the pre-anaesthesia phase, while 53 (44%) showed signs of instability (HR >100 bpm and/or SBP <100 mmHg pre RSI). Hypotension with or without tachycardia was present in 14 (11.6%) patients. This is shown in Table 2.

Glasgow Coma Score (GCS) was similar in patients undergoing pre-hospital anaesthesia for both traumatic brain injury and polytrauma. The distribution of patients by GCS is shown in Table 3.

Injury Severity Score (ISS) was only available in 71 (59%) of patients with confirmed isolated brain injury. ISS for patients with isolated TBI is presented against pre-anaesthesia cardiovascular status in Table 4.

The most frequent mechanism of injury (MOI) for sustaining isolated TBI was a fall (from standing, from height or down stairs), in 72 (59.5%) cases. The other MOIs were Road Traffic Collisions (RTC),  $n = 43$  (35.5%); assault,  $n = 4$  (3.3%) and crush injury,  $n = 2$  (1.7%). The most frequent MOI among the hypotensive patients with isolated TBI were falls down stairs (4/14) and pedestrian vs motorised vehicle (4/14) as shown in Table 5.

In the cohort, the incidence of isolated TBI was higher in patients over the age of 55,  $n = 68$  (56%). The incidence of haemodynamic instability in relation to age group is shown in Table 6.

Out of 121 patients with isolated TBI, 10 (8.2%) received pre-hospital blood products (lyophilised plasma and/or packed red blood cells), 4 of which were hypotensive (SBP <100). This highlights the difficulty in pre-hospital management and decision making for the patients with vasoactive head injury in prehospital setting.

## Discussion

This retrospective, observational study demonstrates that hypotension in the pre-hospital phase following major trauma is relatively common in patients who do not have major haemorrhage.

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