


# Pre-hospital emergency anaesthesia in awake hypotensive trauma patients: beneficial or detrimental?

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## Conflicts of interest

The authors have no conflict of interests to declare.

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**Background:** The benefits of pre-hospital emergency anaesthesia (PHEA) are controversial. Patients who are hypovolaemic prior to induction of anaesthesia are at risk of severe cardiovascular instability post-induction.

This study compared mortality for hypovolaemic trauma patients (without major neurological injury) undergoing PHEA with a patient cohort with similar physiology transported to hospital without PHEA.

**Methods:** A retrospective database review was performed to identify patients who were hypotensive on scene [systolic blood pressure (SBP) < 90 mmHg], and GCS 13–15. Patient records were reviewed independently by two pre-hospital clinicians to identify the likelihood of hypovolaemia. Primary outcome measure was mortality defined as death before hospital discharge.

**Results:** Two hundred and thirty-six patients were included; 101 patients underwent PHEA. Fifteen PHEA patients died (14.9%) compared with six non-PHEA patients (4.4%),  $P = 0.01$ ; unadjusted OR for death was 3.73 (1.30–12.21;  $P = 0.01$ ). This association remained after adjustment for age, injury mechanism, heart rate and hypovolaemia (adjusted odds ratio 3.07 (1.03–9.14)  $P = 0.04$ ).

Fifty-eight PHEA patients (57.4%) were hypovolaemic prior to induction of anaesthesia, 14 died (24%). Of 43 PHEA patients (42.6%) not meeting hypovolaemia criteria, one died (2%); unadjusted OR for mortality was 13.12 (1.84–578.21). After adjustment for age, injury mechanism and initial heart rate, the odds ratio for mortality remained significant at 9.99 (1.69–58.98);  $P = 0.01$ .

**Conclusion:** Our results suggest an association between PHEA and in-hospital mortality in awake hypotensive trauma patients, which is strengthened when hypotension is due to hypovolaemia. If patients are hypovolaemic and awake on scene it might, where possible, be appropriate to delay induction of anaesthesia until hospital arrival.

## Editorial comment

Hypotensive but awake trauma patients in the field sometimes will need to be intubated, and there are alternatives for managing this safely. The study analyses a large experience with this issue, to try to understand how to avoid unnecessary negative circulatory results related to anaesthesia.

The timing, conduct and benefits of pre-hospital emergency anaesthesia (PHEA) remain highly debated<sup>1</sup> despite local and national guidelines addressing these issues.<sup>2,3</sup> There are a small proportion of trauma patients who cannot be managed effectively with basic airway interventions,<sup>4</sup> and in whom indications for PHEA are clear, including patients with airway obstruction, ventilatory failure or significantly reduced level of consciousness. There are also patients in whom the timing of anaesthesia is a more difficult decision and risks and benefits of pre-hospital intervention less clear. The small subgroup of patients with significant hypotension secondary to severe hypovolaemia who are conscious are one such group.

The potential complications of emergency anaesthesia are well described and include hypoxaemia, hypotension, oesophageal intubation and cardiovascular collapse.<sup>5–8</sup> Post-intubation hypotension is reported in 7.3% of patients undergoing PHEA<sup>9</sup> and up to 25% of patients undergoing in-hospital emergency anaesthesia.<sup>6</sup> Hypotension in these circumstances can be difficult to manage and is associated with an increased mortality.<sup>10</sup> It is likely to occur secondary to impairment of sympathetic innervation, vasodilatation from anaesthetic agents, decreased venous return caused by positive pressure ventilation, or further bleeding during an increased scene time. Few studies describe the immediate complications of emergency anaesthesia in the presence of pre-intubation hypotension. Where reported, pre-intubation hypotension is associated with a statistically significant increase in the number of episodes of post-intubation cardiac arrest.<sup>7</sup>

This pragmatic retrospective observational study aimed to compare mortality rates for severely injured hypovolaemic trauma patients (without evidence of major neurological injury) undergoing PHEA with a patient cohort with similar key physiology who were transported to hospital without PHEA. We hypothesized that a proportion of hypovolaemic patients may develop cardiovascular collapse soon after induction of anaesthesia. Collapse outside hospital without access to the full range of hospital-level resuscitation techniques and surgical intervention may result in higher mortality than delayed anaesthesia carried out after arrival in

hospital. The risks in these patients may outweigh the potential benefits of PHEA performed for more subjective indications such as humanitarian need, predicted clinical course or reduced overall time to surgery and definitive intervention. This may influence the practice of PHEA in this trauma patient subgroup.

## Materials and methods

### Study design

A retrospective database review of trauma patient records attended by a physician-led pre-hospital trauma service between 01/09/2009 and 31/08/2014.

### Study setting and pre-hospital care system

The study was conducted in a physician-led, pre-hospital trauma service based in London, UK, which serves a predominantly urban population of up to 10 million in an area of approximately 5000 square kilometres. A doctor-paramedic team is dispatched by helicopter during daylight hours and by fast response car at night. Dispatch is performed by flight paramedics in ambulance control with specific criteria targeting patients with severe injury.<sup>11</sup> A standard ground ambulance is always dispatched in addition to the physician-paramedic team.

### Inclusion criteria

1. Trauma patients attended on scene by the physician-paramedic team and escorted to hospital
2. Initial non-invasive SBP 90 mmHg or less (defined as first reading on arrival of doctor-paramedic team).
3. Initial GCS 13–15

Some patients did not have documented values for initial blood pressure and only the presence or absence of a radial or central pulse was recorded. A SBP of 90 mmHg or less was presumed to be present in those patients with a documented central pulse only. If 'radial pulse' was recorded instead of a numerical value the free text was examined to establish whether the patient had a good/strong radial pulse, which

was estimated to represent an SBP > 90 mmHg and subsequently excluded, or a weak/absent radial pulse which was estimated to represent an SBP < 90 mmHg.<sup>12,13</sup>

### Exclusion criteria

Children <16 years and non-trauma patients (including burns, drowning and hanging) were excluded.

### Data collection

Data from all patients attended by the physician-led pre-hospital trauma service are prospectively collected and recorded on patient report forms at the time of the incident, and subsequently entered into a Microsoft ACCESS™ database shortly after each mission by the attending physician. All physicians receive standardized training on data input and management prior to using the database. Data collected include patient demographics, mission timings and patient physiology. All interventions, including detailed information on the conduct of PHEA, volume and type of drugs and fluid administered, are recorded. A free text section is completed describing injury mechanism, injuries sustained and other relevant information. First and final blood pressure, pulse, oxygen saturations, and GCS are recorded. All observations (updated at 30-s intervals) are downloaded in numerical format from the patient monitor (Propaq MD™; Zoll, Boston, MA, USA) and printed after each mission.

The patient records of all patients included in the study were interrogated to identify patients with severe hypovolaemia on scene rather than mild to moderate hypovolaemia, or hypotension secondary to other causes such as neurogenic or cardiogenic shock, or tension pneumothorax. These patient records were reviewed independently by two pre-hospital clinicians (KC/MR) to determine the presence of severe hypovolaemia based on the factors described below. In two cases where the presence of severe hypovolaemia was still considered to be unclear, a third clinician was consulted (DL).

1. Pre-hospital clinical observations (blood pressure, heart rate and oxygen saturations) recorded on arrival at the patient and hospital

2. Pre-hospital request for blood on arrival at hospital or the need for an urgent blood transfusion on hospital arrival
3. Use of reduced dose of anaesthetic agents or analgesia because of suspected hypovolaemia
4. Suspected and confirmed injuries
5. Documented suspected hypovolaemia
6. Radiology reports
7. Operative findings

Data analysis was performed using statistical tests for categorical variables with Microsoft Excel™ (Microsoft, Redmond, WA, USA) and GraphPad™ (Graphpad Software, La Jolla, CA, USA). Fishers exact test was used to calculate statistical significance of proportions, with statistical significance considered to be  $P < 0.05$ . Exact logistic regression analysis was used to assess the univariate association between mortality and hypovolaemia and PHEA and to obtain univariate odds ratios (OR). For multivariable models, the penalized maximum likelihood estimation (Firth) method was used to compensate for small sample bias. Age, initial heart rate and mechanism of injury were selected for multivariate models as these variables would be readily available to all clinicians on scene making the decision as to whether or not to perform PHEA. The models were fitted in all patients and in PHEA subgroups. Differences in the odds ratio by PHEA were tested by including an interaction term in the model for all patients. Models were fitted using Stata Version 13 (StataCorp, College Station, TX, USA).

### PHEA standard operating procedure

Pre-hospital emergency anaesthesia is performed by the doctor-paramedic team in accordance with current UK recommendations,<sup>3</sup> and local standard operating procedure (SOP).<sup>14</sup> Emphasis is placed on simple reproducible techniques to achieve a high rate of successful first pass intubation and cardiovascular stability. All clinicians working with the trauma service receive intensive training in PHEA. In 1996, the service standardized drug choice for PHEA; etomidate was used for induction of anaesthesia and suxamethonium for muscle relaxation. If the attending clinical team felt addition of an opiate was required, morphine was used. The SOP was

revised in 2012 and standard induction since then has consisted of ketamine, a rapid onset long-acting muscle relaxant (rocuronium), and fentanyl as the opioid of choice. Patients with suspected or confirmed hypovolaemia receive reduced induction doses and in some cases the opioid is omitted altogether. Indications for PHEA include:

1. Actual or impending airway compromise
2. Ventilatory failure
3. Unconsciousness
4. Humanitarian need
5. Patients who are unmanageable or severely agitated after head injury
6. Anticipated clinical course

### Outcome measures

The primary outcome measure was mortality, defined as death before hospital discharge.

### Ethical approval

The study was performed retrospectively on routinely collected data; no additional interventions were performed. The project met local criteria for, and was registered as, a service evaluation project (ID number 5463, Barts Health NHS Trust).

### Results

In total, 9480 patients were attended by the pre-hospital trauma service in the study period; 265 patients (2.8%) met the study criteria and were included. Of these, 118 patients (44%) underwent PHEA; and 147 patients (56%) were managed without PHEA. Eight patients with burns and two patients with a suspected medical event preceding traumatic injury were excluded from final analysis. Outcome data were unavailable for 19 (7%) patients; 236 patients were included in the final data analysis.

Of the 236 patients included in the study, 152 (64.4%) had a numerical value for the SBP documented on arrival of the attending clinical team. In the remaining 84 patients, the initial blood pressure was recorded as radial, central or unrecordable. Of the patients who underwent PHEA, 46 of 101 patients (45.5%) had a

documented numerical value for initial SBP, compared with 106 of 135 patients (78.5%) who were not anaesthetized on scene.

### Mechanism of injury

The most common injury mechanisms were penetrating trauma (100 patients, 42%), and road traffic collision (81 patients, 34%), Table 1.

### Outcome for all patients

The outcome for all patients is described in Fig. 1. Twenty-one of 236 study patients died (8.9%). Pre-hospital emergency anaesthesia was associated with a statistically significant increase in mortality for all patients. The unadjusted OR for death using the exact logistic regression model was 3.73 (1.30–12.21;  $P = 0.01$ ). This association remained after adjustment for age, mechanism of injury, heart rate and hypovolaemia (adjusted OR 3.07 (1.03–9.14)  $P = 0.04$ , Table 2). There were no deaths due to failed intubation. Further analysis of the cause of death for each patient that died showed in all cases except one the death was attributable to hypovolaemia. One patient who did not undergo PHEA was subsequently found to have

**Table 1** Demographics

	Group 1 (No PHEA)	Group 2 (PHEA)	<i>P</i> value
Number of patients	135	101	
Penetrating trauma	81 (60.0%)	19 (18.8%)	< 0.001
Blunt trauma	54 (40.0%)	82 (81.2%)	
Age			
Median (IQR)	25 (20–42)	31 (23.5–43)	0.005
Initial SBP			
Median (IQR)	81 (76–86)	76 (65–85)	0.01
Initial HR			
Median (IQR)	90 (80–107)	114 (94–130)	< 0.001
Initial GCS			
13	7 (5.2)	25 (24.8)	< 0.001
14	38 (28.2)	37 (36.6)	
15	90 (66.7)	39 (38.6)	
Hypovolaemia			
<i>N</i> (%) yes	69 (51.1)	58 (57.4)	0.34

SBP, systolic blood pressure; PHEA, pre-hospital emergency anaesthesia.

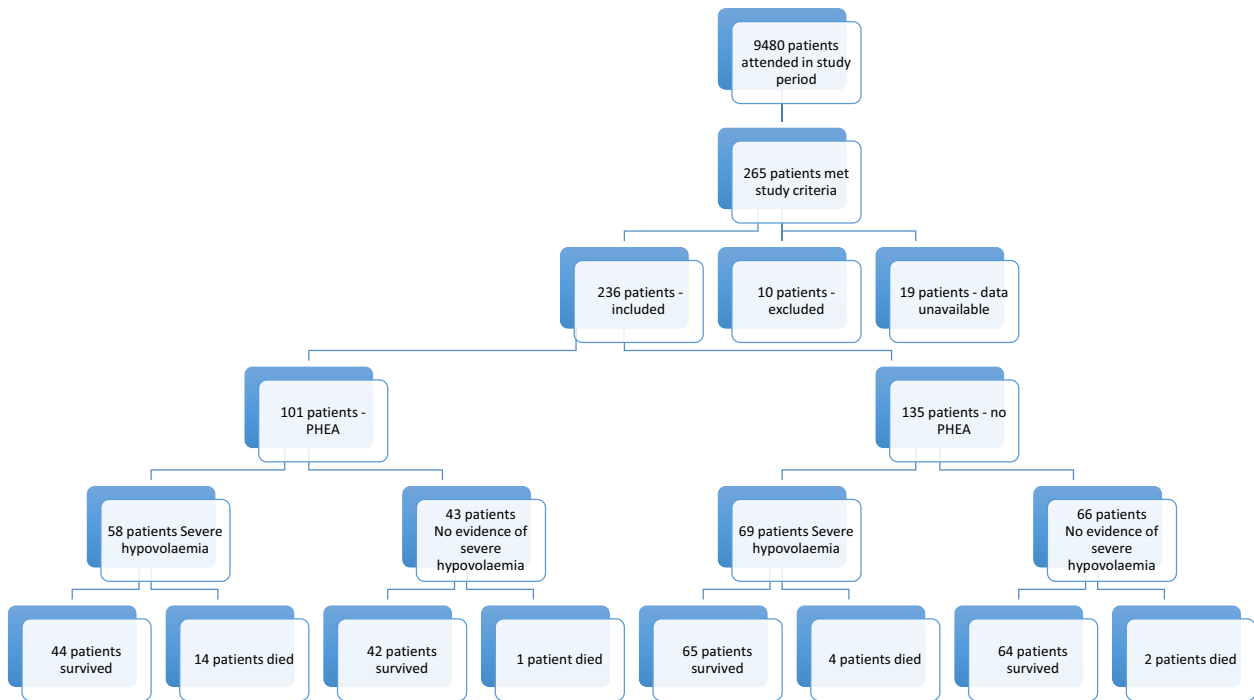


Fig. 1. Outcome for all patients

**Table 2** Multivariable logistic regression model for mortality in all patients.

Variable	Effect	Odds ratio (95% CI)	P value
Age	Per 10 year increase	1.15 (0.89–1.50)	0.29
Mechanism of Injury	Penetrating : Blunt	0.55 (0.17–1.78)	0.32
Initial heart rate	Per 1 standard deviation (SD) increase	0.79 (0.49–1.27)	0.32
Severe hypovolaemia	Yes: No	5.93 (1.74–20.23)	0.004
Intubated	Yes: No	3.07 (1.03–9.14)	0.04

a cardiac tamponade following a stabbing injury.

**Outcome for hypovolaemic trauma patients**

Using the criteria described, 127 of 236 patients had evidence of severe hypovolaemia. The remaining 109 patients were presumed to have

either moderate hypovolaemia, or hypotension secondary to other causes. Eighteen severely hypovolaemic patients died (14.2%) compared with three patients (2.8%) without evidence of severe hypovolaemia, ( $P = 0.003$ ). The unadjusted OR for death using the exact logistic regression model was 5.80 (1.62–31.62;  $P = 0.003$ ). This association remained after adjustment for age, mechanism of injury, heart rate and PHEA (adjusted OR 5.93 (1.74–20.23)  $P = 0.004$ , Table 2).

Of the 101 patients anaesthetized on scene, 58 patients (57.4%) were considered to be severely hypovolaemic prior to induction of anaesthesia, of these 14 died (24%). The unadjusted OR for hypovolaemia was 13.12 (1.84–578.21). After adjustment for age, mechanism of injury and initial heart rate, the OR for mortality remained significant at 9.99 (95% CI 1.69–58.98;  $P = 0.01$ ), Table 3.

There was no statistically significant difference in mortality demonstrated between hypotensive patients with and without additional evidence of severe hypovolaemia who did not undergo PHEA. In this subgroup of 135 patients, 69 (51.1%) were considered to have



**Table 3** Multivariable logistic regression model for mortality in PHEA patients.

Variable	Effect	Odds ratio (95% CI)	P value
Age	Per 10 year increase	1.21 (0.87–1.68)	0.26
Mechanism of injury	Penetrating : Blunt	1.38 (0.33–5.70)	0.66
Initial heart rate	Per 1 SD increase	0.71 (0.42–1.18)	0.18
Severe hypovolaemia	Yes: No	9.99 (1.69–58.98)	0.01

severe hypovolaemia, four (5.8%) died, compared with 66 patients (48.9%) without evidence of severe hypovolaemia, of whom two (3.0%) died,  $P = 0.681$ . The adjusted OR was 1.94 (0.37–10.08)  $P = 0.43$ .

### PHEA protocol

Sixty of 101 patients (59.4%) were intubated prior to the change of PHEA protocol, compared with 41 patients (40.6%) who were intubated using the revised drug protocol. Eleven of the 60 patients (18%) intubated before the change in protocol died, compared with four of 41 (10%) patients intubated following the change,  $P = 0.2693$ . Ninety of the 101 PHEA patients received an opioid. Of these, 49 of 58 (84%) patients who were considered to be severely hypovolaemic were given an opioid during their clinical course compared with 40 of 43 (93%) patients not considered to have severe hypovolaemia. Of the patients that died, 14 were considered to be severely hypovolaemic; 11 of these patients received an opioid. The single patient who died in the group of patients presumed not to be severely hypovolaemic also received an opioid but there was no significant difference in mortality identified that was attributable to opioid use,  $P = 0.3803$ .

### Discussion

This study supports the hypothesis that where patients are hypovolaemic and awake on scene it might be appropriate to delay induction of

anaesthesia until trauma centre arrival. Patients in this study were hypotensive adults with a GCS between 13 and 15 on arrival of the physician-led pre-hospital trauma team. The results demonstrate a statistically significant threefold increase in mortality for trauma patients who are initially hypotensive on scene and undergo PHEA, unadjusted OR 3.73 (1.30–12.21;  $P = 0.01$ ). This association remained after adjustment for age, mechanism of injury, heart rate and hypovolaemia (adjusted OR 3.07 (1.03–9.14)  $P = 0.04$ ). Patients with severe hypovolaemia who underwent PHEA had a 10-fold increase in mortality risk,  $P = 0.004$ , OR 9.99 (95% CI 1.69–58.98;  $P = 0.01$ ), adjusted for age, mechanism of injury and initial heart rate, when compared to PHEA patients without evidence of severe hypovolaemia. Significant haemorrhage was considered to be the cause of death in all but one patient who died, supporting the hypothesis that PHEA is associated with a higher mortality in hypovolaemic patients. These findings are consistent with other published data on this topic.<sup>15</sup> No association could be found between outcome and the change in drug protocol.

The immediate interventions required for each patient, including emergency anaesthesia, are made on an individual patient basis by experienced pre-hospital clinicians, with adherence to SOPs developed by the service. There is significant emphasis placed on performing only urgent interventions on scene to reduce unnecessary delay in transfer of the patient to hospital.

### Indications for PHEA

The indications for PHEA in trauma patients are often straightforward. Deeply unconscious patients or those with airway or ventilatory failure are obvious candidates for PHEA as soon as it is available. There are, however, some patients in whom the indications and timing of PHEA are less straightforward, including hypovolaemic trauma patients with high GCS. The majority of these patients will require surgery or interventional radiology to control bleeding and most will be anaesthetized in the emergency department or operating theatre. Emergency anaesthesia performed in-hospital for patients with cardiovascular compromise is often

delayed until the patient is in theatre and the surgeon is ready to proceed.<sup>16</sup>

### Haemodynamic effect of PHEA

There are potential advantages and disadvantages to PHEA in conscious hypovolaemic patients. Although PHEA inevitably increases scene time, it may improve the ease and speed of passage to the operating theatre, reducing the time spent in the emergency department. Disadvantages to PHEA are mostly related to alterations in patient physiology. Cardiovascular compromise or collapse following induction of anaesthesia is a major concern in hypovolaemic patients. Patients with significant haemodynamic instability rely on high endogenous sympathetic tone to maintain systemic vascular resistance and cardiac output. Drug choice in these patients is critical; ketamine is often considered to be the induction agent of choice in this setting.<sup>17–19</sup> It acts as a sympathomimetic, increasing circulating catecholamines causing direct cardiac stimulation and peripheral vasoconstriction, with preservation of the baroreceptor reflex. The consequences of these effects are observed as an increase in mean arterial pressure (MAP), pulmonary arterial pressure, heart rate and cardiac output.<sup>20</sup> An 10% increase in MAP is reported in emergency surgical patients.<sup>17</sup> Other induction agents often cause a fall in cardiac output and MAP. Thiopentone is associated with negative inotropy, arteriolar vasodilatation and obtunded baroreceptor reflexes.<sup>21</sup> Propofol causes hypotension through reduced systemic vascular resistance and myocardial depression.<sup>20</sup> The use of propofol as an induction agent has a statistically significant association with early post-induction hypotension (in the first 10 min post-induction).<sup>19</sup> Animal data suggest significantly increased blood loss at specific time points post-injury in swine anaesthetized with propofol compared with those managed via face-mask ventilation, without intubation.<sup>22</sup> Etomidate does not demonstrate the same degree of cardiovascular instability but adverse effects on the adrenal axis have limited its popularity.<sup>23,24</sup>

Opioids are administered particularly in patients where the hypertensive response to laryngoscopy is likely to be detrimental. They

can result in reduction of sympathetic tone and contribute to post-induction hypotension.<sup>25</sup> Severe hypovolaemia secondary to haemorrhagic shock results in significant alterations in fentanyl pharmacokinetics showing reduced central clearance and volume of distribution when compared with control subjects.<sup>26</sup> Low protein binding, which may occur in hypovolaemic patients, particularly following crystalloid resuscitation, increases the drug free fraction and results in higher effector site concentration. This may worsen the adverse haemodynamic effects of the drug.<sup>17,19</sup>

There are predictable patterns of ventilation and perfusion observed in anaesthetized, ventilated patients. Perfusion increases from superior to inferior parts of the lungs with some reduction in blood flow in the most inferior parts secondary to atelectasis. Ventilation tends to be greatest in the superior lung and reduced inferiorly. Implementation of positive pressure ventilation in hypovolaemic patients results in reduced venous return<sup>27</sup> which may precipitate cardiac arrest in compromised patients. The application of positive end-expiratory pressure redistributes blood flow to dependent parts of the lungs, usually dorsal in supine anaesthetized patients and may worsen shunting due to limited ventilation, and increase dead space in non-dependent parts of the lung,<sup>28</sup> which may worsen gas exchange. Ventilation strategies to minimize rises in intrathoracic pressure during ventilation might reduce the frequency and degree of post-induction hypotension/cardiovascular collapse. Muscle relaxants and mechanical ventilation decrease the work of ventilation which may be beneficial in hypoxic patients.

### Other factors influencing outcome after PHEA

One major factor affecting outcome from PHEA is the incidence of failed intubation. There were no failed intubations included in this study and the service has previously reported high intubation success rates of 99.3%.<sup>14</sup>

Significant hypothermia is more frequent in anaesthetized patients and may increase bleeding. Previous data from this service demonstrated significantly lower body temperatures at hospital admission in PHEA patients,<sup>29</sup> which

has been shown to correlate with increased mortality.<sup>30</sup>

Few studies focus on the outcome of patients who are hypotensive and hypovolaemic prior to induction of anaesthesia. This study found 2.8% of patients attended were significantly hypotensive prior to induction of anaesthesia. Patients considered to be hypovolaemic secondary to major haemorrhage have a 'care bundle' of interventions carried out on scene including careful handling to minimize clot disruption, use of splints, tourniquets and haemostatic agents to control external haemorrhage, administration of tranexamic acid, pre-hospital activation of massive haemorrhage protocols and administration of on-scene packed red cells. More radical resuscitation interventions, such as pre-hospital thoracotomy for cardiac tamponade and resuscitative endovascular balloon occlusion of the aorta are also considered where indicated.<sup>31</sup> Many patients in this study were attended before this service carried packed red cells. This treatment option was introduced in 2012.<sup>32</sup> It may be that the potential detrimental effects of PHEA in hypovolaemic patients could be mitigated by peri-induction blood transfusion, and this is commenced where time permits. Studies to examine the effects of blood transfusion in our service are in progress.

Prior to providing blood on scene, the concept of declaring a 'Code Red' pre-alert to the hospital receiving the trauma patient, was introduced in 2009. The strategy was designed to permit early activation of hospital major haemorrhage protocols, so packed red cells and other blood products were available for patients when on arrival. It has been shown to improve delivery of blood products and reduce waste.<sup>33</sup> Code red is based on pre-hospital clinical assessment and declared if there is suspected active haemorrhage and a SBP < 90 mmHg.<sup>32</sup> The use of pre-hospital blood is associated with an improved rate of return of spontaneous circulation following traumatic cardiac arrest from hypovolaemia, but a survival benefit in this service has not been demonstrated to date.<sup>34,35</sup>

Use of novel therapies to reduce the incidence of cardiovascular collapse may be considered in this subgroup of trauma patients. Impedance threshold devices (ITDs) have been shown to augment venous return and improve SBP and

pulse pressure in animal studies of hypovolaemic shock.<sup>36</sup> ITD use is described in spontaneously ventilating pigs and it may be possible to translate this to clinical practice in the pre-hospital setting by using the device in awake hypovolaemic trauma patients to improve haemodynamic stability during transfer to hospital and reduce the need for PHEA in this patient subgroup. Alternatively, if PHEA is required the device may be applied with positive pressure ventilation to support the blood pressure and reduce the incidence of cardiovascular collapse.

### Limitations

The study was a retrospective database review based in a pre-hospital trauma service staffed by physicians and paramedics who have undergone extensive training in pre-hospital emergency medicine. It is a pragmatic study based on day-to-day practice and experience gained from the management of pre-hospital trauma patients. One significant flaw is the lack of numerical initial SBP values for all patients but this is a reflection of the patient population attended by the service. Initial identification of patients was objective but subsequent review by the authors to identify hypovolaemia was, in part, subjective, which may cause bias. Data were unavailable for 7% of the study population. The PHEA SOP was revised during the study. Regular SOP review is carried out within the trauma service as part of a clinical governance system. The relevant major changes were a change in induction agent from etomidate to ketamine and a change in opioid from morphine to fentanyl. Another potential limitation is that the exact reasons some patients with hypotension and high GCS were anaesthetized and some were not. Although the authors believe that this was mainly due to different preferences and decision-making of the attending physicians, there is the possibility that other factors – for example, clinical appearance or agitation indicating more severe hypovolaemia or injury severity – may have been present. The only way to eliminate this limitation would be to randomize patients prospectively with the same key physiology. It was not possible to obtain coroner reports identifying the exact cause of death for each patient and the association between



mortality and hypovolaemia is based on physiological observations, the requirement for blood transfusion, radiology reports and operative findings. Finally, the scene times are not reported in this study. However, the system is subject to rigorous governance and any prolonged scene times are identified and thoroughly investigated. The authors do not believe that prolonged scene times are a contributing factor in patient demise. A previous study from the same service reported average scene times of 42 min for patients undergoing PHEA vs. 25 min for patients who do not receive PHEA.<sup>29</sup> However, patients in the PHEA group were more complex and had more on-scene interventions than those without PHEA, so the extended scene times may have been due to more than conduct of anaesthesia.

### Conclusions

This study demonstrates an association between PHEA and mortality in hypotensive trauma patients. The effect on mortality is strengthened when haemodynamic instability is identified by the pre-hospital team as being likely due to significant hypovolaemia. Whilst it is not possible to account for all factors that influence the decision to anaesthetize a patient on scene, the study supports the view that physicians considering PHEA in conscious patients with hypotension secondary to hypovolaemia must be fully conversant with the risks of the procedure and see clear potential benefits in performing PHEA distant from surgical intervention. In these patients, it may be appropriate to delay anaesthesia until arrival at hospital or have in place strategies to reduce the detrimental physiological effects of PHEA.

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### Author contribution

K. C.: Data collection and co-author of first and subsequent drafts.

M. R.: Co-author of first and subsequent drafts, reviewed and constructively criticized the manuscript.

K. B.: Co-author of first and subsequent drafts, reviewed and constructively criticized the manuscript.

D. L.: Conceived the study, co-author of first and subsequent drafts, reviewed and constructively criticized the manuscript.

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