

# Anaesthesiologist-provided prehospital airway management in patients with traumatic brain injury: an observational study

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**Background** Guidelines recommend that patients with brain trauma with a Glasgow Coma Scale (GCS) score of less than 9 should have an airway established. Hypoxia, hypotension and hypertension as well as hypoventilation and hyperventilation may worsen outcome in these patients.

**Objectives** The objectives were to investigate guideline adherence, reasons for nonadherence and the incidences of complications related to prehospital advanced airway management in patients with traumatic brain injury.

**Materials and methods** We prospectively collected data from eight anaesthesiologist-staffed prehospital critical care teams in the Central Denmark Region according to the Utstein-style template.

**Results** Among 1081 consecutive prehospital advanced airway management patients, we identified 54 with a traumatic brain injury and an initial GCS score of less than 9. Guideline adherence in terms of airway management was 92.6%. The reasons for nonadherence were the patient's condition, anticipated difficult airway management and short distance to the emergency department. Following rapid sequence intubation (RSI), 11.4% developed oxygen saturation below 90%, 9.1% had a first post-RSI systolic blood pressure below 90 mmHg and 48.9% had a first post-RSI systolic blood pressure

below 120 mmHg. The incidence of hypertension following prehospital RSI was 4.5%. The incidence of postendotracheal intubation hyperventilation was as high as 71.1%.

**Conclusion** The guideline adherence was high. The incidences of post-RSI hypoxia and systolic blood pressure below 90 compare with the results reported from other physician-staffed prehospital services. The incidence of systolic blood pressure below 120 as well as that of hyperventilation following prehospital endotracheal intubation in patients with traumatic brain injury call for a change in our current practice. *European Journal of Emergency Medicine* 21:418–423 © 2014 Wolters Kluwer Health | Lippincott Williams & Wilkins.

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## Background

Current guidelines for the prehospital treatment of patients with traumatic brain injury (TBI) [1,2] recommend that an airway should be established in TBI patients with a Glasgow Coma Scale (GCS) score of less than 9.

Nonadherence to guidelines may, however, be a problem. In Amsterdam, Franschman *et al.* [3] found that only 56% of patients with TBI and a GCS of less than 9 were treated with prehospital endotracheal intubation (PHETI) according to guidelines [3].

The main reason for performing PHETI on TBI patients is to avoid secondary brain damage by preventing and treating hypoxia [peripheral oxygen saturation (SPO<sub>2</sub>) <90%] and hypoventilation [end-tidal CO<sub>2</sub> (ETCO<sub>2</sub>) >5.3 kPa] [1,2,4]. However, hypoxia [5–7], hypotension [6–8] and hypertension

[9,10] are associated with PHETI even in physician-staffed prehospital services. These complications as well as both hypoventilation and hyperventilation may enhance secondary brain damage [1,2].

We are not aware of any Danish studies investigating the guideline adherence or the incidences of complications related to PHETI in TBI patients.

## Objectives

The objectives of this study were as follows:

- (1) To investigate the guideline adherence to the airway management recommendations in the current guidelines for the prehospital treatment of TBI patients [1,2].
- (2) To investigate the reasons for nonadherence to TBI guidelines.
- (3) To estimate the incidence of hypoxia and hypotension following prehospital rapid sequence intubation (RSI) in both patients with an isolated TBI and an

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initial GCS of less than 9 and patients with TBI, polytrauma and an initial GCS of less than 9.

- (4) To estimate the incidence of hypertension following PHETI in patients with TBI and an initial GCS of less than 9.
- (5) To estimate the incidence of macroscopic aspiration of blood or gastric contents to the lungs following PHETI in patients with TBI and an initial GCS of less than 9.
- (6) To estimate the incidences of hypoventilation and hyperventilation following PHETI intubation in patients with TBI and an initial GCS of less than 9.

## Materials and methods

### Study design

We prospectively collected prehospital advanced airway management (PHAAM) data according to the consensus-based Utstein-style template [11].

### Setting

We investigated the performance of the anaesthesiologist-staffed prehospital critical care teams in the Central Denmark Region, a mixed urban and rural area with a population of 1 270 000 and an overall population density of 97.7 inhabitants/km<sup>2</sup>.

The emergency medical service (EMS) in the region is a two-tiered system based on 64 road ambulances staffed by emergency medical technicians (EMTs) supported by 10 prehospital critical care teams staffed with an anaesthesiologist and a specially trained EMT [7,12].

There is a written local guideline but no standard operating procedures (SOPs) on the prehospital treatment of TBI patients for the prehospital critical care teams in the region. The written guideline is in coherence with the guidelines for the prehospital management of TBI patients [2]. There are no SOP or guidelines for the performance of prehospital RSI in our services.

We collected data between 1 February 2011 and 1 November 2012.

### Participants

*Inclusion criteria:* consecutive prehospital patients of all ages with TBI and an initial GCS of less than 9.

*Exclusion criteria:* interhospital transfers.

### Endpoints and variables

The primary endpoint was the incidence of PHETI.

The secondary endpoints were (a) anaesthesiologists' reasons for not performing PHETI, (b) post-RSI hypoxia, (c) post-RSI hypotension, (d) post-PHETI hypertension, (e) macroscopic aspiration of blood or gastric contents into the lungs during RSI (detected during laryngoscopy), (f) hypoventilation following PHETI and (g) hyperventilation following PHETI.

The anaesthesiologists registered their reasons for not performing PHAAM. We categorized the reasons as follows:

- (1) Expected difficult airway management (as defined by the individual prehospital critical care anaesthesiologist).
- (2) Difficult or limited access to the patient.
- (3) Short transport distance to the emergency department (as defined by the individual prehospital critical care anaesthesiologist).
- (4) Aspects of the patient's current medical condition.
- (5) The patient's comorbidity.
- (6) Physician's lack of training or experience with the type of patient in question.
- (7) Lack of proper equipment.
- (8) No assistance available.
- (9) Other.

Reason 2 would, for instance, include patients in cardiac arrest in a location where PHAAM would be difficult but where bag-mask ventilation could be provided. If the anaesthesiologist then terminated CPR on scene, PHAAM was classified as having been waived because of difficult access to the patient. Reason 4 included both patients in whom advanced critical care was considered futile and patients in whom the attending prehospital critical care anaesthesiologist assessed the risks associated with prehospital PHAAM to outweigh the potential benefits. Reason 5 included patients in whom comorbidity rendered PHAAM unethical and for whom palliative care were more appropriate.

As in the consensus-based template [11], we defined hypoxia as a peripheral oxygen saturation (SpO<sub>2</sub>) below 90% and hypotension as systolic blood pressure (SBP) below 90 mmHg. We have not been able to identify any limits for hypertension in TBI patients and we therefore display the relative changes in SBP following PHETI/prehospital RSI along with the number of patients with a post-PHETI SBP above 180 mmHg. We compared the first SBP measured after arrival of the first EMS unit with the first post-PHETI/post-RSI SBP. We also calculated the incidence of post-RSI SBP below 120 mmHg as recommended by Brenner *et al.* [13], who showed that this may be a better target SBP to avoid secondary brain damage in TBI patients.

We defined hyperventilation as an ETCO<sub>2</sub> less than 4.5 and hypoventilation as an ETCO<sub>2</sub> more than 5.3 kPa [1,2] upon arrival at the emergency department.

Exposure variables were pre-RSI hypoxia, pre-RSI hypotension and induction agents used during RSI. We defined drug-assisted PHETI as PHETI performed with the use of any analgesic or sedative drugs without the use of a neuromuscular blocking agent (NMBA) and RSI as PHETI aided by the use of any combination of (a) a sedative OR an analgesic drug AND (b) an NMBA.

The prehospital critical care teams measured SpO<sub>2</sub>, heart rate and blood pressure using a LifePak 12 monitor (Physio-Control, Redmond, Washington, USA) and monitored ETCO<sub>2</sub> either by the LifePak 12 or by a Nellcor NPB-75 capnograph (Tyco Healthcare Group LP, Pleasanton, California, USA).

### Data sources and data collection

We collected and managed data from eight prehospital critical care teams, including the HEMS [7].

### Bias

To reduce the risk of recall bias and selection bias, the primary investigator reviewed the registration forms on a day-to-day basis. We cross-checked the registration forms with the prehospital records from the prehospital critical care teams to optimize data coverage. In cases of missing data or inconsistencies, we asked the attending anaesthesiologist to provide additional details for clarification.

### Statistical analysis

We analysed the data in the statistical program Stata 12 (StataCorpLP, Lakeway Drive College Station, Texas, USA). In the rare cases of missing data, we carried out complete case analyses.

### Ethics

No patients had their treatment altered because of the study.

All physicians participated in the study on a voluntary basis – there were no refusals.

The study did not involve any alterations from normal practice and according to Danish law, it did not need the approval of the Regional Ethics Committee nor did we need the patients' consent to register and publish the data. The Danish Data Protection Agency approved the study (journal number 2013-41-1462).

## Results

### Participants

Among 1081 consecutive PHAAM patients, we identified 60 TBI patients (5.6%). Of these, 45 patients had an isolated TBI, 15 patients had both a TBI and polytrauma and 54 patients had an initial GCS of less than 9.

### Descriptive data

Table 1 shows demographic data from the 54 TBI patients with an initial GCS of less than 9.

### Adherence to guidelines

The adherence to the airway management recommendations in the guidelines for prehospital treatment of TBI patients is 92.6% in our system; 50 out of the 54 TBI patients with an initial GCS of less than 9 had their tracheas intubated.

### The anaesthesiologists' reason for not performing PHETI

The reasons for not performing PHETI were as follows:

Patient A – patient's condition and short distance to the ED.

Patient B – anticipated difficult intubation and short distance to the ED.

Patient C – patient's condition.

Patient D – patient's condition.

Patient B had his trachea intubated without difficulties in the ED.

### The incidence of post-RSI complications

The incidences of post-RSI hypoxia and post-RSI hypotension in patients with an initial GCS of less than 9 following isolated TBI and TBI + polytrauma are shown in Table 2.

Five patients developed a post-RSI SpO<sub>2</sub> below 90%, whereas four had an initial post-RSI SBP below

**Table 1** Demographic data of patients with TBI and an initial GCS < 9

	N (%)
Total patients included (N)	54
Age [mean (range)] (years)	44.6 (1–86)
< 16	3 (5.6)
< 2	1 (1.9)
Males	33 (61.0)
ASA-PS score [mean (range)]	1.4 (1–3)
Pre-existing cardiac disease	2 (3.7)
Pre-existing hypertension	6 (11.1)
Pre-existing COLD <sup>a</sup>	0 (0)
Pre-existing diabetes	1 (1.9)
Pre-existing neurological disease	1 (1.9)
Other pre-existing disease	5 (9.3)

ASA-PS, American Society of Anesthesiologists Physical Status; GCS, Glasgow Coma Scale score; TBI, traumatic brain injury.

<sup>a</sup>Chronic obstructive pulmonary disease.

**Table 2** The incidences of post-RSI hypoxia and post-RSI hypotension in TBI patients with an initial GCS < 9

Pre-RSI status (N=44)	Post-RSI hypoxia (SpO <sub>2</sub> < 90%)	Post-RSI hypotension (SBP < 90 mmHg)
Isolated TBI (n=26)	0	0
Isolated TBI + hypoxia (n=6)	4	0
Isolated TBI + hypotension (n=1)	0	1
TBI + polytrauma (n=7)	1	0
TBI + polytrauma + hypoxia (n=1)	0	0
TBI + polytrauma + hypotension (n=2)	0	2
TBI + polytrauma + hypoxia and hypotension (n=1)	0	1

GCS, Glasgow Coma Scale score; SpO<sub>2</sub>, peripheral oxygen saturation; RSI, rapid sequence intubation; SBP, systolic blood pressure; TBI, traumatic brain injury.

**Table 3** Relative changes in SBP following PHETI in patients with TBI and a GCS < 9

Changes in systolic blood pressure (%)	Non-RSI		
	RSI (n=44)	drug-assisted PHETI (n=1)	PHETI without drugs (n=5)
>30% decrease	9	–	–
21–30% decrease	4	–	1
11–20% decrease	6	–	–
10% decrease to 10% increase	18	1	2
11–20% increase	1	–	–
21–30% increase	0	–	–
>30% increase	2	–	2 <sup>a</sup>
Incomplete data	–	–	–

GCS, Glasgow Coma Scale score; PHETI, prehospital endotracheal intubation; RSI, rapid sequence intubation; SBP, systolic blood pressure; TBI, traumatic brain injury.

<sup>a</sup>Both resuscitated after cardiac arrest.

**Table 4** The incidences of different degrees of hypotension following RSI with different anaesthesia induction agents

Induction agent	Number of patients	
	Post-RSI SBP < 90 mmHg	Post-RSI SBP < 120 mmHg
Thiopental (n=0)	0	0
Thiopental + opioid (n=16)	2	8
Propofol (n=0)	0	0
Propofol + opioid (n=22)	4	12
S-ketamine (n=4)	2	0
S-ketamine + opioid (n=3)	0	2

RSI, rapid sequence intubation; SBP, systolic blood pressure.

90 mmHg. Twenty-two out of the 45 patients had an initial post-RSI SBP below 120 mmHg.

We present the relative changes in SBP following PHETI and prehospital RSI in Table 3. Two out of the nine patients with a decrease in SBP of more than 30% following RSI had a pre-RSI SBP of more than 220 mmHg. None of the patients with a relative increase in SBP of more than 30% attained an SBP of over 140 mmHg. No patient had a post-PHETI SBP above 180 mmHg.

We show the incidences of both SBP of less than 90 mmHg and SBP of less than 120 mmHg related to the different induction agents used during RSI in Table 4. The prehospital critical care teams performed RSI in 44 cases and drug-assisted PHETI in one case. Three cardiac arrest patients and two noncardiac arrest patients with an initial GCS of 3 had their tracheas intubated without the use of drugs.

Three patients developed macroscopic aspiration of blood or gastric contents into the lungs during RSI.

The incidence of hypoventilation was 4.4% (n = 2) and the incidence of hyperventilation was 71.1% (n = 32) upon arrival at the emergency department. Of the hyperventilated patients, 24 had an ETCO<sub>2</sub> between 4.0 and 4.5; 18 of these patients had an initial GCS of 3. ETCO<sub>2</sub> data were incomplete in seven of the cases.

## Discussion

### Adherence to guidelines

The guideline adherence is high and compares with that reported from the physician-staffed EMS in Amsterdam [3]. The evidence behind these guidelines is still being debated and the decision on whether to perform PHETI or not must be made on the basis of a case-to-case evaluation of the possible advantages and risks associated with PHETI. A 100% guideline adherence may not be appropriate.

### The physicians' reasons for not performing PHETI

Waiving PHETI because of the patient's condition or a suspected difficult PHAAM may be highly appropriate. It is encouraging that a short transport to the ED was not the sole reason for withholding PHAAM in any of the TBI patients in our study as patient packaging and loading as well as unloading and ED handover quickly add minutes to the transport time itself.

### The incidence of post-RSI complications

The overall incidence of post-RSI hypoxia and post-RSI hypotension in TBI patients mirrors what we have reported from the entire prehospital RSI population in our system [7] and compares with those reported from other physician-staffed prehospital services [5,6,8,14]. The incidences of hypoxia following RSI on already hypoxic TBI patients and that of hypotension following RSI on already hypotensive TBI patients are considerably higher than those in patients who were not hypoxic or hypotensive before the RSI. This may indicate a need for better pre-RSI preparations and altered RSI regimes in these patients. We may implement some of the different strategies by Eich *et al.* [15] and Weingart and Levitan [16] to avoid desaturation during RSI.

It may also be, however, that these patients had suffered such extensive injuries that post-RSI hypoxia or hypotension was virtually unavoidable.

The fact that nearly half of the TBI patients with a GCS of less than 9 in our system experience a SBP below 120 is worrying and necessitates changes in our RSI practice in these patients. One may speculate that the introduction of a SOP for the use of S-ketamine as an induction agent in these patients may reduce the incidence of post-RSI hypotension, even though our result shows that the use of S-ketamine is no guarantee against post-RSI hypotension. We did not design this study to detect differences between induction agents; we included the results in Table 4 only to describe the current practice in our system.

The incidence of post-RSI hypertension in our study is considerably lower than the 80% reported from the Kent, Surrey and Sussex HEMS by Perkins *et al.* [9,10]. This may be because the anaesthesiologists in our prehospital critical care teams often used both an opiate (fentanyl or

alfentanil) and an anaesthetic drug when performing RSI. This is in contrast to the SOP for RSI in many British HEMS [6,8,9,17]. There might be a theoretical advantage in not using opiates during RSI if the patient is severely haemodynamically compromised as the use of opiates might increase the risk of post-RSI hypotension. However, both the overall incidence of post-RSI hypotension (SBP < 90 mmHg) [7] and the post-RSI hypotension in TBI patients are lower in our system than the overall incidence of post-RSI hypotension reported from London HEMS [6]. Perkins and colleagues suggest that their routine use of a gum-elastic bougie (GEB) during RSI may be part of the explanation for their high incidence of post-RSI hypertension because the GEB stimulates the trachea and carina more during intubation than the endotracheal tube does on its own. We only use the GEB as an airway back-up device in our system.

We do not know the limit at which hypertension becomes harmful to the injured brain. The relative importance of hypotension versus hypertension in preventing secondary brain damage in TBI patients is, to our knowledge, also unknown.

The incidence of macroscopic aspiration in the current material compares with that in the overall PHAAM population in our system [7]. Our incidence is considerably lower than that found by Lockey *et al.* [18]. A possible explanation for this may be a difference in case mix, but we speculate that it might also be because of the London HEMS SOP for RSI and their routine use of a GEB. This mode of intubation along with the rather extensive check list used may result in a longer time used to establish a secure airway, possibly increasing the risk of aspiration.

The incidence of hyperventilation following PHETI is high, although most hyperventilated patients were only moderately hyperventilated (ETCO<sub>2</sub> 4.0–4.5 kPa). Current guidelines [1,2] state that an ETCO<sub>2</sub> below 4.5 kPa should be avoided unless the patient shows signs of cerebral herniation. It is unlikely that all the patients with an ETCO<sub>2</sub> 4.0–4.5 kPa upon arrival to the ED had signs of cerebral herniation, although most of them had an initial GCS of 3. Warner *et al.* [19] have reported a poor correlation between ETCO<sub>2</sub> and arterial CO<sub>2</sub> in severely injured patients. The authors report that trauma patients ventilated according to the recommended ETCO<sub>2</sub> values are susceptible to hypoventilation, especially if they have signs of poor tissue perfusion. This may complicate the decision-making on what ETCO<sub>2</sub> range is the best for TBI patients. In our study, only four patients had an SBP below 90 after PHETI and the effect of the discrepancy found by Warner *et al.* [19] is probably limited. One may speculate, though, whether the patients ventilated to an ETCO<sub>2</sub> between 4.0 and 4.5 really were hyperventilated. To answer this question, we would need arterial blood gas analyses. The combined incidence of hypoventilation and

hyperventilation in our material is higher than or comparable with those reported from paramedic-based prehospital systems in the USA [20,21]. A change in our clinical practice may be necessary to reduce the incidence of hypoventilation and hyperventilation.

### Limitations

The main limitation of the current analyses is that the attending anaesthesiologists registered all the data. They are therefore subject to registration bias and recall bias. The high capture rate reduces the risk of selection bias. On the basis of the day-to-day cross-check of the registration forms against both the written prehospital journals and the compulsory entries made by the anaesthesiologists in the patients' hospital records, the extent of selection bias is probably limited.

We recognize that the method used for blood pressure measurement in this study is not optimal as short peaks and extremely low values may have been overlooked.

We did not validate the prehospital ETCO<sub>2</sub> values by arterial blood gas analyses upon arrival in the emergency department. This may have influenced the apparent incidences of hypoventilation and hyperventilation [19].

TBI resulting in an initial prehospital GCS of less than 9 is rare in our system. The number of patients in this prospective study is therefore limited and caution is advised when interpreting the results.

### Generalizability

We believe that our results can be valuable to other physician-staffed prehospital services as well as when comparing the performance and patient safety of different prehospital systems with different staffing. The discussion of post-RSI complications is relevant to all prehospital systems performing RSI.

### Perspectives

These results may contribute to the ongoing debate on the optimal prehospital treatment of TBI patients. Further research is needed to identify the effect of different treatment protocols on patient safety and outcome.

### Conclusion

Adherence to the airway management recommendations made in current guidelines for the prehospital treatment of TBI is satisfactory in our anaesthesiologist-staffed prehospital critical care teams. The incidences of oxygen saturation below 90% and SBP below 90 mmHg following prehospital RSI are acceptable but the high incidence of SBP below 120 mmHg as well as that of hyperventilation following PHRTHI necessitate changes in our clinical practice.

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## Conflicts of interest

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