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Prehospital fluid resuscitation in hypotensive trauma patients: Do we need a tailored approach?

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ABSTRACT

Aim: The ideal strategy for prehospital intravenous fluid resuscitation in trauma remains unclear. Fluid resuscitation may reverse shock but aggravate bleeding by raising blood pressure and haemodilution. We examined the effect of prehospital i.v. fluid on the physiologic status and need for blood transfusion in hypotensive trauma patients after their arrival in the emergency department (ED).
Methods: Retrospective analysis of trauma patients ($n = 941$) with field hypotension presenting to a level 1 trauma centre. Regression models were used to investigate associations between prehospital fluid volumes and shock index and blood transfusion respectively in the emergency department and mortality at 24 h.
Results: A 1 L increase of prehospital i.v. fluid was associated with a 7% decrease of shock index in the emergency department ($p < 0.001$). Volumes of 0.5–1 L and 1–2 L were associated with reduced likelihood of shock as compared to volumes of 0–0.5 L: OR 0.61 ($p = 0.03$) and OR 0.54 ($p = 0.02$), respectively. Volumes of 1–2 L were also associated with an increased likelihood of receiving blood transfusion in ED: OR 3.27 ($p < 0.001$). Patients who had received volumes of >2 L have a much greater likelihood of receiving blood transfusion in ED: OR 9.92 ($p < 0.001$). Mortality at 24 h was not associated with prehospital i.v. fluids.
Conclusion: In hypotensive trauma patients, prehospital i.v. fluids were associated with a reduction of likelihood of shock upon arrival in ED. However, volumes of >1 L were associated with a markedly increased likelihood of receiving blood transfusion in ED. Therefore, decision making regarding prehospital i.v. fluid resuscitation is critical and may need to be tailored to the individual situation. Further research is needed to clarify whether a causal relationship exists between prehospital i.v. fluid volume and blood transfusion. Also, prospective trials on prehospital i.v. fluid resuscitation strategies in specific patient subgroups (e.g. traumatic brain injury and concomitant haemorrhage) are warranted.

Introduction

In high-income countries, injury is the leading cause of death among people aged 15–44 years [1]. Approximately, 16% of the global disease burden can be attributed to injuries [1]. Haemorrhage is responsible for 30–40% of trauma mortality, and of these deaths, 33–56% occur during the prehospital period [2]. Moreover, haemorrhage is recognized as the leading cause of preventable death in the initial 24 h after admission to the hospital [3]. Management of haemorrhage and shock in trauma patients comprises many diagnostic and treatment options along the chain of survival [4]. Treatment is initiated in the field where, next to efforts to stop bleeding from external wounds and fractures, resuscitation with intravenous (i.v.) fluid is started before or during rapid transport to a definitive care facility. Historically, i.v. fluid resuscitation consists of early rapid volume replacement (3:1

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volume of blood loss replaced with crystalloids) based on the idea that restoration of circulating volume and blood pressure will help to maintain vital organ perfusion [5–7]. However, there is some evidence that resuscitation with i.v. fluids prior to haemorrhage control may be detrimental in trauma by aggravating bleeding (raising blood pressure) and by worsening of coagulopathy (haemodilution) [5,8]. Also, overzealous resuscitation with i.v. fluids may cause tissue oedema leading to significant morbidity and mortality [5]. A Cochrane Review including three trials was inconclusive about the best i.v. fluid resuscitation strategy in trauma patients[9]. The controversy about prehospital fluid resuscitation in trauma has been acknowledged in the latest guidelines of the European Resuscitation Council [10]. It has been suggested that, in case of uncontrolled haemorrhage, i.v. fluid volume can be restricted by titrating the administration of small aliquots against acceptable vital signs [5,11] but evidence is lacking. We hypothesized that, in bleeding patients, prehospital fluid administration may have volume-dependent effects reducing shock but, on the other hand, may increase blood transfusion upon admission in ED. Therefore, we aimed to study the effects of prehospital i.v. fluid volume on evidence of shock, including the need for blood transfusion and survival outcome in trauma patients delivered to the emergency department (ED) while adjusting for important features influencing prehospital fluid administration.

Patients and methods

Study population

The study was approved by the Human research Ethics Committee of Liverpool Hospital, Liverpool, New South Wales, Australia. This study was designed as a retrospective analysis and included the medical records of patients admitted between 1995 and 2009 to Liverpool Hospital, Sydney, Australia, a level 1 trauma centre within a metropolitan trauma area. During this time period there has not been a change of protocol on the administration of prehospital fluids (e.g. regarding fluid restriction). Patients were identified using the South Western Sydney Regional Trauma Registry. Trauma patients from the major injury database, aged 16 years or older, with a systolic blood pressure of 90 mmHg or less at arrival of the Emergency Medical Services (EMS) at the scene were included. Trauma patients with cardiac arrest at arrival of the EMS and patients with burns and near-drowning were excluded. Trauma patients that were transferred to Liverpool from another hospital were similarly excluded.

Study variables

The following study variables were collected: gender, injury severity score (ISS) [12], prehospital (scene) systolic blood pressure, prehospital (scene) pulse rate, Glasgow Coma Scale (GCS) at the scene, prehospital time (from EMS dispatch until arrival at ED), prehospital airway interventions (endotracheal intubation/cricothyroidotomy), scene entrapment, prehospital blood transfusion, mechanism of injury (MOI): blunt or penetrating, transfusion of bloodproduct(s) in ED, prehospital cardiopulmonary resuscitation (CPR), ED systolic blood pressure, ED pulse rate, and the total volume of prehospital i.v. fluids administered. Prehospital i.v. fluid volumes (PFV) were categorized as follows: volumes of 0–0.5 L ($0 \geq$ volume ≤ 0.5 L), volumes of 0.5–1 L ($0.5 >$ volume ≤ 1 L), volumes of 1–2 L ($1 >$ volume ≤ 2 L) and volumes > 2 L (volume > 2 L).

Mortality at 24 h after admission was derived from the database. The Shock Index (SI) was calculated as follows:

$$\text{Prehospital Shock Index (PHSI)} = \frac{\text{prehospital pulse rate}}{\text{prehospital systolic blood pressure}}$$

and

$$\text{ED Shock Index (EDSI)} = \frac{\text{ED pulse rate}}{\text{ED systolic blood pressure}}$$

The SI is normally 0.5–0.7 and is elevated in the setting of acute hypovolaemia [13]. Furthermore, SI is a clinical indicator of hypovolaemic shock in trauma in respect to transfusion requirements, haemostatic resuscitation and mortality [14]. In our study, shock was defined as SI ≥ 1, indicating moderate to severe shock [14]. The outcome variables were defined as EDSI (as a continuous variable), shock upon arrival at ED (EDSI ≥ 1), any transfusion of blood product(s) during the ED stay (as surrogate for bleeding) and mortality at 24 h after presentation.

Data analysis

Stepwise, backward linear regression analysis was performed to investigate the relationship between PFV (continuous variable) and the shock index in ED (continuous variable) while adjusting for ISS, prehospital shock (PHSI ≥ 1), prehospital blood transfusion, MOI, prehospital time < 1 h, prehospital airway intervention, low GCS (GCS ≤ 8), scene entrapment, and prehospital CPR. Interaction (effect modification) between PFV and, respectively, prehospital shock (PHSI ≥ 1) and prehospital time < 1 h was investigated. Logarithmic transformation to achieve linearity was performed and checked through plot and residual analysis. Data are presented as the retransformed coefficients and 95% confidence intervals (95%-CIs) and also as percentages of increase or decrease of the dependent variable. Significance was set at $p < 0.05$. Stepwise, backward logistic regression analysis was performed to investigate the association between PFV (transformed into an ordinal variable) and shock in ED (EDSI ≥ 1), blood transfusion in ED and mortality at 24 h while adjusting for high ISS (ISS ≥ 16), prehospital shock (PHSI > 1), prehospital blood transfusion, MOI, prehospital airway intervention, low GCS (GCS ≤ 8), scene entrapment, prehospital time <1 h and prehospital CPR. Continuous variables were checked for the linear association assumption and recoded into nominal or ordinal variables if necessary. Data are presented as odds ratio's (ORs) and 95%-CIs. Significance was set at $p < 0.05$. (Software SPSS® 16.0 for Windows®, Microsoft® Corporation, 2007, Mountain View CA, USA).

Results

Group characteristics, prehospital i.v. fluid volumes and haemodynamic parameters

From the trauma registry, 941 eligible patients were identified for analysis. Group characteristics are presented in Table 1A showing that 74.2% of the patients were males. The main mechanism of injury was blunt trauma (79.2%). The median ISS was 13 (range 1–75). 45.2% of the patients had an ISS ≥ 16. A GCS ≤ 8 was noted in 17.5% of patients. The median PFV administered was 0.5 L (range 0–8.1 L) (Table 1A). Volumes of 0–0.5 L were administered in 52.7% of the patients and volumes of 0.5–1 L in 26.9% of patients. Volumes of 1–2 L and volumes of > 2 L were administered in, 16.6% and 3.8% of patients respectively (Table 1A). Mean PHSI and EDSI (SD) were 1.28 (0.60) and 0.82 (0.33) respectively (Table 1B). 69.5% of the patients

Table 1A
Group characteristics of trauma patients ($n = 941$) hypotensive (systolic blood pressure of 90 mmHg or less) at the scene.

Characteristic	Value	n
Male	74.2%	698
Blunt trauma mechanism	79.2%	745
ISS (median, range)	13 (1–75)	941
High ISS (ISS ≥ 16)	45.2%	425
Low GCS (GCS ≤ 8)	17.5%	165
Prehospital airway intervention	4.9%	46
Prehospital CPR	1.3%	12
Entrapment	15.6%	147
Prehospital time (mean, SD)	55.8 (24) min	941
Prehospital time < 1 h	62.0%	583
Prehospital blood transfusion	1.6%	15
Blood transfusion in ED	22.3%	210
Mortality at 24 h	7.2%	68
Mortality until hospital discharge	9.5%	89
Prehospital fluid volume (median, range)	0.5 (0–8.1) L	941
0–0.5 L	52.7%	496
0.5–1 L	26.9%	253
1–2 L	16.6%	156
>2 L	3.8%	36

ISS, injury severity score; GCS, Glasgow Coma Scale; CPR, cardiopulmonary resuscitation.
The grey values emphasize the importance of these values since these represented the independent variable that was of main interest in the study.

were found to be in shock (PHSI ≥ 1) at arrival of the EMS at the scene and 19.8% of the patients were in shock (EDSI > 1) upon arrival at ED (Table 1B). 22.3% of the patients had received a blood transfusion during their ED stay (Table 1A). The mortality at 24 h after arrival in ED was 7.2% (Table 1A).

Relationship between prehospital i.v. fluid volume and shock index upon arrival in ED

The model established by linear regression analysis of the shock index upon arrival in ED (EDSI) on PFV is represented by:

$$EDSI = e^{-0.447-0.073[\mathrm{PFV}]+0.007[\mathrm{ISS}]+0.225[\mathrm{PHSI}\geq 1]-0.059[\mathrm{BLUNT}]+0.318[\mathrm{PHBTF}]+0.180[\mathrm{PHCPR}]}$$

$$R^2 = 0.233, \quad p < 0.001$$

[PFV], prehospital i.v. fluid volume in L; [ISS], Injury Severity Score; [PHSI ≥ 1], prehospital shock; [BLUNT], blunt mechanism of injury; [PHCPR], prehospital CPR; [PHBTF], prehospital blood transfusion.

The R^2 (square of the correlation coefficient of the model) is 0.233 ($p < 0.001$) indicating that 23.3% of the variation in the log shock index upon arrival at ED is explained by the sum total effect of all the variables in this model. The retransformed coefficients and the 95%-CIs are shown in Table 2. PFV is linearly associated with shock index upon arrival at ED: every litre of fluid is associated with a 7% decrease of the shock index upon arrival at ED ($p < 0.001$). Also, blunt trauma mechanism is associated with a 5.7% decrease of the shock index upon arrival at ED with ($p = 0.02$).

Table 1B
Haemodynamic parameters of hypotensive trauma patients ($n = 941$): at the scene and upon arrival at the Emergency Department (ED).

Haemodynamic parameter	Value (mean, SD)
Prehospital systolic blood pressure	81 (12) mmHg
Prehospital pulse rate	99 (26) beats/min
Prehospital shock index	1.28 (0.60)
Patients with prehospital shock (PHSI ≥ 1)	69.5%
ED systolic blood pressure	120 (27) mmHg
ED pulse rate	93 (25) beats/min
ED shock index	0.82 (0.33)
Patients with shock upon arrival at ED (EDSI ≥ 1)	19.8%

PHSI, prehospital shock index; EDSI, shock index upon arrival at ED.

Variables that are associated with an increase of the shock index upon arrival at ED are prehospital blood transfusion (37.4%, $p < 0.001$), ISS (7% per 10 points, $p < 0.001$), prehospital shock (PHSI ≥ 1) (25.2%, $p < 0.001$) and prehospital CPR (19.7%, $p = 0.04$) (Table 2). No effect modification was detected.

Association between prehospital i.v. fluid volumes and shock upon arrival at ED

Stepwise, backward logistic regression analysis of shock at ED arrival (EDSI ≥ 1) on PFV and other covariates was performed. Table 3 shows the adjusted ORs and 95%-CIs. Volumes of 0.5–1 L are associated with an independent, reduced likelihood of shock upon arrival at ED: OR 0.61 ($p = 0.03$) (Table 3) as compared to volumes of 0–0.5 L. This association is also noted for volumes of 1–2 L: OR 0.54 ($p = 0.02$). Volumes of >2 L are also associated with a reduced likelihood of shock upon arrival at ED, but not significantly: OR 0.72 ($p = 0.52$) (Table 3). The covariates prehospital time < 1 h, scene entrapment and prehospital airway intervention were eliminated. No effect modification was detected.

Association between prehospital i.v. fluid volumes and blood transfusion in ED

Stepwise, backward logistic regression analysis of (any) blood transfusion during the ED stay on PFV and other covariates was performed. Table 3 shows the adjusted ORs and 95%-CIs. Volumes of 0.5–1 L are associated with an increased likelihood of receiving blood transfusion in ED, but not significantly: OR 1.27 ($p = 0.31$). However, volumes of 1–2 L are independently associated with an increased likelihood of receiving blood transfusion in ED: OR 3.27 ($p < 0.001$) (Table 3). Also, this independent association appears to be stronger when volumes of > 2 L are administered: OR 9.92 ($p < 0.001$) (Table 3). The covariates prehospital blood transfusion, scene entrapment, prehospital CPR and prehospital airway intervention were eliminated. No effect modification was detected.

Association between prehospital i.v. fluid volumes and mortality at 24 h

Stepwise, backward logistic regression analysis of mortality at 24 h on PFV and other covariates was performed. Table 3 shows the

Table 2
Coefficients of the linear regression model of shock index upon arrival at the Emergency Department (EDSI) on prehospital i.v. fluid volume and other covariates in hypotensive trauma patients.

Variable	Coefficient	Retransformed coefficient ($e^{[coeff]}$)	% change in EDSI	95% CI ($e^{[coeff]}$)	*p*-value
[PFV]	−0.073	0.929	7% decrease per litre	0.903–0.956	<0.001
[ISS]	0.007	1.007	7% increase per 10 points	1.005–1.008	<0.001
[PHSI ≥ 1]	0.225	1.252	25.2% increase if PHSI ≥ 1	1.199–1.306	<0.001
[BLUNT]	−0.059	0.942	5.7% decrease if blunt trauma	0.899–0.988	0.02
[PHBTF]	0.318	1.374	37.4% increase if PHBTF	1.161–1.627	<0.001
[PHCPR]	0.180	1.197	19.7% increase if PHCPR	1.013–1.416	0.04

[PFV] prehospital i.v. fluid volume in L; [ISS] Injury Severity Score; [PHSI≥1] PHSI: prehospital shock index; and PHSI ≥ 1: prehospital shock; [BLUNT] blunt mechanism of injury; [PHBTF] prehospital blood transfusion; [PHCPR] prehospital CPR.

adjusted ORs and 95%-CIs. PFV were not significantly associated with mortality at 24 h (Table 3). In contrast, mortality at 24 h was independently associated with other indicators of injury severity and clinical condition such as high ISS (ISS ≥ 16): OR 25.18 ($p < 0.001$) and low GCS (GCS ≤ 8): OR 3.13 ($p = 0.001$) (Table 3). The covariates prehospital blood transfusion, scene entrapment, prehospital shock (PHSI > 1) and MOI were eliminated. No effect modification was detected.

Discussion

This study shows that there are, significant, contrasting associations (reversal of shock vs increased allogeneic blood transfusion requirements) of i.v. fluid resuscitation during the prehospital phase in hypotensive trauma patients and may support the hypothesis that a more tailored approach is needed as opposed to the traditional, rapid infusion of fixed and/or substantial amounts of fluids [7].

Mortality

A review of the literature suggests that the association between prehospital fluid volumes and mortality remains unclear. It has been reported that in patients with penetrating torso injury, field hypotension and uncontrolled haemorrhage, restriction of i.v. fluid resuscitation until haemorrhage control has been achieved improves survival [8]. However, another study in this patient subgroup showed no difference in survival between patients that received or did not receive prehospital i.v. fluids [15]. Sampalis et al. [16] and Haut et al. [17] concluded from their observational studies that the administration of prehospital i.v. fluids in trauma patients is associated with an increased risk of mortality. However, because the authors did not differentiate between the sole prehospital placement of i.v. catheters on the one hand and the actual administration of i.v. fluids on the other hand, their findings can not be attributed to effects of prehospital i.v. fluid volume. In contrast, Kaweski et al. [18] found that the presence of prehospital i.v. fluid administration did not influence the mortality rate in trauma patients. Hampton et al. [19] found that prehospital i.v. fluid (median 0.7 L) is associated with increased survival in trauma patients that were enrolled earlier for a multicenter study of massive transfusion (PROMMTT). However, as indicated by the authors, multilevel analysis was not performed and missing data (e.g. on on-scene SBP) were substantial. In a matched-pair analysis, Hussman et al. [20] found that high-volume prehospital fluid replacement (>1.5 L) increased the mortality rate when compared to low-volume fluid replacement (<1.5 L). Dula et al. [21] found no differences in survival in hypotensive, blunt trauma patients that had received either none or more than 0.5 L of prehospital i.v. fluids. Talving et al. [22] also found no association between different prehospital fluid volumes and mortality while adjusting for injury severity and severe hypotension at the scene. Also, the mortality rate in trauma patients (ISS ≥ 16) without head injury was not different between those patients that had received up to 1.5 L of prehospital i.v. fluids and those that had received more than 2 L. [23] In our study, mortality at 24 h following admission was not associated with the administration of any volume of prehospital i.v. fluids and supports the findings of the latter studies.

Shock and blood transfusion in ED

In the prehospital arena and in ED initially, clinical decisions are made on basis of the haemodynamic parameters. SI is a useful an

Table 3
Logistic regression analysis of shock (EDSI ≥ 1) upon arrival in the Emergency Department (ED), bloodtransfusion in ED and mortality at 24 h, respectively,on prehospital i.v. fluid volumes and other covariates in hypotensive trauma patients.

Shock (EDSI ≥ 1)				Blood transfusion (ED)				Mortality at 24 h			
Variable	OR	95%-CI	*p*-value	Variable	OR	95%-CI	*p*-value	Variable	OR	95%-CI	*p*-value
0–0.5 L	REF		0.05[a]	0–0.5 L	REF		<0.001[a]	0–0.5 L	REF		0.39[a]
0.5–1 L	0.61	0.34–0.96	0.03	0.5–1 L	1.27	0.80–1.99	0.31	0.5–1 L	0.97	0.45–2.09	0.93
1–2 L	0.54	0.32–0.91	0.02	1–2 L	3.27	2.02–5.31	<0.001	1–2 L	1.94	0.86–4.35	0.11
>2 L	0.72	0.26–1.97	0.52	>2 L	9.92	4.00–24.60	<0.001	>2 L	0.94	0.28–4.08	0.94
Blunt MOI	0.43	0.28–0.66	<0.001	Blunt MOI	0.63	0.39–1.01	0.06	High ISS (ISS ≥ 16)	25.18	5.96–106.44	<0.001
Prehospital CPR	56.30	6.33–500.53	<0.001	High ISS (ISS ≥ 16)	6.89	4.45–10.59	<0.001	Low GCS (GCS ≤ 8)	3.13	1.62–6.03	0.001
High ISS (ISS ≥ 16)	3.18	2.12–4.76	<0.001	Low GCS (GCS ≤ 8)	1.59	1.03–2.46	0.04	Prehospital time < 1 h	5.40	2.21–13.21	<0.001
Prehospital shock (PHSI ≥ 1)	6.72	3.52–12.82	<0.001	Prehospital shock (PHSI ≥ 1)	2.75	1.69–4.48	<0.001	Prehospital CPR	23.76	2.70–209.22	0.004
Low GCS (GCS ≤ 8)	1.80	1.15–2.83	0.01	Prehospital time < 1 h	1.53	0.98–2.37	0.06	Prehospital airway intervention	3.88	1.46–10.34	0.007
Prehospital blood transfusion	3.33	0.79–13.93	0.10								

EDSI: Shock index upon arrival at ED, MOI: mechanism of injury, CPR: cardiopulmonary resuscitation, ISS: injury severity score, PHSI: prehospital shock index, GCS: Glascow Coma Scale.

[a] *p*-value represents the overall significance across the four fluid volume categories.

indicator of shock in early haemorrhage, especially when point-of-care-tests are not avialable [14,24]. In our study linear relationship was found between prehospital i.v. fluid volume and SI upon arrival at ED showing that i.v. fluid resuscitation is associated with a decrease in the shock index and so, reversal of shock. Dula et al. [21] found that hypotensive, blunt trauma patients that had received a prehospital fluid volume of more than 0.5 L, were more likely to have an increase in systolic blood pressure upon arrival in ED. In our study, the likelihood of shock was significantly decreased in patients that received volumes of 0.5–1 L and 1–2 L but not by any larger volumes. This finding is concordant with the concept that failure to respond to the initial administration of a bolus of 2 L of i.v. fluids may indicate ongoing haemorrhage. Continuation of i.v. fluid resuscitation may then promote further bleeding and/or increase the likelihood for transfusion of blood products [7]. Our study shows that administration of volumes of 1–2 L halved (OR 0.54) the likelihood for shock but tripled the likelihood of receiving blood transfusion in ED (OR 3.27). When administering volumes of >2 L, this likelihood is increased almost ten times (OR 9.92). Hussman et al. [23] also found that trauma patients who had received volumes of 2 L or more, received significantly more packed red blood cells than patients who received volumes of up to 1.5 L. However, a possible causal relationship between i.v. fluid volume and blood transfusion still needs to be proven.

Limitations

Our study has some limitations. Inclusion was based on hypotension at the scene and not necessarily hypotension caused by haemorrhagic shock. However, in trauma, hypotension is considered to be caused by haemorrhagic shock, until proven otherwise and the decision to start i.v. fluid resuscitation is triggered by observed scene hypotension. Hypotensive trauma patients are most likely to benefit from fluid resuscitation and also, by using this common criterion for inclusion, comparison with other studies is possible. In our study, the included number of trauma patients with scene hypotension is substantial ($n = 941$) and more than half of our study population had an ISS < 16. To adjust for injury severity, we incorporated high ISS (ISS ≥ 16) in our analyses. We adjusted for low GCS (as an indicator for head injury) as trauma patients with head injury may receive more aggressive fluid resuscitation in an effort to maintain adequate cerebral perfusion pressure. Low GCS could also be caused by lowered cerebral perfusion due to severe hypotension but, in the field, clinical decisions regarding potential head injury are often made primarily based on GCS. We did not adjust for types of resuscitation fluids (e.g. crystalloids and/or colloids) for the sake of comparability with others studies. Also, in this early phase of the resuscitation, the clinical effects of the fluids probably depend more on the administered volumes rather than on specific chemical differences between these fluids [25].

Prehospital fluid resuscitation strategies

Haut et al. [17] conclude from their research that routine use of prehospital i.v. fluid administration should be discouraged for all trauma patients. However, such an oversimplified approach of withholding i.v. fluids may in some cases, lead to underresuscitation and could be detrimental. No simple strategy to approach all trauma patients exists. Our findings support the tailored approach in which critical decisions have to be made in individual trauma cases weighing the risk of aggravating bleeding against optimizing haemodynamic parameters while administering i.v. fluids. Revell et al. [11] presented a consensus view on i.v. fluid resuscitation in prehospital trauma care advising boluses of 250 mL fluid to be titrated against the presence or absence of a radial pulse (caveats; penetrating torso injury, head injury and infants). Sumann et al. [26] proposed an algorithm regarding prehospital fluid management strategy in traumatic shock differentiating for head injury. Søreide et al. [27] have also suggested a differentiated approach using 500 mL boluses and present infusion of a hypertonic fluid bolus to maintain blood pressure above 80 mmHg as an alternative. Cotton et al. [28] also recommend administering small boluses of 250 mL to return the patient to a coherent mental status or a palpable radial pulse. In the setting of traumatic brain injury, a prehospital systolic blood pressure greater than 90 mmHg [28] or 110 mmHg [27] seems well advised. In the meantime, prospective research on the ideal prehospital fluid resuscitation strategy of trauma patients is urgently needed.

Conclusion

Decision making regarding prehospital i.v. fluid resuscitation is critical. We found that fluid volumes were associated with a decrease of the likelihood of shock. On the other hand, increasing fluid volumes were associated with an increase in the likelihood of receiving blood transfusion. This suggests that treatment may need to be tailored to the individual situation. Further research is needed to clarify whether a causal relationship between prehospital i.v. fluid volumes and subsequent blood transfusion exists. Also, prospective trials on optimal prehospital fluid resuscitation strategies in specific patient subgroups (e.g. traumatic brain injury and concomitant haemorrhage) are warranted.

Conflict of interest

None.

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