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## REVIEW

# Pre-hospital fluid therapy in the critically injured patient—a clinical update

Eldar Søreide<sup>a,b,\*</sup>, Charles D. Deakin<sup>c,d,e</sup><sup>a</sup> Medical Director ICU, Division of Acute Care Medicine, Stavanger University Hospital, Stavanger, Norway<sup>b</sup> Institute of Surgical Sciences, University of Bergen, Norway<sup>c</sup> Medical Director, Hampshire Ambulance Service NHS Trust, Winchester, UK<sup>d</sup> Helicopter Emergency Medical Service, Royal London Hospital, London, UK<sup>e</sup> Southampton University Hospital, Southampton, UK

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**Summary** Venous access and fluid therapy should still be considered to be essential elements of pre-hospital advanced life support (ALS) in the critically injured patient. Initiation of fluid therapy should be based on a clinical assessment, most importantly the presence, or otherwise, of a radial pulse. The goal in penetrating injury is to avoid hypovolaemic cardiac arrest during transport, but at the same time not to delay transport, or increase systolic blood pressure. The goal in blunt injury is to secure safe perfusion of the injured brain through an adequate cerebral perfusion pressure, which generally requires a systolic blood pressure well above 100 mmHg. Patients without severe brain injury tolerate lower blood pressures (hypotensive resuscitation). Importantly, using systolic blood pressure targets to titrate therapy is not as easy as it seems. Automated (oscillometric) blood pressure measurement devices frequently give erroneously high values. The concept of hypotensive resuscitation has not been validated in the few studies done in humans. Hence, the suggested targeted systolic blood pressures should only provide a mental framework for the decision-making. The ideal pre-hospital fluid regimen may be a combination of an initial hypertonic solution given as a 10–20 minutes infusion, followed by crystalloids and, in some cases, artificial colloids. This review is intended to help the clinician to balance the pros and cons of fluid therapy in the individual patient.

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\* Corresponding author.

E-mail address: [soed@sir.no](mailto:soed@sir.no) (E. Søreide).

## Contents

Introduction . . . . .	1002
Recent controversies . . . . .	1002
Recent recommendations . . . . .	1004
Hypotensive resuscitation—experience and problems . . . . .	1005
Targeted therapy based on the use of systolic blood pressure—technical pitfalls. . . . .	1005
Clinical assessment based on presence of peripheral pulse . . . . .	1005
Types of intravenous fluid . . . . .	1006
Crystalloid versus colloid . . . . .	1006
Hypertonic saline. . . . .	1006
Artificial blood . . . . .	1006
Summary and future improvements . . . . .	1007
References. . . . .	1008

## Introduction

As the care delivered by emergency medical service (EMS) systems evolved from the basic life support of the 1960's, intravenous fluid therapy was one of the first 'advanced' skills to be introduced. It was regarded as an important element of pre-hospital advanced life support (ALS) in critically injured patients.<sup>12</sup> The reasonable basic physiological premise that hypovolaemia should be corrected immediately formed the basis for aggressive pre-hospital intravenous fluid therapy for several decades, aiming to restore patients to a normovolaemic state as soon as possible. However, some of the earliest studies, examining outcome in relation to pre-hospital fluid volume, failed to show any benefit and most clinical and animal studies have been consistent with those findings. Several authors<sup>1,19,35,42,44,45,51,64,65,72,74,79,83</sup> have challenged the concept of the liberal use of pre-hospital fluids, especially in patients with penetrating trauma. Clinical practice guidelines<sup>19,56,82</sup> and some national guidelines<sup>60</sup> are now moving away from this traditional strategy and recommending a more judicious use of fluids. Due to the lack of randomised controlled studies, these guidelines are primarily based on animal research and observational studies, combined with pathophysiological rationale and the consensus of experts in the field. It is, however, not clear to what extent these guidelines have been implemented in EMS systems world-wide. Further, it is not yet documented that the proposed changes in patient management actually will improve patient outcome.

The aim of this clinically oriented update is to present an overview of the recent controversies and developments related to pre-hospital fluid therapy in critically injured patients. We also present some suggestions for best clinical practice and further improvements.

## Recent controversies

In all forms of trauma, tissue oxygenation is compromised not only by a reduction in tissue oxygen delivery, the result of haemorrhage, but also by the associated increase in tissue oxygen consumption, due to the inflammatory response. Trauma, however, is not a generic disease. In blunt trauma, a mixture of bleeding, tissue oedema, neurogenic factors and pain, combined with a tension pneumothorax, or spinal injury, may cause traumatic shock (circulatory failure).<sup>31,67</sup> Furthermore, the bleeding frequently occurs at multiple sites and is self-limiting. In penetrating trauma (stab and gunshot wounds), rupture of arteries or veins may be associated with little tissue damage, but major bleeding that rapidly leads to hypovolaemic cardiac arrest. The major controversy in intravenous fluid resuscitation relates to patients with uncontrolled haemorrhage. The optimal volume of intravenous fluid administered is a balance between improving tissue oxygen delivery against increasing the blood loss by raising systolic blood pressure. Where this balance lies has yet to be determined.

When Bickell et al.<sup>1</sup> published their now classical paper<sup>54</sup> in 1994, showing an absolute increase of 8% in survival with delayed fluid resuscitation in hypotensive trauma patients, they fuelled an already established debate. In their randomised, controlled study from urban Houston, fluid therapy was delayed all the way to arrival in the operating room. The short transport times and the young population of hypotensive patients with penetrating torso injuries only, made it hard to draw conclusions that would be valid for older patients, rural EMS systems and, most importantly, for blunt trauma with, or without, head-injury.<sup>54,82</sup> A later subgroup analysis showed that the difference in survival was only seen in patients with cardiac injuries. The study was also

criticized for the long delays in time to surgery. Although the concept of delayed resuscitation is still fiercely advocated by a few trauma surgeons,<sup>51</sup> the complete withholding of pre-hospital, or in-hospital, intravenous fluids is not generally recommended. Ten years later, it appears fair to say that the main message of the Bickell et al. study is that control of haemorrhage is an imperative priority in patients with penetrating trauma,<sup>54</sup> while normalization of systolic blood pressure is not!

Intravenous fluids cause haemodilution (a fall in haematocrit and clotting factors), as well as an increase in the extra-cellular fluid compartment. The actual effect and distribution of the infused fluid will vary with the circulatory status of the patient.<sup>30,31</sup> Hence, the relative expansion of the intravascular volume with 500 ml of an isotonic crystalloid is much greater in a shocked person than in a healthy volunteer. This is an important concept to remember, since the expansion of the intravascular volume leads to an increase in systolic blood pressure. Although this increase secures adequate perfusion and is good for the injured brain, it may cause disruption of haemostatic clots in damaged blood vessels, thereby causing further bleeding. To what extent the latter truly represents a real threat, and not just a theoretical worry in patients with blunt trauma, is not well documented.<sup>67,79</sup> Several animal models of uncontrolled bleeding have found that an increase in systolic blood pressure does increase bleeding.<sup>49,71</sup> Nevertheless, in a recent systematic review<sup>49</sup> of controlled animal studies, Mapstone et al. concluded that moderate volumes of fluid improved survival in all models, but that excessive amounts were detrimental in some situations. Another recent animal study found that the systolic blood pressure threshold for provoking new bleeding was 90 mmHg, independent of time from start of bleeding.<sup>77</sup>

Another criticism of pre-hospital fluid therapy is that establishing an intravenous-line may take time and may delay transport and definite intervention; this in turn may increase mortality.<sup>74</sup> Most authors, however, have found this association not to hold true.<sup>79</sup> The debate on what should constitute best practice for fluid therapy in penetrating versus blunt trauma, in rural versus urban settings, in younger versus older patients and in head-injured versus non-head injured patients is very much alive. In a recent systematic (Cochrane) review<sup>42</sup> of the topic, the main problem was the obvious lack of randomised, controlled studies. For this reason, the authors had no choice but to conclude that there is “continuing uncertainty about the best fluid administration strategy in bleeding trauma patients” and that “more randomised controlled

trials are needed”. Numerous observational studies<sup>32,34–36,89</sup> have found pre-hospital traumatic shock (hypotension) to be associated with an increase in hospital mortality, but to what extent pre-hospital fluid therapy improves, or actually worsens, the prognosis in these patients, is still under debate. Importantly, fluid resuscitation is only one of many aspects of ALS in these studies. Further, the ALS providers differed, as did the trauma populations and the organization of the trauma systems. Not surprisingly, the results in terms of mortality and morbidity have been inconsistent.<sup>3,15,22,25,26,29,44,45,53,55,57,63,65,74,75,79,80,83,84</sup>

To define the role of fluid therapy better, we also need to look at the magnitude of the problem of pre-hospital haemorrhagic shock in trauma patients. A pre-hospital systolic blood pressure of  $\leq 90$  mmHg is most frequently used as a cut-off point to define pre-hospital hypotension. However, the sensitivity and specificity of this cut-off blood pressure value to define continuing bleeding, the need for intravenous fluid resuscitation and later hospital interventions to stop bleeding, have not been as widely studied as imagined. Although both pre-hospital hypotensive episodes<sup>76</sup> and a low systolic blood pressure at arrival are associated with the need for surgical interventions and a higher mortality, the relationship is not clear-cut. Pre-hospital hypotension may not always be a sign of haemorrhage, but may have other causes, such as a tension pneumothorax, or spinal injury. Importantly, Lechleutner et al.<sup>43</sup> found that only 50% of patients with pre-hospital hypotension later needed surgery. In EMS systems, where pre-hospital fluid therapy is used, the incidence of hypotension at hospital admission has consistently been found to be lower than 10%.<sup>35,51</sup> Even in this population, there is less frequent indication for immediate haemostatic surgery than one would believe. Recent publications have reinforced the impression that fluid resuscitation and blood transfusion in the Emergency Department are still essential elements of the early hospital management of critically injured patients.<sup>7,8,33–35,46,58,81,82,90</sup>

Patients with severe traumatic brain injury (TBI), defined as a Glasgow Coma Score  $< 9$ , deserve special comment as the injured brain does not tolerate even short periods of hypotension (hypoperfusion).<sup>4</sup> Several studies have found a significant association between pre-hospital hypotension and worse outcome in severe TBI.<sup>5,23,48,88</sup> Studies<sup>25,26,63</sup> have also linked pre-hospital ALS to improved outcome in this trauma sub-population. A recent study<sup>10</sup> from Melbourne, Australia found an incidence of severe TBI around 20 per 1 million inhabitants/year. Almost half (45%) of these patients had an episode of

**Table 1** A suggested differential approach to pre-hospital fluid therapy in critically injured patients

Type of injury	Fluids	Pathophysiological rationale
Blunt trauma with clinical suspicion of severe brain injury	The minimal amount needed, as rapid infusion aliquots of 500 ml isotonic crystalloids, to restore and maintain systolic blood pressure $\geq 110$ mmHg	Perfusion of the traumatised brain despite the loss of auto-regulation
Blunt injury without suspicion of severe brain injury	The minimal amount needed, as rapid infusion aliquots of 500 ml isotonic crystalloids, to restore a peripheral pulse, but keep systolic blood pressure $\leq 90$ mmHg	Perfusion of vital organs without contributing to continuing or recurring bleeding
Penetrating trauma	The minimal amount needed, as rapid infusion aliquots of 500 ml isotonic crystalloids, to restore some eye opening on stimulation with, or without, a peripheral pulse, but keep systolic blood pressure $\leq 90$ mmHg	Perfusion of at least the brain and heart to avoid hypovolaemic cardiac arrest during transport, but without increasing further blood loss

Based on Nardi and co-workers.<sup>56</sup>

pre-hospital hypotension. In this group of patients, the use of early fluid therapy is considered standard treatment by most authors.<sup>19,56,65,81,82</sup> The discussion has been centred more around what systolic blood pressure to aim for and what fluid to use.<sup>4</sup> The Australian study<sup>10</sup> compared hypertonic saline plus isotonic saline to isotonic saline alone, and found no difference in outcome. The authors explained this lack of difference in outcome with by fact that both fluid regimens produced the same improvement in systolic blood pressure.

Recently, who provides care and the quality of that care have been brought into the discussion on clinical effectiveness of pre-hospital ALS.<sup>16,26,53,78,84</sup> The same logic should apply to pre-hospital fluid therapy, as part of the pre-hospital ALS. It is not obvious that a strict, protocol-driven therapy by paramedics, with limited clinical experience, will produce the same results as pre-hospital EMS systems using specialised emergency physicians<sup>47,63,84</sup> able to individualise therapy.<sup>17,26,64</sup> In latter case, advanced therapy is provided by experienced hospital clinicians working in the pre-hospital phase and applying the same emergency and critical care skills and therapies as they would do when receiving a patient in the emergency department. Applying that same therapy, but earlier, seems logical if the whole process of care is to be speeded up.<sup>2,33,58,79</sup> However, studies trying to compare physician-based ALS with paramedic, or nurse, based ALS have shown conflicting results in terms of survival benefits.<sup>25,26,44,63,80</sup> Employing physicians may not, per se, make a difference if the overall patient care is no different, either in terms of diagnostic and thera-

peutic quality, or patient safety (less complications).<sup>9,84</sup> Nevertheless, it is self-evident that the clinical skills and experience of the care provider must be important.

## Recent recommendations

Recently, several groups<sup>4,19,31,56,67,68,70,82,83</sup> have published clinical recommendations for pre-hospital fluid therapy in trauma patients. Due to the lack of well-performed, randomised, controlled trials, the recent guidelines are based on a combination of expert opinions, pathophysiological rationale and the results of observational cohort studies in humans and controlled studies in animal models.

The pathophysiological rationale is used to strike a balance between the risks and the benefits of fluid infusion (Table 1). The goal is to secure perfusion of vital organs without increasing the risk of more bleeding and of delaying transport to hospital. To what extent such guidelines cause a significant change in clinical practice, when it comes to limiting the volume and speed of pre-hospital intravenous infusion, has not been documented. Still, the authors have a distinct impression that there is a trend towards limiting the volume and speed of pre-hospital intravenous fluids, based on the experience from our own pre-hospital EMS systems. Some reported data from the UK also indicate this.<sup>72,73</sup>

The new fluid therapy concept has been named “hypotensive resuscitation” (also permissive hypotension (hypovolaemia))<sup>20,40,82</sup> because it aims at

limiting the volume of fluid given and maintaining the systolic blood pressure as low as considered safe in the specific situation (Table 1). To titrate the fluid therapy, aliquots as low as 25 ml, and up to 500 ml, of crystalloids<sup>19,20,51</sup> have been suggested. This in contrast to (a) traditional fluid resuscitation, which starts with 2 l of crystalloid and aims at normalization of systolic blood pressure, or (b) delayed resuscitation where no fluid at all is given until the time of surgery.<sup>40,67,68</sup> Before fully accepting the new concept of hypotensive resuscitation, it is important to bear in mind that clinical experience with it is still very limited. Additionally, the use of measured systolic blood pressure has been reported as having major limitations as a clinical end-point for fluid resuscitation.<sup>67,68,90</sup>

### Hypotensive resuscitation—experience and problems

Although systolic blood pressure is known to be a poor surrogate measure for tissue perfusion, especially in younger patients with well-preserved sympathetic vasoconstriction responses, it is still widely used, both for study and clinical purposes. To test whether the new concept of hypotensive resuscitation was feasible in clinical practice and actually improved survival, Dutton et al.<sup>20</sup> randomised 110 hypotensive patients with suspected continuing haemorrhage to a target systolic blood pressure of either >100 mmHg, or >70 mmHg. They found no difference in survival. Furthermore, the patients ended up with rather similar blood pressures. The authors ascribed this to the “dynamic interaction between fluid administration, anaesthetic agents and the patient’s own auto-regulatory mechanisms”. This study elegantly showed that the systolic blood pressure end-points are not easy to achieve, despite only giving fluid in small, titrated aliquots of 200–500 ml. The study also indicated that the concept of hypotensive resuscitation is unlikely to have an impact on the in-hospital mortality of trauma victims in a modern trauma centre. Lastly, it illustrates the fact that modern trauma care is not the same as an immediate operation in most patients, but a differentiated and dynamic damage limitation approach of volume resuscitation, transfusion, embolization, critical care and sometimes haemostatic surgery.<sup>2,4,7,8,20,34,35,37,58,90</sup>

To the authors’ knowledge, no similar study in the pre-hospital phase has been carried out. In their review, Kreimeier et al.<sup>40</sup> concluded that the general application of hypotensive resuscitation concept cannot be recommended at the present time, especially in patients with severe TBI.

### Targeted therapy based on the use of systolic blood pressure—technical pitfalls

The measured systolic blood pressure is a result of cardiac output, peripheral vascular resistance and how we measure it.<sup>66</sup> Systolic blood pressure has been used extensively to assess volume status in trauma patients, both for triage, treatment and study protocols.<sup>1,3,5,10,12,13,19,20,22,23,31,35,39,40,43,45,51,56,67,68,70,72,76,77,82,85,88,91</sup> This use, however, is not without problems. The sensitivity and specificity of certain cut-off values to predict acute blood loss are poor. More importantly, the measured values vary with the method applied. Invasive measurement is considered to be the gold standard, but is not feasible for pre-hospital use. Both auscultatory (manual) and oscillometric (automated), non-invasive methods are in common use, but the results of concomitant measurement in the same patient will vary significantly.<sup>66</sup> Only recently has this aspect of the initial trauma management been studied. Davis et al.<sup>11</sup> found that the systolic blood pressure measured with the oscillometric method was consistently higher than the manually measured systolic blood pressure, especially in hypotensive patients (defined as systolic blood pressure <90 mm Hg by the auscultatory method). Hence, they suggest that only manual systolic blood pressure should be used for pre-hospital, or hospital, triage decisions. The wide-spread use of automated blood pressure devices, both in pre-hospital and hospital EMS, should prompt us to re-evaluate the use of specific systolic blood pressure values, both in treatment algorithms and study protocols. Furthermore, the method used for blood pressure measurement should always be reported in treatment algorithms and study protocols.

### Clinical assessment based on presence of peripheral pulse

In the pre-hospital situation, the use of automated, or manual, blood pressure measurement methods may not always immediately be available, or feasible. The initial assessment of the patient’s volume status should always include heart rate, capillary refill, skin temperature and dryness, as well as the mental status.<sup>90</sup> The presence of a radial pulse has been suggested as a reliable indicator of specific systolic blood pressure values.<sup>19,67,68</sup> The presence of a radial pulse has traditionally been taught to correspond to a systolic blood pressure of no less than 80 mmHg. Deakin and Low<sup>13</sup> questioned this dogma, as they found lower systolic blood pressure than this in 80% of their hypovolaemic patients with a peripheral pulse. Palpation of the radial pulse is easier than the brachial, femoral and carotid

pulses.<sup>50</sup> Certainly, palpating pulses is a clinical skill that needs practice like any other skill.<sup>21</sup> It is important to remember that continuing bleeding may not necessarily be associated with tachycardia.<sup>14,38,43</sup>

Although the traditional trauma teaching may overestimate the systolic blood pressure at which the radial artery pulse disappears, there seems to be good clinical agreement between the degree of haemorrhagic shock and the presence of a peripheral pulse. Lately, more authors suggest that the initial assessment of the C' (Circulation) component of the Airway, Breathing, Circulation (ABC) should focus on mental status, skin, respiratory rate and the peripheral pulse, more than the measured systolic blood pressure in isolation.<sup>19,67,68,90</sup> The combination of an altered mental state, or unconsciousness, combined with cool, clammy skin and an absent radial pulse is a well established triad indicating hypovolaemic shock. Using this, a rapid diagnosis of coma due to circulatory failure (shock) can be differentiated from coma due to traumatic brain injury and initial care be targeted accordingly. This has been the practice of the authors of this review for many years. After the initial assessment and therapy is started, we use systolic blood pressure values to monitor further responses.

## Types of intravenous fluid

### Crystalloid versus colloid

The optimal type of fluid for intravenous fluid replacement is debated. Theoretical advantages of crystalloids are that they replace interstitial, as well as intravascular, fluid loss, they do not impair coagulation, do not cause allergic reactions and are inexpensive. Their limitations include limited intravascular expansion and tissue oedema, which may contribute to impaired gaseous exchange in the lungs, increased bacterial translocation in the gut and reduced capillary blood flow, impairing wound healing. Proponents of colloids cite the advantage that they have a longer intravascular effect, which may improve organ perfusion and that they cause less tissue oedema, which may improve gaseous exchange. Against this must be weighed the disadvantages of an increased incidence in allergic reactions (due to gelatins), impaired blood cross-matching (due to dextrans), influences on coagulation (dextrans, hetastarches), possible renal impairment (hetastarches) and accumulation (hetastarches). A few meta-analyses have purported to show increased mortality with colloids, but these studies have been grossly underpowered and included study designs of very poor quality.<sup>6</sup> A recent,

prospective, blinded Australian study randomised 6833 hypotensive trauma patients to receive either saline or 4% albumin in the initial management of their hypovolaemic shock.<sup>24</sup> There was neither any overall difference in 28 day mortality between the two groups, nor any difference in rates of organ failure. Subgroup analysis did suggest that there may have been a survival advantage for patients with head injuries receiving saline, but these results must be interpreted with caution.

### Hypertonic saline

Hypertonic saline is a relatively new fluid available for resuscitation of hypovolaemic shock. Its osmotic properties attract fluid into the intravascular compartment, where the addition of a dextran, or hetastarch, helps to prolong its effects through binding of the recruited water.<sup>69</sup> Fluid resuscitation using hypertonic saline/dextran (approximately 4 ml/kg) is associated with improved haemodynamics and rapid correction of blood pressure. Importantly, it enhances microcirculatory flow by drawing fluid from oedematous endothelium, thereby improving tissue perfusion. Most publications<sup>10,39</sup> have concluded that there is no clear benefit of hypertonic saline solutions in terms of survival, or reduced morbidity. However, a re-analysis of individual data from previous studies of hypertonic saline/dextran found an improved survival in both hypotensive patients with head injury, where it acts to increase cerebral perfusion pressure, and in those with penetrating injuries needing immediate surgery.<sup>41,85–87,91</sup> The pre-hospital use of hypertonic saline/colloid solutions is limited to a few countries, most of them in Europe,<sup>52,61,70</sup> and further studies are needed to elicit their potential benefits over standard fluid therapy. To define their exact role better in pre-hospital fluid therapy, the authors think that more pre-hospital EMS systems should gain clinical experience with the use of these hypertonic fluids.<sup>61</sup> If hypertonic solutions are used, they should probably be given as a 4 ml/kg infusion over 10–20 minutes, rather than as rapid boluses.<sup>39</sup>

### Artificial blood

Mild anaemia may actually improve oxygen delivery by reducing blood viscosity, providing the cardiac output remains adequate. Once haemoglobin falls below 8.0 g/dl, oxygen delivery is impaired and the oxygen-carrying capacity of the blood needs to be increased, ideally with blood, but artificial solutions are approaching clinical use. Although pre-hospital transfusion with type O, Rh negative packed red blood cells has been used in some systems and

situations,<sup>27</sup> for obvious reason this practice is not common. There are several problems with pre-hospital transfusions. A major concern is the lack of re-warming before use, which may lead to potentially dangerous hypothermia. Haemoglobin substitutes (artificial blood) have been tested in trauma patients. Earlier studies have been stopped due to serious complications, but new studies<sup>28</sup> are under way. In the future, the use of artificial oxygen carriers may become an integral part of pre-hospital fluid therapy.

## Summary and future improvements

Yesterday's dogma that fluid therapy and other pre-hospital ALS interventions are always of benefit, has been replaced with a major concern that pre-hospital ALS may actually do more harm than good. This raises serious concern over the question of the widespread use of pre-hospital fluid therapy in paramedic-run EMS systems. The number of critically injured patients is limited and to secure high quality care and improve patient safety, the authors are of the view that a limited number of clinicians, medical or otherwise, should provide pre-hospital ALS. We also think that more qualitative studies, looking

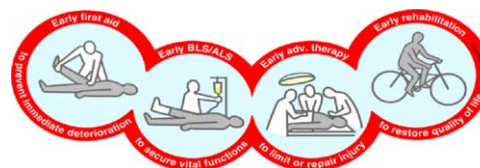


Figure 1 Trauma Chain of Survival.

into training aspects and how best to gain experience are needed before we can define the exact role of pre-hospital fluid therapy.

Furthermore, the era of regarding trauma care as a fragmented process, split into a pre-hospital phase, the emergency department, the operating room, the ICU and so on, should end. The figure of the Trauma Chain of Survival (Fig. 1) illustrates the integrated trauma care approach, in which all the links fit together. Patient survival is linked to the overall quality, integration, communication and process of care in a trauma system. Instead of merely focusing on the minutes used to start fluid therapy, or the time spent at the scene, the total pre-hospital time, total time to reach certain clinical end-points and total time to the start of treatment of continuing bleeding are of greater importance. The majority of severely injured patients do not need surgical interventions to stop

**Table 2** A suggested step-wise and minimised approach to circulatory support in the critically injured trauma patients

- (1) When assessing mental status, airway and breathing, find out what injury mechanism (blunt or penetrating) is most likely and start basic life support. This includes external compression to stop bleeding from the scalp and other wounds.
- (2) Check skin temperature and feel for the radial pulse. Present, weak, absent?
- (3) Indication for analgesia and/or anaesthetic-assisted endotracheal intubation?
- (4) Insert at least one peripheral venous cannula. Use a large-bore catheter if possible. Secure the intravenous line!
- (5) Start an isotonic crystalloid infusion. If absent peripheral pulse and other signs of haemorrhagic shock, begin a rapid infusion (see Table 1). Consider the use of hypertonic solutions instead of crystalloids.
- (6) Always seek alternative explanations other than bleeding (such as tension pneumothorax, hypothermia or pain) when signs of reduced peripheral circulation (shock) is present.
- (7) Always have a rapid infusion running during endotracheal intubation procedure, in order to counteract the effects of anaesthetic drugs and positive pressure ventilation. Consider the use of monitoring equipment before this procedure (pulse oxymetry and systolic blood pressure). Use manual blood pressure measurements initially.
- (8) Re-assess the circulation by the repeated use of the radial artery pulse.
- (9) In penetrating trauma and haemorrhagic shock, first priority is rapid transport to a hospital, ready for surgical intervention.
- (10) In blunt trauma, especially after endotracheal intubation, always suspect a tension pneumothorax.
- (11) Over zealous infusion, especially in trapped and exposed patients, will result in hypothermia. Be aware! Keep the patient warm.
- (12) Before transport, intensify level of monitoring. This is especially important with prolonged transport times. Automatic blood pressure devices are easy to use but frequently overestimate systolic blood pressure.
- (13) In patients with signs of haemorrhagic shock, information about the volumes of fluids given and their effect on vital signs is very important for the hospital trauma team. To avoid delays in the process of care, make sure to present this information as soon as possible.

bleeding, but other types of surgery and longitudinal critical care, including intravenous fluids and transfusion, to secure functional survival. The major challenge, therefore, is to make sure that the treatment plan is adapted to individual needs and not only the few patients with an immediate need for haemostatic surgery. What is said here holds true for most adult patients in general, but certainly also for the sub-group of geriatric and pregnant patients,<sup>38,59</sup> as well as children, in whom pre-hospital fluid therapy seems to play an even more important role.<sup>62</sup>

When comparing the results in different systems, both system factors<sup>18,46</sup> and the background and training of those who provide pre-hospital ALS should be taken into account.<sup>53,84</sup> For the pre-hospital ALS provider we suggest the following step-wise framework for fluid therapy decision making (Table 2). A key issue always to remember is that pre-hospital trauma care is a very dynamic process, with constantly changing conditions in an uncontrolled environment.<sup>9</sup>

In conclusion, we believe that venous access and fluid therapy should still be considered essential elements of pre-hospital ALS in the critically injured patient. Initiation of fluid therapy should be based on a clinical assessment, most importantly the presence, or otherwise, of a radial pulse. The goal in penetrating injury is to avoid hypovolaemic cardiac arrest during transport, but not to delay transport, or to increase the systolic blood pressure above 80 mmHg. The goal in blunt injury is to secure sufficient perfusion of the injured brain through an adequate cerebral perfusion pressure, which generally requires a systolic blood pressure well above 100 mmHg. Patients with blunt trauma, without severe brain injury, tolerate lower blood pressures. Automated (oscillometric) blood pressure measurement devices are not accurate in hypovolaemic patients and frequently give erroneously high values. Hence, the suggested targeted systolic blood pressures should only provide a mental framework for the decision making. The optimal pre-hospital fluid may be a combination of an initial hypertonic solution given as a 10–20 min infusion, followed by crystalloids and, in some cases, artificial colloids. The role of artificial blood in this context is not clear, but controlled studies are under way. As the initial assessment and response to fluid therapy is of great importance for monitoring the in-hospital treatment plan, a short and highlighted pre-hospital report is mandatory. Under optimal conditions, this information should be transmitted to the hospital trauma team before arrival. This will help secure a continuum of care. To help the clinician to balance the pros and cons of fluid therapy in the individual

patient, more studies on the relationship between pre-hospital systolic blood pressure measurements, volume status and fluid therapy response are needed.

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