REVIEW ARTICLE

Revista de Medicină de Urgență, Vol. 3, Nr. 1: 21-26

A review of the fluid resuscitation strategies in trauma care-the concept of permissive hypotension

Anca Bîrliga*

Abstract

The aggressive fluid therapy (two large-bore i.v. lines, rapid infusion), which was standard of care until recently in trauma cases with signs of shock, is more and more replaced with the permissive hypotension concept.

This concept refers to the practice of restricting fluid resuscitation and keeping a lower than normal arterial pressure until hemorrhage is stopped. The hemodynamic goal in permissive hypotension is maintaining systolic blood pressure between 80 and 90 mm Hg.

Actually, fluid resuscitation should be titrated to individual needs, following the principle: enough fluid to maintain organ perfusion, but not so much as to exacerbate the bleeding.

Permissive hypotension is not an option for patients with traumatic brain injury or spinal cord injury, as in these cases it may worsen the neurological outcome. Also patients with preexistent vascular diseases will probably not benefit from the concept, due to fast exhaustion of physiologic compensatory mechanisms.

The new strategies for fluid resuscitation have focused on enhancing oxygen delivery to the tissues, and microcirculatory resuscitation, which is represented by the concept of small-volume hyperosmolar resuscitation.

The hyperosmolar saline colloid solutions, which combine hypertonic saline solution with colloids, are becoming the best choice for small-volume resuscitation in prehospital setting.
(Revista de Medicină de Urgență, Vol. 3, Nr. 1: 21-26)

Key words
Fluid resuscitation, permissive hypotension, small volume resuscitation, hyperosmolar saline colloid solutions.

Introduction

In the setting of trauma victim with signs of hemorrhagic shock, the standard approach recommended by ATLS course is aggressive fluid therapy. This means 2 large-bore intravenous lines and rapid infusion of large volume of fluids. The goals of this treatment strategy are rapid restoration of intravascular volume and vital signs towards normal, and maintenance of vital organ perfusion[1]

A long list of the most frequently used techniques to normalize the blood pressure in a short time can be recalled:
- elevation of the legs or Trendelenburg position(the goal is to increase the venous return;
- use of aggressive crystalloid fluid administration;
- use of colloids;
- use of rapid infusers;
- use of vasopressors;
- use of hypertonic saline [2].

But the evolving evidence suggests that aggressive fluid resuscitation prior to hemostasis leads to additional bleeding through hydraulic acceleration of hemorrhage, soft clot dissolution and dilution of clotting factors[3].

In contrast, there is growing evidence that small-volume resuscitation and maintenance of the blood pressure under normal values results in improved long-time survival[4].

Defining the concepts-deliberate hypotension-delayed fluid resuscitation-permissive hypotension

1. Deliberate hypotension in the intra-operative setting is defined as a reduction of the blood pressure (by pharmacological means) before surgery, with the goal of decreasing blood loss [5].

This concept was proposed by Cushing [6] in 1917 for intracranial surgery.

The correlation between the blood pressure values and blood loss is obvious; using deliberate hypotension technique, blood loss decreases by 50% or more in some procedures.

Most studies define deliberate hypotension as a reduction in systolic blood pressure to 80-90 mm Hg, or a decrease of mean arterial pressure (MAP) to 50-65 mm Hg in normotensive patients [7].

Because deliberate hypotension is designed to decrease arterial blood pressure preserving organ blood flow, it must be emphasized that this procedure requires constant assessment of intravascular volume by invasive hemodynamic monitoring throughout surgery, to ensure optimal organ function [5].

The method is not suitable for patients with diminished reserves for adequate organ perfusion-patients with a history of cerebrovascular disease; renal dysfunction; liver dysfunction; ischemic heart disease; severe peripheral claudication [7].

* MD, Emergency Physician
Emergency Department, Bucharest Clinical Emergency Hospital, 8 Calea Floreasca, Sector 1, Bucharest, Romania. E-mail: abirliga@yahoo.com.
Recived in 15 april, accepted in 20 mai.

Situations needing deliberate hypotension are neurosurgery; large orthopedic procedures; surgery on large tumors; or even when religious beliefs preclude blood transfusion [5,7].

II. Delayed fluid resuscitation-versus immediate standard resuscitation.

In 1994, Bickell and coworkers [8] published a study that included 598 patients with penetrating torso injuries and a prehospital systolic blood pressure of less than 90 mm Hg. They were divided into 2 groups.

Patients assigned to the first group-the immediate resuscitation group-received standard fluid resuscitation before they reached the hospital.

Patients assigned to the second group-the delayed resuscitation group-received IV cannulation, but no fluid resuscitation until they reached the operating room.

In the second group, mortality was lower, hospitalization was shorter and there were fewer post-operatory complications.

The study addressed the point that intravenous fluid infusion may be detrimental in the clinical setting, if administered before hemorrhage is surgically controlled.

But the interpretation of the results is restricted to penetrating trauma (significant and uncontrollable blood loss) and to a prehospital scenario in big cities, where trauma centers may be reached within a few minutes[5].

There are studies indicating that delayed resuscitation produces a more profound shock insult and a more severe metabolic acidosis than traditional resuscitation, characterized by repeated episodes of alternating hypotension and normotension[9].

III. The concept of permissive hypotension does not exclude immediate fluid replacement, the only restriction is to avoid completely normalizing blood pressure in a situation where blood loss may be enhanced.

Thus, the risk of organ ischemia is balanced against the possibility of provoking more bleeding with fluids[1,10].

Once hemorrhage is controlled-this remains the most important intervention!-the blood pressure and normovolemia should be restored. Fluid resuscitation will be now targeted against conventional endpoints, the base deficit and plasma lactate [10].

Determining the optimum target blood pressure

In most cases the hemodynamic goal in permissive hypotension is maintaining systolic blood pressure between 80 and 90 mm Hg. This translates into tolerating class III hemorrhagic shock and resuscitating class IV shock (differentiated bellow) [11].

Class III Shock	Class IV Shock
Vital signs ➢ SBP<90 mm Hg ➢ HR>120 ➢ RR>30 Mental status ➢ Anxious or confused Skin ➢ Capillary refill delayed	Vital signs ➢ SBP<70 mm Hg ➢ HR>140 ➢ RR>35 Mental status ➢ Combative or comatose Skin ➢ Cool, diaphoretic ➢ Mottled, ashen, gray, pale ➢ Capillary refill absent

SBP-systolic blood pressure; HR-heart rate; RR-respiratory rate

In fact, fluid resuscitation should be titrated to individual needs, based on the principle: enough fluid to maintain organ perfusion, but not so much as to exacerbate the bleed[12].

Many protocols have begun to shift from an algorithmic general response to trauma to a treatment protocol responsive to patient's needs, determined by careful monitoring.

There are three distinct situations:

a)Young patients, without preexistent pathology.

For them, the resuscitation endpoint is a systolic blood pressure of 80 mm Hg-which is enough for an adequate organ perfusion [2,13,14].

b) Patients with preexistence of cardiovascular disease; hypertensive; with angina; with history of cerebrovascular disease or carotid artery stenosis; with compromised renal function due to renal artery stenosis; with severe peripheral vascular disease.

In all these cases, low blood pressure may induce sludge, thrombosis and lead to occlusion of the vessel lumen[5].

The target systolic blood pressure is recommended to be at least 100 mm Hg[14].

c) Patients with severe traumatic brain injury and hemorrhagic shock.

In these cases, permissive hypotension should best be avoided[13,14]; the systolic blood pressure must be maintained at least 120 mm Hg[14].

Because of impaired cerebral hemodynamics autoregulation after trauma, CPP will become dependent on blood pressure values, according to the formula[13]:

CPP=MAP-ICP

CPP-cerebral perfusion pressure;

MAP-mean arterial pressure;

ICP-intracranial pressure.

As a consequence, hypovolemic hypotension that would not otherwise reduce cerebral blood flow may lead to brain ischemia[5].

Recent studies have identified hypotension as the predominant cause of secondary brain injury in severe head trauma and as the most important extra-cerebral factor affecting outcome after traumatic brain injury[5,15].

Patients with traumatic brain injury who were hypotensive at the time of admission had twice the mortality and a significant increase in morbidity, when compared with the patients who were normotensive. The concomitant presence of hypoxia(PaO_2<60 mm Hg) and hypotension (systolic blood pressure = 90 mm Hg) upon admission resulted in a 75% mortality[5].

Thus, prompt application of life support measures: tracheal intubation, mechanical ventilation, IV fluid resuscitation will limit secondary hypoxic brain damage [5]. Concerns that adequate fluid resuscitation results in increased intracranial pressure(ICP) after head injury appear to be unfounded[16].

When targeted systolic blood pressure can not be achieved by volume expansion therapy alone, the use of vasopressors may be warranted[14].

The medication of choice is dopamine(10-15µg/kgc/min) but its efficacy can be insufficient or decreased in cases of prolonged use. In such conditions, norepinephrine(0,5-2µg/min) is used[15].

The concept of maintaining cerebral perfusion pressure, which in case of traumatic brain injury needs normotensive systemic pressures, has been extended to patients with spinal cord injury.

Maintaining spinal cord blood flow reduces the risk of secondary ischemic injury and may improve the outcome[5].

Fluid resuscitation in prehospital trauma setting-which is the most appropiate protocol?

Until now, early venous access and administration of fluids has been regarded as of paramount importance.

However, a retrospective study from 1996 [17,18] makes a comparison between 2 groups: patients brought to hospital by paramedics, and patients brought in by bystanders, relatives, and the police.

Outcome was worse in the first group; the poor outcomes can be related to detrimental effects of aggressive fluid resuscitation, and to the delays in transfer to hospital because of the interventions performed on scene.

Another study indicates that fluid replacement in injured patients at the accident scene does not significantly influence the outcome[19].

A conclusion may be that the ambulance crews should concentrate on avoiding unnecessary delays and speeding up transfer to definitive care in hospital, rather than concentrate on their fluids protocols.

Regarding the cannulation on scene issue, of course, cannulation at an early stage is desirable-but without transfer times being prolonged by attempts to gain an iv line.

One way to balance the benefits to be gained by obtaining venous access prehospital with the risk of lengthening transfer times is to attempt cannulation en route.

This should be considered every time appropriate expertise and training are available. A limit of two attempts en route is reasonable[18].

The management of entrapped patients is a special situation-the efforts to cannulate will not extend transfer time, and there are compelling reasons for obtaining a line on scene; principally, the need for analgesia, also for resuscitation drugs and fluids. So, it is recommended, in cases of entrapment, to gain circulatory access on scene[18].

Regarding fluid replacement protocols in trauma patients, the most appropriate prehospital approach involves: determining the mechanism of injury (blunt versus penetrating); identifying anatomic involvement(truncal versus isolated head injury versus isolated extremity injury); and staging the condition (hemodynamic stability versus instability versus periarrest state) [20].

It must be also emphasized that, in the field or in the emergency department, blood pressure measured values reflect only approximately the level of shock and the adequacy of fluid resuscitation[2,19].

So, protocols will be based on the clinical examination of the patient.

The presence or absence of a radial pulse gives an approximate guide to whether the systolic blood pressure is above or below 80-90 mm Hg[18].

Brachial pulse corresponds to about 70-80 mm Hg and a central pulse (femoral or carotid) to 60-70 mm Hg. On children less than 1 year old, the use of a brachial pulse is more practical and it is easier to feel [18].

The next clinical scenarios can be limitated:

a) If the patient cerebrates normally and the peripheral pulse can be felt, it is recommended just to put an intravenous catheter; if a line is started, the rate of fluid administration should be to keep open only. Some clinicians would desire an iv portal just to be available, in case the patient deteriorates[2].

b) If the patient does not cerebrate normally, the examiner looks for the pressure of peripheral pulse. If present a line is started with a rate just to keep open only, and transport is determined on the basis of diagnosed injury[2].

If the peripheral pulse is absent, the fluid resuscitation will start by giving boluses of 250 ml and monitoring the hemodynamic impact; once the peripheral pulse returns,

fluid replacement can be suspended and the situation monitored [2,20].

c) A different category includes patients with uncontrollable and ongoing hemorrhage(those with penetrating torso injuries). For them, it is recommended to delay or to limit iv fluid resuscitation preoperatively even if they have signs of hypoperfusion [3,18,21]. The presence of a central pulse should be considered the adequate endpoint of resuscitation[18].These patients must arrive as soon as possible in the operating room, eventually without passing through the Emergency Department.[14].

d) It is still available the recommendation to reestablish normotension through fluid therapy in cases with controllable hemorrhage, isolated head injury or isolated extremity injury[3,20].

What fluid shall we choose? The new volemic resuscitation strategies

The choice of the fluid is a complex one; the following factors must be taken into account[19]:

- early hemodynamic effects
- effects on haemostasis
- oxygen carriage
- distribution and capillary endothelial leak
- modulation of inflammatory response
- safety of administration
- pH buffering capacity
- method of elimination
- cost and stocking problems.

In the initial stages of trauma patient resuscitation, the precise fluid used is probably not important, as long as an appropriate volume is given [10, 22].

I) The most available solutions are isotonic crystalloid solutions-lactated Ringer's and normal(0,9%) saline. They are cheap, easy to store and to warm; they do not draw water out of the intravascular space; they are distributed throughout the extracellular space, across the intravascular and interstitial spaces[14,19].

They are also free of side-effects.

Lactated Ringer's solution has some advantages regarding buffering capacity, but carries a theoretical risk of iatrogenically increasing lactic acidosis in large doses or in patients with liver failure[19].

But also saline in large amounts may produce a hyperchloraemic acidosis [19, 23].

At present, isotonic saline is recommended as the first line fluid in the resuscitation of a hypovolaemic trauma patient[19].

II) Regarding colloids, the most used are polygelatin solutions (Haemaccel, Gelofusine) and hydroxyethyl starches(HES).

There are studies emphasizing that trauma patients mortality is lower in cases where crystalloid solutions were used in early stages of resuscitation, instead of colloids [14, 24, 25].

Colloids vary substantially in their pharmacology and pharmacokinetics and the experimental findings based on one colloid cannot be extrapolated reliably to another[10,22].

In the presence of Systemic Inflammatory Response Syndrome(SIRS), hydroxyl ethyl starch may reduce capillary leak[10].

There may be problems regarding storage and costs[12].

III) The small volume resuscitation concept

The concept of „small volume resuscitation" refers to the use of hypertonic saline in trauma.

Actually, intravenous hypertonic saline has been used in ICU for many years, to treat raised ICP in critically ill patients[26].

It increases cerebral perfusion and decreases brain swelling more effectively than conventional resuscitation fluids.

Hypertonic solutions extract fluid from cells, expanding intravascular volume by considerably more than the volume infused. For example, hypertonic saline 7,5% expands intravascular volume 8-10 times more than the equivalent volume of normal(0,9%) saline[15].

In prehospital trauma resuscitation setting, hypertonic saline has some clear advantages compared to conventional fluids:

1) The established prehospital infusion volume is 250 ml 7,5% hypertonic saline-this volume is easy to transport and easy to infuse by peripheral intravenous catheters. In the concentration of 7,5%, it does not damage peripheral veins[27].

2) Hypertonic saline has an established safety record-no adverse reactions have been reported in human studies using the recommended dose, which is 4ml/kgc of 7,5% hypertonic saline .

Despite frequent usage in patients with penetrating trauma, no evidence of increased bleeding had been reported [15,27].

3) Hypertonic saline improves regional blood flow to brain, renal and mesenteric vascular beds. It also decreases endothelial edema and improves capillary blood flow more effectively than alternative solutions [15,28].

4) Hypertonic saline may be anti-inflammatory and may decrease white blood cell adherence to capillary endothelium in each of the important microcirculatory beds. By limiting the inflammatory reaction, it may decrease the ARDS incidence[14,15,29]. Therefore, hypertonic saline may decrease Multiple Organ Dysfunction Syndrome (MODS) incidence.

Potential side effects of hypertonic saline have been carefully assessed and excluded. One of them might be hypernatraemia-but the highest value of serum sodium after administration of 250 ml 7,5% hypertonic saline was 155 mmol/l, and the mean value was 151 mmmol/l[15].

Hyperosmolality can also occur-the mean serum osmolarity was about 390 mOsm/l[14].

The high sodium load in elderly patient with impaired cardiac function might worsen congestive heart failure; convulsions and hyperchloremic acidosis are also possible side effects, but neither have been reported in patients[15].

Hyperchloremic acidosis occurs with hypertone saline, but lactic acidosis is simultaneously decreased due to better shock resuscitation. On balance, acidosis is therefore not worsened by hypertonic saline[15].

7,5% hypertonic saline has only a transitory effect on expanding the intravascular volume; the addition of colloids will determine this effect to last.

The result of this combination will be the hyperosmolar saline colloid solutions; examples are the 7,5%NaCl/6%dextran 70 solution called RescueFlow, or the 7,2%NaCl/6% HES 200.000 solution called HyperHAES.

These solutions are the best choice for small volume resuscitation in prehospital setting[30].

In a large clinical trial, the patients with penetrating trauma needing surgery had a significantly higher survival rate if treated with hyperosmolar saline dextran (RescueFlow), compared with those given standard of care [31].

Another study concluded that patients who have traumatic brain injuries in the presence of hypotension and receive small volume hyperosmolar resuscitation are about twice as likely to survive as those who receive standard of care[32].

The hyperosmolar saline colloid solutions do not compromise homeostasis, and are rarely associated with clinically manifest side-effects[33].

In a prospective study, 5% of the patients developed heat sensation, restlessness, poor taste and vomiting, which may have been solution-related[33].

4) Other options-which are not widely used-are oxygen carrying blood substitutes (perfluorocarbons)and Hb solutions.

Regarding the use of polymerized hemoglobin solutions, a study indicates that their use does not improve survival and results in a significant metabolic acidosis[30].

Regarding the use of perfluorocarbons, they may have an important role in improving oxygen transport to the tissues and survival in trauma patients with severe uncontrolled bleeding [5, 34].

Conclusion

In our times, the new volemic resuscitation strategies have become quite different from the well-known practice „2 large-bore intravenous lines running wide open".

The concepts of permissive hypotension and small volume hyperosmolar resuscitation have focused on enhancing oxygen delivery to the tissues and on microcirculatory resuscitation; must also be avoided the pressure-induced enhanced bleeding.

To reestablish the normotension is not anymore a target for the practitioner involved in trauma emergency care. Protocols must evolve from an algorithmic general response to a treatment strategy more and more adapted to patients individual needs.

References

1 Stern SA. Low volume fluid resuscitation for presumed hemorrhagic shock: helpful or harmful? Current Opinion in Critical Care.2001 Dec;7(6):422-30

2 Mattox KL. Permissive Hypotension. From Trauma Wiki[cited 2005 April 20]. Available from:http://beta.trauma.org/traumawiki/index php?title=permissive_Hypotension

3 Fowler R, Pepe PE. Fluid resuscitation of the patient with major trauma. Current Opinion in Anesthesiology 2002 April,15:173-178.

4 Brett AS. Fluid resuscitation in the initial management of posttraumatic shock: the concept of permissive hypotension. Clinical Intensive Care 2000 June;11(3):121-6.

5 Kreimeier U, Pruckner S, Peter K. Permissive hypotension during primary resuscitation from trauma and shock. In: Yearbook of Intensive Care and Emergency Medicine, 2001. Ed. Springer: 331-341.

6 Cushing H. Tumors of the nervus acusticus. 1917. WB Saunders, Philadelphia.

7 Van Aken H, Miller ED. Deliberate Hypotension. In Miller RD(ed).Anesthesia. 2000 Churchill Livingstone, Philadelphia, pp 11470-1490.

8 Bickell WH, Wall MJ Jr, Pepe Pe, et al. Immediate versus delayed fluid resuscitation for hypotensive patients with penetrating torso injuries. N. Engl. J. Med .1994 331:1105-1109.

9 Baron BJ, Sinert RH, Sinha AK et al. Effects of traditional versus delayed resuscitation on serum lactate and base deficit. Resuscitation. 1999 Dec; 43(1):39-46.

10 Nolan J. Fluid resuscitation for the trauma patient. Resuscitation 2001 Jan;48(1):57-69.

11 Spector SA, Rabinovici R. Initial Evaluation and Resuscitation of the Trauma Patients. In: Cameron JL(editor): Current Surgical Therapy. St Louis Mosby Seventh Edition 2001; pag 1050-1062.

12 Fluid resuscitation. In:EMS Magazine, 2002 March[cited 2005, 27 July]. Cited from:http://www. ems magazine.com./issues/article 0019b.html.

13 Scaletta T. Evolving Concepts in Trauma Management. From Emed Home[cited 2005 16 July]. Available from: http: //www.emed.home.come/features_archive-detail.cfm?FID=51

14 Pitti R, Steiner T, Da Conceicao M. Le choc hémorragique traumatique:principes de prise en charge. In: Choc hémorragique, Trauma. [cited 2005 August 9]. Available from:http://www.urgence-pratique. com/2 articles/medic/choc_hemo.htm.

15 Murray L, Cooper DJ. Hypertonic Saline Resuscitation for Traumatic Brain Injury? In: Yearbook of Intensive Care and Emergency Medicine, 2001. Ed. Springer:342-349.

16 Prough DS, Lang J. Therapy of patients with head injuries: key parameters for management.J. Trauma 1997. 42:S10-S18.

17 Demetriades D, Chan L, Cornwell E, et al. Paramedic versus private transportation of trauma patients. Effect on outcome. Arch. Surg. 1996;131:133-8.

18 Revell M, Porter K, Greaves I. Fluid resuscitation in prehospital trauma care: a consensus view. Emerg. Med. J. 2002; 19: 494-498.

19 Turner J, Nicholl J, Webber L et al. A randomized controlled trial of prehospital intravenous fluid replacement therapy in serious trauma. Health Technology Assessment 2000 Nov;4(31):1-57.

20 Pepe PE, Masesso JR JV, Falk JL. Prehospital fluid resuscitation of the patient with major trauma. In: Prehospital Emergency Care. 2002. Jan-Mar;6:81-91.

21 Hyde AJ, Graham TR. Prehospital Fluid Administration for Thoracic Trauma. Pre-Hospital Immediate Care, 1999 June;3:99-101.
22 Nolan J. Fluid Replacement. British Medical Bulletin. 1999 Dec; 55(4):821-43.
23 Williams EL, Hildebrand KL, Mc Cormick SA, et al.The effect of intravenous lactated Ringer's solution versus 0,9% sodium chloride solution on serum osmolality in human volunteers. Anesth Analg 1999;88:999-1003.
24 Choi PT, Yip G, Kuinonez LG. et al. Crystalloids vs. colloids in fluid resuscitation: a systematic revue. Crit. Care. Med.1999;27:p 200-210.
25 Schierhout G, Roberts I. Fluid resuscitation with colloid or crystalloid solutions in critically ill patients: a systematic revue of randomized trials. B.M. J. 1998;316:p 961-964.
26 Worthley L, Cooper D, Jones N. Treatment of resistant intracranial hypertension hypertonic saline.Neurosurgery.1988;68:478-481.
27 Dubick M, Wade C. A review of the efficacy and safety of 7,5% NaCl /6% dextran 70 in experimental animals and in humans. J. Trauma 1994;36:323-330.
28 Mazzoni M, Borgastrom P, Intaglietta M et al. Capillary narrowing in hemorrhagic shock is rectified by hyperosmotic saline –dextran re-infusion. Circ. Shock .1990;31:407-418.
29 Rotstein DO. Hypertonic saline as a resuscitation strategy for hemorrhagic shock-4-th international therapeutic fluids group meeting. 2000, June 14-15. Paris, France.
30 Handrigan Mt, Bentley TB, Oliver JD et al. Choice of fluid influences outcome in prolonged hypotensive resuscitation after hemorrhage in awake patients. Shock.23(4):337-343, April 2005.
31 Mattox KL, Maningas PA, Moore EE, et al. Prehospital hypertonic saline/dextran infusion for post-traumatic hypotension-the U.S.A. multicenter trial. Ann. Surg.1991; 213: 482-491.
32 Wade CE, Grady JJ, Kramer GC et al. Individual patient cohort analysis of the efficacy of hypertonic saline/dextran in patients with traumatic brain injury and hypotension. J Trauma 1997; 42: S61-S65.
33 Mauritz W, SchimettaW, Oberreither S.et al. Are hypertonic hyperoncotic solutions safe for prehospital small volume resuscitation? Results of a prospective observational study. European Journal of Emergency Medicine. 9(4):315-319, December 2002.
34 Stern SA, Dronen SC, McGoron AJ, et al.Effect of supplemental perfluorcarbon administration on hypotensive resuscitation of severe uncontrolled hemorrhage. Am J Emerg Med. 1995; 13: 269-275.