Prehospital Fluid Resuscitation of the Patient with Major Trauma

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Abstract

The most appropriate prehospital approach to resuscitative fluid interventions for trauma patients involves: determining the mechanism of injury (i.e., blunt versus penetrating versus thermal injury); identifying anatomic involvement (i.e., truncal versus isolated head injury versus isolated extremity injury); and staging the condition (i.e., hemodynamic stability versus instability versus moribund state). Based on available data, the liberal use of fluid infusions for presumed uncontrolled internal hemorrhage, such as that usually occurring after penetrating abdominal and thoracic injuries, is no longer advised. Although some infusion might be appropriate in patients with extremely severe hemorrhage (i.e., no palpable blood pressure, unconscious), the priority in such patients is rapid evacuation to definitive surgical intervention, with airway control and intravenous access provided en route. The data are less clear for patients with blunt injuries, particularly those with closed head injury. Most researchers would still recommend that patients with isolated extremity and head injuries, either blunt or penetrating, are candidates for immediate support of blood pressure through fluid infusions. However, the addition of potential intra-abdominal, intrapelvic, or intrathoracic injuries with uncontrolled hemorrhage confounds the decision-making process. Although conventional wisdom has been to provide aggressive blood pressure support under these circumstances through judicious use of isotonic, or perhaps hypertonic, fluid resuscitation, recent experimental data challenge even this philosophy. Use of new blood substitutes might help to resolve some of these issues by providing oxygen delivery with limited volume in the face of uncontrolled hemorrhage.
Key words: blood substitute; blunt injury; head injury; hemoglobin-based oxygen carrier; hemorrhage; injury; intravenous fluid; penetrating injury; resuscitation; shock; trauma.

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For more than three decades, the traditional approach to prehospital treatment of trauma patients with low blood pressure resulting from obvious or presumed hemorrhage has been to attempt to restore normal systemic arterial blood pressure.[1–7] The rationale for this approach has been to ensure and maintain vital organ perfusion while awaiting definitive surgical intervention and hemostasis.[1–3] The two modalities most often used to achieve this goal are: 1) rapid intravenous infusions of isotonic crystalloid or colloid solutions (normal saline, lactated Ringer's solution, albumin, or hetastarch); and 2) use of the pneumatic antishock garment (PASG), also known as military antishock trousers (MAST).[1–7]

The basis for this approach was largely established by the results of several elegant animal studies performed in the 1950s and 1960s.[8–10] Researchers found that animals receiving both blood and intravenous isotonic fluid as resuscitative measures had a greater likelihood of survival after severe hemorrhage when compared with animals receiving blood alone. The animals left untreated usually died or sustained irreversible organ damage. In addition, restoring blood pressure to normal or close to normal was associated with an improved outcome. The results of these studies immediately affected the standard treatment of wounded soldiers in Vietnam by battlefield medics, which contrasted with previous battlefield approaches.[11] In turn, the infusions of intravenous fluids to normalize blood pressure eventually was transferred to the streets of the United States and other western societies by the 1970s with the development of modern paramedic services.

The PASG was developed as a modification of the jet aviator's G-suit.[4,12–18] It was designed to help normalize blood pressure in the face of post-traumatic hypotension[4,13,19] through its ability to increase peripheral vascular resistance. The PASG also had the theoretic advantage of providing a potential tamponade effect for underlying injuries with active internal bleeding.[20] By the 1980s, the PASG and aggressive intravenous fluid resuscitation had become the standard of care for all trauma patients with potential signs or symptoms of presumed hemorrhagic shock.[1–4,6,7]

Despite these longstanding traditional management approaches, recent experimental and clinical data have indicated a modification to this universal approach to the trauma patient.[11,21–31] Although raising blood pressure and restoring

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perfusion to vital organs are clearly believed to be beneficial *after* hemorrhage is controlled, growing evidence indicates that raising blood pressure *before* achieving adequate hemostasis may be detrimental.[21–33] While the original animal studies that laid the groundwork for fluid resuscitation more or less involved controlled hemorrhage models with fixed amounts of blood loss,[8–10] more current studies have begun to examine the effects of raising blood pressure during uncontrolled hemorrhage.[21–31] These studies and their potential implications for clinical epidemiology, research, and management are discussed in detail.[34]

EVIDENCE AGAINST PREOPERATIVE BLOOD PRESSURE ELEVATION

Several animal studies performed in the 1980s and 1990s found that treatment with intravenous fluids before hemorrhage is controlled increases the mortality rate, especially if blood pressure is elevated.[21–27] Possible mechanisms responsible for worse outcomes include hydraulic acceleration of ongoing hemorrhage as a result of the elevated systemic blood pressure, mechanical dislodgment of active soft clot formation, and dilution of existing clotting factors from administration of large volumes of intravenous fluids.[21,33]

Research in humans, although limited, has supported this concept. A large, prospective, controlled clinical trial comparing immediate prehospital and emergency department intravenous fluid resuscitation with fluid resuscitation delayed until arrival in the operating room was conducted in Houston, Texas, in the 1980s and early 1990s.[33] In this study, hypotensive (systemic blood pressure <90 mm Hg) patients with penetrating torso injuries received either aggressive isotonic fluid resuscitation preoperatively (immediate group) or were given fluids only on arrival in the operating room (delayed group). Patients in the immediate resuscitation group had a higher mortality rate and a higher rate of postoperative complications compared with patients in the delayed resuscitation group. The authors of this study concluded that rapid administration of intravenous fluids before hemorrhage is controllable results in worse outcomes in this subpopulation of hypotensive patients with penetrating truncal injuries.

Despite some negative reaction to this initial clinical effort to resolve the question of when to provide fluid resuscitation,[35] the study remains the strongest available evidence to date regarding the issue. Also, counterarguments to the initial critiques of the study have noted that, at the very least, there is no demonstrable advantage to administering fluids in this subpopulation of trauma patients.[36] In addition, experimental studies in multiple animal models have all supported the concept of the detrimental effect of fluid resuscitation in uncontrolled hemorrhage.[21–31] Furthermore, this effect is found regardless of the solution used (i.e., blood, lactated Ringer's solution, hypertonic saline[22,26,29]). More importantly, in other prehospital studies, fluid resuscitation has yet to be correlated scientifically with improved survival in the clinical setting, particularly in moribund patients who intuitively would benefit the most.[37,38] The key factors in the survival of moribund patients appear to be limited to rapid transport to an appropriate trauma facility and aggressive airway control.[34,37,38]

Therefore, the working hypothesis at this time is that intravenous fluid resuscitation should probably be delayed until hemostasis is achieved. Following this line of thinking, if the anatomic site of bleeding is a large vessel within the thoracic or abdominal cavity, bleeding control will usually require surgical hemostasis. Although large intravenous fluid infusions may be necessary as soon as bleeding is controlled, aggressive fluid resuscitation in the prehospital or emergency department settings might still be detrimental in such patients.[28,33] In contrast, a hypotensive trauma patient with isolated severe hemorrhage from an extremity probably would benefit from immediate prehospital fluid resuscitation because the bleeding can be controlled outside of the operating room setting.

The consideration that hemostasis must be achieved before blood pressure is raised might also explain why antishock garments have not provided the anticipated benefit, particularly in patients with penetrating abdominal injuries.[39–44] In prospective trials, one difference between PASG patients and control patients has been a marked elevation in systolic blood pressure among the patients who had prehospital PASG application.[39] This elevation in systolic blood pressure is consistent with findings of retrospective studies and the initial anecdotal reports that originally espoused the widespread use of the PASG device. Based on traditional wisdom, most clinicians might anticipate that elevating blood pressure in the face of presumed hemorrhagic shock (post-traumatic hypotension) would be beneficial and improve patient outcome.[4,12,13,17] In these clinical trials, however, patients with major vascular injuries, both arterial and venous, were found to trend toward decreased survival.[39] Survival rates were approximately 90% for patients with solid organ, abdominal wall, or bowel injuries, with or without PASG application. When the subset of patients with large-vessel involvement (e.g., inferior vena cava, renal vein, hepatic artery) was examined, survival rates were 49% for the PASG group and 65% for the control group.[39] Again, this observation is compatible with the evolving paradigm that blood pressure elevation before hemostasis may be detrimental. It must be kept in mind, however, that through peripheral vascular compression, the PASG lowers total body cardiac output and that other mechanisms may also explain the disadvantages of this device.

BLUNT VERSUS PENETRATING TRAUMA

Although both experimental and clinical studies supporting delay of fluid resuscitation are somewhat compelling, the clinical studies generally involved patients with penetrating injuries. The role of fluid resuscitation in patients with blunt trauma is less clear. For example, after motor vehicle collisions, patients with multisystem injuries traditionally have been provided blood pressure support for major fractures and closed head injury.[45–52] However, a patient trapped in a vehicle who has altered mental status, left upper quadrant pain, a closed femur fracture, and hypotension presents a challenging dilemma for clinicians. Although aggressive preoperative fluid resuscitation theoretically could accelerate hemorrhage from a major splenic rupture or avulsion (a potential here), blood pressure support for brain injury is also considered to be a key therapeutic intervention. Also, a femur fracture can result in massive blood loss and fluid sequestration leading to profound shock conditions.

Nevertheless, recent experimental models of head injury have refuted the traditional universal use of aggressive blood pressure support in patients with head injury.[31] Although better outcomes have been correlated with increased systemic blood pressure in patients with severe head injury,[46–52] a higher blood pressure might simply be a marker for less severe head and systemic injury. Conversely, hypotension might be a marker for other factors that lead to a bad outcome and in itself is not necessarily detrimental.[31]

Therefore, further clinical and experimental studies are necessary to better delineate the role of fluid resuscitation in these complicated patients. The type of injuries, their anatomic location, and their severity must be kept in mind when such research efforts are designed.[34] Specifically, stratification of patients with and without head injuries must be made clear, as should stratification of those in extremis conditions.[32]

FLUID RESUSCITATION IN THE MORIBUND PATIENT

Although experimental evidence has demonstrated the potential detrimental effects of aggressive fluid resuscitation in uncontrolled hemorrhage, many animal studies have suggested that blood or fluid administration may be of value in patients with "severe circulatory compromise" (i.e., mean systemic arterial blood pressure <40 mm Hg).[23–25] However, patients with such a degree of hypotension typically present without a measurable blood pressure and are usually unconscious.

One retrospective study found that patients with such severe circulatory compromise might benefit from application of the PASG.[53] However, that conclusion may have been limited by the study design (selective retrospective analysis, accuracy of prehospital blood pressure measurements <70 mm Hg, and statistical power). In the prospective clinical trial of immediate versus delayed intravenous fluid resuscitation for penetrating torso injury,[33] patients who had a systemic arterial blood pressure of < 70 mm Hg were generally pulseless and clinically moribund. These patients' chance of survival was very low regardless of their prehospital treatment. At the same time, a retrospective subanalysis of all patients with injury severity scores > 26 showed that patients had worse outcomes with early fluid resuscitation.[33,36]

Despite the animal data, statistically significant evidence from prospective clinical trials is still lacking regarding the value of fluid resuscitation for the most severely injured patients. Nevertheless, considering the animal data and grim outlook for these patients, rapid fluid infusions still might be empirically reasonable in the absence of pulse and consciousness.

One consideration that might help guide such therapy for severely compromised patients is the use of end-tidal carbon dioxide ($ETCO_2$) measurements to detect critical perfusion levels in which total body oxygen consumption and CO_2 production begin to fall significantly.[54–56] End-tidal carbon dioxide levels are affected primarily by the level of pulmonary blood flow.[57] Thus in severely injured patients whose cardiac output is very low, little CO_2 is delivered from tissues to the pulmonary circuit and the exhalation of CO_2 is minimal.[57] Measurements of $ETCO_2$ therefore reflect total body cardiac output and can be used as one noninvasive means of monitoring directional changes in blood flow during cardiopulmonary resuscitation and fluid resuscitation (Figs. 1 and 2).[54,58] As shown in Figure 2, $ETCO_2$ can increase in response to aggressive fluid resuscitation in a patient with severe trauma.[58] However, the suggestion to begin fluid resuscitation according to $ETCO_2$ measurement in severe trauma is still only empiric and unstudied, particularly in those patients with uncontrolled hemorrhage. It is still not known whether $ETCO_2$ levels should constitute trigger points for fluid infusions or how much or how fast the fluids should be infused. Studies examining these issues are strongly encouraged.

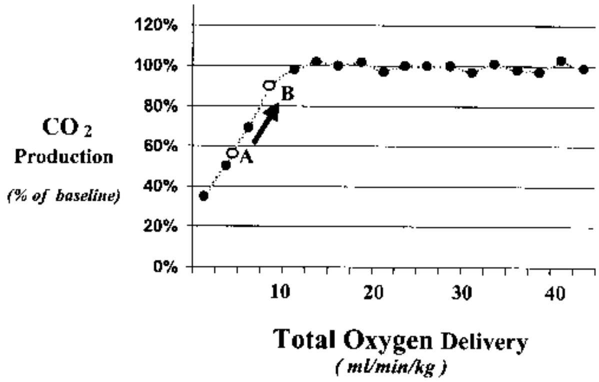

FIGURE 1. As total body oxygen (O_2) delivery falls below an observed threshold, tissue O_2 extraction is maximized and O_2 consumption (and the corresponding carbon dioxide [CO_2] production) decreases linearly. However, CO_2 production improves (point A to point B) through saline infusions (25 mL/kg) in a closed cardiovascular system (i.e., no uncontrolled hemorrhage). Based on data obtained from: Pepe PE, Culver BH. Independently measured oxygen consumption during reduction of oxygen delivery by positive end-expiratory pressure. Am Rev Respir Dis. 1985;132:788-92.

One confounding factor to be considered in these and any other studies of severe circulatory compromise is the detrimental effects of positive-pressure ventilation, particularly in patients with presumed hemorrhagic shock and severely depressed preload.[32,56] This additional controversy demonstrates how stratification of the severity (staging) of injuries is important in future research endeavors. Confounding the situation further is the recent experimental evidence that bolus infusions might be more detrimental than slow infusions in near-fatal models of uncontrolled hemorrhage.[28] In addition, such investigations have also used hypertonic solutions.[22,28] Therefore, the choice of fluid may also have to be considered when analyzing the results of these studies.

RESUSCITATION WITH NONCRYSTALLOID FLUIDS

The type of fluid that should be administered to trauma patients, even those with controlled hemorrhage, has been the subject of considerable debate. In North America, crystalloids are typically given to replace blood loss, but several studies have examined the use of fluid substitutes, such as colloids and hypertonic saline, for prehospital use.[46,59-66] Although isotonic crystalloids (e.g., normal saline or lactated Ringer's solution) remain the principal choice today in the United States, various types of colloids (all very different in themselves) are often the fluid of choice in other countries, such as Australia and European nations. Obviously, this further complicates the interpretation of meta-analysis and cross-study comparisons.

Proponents of the use of colloids, such as albumin, argue that fluids given to replace blood loss from the intravascular space should be designed to remain in that space.[67] The traditional teaching concerning crystalloid infusions has been the "3:1 rule," whereby 1 liter of crystalloid remains in the vascular space for every 3 liters infused.[1–3] This approach requires that large volumes of balanced salt solutions be administered to replace blood loss. It could be further argued that such infusions carry inherent risks. For example, infusions of such large volumes of crystalloid might decrease intravascular colloid osmotic pressure, potentially increasing the risk of developing or exacerbating pulmonary or cerebral edema. Nevertheless, most analyses have not yet proven definitively the advantage of albumin over crystalloids.[67,68]

The main alternative solutions studied for trauma resuscitation in the United States are hypertonic saline and nonprotein plasma expanders, such as dextran and hetastarch.[60–66] These fluids are less antigenic and less expensive than albumin but still may induce allergic reactions, coagulopathies, and seizures. Limiting the volumes infused may avoid some of these problems.[28,62]

Some clinical trials have specifically examined the efficacy of hypertonic saline.[61,62,64–66] Experimentally, hypertonic saline increases myocardial contractility, induces vasodilation to precapillary resistance vessels, and improves redistribution of fluid from the extravascular to the vascular compartments. In addition, because considerably smaller volumes are needed to restore intravascular volume, hypertonic saline solutions have been advocated as a resuscitative agent for field use, particularly for hypotensive patients with severe head injury.[46,66]

A prospective, multicenter trial from 1991 compared the outcomes of hypotensive trauma patients treated with normal saline or hypertonic saline in dextran (e.g., 7.5% NaCl in 6% dextran 70).[62] The two groups received equal volumes of fluid in the prehospital phase, followed by standard isotonic infusions in the emergency department. Although the patients in the hypertonic saline in dextran group had higher systemic blood pressures on arrival at the hospital, no significant difference in overall mortality was seen between the two groups at 24 hours. There were no clinically significant complications of hypernatremia or dextran-related allergic reactions among the patients who received hypertonic saline in dextran. The authors concluded that despite theoretic concerns, hypertonic saline in dextran was safe and further study was warranted.

However, one of the problems with this study was that relatively small volumes of fluid were administered in the prehospital setting to a group of patients with predominantly penetrating injuries. Both groups of patients received only 250 mL of either fluid, and the two groups were then given standard isotonic fluid infusions in similar fashions, confounding the results.

Future research could repeat this protocol with a third study arm that includes a group receiving no fluid in the field, or even a fourth group of patients who receive the single dose of 250 mL of hypertonic saline in dextran alone. This would help to address the question of whether any prehospital administration of fluids is warranted. Another consideration would be a comparison of preoperative versus postoperative infusion of hypertonic saline in dextran. Finally, use of hypertonic saline in dextran in patients with or without concomitant head injury (without any additional isotonic fluid infusions) should be examined more closely. Although other data seem to support its efficacy,[64–66] the general consensus is that definitive clinical trials are lacking.

In the meantime, most experts in the United States generally hold that limited crystalloid infusions are preferable to most other colloid infusions in the prehospital setting and in the early resuscitative phases of trauma care.[5] The rationale is that crystalloid is inexpensive, readily available, and nonantigenic and that administration of any isotonic infusions might be followed by plasma expanders or blood products in the emergency department or operating room. Also, no firm data support the use of the more expensive colloid solutions.[67] Nevertheless, the potential anti-inflammatory properties of some colloids also create some appeal for researchers and further study.[68]

The hetastarches, which are commonly used in Europe, and artificial hemoglobins are not as well studied as crystalloids and other

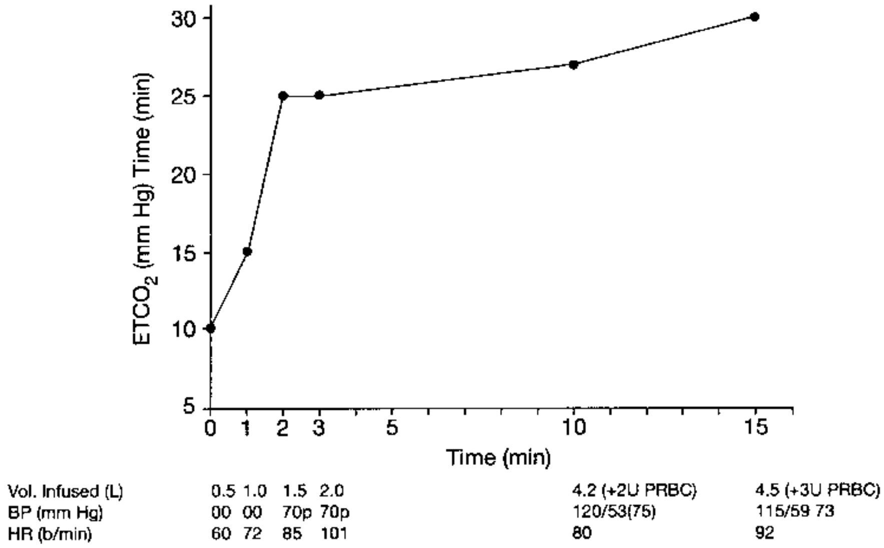

FIGURE 2. Response of end-tidal carbon dioxide ($ETCO_2$) to fluid resuscitation in a patient with severe trauma. BP = blood pressure; HR = heart rate; PRSC = prehospital fluid resuscitation. Reprinted with permission from: Falk JL, O'Brien JF, Kerr R. Fluid resuscitation in traumatic hemorrhagic shock. Crit Care Clin. 1992;8:323-40.

intravascular infusions.[59,63,69–78] Although further research into these types of resuscitative agents can be anticipated, many of these agents have been demonstrated to have a potential for raising blood pressure, a factor to consider in patients with uncontrolled hemorrhage.[79] For example, many of the hemoglobin-based oxygen-carrier products are thought to have nitric-oxide scavenging effects that lead to smooth-muscle constriction and subsequent blood pressure elevation.[79] However, it is hoped that the oxygen-carrying properties will outweigh the risk of secondary hemorrhage.[70,71]

One trial of the artificial hemoglobin diaspirin cross-linked hemoglobin (DCLHb) for trauma patients was terminated prematurely because of an increased number of deaths in the experimental group.[69] However, study bias and other factors may have led to this unexpected observation with this particular product.[69]

The recent introduction of another compound, HBOC-201, a purified form of bovine hemoglobin, has sparked renewed interest in artificial hemoglobin. HBOC-201 has been shown to have some promise in the laboratory.[70,71] In a swine model of trauma, Manning et al.[70] demonstrated the successful resuscitation and survival of asanguinous animals with uncontrolled hemorrhage that were given HBOC-201 compared with the high mortality found in the control group given standard crystalloid infusions.

In addition to improved oxygen-carrying capacity, HBOC-201 is relatively temperature stable[75] and could be stored in ambulances or in military far-forward positions, thus making it attractive to researchers. In terms of human data, HBOC-201 has been used successfully as a short-term blood substitute in hundreds of surgical patients[72–78] but has not yet been tested in any of the various subpopulations of trauma patients. Nevertheless, the concept of providing improved oxygen transport in the face of uncontrollable hemorrhage is appealing, and future clinical trials of this new compound would be worthwhile.

COMPLICATIONS OF PREOPERATIVE FLUID RESUSCITATION

Blood pressure alone is a poor predictor of shock (defined as inadequate perfusion of tissues). Despite the recently evolving paradigm that limiting preoperative fluid is preferable in most patients with internal hemorrhage to prevent secondary bleeding or acceleration of ongoing hemorrhage, concern still exists over the potential for other sequelae of shock. The duration and degree of systemic hypotension that a patient can withstand are the issue.

The clinical study comparing immediate with delayed fluid resuscitation showed a higher incidence of postoperative complications in the immediate resuscitation group (i.e., patients who received aggressive fluid administration in the prehospital and emergency department settings).[33] The incidence of acute respiratory distress syndrome, sepsis, coagulopathies, and renal failure was greater in the immediate resuscitation group compared with the delayed resuscitation group.[33] Several possible explanations have been offered for this observation, including decreased oxygen-carrying capacity from accelerated hemorrhage. The statistically lower hemoglobin levels on arrival in the emergency department for patients who received prehospital fluid resuscitation was more pronounced than those predicted by simple hemodilution alone.[33]

Again, these findings suggest that prehospital fluid might be harmful in patients with penetrating injuries to the torso and presumed internal bleeding in an urban emergency medical services (EMS) system with rapid transport intervals. Concern over more prolonged periods of hypovolemia without fluid resuscitation still exists, however, particularly in patients with blunt injury. More importantly, the definition of "prolonged" must be clarified. Above all, the dilemma of maintaining perfusion to the brain and other vital organs in patients with closed head injury remains a significant worry. Further study is required in venues in which there are prolonged transport times and potentially long delays until definitive surgical hemostasis. Also, many investigators consider blunt trauma a disease of massive soft-tissue injury and systemic inflammation, making it much different from the more focused penetrating injury that primarily leads to more localized injury and bleeding.[80]

CONCLUSION

The liberal use of fluid infusions for patients with presumed uncontrolled internal hemorrhage, such as that usually occurring after penetrating abdominal and thoracic injuries, is no longer advised. In fact, this recommendation is not new but actually long established, if one considers the observations made during past wars.[11,81] Although some infusion may be appropriate in patients with extremely severe internal hemorrhage, the priority in such patients is rapid transport to definitive surgical hemostasis.

The use of fluid infusions in patients with blunt trauma is not always clear. For patients with isolated extremity and head injuries (blunt or penetrating), immediate support of blood pressure through fluid infusions is considered beneficial when the bleeding is controllable. However, potential intra-abdominal, intrapelvic, or intrathoracic injuries and bleeding complicate the picture. Although blood pressure support through judicious use of isotonic or hypertonic fluid resuscitation generally has seemed reasonable, some recent experimental data even challenge this approach.

As implied in the preceding discussion, the traditional management of trauma in the prehospital setting paradoxically has been complicated by well-intentioned attempts to simplify it. Most EMS systems worldwide have developed treatment algorithms that often do not delineate between the different mechanisms of injury or anatomic location of wounds. The result has been mistreatment or misunderstanding of conflicting study data. In that respect, the classic debate of "scoop and run" versus scene stabilization has by definition oversimplified prehospital care strategies. Confounding the discussion further, only a few prospective controlled clinical trials have been conducted to validate or refute the prehospital interventions currently used for major trauma patients. Future discussions of the epidemiology, research, and ultimately management of trauma should include discrimination of the mechanism of injury (i.e., blunt versus penetrating versus thermal injury), anatomic involvement (i.e., truncal versus extremity versus isolated head injury), and staging of the condition (i.e., hemodynamic stability versus instability versus moribund state). In particular, the controversial issue of prehospital care for trauma patients with potential internal hemorrhage, with or without head injury, needs to be examined more closely. Finally, the various types of colloid and crystalloid fluids also need to be examined more closely, as does the development of oxygen-carrying media. In all of these cases, the confounding factor of current ventilatory techniques for severely injured patients must be considered as well.

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CONSENSUS PRESENTATION

The consensus group agreed that both animal and human trials indicate that patients respond differently to the various types of major trauma. Trauma patients are not a homogeneous group, and they vary in their physiologic reactions to the injury according to type and severity of the injuries they sustain. Some patients deteriorate into circulatory arrest, others develop severe shock states, and some remain in a hemodynamically stable condition. The mechanism of trauma and its anatomic involvement can significantly affect these responses and, in turn, influence the appropriate approach to fluid resuscitation. In addition, age and comorbidities can be significant factors. Children may have different fluid replacement requirements than do adults. Even among adults, the fluid replacement requirements of a younger person, both in terms of rates and absolute amounts, may differ from those of an elderly person, who might have several comorbid conditions. Thus no single recommendation can be made for prehospital fluid resuscitation of trauma patients.

In addition to patient variables, the resources and characteristics of the EMS system influence prehospital fluid resuscitation decisions. For example, the time it takes to transport the patient from the scene to the operating room may be a critical factor in determining appropriate fluid volumes and rates. Therefore, the consensus participants suggested that EMS providers collaborate with local and regional trauma services to develop a coordinated approach to fluid management in the trauma patient.

CONTROLLABLE HEMORRHAGE

The consensus group noted that a truly controlled hemorrhage is rare in the prehospital setting. A better term might be controllable hemorrhage, which reflects the likelihood that if bleeding were to occur, EMS providers would probably be able to control it. This generally applies to extremity injuries or superficial soft-tissue wounds of the trunk.

In the trauma patient with controllable hemorrhage, the consensus group recommended resuscitation with intravenous fluids if the patient shows clinical signs of shock, such as altered mental status or poor peripheral perfusion. Use of capnography, if available, was recommended as a potential means of evaluating perfusion.

Adequate volume should be infused to reverse clinical signs of shock while ensuring continued hemostasis. Fluid could be administered as intravenous boluses, with the volume based on factors such as the patient's hemodynamic status, age, and comorbidities and the amount of the hemorrhage. After each bolus, patients should be reassessed and given additional boluses as indicated. Reassessment should include examination of lung sounds and respiratory status for evidence of pulmonary edema.

UNCONTROLLABLE HEMORRHAGE

Based on the best available data, the appropriate approach to fluid resuscitation of a trauma patient with presumably uncontrollable hemorrhage largely depends on the mechanism of injury, be it penetrating or blunt, the anatomic involvement, and the severity of the physiologic compromise.

Penetrating Trauma

For the patient with penetrating trauma and presumed uncontrollable hemorrhage, the goal is to keep the out-of-hospital time as short as possible. Along with rapid evacuation, maintaining an open airway and ensuring adequate ventilation and oxygenation are the first priorities. Intravenous access, preferably with large-bore catheters, should be established en route to the hospital.

Although the available data are not definitive, for patients with presumed uncontrollable hemorrhage who show some signs of shock but are not near imminent circulatory arrest (e.g., responsive to verbal stimuli, palpable pulse), the amount of fluid administered should be limited. Intravenous access with two large-bore catheters should still be established en route to the hospital, but fluids should be limited to "keep vein open" (KVO). Small titrated boluses may be considered in some patients with severe tachycardia and those who show signs of further hemodynamic deterioration. However, there is insufficient clinical research to define the role of fluid administration more precisely in these patients.

Moribund patients with near-fatal injuries are reasonable candidates for more immediate fluid resuscitation. These patients typically have signs of extreme shock (e.g., unresponsive to verbal stimuli, no palpable peripheral pulse). As fluid is being administered, the patient's status should be continuously assessed. Once signs of improvement are noted, such as return of pulses or improved mental status, the rate of fluid administration can be tempered. The overall goal is not to return patients to a state of normal perfusion but rather to achieve perfusion adequate enough to maintain viability of vital organs until the time of definitive intervention.

Blunt Trauma

Although many trauma life support courses recommend that 2 liters of fluid be given to all

TABLE 1. Prehospital Fluid Resuscitation of Trauma Patients: Summary of Consensus Group Recommendations

- Discriminate between blunt, penetrating, and thermal injury or combinations thereof (e.g., blast injury).
- If there are concerns about potential internal or other uncontrollable hemorrhage, rapidly evacuate and transport the patient to definitive surgical facilities where hemostasis can be achieved.
- Manage the airway as indicated. Maintain airway patency, adequate oxygenation, and adequate ventilation. Avoid hyperventilation with positive-pressure breaths (either high respiratory rates or excessively large tidal volumes [>15 mL/kg]) because they can compromise cardiac output (due to decreased venous return) and cerebral perfusion (due to cerebral vasoconstriction). Respiratory rates of 8 to 10 breaths/min are generally adequate for patients with shock and even fewer (6 to 8 breaths/min) for those with circulatory or near-circulatory arrest.
- Establish intravenous access with large-bore catheters.
- If bleeding can be controlled (e.g., an isolated extremity injury), provide rapid intravenous fluid infusions for patients with blunt or penetrating trauma who show signs or symptoms of compromised circulation.
- In patients with penetrating trauma, infuse isotonic intravenous fluids if the patient is moribund (unconscious, no palpable pulses); otherwise, restrict fluid infusions, particularly in patients with penetrating torso injuries.
- In patients with blunt trauma, especially those with severe head injury, provide enough intravenous fluid infusion to maintain perfusion using clinical judgment but avoid excessive fluid administration (because of the theoretical risks of cerebral and pulmonary edema and secondary hemorrhage). Recognize that proper endpoints for determining adequacy of fluid resuscitation are still unknown.
- Consider end-tidal carbon dioxide ($ETCO_2$) monitoring as an adjunct to identify patients with severe circulatory compromise.
- Establish a coordinated prehospital treatment protocol with local and regional trauma services.

patients with blunt trauma, particularly those with signs of shock, the consensus group recommended that intravenous fluid administration be based more on the patient's clinical condition. Although underresuscitation must be avoided, providers should recognize that overresuscitation can also be harmful. The group agreed that the cookbook approach of at least 2 liters of fluid to all trauma patients and the approach that volume can be administered copiously without consequence should be discouraged.

Although the consensus was that fluid administration generally should be more liberal in patients with blunt injury than in those with penetrating injury, most patients with blunt trauma still should receive just enough prehospital fluid to maintain perfusion. Volume overload should be avoided to prevent complications such as pulmonary edema and the potential for worsening hemorrhage. The difficulty remains in terms of understanding how much and how fast to infuse, if at all.

Patients with closed head injury present a particular challenge. Intravascular volume support may be necessary to maintain cerebral perfusion and prevent secondary cerebral injury, but excess volume can increase cerebral edema and intracranial pressure. Pending further research to determine best clinical practices, EMS providers should consider these concerns when assessing and treating individual patients.

Again, the principal difficulty faced by the consensus group in discussing fluid resuscitation in blunt trauma is that the proper volume and rate for initial resuscitation in such patients, using current techniques, are unknown. More importantly, just as elusive are the proper endpoints to be measured that would guide such therapy. Overall, the group generally would err on the side of beginning to give fluids en route to hypotensive patients with blunt trauma, especially those with severe head trauma and those with any clear clinical signs of shock.

PREHOSPITAL FACTORS

The consensus group considered how EMS providers can determine the patient's perfusion and metabolic status using available techniques. Checking pulses and blood pressure can be difficult in the trauma patient, and these measurements are crude estimates of actual perfusion. Assessment of mental status as a sign of shock can be confounded by the presence of head injury, drugs, or alcohol. The participants agreed that $ETCO_2$ can be a useful adjunct, particularly in patients who have already been intubated, to assess perfusion in low-flow states. End-tidal carbon dioxide measurements provide an indication of changes in cardiac output and tissue perfusion. If a patient is in severe shock, $ETCO_2$ levels will be very low. Ventilation generally should be kept constant when using capnometry to assess perfusion. However, positive-pressure breaths can be detrimental in patients with severely compromised circulation, serving to confound the shock state even further. Therefore, in trauma patients with presumed hypovolemia and severe circulatory compromise, infrequent breaths, using 10 to 15 mL/kg tidal volume, are recommended as the constant. In EMS systems that have capnometry available, EMS providers should receive specific training in its use.

In some EMS systems, transport to the hospital may be prolonged, particularly if air medic units are unavailable or the system is located in a rural setting. This extended prehospital phase places an even greater burden on EMS personnel.

The risks to the patient become greater, and prehospital providers must be more rigorous and careful in their assessment and management. Because patients may be in severe shock for an extended period, the possibility of underresuscitating with fluids may be greater. Paradoxically, these patients are at higher risk for overresuscitation with fluids as well. Therefore, the recommendation for those experiencing extended prehospital phases of care is to limit intravenous fluids to the amount needed to reverse severe shock and to prevent the possible risk of excess fluid. Rescuers should also keep in mind that patients with crush syndrome may have a greater need for fluids than those without crush syndrome.

CONCLUSION

Because trauma patients are not a homogeneous group, fluid resuscitation in the field must be individualized according to the mechanism, anatomic involvement, and severity of the injury, as well as certain patient characteristics and estimated transport time to definitive surgical intervention. The consensus group agreed on certain points for prehospital fluid resuscitation, as summarized in Table 1. However, they also agreed that many questions remain unanswered regarding optimal prehospital fluid management. For example, proper clinical endpoints for the optimal type, amount, and rate of fluid to be administered are unknown. Clinical markers of adequate perfusion also are suboptimal. Consequently, no firm recommendations can be made as to what markers EMS providers should use to determine the appropriate use of fluid resuscitation.

References

1. American College of Surgeons Committee on Trauma. Advanced Trauma Life Support (ATLS) Course. Chicago, IL: American College of Surgeons, 1981, p 17.
2. American College of Surgeons Committee on Trauma. Advanced Trauma Life Support Program for Physicians: Instructor Manual. Chicago, IL: American College of Surgeons, 1984, pp 189-91, 201-3.
3. Fowler RL. Shock. In: Campbell JE (ed). Basic Trauma Life Support: Advanced Prehospital Care. 2nd ed. Englewood Cliffs, NJ: Prentice-Hall, 1988, pp 107-19.
4. McSwain NE. Pneumatic trousers and the management of shock. J Trauma. 1977;17:719-24.
5. American College of Surgeons Committee on Trauma. Shock. In: Advanced Trauma Life Support Program for Physicians: Instructor Manual. Chicago, IL: American College of Surgeons, 1993, pp 75-110.
6. Trunkey DD, Lewis FR. Current Therapy of Trauma 1984–85. Philadelphia, PA: B. C. Decker, 1984, p 1.
7. Caroline NL. Emergency Care in the Streets. 2nd ed. Boston, MA: Little, Brown, 1983, p 57.
8. Wiggers C. Physiology of Shock. New York, NY: Commonwealth Fund, 1950, pp 121-46.
9. Shires T, Coln D, Carrico CJ, et al. Fluid therapy in hemorrhagic shock. Arch Surg. 1964;88:688-93.
10. Dillon J, Lynch C Jr, Myers R, et al. A bioassay of treatment of hemorrhagic shock. Arch Surg. 1966;93:537-55.
11. Bickell WH, Shaftan GW, Mattox KL. Intravenous fluid administration and uncontrolled hemorrhage [editorial]. J Trauma. 1989;29:401.
12. Cutler BS, Daggett WM. Application of the G-suit to the control of hemorrhage in massive trauma. Ann Surg. 1971;173:511-4.
13. Civetta JM, Nussenfeld SR, Rowe TR, et al. Prehospital use of the military antishock trouser (MAST). J Am Coll Emerg Phys. 1976;5:581-7.
14. Gaffney FR, Thal ER, Taylor WR, et al. Hemodynamic effect of medical antishock trousers (MAST garment). J Trauma. 1981;21:931-7.
15. Gardner WJ, Storer J. The use of the G-suit in control of intra-abdominal bleeding. Surg Gynecol Obstet. 1966;123:792-8.
16. Kaplan BH. Emergency autotransfusion by medical pneumatic trouser: disclosure of invention, logbook entry 21. Ft. Rucker, AK: U.S. Army Aeromedical Research Unit, 1971.
17. Lilja GP, Batalden DJ, Adams BE, et al. Value of the counterpressure suit (MAST) in prehospital care. Minn Med. 1975;58:540-3.
18. Wayne MA, MacDonald SC. Clinical evaluation of the antishock trouser: retrospective analysis of five years of experience. Ann Emerg Med. 1983;12:342-7.
19. Niemann JT, Stapczynski JS, Rosborough JP, et al. Hemodynamic effects of pneumatic external counter pressure in canine hemorrhagic shock. Ann Emerg Med. 1983;12:661-7.
20. Ludewig RM, Wangensteen SL. Aortic bleeding and the effect of external counterpressure. Surg Gynecol Obstet. 1969;128:252-9.
21. Bickell WH, Bruttig SP, Millnamow GA, et al. The detrimental effects of intravenous crystalloid after aortotomy in swine. Surgery. 1991;110:529-36.
22. Bickell WH, Bruttig SP, Millnamow GA, et al. Use of hypertonic saline/dextran versus lactated Ringer's solution as a resuscitation fluid after uncontrolled aortic hemorrhage in anesthetized swine. Ann Emerg Med. 1992;21:1077-85.
23. Capone A, Safar P, Stezoski W, et al. Improved outcome with fluid restriction in treatment of uncontrolled hemorrhagic shock. J Am Coll Surg. 1995;180:49-56.
24. Kowalenko T, Stem S, Dronen S, et al. Improved outcome with hypotensive resuscitation of uncontrolled hemorrhagic shock in a swine model. J Trauma. 1992;33:349-53.
25. Stern SA, Dronen SC, Birrer P, et al. Effect of blood pressure on hemorrhage volume and survival in a near-fatal hemorrhage model incorporating a vascular injury. Ann Emerg Med. 1993;22: 155-63.
26. Stern SA, Dronen SC, Wang X. Multiple resuscitation regimens in a near fatal porcine aortic injury hemorrhage model. Acad Emerg Med. 1995;2:89-97.
27. Gross D, Landau EH, Assalia A, et al. Is hypertonic saline resuscitation safe in uncontrolled hemorrhagic shock? J Trauma. 1988;28:751-6.
28. Stern SA, Kowalenko T, Younder J, Wang A, Dronen SC. Comparison of the effects of bolus vs slow infusion of 7.5% NaCl/6% dextran-70 in a model of near-lethal uncontrolled hemorrhage. Shock. 2000;14:616-22.
29. Stern SA, Jwayyed S, Dronen SC, Wang X. Resuscitation of severe uncontrolled hemorrhage 7.5% sodium chloride/6% dextran 70 vs 0.9% sodium chloride. Acad Emerg Med. 2000;7:847-56.
30. Stern SA, Wang X, Mertz M, et al. Underresuscitation of near-lethal uncontrolled hemorrhage: effects on mortality and end-organ function at 72 hours. Shock. 2001;15:16-23.
31. Stern SA, Zink BJ, Mertz M, Wang Z, Dronen SC. Effect of initially limited resuscitation in a combined model of fluid-percussion brain injury and severe uncontrolled hemorrhagic shock. J Neurosurg. 2000;93:305-14.

32. Pepe PE. Resuscitation of the patient with major trauma. Curr Opin Crit Care. 1995;1:479-86.
33. 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;331:1105-9.
34. Pepe PE, Maio RF. Evolving challenges in prehospital trauma services. Prehosp Disaster Med. 1993;8:25-34.
35. Dick WF. Controversies in resuscitation: to infuse or not to infuse (1). Resuscitation. 1996;31:3-6.
36. Pepe PE. Controversies in resuscitation: to infuse or not to infuse (2). Resuscitation. 1996;31:7-10.
37. Copass MK, Oreskovich MR, Blaedogroen MR, et al. Prehospital cardiopulmonary resuscitation of the critically injured patient. Am J Surg. 1984; 148:20-4.
38. Durham LA, Richardson RJ, Wall MJ, et al. Emergency center thoracotomy: impact of prehospital resuscitation. J Trauma. 1992;32:775-9.
39. Bickell WH, Pepe PE, Bailey ML, et al. Randomized trial of pneumatic antishock garments in the prehospital management of penetrating abdominal injuries. Ann Emerg Med. 1987;16:653-8.
40. Honigman B, Lowenstein SR, Moore EE, et al. The role of the pneumatic antishock garment in penetrating cardiac wounds. JAMA. 1991;266:2398-401.
41. MacKersie RC, Christensen JM, Lewis FR. The prehospital use of external counter-pressure: does MAST make a difference? J Trauma. 1984;24:882-8.
42. Mattox KL, Bickell WH, Pepe PE, et al. Prospective MAST study in 911 patients. J Trauma. 1989;29:1104-12.
43. Pepe PE, Bass RR, Mattox KL. Clinical trials of the pneumatic antishock garment in the urban prehospital setting. Ann Emerg Med. 1986;15:1407-10.
44. Pepe PE, Wyatt CH, Bickell WH, et al. Use of MAST in penetrating cardiac injuries [abstract]. Chest. 1986;89:452.
45. Fowler RL, Pepe PE, Lewis RJ. Shock Evaluation and Management. In: Campbell JE (ed). Basic Trauma Life Support. 3rd ed. Upper Saddle River, NJ: Prentice-Hall, 1995, pp 111-24.
46. Zornow MH, Prough DS. Fluid management in patients with traumatic brain injury. New Horiz. 1995;3:488-98.
47. Miller JD, Butterworth JF, Gudeman SK, et al. Further experience in the management of severe head injury. J Neurosurg. 1981;54:289-99.
48. Joint Section on Trauma and Critical Care of the American Association of Neurological Surgeons and the Brain Trauma Foundation. Guidelines for the Management of Severe Head Injury. Park Ridge, IL: American Association of Neurological Surgeons, 1995.
49. Chestnut RM, Marshall LF, Klauber MR, et al. The role of secondary brain injury in determining outcome from severe head injury. J Trauma. 1993;34: 216-22.
50. Miller JD, Sweet RC, Narayan R, et al. Early insults to the injured brain. JAMA. 1978;240:439-42.
51. Siegel JH, Gens DR, Mamantov T, et al. Effect of associated injuries and blood volume replacement on death, rehabilitation needs, and disability in blunt traumatic brain injury. Crit Care Med. 1991;19:1252-65.
52. Wald S, Shackford ST, Fenwick J. The effect of secondary insults on mortality and long-term disability of severe head injury in a rural region without a trauma system. J Trauma. 1993;34:377-82.
53. Cayten CG, Berendt BM, Byrne DW, et al. A study of pneumatic antishock garments in severely hypotensive patients. J Trauma. 1993;34:728-35.
54. Pepe PE, Culver BH. Independently measured oxygen consumption during reduction of oxygen delivery by positive end-expiratory pressure. Am Rev Respir Dis. 1985;132:788-92.
55. Levine RL, Wayne MA, Miller DD. End-tidal carbon dioxide and outcome of out-of-hospital cardiac arrest. N Engl J Med. 1997;337:301-6.
56. Pepe PE, Swor RA, Ornato JP, et al. Resuscitation in the out-of-hospital setting: medical futility criteria for on-scene pronouncement of death. Prehosp Emerg Care. 2001;5:79-87.
57. Falk JL, O'Brien JF, Kerr R. Fluid resuscitation in traumatic hemorrhagic shock. Crit Care Clin. 1992;8:323-40.
58. Falk JL. End-tidal carbon dioxide monitoring during cardiopulmonary resuscitation. In: Advances in Anesthesia. St. Louis, MO: Mosby–Year Book, 1993, pp 275-88.
59. Gold MS, Russo J, Tissot M, et al. Comparison of hetastarch to albumin for perioperative bleeding in patients undergoing abdominal aortic aneurysm surgery. Ann Surg. 1990;211:482-5.
60. Griswold JA, Anglin BL, Love RT, et al. Hypertonic saline resuscitation: efficacy in a community based burn unit. South Med J. 1991;84:692-6.
61. Maningas PA, Bellamy RF. Hypertonic sodium chloride solutions for the prehospital management of traumatic hemorrhagic shock: a possible improvement in the standard of care? Ann Emerg Med. 1986;15:1411-4.
62. Mattox KL, Maningas PA, Moore EE, et al. Prehospital hypertonic saline/dextran infusion for post-traumatic hypotension: the USA multicenter trial. Ann Surg. 1991;213:482-91.
63. Munsch CM, MacIntyre E, Machin SJ, et al. Hydroxyethyl starch: an alternative to plasma for postoperative volume expansion after cardiac surgery. Br J Surg. 1988;75:675-8.
64. Vassar MJ, Perry CA, Holcroft JW. Prehospital resuscitation of hypotensive trauma patients with 7.5% NaCl versus 7.5% NaCl with added dextran: a controlled trial. J Trauma. 1993;34:622-33.
65. Vassar MJ, Fischer RP, O'Brien PE, et al. A multicenter trial for resuscitation of injured patients with 7.5% NaCl: the effect of added dextran 70. Arch Surg. 1993;128:1003-13.
66. Wade CE, Grady JJ, Kramer GC, Younes RN, Gehlsen K, Holcroft JW. Individual patient cohort analysis of the efficacy of HSD in patients with traumatic brain injury and hypotension. J Trauma. 1997;42:S61-S65.
67. Roberts I. Human albumin administration in critically ill patients. BMJ. 1998;317:235-40.
68. Cook D, Guyatt G. Colloid use for fluid resuscitation: evidence and spin. Ann Intern Med. 2001;135:205-8.
69. Sloan E, Koenigsberg MD, Gens D, et al. Diaspirin cross-linked hemoglobin (DCLHb) in the treatment of severe traumatic hemorrhagic shock: a randomized controlled efficacy trial. JAMA. 1999;282:1857-64.
70. Manning JE, Katz LM, Brownstein MR, Pearce LB, Gawryl MS, Baker CC. Bovine hemoglobin-based oxygen carrier (HBOC-201) for resuscitation of uncontrolled, exsanguinating liver injury in swine. Carolina Resuscitation Group. Shock. 2000;13:152-9.
71. McNeil JD, Smith DL, Jenkins DH, York GB, Josephs JJ. Hypotensive resuscitation using a polymerized bovine hemoglobin-based oxygen-carrying solution (HBOC-201) leads to reversal of anaerobic metabolism. J Trauma. 2001;50:1063-75.
72. Kasper SM, Grune F, Walter M, Amr N, Erasmi H, Buzello W. The effects of increased doses of bovine hemoglobin on hemodynamics and oxygen transport in patients undergoing preoperative hemodilution for elective abdominal aortic surgery. Anesth Analg. 1998;87:284-91.
73. Kasper SM, Walter M, Grune F, Bischoff A, Erasmi H, Buzello W. Effects of a hemoglobin-based oxygen carrier (HBOC-201) on hemodynamics and oxygen transport in patients undergoing preoperative hemodilution for elective abdominal aortic surgery. Anesth Analg. 1996;83:921-7.
74. LaMuraglia GM, O'Hara PJ, Baker WH, et al. The reduction of the allogenic transfusion requirement in aortic surgery with hemoglobin-based solution. J Vasc Surg. 2000;31:299-308.
75. Levy JH, Goodnough LT, Greilich P, et al. A room-temperature stable hemoglobin (HBOC-201) eliminates allogeneic

red blood cell (RBC) transfusion in post-operative cardiac surgery patients [abstract]. Circulation. 1998;17(suppl 1):132.
76. Monk TG, Goodnough L, Hughes G, Jacobs EE. Evaluation of the safety and tolerance of hemoglobin-based oxygen carrier-201 [abstract]. Anesthesiology. 1995;83:A285.
77. Sprung J, Popp H, O'Hara P, Woletz J. The successful use of hemoglobin-based oxygen carrier as a primary blood substitute during abdominal aneurysm repair with large blood loss. Anesth Analg. 2001;92:1413-5.
78. Wahr JA, Levy JH, Kindscher J, et al. Hemodynamic effects of a bovine hemoglobin based oxygen carrying solution in surgical patients [abstract]. Anesthesiology. 1996;85:A347.
79. Ketcham EM, Cairns CB. Hemoglobin-based oxygen carriers: development and clinical potential. Ann Emerg Med. 1999;33:326-37.
80. Santibanez-Gallerani AS, Barber AE, Williams SJ, Zhoa Y, Shires GT. Improved survival with early fluid resuscitation following hemorrhagic shock. World J Surg. 2001;25:592-7.
81. Cannon WB, Fraser J, Cowell EM. The preventive treatment of wound shock. JAMA. 1918;70:618-21.