Volume Replacement in Critically Ill Patients with Acute Renal Failure

MAXIMILIAN J. R. RAGALLER, HERMANN THEILEN, and THEA KOCH
Department of Anesthesiology and Intensive Care Medicine, University Hospital Carl Gustav Carus Medical Faculty, Technical University Dresden, Harvard Medical International Associated Institution, Dresden, Germany.

Abstract. Maintenance and restoration of intravascular volume are essential tasks of critical care management to achieve sufficient organ function and to avoid multiple organ failure in critically ill patients. Inadequate intravascular volume followed by impaired renal perfusion is the predominant cause of acute renal failure. Crystalloid solutions are the first choice to correct fluid and electrolyte deficits in these patients. However, in case of major hypovolemia, particularly in situations of increased capillary permeability, colloid solutions are indicated to achieve sufficient tissue perfusion. Whereas albumin should be avoided for correction of intravascular hypovolemia, synthetic colloids can restore intravascular volume and stabilize hemodynamic conditions. In addition to a faster, more effective and prolonged restoration of intravascular volume, colloid solutions are able to improve microcirculation. Of the synthetic colloids, hydroxyethyl starch (HES) solutions with a low in vivo molecular weight, such as HES 200/0.5, offer the best risk/benefit ratio. These solutions are safe with respect to effects on coagulation, platelets, reticuloendothelial system, and renal function, if used below their upper dosage limits. For patients with acute renal dysfunction, daily monitoring of renal function is necessary if colloids are required to stabilize hemodynamic conditions. In these patients, measurement of the colloidal osmotic pressure and adequate amounts of crystalloid solutions will reduce the risk of hyperoncotic renal failure. Of all colloids, gelatin and HES solutions with low in vivo molecular weight are preferred in these cases. In the very specific situation of kidney transplantation, colloid solutions should be administered in a restricted manner to organ donors and kidney recipients.

Intravascular volume is a crucial factor in the maintenance of hemodynamic stability, tissue oxygenation, and organ function. In principal, fluid intake and renal function govern fluid homeostasis. The intravascular volume, in particular, is regulated very closely by different mechanisms, including the transmembrane filtration pressure, interstitial hydrostatic pressure, colloid osmotic pressure (COP), lymphatic transport, sympathoadrenergic system, and renin-angiotensin system. Patients in intensive care units (ICU) experience conditions such as systemic inflammatory response syndrome (SIRS), sepsis or septic shock, ileus and subileus, acute bleeding, severe diarrhea, dehydration, fever, and therapy with diuretics or other compounds that reduce the intravascular volume. An extensive loss of intravascular fluid that cannot be compensated for by physiologic regulatory mechanisms in these critical situations leads to maldistribution of nutritional blood flow and generalized tissue hypoxia. If the volume loss is not adequately treated, multiple organ dysfunction and subsequent multiple organ failure may follow. It is therefore an essential task of critical care management to restore intravascular volume, to achieve adequate systemic circulation as well as sufficient microcirculation. This crucial situation becomes even more complex in patients who develop acute renal failure (ARF), which is often induced by intravascular hypovolemia and deterioration of renal perfusion. With respect to the importance of the kidney in fluid regulation, adequate renal function in critically ill patients in the ICU is inseparable from euvoemia and sufficient volume resuscitation.

Treatment of intravascular hypovolemia has changed significantly in the past decades. Whereas in the past blood and fresh-frozen plasma or plasma preparations were used as the main substitutes, crystalloids and synthetic colloids are now preferred. Despite several studies and consensus conference recommendations, neither the ideal resuscitation fluid for critically ill patients nor the best resuscitation fluid for patients in the ICU with ARF has been found. This review focuses on strategies for volume replacement in critically ill patients with accompanying ARF.

Hypovolemia in Critically Ill Patients
An extensive description of the pathophysiologic features of hypovolemia in different clinical situations is beyond the scope of this article and is presented elsewhere (1–3). Briefly, a lack of intravascular volume may be the result of acute hemorrhage, trauma, burns, excessive vomiting, diarrhea, or situations involving increased microvascular permeability, such as SIRS, sepsis, and anaphylaxis. Acute hypovolemia induces stimulation of the sympathoadrenergic and renin-angiotensin systems,
to maintain both cardiac output and arterial BP. These compensatory mechanisms lead to redistribution of blood flow to the cerebral, coronary, and renal circulations. However, if extensive blood loss (>30% of total blood volume) occurs and remains untreated, a number of pathophysiologic abnormalities can be observed on the microcirculatory level (e.g., hypoxia, endothelial cell swelling, capillary leakage, and release of cytokines) and promote multiple organ failure (1–4). In severe sepsis or septic shock, hypovolemia is attributable to the effects of endotoxins or antigens released from microorganisms, with subsequent liberation of a variety of vasoactive mediators. Venous pooling and increased microvascular permeability, with extravasation of fluid and macromolecules such as albumin, then occurs, followed by maldistribution of nutritional blood flow and disturbance of oxidative processes in the mitochondria (4–8). Most critically ill patients, however, suffer from the common pathophysiologic process termed SIRS. The inflammatory response, which is caused by surgery, trauma, or infection and is mediated by cytokines (e.g., interleukins and tumor necrosis factor-α), nitric oxide, metabolites of arachidonic acid, the complement system, and other compounds, is an essential process in critically ill patients that induces endothelial cell damage, followed by enhanced microvascular permeability. In this syndrome, which may vary with respect to the intensity of the inflammatory response, extravasation of fluid into the tissues is promoted. Therefore, the major goal of fluid management in critically ill patients is to compensate for intravascular hypovolemia, especially in cases of generalized capillary leakage (9). Situations involving intravascular dysvolemia are more complex if ARF aggravates the course of the disease. The incidence of ARF in ICU varies between 7 and 23%, depending on the case mixture, the severity of the underlying disease, and the definitions used to characterize ARF (10,11). Acute tubular necrosis, primarily induced by ischemia and nephrotoxic substances (drugs), is the predominant cause of ARF (11). The risk factors for ARF are volume depletion, sepsis, septic shock, hemorrhagic shock, contrast exposure, aminoglycoside therapy, age, and previous chronic renal disease or heart failure. The predominance of prerenal risk factors in the ICU setting highlights the importance of maintaining adequate renal perfusion for renal protection (11). Therefore, adequate volume replacement plays a crucial role in the treatment of critically ill patients with acute renal dysfunction or ARF. The essential aims of volume replacement therapy are presented in Table 1.

**Crystalloids**

Isotonic crystalloid solutions (Ringer’s lactate solution and 0.9% saline solution) are very commonly used to compensate for general losses of water and electrolytes and are usually the first choice for fluid replacement. Isotonic crystalloid solutions do not contain oncotically active macromolecules. Therefore, their effect on plasma volume expansion of approximately 200 ml for every 1000 ml administered, with an intravascular half-life of 20 to 30 min, is very limited (5). To substitute for blood loss, crystalloid solutions must be infused in four- to fivefold greater amounts, compared with colloid solutions, to exert the same volume effects. Moreover, it was demonstrated that crystalloids could not effectively restore microcirculatory blood flow in several organs in models of hemorrhagic shock (12,13). In the dynamic processes of SIRS or sepsis, with increased transmembrane fluid flux and low plasma COP, fluid shift from the intravascular compartment to the interstitial compartment is promoted if crystalloids are exclusively infused. In addition to their ineffectiveness in restoring sufficient tissue perfusion, this phenomenon increases the risk for tissue edema, particularly in the lung and gut mucosa (5). However, despite these results from experimental trials, an ongoing controversy exists regarding the use of crystalloids or colloids for adequate fluid replacement. To date, there has been no clinical trial with sufficient statistical power to compare the different fluids with respect to mortality rates. In two recent meta-analyses, published by Choi et al. (14) and Schierhout et al. (15), either no difference (14) or a 4% increase (15) in the absolute risk of death with colloid use was reported. However, the analysis by Schierhout et al. (15) is not free of important limitations, because it included several studies that used experimental colloid solutions and hypertonic saline solutions. Furthermore, none of the studies analyzed used modern colloid solutions, which are the current standard of care, particularly in Europe (4,16). Crystalloids have no specific nephrotoxic effects and are the basic fluids to fulfill the requirements for water and electrolytes in critically ill patients. In cases of major intravascular hypovolemia or severe sepsis, the exclusive administration of crystalloids is not appropriate because they are not able to sufficiently restore microcirculation, which is the major pathogenic factor in the development of multiple organ failure. Therefore, crystalloids should be used in conjunction with colloids to restore intravascular volume.

**Table 1. Goals of volume and fluid replacement for critically ill patients**

<table>
<thead>
<tr>
<th>Goal</th>
</tr>
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<tbody>
<tr>
<td>Achieve and maintain normovolemia and hemodynamic stability</td>
</tr>
<tr>
<td>Optimize oxygen delivery and oxygen consumption</td>
</tr>
<tr>
<td>Restore fluid homeostasis in the different fluid compartments</td>
</tr>
<tr>
<td>Ensure adequate plasma COP</td>
</tr>
<tr>
<td>Improve microcirculatory perfusion</td>
</tr>
<tr>
<td>Ensure appropriate inflammatory response and endothelial cell-leukocyte interactions</td>
</tr>
</tbody>
</table>

*a Adapted from reference 17. COP, colloid osmotic pressure.*

**Colloids**

**General Considerations**

The controversy regarding the use of colloid or crystalloid solutions for volume resuscitation in critically ill patients has been reinitiated by the meta-analyses mentioned above (14,15,17). Because of their content of macromolecules, colloids are retained within the intravascular space to a much greater extent, resulting in a greater intravascular volume effect. The volume effect exerted by colloids and their volume-supporting capacity with time depend on their concentration,
mol wt, molecular structure, COP, metabolism, and elimination rate. The characteristics of several colloid solutions, with respect to their volume effects and anaphylactic risk, are presented in Table 2.

**Albumin**

The administration of albumin, as the “natural colloid,” has been the standard method for the treatment of hypovolemia in critically ill patients in past decades. However, albumin administration is expensive and offers no apparent advantage, with respect to outcomes, for patients with hypovolemia or hypoalbuminemia (9,18,19). Albumin, which is usually used in 5% or 20% solutions, is thought to increase COP, which prevents extravasation of fluid from the intravascular space. In contrast, in situations with increased capillary permeability, the shift of albumin into the interstitial space is enhanced, and albumin thus aggravates interstitial edema formation (5,9).

With respect to the effect of albumin administration on patient outcomes, no evidence currently exists to support the use of albumin in patients in the ICU (9,18,19). Furthermore, a meta-analysis published by the Cochrane Injuries Group (20) indicated a 6% increase in mortality risk attributable to albumin administration in cases involving hypovolemia, hypoalbuminemia, or burns. When the available data are summarized, there is no convincing indication for the use of albumin in the treatment of hypovolemia or hypoalbuminemia in critically ill patients (9,17). According to the University Hospital Consortium Guidelines, albumin use should be restricted to cases in which nonprotein colloids are contraindicated (21).

**Gelatin**

Three different gelatin preparations (polypeptides from bovine raw material) are currently available, with a relatively low average mol wt of approximately 35,000. They contain a high proportion of low-mol wt components that are poorly retained in the intravascular space. Therefore, their effect on volume expansion is limited, and the duration of the effect does not exceed 2 h (Table 2). The low volume efficacy outweighs the advantage of the absence of dose limitations. Therefore, a colloid fluid regimen restricted to gelatin alone may be of limited value for patients with severe hypovolemia (5,17). Gelatin solutions were long thought not to interfere with plasma coagulation factors or platelets. Recent studies, however, demonstrated that gelatin compromises platelet function and the activity of the plasma von Willebrand’s factor and interferes with the polymerization of fibrin monomers, thus reducing the quality of clot formation (22,23). Because of their capacity for histamine release, gelatin solutions are associated with a higher incidence of anaphylactoid reactions, compared with hydroxyethyl starch (HES) solutions (24). In addition, the high potassium and calcium contents of 3.5% urea-cross-linked gelatin explain the unsuitability of this colloid, especially for patients with ARF or those undergoing digitalis therapy (25). Furthermore, the bovine raw material of gelatin solutions is associated with the theoretical risk of transmission of bovine spongiform encephalopathy, which must be taken into account in benefit/risk analyses (4). Because of these issues, gelatin must be considered a colloid of second choice for critically ill patients.

**Dextran**

Dextran, a single-chain polysaccharide of bacterial origin, is still commonly used for plasma expansion in many countries. Three preparations are currently available, i.e., 6% dextran 70, 6% dextran 60, and 10% dextran 40. Dextran solutions have sufficient initial plasma volume expansion effects, as well as prolonged intravascular volume effects because of a high water-binding capacity (approximately 20 to 25 ml/g dextran). However, these solutions have been associated with serious side effects, such as coagulation abnormalities (dose, >1.5 g/kg per d) (2,4,26), anaphylactic/anaphylactoid reactions (24), and the onset of oliguric or anuric renal failure (27). The implementation of hapten prophylaxis reduced the incidence of severe dextran-induced anaphylactic reactions from 1/2000

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**Table 2. Characteristics of colloid solutions**

<table>
<thead>
<tr>
<th>Colloid Solution</th>
<th>Maximal Volume Effect (%)</th>
<th>Duration of Volume Effect (100%) (h)</th>
<th>Risk of Anaphylaxis (Grade III or IV) (%)</th>
<th>COP (mmHg)</th>
<th>Dose Limit (ml/kg per d)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Albumin, 5%</td>
<td>100 to 150</td>
<td>4</td>
<td>0.032</td>
<td>20</td>
<td>None</td>
</tr>
<tr>
<td>Albumin, 20%</td>
<td>Up to 400</td>
<td>4 to 6</td>
<td>0.032</td>
<td>74</td>
<td>None</td>
</tr>
<tr>
<td>Gelatin MF, 3%</td>
<td>100</td>
<td>1 to 2</td>
<td>0.056</td>
<td>24</td>
<td>None</td>
</tr>
<tr>
<td>Dextran 60, 6%</td>
<td>130</td>
<td>4 to 6</td>
<td>0.067&lt;sup&gt;b&lt;/sup&gt;</td>
<td>26</td>
<td>20</td>
</tr>
<tr>
<td>Dextran 40, 10%</td>
<td>175</td>
<td>3 to 4</td>
<td>0.067&lt;sup&gt;b&lt;/sup&gt;</td>
<td>170</td>
<td>20</td>
</tr>
<tr>
<td>HES 200/0.5, 6%</td>
<td>100</td>
<td>4</td>
<td>0.023</td>
<td>26</td>
<td>33</td>
</tr>
<tr>
<td>HES 200/0.5, 10%</td>
<td>150</td>
<td>4</td>
<td>0.023</td>
<td>64</td>
<td>20</td>
</tr>
<tr>
<td>HES 200/0.62, 6%</td>
<td>110</td>
<td>8</td>
<td></td>
<td>28</td>
<td>20</td>
</tr>
</tbody>
</table>

<sup>a</sup> HES, hydroxyethyl starch. The pharmacological characteristics of colloid solutions were observed in healthy patients. The maximal volume effect represents the intravascular volume expansion related to the infused volume. Data were taken from references 2, 4, 9, 17, and 24 and from product information.

<sup>b</sup> With hapten prophylaxis.
(1975 to 1979) to 1/70,000 (1983 to 1992) (28). However, dextran solutions are still the colloids associated with the most severe anaphylactic reactions, because hapten prophylaxis cannot fully eliminate this phenomenon (24). In some case reports (29,30) and in a study of dextran treatment in stroke (31), the induction of oliguric or anuric renal failure was associated with the use of 10% dextran 40 solutions. Because dextrans can cause uterine hypertension with signs of acute fetal distress, when used during delivery, they are absolutely contraindicated for correction of hypovolemia in pregnant patients (32). However, dextrans have well documented effects in the reduction of endothelial cell-blood cell interactions, which are important for adequate capillary perfusion and may be of value in preventing excessive activation of inflammatory cascade systems (17). Recently, an experimental study demonstrated a beneficial effect of dextran on leukocyte adherence in an animal model of ischemia-reperfusion injury (33). When the data on dextran solutions are summarized, the potential advantages are completely outweighed by the serious side effects. Therefore, the routine use of dextran solutions is no longer warranted for intravascular volume expansion in critically ill patients. The current use of dextran is <1% of total colloid consumption in German ICU and is also decreasing in almost all other European countries (4,16,17).

**Hydroxyethyl Starches**

Polydisperse HES, as modified natural polymers of amylopectin, are available in different preparations in several countries (6% HES, 450/0.7; 6 or 10% HES, 200/0.5; 6% HES, 200/0.62; 6 to 10% pentastarch, 264/0.45; 6% HES, 130/0.4; 6% HES, 70/0.5). According to the number of hydroxyethylations at carbon positions C2, C3, or C6 (degree of substitution), the HES are more or less resistant to degradation by plasma α-amylase. The degree of substitution and the C2/C6 hydroxyethylation ratio, in combination with the mol wt, are responsible for the pharmacokinetic characteristics of HES solutions. A detailed overview of their pharmacokinetics and elimination pathways is provided elsewhere (4,34). However, it must be emphasized that the *in vivo* mol wt after partial hydrolysis of the original solution is the critical parameter that determines both the effects on intravascular volume expansion and the side effects. The *in vivo* mol wt depends on the original mol wt, the extent of hydroxyethylation, and the C2/C6 ratio. The incidences of coagulation disorders and renal dysfunction were found to be associated with increasing values for these parameters (30,34,35). Accordingly, the ideal HES should have the lowest *in vivo* mol wt but should be above the threshold for renal elimination (50 to 60 kD) to guarantee effective intravascular volume expansion and restoration of an adequate COP, combined with a low rate of side effects. Compared with other HES solutions, HES 200/0.5, as a middle-mol wt and low-substituted solution in 6% (iso-oncotic) or 10% (hyperoncotic) preparations, seems to have some advantages (4,17,34,35). The successful use of HES 200/0.5 for intravascular volume expansion has been demonstrated in several settings, such as isovolemic hemodilution, perioperative volume replacement, cardiac surgery, trauma, and sepsis (4,19,36,37). Furthermore, experimental studies showed beneficial effects of HES solutions on inflammatory processes such as endothelial cell activation and monocyte chemotaxis or chemotactic cytokine release (38,39). Other authors suggested that HES may be able to ameliorate capillary leakage secondary to inflammation (40,41). However, whether HES solutions are able to “seal the leak” in human patients with severe sepsis, as proposed by these results, must be clarified by additional studies. A recent clinical trial that compared HES 200/0.5 with 20% albumin for volume replacement in septic patients demonstrated better regional microcirculation [splanchnic perfusion, measured by gastric tonometry (pH measurements)] in the group of patients treated with HES (19). Although HES seems to be very effective in correcting hypovolemia, potential side effects must also be stressed (42), such as coagulation disorders. Reduction of factor VIII and von Willebrand’s factor activity, impairment of platelet function, or increases in the activated partial thromboplastin time were observed after repeated treatment with HES preparations with high *in vivo* mol wt (HES 450/0.7 and HES 200/0.62) (34,42). The incidence of coagulation disorders after administration of rapidly degradable HES solutions with low *in vivo* mol wt, up to their dosing limits, was reported to be very low (34,35). For these HES solutions, clinically relevant bleeding complications have not yet been reported (34). The risk of anaphylactic reactions, and severe anaphylactic reactions (grade III/IV) in particular, seems to be the lowest among all synthetic colloids. Lethal anaphylaxis has never been observed (24). Another specific side effect is the accumulation and slow degradation of HES in the cells of the mononuclear phagocytic system. The determinants for the ingestion of HES by the reticuloendothelial system remain unclear. However, several *in vitro* studies failed to demonstrate an impairment of mononuclear phagocytic system function after HES exposure (4,39,43). Overall, there is considerable evidence that HES 200/0.5 is an effective, safe, and economically attractive colloid solution for volume replacement in critically ill patients.

**Colloids and ARF**

After the first description of ARF occurring after the infusion of dextran (27,29), additional case reports of ARF occurring after gelatin, 10% HES, 20% mannitol, or concentrated (20%) albumin solution administration were published, as summarized by Baron (30). However, most cases of dextran-induced renal failure were associated with several other patient risk factors, such as age, arteriosclerosis, preexisting renal insufficiency, colloid use for nonsurgical reasons, and dehydration before colloid use. In addition, high doses of 10% dextran 40 were administered for several days (29). Currently, three hypotheses are presented to explain the mechanisms of ARF associated with colloid use, *i.e.*, accumulation of a low-mol wt fraction in the renal tubules, induction of osmotic nephrosis-like lesions (vacuolization of the proximal tubular cells), and hyperoncotic renal failure (30). The intratubular accumulation, hyperviscosity, and precipitation of low-mol wt fractions of dextran in the presence of decreased transglomerular filtrate seem to be specific for dextran solutions (27). The induction of osmotic nephrosis-like lesions has been reported for dextran, as well as for gelatin and HES (30,44). Although these lesions were initially considered to be responsible for the deterioration in renal function, the significance of vacuoliza-
tion of the proximal tubular cells remains incompletely understood, because these alterations were also observed without accompanying ARF. Furthermore, HES was never detected in these vacuoles (4,30). The hypothesis of hyperoncotic renal failure, which was first described by Moran and Kapsner (45), seems to fit best to the pathophysiologic considerations and clinical data. The GFR depends on the imbalance between positive hydrostatic pressure (renal perfusion pressure) and oncotic forces at the membrane of the glomeruli. In cases of low renal perfusion pressure in the glomerular arterioles, an increase in the COP (attributable to an accumulation of unfilterable, osmotically active substances in the plasma) induces reduction or cessation of glomerular filtration. The back-leakage of filtrate across ischemic or otherwise damaged tubular epithelium additionally reduces renal excretory function (30,45,46).

Considering this pathogenesis, it can be hypothesized that all hyperoncotic colloid solutions (such as 20 or 10% albumin, dextran 40, 3.5% gelatin, 10% HES 200/0.5 to -62, and 6% HES 450/0.7) can induce ARF. In the case of HES, the risk of high plasma COP and thus the risk of ARF are probably increased by high concentrations of the colloid (10% HES) or repeated administration of HES with a high in vivo mol wt (4,30). However, as pointed out earlier, risk factors such as hemodynamic instability, obstructive vascular disease, dehydration, and preexisting renal insufficiency seem to have greater predisposing effects on the development of ARF than does the type of colloid administered (29,30). Furthermore, HES solutions with a low in vivo mol wt, such as HES 200/0.5, did not increase the risk for ARF even when used in large amounts intraoperatively (47,48) or postoperatively (49).

In a retrospective study, Legendre et al. (44) reported an 80% rate of osmotic nephrosis-like lesions in transplanted kidneys after routine administration of HES 200/0.62 to brain-dead donors. However, the lesions did not induce significant impairment of transplant function or increases in serum creatinine levels in the recipients (44). In a prospective randomized study reported by Cittanova et al. (50) in 1996, which compared HES 200/0.62 (33 ml/kg per d; Elohaes Laboratoires Fresenius, Louvier, France) with gelatin, slower recovery of serum creatinine levels and a greater number of patients requiring hemodialysis were observed in the early phase after transplantation if kidneys were exposed to HES in the brain-dead donors. Osmotic nephrosis-like lesions were detected only in biopsy specimens from the HES-treated group. Although no HES was found in these lesions, the authors suggested that HES solutions should be avoided for volume replacement in brain-dead organ donors (50). However, in two other studies, neither the incidence of osmotic nephrosis-like lesions nor any adverse effects of HES 200/0.5 on early graft function after kidney transplantation could be demonstrated (51,52). These data suggest that HES 200/0.62, with a higher in vivo mol wt, should be avoided in organ donors, whereas HES 200/0.5 seems to be safe for volume replacement in brain-dead organ donors, with respect to early transplant function. Considering the exclusive situation of kidney transplantation, with several other factors potentially causing early graft dysfunction (cyclosporine use, the hemodynamic status of the donor and recipient, and the use of preservation solution), and the limitations of these small studies, restricted use of HES can be proposed. The use of HES should be limited to iso-oncotic HES solutions with low in vivo mol wt, at a maximum dose of 15 ml/kg per d (52).

Conclusion

Resuscitation of intravascular volume and fluid replacement are cornerstones in the treatment of critically ill patients. Restoration of macrocirculation and microcirculatory perfusion is the primary goal of volume therapy, to prevent deleterious consequences such as organ dysfunction or multiple organ failure. Because there are no pharmacologic strategies to prevent or even treat ARF, the maintenance of sufficient renal perfusion, supported by adequate intravascular volume expansion, is of substantial importance for the avoidance of ARF.

Crystalloid solutions represent the basic treatment to correct water and electrolyte deficits after blood or fluid loss. Therefore, they are always the first choice for treatment of intravascular hypovolemia. However, for major hypovolemia, and particularly in cases with microcirculatory disturbances and increased capillary permeability, colloid solutions are necessary to guarantee sufficient tissue perfusion. There is growing evidence that albumin should not be routinely used for correction of hypovolemia or hypoalbuminemia in critically ill patients. Albumin should be restricted to precisely defined indications, e.g., situations in which synthetic colloids are contraindicated or have been used to their maximal doses. In cases of intravascular hypovolemia with reduced cardiac output and compromised tissue perfusion, all synthetic colloids can restore intravascular volume and stabilize the hemodynamic conditions. Of the synthetic colloids, HES solutions with low in vivo mol wt, such as HES 200/0.5, demonstrate the best risk/benefit ratios. Faster, sufficient, prolonged restoration of intravascular volume, attributable to their osmotic power, can be predicted, as can improved microcirculation. If used in dosages below their upper limit (33 ml/kg per d), HES solutions are obviously safe with respect to effects on coagulation, platelets, the reticuloendothelial system, and renal function. All synthetic colloids, as well as albumin, may produce anaphylactoid reactions, which are generally very rare. A lower incidence seems to be associated with HES, compared with other colloid solutions.

For patients with acute renal dysfunction, careful daily monitoring of renal function is required if colloids are used. The risk of hyperoncotic renal failure can be further reduced by the administration of adequate amounts of crystalloid solutions. The measurement of COP may facilitate safe fluid management in these cases. Of all colloids, gelatin and HES solutions with low in vivo mol wt should be preferred in these cases. On the basis of the currently very limited data regarding the specific situation of kidney transplantation, colloid solutions should be administered in a restricted manner to organ donors and kidney recipients.
References


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