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Review

Unnecessary fluid therapy in patients with shock: Five classic fluid pitfalls and five ways to do it better

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ABSTRACT

Intravenous fluid therapy remains a cornerstone of shock management, yet is frequently misapplied. Unnecessary administration can lead to avoidable harm, including fluid accumulation, electrolyte disturbances, and delayed recovery. Despite good intentions, clinicians often fall into predictable patterns that drive overinfusion. Recognizing and correcting these habits is an important step toward safer and more effective fluid management, which may contribute to better patient outcomes.

This narrative review highlights five recurring pitfalls in fluid management: confusion between resuscitation, maintenance, and replacement indications; failure to account for hidden fluid sources; underestimation of the impact of sodium burdens; reflexive fluid administration in response to misinterpreted markers such as hyperlactatemia, low central venous pressure or reduced urine output; and finally, a narrow focus on fluid responsiveness without consideration of fluid tolerance.

To address these challenges, we propose a pragmatic, physiology-based approach to fluid stewardship. Key strategies include adopting a phase-based model for fluid therapy, minimizing unnecessary sodium and chloride exposure, interpreting fluid balance data with caution, initiating early vasopressor support when appropriate, and employing structured de-resuscitation (and preventive) strategies rather than relying solely on loop diuretics.

Thoughtful, physiology-driven fluid therapy requires more than reflexes, fixed volumes and flowcharts, it demands context-sensitive, physiology-informed decision-making. By recognizing common mistakes and adopting simple yet powerful principles, clinicians can shift from automatic to analytic and from flood to finesse.

Background

Intravenous fluid therapy is often initiated with the best intentions. However, when resuscitation fluids are administered without clear physiological targets, or when maintenance fluids are misused in the context of shock, these good intentions can harm the patient.

Historically, early goal-directed therapy was considered standard of care, guided by protocols aiming to optimize arterial oxygen delivery. In practice, this often translated into rapid fluid administration and a focus on urine output as surrogate indicators of treatment success. A continuous stream of evidence in recent years has shown that various aspects of fluid therapy—such as fluid type, sodium content, dosing strategies and guiding parameters such as fluid responsiveness—can influence outcomes and that inappropriate or excessive fluid administration may be harmful in ICU patients Injudicious fluid administration has been linked to fluid accumulation syndrome with longer durations of mechanical ventilation, increased risk of acute kidney injury, and prolonged hospital stays [1].

This review, summarized in Fig. 1, highlights common misconceptions and frequently overlooked insights, and proposes practical guiding principles to help avoid these classic pitfalls.

Abbreviations: AKI, acute kidney injury; CVP, central venous pressure; DPB, diastolic blood pressure; EVLW, extravascular lung water; FR, fluid responsiveness; FT, fluid tolerance; GEDV, global end diastolic volume; IAP, intra-abdominal pressure; ICU, intensive care unit; IV, intravenous; IVC, inferior vena cava; PAOP, pulmonary artery occlusion pressure; PCO_2 gap, central venous–arterial pCO_2 gradient; RRT, renal replacement therapy; US, ultrasound; VEXUS, venous excess ultrasound score.

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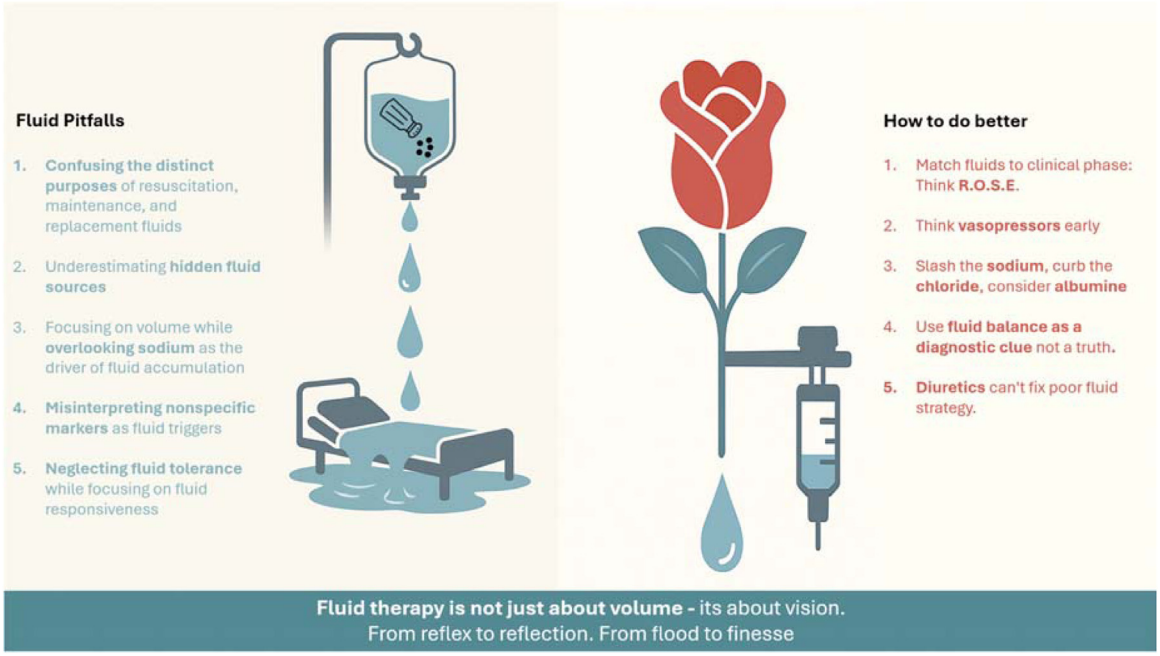

Fig. 1. Five classic fluid pitfalls and five ways to do it better.

Five classic pitfalls in fluid therapy

Confusing the distinct purposes of resuscitation, maintenance, and replacement fluids

In the high-stakes environment of shock, the administration of intravenous fluids is often viewed as universally beneficial, but this assumption is dangerously flawed. A common clinical error is the failure to distinguish between the three different reasons for which intravenous fluids are prescribed: resuscitation fluids in case of shock, maintenance fluids to cover basic needs and replacement fluids to correct past or ongoing fluid losses. These are three very distinct indications and should be in separate mental buckets, each with their own considerations. While it is increasingly accepted that resuscitation fluids must be isotonic and administered as a rapid bolus, it is often overlooked that maintenance fluids should closely match the water and electrolyte requirements of a healthy human diet. Typically, these needs include approximately 25–30 mL/kg/day of water (making maintenance fluids unnecessary when other fluid sources or oral intake are sufficient), 1 mmol/kg/day of sodium and potassiumand 50–100 g of glucose [2,3]. Replacement fluids cover losses that may originate from various sources [4]. Fluids used to replace these losses should closely match the composition of the fluid lost. Using the same fluid to treat all types of losses will inevitably disrupt homeostasis. Table 1 summarizes the composition of various types of fluid losses and provides corresponding recommendations for fluid replacement. A further contributor to fluid accumulation is the common omission of stop dates or reassessment intervals in maintenance and replacement prescriptions. Maintenance and replacement fluid orders often continue unnoticed for days, with no automatic trigger to stop or review them: a “set it and forget it” approach. Without clear tracking and accountability, fluid accumulates quietly, making meaningful fluid stewardship nearly impossible.

Our advice: Clearly differentiate between resuscitation, maintenance, and replacement fluids, as each fulfills a distinct physiological purpose. Avoid using a one-size-fits-all approach, which can disrupt homeostasis and unnecessarily increase electrolyte load.

Underestimating hidden fluid sources

While fluid resuscitation often draws the most attention, it represents only a small part of the total fluid volume ICU patients receive. In practice, resuscitation fluids account for a mere 6–10% [5,6]. Frequently overlooked are background fluids, or “fluid creep” – a term that encompasses flushes, line patency fluids and medication diluents – which account for roughly one third of water and sodium burdens administered to ICU patients. Fluid creep can easily provide enough daily fluid to make maintenance fluids redundant—but this overlap often goes unrecognized. An observational study found that ICU patients received a median of 645 mL of these hidden fluids and an additional 2592 mL of discretionary fluids over just 24 h [7]. Failing to account for this hidden volume could turn your maintenance strategy into an accidental flood.

Our advice: Always account for fluid creep (and implement this in fluid balance calculations). It may render additional maintenance or even replacement fluids unnecessary.

Focusing on volume while overlooking sodium as the driver of fluid accumulation

While the sheer volume of fluid administered in the ICU often gets the blame for fluid overload, excessive sodium intake may play an even more important role. From a physiological standpoint, the kidneys regulate water balance efficiently and rapidly, yet respond surprisingly slowly to changes in sodium intake. Classic experiments demonstrated that, in healthy individuals, increasing sodium intake from 0.5 g/day to 3.2 g/day (a common amount in many parts of the world) required roughly five days for the kidneys to re-establish sodium balance, a relatively slow adaptive process despite the kidneys’ high filtration capacity [8]. During this period, fluid retention led to a weight gain of over 1 kg. Once the sodium load was reduced, shedding this weight took just as long. It is readily apparent that even isotonic fluids— including balanced alternatives to NaCl 0.9%—can exceed a patient’s typical daily sodium intake with no more than one liter [9].

Table 1
Composition of common fluid losses and suggested intravenous replacement fluids [2].

	Sodium (mmol/L)	Chloride (mmol/L)	Potassium (mmol/L)	Suggested IV fluid
Maintenance fluids	40	40	40	Hypotonic balanced crystalloid with potassium (e.g. Maintelyte®) or NaCl 0.18–0.45% with added KCl[a]
Replacement fluids				
Biliary drainage loss	145	105	5	Isotonic balanced crystalloid (e.g Ringer's, PlasmaLyte®)
Jejunal loss via stoma or fistula	140	135	5	NaCl 0,9%
Pancreatic drain or fistula	125–138	56	8	Isotonic balanced crystalloid (e.g Ringer's, PlasmaLyte®)
High volume ileal loss via new or high stoma or fistula	100–140	75–125	4–5	Isotonic balanced crystalloid (e.g Ringer's, PlasmaLyte®)
Low volume ileal loss via established or low stoma or fistula	50–100	25–75	25–75	Hypotonic balanced crystalloid with potassium (e.g. Maintelyte®) or NaCl 0.45% with added KCl
Diarrhea or excess colostomy loss	30–140	20–80	30–70	Hypotonic balanced crystalloid with potassium (e.g. Maintelyte®) or NaCl 0.45% with added KCl
Vomiting / nasogastric tube loss	20–60	140	14	NaCl 0,9% or NaCl 0.45%
Pure water loss (fever, hypernatremia, osmotic diuresis or diabetes insipidus	0	0	0	Glucose or dextrose 4–5%

[a] Commercially available premixed solutions often fall just slightly short on potassium to fully meet guideline recommendations, while 0.45% NaCl with extra KCl contains an excessive amount of sodium and chloride.

Our advice: Because human physiology is adapted to conserve sodium, the kidneys have difficulty excreting excess salt rapidly. Minimizing sodium exposure—from maintenance fluids, replacement fluids, and fluid creep—may be as important as limiting fluid volume, if not more so.

Misinterpreting nonspecific markers as fluid triggers

Administering a fluid bolus is a common and often instinctive intervention in critical care, as it can produce an immediate and observable hemodynamic response. However, such reflexive use should be avoided unless guided by physiological assessment. Relying on single parameters such as lactate, central venous pressure (CVP), urine output, or tachycardia can be misleading, as none of these reliably indicate true hypovolemia. Each should be interpreted within a broader hemodynamic and physiological assessment rather than used as a trigger for fluid administration. Recognizing the limitations of these markers is key to preventing unnecessary fluid overload, as outlined in the four guiding tenets below.

- Hyperlactatemia

Increased lactate levels are often interpreted as a marker of hypoperfusion, yet hyperlactatemia is multifactorial and does not always reflect poor perfusion and thus the need for fluids [10]. The ANDROMEDA-SHOCK trial showed that targeting peripheral perfusion (via capillary refill time) led to 400 mL less fluid administration over 8 h compared to lactate-guided therapy, with no worse outcome [11]. Other reviews have emphasized that elevated lactate levels in sepsis can result from diverse mechanisms—including altered cellular metabolism, adrenergic stimulation, or impaired clearance—many of which are unrelated to tissue hypoxia or hemodynamic compromise [12,13]. A more informative approach may be to interpret lactate levels alongside flow-sensitive parameters such as $ScvO_2$, pCO_2 gap, and clinical assessment of peripheral perfusion. Although evidence remains limited, the pCO_2 gap has been suggested as a potentially useful indicator of low cardiac output in this context. A pCO_2 gap > 6 mmHg may indicate inadequate flow or low cardiac output, even when $ScvO_2$ exceeds 70%. However, this finding should always be interpreted in conjunction with clinical signs of hypoperfusion and the broader hemodynamic context before considering therapeutic interventions such as fluid administration [14–17].

- Low urine output

Oliguria is often interpreted as a sign of hypovolemia, prompting fluid administration. However, the concept of *acute renal success*, introduced by Thurau and Boylan in 1976 challenges this oversimplified assumption [18]. While oliguria may sometimes reflect true hypovolemia or impaired perfusion, it can also occur despite preserved hemodynamics—for example, due to excess ADH secretion without a volume or osmolar stimulus—or as a physiological, protective response aimed at conserving water and sodium and allowing renal recovery. Most oliguric episodes are not followed by significant increases in creatinine, highlighting the limited specificity and predictive value of oliguria in ICU patients [19] Interpretation should therefore always consider the hemodynamic context. Forcing fluids solely to increase urine output may disrupt this adaptive process, exacerbate congestion, and impair recovery. The RESPONSE trial showed that a fluid bolus led to only a modest rise in urine output and shorter oliguria, but at the cost of a significantly more positive fluid balance—highlighting that low urine output alone should not prompt fluid administration without clinical context [20].

- Tachycardia

Tachycardia is often seen as a marker of hypovolemia, leading clinicians to administer fluids in hopes of slowing the heart rate [21,22]. However, isolated tachycardia has limited diagnostic utility across various shock states [23]. Furthermore, evidence suggests that heart rate changes post-fluid bolus are minimal and unreliable [24,25]. Tachycardia can result from a range of factors including pain, fever, or adrenergic stress, none of which are corrected by volume expansion alone.

- Central venous pressure

Modern guidelines have moved away from using CVP thresholds for resuscitation decisions, recognizing that CVP alone does not accurately reflect preload or predict fluid responsiveness. Rather than reflecting true intravascular volume, CVP more accurately represents the interplay between compartment pressures (abdominal and thoracal pressures), venous tone and right heart function [26,27]. Its primary utility now is as a safety parameter to avoid fluid overload, not as a resuscitation trigger [28]. A better understanding of its physiologic significance—as a dynamic reflection of cardiac-venous interactions—could help restore its appropriate role in identifying fluid intolerance, as demonstrated in the next section.

Our advice: Treat lactate levels, urine output, heart rate and CVP as clues, not cues for fluid boluses.

Neglecting fluid tolerance while focusing on fluid responsiveness

In hemodynamic resuscitation, the concept of fluid responsiveness (FR) or the ability of the heart to increase stroke volume in response to

Table 2
Framework for guiding fluid therapy based on fluid responsiveness (FR) combined with fluid tolerance (FT).

	Fluid responsive	Fluid unresponsive
Fluid tolerant	Profile A - Fluid administration is beneficial with low risk.	Profile B: - Fluids unlikely to improve hemodynamics - Conservative management advised to avoid FT erosion
Fluid intolerant	Profile D - Despite FR, fluids pose high risk. Consider additional hemodynamic monitoring, smaller crystalloid boluses or even hyperoncotic albumin - Prioritize vasoactive support over additional volume loading	Profile C - Fluids should be avoided - Fluid removal strategies may be beneficial

fluid administration, has long dominated clinical decision-making [29]. However, an equally important and often overlooked concept is fluid tolerance (FT), defined as the body's ability to receive fluids without progressing to organ dysfunction [30]. While many clinicians remain focused on markers of forward flow, such as cardiac output and other microcirculatory parameters, insufficient attention is given to venous congestion, microcirculatory flow, and their relationship to organ function [31].

Critically ill patients may be fluid responsive but not fluid tolerant, meaning that while fluid administration increases cardiac output, it simultaneously induces or exacerbates organ dysfunction through venous congestion. This disconnect underscores the need for a combined evaluation of both FR and FT. As Melo et al. emphasize, even when FR is present, the absence of FT can neutralize the benefits of volume expansion, or worse, trigger new complications [32].

From this combined assessment, four hemodynamic profiles emerge, each requiring a tailored approach as depicted in Table 2. Understanding and applying these profiles can help individualize fluid therapy and prevent or reverse organ dysfunction in hemodynamically unstable patients.

Evaluating fluid intolerance requires identifying signs of organ congestion and congestion-prone states across multiple organ systems. This includes assessment of the heart, lungs, abdomen, kidney/venous compartment, and peripheral tissues using both clinical indicators and point-of-care ultrasound (POCUS). Fig. 2 summarizes commonly used indicators that may suggest developing fluid intolerance.

Point-of-care ultrasound is increasingly available in the ICU and offers valuable insights into both left and right-sided FT. Lung ultrasound is gaining widespread use for assessing pulmonary congestion by detecting B-lines and pleural effusions [33]. For right-sided evaluations, the Venous Excess Ultrasound Score (VExUS) is a dynamic and integrative tool that combines measurements of the inferior vena cava diameter with Doppler flow patterns in the portal, hepatic, and intrarenal veins [34]. VExUS has demonstrated strong correlation with renal dysfunction, particularly in post-cardiac surgery patients [35], and is proving valuable in guiding fluid removal in dialysis patients and resuscitation strategies in the critically ill and in heart failure patients with cardiorenal syndrome [34–37]. As such, the quantification of organ congestion using venous

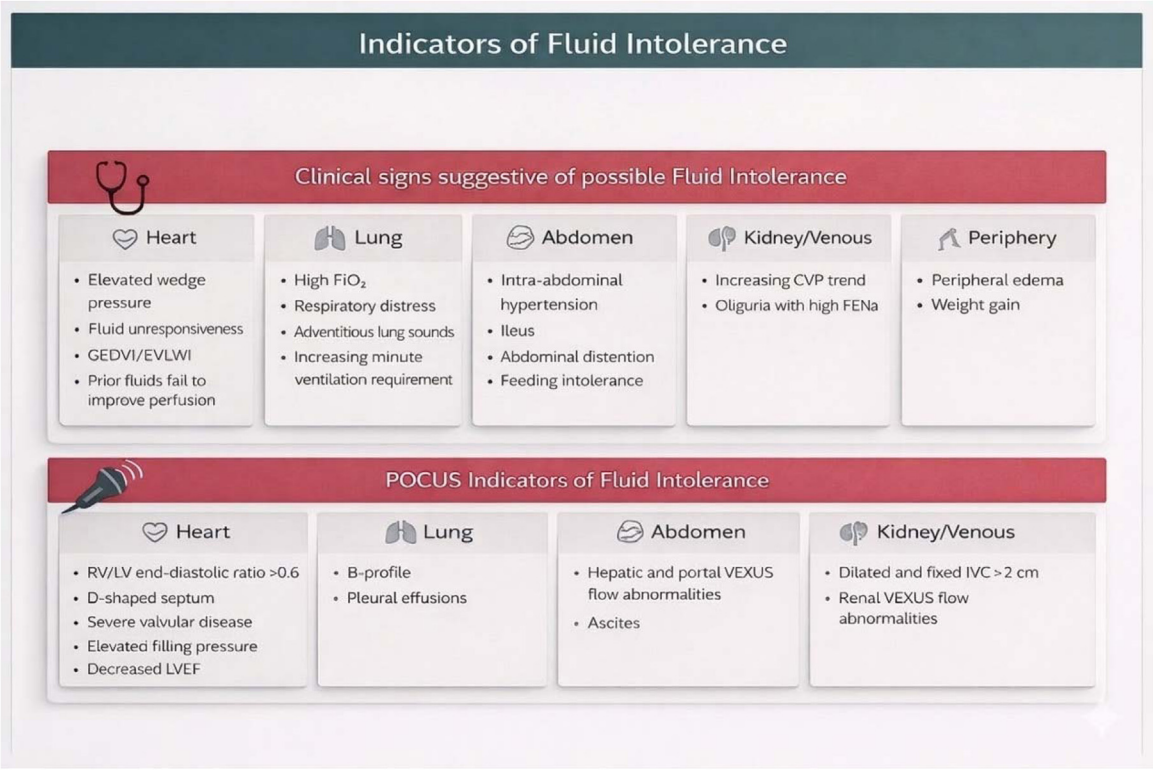

Fig. 2. Common examples of clinical and point-of-care ultrasound (POCUS) findings that may suggest reduced tolerance to further fluid administration, grouped by organ system. Findings should be interpreted in the overall clinical and hemodynamic context. RV: Right ventricle; LV: Left ventricle; LVEF: Left ventricular ejection fraction; LA: Left atrium; FENa: fractional excretion of sodium; GEDVI global end diastolic volume index (PiCCO-device) EVLWI: extravascular lung water index; IVC: inferior vena cava; VExUS: Venous Excess Ultrasound Score; CVP: central venous pressure.

Doppler is becoming increasingly more relevant for assessing fluid tolerance [38].

Our advice: Assess fluid tolerance in conjunction with fluid responsiveness and incorporate both into bedside decision-making about fluid administration.

From excess to precision: five ways to do it better

Let the clinical phase of shock guide the fluid—apply the ROSE model

Like any drug, fluid therapy requires careful consideration of dose, timing, and context [39]. The ROSE model—Resuscitation, Optimization, Stabilization, and Evacuation—offers a structured, phase-based approach to fluid therapy, allowing clinicians to align fluid strategies with the evolving physiology of critical illness [40]. Recognizing a patient's position along this continuum is essential for guiding safe and effective fluid therapy.

For a detailed description, we refer to the original ROSE model publication [39]. Briefly, the Resuscitation phase occurs in the first minutes after shock diagnosis and involves rapid, often empiric administration of small fluid boluses, typically not exceeding 4 mL/kg (or 250–500 mL) at a time. While such boluses may not be sufficient in all patients and may need to be repeated depending on clinical response, this incremental approach may ultimately result in lower total fluid administration during the early resuscitation phase compared with fixed-dose strategies such as the commonly cited 30 mL/kg initial target in septic shock. If shock is not reversed, management transitions to the Optimization phase, where fluid therapy is tailored using dynamic measures (e.g., Passive Leg Raise Test, End Expiratory Occlusion Test, PPV/SVV). Vasopressors are initiated if needed (see the next section). In the Stabilization phase, once hemodynamic stability is achieved, fluids are de-escalated to maintenance or replacement as needed, with reassessment of fluid balance, body weight, kidney function (including sodium avidity), and fluid tolerance. The Evacuation phase focuses on active fluid removal, ideally after demonstrating fluid unresponsiveness.

Our advice: The ROSE concept encourages clinicians to approach fluid therapy with care and balance, nurturing recovery by tailoring interventions to each phase of illness, rather than overwhelming the system through excess.

Treat vasoplegia with vasopressors, not with more fluids

Vasoplegic shock represents a state of profound circulatory collapse characterized not by pure volume loss, but by pathological vasodilation and capillary leakage due to endothelial dysfunction [41]. When the vascular endothelium is compromised, it becomes more permeable, allowing fluid to escape into the interstitial space. The result is tissue oedema, which can paradoxically coexist with intravascular hypovolemia. In this context, further liberal fluid administration may aggravate interstitial congestion without restoring effective circulatory pressure. Emerging evidence suggests that early initiation of vasopressors, particularly norepinephrine, may curb this fluid mismanagement. The CENSER trial, a landmark study by Permpikul et al., demonstrated that administering norepinephrine within the first hour of shock significantly improved shock control rates by six hours [42]. Importantly, low-dose, dilute norepinephrine was safely administered through a peripheral line without adverse effects, supporting the notion that vasopressor therapy should not be delayed while awaiting central access. More recently, a large study [43] further reinforced the safety of peripheral norepinephrine, even at standard concentrations (16 mg/250 mL), with a low incidence of clinically significant extravasation. Furthermore, a contemporary meta-analysis [44] reported a possible association between early vasopressor initiation and decreased fluid volume administered at 6 h, reinforcing the potential benefit of this strategy.

Additionally, in a propensity-matched analysis of 337 patients, early norepinephrine not only reduced cumulative fluid balance but also appeared to augment the effects of fluid boluses by recruiting stressed volume [45]. Adda et al. further supported this by showing that early vasopressor use rapidly increases mean systemic filling pressure, enhancing cardiac output in volume-responsive patients with less fluid administration [46].

The optimal timing for vasopressor initiation remains uncertain, but diastolic blood pressure (DBP) may serve as a pragmatic bedside indicator of vasoplegia. Holder et al. found that lower DBP values independently predicted progression to full-blown septic shock [47]. Thus, DBP values below 50–55 mmHg could serve as an early signal to clamp down on leaky pipes with norepinephrine, rather than reflexively reaching for another fluid bolus. Findings from ANDROMEDA-2 [48] further support this approach, as a DBP-targeted challenge was associated with improvement or normalization of capillary refill time. However, DBP must be interpreted in clinical context, taking into account heart rate, perfusion markers, and the persistent uncertainty regarding the ideal moment to initiate vasopressor therapy.

While evidence is still evolving, several studies suggest that non-adrenergic vasopressors may complement adrenergic agents within a multimodal vasopressor approach. A recent meta-analysis found a possible association between their use and reduced mortality in septic shock. Although the results also suggested potential benefit—or at least no measurable harm—in cardiac and non-cardiac surgical populations, high-quality trials are needed to confirm these observations [49].

Nevertheless, early vasopressor initiation is not without potential drawbacks. Critics argue that correcting hypotension with vasopressors may mask underlying hypovolemia, creating a false sense of hemodynamic stability while tissue perfusion remains inadequate [50]. Moreover, increasing vascular tone in the absence of sufficient preload could paradoxically worsen tissue hypoperfusion, potentially resulting in digital or mesenteric non-occlusive ischemia. In patients with marginal cardiac output or underlying peripheral arterial disease. Let's not forget that pressure does not equal perfusion.

Our advice: Don't hesitate to start vasopressors early – even peripherally; effective distributive shock management lies in striking the right balance between volume expansion and vascular tone. However, decisions should be individualized to balance the need for volume expansion with the restoration of vascular tone.

Slash the sodium, curb the chloride and carefully consider albumin

Emerging data indicate that limiting sodium exposure, where clinically appropriate, could help improve fluid management and patient outcomes. From healthy volunteers in the MIHMoSA experiment to patients undergoing major surgery in the TOPMAST trial, hypotonic maintenance fluids have consistently been shown to cause significantly less fluid and sodium accumulation than their sodium-rich alternatives [51–53]. The risk of clinically relevant hyponatremia is negligible [54]. In ICU patients with septic shock, the DEMANDS trial demonstrated that dextrose 5% resulted in a significantly lower cumulative fluid balance—2.06 L less—compared to 0.9% NaCl, and even showed a potential mortality benefit [55]. The upcoming CReep and maintenance flUid Salt ADministration rEduction in cRitically ill adultS (CRUSADERS) trial in critically ill patients (EuCT 2025–520744-14–00) is designed to provide further clarity in critically ill patients. Of note, this sodium-restrictive strategy should not be extrapolated to patients with intracranial hypertension, traumatic brain injury, or acute hyponatremia, in whom hypotonic fluids may exacerbate cerebral edema or worsen neurologic outcomes;

in these populations, avoidance of hypotonic solutions remains standard practice.

Excessive chloride exposure also warrants cautious attention, as it has been associated with hyperchloremic metabolic acidosis, impaired renal perfusion, and an increased risk of acute kidney injury [56,57]. Hyperchloremia promotes afferent arteriolar vasoconstriction, which reduces glomerular filtration and impairs sodium and water excretion [58]. As a consequence, NaCl 0.9% leads to more fluid retention than balanced solutions [59]. In the debate between balanced and unbalanced fluids, it is often overlooked that far more chloride can be spared by switching from isotonic to hypotonic solutions for maintenance, replacement (when appropriate), and fluid creep than by switching from NaCl 0.9% to balanced solutions for resuscitation—because the chloride difference is smaller in resuscitation fluids, and resuscitation represents only a small fraction of total fluid administration (Fig. 3). In a large retrospective study of 14,654 patients, Van Regenmortel et al. showed that using unbalanced instead of balanced resuscitation fluids would have added just 3.0 mmol of chloride daily, while replacing hypotonic maintenance fluids with isotonic ones would have added 30.8 mmol/day [6]. According to Magee et al., replacing saline with dextrose 5% as the primary medication diluent reduced the incidence of hyperchloremia and was associated with a lower rate of acute kidney injury, highlighting the potential harm of chloride from fluid creep in critically ill patients [60].

In parallel, the role of colloids such as albumin continues to be debated. Although significantly more expensive than crystalloids, albumin may offer benefits in selected scenarios [61]. Hypoalbuminemia in critical illness typically results from redistribution rather than loss or impaired synthesis. Preclinical evidence suggests that albumin may help stabilize the endothelial glycocalyx, thereby reducing capillary leakage [62]. While current SCCM guidelines do not recommend its use during initial resuscitation in sepsis, albumin may be beneficial in later phases, when large amounts of crystalloids have already been administered. In septic shock, albumin has been associated with lower net fluid balance and possibly improved outcomes [63–65]. In the Surviving Sepsis Campaign, this led to cautious support for the use of albumin in patients with sepsis or septic shock who had already received large volumes of crystalloids [66]. The soon-to-be-published ALBIOSS-BALANCED Trial (NCT03654001) aims to provide further clarity. Although albumin has proven benefits in specific conditions, there is need for continued scrutiny of albumin use in different clinical contexts [67]

Our advice: Efforts to reduce sodium and chloride exposure should extend beyond resuscitation fluids to include consideration of hypotonic solutions for maintenance, replacement, and medication dilution when suitable. In selected cases and awaiting further evidence, thoughtful use of albumin may enhance certain aspects of fluid therapy.

Use fluid balance as a guide, not a goal

Fluid balance may appear neat on charts, but in practice, ICU measurements are often unreliable. Nurse-recorded cumulative balances frequently fail to match body weight trends [68], and insensible losses—such as those from respiration and the skin—are nearly impossible to quantify [69]. Capturing pre-admission fluid shifts is challenging: patients may present with a falsely reassuring zero or negative fluid balance in the ICU simply because they are excreting a pre-admission fluid load. Conversely, in cases of dehydration, fluid depletion, or ongoing extensive skin losses, a positive fluid balance may be appropriate—and even a therapeutic goal—further limiting its reliability as a marker of capillary leakage, third-spacing, or tissue redistribution. Aiming for 'zero balance' without accounting for these limitations may be misleading. The RELIEF trial demonstrated that rigid fluid restriction increased the risk of acute kidney injury without improving disability-free survival in major abdominal surgery [70]. Daily weighing remains a pragmatic and high-yield measure for tracking fluid accumulation and guiding individualized fluid management decisions.

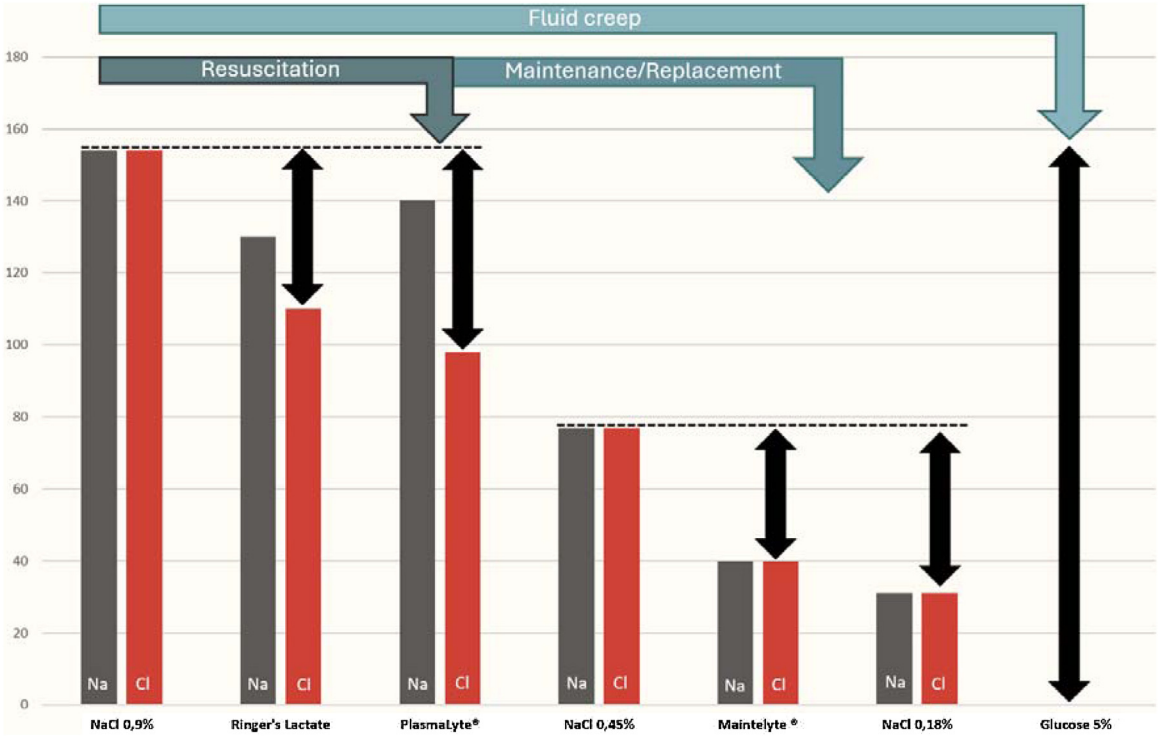

Fig. 3. Sodium and chloride content (mmol/L) of commonly used fluids and the projected impact of switching to sodium- and chloride-poor alternatives for resuscitation, maintenance, and fluid creep prescriptions.

Our advice: Don't use fluid balance as a treatment goal in itself and interpret it alongside body weight trends, clinical assessment of insensible losses, and disease-specific context.

Furosemide can't easily reverse poor fluid choices

Neglecting sodium and chloride minimization during fluid therapy sets the stage for diuretic-induced hypernatremia, since loop diuretics produce hypotonic urine, which worsen sodium imbalances from earlier mismanagement. Hypernatremia is an often preventable complication linked to increased mortality, prolonged ICU stays, and worse neurological outcomes [71]. Managing fluid removal requires more than diuresis, it demands a strategic approach to electrolyte handling.

While loop diuretics can achieve a negative fluid balance by inhibiting the $Na^+/K^+/2Cl^-$ transporter in the thick ascending limb and promoting diuresis, it cannot effectively compensate for earlier excessive sodium and chloride resuscitation [72]. In the setting of established sodium overload, isolated fluid removal is often insufficient. A desalination strategy aimed at eliminating both sodium and water is preferable. This demands a combined diuretic strategy, as the synergistic effect of agents like acetazolamide and thiazides with loop diuretics produces a more isotonic diuresis—a 'balanced natriuresis'—that is crucial for achieving a negative sodium balance [73–76] This strategic pairing is necessary because furosemide alone, by primarily removing hypotonic fluid, is poor at correcting the initial sodium and chloride imbalance [75,76].

It is frequently overlooked that sodium excretion is more effective in well-hydrated patients with an adequate urine flow [77]. Given that urine sodium concentration typically does not exceed twice the plasma concentration, enhancing urine output through improved hydration—alone or in combination with diuretics—will often facilitate sodium removal more effectively than restricting water intake. Even the best diuretic strategy often needs a sidekick: electrolyte-free water.

Our advice: When fluid accumulation cannot be prevented, understand that loop diuretics alone are inadequate to reverse the damage. A more physiological approach might be to address the established sodium overload with combination diuretic therapy and free water replacement.

Conclusion

Fluids are medications and we need to treat them as such. We should be considering fluid prescriptions with the amount of mental energy that we use for any other medication. By understanding common pitfalls and applying straightforward principles, we can improve fluid management and provide better patient care

CRediT authorship contribution statement

M.V.E. and N.V.R. contributed to the concept of the review, drafted the manuscript and approved the final manuscript. ChatGPT (OpenAI) was employed solely for linguistic enhancement and to assist with figure creation.

Ethics approval and consent to participate

Not applicable.

Consent for publication

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Declaration of competing interest

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