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Editorial

Fluid Therapy

Fluid Therapy in Critically Ill Patients: From Liberal Resuscitation to Precision Hemodynamic Management - An Update

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Background: Intravenous fluid therapy is among the most frequently administered yet least rigorously prescribed interventions in critical care. Historically guided by tradition and fixed-volume protocols, fluid resuscitation has long been associated with the unintended consequences of fluid overload, including organ dysfunction and increased mortality. A paradigm shift toward individualized, physiology-guided fluid management has emerged over the past two decades, driven by accumulating trial evidence and advances in hemodynamic monitoring.

Objective: To review the evolution of fluid resuscitation practice in critically ill patients, examine landmark trial evidence reshaping current practice, appraise dynamic tools for assessing fluid responsiveness, and outline a precision framework for fluid administration, titration, and de-escalation across the phases of critical illness.

Evidence Review: Major randomized controlled trials and consensus guidelines were reviewed, including the SMART, SALT-ED, PLUS, SAFE, CHEST, SPLIT, and CLASSIC trials, alongside Surviving Sepsis Campaign and European Society of Intensive Care Medicine recommendations. The physiological basis of fluid responsiveness, the limitations of static preload markers, and the clinical utility of dynamic indices, including passive leg raising, pulse pressure variation, stroke volume variation, and point-of-care echocardiography, are discussed. Conceptual frameworks including the Four D's of fluid therapy and the ROSE model are appraised as structured approaches to individualized management.

Results: Balanced crystalloids are associated with reduced rates of acute kidney injury and renal replacement therapy compared with normal saline, though the magnitude of benefit varies across populations. Hydroxyethyl starches are definitively contraindicated in critical illness. Albumin demonstrates equivalence to saline in heterogeneous ICU populations with selective benefit in septic and cirrhotic physiology. Fluid overload is established as an independent driver of pulmonary, renal, and microcirculatory injury. The CLASSIC trial supports the safety of a restrictive fluid strategy beyond the initial resuscitation phase. Dynamic indices of fluid responsiveness consistently outperform static preload markers in predicting hemodynamic benefit from fluid administration.

Conclusions: Fluid therapy in critical illness must be approached with the same rigor applied to any pharmacological intervention, with explicit indications, individualized dosing, active reassessment, and planned de-escalation. No protocol or biomarker substitutes for bedside clinical judgement informed by dynamic hemodynamic assessment. Emerging technologies including artificial intelligence-assisted monitoring and closed-loop systems hold promise for further personalization, but the central task of modern intensive care remains physiological titration rather than volume maximization or minimization.

Keywords: fluid resuscitation, fluid responsiveness, fluid overload, balanced crystalloids, hemodynamic monitoring, septic shock, critical care

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Few interventions in critical care medicine are administered as frequently, and scrutinized as inadequately, as intravenous fluid therapy. Almost every patient who passes through an intensive care unit receives fluids at some point in their trajectory, yet for decades the decision of how much, what type, and for how long was guided more by tradition and reflex than by physiological reasoning. Fluids have long occupied an ambiguous space in critical care: simultaneously life-saving and potentially harmful, indispensable in shock yet implicated in organ dysfunction when given indiscriminately. The growing recognition that fluid administration carries a dose-dependent risk profile, much like any pharmacological agent, has catalyzed a paradigm shift away from liberal, protocol-driven resuscitation toward individualized, physiology-guided, and increasingly technology-assisted fluid management. This editorial examines that evolution, situates it within the landmark trial evidence that has reshaped practice, and argues that guidelines inform but do not replace bedside judgement: the central task of modern intensive care is not simply to resuscitate, but to titrate.

The history of fluid resuscitation is, in many respects, a history of overcorrection. Early twentieth-century shock physiology emphasized the restoration of circulating volume as the primary therapeutic goal, and this principle was reinforced by battlefield and trauma experience through the mid-century, which demonstrated unequivocally that exsanguinating patients died without volume replacement. This foundational truth, however, was gradually generalized beyond its original context. By the late twentieth century, "more fluid is better" had become an unspoken axiom of critical care, embedded in early goal-directed therapy protocols that mandated fixed volumes of crystalloid within rigid time windows.

The Surviving Sepsis Campaign's early iterations, while instrumental in standardizing sepsis recognition and reducing time to treatment, inadvertently entrenched a culture of aggressive, often unmonitored fluid boluses,(1) and a positive fluid balance accrued in this era is best understood as a physiological signal requiring reassessment rather than a therapeutic endpoint in itself.(2) It took nearly two decades of accumulating observational and trial data on fluid overload, capillary leak, and organ congestion before the pendulum began to swing back toward restraint. Current Surviving Sepsis Campaign recommendations now favor initial crystalloid administration in selected patients with septic shock, followed by frequent reassessment of responsiveness, rather than the fixed-volume boluses of earlier protocols.(1)

The physiological rationale for fluid administration remains, at its core, the optimization of cardiac preload to augment stroke volume and oxygen delivery to tissues, in accordance with the Frank-Starling relationship. The fundamental error of historical practice was conflating volume status with fluid responsiveness. A patient may be intravascularly replete, or even overloaded, while still appearing clinically "dry" on traditional static indices such as central venous pressure or jugular venous distension; conversely, a hypovolemic patient on the steep portion of the Frank-Starling curve will respond to fluid with a meaningful rise in stroke volume, while one on the flat portion will not, regardless of apparent volume deficit. Central venous pressure alone should not be relied upon to predict fluid responsiveness, a limitation now well established across multiple meta-analyses.(3) Fluid responsiveness is therefore not a fixed patient characteristic but a dynamic physiological state dependent on ventricular function, vascular tone, and the position of the heart on its preload-dependent curve at a given moment, and a patient's fluid responsiveness today does not guarantee fluid responsiveness tomorrow.(4) Hypotension alone should not automatically trigger fluid administration, since tachycardia, oliguria, and low blood pressure are nonspecific signs that may equally reflect vasoplegia, cardiac dysfunction, or simple pain and agitation. Administering fluid to a non-responsive heart accomplishes nothing therapeutically while still incurring the full burden of volume-related harm, a distinction that lies at the heart of the modern precision approach; indeed, the absence of fluid responsiveness is often more informative than its presence, since it redirects the clinician toward vasopressor support or a search for an alternative diagnosis. Individual patient physiology remains the ultimate determinant of therapy, and no biomarker or formula can substitute for this individualized assessment.

To operationalize this distinction, clinicians have increasingly adopted the conceptual framework of the *Four D's of fluid therapy: Drug, Dose, Duration, and De-escalation.*(5)

Conceiving of fluid as a drug compels the clinician to define an indication before infusion, just as one would before prescribing an antibiotic or vasopressor. The decision to withhold fluid can be as therapeutically important as the decision to administer it. The dose must be individualized rather than weight-based or protocolized, the duration of therapy must be actively reassessed rather than open-ended, and de-escalation, including active fluid removal, must be planned as deliberately as initiation. Fluid therapy should be individualized rather than protocolized, a principle that this framework operationalizes at the bedside. This framework dovetails with the ROSE model of fluid management, which delineates four temporally distinct phases of critical illness: Resuscitation, in which fluid boluses address life-threatening hypoperfusion; Optimization, in which fluid administration is titrated against dynamic markers of responsiveness to fine-tune oxygen delivery; Stabilization, in which fluid balance is maintained near neutral while organ support continues; and Evacuation, in which active de-resuscitation, often pharmacological diuresis or renal replacement therapy, is employed to correct the cumulative positive fluid balance accrued during earlier phases.(6) The ROSE model reframes fluid therapy as a continuum requiring a different therapeutic posture at each stage, rather than a single undifferentiated intervention applied uniformly throughout the ICU stay.

The choice of fluid type has itself undergone substantial reappraisal. Normal saline, long the default crystalloid by virtue of historical inertia rather than physiological superiority, has been increasingly displaced by balanced crystalloid solutions whose electrolyte composition more closely approximates plasma, thereby avoiding the hyperchloremic metabolic acidosis and renal vasoconstriction associated with large-volume saline administration. The **SMART** trial demonstrated that balanced crystalloids reduced the composite outcome of death, new renal replacement therapy, or persistent renal dysfunction among critically ill patients compared with saline,(7) while the **SALT-ED** trial extended a similar, albeit more modest, signal to non-critically ill emergency department patients.(8) The subsequent **PLUS** study, a large multicenter trial comparing Plasma-Lyte (148) with saline in ICU patients, did not demonstrate a significant difference in ninety-day mortality, illustrating that the magnitude of benefit from balanced solutions may be more modest, or more population-dependent, than initially suggested, and that this question remains incompletely resolved.(9) Balanced crystalloids may be considered the preferred first-line resuscitation fluid in many critically ill patients, although patient-specific factors remain paramount. Colloid therapy has followed its own trajectory of disillusionment. The SAFE study established that albumin and saline produced equivalent outcomes in heterogeneous ICU populations, with a signal of harm in traumatic brain injury, tempering enthusiasm for albumin as a universal resuscitation fluid while leaving open more selective indications such as cirrhotic and septic shock physiology.(10) Hydroxyethyl starches, once attractive for their theoretical plasma-expanding efficiency, have been definitively abandoned in critical illness following the CHEST(11) and SPLIT(12) trials and earlier European data(13) demonstrating increased rates of acute kidney injury and renal replacement therapy without offsetting survival benefit, a body of evidence so consistent that regulatory agencies in multiple jurisdictions have restricted starch use in critically ill and septic patients.

Perhaps the most consequential evidentiary contribution of the past decade has been the demonstration that fluid overload itself constitutes an independent driver of morbidity and mortality, rather than a benign epiphenomenon of severe illness.(2,14) Excess interstitial fluid disrupts the glycocalyx, worsens tissue edema, impairs microcirculatory flow paradoxically by raising interstitial pressure, and contributes mechanically to abdominal compartment syndrome, impaired lymphatic drainage, and delayed wound healing. Pulmonary consequences are particularly prominent, with positive fluid balance strongly associated with prolonged mechanical ventilation, acute respiratory distress syndrome, and failure to wean. Renally, venous congestion from fluid overload is now understood to impair glomerular filtration through elevated renal venous pressure, compounding rather than ameliorating acute kidney injury when fluid is given beyond the point of physiological benefit;(15) indeed, venous congestion can contribute to organ dysfunction even when arterial perfusion appears adequate, a distinction easily missed when blood pressure alone is used as the marker of adequate resuscitation.

The **CLASSIC** trial, which randomized septic shock patients to restrictive versus standard fluid strategies after initial resuscitation, found no significant difference in mortality but reinforced the safety of a more conservative approach beyond the initial resuscitation phase,(16,17) adding to a now substantial body of evidence that "less may be at least as good, and possibly better" once the resuscitation phase has concluded. De-resuscitation should be contemplated once hemodynamic stability has been achieved and ongoing fluid accumulation no longer provides physiological benefit, though diuresis or fluid removal should be guided by ongoing hemodynamic tolerance rather than by a fixed fluid balance target.

These risks have driven the adoption of dynamic, rather than static, indices of fluid responsiveness at the bedside. Dynamic indices should generally be preferred over static preload markers when evaluating fluid responsiveness. Passive leg raising, which transiently auto-transfuses approximately three hundred milliliters of venous blood toward the central circulation, functions as a reversible "fluid challenge" without the risk of fluid accumulation, and changes in cardiac output or stroke volume following this maneuver predict true fluid responsiveness with considerably greater accuracy than central venous pressure ever achieved;(3,18) passive leg raising provides this assessment without committing the patient to a non-retractable volume load, a property that distinguishes it from a conventional fluid bolus. In mechanically ventilated patients without arrhythmia, pulse pressure variation and stroke volume variation exploit the cyclical changes in venous return induced by positive-pressure ventilation, with variation thresholds reliably distinguishing responders from non-responders, though their utility diminishes in spontaneously breathing patients, those with low tidal volumes, or right ventricular dysfunction.(4) Point-of-care and transesophageal echocardiography have become indispensable adjuncts, allowing direct visualization of ventricular filling, contractility, inferior vena cava distensibility, and the velocity-time integral response to fluid or passive leg raising, while simultaneously excluding occult cardiac dysfunction that would otherwise be missed by hemodynamic numbers alone.(15) A fluid challenge, whichever modality is used to assess it, should always be paired with a predefined safety limit and a clear physiological endpoint,(19) since administering fluid without a defined stopping rule risks progressive accumulation with diminishing returns.

The convergence of these tools has enabled a genuinely individualized, precision-medicine approach to fluid therapy, one in which the decision to administer fluid is made only when a dynamic test predicts a meaningful hemodynamic response, and even then is bounded by predefined safety limits and a clear endpoint.(20) Equally important is the recognition that fluids and vasopressors are not sequential but parallel therapeutic levers; early, judicious use of norepinephrine to restore vascular tone may reduce cumulative fluid exposure without compromising perfusion, a strategy increasingly termed fluid stewardship in analogy to antimicrobial stewardship.(5) Current Surviving Sepsis Campaign guidance reflects this shift, recommending a more cautious, reassessment-driven approach to initial fluid boluses than earlier iterations, with explicit encouragement of dynamic measures over static ones,(1) while European Society of Intensive Care Medicine consensus statements have gone further in articulating de-resuscitation as a formal component of ICU care, advocating active fluid removal once hemodynamic stability permits.(15) The decision to administer, withhold, or remove fluid should be guided by the patient's evolving hemodynamic profile rather than by a fixed protocol or an arbitrary cumulative volume target. Looking forward, the integration of artificial intelligence and machine learning into hemodynamic monitoring platforms holds promise for moving beyond reactive, clinician-triggered fluid challenges toward predictive models capable of forecasting fluid responsiveness from continuous waveform data before a challenge is even performed. Closed-loop and decision-support systems that integrate arterial waveform analysis, echocardiographic parameters, and biomarkers of renal and pulmonary congestion may ultimately permit truly personalized resuscitation algorithms, tailored not merely to a diagnosis but to a specific patient's hemodynamic phenotype at a specific moment in their illness trajectory. Realizing this potential will require prospective validation at a scale comparable to the landmark trials that reshaped crystalloid practice, but the trajectory of field, from empirical bolusing toward algorithmic, physiology-anchored titration, is unmistakable; even most sophisticated algorithm, however, will refine rather than replace clinical reasoning at the bedside.

The cumulative weight of three decades of trial evidence and physiological research converges on a single, unifying message: fluids are drugs, and like all drugs they possess indications, contraindications, dose-response relationships, and adverse effect profiles that demand the same rigor in prescribing as any vasopressor, antibiotic, or sedative. The question facing the modern intensivist is no longer how much fluid a patient requires in absolute terms, but whether this patient, at this moment, in this physiological state, will benefit from this volume of this fluid, and whether a clear plan exists for de-escalation once the therapeutic window has closed. Optimal outcomes in critical illness will not be achieved by maximizing or minimizing fluid volume as a matter of dogma, but by consistently asking, and rigorously answering, whether the right fluid is being given to the right patient, at the right time, and in the right amount. Guidelines, landmark trials, and emerging technologies all serve this single end: they refine the question, but it is the clinician, at the bedside, who must still answer it.

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