

Perioperative Fluid Therapy: From Restrictive/Liberal Labels to Physiology-Guided Individualization

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ABSTRACT

Perioperative fluid therapy has evolved from fixed replacement of fasting and presumed third-space losses toward restrictive, zero-balance, and increasingly individualized strategies. The therapeutic window is narrow: inadequate circulating volume may impair organ perfusion and contribute to acute kidney injury (AKI), whereas excessive fluid can cause interstitial oedema, pulmonary complications, gastrointestinal dysfunction, and delayed recovery. Enhanced Recovery After Surgery (ERAS) pathways have reduced unnecessary intravenous fluid administration, but the optimal balance remains uncertain. The RELIEF randomized trial found no disability-free survival benefit from a restrictive strategy and reported more postoperative AKI than with liberal therapy. [1] Contemporary PeriOperative Quality Initiative (POQI) recommendations emphasise individualised management according to surgical factors, intravascular volume status, arterial pressure, and comorbidities. [2,3] This narrative review synthesizes evidence on restrictive versus liberal strategies, goal-directed haemodynamic therapy (GDHT), fluid responsiveness, AKI, fluid accumulation, fluid composition, vasopressors, and ERAS integration. The emerging paradigm is not simply restrictive or liberal therapy, but dynamic physiology-guided treatment that seeks adequate organ perfusion while avoiding unnecessary fluid accumulation.

Keywords: perioperative fluid therapy; restrictive fluid therapy; liberal fluid therapy; individualised fluid management; goal-directed haemodynamic therapy; acute kidney injury; ERAS; fluid responsiveness; balanced crystalloids.

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INTRODUCTION

Intravenous fluid is among the most frequently administered perioperative therapies, yet hypotension does not necessarily indicate hypovolaemia. Anaesthetic-induced vasodilation, blood loss, myocardial dysfunction, altered venous return, and mechanical ventilation can produce different haemodynamic phenotypes that may respond very differently to fluid. A patient may therefore be hypotensive and fluid responsive, hypotensive but adequately filled, or hypotensive primarily because of vasodilation.

Historically, fluid therapy was based on calculated fasting deficits, maintenance requirements, urine output, blood loss, evaporation, and presumed third-space losses.

Recognition of fluid-associated oedema led to restrictive and zero-balance approaches, particularly within ERAS.^{4,5}

However, the RELIEF trial challenged excessive restriction by demonstrating more postoperative AKI without an improvement in disability-free survival.¹

The question has therefore shifted from 'How much fluid should every patient receive?' to 'Does this patient need fluid now, and is fluid likely to improve perfusion?' Contemporary POQI guidance reflects this transition.^{2,3}

METHODS

This manuscript is a narrative review and does not

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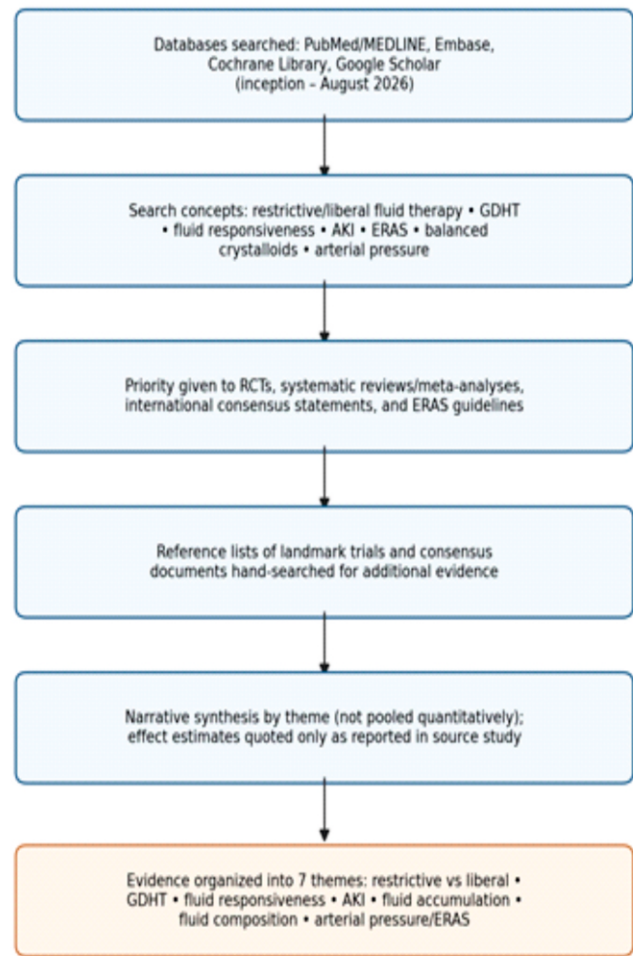
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constitute a systematic review or meta-analysis. No original patient-level data were collected or analyzed. Accordingly, study-selection flow counts, statistical power calculations, formal hypothesis testing, and de novo pooled effect estimates were not performed or reported, as their inclusion would be methodologically inappropriate for a narrative review.

A structured literature search was conducted across PubMed/MEDLINE, Embase, the Cochrane Library, and Google Scholar, covering the period from database inception through August 2026. The search strategy incorporated key concepts related to perioperative fluid therapy, including restrictive and liberal fluid strategies, individualized fluid management, goal-directed haemodynamic therapy, fluid responsiveness, acute kidney injury, enhanced recovery after surgery (ERAS), balanced crystalloids, postoperative complications, and perioperative arterial pressure. Studies were selected according to their relevance to the predefined themes of the review, with emphasis on randomised controlled trials, systematic reviews/meta-analyses, international consensus statements, ERAS guidelines, and influential observational studies. Reference lists of landmark trials and consensus documents were screened for additional relevant evidence. Relevant literature was synthesized narratively to provide a contemporary, clinically focused overview of the evidence, with emphasis on areas of agreement, ongoing controversy, and implications for individualised perioperative fluid management.

Fig. 1 summarises this process as an evidence-selection map.

Statistical considerations: because this is a narrative review without a new quantitative synthesis, conventional sample-size calculation, statistical power analysis, hypothesis testing, pooled confidence intervals, and I² calculations are not applicable. All percentages, p-values, and effect estimates reported below are reproduced from the original studies as published, with the time point of measurement specified wherever a trial reported effects that varied by follow-up interval (see OPTIMISE II, below).



Note: this is a narrative-review evidence map, not a PRISMA systematic-review flow diagram — no screening/exclusion counts are implied.

Fig. 1. Literature search and evidence-selection process. This is a narrative-review evidence map, not a PRISMA systematic-review flow diagram; no screening or exclusion counts are implied.

RESULTS: Evidence Synthesis

The literature demonstrates a shift from fixed-volume prescriptions toward individualized physiological management. The labels restrictive and liberal remain useful for describing trials but are insufficient as individual prescriptions.

. Comparison of restrictive, liberal, and individualized perioperative fluid strategies.

Strategy	Potential benefit	Potential harm	Interpretation
Restrictive	Less cumulative fluid	Hypoperfusion/AKI if excessive	Avoid unnecessary fluid, but restriction should not become the sole target.
Liberal	Protects against untreated hypovolaemia	Oedema, congestion, delayed recovery	Use when genuine deficits/losses justify replacement.
Individualized/GDHT	Targets physiology and response	Requires expertise; evidence heterogeneous	Most physiologically rational for selected higher-risk patients.

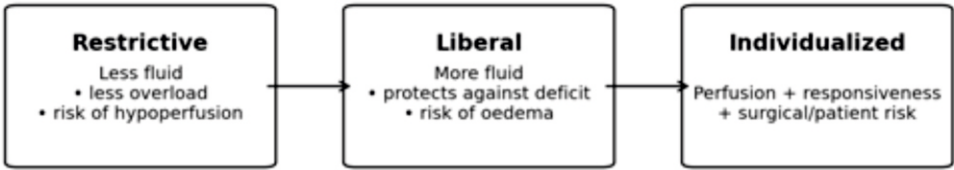


Fig. 2. Evolution of perioperative fluid strategies. Individualised therapy is not simply a midpoint between restrictive and liberal volumes; it is physiology-based.

Restrictive versus liberal therapy

RELIEF randomized 3000 patients undergoing major abdominal surgery to restrictive or liberal intravenous fluid therapy. Restrictive therapy did not improve disability-free survival and was associated with more postoperative AKI.¹ The trial therefore argues against excessive fluid avoidance, but it does not establish liberal loading as a universal strategy because the intervention was a predefined regimen

rather than physiology-guided treatment. Earlier randomized evidence was heterogeneous because restrictive and liberal strategies varied substantially in both volume and implementation. [6] Thus, fluid strategy should always be interpreted in the context of the operation, patient risk, haemodynamic phenotype, and the rest of the perioperative pathway.

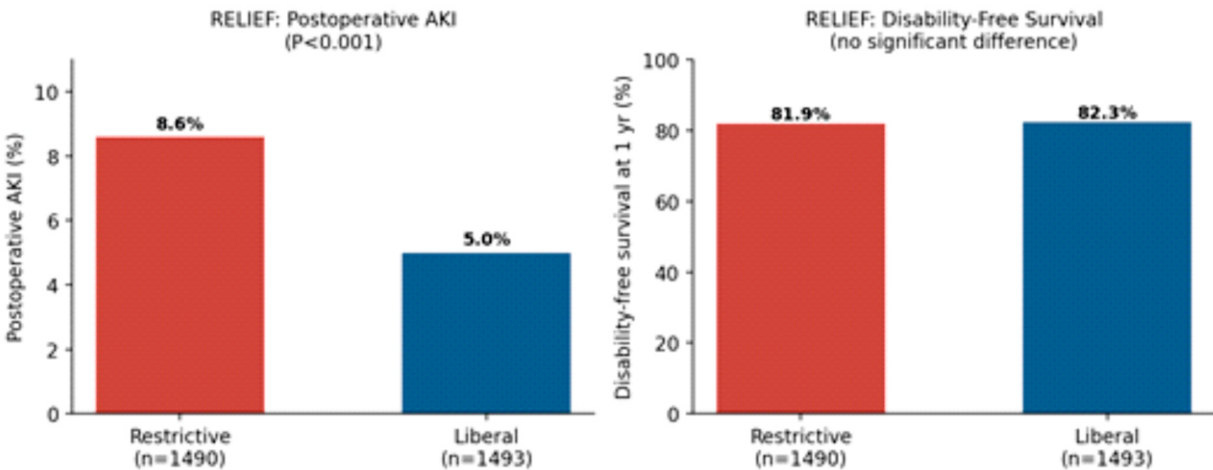


Fig. 3. RELIEF trial: postoperative AKI (8.6% restrictive vs 5.0% liberal, P<0.001) and 1-year disability-free survival (no significant difference). [1]

Goal-directed haemodynamic therapy

GDHT uses cardiac output, stroke volume, dynamic fluid responsiveness, arterial pressure, and sometimes oxygen-delivery targets to guide therapy. OPTIMISE reported fewer complications with cardiac-output-guided therapy in major gastrointestinal surgery.⁷ OPTIMISE II, however, found no reduction in its primary postoperative infection outcome

and reported more acute cardiac events — mainly arrhythmias — in the intervention group at 24 hours (3.0% vs 1.7%, P=0.03); this difference did not persist to 30 days (6.8% vs 6.3%, P=0.71).⁸ These findings demonstrate that a haemodynamic protocol is not automatically beneficial and that algorithm design and patient selection matter.

Table 2. Key statistical findings from landmark evidence.

Evidence	Design/sample	Key reported statistical finding	Clinical interpretation
RELIEF ^[1]	RCT; n=3000	Postoperative AKI: 8.6% restrictive vs 5.0% liberal (P<0.001)	Excessive restriction may increase AKI.
OPTIMISE [7]	RCT; major GI surgery	Original trial reported fewer complications with cardiac -output-guided therapy	Supports selected GDHT.
OPTIMISE II [8]	RCT; n=2498	No reduction in primary infection outcome; more acute cardiac events at 24h (3.0% vs 1.7%, P=0.03), not sustained at 30d (P=0.71)	Routine protocolization is not universally beneficial.
Balanced crystalloids [12,13]	Large pragmatic RCTs	Modest kidney -related differences in some populations	Composition matters, but indication and volume remain fundamental.
GDHT meta-analyses [17–19]	Systematic reviews/meta-analyses	Benefits vary with heterogeneity in populations and algorithms	Avoid overinterpreting pooled rankings.

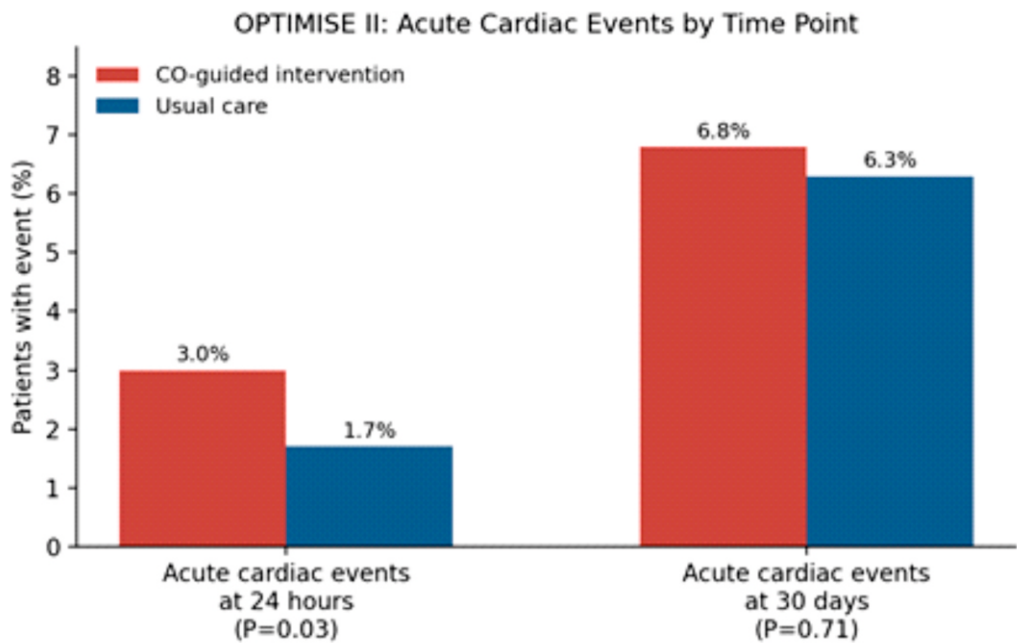


Fig. 4. OPTIMISE II: acute cardiac events (mainly arrhythmias) by time point. The significant difference at 24 hours was not sustained at 30 days. [8]

Fluid responsiveness versus fluid requirement

Fluid responsiveness means that stroke volume can increase after an increase in preload; it does not mean that additional fluid is clinically required. Passive leg raising, a small fluid challenge, pulse-pressure variation, stroke-volume variation, and echocardiography can assist when their physiological assumptions are satisfied.

The clinically useful sequence is to identify the cause of instability, assess perfusion, assess responsiveness when appropriate, intervene selectively, and reassess. This reduces the tendency to treat every episode of hypotension with crystalloid.

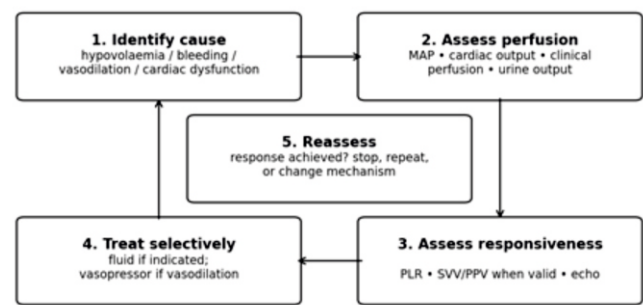


Fig. 5. Physiology-guided perioperative decision loop. This is a conceptual framework and not a validated clinical score

Acute kidney injury

AKI is central to the fluid debate because renal function is vulnerable to both inadequate perfusion and venous congestion. RELIEF demonstrated more AKI with restrictive therapy.¹ The ADQI/POQI consensus emphasizes baseline risk assessment, avoidance of nephrotoxins, adequate haemodynamic management, and prevention of secondary kidney injury.⁹ Oliguria alone should not automatically trigger fluid because urine output is influenced by anaesthesia, neuroendocrine responses, surgical stress, pneumoperitoneum, and renal haemodynamics.

A 2024 secondary analysis of RELIEF explored candidate kidney-protective factors.¹⁰ Long-term follow-up published in 2026 found no significant difference in new or progressive stage ≥3 chronic kidney disease between restrictive and liberal groups despite the original difference in postoperative AKI.¹¹ Early AKI and long-term CKD should therefore not be treated as interchangeable

outcomes.

Fluid accumulation and postoperative complications

Excess fluid increases interstitial water and may contribute to pulmonary oedema, bowel-wall oedema, impaired wound healing, and venous congestion. Conversely, insufficient circulating volume can compromise organ perfusion. The appropriate target is therefore neither indiscriminate liberal therapy nor an absolute zero-balance target, but adequate perfusion without avoidable accumulation

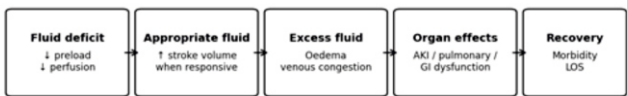


Fig. 6. Conceptual pathway linking inadequate or excessive fluid exposure with postoperative outcomes.

Fluid composition

Balanced crystalloids contain less chloride than 0.9% saline and may reduce hyperchloremia and metabolic acidosis. Large pragmatic trials have suggested modest differences in kidney-related outcomes in some populations, although results are not uniform.^{12,13} A 2024 meta-analysis in noncardiac surgery found less acidosis with balanced crystalloids but no clear reduction in renal insufficiency or mortality, with low certainty of evidence.¹⁴

Arterial pressure and vasopressors

Perioperative hypotension often reflects anaesthetic-induced vasodilation rather than absolute hypovolaemia. Repeated fluid boluses in an adequately filled, vasodilated patient can increase fluid accumulation without correcting the dominant mechanism.

The 2024 POQI arterial pressure consensus recommends maintaining intraoperative MAP at or above 60 mm Hg in at-risk patients, with higher targets when venous or compartment pressures are elevated, and treating hypotension according to its presumed cause.¹⁵ Fluid and vasopressors should therefore be complementary: fluid for clinically important preload deficiency when responsiveness is likely, and vasopressors when vasodilation predominates.

ERAS integration

ERAS is not synonymous with fluid restriction. Appropriate preoperative oral intake, avoidance of unnecessary replacement of theoretical fasting deficits, individualized

intraoperative haemodynamic management, and early postoperative oral intake reduce unnecessary fluid exposure.^{4,16}

DISCUSSION

The central message is that restrictive and liberal are trial labels, not universal prescriptions. Fluid requirements change with induction-related vasodilation, blood loss, pneumoperitoneum, surgical stress, cardiac reserve, and renal reserve. A fixed infusion rate therefore risks treating the protocol rather than the physiology.

RELIEF shows that excessive restriction may increase AKI without improving disability-free survival.¹ The appropriate interpretation is not that liberal fluid loading is universally superior, but that there is a lower boundary below which fluid avoidance may become harmful.

Mixed GDHT evidence also supports individualization. OPTIMISE suggested benefit, whereas OPTIMISE II did not reduce its primary endpoint and raised concern about arrhythmias associated with the intervention algorithm at 24 hours, a difference that did not persist to 30 days.^{7,8} The 2025 POQI consensus therefore supports selective, individualized use of GDHT rather than routine protocolization across heterogeneous surgical populations.³

Renal protection further supports mechanism-based treatment. Kidney injury may follow hypotension, blood loss, nephrotoxins, inflammation, and venous congestion. Fluid should not be administered solely to increase urine output, but genuine hypovolaemia should not be ignored. The dissociation between early AKI and long-term CKD in the RELIEF cohort^{10,11} reinforces this point: a strategy that reduces one physiological insult (hypoperfusion) or another (venous congestion) may still leave long-term renal trajectory largely unchanged, which argues against using short-term AKI incidence as a complete proxy for renal outcome when comparing fluid strategies.

Fluid composition and arterial pressure management are best understood as adjuncts to, rather than substitutes for, correct volume decision-making. Balanced crystalloids may reduce hyperchloraemic acidosis, but the evidence for a clinically important reduction in renal or mortality outcomes remains of low certainty¹⁴, so composition should not be expected to compensate for an incorrect volume or timing decision. Likewise, the POQI arterial pressure

consensus¹⁵ and the ERAS literature^{4,16} converge on the same underlying principle as the AKI and GDHT evidence: hypotension and oliguria are signals to be interpreted, not default triggers for fluid, and vasopressor support is frequently the more mechanistically appropriate response when vasodilation rather than hypovolaemia predominates.

Taken together, the restrictive-versus-liberal trials, the divergent GDHT results, the renal evidence, and the fluid-composition and arterial-pressure literature all point to the same conclusion from different directions: no single fixed volume, algorithm, or fluid type performs consistently across heterogeneous surgical populations. What varies between studies is not necessarily the correctness of the underlying physiology but the fidelity with which each protocol identified the right patient, at the right time, with the right intervention. This reframes the restrictive-versus-liberal debate as a question of diagnostic accuracy — correctly identifying the cause of haemodynamic instability — rather than one of selecting the correct default volume.

This synthesis also clarifies why protocolized GDHT has produced inconsistent results across trials^{7,8,17–19}: a rigid algorithm applied irrespective of patient phenotype inherits the same weakness as a rigid restrictive or liberal volume target. The clinical implication is that monitoring technology and consensus targets are enabling tools for individualization, not replacements for it.

Future practice is likely to combine clinical examination, point-of-care ultrasound, minimally invasive cardiac-output monitoring, arterial waveform analysis, and predictive tools. Technology should be used only when it changes management; sophisticated monitoring cannot compensate for an incorrect physiological diagnosis.

Clinical Contribution

- Reframes the restrictive-versus-liberal debate around individual physiology.
- Integrates fluid therapy with ERAS, AKI prevention, arterial pressure management, and vasopressor therapy.
- Distinguishes fluid responsiveness from the clinical indication to administer fluid.
- Explains why contemporary GDHT evidence supports selective rather than universal protocolization.

- Provides a practical decision loop adaptable to different surgical risk profiles.
- Identifies priorities for future trials of individualized perioperative haemodynamic care.

LIMITATIONS

- This is a narrative review rather than a systematic review or meta-analysis; no formal pooled estimate or GRADE assessment was performed.
- The search was structured but was not designed to generate a PRISMA systematic-review flow diagram with independently verified screening counts; Figure 1 instead summarizes the evidence-selection process as a narrative map.
- Evidence is heterogeneous in surgical population, fluid definitions, monitoring technology, haemodynamic targets, and outcome definitions.
- Some evidence for balanced crystalloids is indirect because it derives from critically ill or non-operative populations.
- Several GDHT studies evaluated bundled algorithms, making it difficult to isolate the effect of fluid administration alone.
- Long-term patient-centred outcomes remain less studied than short-term AKI and morbidity.

Future Research Priorities

Future trials should compare individualized fluid-and-vasopressor algorithms with contemporary usual care rather than arbitrary fluid volumes. Studies should stratify patients by renal and cardiac reserve, surgical magnitude, expected blood loss, baseline arterial pressure, and haemodynamic phenotype. Outcomes should prioritize AKI severity, major postoperative complications, quality of recovery, disability-free survival, length of stay, and long-term kidney and cardiovascular outcomes.

CONCLUSION

Perioperative fluid therapy should move beyond the binary labels of restrictive and liberal. Evidence from RELIEF shows that excessive restriction may increase AKI, while excessive administration may promote fluid accumulation and postoperative morbidity. ERAS supports avoidance of unnecessary intravenous therapy but should not be equated with near-zero fluid balance. Contemporary POQI guidance

supports individualized management based on surgical factors, intravascular volume status, comorbidities, arterial pressure, and haemodynamic response.^{1-3,15}

The most defensible modern paradigm is physiology-guided therapy: identify the cause of haemodynamic instability, assess perfusion and fluid responsiveness when appropriate, administer fluid only when likely to provide benefit, use vasopressors when vasodilation is dominant, and reassess continuously.

DECLARATIONS

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Data availability: No original dataset was generated or analyzed for this narrative review.

Author contributions: All three authors contributed to the conception and literature synthesis, manuscript preparation and critical revision, and approved the final manuscript.

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