Fluid Management in Abdominal Surgery

What, When, and When Not to Administer

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KEYWORDS

- Intravenous fluids • Crystalloids • Colloids • Balanced fluids • Fluid responsiveness
- Noninvasive monitoring

KEY POINTS

- Intravenous fluids are drugs with predominantly cardiovascular and renal effects, potentially significant gastrointestinal effects, and possible immune effects.
- Distribution of administered fluid volume across compartments (such as the intravascular, interstitial, and intracellular spaces) depends on several factors, including the integrity of the endothelial glycocalyx and intravascular volume context.
- Before the administration of fluid therapy, determination of volume responsiveness and volume status is recommended.
- Balanced crystalloids, with a physiologic strong ion difference and chloride content, may avoid the potentially deleterious effects of chloride-rich isotonic fluids like normal (0.9%) saline.
- Intravascular volume status may be assessed with variable accuracy using minimally invasive or noninvasive technologies.

INTRODUCTION

Intravenous fluid therapy is a key part of perioperative care, and surgical outcomes have been shown to be affected by the type and volume of fluid used. This review

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presents an overview of the basic principles that underlie fluid management in the perioperative setting, includes evidence-based recommendations (where tenable), and suggests a rational approach to the timing and choice of fluids for administration.

TYPE OF FLUID

A variety of fluid types are available, including different types of crystalloids, colloids, blood products, and even hemoglobin-based oxygen-carrying solutions. The use of normal or isotonic (0.9%) saline solution dates to the work of a Dutch chemist named Hartog Hamburger in 1896.[1] In an in vitro study of red blood cell (RBC) lysis in response to changes in tonicity, human RBCs were found to be most stable in a preparation of 0.92% saline. More recently, studies have reported an association between resuscitation with isotonic saline and several undesirable effects when compared with resuscitation with physiologically balanced crystalloids (eg, lactated Ringer solution, Plasma-Lyte, Hartmann solution).[2] Administration of 0.9% saline results in hyperchloremia and a decrease in the plasma strong ion difference with consequent metabolic acidosis.[3] This condition, in turn, has been associated with reduced cardiac contractility, decreased renal perfusion, reduced gastric blood flow, and impaired gastric motility.[4–7] Elevated serum chloride concentrations have been associated with renal vasoconstriction and renal parenchymal swelling in animal studies[8,9] and an increase in postoperative 30-day mortality in large database analyses.[10] The deleterious effects of administration of large volumes of 0.9% saline on the kidney have also been shown in a human study that demonstrated decreased renal blood flow velocity and cortical tissue perfusion.[11] Acknowledging potential clinical implications, the British Consensus Guidelines on Intravenous Fluid Therapy for Adult Surgical Patients recommended the use of balanced crystalloids rather than isotonic saline in most routine clinical settings.[12] The case for balanced crystalloids has also been presented comprehensively in a review.[13] Populations for whom isotonic saline remains a reasonable choice include patients with nausea/vomiting or gastric suction (and thus hypochloremic alkalosis) and neurosurgical patients for whom avoiding other hypotonic crystalloids may be reasonable.

Hadimioglu and colleagues[14] conducted a double-blind study randomizing kidney transplant recipients to receive isotonic saline, lactated Ringer solution, or Plasma-Lyte and compared subsequent changes in acid-base balance and potassium and lactate levels. No significant changes in pH or acid-base measures were seen in patients receiving lactated Ringer solution or Plasma-Lyte as opposed to those who received saline (7.44 ± 0.50 vs 7.36 ± 0.05, and 0.4 ± 3.1 vs -4.3 ± 2.1, respectively). However, there were no subsequent significant differences in postoperative renal function. The best metabolic profile was seen in patients receiving Plasma-Lyte. Shaw and colleagues[15] conducted an observational study evaluating the use of normal saline versus a calcium-free isotonic balanced crystalloid solution in adult patients undergoing major abdominal surgery using the Premier Perspective Comparative Database. A total of 926 patients who received Plasma-Lyte on the day of surgery were propensity-matched (in a 3:1 ratio) with 2778 patients who received saline. Hemodialysis occurred approximately 5 times more often in the matched saline group (1.0% [95% confidence interval (CI) 0.05–1.8] vs 4.8% [95% CI 4.1–5.7], $P<.001$); the matched saline group also had significantly increased odds of postoperative infection, blood transfusion, and electrolyte disturbance (sodium, potassium, and/or magnesium). In addition, in-hospital mortality was higher for the saline group (5.6%) than for the balanced crystalloid–Plasma-Lyte group (2.9%), although the difference was not significant after correcting for confounders. Further literature has confirmed the association between fluids with high chloride ion content (and a lower strong ion difference) and renal vasoconstriction and decreased glomerular filtration rate.[16]

Clinicians may prefer colloids (such as albumin, starches, and gelatins) based on the theory that intravascular retention is prolonged as compared with crystalloids (a theoretic premise that larger particles are trapped in the vascular space by an intact endothelial barrier). However, large multicenter clinical trials suggest that the advantage in volume expansion is usually only about 30% to 40%.[17,18] In a hypovolemic context, the potency of colloids is perhaps up to twice that of crystalloids.[19] Using a prospective study setup with double-tracer blood volume measurements (to assess volume status before and after crystalloid administration), Jacob and colleagues[20] evaluated the traditional model of replacement of blood loss with 3 times as much crystalloid. The volume effect of lactated Ringer solution was less than 20% (and additional infusions of hypertonic albumin were used to restore blood volume to original value) under these conditions.

The major drawbacks of colloid use include increased cost, potentially limited availability (with albumin, which is a blood product), possibly impaired coagulation (notable with larger quantities of hetastarch), and persistent evidence of renal injury seen with starch solutions.[21] Hetastarch use has been associated with increased bleeding in cardiac and neurologic surgery[22,23] and may increase the incidence of renal failure in septic patients[24] as well as in patients undergoing renal transplantation and cardiac surgery.[25,26] Meta-analyses show no improvement in survival with the use of colloids versus crystalloids among patients with trauma, burns, and following surgery.[27] Specific blood products (packed RBCs, fresh frozen plasma, cryoprecipitate, platelets, specific factor solutions) may be used for selected indications such as anemia or coagulation factor deficiencies in respective subgroups or during resuscitation (eg, hemorrhagic shock). Perioperative cell salvage and hemoglobin-based oxygen-carrying solutions are less commonly used.

AMOUNT OF FLUID

Various volume strategies have been studied including individualized goal-directed therapy (iGDT) and liberal, zero-balance, or restrictive approaches.[28] However, there is no uniform definition for what constitutes a liberal versus restrictive approach. Varadhan and Lobo[28] developed definitions to better compare outcomes: liberal was defined as greater than 2.75 L/day, zero-balance as 1.75 to 2.75 L/day, and restrictive as less than 1.75 L/day in the postoperative period. Brandstrup and colleagues[29] defined fluid management strategies for elective colorectal surgery based on quantities transfused for preloading with epidural analgesia, replacement of so-called third space losses, replacement of fasting deficits (maintenance requirements), and replacement of estimated blood losses. Fluid restriction resulted in fewer complications and earlier return of bowel function. Other studies have used different definitions of liberal versus standard fluid management approaches (incorporating the quantity of electrolytes received in a given day). Lobo and colleagues[5] evaluated the recovery of gastrointestinal function after elective colonic resection with a standard group receiving 3 L of intravenous fluids with 154 mmol sodium daily versus a restricted group receiving no more than 2 L of fluid with 77 mmol sodium. Patients in the standard group gained more weight, suffered delayed recovery of gastrointestinal function, endured increased overall complications, and had an extended hospital length of stay.

Perioperative iGDT aims to optimize circulation in the operating room via real-time individualized hemodynamic monitoring and therapeutic interventions to maximize stroke volume (SV).[30] iGDT may incorporate the use of fluid boluses, inotropic medications directed to maximize specific parameters such as cardiac index, SV, or minimize pulse pressure variation (PPV), or oxygen extraction ratio. A systematic review and meta-analysis of 29 trials involving 4805 moderate- and high-risk surgical patients by Hamilton and colleagues[31] and by Pearse and colleagues[32] showed a significant reduction in morbidity with the use of both fluids and inotropes (as opposed to fluids alone). Other studies evaluating preemptive iGDT have shown earlier return of bowel function, decreased incidence of nausea and vomiting, and reductions in hospital length of stay.[33,34] Although there are no standardized recommendations for the type and amount of fluid or inotrope to administer, therapy should be guided with real-time monitoring of fluid responsiveness so that the administration of fluid to a volume nonresponsive patient is avoided.[35,36]

A review of the literature on iGDT in colorectal surgery[37] drew attention to the fact that comparisons had not been made in the setting of restrictive fluid therapy (near-zero fluid balance) in the postoperative period and patients had not been managed within an enhanced recovery after surgery pathway.[38] This review also noted the heterogeneity in trials involving iGDT. The benefits observed in initial trials may be minimized by advances in surgical techniques and perioperative care.[37] Two trials support this position.[39,40] In a double-blind multicenter trial on 150 patients undergoing elective colorectal surgery, Doppler-guided iGDT to near-maximal SV added no benefit compared with the use of a zero-balance approach (ie, maintenance of near-normal body weight).[39] There were no significant differences in complications or duration of hospital stay. These results were confirmed in another study that randomized 85 patients undergoing elective colectomy within an established enhanced recovery protocol (including fluid restriction) to flow-guided or no flow-guided fluid therapy.[40] Based on these consistent findings, it may not be necessary to use flow-directed fluid volume therapy in all patients undergoing major surgery, particularly in the context of an existing enhanced recovery protocol in which postoperative fluid overload is avoided.[39,40] However, when blood loss is expected to be in excess of 500 mL, or if preoperative volume status is uncertain, Doppler-guided iGDT may remain potentially useful. The current safety warnings on hydroxyethyl starch (HES) add to the uncertainty surrounding iGDT because most trials were conducted with colloid. However, one study has suggested that either crystalloid or HES may be used with equal efficacy for flow-directed fluid therapy.[41]

A fluid challenge may be administered to evaluate hemodynamic response, with the volume administered being delivered rapidly (studies report timing the challenge over 5–10 minutes) and the bolus being large enough to recruit preload (inducing myocardial stretch), thereby increasing end-diastolic volume and SV.[35] In most iGDT protocols, SV maximization continues (with boluses of approximately 3 mL/kg) until SV no longer increases with volume loading (with a 10%–15% increase in SV set as the threshold for responsiveness). This concept differs from liberal strategies whereby the proximate dynamic hemodynamic response is not routinely monitored in real time. The overall goal includes maximization of cardiac output (CO) to preempt the development of an oxygen debt. Most studies support avoiding routine empirical fluid loading. Zero-balance techniques avoid net weight gain due to fluids with the use of volume removal where necessary (eg, with diuretics when there is a weight gain of approximately 2.5–3 kg).[42]

FLUID THERAPY WITHIN ENHANCED RECOVERY AFTER SURGERY

Traditional intravenous fluid regimens for patients undergoing abdominal surgery have incorporated large volumes often exceeding actual/measured fluid losses.[43] Patients may have received 3.5 to 7 L of fluids intraoperatively and up to 3 L per day on the surgical wards with ramifications as discussed earlier.[5,42] For many years, a third space was believed to exist and has been described in textbooks as a fluid compartment that needed to be replete.[38,44,45] Studies have confirmed that damage to the endothelial glycocalyx occurs with such administration of crystalloids and could result in accumulation of fluid in the interstitial space with consequent complications.

Concerns pertinent to patients undergoing elective abdominal surgery may be considered in 3 stages. In the preoperative setting, possible depletion of circulating volume secondary to prolonged fasting, mechanical bowel preparation, nausea/vomiting/diarrhea, or decreased oral intake due to gastrointestinal pathology may occur, resulting in a volume-responsive hypovolemic patient presenting for surgery. Intraoperatively, large incisions (during open laparotomy) resulting in evaporative fluid loss may be compounded by surgical blood loss. Postoperatively, patients may experience additional fluid depletion from a lack of oral intake, drains, fistulae, and/or increased ileostomy output. Hypotension due to hypovolemia is exacerbated by internal redistribution of effective circulating blood volume from thoracic epidural analgesia-induced sympathectomy (ie, there may be an increase in venous pooling of circulating blood volume leading to reduced preload).

In 2012, the Enhanced Recovery after Surgery (ERAS) Society, the European Society of Clinical Nutrition and Metabolism, and the International Association for Surgical Metabolism and Nutrition issued updated consensus recommendations: ERAS in colonic,[46] pelvic/rectal,[47] and pancreatic surgery.[48] The goal was to decrease hospital length of stay and time to resumption of normal activities and improve survival.[38] Preoperative optimization of the patient's fluid status before gastrointestinal surgery is achieved via 2 major initiatives: (1) avoiding excessive starvation by allowing solid food intake up to 6 hours and clear liquids up to 2 hours before anesthetic induction and (2) avoiding routine mechanical bowel preparation. Prolonged starvation (beyond 8 hours) may place patients in a catabolic state, increasing insulin resistance and prolonging hospital length of stay. Preoperative carbohydrate intake has been shown to reduce insulin resistance[49] without increasing the risk of aspiration[50] and may reduce hospital stay by a day in patients undergoing major abdominal surgery.[51] Bowel preparation has been shown to cause significant fluid and electrolyte imbalance, including hypocalcemia and hypophosphatemia.[52] Bucher and colleagues[53] performed a meta-analysis of 4 randomized controlled trials with a total of 1297 patients undergoing elective colorectal surgery. Anastomotic leakage was significantly more common in the bowel preparation group (5.6% vs 2.8%) in addition to general morbidity and mortality rates. Thus, the guidelines recommend bowel preparation solely for those patients undergoing low rectal resection with a diverting stoma.

Intraoperatively, the ERAS guidelines recommend the use of warmed intravenous fluids, preferring boluses with colloid and crystalloids for background maintenance. Thus, iGDT is recommended to optimize intravascular volume status and ensure an optimized SV while minimizing fluid overload. Targeting supranormal global oxygen delivery was demonstrated to have a significant survival benefit (by Shoemaker and colleagues[54] who conducted a prospective, longitudinal study analyzing hemodynamic and oxygen transport variables in 708 high-risk surgical patients). Cardiac index, oxygen delivery, and oxygen consumption were found to increase in the immediate postoperative setting, more dramatically in survivors as compared with nonsurvivors. Shoemaker and colleagues hypothesized that the need for increased CO may be due to increased metabolic demands after surgical trauma in the setting of previously low and maldistributed intraoperative blood flow due to neural and hormonal mechanisms. The combined use of fluids and inotropes (as opposed to fluids alone) has been shown to better facilitate achievement of supranormal oxygen delivery and reduce mortality (odds ratio, 0.41; 95% CI 0.23–0.73), although the exact doses of inotropes used were not evaluated. In addition, vasopressors should be administered to normovolemic hypotensive patients to avoid complications associated with using fluids alone (such as bowel edema and increased extravascular lung water).[46] Large-volume blood loss should prompt replacement (1:1) with allogeneic packed RBCs and fresh frozen plasma, with platelets administered as needed.

Postoperatively, no more than 2 to 2.5 L of water and 70 to 100 mmol sodium should be administered per day for most patients who require only maintenance fluid replacement (ie, without volume deficits or ongoing fluid and electrolyte losses). The ERAS guidelines advocate avoiding nasogastric tubes and the aggressive treatment of postoperative nausea and vomiting to enhance oral intake and wean intravenous fluids (ideally within 48 hours after colonic surgery). Optimal management thus includes the combination of iGDT with an overall aim to achieve a state of zero-balance in terms of weight gain. Patients undergoing laparotomy have fluid requirements different from those undergoing laparoscopic surgery. Increased fluid requirements in open laparotomy may result from a greater systemic inflammatory response syndrome (with greater loss of the endothelial glycocalyx), increased evaporative losses, and increased bowel handling/manipulation.[46] Patients undergoing laparotomy are more likely to have thoracic epidural analgesia and require intravenous fluids to counteract the sympathectomy-induced hypotension (from increased venous capacitance and relative hypovolemia), further exacerbated by the increased release of inflammatory mediators. Abdominal insufflation and Trendelenburg positioning during laparoscopic surgery have been shown to reduce tissue oxygen delivery.[55] Thus, appropriate decisions regarding fluid therapy must be made to augment SV during laparoscopy based on individualized assessments of hemodynamic variables.

CLINICAL ASSESSMENT AND MONITORING TO GUIDE FLUID THERAPY

Assessment of fluid status can be difficult in the operative setting where a formal physical examination cannot always be conducted. Traditional evaluation has focused on assessment of the heart rate, blood pressure, and urine output.[56] Volume deficits may not become apparent until they exceed 10% of body weight.[57] As noted by Vincent and Weil,[57] hypotension can be a nonspecific sign due to vascular inflow obstruction, heart failure, or a vasodilatory process. Changes in heart rate to maintain CO may be skewed by medications such as β-blockers and vasopressors. Other common intraoperative events such as surgical stimulatory actions activating nociceptive pathways and changes in body temperature may distort an anesthesiologist's interpretation of the patient's real-time volume status. Static measurements such as end-diastolic pressure and central venous pressure can also be influenced by a myriad of factors including patient comorbid cardiovascular pathologies and, thus, may not accurately reflect volume responsiveness.

Assessment of volume responsiveness may be performed with dynamic methods such as the administration of a rapid fluid bolus (3 mL/kg as earlier in the context of iGDT) or by a passive leg raise maneuver. Stroke volume variation (SVV) and/or PPV (with pulse pressure equal to the difference between systolic and diastolic pressure) can also be formally assessed. Analysis of various systems that quantify SVV, including the PiCCO plus system (Pulsion Medical Systems, Munich, Germany), FloTrac/Vigileo (Edwards Lifesciences, Irvine, CA, USA), and LiDCO plus technique (LiDCO Ltd, London, UK), revealed fluid responsiveness with SVV threshold values between 10% and 13%.[56,58–66] Marik and colleagues[67] conducted a meta-analysis of 29 studies encompassing a total of 685 patients to compare the accuracy of SVV, PPV, and systolic pressure variation (SPV) in predicting change in SV index or cardiac index after a fluid challenge during controlled mechanical ventilation. Roughly 56% of patients showed a demonstrable response to a fluid bolus. Pooled correlation coefficients between baseline SVV, PPV, and SPV and change in stroke/cardiac index were 0.72, 0.78, and 0.72, respectively. Areas under the receiver operating characteristic curves were 0.84, 0.94, and 0.86, respectively. This result favored PPV in comparison with SVV.

Monitors can help distinguish between hypovolemia and other causes of hypotension (such as cardiogenic, neurogenic or distributive, and obstructive). Pulmonary artery catheters (PACs), although frequently used in the past, have been associated with conflicting data.[68,69] Esophageal Doppler-based monitoring (EDM, Deltex Medical Inc, Chichester, West Sussex, United Kingdom) directly measures blood flow velocity in the descending thoracic aorta. Then, using a nomogram-based estimate of aortic cross-sectional area, CO and SV are estimated. Fluid boluses may be titrated to target an increase of at least 10% in the SV and CO. Dynamic arterial pressure–based computations such as SPV, PPV, and SVV may also help in defining fluid responsiveness (as described earlier). Noblett and colleagues[70] conducted a double-blind prospective randomized controlled trial with 108 patients randomized to either receive perioperative fluid at the discretion of the anesthetist (control group) or EDM-based optimization of SV. The intervention group had overall higher average SV, had shorter hospital length of stay (7 vs 9 days), tolerated enteral nutrition earlier, had a reduced increase in interleukin 6 level, and had decreased morbidity. Abbas and Hill[71] conducted a systematic review of EDM-guided therapy in major abdominal surgery and reported reduced hospital length of stay, faster return of gastrointestinal function, and reduced requirement for inotropes postoperatively. These studies were conducted before ERAS guidelines and fluid-restrictive strategies were commonplace.

The bioreactance-based noninvasive cardiac output monitor (NICOM, Cheetah Medical, Vancouver, WA, USA) uses 4 surface electrodes across the chest to compute an approximation of the CO, SV, and SVV. Waldron and colleagues[72] compared 100 adult patients undergoing elective colorectal surgery with goal-directed fluid therapy (using 250-mL colloid boluses) guided by either EDM or NICOM. Both monitors were used to assess monitor discrepancies in both patient groups. A 10% increase in SV was used for the fluid challenge. There was no statistically significant difference between monitor readings and no clinically significant differences in outcomes including postoperative pain, nausea, return of bowel function, organ dysfunction, and hospital length of stay. Critchley and Critchley[73] conducted a meta-analysis using Bland-Altman statistics (bias and precision) to compare novel CO measurement technology against gold standard techniques. They determined that a lack of precision of up to 30% was acceptable for routine clinical purposes. Peyton and Chong[74] conducted a meta-analysis reviewing data on EDM, NICOM, pulse contour techniques, and partial carbon dioxide rebreathing to assess percentage error versus PAC-based thermodilution. None of the techniques had less than 30% error (most were only about 45% precise).

New noninvasive systems display continuous noninvasive arterial pressures (CNAPs) recreating a beat-to-beat waveform. This technology is based on arterial tonometry and the volume clamp method. Current devices include Nexfin (BMEYE B.V., Amsterdam, The Netherlands), CNAP (CNSystems, Graz, Austria), and T-line (Tensys Medical, Inc, San Diego, CA, USA). Kim and colleagues[75] conducted a systematic review and meta-analysis of 28 studies (919 patients) reporting pooled random-effects bias and standard deviation (SD) measures of systolic arterial pressure, diastolic arterial pressure, and mean arterial pressure. Acceptable standards

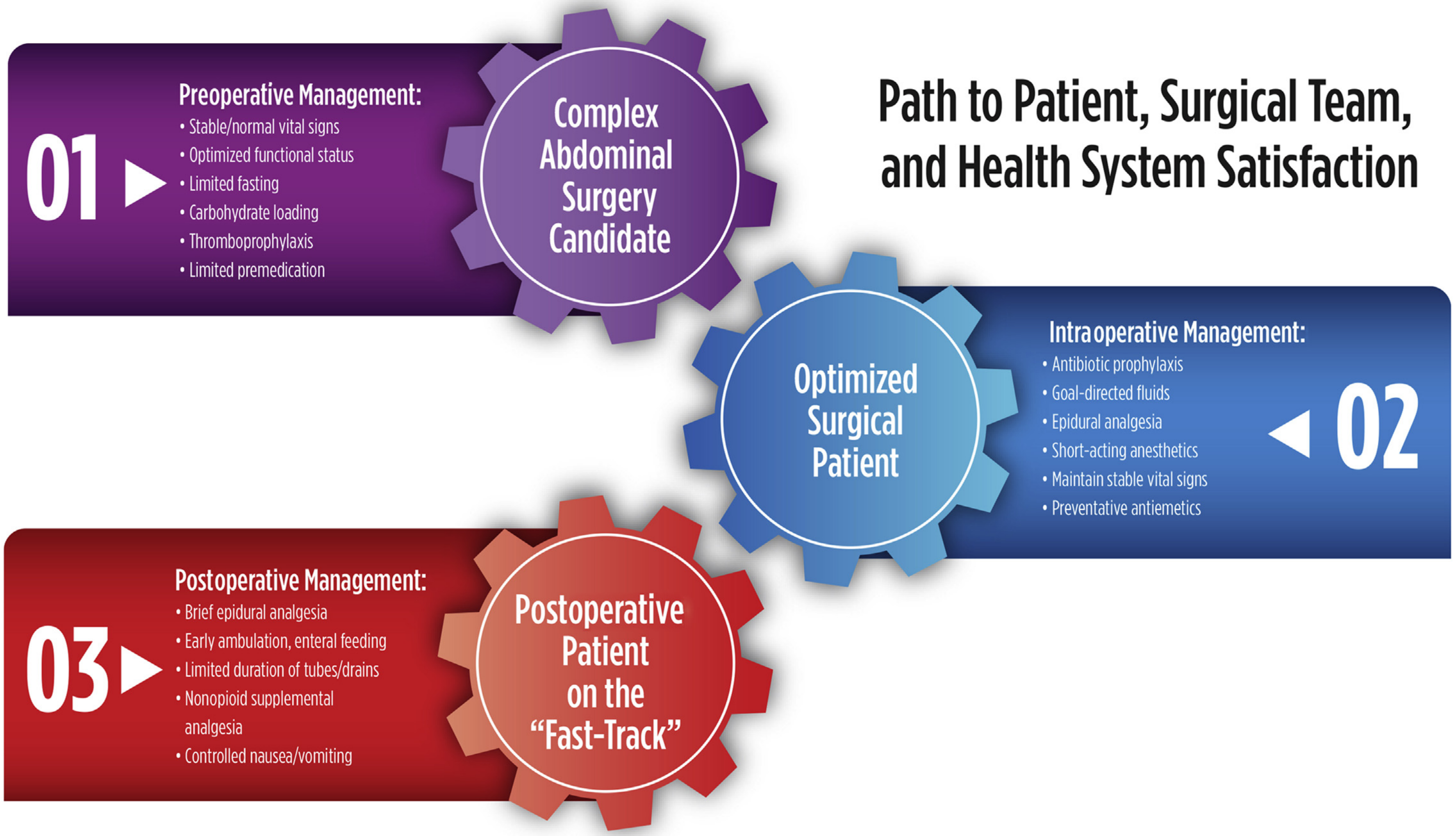

Fig. 1. Perioperative management of the complex abdominal surgery candidate.

for bias and SD were less than 5 and 8 mm Hg, respectively. The overall pooled bias and SD were -1.6 ± 12.2 mm Hg (95% limits of agreement -25.5 to 22.2 mm Hg) for systolic arterial pressure, 5.3 ± 8.3 mm Hg (-11.0 to 21.6 mm Hg) for diastolic arterial pressure, and 3.2 ± 8.4 mm Hg (-13.4 to 19.7 mm Hg) for mean arterial pressure. Kim and colleagues stressed the importance of this finding, noting that a patient with an invasive systolic arterial pressure reading of 100 mm Hg could have a CNAP reading of systolic arterial pressure anywhere from 74 to 123 mm Hg. These devices thus do not comply with standards created by the Arlington Association for the Advancement of Medical Instrumentation but are nevertheless clinically useful to monitor trends in certain settings.

In the late 1990s, physicians began analyzing the pulse oximetry waveform to obtain noninvasive data regarding volume status in mechanically ventilated patients in normal sinus rhythm.[76] Indices of fluid responsiveness include respiratory variation in pulse oximetric plethysmographic (POP) waveform amplitude (delta POP; manually calculated) and pleth variability index (PVI; continuous/automated calculation; measure of dynamic change in perfusion index occurring during the respiratory cycle where perfusion index is defined as the ratio of nonpulsatile to pulsatile blood flow through the peripheral capillary bed). Sandroni and colleagues[77] reported results of a meta-analysis of 10 studies with 233 patients that evaluated the accuracy of delta POP and/or PVI in predicting the hemodynamic response to a large or small fluid bolus (approximately 500 mL vs 250 mL, respectively). There was no major difference in change in the cardiac index, SV, or SV index found within studies incorporating the same-size fluid bolus. There was also no significant difference found between studies using delta POP and those using PVI. Sensitivity and specificity of the diagnostic techniques were more accurate with studies incorporating larger boluses, which likely indicates inaccuracy with poor capillary perfusion (eg, low CO, peripheral vasoconstriction, hypothermia). Analysis by Sandroni and colleagues suggests that plethysmographic indices are adequate for detecting fluid responsiveness, but less accurate in quantifying the magnitude of change in CO after fluid administration.

Fluid and electrolyte overload may result from liberal fluid replacement strategies or from moderate strategies in patients with comorbidities (such as congestive heart failure or end-stage renal disease). Patients may present with hypertension and significant weight gain (>2.5 kg), display jugular venous distention, develop pitting/peripheral edema, demonstrate a third heart sound on auscultation, have increased urine output, or accumulate ascites or pulmonary edema. Excessive crystalloid or colloid administration may result in a dilutional coagulopathy, which can be worsened by hypothermia if fluids are not properly warmed. Lack of fluid responsiveness (as defined previously) may indicate poor cardiac reserve or adequate resuscitation. EDM may show profiles of blood flow velocities consistent with euvolemia, whereas euvolemic mechanically ventilated patients have minimal PPV, SVV, and SPV on arterial waveform analysis. Changes in central venous pressure are neither sensitive nor specific for the identification of fluid responsiveness.[78] Inferior vena cava (IVC) diameter may be increased with minimal respiratory variation on bedside transthoracic echocardiogram (on standardized M-mode–based IVC variability measures). Multifrequency bioelectrical impedance analysis is currently used by some nephrologists to evaluate both extracellular and intracellular water volumes with proven accuracy.[79] Pharmacologic treatment of volume overload includes administration of diuretics such as furosemide (Lasix) or metolazone (Zaroxolyn) in patients with intact renal function.[80] Nonpharmacologic fluid removal (eg, hemodialysis) may be indicated in patients unresponsive to medications or intolerant to medication side effects who have signs of symptomatic renal failure.

SUMMARY

Optimal management of volume status before, during, and after abdominal surgery involves a combination of the recommendations discussed under ERAS protocols, iGDT in patients at moderate to high risk for perioperative complications, and a zero-balance goal in patients with significant physiologic reserve at low risk for complications (**Fig. 1**). Therapy may be titrated to fluid responsiveness or may be continued as long as weight gain is less than 2.5 kg (when dynamic measures are not being used). Measures such as mixed venous oxygen saturation, arterial lactate concentrations, or base deficit could be used as markers for globally adequate oxygen delivery. Ideally, patients are normotensive, with a cardiac index greater than 2.5 L/min/m^2 and a normal heart rate.

Future pathways for optimal fluid management may include the development of potentially noninvasive hemodynamic monitoring systems coupled with closed-loop fluid administration algorithms that would assess patients in the preoperative, intraoperative, and postoperative settings. At present, no single monitor can replace a vigilant anesthesiologist and surgeon working together to interpret hemodynamic data within the context of the patient's history and surgical procedure. Key decisions rely on experience with resource availability at the given perioperative setting and interventions with judicious intravenous fluid support.

REFERENCES

1. Lazarus-Barlow WS. On the initial rate of osmosis of blood-serum with reference to the composition of "physiological saline solution" in mammals. J Physiol 1896; 20(2-3):145–57.
2. Guidet B, Soni N, Della Rocca G, et al. A balanced view of balanced solutions. Crit Care 2010;14(5):325.
3. Kim SY, Huh KH, Lee JR, et al. Comparison of the effects of normal saline versus Plasmalyte on acid-base balance during living donor kidney transplantation using the Stewart and base excess methods. Transplant Proc 2013;45(6):2191–6.
4. Ho AM, Karmakar MK, Contardi LH, et al. Excessive use of normal saline in managing traumatized patients in shock: a preventable contributor to acidosis. J Trauma 2001;51(1):173–7.
5. Lobo DN, Bostock KA, Neal KR, et al. Effect of salt and water balance on recovery of gastrointestinal function after elective colonic resection: a randomized controlled trial. Lancet 2002;359(9320):1812–8.
6. Mecray PM, Barden RP, Ravdin IS. Nutritional edema: its effect on the gastric emptying time before and after gastric operations. Surgery 1937;1(1):53–64.
7. Tournadre JP, Allaouiche B, Malbert CH, et al. Metabolic acidosis and respiratory acidosis impair gastric-pyloric motility in anesthetized pigs. Anesth Analg 2000; 90(1):74–9.
8. Hansen PB, Jensen BL, Skott O. Chloride regulates afferent arteriolar contraction in response to depolarization. Hypertension 1998;32(6):1066–70.
9. Stone HH, Fulenwider JT. Renal decapsulation in the prevention of post-ischemic oliguria. Ann Surg 1977;186(3):343–55.
10. McCluskey SA, Karkouti K, Wijeysundera D, et al. Hyperchloremia after noncardiac surgery is independently associated with increased morbidity and mortality: a propensity-matched cohort study. Anesth Analg 2013;117(2):412–21.
11. Chowdhury AH, Cox EF, Francis ST, et al. A randomized, controlled, double-blind crossover study on the effects of 2-L infusions of 0.9% saline and Plasma-lyte® 148 on renal blood flow velocity and renal cortical tissue perfusion in healthy volunteers. Ann Surg 2012;256(1):18–24.

12. Powell-Tuck J, Gosling P, Lobo DN, et al. British consensus guidelines on intravenous fluid therapy for adult surgical patients (GIFTASUP). London: NHS National Library of Health; 2009.
13. Lobo DN, Awad S. Should chloride-rich crystalloids remain the mainstay of fluid resuscitation to prevent 'pre-renal' acute kidney injury?: con. Kidney Int 2014; 86(6):1096–105.
14. Hadimioglu N, Saadawy I, Saglam T, et al. The effect of different crystalloid solutions on acid-base balance and early kidney function after kidney transplantation. Anesth Analg 2008;107(1):264–9.
15. Shaw AD, Bagshaw SM, Goldstein SL, et al. Major complications, mortality, and resource utilization after open abdominal surgery: 0.9% saline compared to Plasma-Lyte. Ann Surg 2012;255(5):821–9.
16. Raghunathan K, Shaw AD, Bagshaw SM. Fluids are drugs: type, dose and toxicity. Curr Opin Crit Care 2013;19(4):290–8.
17. Finfer S, Bellomo R, Boyce N, et al. A comparison of albumin and saline for fluid resuscitation in the intensive care unit. N Engl J Med 2004;350(22): 2247–56.
18. Brunkhorst FM, Engel C, Bloos F, et al. Intensive insulin therapy and pentastarch resuscitation in severe sepsis. N Engl J Med 2008;358(2):125–39.
19. Annane D, Siami S, Jaber S, et al. Effects of fluid resuscitation with colloids vs crystalloids on mortality in critically ill patients presenting with hypovolemic shock: the CRISTAL randomized trial. JAMA 2013;310(17):1809–17.
20. Jacob M, Chappell D, Hofmann-Kiefer K, et al. The intravascular volume effect of Ringer's lactate is below 20%: a prospective study in humans. Crit Care 2012; 16(3):R86.
21. Hartog CS, Bauer M, Reinhart K. The efficacy and safety of colloid resuscitation in the critically ill. Anesth Analg 2011;112(1):156–64.
22. Wilkes MM, Navickis RJ, Sibbald WJ. Albumin versus hydroxyethyl starch in cardiopulmonary bypass surgery; a meta-analysis of postoperative bleeding. Ann Thorac Surg 2001;72(2):527–33.
23. Jonville-Bera AP, Autret-Leca E, Gruel Y. Acquired type I von Willebrand's disease associated with highly substituted hydroxyethyl starch. N Engl J Med 2001;345(8):622–3.
24. Schortgen R, Lacherade JC, Bruneel F, et al. Effects of hydroxyethyl starch and gelatin on renal function in severe sepsis: a multicentre randomised study. Lancet 2001;357(9260):911–6.
25. Cittanova ML, Leblanc I, Legendre C, et al. Effect of hydroxyethyl starch in brain-dead kidney donors on renal function in kidney-transplant recipients. Lancet 1996;348(9042):1620–2.
26. Rioux JP, Lessard M, De Bortoli B, et al. Pentastarch 10% (250 kDa/0.45) is an independent risk factor of acute injury following cardiac surgery. Crit Care Med 2009;37(4):1293–8.
27. Perel P, Roberts I, Ker K. Colloids versus crystalloids for fluid resuscitation in critically ill patients. Cochrane Database Syst Rev 2013;(2):CD000567.
28. Varadhan KK, Lobo DN. A meta-analysis of randomized controlled trials of intravenous fluid therapy in major elective open abdominal surgery: getting the balance right. Proc Nutr Soc 2010;69(4):488–98.
29. Brandstrup B, Tonnesen H, Beier-Holgersen R, et al. Effects of intravenous fluid restriction on postoperative complications: comparison of two perioperative fluid regimens: a randomized assessor-blinded multicenter trial. Ann Surg 2003; 238(5):641–8.

30. Cecconi M, Corredor C, Arulkumaran N, et al. Clinical review: goal-directed therapy - what is the evidence in surgical patients? The effect on different risk groups. Crit Care 2013;17(2):209.
31. Hamilton MA, Cecconi M, Rhodes A. A systematic review and meta-analysis on the use of preemptive hemodynamic intervention to improve postoperative outcomes in moderate and high-risk surgical patients. Anesth Analg 2011;112(6): 1392–402.
32. Pearse RM, Harrison DA, MacDonald N, et al. Effect of a perioperative, cardiac output-guided hemodynamic therapy algorithm on outcomes following major gastrointestinal surgery: a randomized clinical trial and systematic review. JAMA 2014;311(21):2181–90.
33. Gan TJ, Soppitt A, Maroof M, et al. Goal-directed intraoperative fluid administration reduces length of hospital stay after major surgery. Anesthesiology 2002;97(4): 820–6.
34. Pearse R, Dawson D, Fawcett J, et al. Early goal-directed therapy after major surgery reduces complications and duration of hospital stay. A randomized, controlled trial. Crit Care 2005;9(6):R687–93.
35. Cecconi M, Parsons AK, Rhodes A. What is a fluid challenge? Curr Opin Crit Care 2011;17(3):290–5.
36. Bennett-Guerrero E. Hemodynamic goal-directed therapy in high-risk surgical patients. JAMA 2014;311(21):2177–8.
37. Srinivasa S, Taylor MH, Sammour T, et al. Oesophageal Doppler-guided fluid administration in colorectal surgery: critical appraisal of published clinical trials. Acta Anaesthesiol Scand 2011;55(1):4–13.
38. Lassen K, Soop M, Nygren J, et al. Consensus review of optimal perioperative care in colorectal surgery: Enhanced Recovery After Surgery (ERAS) Group recommendations. Arch Surg 2009;144(10):961–9.
39. Brandstrup B, Svendsen PE, Rasmussen M, et al. Which goal for fluid therapy during colorectal surgery is followed by the best outcome: near-maximal stroke volume or zero fluid balance? Br J Anaesth 2012;109(2):191–9.
40. Srinivasa S, Taylor MH, Singh PP, et al. Randomized clinical trial of goal-directed fluid therapy within an enhanced recovery protocol for elective colectomy. Br J Surg 2013;100(1):66–74.
41. Yates DR, Davies SJ, Milner HE, et al. Crystalloid or colloid for goal-directed fluid therapy in colorectal surgery. Br J Anaesth 2014;112(2):281–9.
42. Lobo DN, Macafee DA, Allison SP. How perioperative fluid balance influences postoperative outcomes. Best Pract Res Clin Anaesthesiol 2006; 20(3):439–55.
43. Hannemann P, Lassen K, Hausel J, et al. Patterns in current anaesthesiological peri-operative practice for colonic resections: a survey in five northern European countries. Acta Anaesthesiol Scand 2006;50(9):1152–60.
44. Jacob M, Chappell D, Rehm M. The ‘third space’ – fact or fiction? Best Pract Res Clin Anaesthesiol 2009;23(2):145–57.
45. Shires T, Williams J, Brown F. Acute change in extracellular fluids associated with major surgical procedures. Ann Surg 1961;154:803–10.
46. Gustafsson UO, Scott MJ, Schwenk W, et al. Guidelines for perioperative care in elective colonic surgery: Enhanced Recovery After Surgery (ERAS®) Society recommendations. Clin Nutr 2012;31(6):783–800.
47. Nygren J, Thacker J, Carli F, et al. Guidelines for perioperative care in elective rectal/pelvic surgery: Enhanced Recovery After Surgery (ERAS®) Society recommendations. Clin Nutr 2012;31(6):801–16.

48. Lassen K, Coolsen MM, Slim K, et al. Guidelines for perioperative care for pancreaticoduodenectomy: Enhanced Recovery After Surgery (ERAS®) Society recommendations. Clin Nutr 2012;31(6):817–30.
49. Kingsnorth A, Bowley D. Fundamentals of surgical practice: a preparation guide for the intercollegiate MRCS examination. 3rd edition. New York: Cambridge University Press; 2011. p. 181–9.
50. Brady M, Kinn S, Stuart P. Preoperative fasting for adults to prevent perioperative complications. Cochrane Database Syst Rev 2003;(4):CD004423.
51. Awad S, Varadhan KK, Ljungqvist O, et al. A meta-analysis of randomised controlled trials on preoperative oral carbohydrate treatment in elective surgery. Clin Nutr 2013;32(1):34–44.
52. Holte K, Nielsen KG, Madsen JL, et al. Physiologic effects of bowel preparation. Dis Colon Rectum 2004;47(8):1397–402.
53. Bucher P, Mermillod B, Gervaz P, et al. Mechanical bowel preparation for elective colorectal surgery: a meta-analysis. Arch Surg 2004;139(12):1359–65.
54. Shoemaker WC, Appel PL, Kram HB. Hemodynamic and oxygen transport responses in survivors and nonsurvivors of high-risk surgery. Crit Care Med 1993;21(7):977–90.
55. Levy BF, Fawcett WJ, Scott MJ, et al. Intra-operative oxygen delivery in infusion volume-optimized patients undergoing laparoscopic colorectal surgery within an enhanced recovery programme: the effect of different analgesic modalities. Colorectal Dis 2012;14(7):887–92.
56. Hofer CK, Cannesson M. Monitoring fluid responsiveness. Acta Anaesthesiol Taiwan 2011;49(2):59–65.
57. Vincent JL, Weil MH. Fluid challenge revisited. Crit Care Med 2006;34(5):1333–7.
58. Hofer CK, Muller SM, Furrer L, et al. Stroke volume and pulse pressure variation for prediction of fluid responsiveness in patients undergoing off-pump coronary artery bypass grafting. Chest 2005;128(2):848–54.
59. Berkenstadt H, Margalit N, Hadani M, et al. Stroke volume variation as a predictor of fluid responsiveness in patients undergoing brain surgery. Anesth Analg 2001;92(4):984–9.
60. Preisman S, Kogan S, Berkenstadt H, et al. Predicting fluid responsiveness in patients undergoing cardiac surgery: functional haemodynamic parameters including the respiratory systolic variation test and static preload indicators. Br J Anaesth 2005;95(6):746–55.
61. Reuter DA, Kirchner A, Felbinger TW, et al. Usefulness of left ventricular stroke volume variation to assess fluid responsiveness in patients with reduced cardiac function. Crit Care Med 2003;31(5):1399–404.
62. Biais M, Nouette-Gaulain K, Cottenceau V, et al. Uncalibrated pulse contour-derived stroke volume variation predicts fluid responsiveness in mechanically ventilated patients undergoing liver transplantation. Br J Anaesth 2008;101(6):761–8.
63. Cannesson M, Musard H, Desebbe O, et al. The ability of stroke volume variations obtained with Vigileo/FloTrac system to monitor fluid responsiveness in mechanically ventilated patients. Anesth Analg 2009;108(2):513–7.
64. Hofer CK, Senn A, Weibel L, et al. Assessment of stroke volume variation for prediction of fluid responsiveness using the modified FloTrac and PiCCOplus system. Crit Care 2008;12(3):R82.
65. Belloni L, Pisano A, Natale A, et al. Assessment of fluid-responsiveness parameters for off-pump coronary artery bypass surgery: a comparison among LiDCO, transesophageal echocardiography, and pulmonary artery catheter. J Cardiothorac Vasc Anesth 2008;22(2):243–8.

66. de Wilde RB, Geerts BF, van den Berg PC, et al. A comparison of stroke volume variation measured by the LiDCOplus and FloTrac-Vigileo system. Anaesthesia 2009;64(9):1004–9.
67. Marik PE, Cavallazzi R, Vasu T, et al. Dynamic changes in arterial waveform derived variables and fluid responsiveness in mechanically ventilated patients: a systematic review of the literature. Crit Care Med 2009;37(9):2642–7.
68. Harvey S, Young D, Brampton W, et al. Pulmonary artery catheters for adult patients in intensive care. Cochrane Database Syst Rev 2006;(3):CD003408.
69. Takala J. The pulmonary artery catheter: the tool versus treatments based on the tool. Crit Care 2006;10(4):162.
70. Noblett SE, Snowden CP, Shenton BK, et al. Randomized clinical trial assessing the effect of Doppler-optimized fluid management on outcome after elective colorectal resection. Br J Surg 2006;93(9):1069–76.
71. Abbas SM, Hill AG. Systematic review of the literature for the use of oesophageal Doppler monitor for fluid replacement in major abdominal surgery. Anaesthesia 2008;63(1):44–51.
72. Waldron NH, Miller TE, Thacker JK, et al. A prospective comparison of a noninvasive cardiac output monitor versus esophageal Doppler monitor for goal-directed fluid therapy in colorectal surgery patients. Anesth Analg 2014;118(5):966–75.
73. Critchley LA, Critchley JA. A meta-analysis of studies using bias and precision statistics to compare cardiac output measurement techniques. J Clin Monit Comput 1999;15(2):85–91.
74. Peyton PJ, Chong SW. Minimally invasive measurement of cardiac output during surgery and critical care: a meta-analysis of accuracy and precision. Anesthesiology 2010;113(5):1220–35.
75. Kim SH, Lilot M, Sidhu KS, et al. Accuracy and precision of continuous noninvasive arterial pressure monitoring compared with invasive arterial pressure: a systematic review and meta-analysis. Anesthesiology 2014;120(5):1080–97.
76. Shamir M, Eidelman LA, Floman Y, et al. Pulse oximetry plethysmographic waveform during changes in blood volume. Br J Anaesth 1999;82(2):178–81.
77. Sandroni C, Cavallaro F, Marano C, et al. Accuracy of plethysmographic indices as predictors of fluid responsiveness in mechanically ventilated adults: a systematic review and meta-analysis. Intensive Care Med 2012;38(9):1429–37.
78. Marik PE, Cavallazzi R. Does the central venous pressure predict fluid responsiveness? An updated meta-analysis and a plea for some common sense. Crit Care Med 2013;41(7):1774–81.
79. Davenport A. Dialysis: bioimpedance spectroscopy for assessment of fluid overload. Nat Rev Nephrol 2013;9(5):252–4.
80. Goldstein S, Bagshaw S, Cecconi M, et al. Pharmacological management of fluid overload. Br J Anaesth 2014;113(5):756–63.