EMERGING TECHNOLOGY REVIEW

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Physiologic Goal-Directed Therapy in the Perioperative Period: The Volume Prescription for High-Risk Patients

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CONVENTIONAL PERIOPERATIVE hemodynamic management based on assumed preoperative volume deficits, blood pressure, heart rate, and urine output is not reliable for guiding volume therapy to avoid inadequate perioperative tissue perfusion. Goal-directed therapy (GDT) has been used to describe a wide variety of interventions and approaches to hemodynamic management tailored to patient-specific physiologic goals. GDT in the perioperative period involves measurement of an individual patient's oxygen flux or reliable surrogates and interventions objectively directed to augment flow. Successful GDT includes assessment of the adequacy of these interventions in a patient-specific context. The authors present the Physiologic Optimization Program (POP) as a rational physiologic approach to perioperative hemodynamic management.

Goal-directed therapy (GDT) in the perioperative period has been demonstrated to save lives, decrease complications, and save money.[1–5] However, despite overwhelming evidence to support the practice, its adoption by the anesthesia/perioperative care community generally is poor.[1] Barriers to its translation into clinical practice may involve administrative, economic, physician-autonomy, and institutional issues.[1] The cost of technologies as well as lack of familiarity and training with the necessary hardware also may be obstacles to the widespread adoption of GDT in the perioperative period. Current anesthetic practice has evolved to the point at which even the highest-risk surgeries are accomplished routinely without significant morbidity or early mortality.[6] Monitoring technologies may have contributed to these successes by instantaneously identifying complex physiologic derangements so that they can be addressed quickly. For less ill patients who still meet high-risk criteria, physiologic stability is expected and achieved in the majority.

Therapy aimed at improving hemodynamics is used routinely in the intensive care unit (ICU) and the operating room (OR), especially for the critically ill. Unfortunately and surprisingly, this often is done without verification or knowledge of its impact on cardiac performance or oxygen delivery.[1] Even though perioperative goal-directed therapies have shown improved patient outcomes and despite recommendations from prominent experts in the United States and British Consensus Guidelines,[7,8] assessment of oxygen delivery during high-risk surgery remains inconsistent.[1] Failure to assess the impact of patient physiology and interventions on oxygen delivery misses a significant opportunity to improve care for these patients.

The complications and mortality that may be influenced by the application of GDT to perioperative care extend beyond the immediate postoperative period.[3,9] Despite evidence to the contrary, these deaths and complications simply are not considered to be related to perioperative management.[3,9] Complications determine length of stay, and readmission is a primary driver of cost for the surgical patient.[10] Thus, the mechanism by which perioperative GDT may have extended benefits beyond the immediate intraoperative and ICU recovery periods may be a reduction in hospital length of stay and in readmissions.

Cost accounting that assesses the entire cost of care without assigning it to specific cost centers may further understanding in this area and better inform decision-makers about the cost-effectiveness of GDT. A corollary issue hindering better diffusion of GDT into clinical practice is the myriad of protocols and devices available to practitioners and the confusion this may engender when selecting an intervention.

FUNCTIONAL HEMODYNAMICS

Considering the increasing complexity of surgery and the advancing age and increased co-morbidities in surgical populations,[11,12] the need to understand the underlying physiology of functional hemodynamics is growing in importance. Also, the use of monitoring devices that allow clinicians to track and treat flow-related variables during the perioperative period is growing. Vigilant, well-trained clinicians remain indispensible for the success of algorithms.

Functional hemodynamic monitoring encompasses the measurement of physiologic variables such as the plethysmographic variability index (PVI), invasive arterial pressure- based parameters such as the stroke volume (SV), stroke volume variation

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(SVV), pulse pressure variation (PPV) or systolic pressure variation (Delta-down) and Doppler measures, SV and flow time (FTc) or echo assessment of SV and inferior vena cava compressibility (IVC variability). Functional variables are influenced by both the heart-lung interaction and volume status.[13] These parameters can determine volume responsiveness, ie, the extent to which the administration of fluids will augment cardiac output (CO). Volume responsiveness reveals the ability of the myocardium to increase contractility when sarcomere stretch occurs. These variables aid prediction of the effects of fluid therapy, answering the question, "Will fluid administration augment flow?" It has become clear that application of the right kind of fluid therapy at the right time in the right amount is needed for optimal patient outcomes.[14] However, equating volume responsiveness to the volume requirement is not appropriate either, as such volume treatment adds cost and, potentially, complications without the possibility of significant benefit (for example, the application of functional hemodynamics to optimize volume for the elective laparoscopic cholecystectomy in patients with little or no pre-morbidity [ASA PS 1-2]).

GOAL-DIRECTED THERAPY

GDT approaches prescribe fluids based on an individualized demonstration of flow augmentation. Volume therapy can be optimized and individualized with this approach. As volume therapy generally is the primary route of DO_2 augmentation for the majority of high-risk surgical patients, this approach allows precision and eliminates guesswork. The balance between volume therapy and vasoactive treatment to achieve oxygen delivery and other physiologic targets ultimately remains a clinical decision requiring clinical skill and thorough knowledge of patient physiology.

Fluids are given only to patients showing at least a 10% increase in SV, or fluid therapy initiated when PPV is $>12\%$ or SVV is $>10\%$ in appropriate patients.[3,15–17] Patients receive fluids based on an assessment of left ventricular (LV) preload responsiveness (Fig 1). Flow is recruited continuously to meet an individualized goal, and hypoperfusion is avoided. In addition, fluids are restricted in nonresponders, and hypervolemia is mitigated. Volume limitation is desirable in patients undergoing thoracic or hepatic surgery (those with acute lung injury/acute respiratory distress syndrome or dilutional coagulopathy, as common examples). Vasoactive therapy to augment DO_2 may be preferable to volume for individual patients. Calculations and knowledge of DO_2 with either approach simplifies decision-making, adding a further degree of precision to care.

SVV/SV pairs allow individual determination of a patient's position on the Frank-Starling curve that can indicate when volume is required to improve cardiac performance and, conversely, when volume can be removed safely (ie, patients can be individually "Starling-ized").

Titrated volume management using an SVV target is available only for patients with controlled positive-pressure ventilation with adequate tidal volumes and without significant arrhythmias. The utility of SVV or PPV to assess volume responsiveness has not yet been validated adequately in patients without these conditions. Resuscitation of patients with a CO

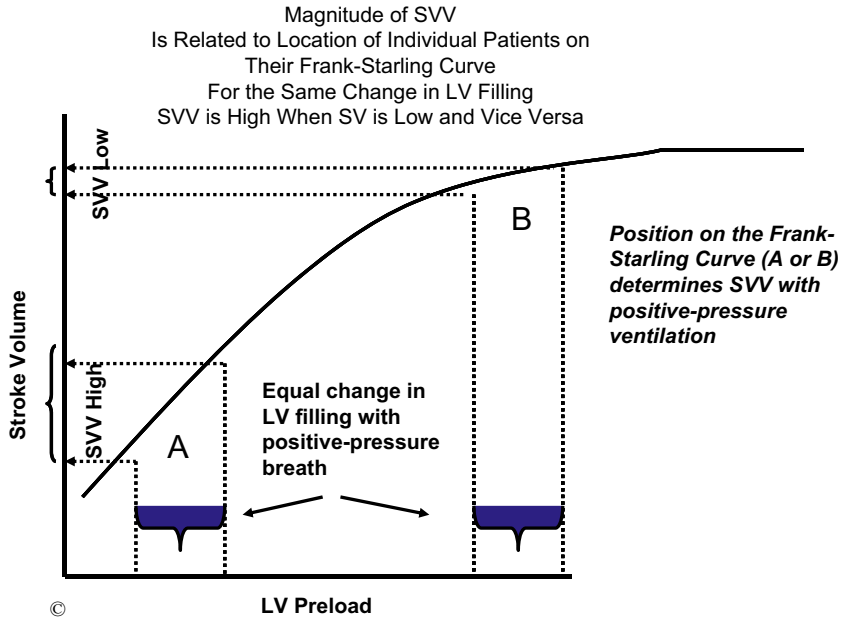

Fig 1. A patient's position on the Frank-Starling curve determines cardiorespiratory changes with positive-pressure ventilation. A and B represent different locations on the Frank-Starling curve. Positive-pressure ventilation produces the same change in preload at either A or B; however, the impact on SV varies. The change in SV induced by 1 positive-pressure breath is proportional to SVV. SVV is determined by the magnitude of preload dependency. Patients with higher SVV are more volume responsive (A, preload-dependent), functioning on the steeper portion of the Frank-Starling curve. SVV decreases as preload-dependent LV function is optimized (B. preload-independent). In these patients (B), other therapy is required to augment cardiac performance. Alternatively, volume may be removed safely if necessary, as cardiac performance is not influenced by changes in preload on this part of the Frank-Starling curve. Abbreviations: SV, stroke volume; SVV, stroke volume variation; LV, left ventricle. (Color version of figure is available online.)

measure, however, is uncomplicated, relying on the titration of volume and vasoactive therapy against cardiac performance, as measured by SV and CO. This approach provides assurance that preload optimization has occurred before implementation of pharmacotherapy when volume is the primary treatment. Vasoactive titration is accomplished similarly, with continuous assessment of meaningful physiologic targets: blood pressure, SV and cardiac output, and oxygen delivery.

With decreasing use of the pulmonary artery catheter, minimally invasive technologies have become popular to assess flow and fluid responsiveness.[18] Monitoring has to be coupled with a physiologically rational treatment algorithm to improve outcomes. The method used to measure flow may be important, as well as the specific parameter being optimized.[3,19] Greater support exists for esophageal Doppler,[20,21] echocardiographic assessment of inferior vena cava respiratory variability,[22] and PPV[17] than for arterial pulse-contour-based methods[23] or venous oximetry.[24]

PHYSIOLOGIC OPTIMIZATION PROGRAM

The authors developed the POP aimed to titrate preload and, ultimately, oxygen delivery (DO_2) at the patient level and thereby promote individualized therapy.[25] Although this approach has not been compared with another goal-directed approach in a randomized prospective trial, clinicians can apply the POP using functional hemodynamics as founding rationale for decision-making. Hemodynamic performance at the bedside may be monitored using targets amenable to iterative preload challenge based on objective measures of cardiac performance.

Many clinical studies revealed a linear relationship between SVV or PPV and preload responsiveness.[26–29] Figure 2 describes the utility of this approach independent of the patient-specific Frank-Starling curve.

As Figure 2 implies, the relationship between SV and preload responsiveness is a continuous, not dichotomous, relationship. A lesser definition of volume responsiveness, ie, 10% change in CO, likely would yield a lower value for SVV; whereas a higher value, ie, 20% change, would produce a higher threshold and improved specificity for SVV. Therefore, it is best to think of the range between 10%-15% as providing the best discrimination[17] (ie, most patients with SVV $>15\%$ will respond to volume with a meaningful change in cardiac performance, whereas those with SVV $<10\%$ will not. The most relevant question regarding intelligent application of functional hemodynamics is the combination of magnitude of change and the direction of change in the dynamic parameter. Looking at the variables together adds precision to the more typical empiric approach to volume management.

Thus the POP answers three questions: (1) What is the patient's current state of volume responsiveness? (2) What is the goal of volume loading in the perioperative setting? (intuitively, it is to obtain the greatest benefit from preload, adhering to the Frank-Starling mechanism) and (3) What is the impact of therapy on cardiac performance as measured by the change?

The POP is not device-dependent and is based on well-established physiology. This program is further simplified by the fact that it uses physiologic targets—SV and CO as the cardiac perfusion measure and SVV as the volume-responsiveness indicator—that rely on functional hemodynamics. This approach encourages individualization based on the unique physiology encountered with each patient (Fig 3).

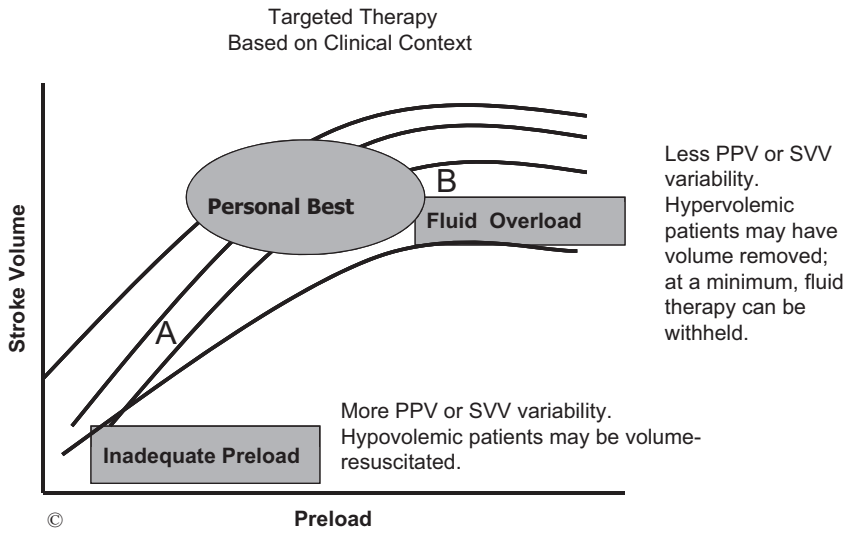

Fig 2. Multiple Frank-Starling curves represent individual patients or a single patient whose cardiac performance varies throughout the course of illness and treatment. Determining where a patient is on one's own Frank-Starling curve at any point in their illness is what makes this data pair (SVV:SV) a major breakthrough in the ability to care for high-risk surgical patients and the critically ill. When SVV is elevated >10%-15%, (A) patients are on the preload-dependent part of the curve where they will respond to volume. B represents the preload-independent portion of the curve, with SVV <10%; if cardiac performance (SV) is inadequate (determined clinically), therapy other than volume is necessary to augment cardiac performance. The inflection point of the Frank-Starling curve defines the transition between the preload dependence and independence. When this point is reached, preload-dependent cardiac function has been optimized, defined as the administration of just enough fluid to reach an individual's "personal best". Abbreviations: SV, stroke volume; SVV, stroke volume variation, PVV = pulse pressure variation. (Color version of figure is available online.)

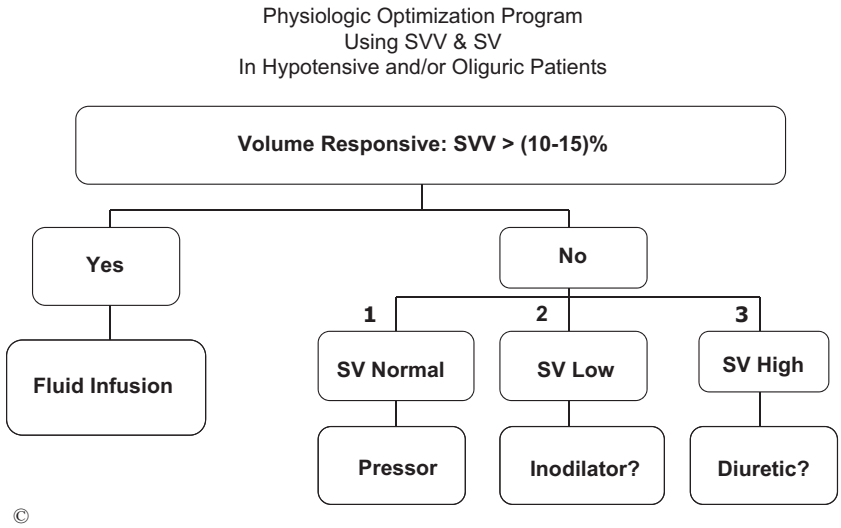

Fig 3. Pathway 1 may represent a vasodilated patient; resuscitated septic shock is a common example. Pathway 2 may represent a patient with congestive heart failure; determination of ejection fraction is very helpful in selecting appropriate therapy for this situation. Pathway 3 often represents the aftermath of successful resuscitation. For patients with acute lung injury or acute respiratory distress syndrome, diuretics are appropriate. For patients with clear lungs, this remains an open question but, at a minimum, further volume therapy should be withheld. Abbreviations: SV, stroke volume; SVV, stroke volume variation. (Color version of figure is available online.)

Importantly, use of the POP typically is initiated by a clinical question—often hypotension and/or oliguria—and is designed to augment, not supplant, clinical decision-making. These three questions are not asked in a vacuum. Physicians know much more about their patients than the SV–SVV data pair. However, these data pairs provide important information about the best approach to augment cardiac performance; (Fig 3). There is little intrinsic value in the treatment of hypotension or oliguria in the absence of contextual information. For instance, the use of hypotension as a surrogate for hypoperfusion in the patient receiving elective neuraxial analgesia (via epidural local anesthetic) is not necessarily the right choice, as the context here actually might be a better-than-normal cardiac output. Vasopressors (requiring low-dose alpha-agonists) might be indicated rather than volume therapy, as there is relative and not absolute hypovolemia. Vasoconstriction corrects the redistribution of volume between stressed and unstressed compartments, an "internal volume bolus" that returns hemodynamics to the homeostatic center while avoiding the application of "external-volume" therapy.[30]

POP in the OR

Although the POP has improved clinicians' ability and confidence in applying physiology-based therapies for the critically ill or injured, the best evidence of the utility of manipulating cardiac output and oxygen delivery to improve outcomes exists for operative patients. In the operating room, the program has been simplified to emulate routine anesthetic practice regarding the use of volume therapy and vasopressors; it enables physiology-based volume management for most high-risk patients.[31,32] Importantly, considering the negative implications of excess fluid, it provides endpoints for volume therapy.

In the OR, maintenance of hemodynamic homeostasis with appropriate application of volume, inotrope, and pressor therapy is a primary goal. The OR typically provides the ideal environment for physiologic treatment based on application of functional hemodynamics: Controlled mechanical ventilation with an adequate tidal volume. As it is difficult to describe all the intraoperative scenarios that ultimately would lead to a clinical choice to use volume or pressors, suffice it to say that this approach is most helpful whenever the following questions arise: (1) Is the patient at-risk for accruing tissue oxygen debt? (2) If so, is the patient volume-responsive? and (3) If so, is this because of absolute hypovolemia (needs volume boluses) or relative hypovolemia (needs to recruit volume from unstressed reservoirs such as the splanchnic circulation)?

The POP provides assurance that hemodynamic therapy is appropriate and eliminates the guesswork that invariably

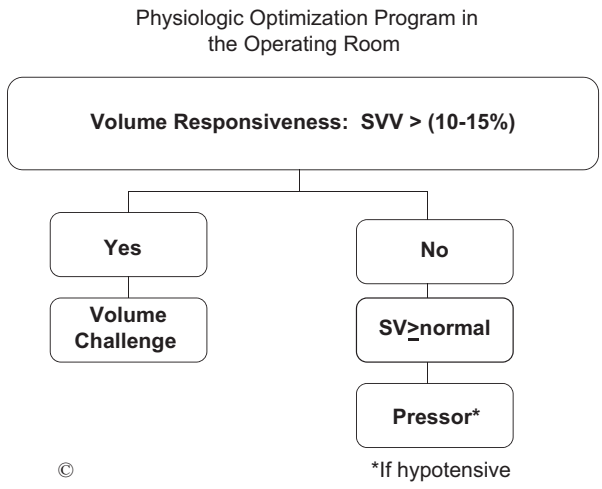

Fig 4. For most high-risk surgical patients, this very simple physiologic algorithm enhances and simplifies clinical decision-making to a greater extent than using information based on arterial pressure monitoring alone. Abbreviation: SV, stroke volume; SVV, stroke volume variation. (Color version of figure is available online.)

accompanies decision-making in the absence of flow measures (Fig 4).

Applied physiology in the operating room increases confidence that hemodynamic therapy is achieving desired goals. Additional outcome parameters that are assessed individually include vital signs, peripheral perfusion, urine output, base excess, and lactate, along with O_2 extraction in the more critically ill. An example using the POP in a complex patient undergoing major abdominal surgery throughout the perioperative period is included as Appendix A.

Limitations

It is important to note that SVV or PPV does not always predict volume responsiveness. The cardiorespiratory interactions that underlie SVV and PPV will vary based on irregular cardiac or respiratory cycles without implying the need for volume. Also, if the cardiorespiratory interactions are not of adequate magnitude (low tidal volume, open chest) or are excessive (abdominal compartment pressures are high), then the dynamic parameters may not be reliable. A list of limitations is shown in Table 1. If filling of the ventricles is changing on a beat-to-beat interval because of a significant arrhythmia (atrial fibrillation is the best example of this), there is significant variation in SV that is related simply to the variability in filling time caused by the irregular rhythm and will not reflect volume responsiveness. Current algorithms attempt to assess the respirophasic change in SV from that induced by the cardiac rhythm but have yet to be evaluated rigorously.[33]

Similarly, albeit through a different mechanism, an irregular breathing pattern, ie, spontaneous breathing, both the tidal volume and cycle length may vary from breath to breath. This phenomenon will induce SVV that is not related to volume responsiveness but is instead related to variable changes and time cycling in pleural pressure. In these clinical settings, how is volume responsiveness determined? As long as SV can be measured, this question can be answered simply using either a volume challenge or the passive leg raise (PLR).[25,34–36]

Table 1. Limitations of the Use of SVV and PPV for Fluid Responsiveness

Patient must have mechanically controlled positive pressure ventilation
Respiratory rate (RR) of <35
Tidal volume ≥ 8 ml/kg of ideal body weight
No significant dysrhythmias: HR <150
HR:RR ratio below 3:1
Chest must be closed
Raised intra-abdominal pressure will exaggerate the cardio-pulmonary interaction
Raised intra-thoracic pressure may exaggerate the cardio-pulmonary interaction
No right ventricular failure
Good arterial waveform

Abbreviations: SVV, stroke volume variation; PPV, pulse pressure variation; RR, respiratory rate; HR, heart rate.

IMPROVING OUTCOMES

The importance of precise application of fluid therapy for the critically ill or injured is becoming increasingly recognized as more reports highlight the adverse outcomes related to excessive volume.[37–39] As illustrated in Figure 5, both under-

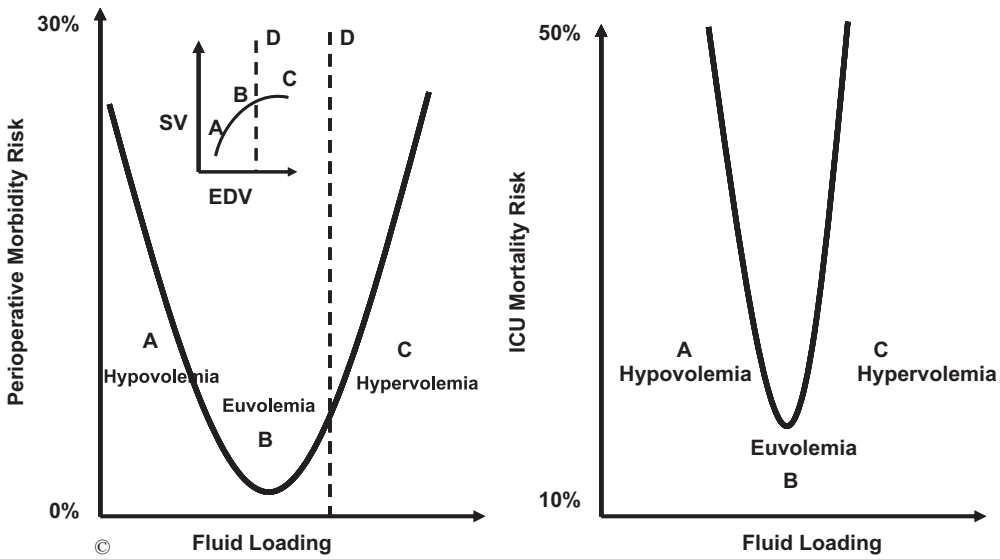

Fig 5. Left side panel shows the Frank-Starling model underlying Stroke Volume Optimization Programs. Unrecognized hypovolemia (A) when corrected by goal-directed therapy programs (B) will result in lower perioperative morbidity. Patients will be moved from left to right along the curve. However, equally important is the prevention of unnecessary volume excess (C). During goal-directed therapy, this is done by stopping the use of volume loading when patients are not volume responsive, hence staying to the left of line D. Right side panel: In the ICU, risk of morbidity and mortality are higher as shown by the difference in the y-axis scale. Margin for error is narrower as the curve shifts from being U-shaped to V-shaped. Precision with regard to volume therapy becomes even more important as physiologic reserve declines. Line D is missing as common volume management practice often proceeds without physiologic guidance. The end result is that outcomes are worse when sicker patients receive volume therapy without physiologic flow-based algorithms. (Color version of figure is available online.)

resuscitation and over-resuscitation are associated with morbidity and mortality in high-risk patients. The margin for error with regard to volume therapy becomes smaller as the patient's comorbidities and severity of illness increase.

A growing body of evidence supports the concept that physiologic optimization of fluid management can minimize these risks. Although this program has not been evaluated rigorously, a physiologically rational approach to perioperative volume management is advocated to both improve outcomes and decrease costs.[7,8] The authors encourage further clinical research to assess physiologic endpoints, clinical outcomes, and costs associated with the application of this program and others that apply individualized goals in the perioperative period.

CONCLUSIONS

Individualized goals based on physiology in the context of specific patient anesthetic technique and surgical procedure now can be obtained easily for most high-risk patients. Achievement of appropriate individualized goals demands clinical skill. Utilization of tested physiologic principles in the care of patients strengthens clinical decision- making. GDT in the perioperative period incorporates the application of a variety of technologies to measure an individual patient's flow or surrogates for flow adequacy relative to demands. At a minimum, interventions should be directed to augment flow to match existing or anticipated demand.[30] Finally, the adequacy of such interventions needs to be tested continually. The POP represents one possible simple approach to perioperative hemodynamic management, incorporating basic physiologic tenets.

APPENDIX A. ILLUSTRATIVE CASE

Part 1

A 70-year-old male, 5'11" and 90 kg, with a history of hypertension, diabetes, and chronic kidney disease (eGFR of 30 mL/min), and a past surgical history notable for exploratory laparotomy for a gunshot wound to the abdomen 35 years ago, was scheduled to undergo laparoscopic surgery for colorectal cancer. He was compliant with his prescribed medications and reported no significant cardiopulmonary restriction of physical activity, although he led a sedentary lifestyle. His cardiologist cleared him preoperatively, finding him fit to undergo the procedure without further testing. He noted that the patient was at intermediate risk for major adverse cardiovascular events.

In preoperative holding, a low thoracic epidural was placed. An arterial line was attached to an arterial-pressure-based cardiac output device. Controlled mechanical ventilation was initiated with tidal volumes of 8 mL/kg predicted body weight. No significant arrhythmias were noted.

The patient underwent placement of ureteral stents by a urologist before the start of the colorectal operation. During this 30-minute period, the stroke volume (SV) was noted to be 70 mL and stroke volume variation (SVV) was 8%. An NPO deficit of approximately 1 liter and an additional 1 liter deficit due to the bowel prep were estimated. Two liters of IV normal saline were infused over the next 30 minutes. Was this appropriate?

SV- and SVV-Guided Avoidance of Inappropriate Volume Therapy

Typical clinical practice during high-risk surgery often includes formulaic volume replacement of presumed NPO deficits based on absolute body weight (for example, weight × NPO time × hourly requirement calculated by the 4:2:1 rule = volume needed for replacement of NPO deficits). Neither NPO deficits nor bowel prep losses need automatic empiric replacement. In this case, the SVV showed the absence of volume responsiveness, and exposing the patient to a volume load merely resulted in loading the interstitium and hemodilution.

Part 2

After 60 minutes, the surgeon made the decision to convert to open laparotomy. The epidural catheter was dosed before incision with 10 mL of 1% lidocaine. Twenty minutes later, the patient was hypotensive; urine output had been 45 mL over the past hour, with SVV 15% and SV 60 mL. House staff decided to volume load with isotonic saline. What are the endpoints that may be used to guide start and stop points for volume therapy?

SV- and SVV-Guided Volume Therapy for Relative Hypovolemia

In clinical practice, urine output traditionally is used as a gauge of cardiac output. It has been shown repeatedly that urine output in the mechanically ventilated patient under anesthesia is unreliable as a predictor of volume status. This problem may be further complicated by hyperglycemia, a common cause of osmotic diuresis. As per this protocol, serial volume loading in bolus fashion may be used, guided by SVV. If the patient remains hypotensive after volume loading with SV that is at least normal and SVV below the threshold, then pressors may be appropriate to reverse the redistribution of blood volume produced by neuraxial local anesthesia.

Part 3

After another 60 minutes of open surgery, blood loss was estimated at 1800 mL, and the patient was hypotensive with SVV 20% and SV 50 mL. House staff decided to volume load. What are the endpoints that may be used to start and stop volume resuscitation?

SV- and SVV-Guided Therapy for Absolute Hypovolemia

Presumed crystalloid:colloid ratios for blood volume replacement (for example, weight × estimated blood loss × 1:1 ratio for colloids or 1:3-4 for crystalloids = volume needed for replacement of blood loss) have been advocated and used with limited evidence to support the rationale. Per this protocol, objective precise decision-making will occur regarding the effects of volume therapy with crystalloids, colloids, or blood products. The authors do not wish to engage in "type of fluid" or "target hemoglobin" debates, but rather just point out that regardless of the specific fluid type, the protocol provides an objective guide to initiate and cease volume therapy.

Furthermore, determination of cardiac output allows oxygen delivery to be targeted when desired.

Part 4

Sixty minutes later, the SVV was 10% and SV was 75 mL, and the patient was hypotensive with lower-than-expected urine output. House staff decided to replace so-called "third-space" losses calculated arbitrarily based on absolute body weight, duration of the procedure, and extent of the surgical exposure. What are the endpoints that may be used to start and stop volume resuscitation?

SV- and SVV-Guided Therapy to Avoid Hypervolemia

Adverse effects of hypervolemia have been shown in the colorectal surgical population. Presumed formulae for replacement of third-space losses stem from theoretic constructs that largely are unsupported by evidence (for example, during a major open abdominal operation, weight × 6-10 mL/kg × hours of surgical exposure = volume needed for replacement of third-space losses). As per this protocol, the effects of unnecessary volume therapy will be avoided.

Part 5

Postoperatively, the patient was taken to the surgical ICU, intubated, and mechanically ventilated. Overnight, urine output fell to under 0.5 mL/kg/h, and the patient showed SVV of 7% and SV 55 mL with hypotension. House staff decided to further volume load the patient. Was this appropriate?

SV- and SVV-Guided Therapy to Augment Cardiac Output

These parameters suggest the need to augment cardiac output by increasing inotropic function. There is no evidence of volume responsiveness and there is a reduction in SV, suggesting reduced inotropism. TEE/TTE may be used at the bedside to evaluate whether there are new regional wall motion abnormalities and ejection fraction.

Part 6

On POD 2, the patient was mechanically still ventilated with evidence of bilateral alveolar infiltrates. Urine output was 0.5 mL/kg/h with SVV of 8% and SV 85 mL. House staff decided to initiate diuresis. Was this appropriate?

SV- and SVV-Guided Therapy to Deresuscitate

A low SVV and higher SV suggest the need for diuresis, with early liberation from mechanical ventilation as a possible benefit. Diuretic therapy is indicated, especially with the presence of a "wet" chest x-ray, and any ongoing "maintenance" fluids certainly should be stopped.

Final Comment

For those conditions in which SVV is not useful in predicting volume responsiveness (Table 1), clinicians simply can follow the change in SV with any of the interventions outlined in this case. Although the ability to predict what will occur is lost, that knowledge of SVV provides clinicians assessing the impact of the interventions on the ultimate outcome attempting to estimate and manipulate (SV) is far superior than common clinical practice—guessing!

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