Review Article

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Intraoperative Hemodynamic Fluid Therapy for Immediate- to High-risk Non-cardiac Surgery: A Narrative Review

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Abstract:

The rates of perioperative morbidity and mortality during major surgery have shown a declining trend due to improvements in hemodynamic monitoring and fluid assessment. However, several million surgical procedures involving aged patients and those with multiple comorbidities are performed every year worldwide. Thus, the establishment and constant re-evaluation of appropriate threshold values of perioperative hemodynamic parameters for the management of immediate- to high-risk patients with a narrow safety margin are especially important. Perioperative fluid balance is an important independent risk factor of postoperative morbidity and mortality. In this article, we provide an overview of intraoperative hemodynamic fluid resuscitation and fluid-response monitoring during non-cardiac surgery. We also focus on targets at the macrocirculatory, microcirculatory, and cellular levels.

Keywords: fluid responsiveness, fluid therapy, hemodynamic monitoring, non-cardiac surgery

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Introduction

The reported incident mortality rate associated with elective surgery in patients under 60 years of age in USA, Europe, and Brazil ranges from 0.4% to 0.6%. However, 30.0–40.0% of the patients undergoing these procedures are at a high risk for postoperative complications and death, and up to 20.0% of them experience severe and possibly life-threatening complications.[1–3] Evidence from large studies suggests that patients with multiple comorbidities tend to show a greater incidence or longer durations of intraoperative hypotension in non-cardiac surgery and higher rates of 30-day mortality.[4] To reduce these complications, perfusion pressure and oxygen delivery should be optimized to maintain adequate cellular metabolism.[5]

Goal-directed therapy (GDT) techniques enable flow monitoring for perioperative fluid management, and the individualized assessment and treatment facilitated by these techniques make them more advantageous than conventional or uniform interventions. Fluid resuscitation is an important part of personalized hemodynamic management for the maintenance or restoration of tissue perfusion using minimally invasive or non-invasive monitoring devices; it allows for the application of precise control, tracking, and observational evaluations of broad patient populations.[6]

Effects of anesthesia and surgery on the cardiovascular system

Although blood pressure (BP) is influenced by cardiac output (CO) and systemic vascular resistance (SVR) and CO is the product of stroke volume (SV) and heart rate, increased CO does not directly result in increased BP. Considering the equation SV = end-diastolic volume (EDV)– end-systolic volume (ESV)[7], the BP response during anesthesia and surgery will be affected by the following four parameters.

Preload

Preload will depend upon the degree to which the constituent cardiomyocytes are stretched, which, in turn, is related to the chamber volume. In clinical practice, the relationship between EDV and cardiac work is difficult to discern since direct measurements of hydraulic work and cardiac volume cannot be obtained easily. Thus, SV and end-diastolic pressure (EDP) are often used erroneously as surrogates for cardiac work and cardiac volume, respectively.[8] Anesthesia induction induces vasodilation and decreases venous return by increasing venous capacitance without any reduction of the intravascular volume or volume stress. A reduction in the mean systemic filling pressure (MSFP) can reduce preload. Moreover, during surgery, fluid loss may occur as a result of bleeding, endothelial glycocalyx destruction, or insensible loss. Some types of surgery that involve pneumoperitoneum induction, such as a laparoscopic surgery with CO_2 insufflation, can also result in reduced preload because of the increased abdominal pressure. Such procedures would require interventions that help predict fluid responsiveness through the identification of fluid-responder patients.[9]

Contractility

The relationship underlying contractility is described by the Frank-Starling curve, in which an increase in the left ventricle (LV) preload is associated with an increase in SV until a plateau is reached; the position of the curve is also affected by ventricular compliance or distensibility, and compliance is also affected by disease. The LV exposed to lifelong pressure overload, e.g., as is the case in chronic hypertension, may show increased wall thickness and altered wall composition (with substantial collagen deposition); in patients with such an altered LV, high EDVs may yield high intracavity pressure but show limited capacity to stretch individual myocytes, potentially decreasing contractility.[8] Almost all anesthetic drugs, such

as volatile anesthetics, opioids, and induction agents, depress heart contractility to varying degrees depending on individual patient characteristics.

The direct measurement of contractility by pressure–volume curves is difficult in the clinical setting. Indirect measurements include the echocardiographic determination of the ejection fraction and the measurement of the CO, SV, and the right as well as the left ventricular stroke work indices in relation to SVR and pulmonary vascular resistance.[7]

Afterload

Afterload is commonly described as the resistance, impedance, or pressure that the ventricles must overcome to eject their blood volumes. In the clinical setting, the most sensitive measure of afterload is the SVR for the LV and the pulmonary vascular pressure for the right ventricle (RV). Anesthesia induction and regional anesthesia influence systemic vasodilation. Since afterload refers to the tension across the ventricular wall, it is influenced by pleural pressure. Thus, while large negative pleural pressures increase afterload and reduce CO, positive pressure ventilation can increase CO.[8] Some surgeries or interventions, such as bone cement implantation or tourniquet release during orthopedic surgery or reperfusion syndrome, induce the release of cytokines, which can also reduce cardiac afterload.[10]

Heart rate

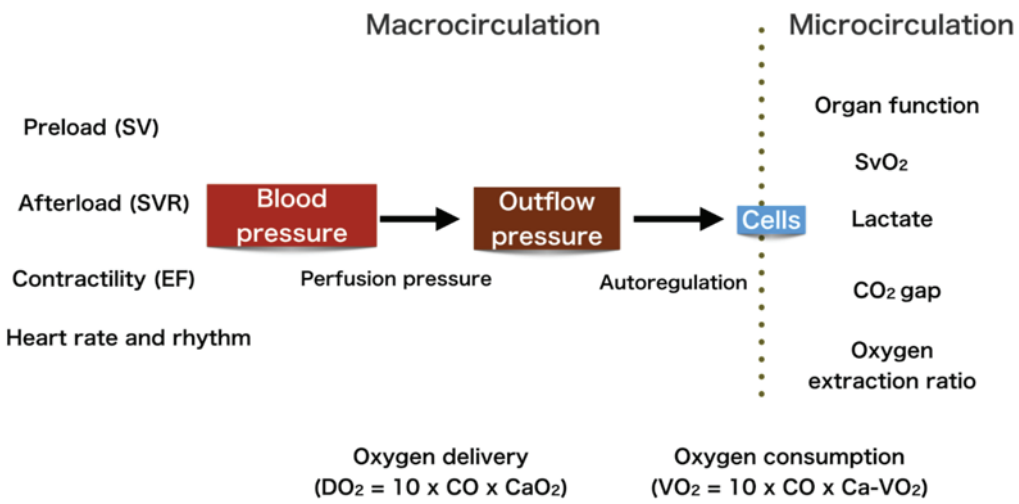
The differences in hormone secretion and autonomic innervation associated with comorbidities, such as diabetes, liver dysfunction, and advanced atherosclerosis, may cause variations in the autonomic response during preoperative volume depletion. Moreover, the depth of anesthesia, peripheral chemoreceptors (e.g., neuromuscular blockade), baroreflex (e.g., opioid use), impaired cardiac contractility (e.g., volatile agent use), or sympatholysis (e.g., intravenous anesthesia) are associated with a change in heart rate. Consequently, hemodynamic parameters, like the ones mentioned above, may not necessarily relate to fluids.[7]

Goal of intraoperative fluid therapy

Fluid therapy, in which combinations of vaso–constrictors and inotropic agents are used to enable normal macrocirculation, microcirculation, and cellular metabolism (Figure 1), is an important part of intraoperative hemodynamic management. Pressure, volume, and flow are related in dynamic systems (as represented by the elastance and capacitance of vessels). Currently, due to limitations in the measurement of microcirculation and cellular–level flow in clinical settings, the first step of therapeutic fluid management is restoration of macrocirculation. However, macrocirculatory variables, such as CO, SV, mean arterial pressure (MAP), central venous pressure (CVP), and heart rate, show poor sensitivity in indicating the presence of risks for microcirculation or cellular dysfunction.[9,11] Therefore, although restoration of macrocirculation cannot guarantee normal microcirculation and cellular fluid levels, especially in hemodynamically incoherent conditions, a hypervolemic status caused by excess fluid administration may induce cellular oedema, which is a manifestation of hemodynamic incoherence.

Relationship between pressure and flow

The maintenance of perfusion is essential to life. Although the maintenance of an adequate global blood flow can be achieved via a wide range of different pressure/resistance combinations, this is not universally true when specific organs are considered. The phenomenon of the autoregulation of flow refers to the ability of different vascular beds to maintain a constant regional flow across a restricted range of pressures through adjustments of local resistance. Thus, a combination of metabolic and arterial myogenic/neurogenic mechanisms can maintain constant



CO=cardiac output, CaO_2=arterial oxygen content, Ca-VO_2=arterial-venous oxygen content difference, CO_2=carbon dioxide, EF=ejection fraction, SV =stroke volume, SVR=systemic vascular resistance, SvO_2=mixed venous oxygen saturation

Figure 1 Hemodynamic variables that serve as targets for hemodynamic management

cerebral blood flow across MAP values ranging from approximately 60 to 140 mmHg. This range is influenced by various neuroendocrine and biochemical factors and by the disease state. Similarly, for the renal vasculature, a combination of myogenic mechanisms and tubuloglomerular feedback can maintain a constant flow until the MAP falls below approximately 70 mmHg.[7,8] A loss of coronary circulation autoregulation at an MAP of 50–55 mmHg has been reported to associate with myocardial ischemia and acute kidney injury in patients showing a few minutes of intraoperative hypotension below this level.[12] Therefore, to ensure organ perfusion, the intraoperative BP should be maintained above 65–70 mmHg, except in cases with other limiting conditions such as chronic hypertension, stroke, or critical illness.[13]

The differences between MAP (i.e., the inflow pressure) and outflow pressure determine the tissue perfusion pressure. The outflow pressure differs across organ systems; it can even differ in the same organ in the same patient over time. For example, the outflow pressure for the brain is either CVP or intracranial pressure (whichever is higher).[7,14]

Relationship between pressure and volume

Cardiac performance according to the Frank-Starling curve varies among individuals, and the relationship between preload and volume (steep or plateau regions) can enable the identification of fluid responders. Static parameters, measured under a single ventricular loading condition, are presumed to provide a reliable estimate of the preload of one or both ventricles. However, these parameters have poor reliability for identifying fluid responders.[13,15] Moreover, conservative fluid challenge tests cannot distinguish between fluid responders or nonresponders, and the hypovolemic and hypervolemic statuses in these tests may harm sensitive patients. An unsuccessful fluid challenge test result does not significantly increase CO, which might decrease oxygen delivery due to hemodilution.[16] Dynamic parameters are used to determine whether the findings for a specific patient are on the ascending or flat portion of

the Frank–Starling curve. Several approaches can be used to ascertain the functioning preload/SV relationship in the ventricle, facilitating the diagnosis of preload dependence or independence. Typically, the findings for fluid responders will be located in the steep part of the Frank–Starling curve, with an increase in EDV and an increase of more than 10.0–15.0% in CO.[12,13] Moreover, factors causing changes in venous capacitance or tonicity, such as vasopressor administration, can increase the volume or preload through elevations in volume stress.[10,16]

Static parameters

Static parameters, such as CVP and its surrogates, are influenced by multiple factors that are not related to the fluid status or fluid responsiveness, including cardiac compliance, intra-abdominal pressure, airway pressure, and positive end-expiratory pressure (PEEP). Therefore, static parameters should not be used to determine the need for additional fluid requirements.[14–16] However, CVP reflects right atrial pressure (RAP), which indicates EDP; therefore, an increase in EDP due to fluid challenge should result in a reduction in venous return.[17–20] Many studies have observed that fluid challenge is associated with an elevated CVP in responders, resulting in an increase in the gradient of venous return and CO.[18,21] Greater changes in CVP during fluid challenge without CO monitoring may effectively indicate fluid requirements; however, patient responsiveness to fluids will still remain unknown since this factor also depends on cardiac tolerance.[19] In clinical settings, where CO monitoring is not easily available, extreme CVP values may be more helpful to guide fluid administration than intermediate values.[19,22] In an earlier study, a positive response to fluids was observed when CVP values were less than 6 mmHg, but it was unlikely when values were greater than 15 mmHg.[23] Nevertheless, the obviously lower risk of edema in patients with a low CVP implies that the administration of fluids to a nonresponsive patient with a low CVP is less risky than that to patients with a high CVP.[19,23,24]

Dynamic parameters

The dynamic preload parameters can be used to predict fluid responsiveness due to their better accuracy and lower positive fluid balance. The parameters representing the heart–lung interaction, including the pulse pressure variation (PPV) and the stroke volume variation (SVV), can be easily evaluated intraoperatively in intubated patients receiving controlled ventilation; they have been reported to show area under the curve (AUC) values of 0.94 and 0.84, respectively.[25,33] The cut-off value for SVV is between 10.0% and 12.0%.[26,27] Although parameters based on the heart–lung interaction, such as PPV and SVV, can predict fluid responsiveness with good reliability, they are invalid in patients undergoing open chest procedures or low-volume ventilation and those with low heart rate/respiratory ratios, increased abdominal pressure, or cardiac arrythmias.[24,26] One retrospective study showed that the use of non-supine positions, including the prone and Trendelenburg positions, as well as the lack of preoperative β-blocker administration were independent factors altering PPV and systolic pressure variation (SPV).[28,29] Therefore, the tidal volume challenge test was developed to predict fluid responsiveness in patients showing limitations in heart–lung interactions and those in the grey zone of PPV (between 9.0% and 13.0%), by monitoring changes in the PPV or SVV.[28] This test was found to be useful, especially in patients requiring lung-protective strategies such as those with acute respiratory distress syndrome.[17,30] One study reported that increasing the tidal volume from 6 to 8 mL/kg could change the PPV by 2.5% and the SVV by 3.5%, with AUC values of 0.99 and 0.97, respectively.[25] The evaluation of patients in the Trendelenburg position in robot-assisted laparoscopic surgery has shown the tidal volume challenge test to be reliable, and the reliability of this test was even confirmed

in neurosurgical patients in the prone position; however, evaluations involving patients in the extreme Trendelenburg position are quite limited.[31,32]

Continuous CO monitoring based on calibrated and a non-calibrated pulse-wave analysis has been developed to facilitate the co-evaluation of fluid responsiveness with tests such as the passive leg rising test (PLRT), end-expiratory occlusion test (EEOT), and mini-fluid challenge test. PLRT has an autotransfusion of approximately 300 mL of blood and is accurate in conditions involving an increased CO of more of than 10.0% (sensitivity, 86.0%; specificity, 92.0%).[33,34] Additionally, PLRT is not dependent on sinus rhythm or low tidal volume ventilation. On the other hand, the procedure is contraindicated in conditions with limited leg movement, stimulation of sympathetic activation, and an intra-abdominal hypertension of more than 16 mmHg.[20,34]

EEOT, which was introduced by Monnet et al., is based on the influence of the deep inspiration of a cardiac preload within 15 s of end-expiratory occlusion in mechanically ventilated patients, which can induce a change of 5.0% in CO.[35,36] Low-volume ventilation was evaluated in systematic reviews with a volume of <7 mL/kg (ROC 0.96) and a PEEP level of 4-14 cmH_2O (median, 7 cmH_2O), and its accuracy to predict fluid responsiveness and the type of CO monitoring were not shown to be different.[36,37] Hence, further studies are required to evaluate the accuracy of EEOT in other positions beside the supine position in the operating room.

The mini-fluid challenge test involves increasing CO by a 10.0% threshold via the velocity time integral (VTI) obtained via echocardiograms or continuous CO monitoring, with the smallest colloid volume of 100 mL.[38]

Pulse oximetry can be universally performed in patients during an operation and is useful in patients receiving intubation and controlled ventilation. The Pleth variability index (PVI) is a parameter that allows for the continuous and automatic estimation of respiratory variation in the pulse oximeter waveform amplitude.

In cases with a lack of continuous CO monitoring and PVI, novel methods such as the evaluation of end-tidal carbon dioxide concentration ($P_{ET}CO_2$) are used to predict fluid responsiveness during PLRT, with the $P_{ET}CO_2$ changing by ≥5.0% (sensitivity, 71.0%; specificity, 100%) in response to a provoked PEEP challenge from 5 to 10 mmH_2O for 1 min.[39] According to another study, the exhaled CO_2 volume also decreased by 11.0% (sensitivity, 0.9; specificity, 0.95), but this measurement was not reliable when assessed via the mini-fluid challenge test.[35]

Concerning one-lung ventilation, an observational study reported the use of a lung recruitment maneuver prior to thorax closure. During this procedure, one breath transition was held from one- to two-lung ventilations at 30 cmH_2O for 10 s and then repeated three times, and changes in the MAP served as the most successful predictor of fluid responsiveness (AUC=0.852; optimal threshold, 9.5 mmHg).[40,41] This was similar to SVV decreasing by 30.0% during the lung recruitment maneuver (continuous airway pressure of 30 cmH_2O for 30 s) under neurosurgery, without intracranial hypertension, indicating the predicted fluid responsiveness.[40] However, recruitment maneuvers should be performed cautiously in patients with low CO because they increase the afterload in the right ventricle, which can be especially deleterious in such patients.[40,42]

RV dysfunction is associated with a high risk of mortality in non-cardiac surgery, and only a few studies have evaluated fluid responsiveness in association with this condition. Transthoracic or transesophageal echocardiography can be used to monitor RV dilation, RV systolic function, LV function, and the central vein, and in selected cases, monitoring pulmonary artery pressure can be useful.[43–45]

During intraoperative hemodynamic management, not all patients who show fluid responsiveness require fluids; for example, patients with adequate or high SV or CO may not require fluid administration even if they are fluid responders. Assessments of fluid responsiveness should

be a part of personalized hemodynamic management with vasopressor and inotropic drugs, with the ultimate aim of improving postoperative pulmonary complications.

Relationship between macrocirculation, microcirculation, and cellular fluid levels

Disruptions in the hemodynamics of microcirculation and cellular fluid levels can influence the balance of oxygen delivery and oxygen consumption even when BP, preload, afterload, and contractility are stable and normal. Microcirculation can be assessed by direct and indirect methods such as intravenous microscopy, laser Doppler flowmetry, or tissue oximetry. The biomarkers of the tissue perfusion include lactate and oxygen levels, oxygen extraction ratio, central venous oxygen saturation, and carbon dioxide gap (Figure 1). Clinical examinations can also involve the assessment of capillary refill time or skin mottling.[11,46] Moreover, similar to the difference between arterial and venous blood flow, which drives organ blood flow, the differences between post-arteriolar and venular pressure drive the microcirculation flow; thus, microcirculation flow can decrease when venous pressure increases.[9,10]

These parameters should be checked when the patients show a critical state and adjusted accordingly to ensure an optimal oxygen delivery balance and oxygen consumption. Although these parameters have been used in the management of patients with sepsis, studies on their perioperative outcomes in non-cardiac surgery are limited.[11]

Conclusion

Standard fluid resuscitation approaches cannot improve circulatory instability in all intermediate- to high-risk surgical patients because of individual differences in cardiovascular physiology. Thus, the complications in these patients can be reduced by the careful evaluation of fluid responders as a part of personalized hemodynamic monitoring. However, there is no single monitoring approach that can comprehensively identify the spectrum of pathophysiologic changes in these patients. Finally, further studies are needed to evaluate the point-of-care correlations among macrocirculation, microcirculation, and cellular metabolism in the perioperative guidance of fluid therapy.

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