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Review Article

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Management of perioperative volume therapy – monitoring and pitfalls

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Over 300 million surgical procedures are performed every year worldwide. Anesthesiologists play an important role in the perioperative process by assessing the overall risk of surgery and aim to reduce the risk of complications. Perioperative hemodynamic and volume management can help to improve outcomes in perioperative patients. There has been ongoing discussion about goal-directed therapy. However, there is a consensus that fluid overload and severe fluid depletion in the perioperative period are harmful and can lead to adverse outcomes. This article provides an overview of how to evaluate the fluid responsiveness of patients, details which parameters could be used, and what limitations should be noted.

Keywords: Cardiac output monitoring; Colloids; Crystalloid solutions; Hemodynamic monitoring; Hypotension; Volume therapy.

Introduction

Anesthesiologists play an important role in the perioperative process by assessing the overall risk of surgery, including the risk factors of the surgical procedure and those of the patient. There have been substantial developments over the last few decades regarding the risk of anesthesia. In the early days of anesthesia, the risk of the anesthetic procedure itself was high and led to a number of fatalities [1,2]. This has changed dramatically, and these days anesthesia-related deaths are extremely low. Such advances are likely related to drug advancements, an improved understanding of physiology, and better monitoring and management. However, death after surgery, including the whole perioperative process, has not substantially declined. The risk of death during the 30 days following surgery is 1,000 times more likely than during surgery itself [3]. According to recent data, death within 30 days of surgery is the third leading cause of death in the United States of America, behind cardiac diseases and malignancy [4].

Some recent studies have assessed this risk in detail. According to Weiser et al. [5], it is estimated that more than 310 million surgical procedures are performed worldwide. The exact complication rate and risk of death after these procedures are largely unknown. However, it is estimated that depending on the type of surgery and patient's comorbidities that about 30–40% of patients develop complications, of which up to 20% are severe and possibly life-threatening [6–8]. Besides, the mortality risk of surgery is not precisely known. It has been estimated that between 3 and 12 million patients die worldwide after surgery. The European Surgical Outcome study evaluated this in detail in a seven-day cohort study that included 46,539 patients from 498 European centers. Patients were followed for up to 60 days after their surgical procedure. Of this cohort, 1,855 patients died while in hospital, giving an in-hospital mortality rate of 4.7%. Additionally, the study

found that there was substantial variation in the risk-adjusted mortality between European centers. These results highlight an opportunity to learn from those centers with lower risk-adjusted mortality rates to improve patient outcomes in other centers [9].

Perioperative hemodynamic and volume management are important considerations in improving outcomes in perioperative patients. There has been ongoing discussion on which fluid should be used, and at what rate it should be administered to particular patients. There is a consensus that fluid overload and severe fluid depletion in the perioperative period is harmful and leads to adverse outcomes [10]. Unfortunately, adequate management of volume therapy is challenging and requires additional testing and monitoring that is seldom used in clinical practice, even in high-risk patients [11,12].

Physiology of volume replacement

Adequate fluid and volume therapy during and after anesthesia is important for improving perioperative outcomes. Without a doubt, the most common intervention done by anesthesiologists is prescribing fluids. Fluids are important as normovolemia is an essential factor of hemodynamic stability and homeostasis between the intravascular fluid and extravascular space. However, the traditional concept to give fluids where hemodynamic compromise is recognized (e.g., hypotension), following the principle "in doubt give volume", has been proven to be incorrect [13]. Notably, in abdominal surgery, the concept of "restrictive" fluid therapy that was introduced in the early 2000s was quite successful and led to better outcomes compared to the traditional liberal volume therapy [14]. In particular, complications that were associated with fluid overload like pulmonary edema, anastomotic leakage, anemia, coagulopathy, and cardiovascular compromise dramatically reduced, which led to better outcomes overall. However, in further studies, "restrictive" and "liberal" were not well defined, and what was considered restrictive in one study was deemed liberal by others [15]. In some studies, an extremely restrictive approach led to severe hemodynamic compromise with decreased perfusion, decreased oxygen delivery, and complications like acute kidney injury (AKI) [16].

Fluid overload is recognized as being harmful. Unfortunately, fluid overload is common, silent, and deadly. Bellamy [17] put together a theoretical framework based on their concept that there is a U-shape relationship between fluid therapy and outcome. Excess fluid overload and severe fluid restriction can both lead to adverse outcomes. Therefore, anesthesiologists need to find a balance and ascertain the ideal volume status for individual patients. This is termed normovolemia (Fig. 1). Two retrospective studies

recently showed that fluid overload and hypovolemia are associated with unfavorable outcomes such as AKI, pulmonary complications, and even mortality [10,13]. Therefore, it is important to recognize the need for fluid in some patients and deresuscitation in others. Thus, we need to clearly define our aim when giving patients fluids. Do we want to expand the extracellular space to compensate for losses, or do we want to increase the intravascular space to improve the filling pressures and potentially cardiac output (CO)? In this review, only volume therapy, giving additional fluids to improve hemodynamic parameters, will be discussed. Fluid therapy, which is mostly used to compensate extravascular losses and regain fluid homeostasis in internal medicine patients, is beyond the scope of this article and will not be discussed.

Before giving patients additional fluid, we need to ascertain whether the issue can be solved by increasing stroke volume and cardiac output. However, usually, we do not want to increase only cardiac output. In most cases, we aim to increase oxygen delivery to the tissues. However, to achieve this, global oxygen delivery needs to first be increased. Currently, we cannot be sure that this will also lead to increased oxygen delivery to individual tissues as monitoring of the microcirculation, while possible, has not gained general acceptance in clinical practice [18]. In a review article, Monnet and Teboul [19] detailed all of the circumstances in which a volume bolus will lead to increased tissue perfusion and function. The first step is to increase the mean systemic filling pressure, which can be counteracted by capillary leakage and ven-
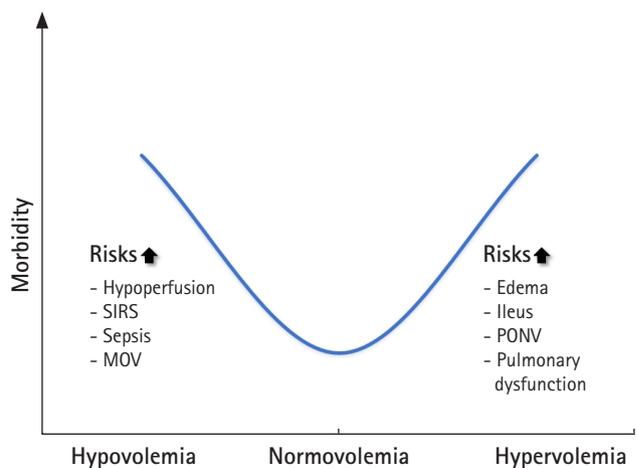
Fig. 1. Relationship between morbidity and hypo- normo- and hypervolemia. Bellamy [17] developed a theoretical framework based on their concept that there is a U-shape relationship between fluid therapy and outcome. Therefore, prevention of hypo- and hypervolemia by goal-directed fluid management is essential. SIRS: systemic inflammatory response syndrome, MOV: multiple organ failure, PONV: postoperative nausea and vomiting.

odilatation. The role of making artificial and natural colloids more effective so as to increase the mean systemic filling pressure has been extensively discussed. However, as yet, no conclusions have been reached. Nevertheless, colloids appear to be more able to achieve this with less fluid and a longer intravascular half-life. Therefore it is only moderately surprising that colloids, including starches, were used in 86% of the included studies in a review of fluid boluses [20]. This was done despite several studies showing that in critically ill patients, the use of colloids, and in particular starches, can result in an increased risk of renal failure and death [21–23]. However, a recent meta-analysis did not confirm these findings in surgical patients [24,25]. It is paramount that the patient is fluid responsive and that the stroke volume can be increased by additional fluid loading, given that the aim is to increase global oxygen delivery. After fluid loading, not all patients that have increases in their stroke volume and cardiac index shows better microcirculatory flow and increased oxygen consumption. Further research is needed to identify the mechanisms for uncoupling the global perfusion indices from regional indexes and identifying suitable treatment algorithms.

As optimized global perfusion is a prerequisite to optimizing microperfusion, in the following section, methods to assess the need for intravascular volume therapy to increase stroke volume will be critically discussed.

How to assess fluid responsiveness

The concept of determining the treatment effects of therapies is not new. If we administered vasopressors and did not measure arterial blood pressure before and after the intervention, we would be accused of malpractice. However, when we administer fluids during surgery, the verification of a positive drug effect, and the decision to give fluids is often made with little testing or indications. Almost a hundred years ago, Prof. Jarisch [26] asserted that our understanding of circulation was limited as while blood pressure is easily measured, blood flow is not. This is why blood pressure monitoring is so prevalent despite most organs requiring blood flow, not pressure. However, if we do not measure blood flow, how can we know that additional volume given to patients is actually increasing blood flow? A recent study by Cecconi et al. [27] tried to elucidate what drives the decision to give additional volume to intensive care unit (ICU) patients. In 42.7% of the patients, no testing of fluid responsiveness took place, and the decision was only based on clinical experience. In another 35.5%, the decision was based on static parameters like central venous pressure (CVP) or atrial blood pressure that we will discuss below. The second most interesting finding of this study was that despite

the results of testing, about 50% in all groups (positive, negative, and uncertain) received additional fluids.

Pressure based volume therapy – arterial blood pressure, CVP

Generally, in recent years, a large amount of fluids has been given to patients undergoing surgery, especially when there was some sort of hemodynamic deterioration like hypotension. The idea behind this was that a "liberal" policy of fluid management in surgical patients is required. This concept is based on ideas and studies from Tom Shires, Chief of Surgery at the University of Texas Southwestern, Dallas, Texas [28]. His work led him to conclude that an extracellular fluid deficit in surgical patients and the consequent elevations of aldosterone and antidiuretic hormone is caused by extravasations of fluid from the extracellular compartment to the third space along with evaporative losses [29,30]. A strategy of aggressive fluid replacement emerged as the mainstay of perioperative care to compensate for these losses [31,32]. However, hypotension can occur quite often during surgical procedures, and in many cases, hypotension is not linked with hypovolemia. Intraoperative hypotension has been studied for many years. Therefore, it is surprising that there is still no clear definition of intraoperative hypotension. In a review by Bijker et al. [33] of 130 studies, 140 different definitions of hypotension were described. Risk factors for hypotension besides hypovolemia are increased age, a higher American Society of Anesthesiologists score, induction medication that might lead to vasodilatation, and neuro-axial anesthesia. Also, during different time frames of anesthesia, various risk factors have been published, describing post-induction hypotension, early intraoperative hypotension, and late intraoperative hypotension [34,35]. Hypovolemia is only one potential cause of hypotension. Therefore, any given arterial blood pressure cannot be used to decide whether additional fluid should be provided to a patient to increase cardiac output. Nevertheless, hypotension, in conjunction with the wider clinical picture, can help to find an indication to give fluid. In polytraumatized patients with ongoing bleeding, the first step is to give fluids. However, during procedures, it is not possible to tell when resuscitation is complete, and normovolemia is reached just by measuring the arterial blood pressure.

Another option might be to measure venous filling pressures like CVP or pulmonary artery occlusion pressure (PCWP). The measurement of filling pressures was long advocated for in many guidelines, such as the surviving sepsis campaign [36]. This guideline recommended that patients should receive additional fluids to optimize perfusion until their CVP was 8–12 or 12–15

cmH$_2$O, if mechanically ventilated. Unfortunately, this has been proven to be incorrect. Filling pressures like CVP and PCWP are influenced by many other factors that are not related to the fluid status or fluid responsiveness such as cardiac compliance, intra-abdominal pressure, airway pressure and positive end-expiratory pressure (PEEP), pulmonary vascular resistance, and cardiac pathologies such as mitral/tricuspid regurgitation and congestive heart failure. Extensive research, including several meta-analyses, have been conducted on this subject and have concluded that CVP and PCWP should not be used to decide whether to give additional fluids [37,38].

Nevertheless, there is some value in measuring the CVP curve. Recent work that focused on different waves of the CVP curve found some association with preload dependence compared to no preload dependence [39,40]. However, this work must still be viewed as preliminary, particularly as no study has tested these findings with a large number of patients using a multicenter approach. Yet, the absolute number of CVP might also play an important role. Even when an absolute number of CVP does not preclude fluid-responsiveness, it can be used to assess the risk of adverse outcomes. As the CVP is the "zero-mark" of the cardiovascular system, it plays an important role in venous return and microcirculation. Therefore, severely elevated CVP values can be used as a symptom of fluid challenges, even in patients who remain fluid responsive [41]. It has been shown that CVP values over 15 mmHg are associated with increased rates of unfavorable outcomes like AKI [42].

Therefore, in these patients, CVP can be used as a marker of when to stratify increased risk versus the benefits of further fluid loading.

Stroke volume-based volume therapy

One easy method to test whether stroke volume can be increased through fluid loading is to give patients a defined volume bolus and measure it before and after the intervention. This concept is based on the physiological framework of Frank and Starling. Until a certain cut-off regarding the preload of the left ventricle, it can increase its stroke volume. Therefore, only patients that are below this cut-off should receive additional fluids, and this is best estimated by using the steep part of the Frank-Starling curve. Small increases in preload will lead to relatively large increases in stroke volume. Unfortunately, this cut-off varies between people and can also change during different loading conditions. This is especially troublesome as, therefore, all static parameters like filling pressures (CVP and PCWP), and volumetric measures such as global end-diastolic volume cannot provide a specific cut-off

number for fluid responsiveness.

A fluid challenge is a maneuver in which a defined bolus of fluid is given within a short time frame. In most cases, this is an artificial colloid. In a recent review, it was asserted that the bolus is relatively standardized within the goal-directed hemodynamic therapy (GDT) literature, and is 250 ml [20]. In 86% of the studies, a colloid was used. It is important that the fluid bolus is given relatively rapidly so that it can stretch the right ventricle to detect an increase in stroke volume in responders. Therefore, most authors apply the bolus within 5–10 minutes or less. If the bolus is too small or given too slowly so that an acute increase of the right ventricular end-diastolic volume is not reached, there is a risk of a false negative test. Most authors recommend measuring stroke volume before and after the fluid challenge. An increase in stroke volume of at least 10–15% is considered a positive response [43]. Theoretically, any device that can measure stroke volume could be used. However, most studies use uncalibrated pulse wave analysis technology.

A fluid challenge is included in many algorithms used to optimize hemodynamics, also called hemodynamic GDT [44]. One of the simplest algorithms is to measure stroke volume, give a fluid challenge, and repeat this until the stroke volume no longer increases by more than 10%. These simple algorithms are easy to follow with high implementation rates. However, if the trigger is hypotension, repeated negative fluid challenges, especially in the ICU, can lead to a substantial positive fluid balance. An unsuccessful fluid challenge does not significantly increase stroke volume and, therefore, might decrease oxygen delivery due to inherent hemodilution if blood is not used for the fluid challenge.

Volume therapy based on dynamic parameters

Another way to optimize the fluid status of patients is by using dynamic parameters like stroke volume variation (SVV), pulse pressure variation (PPV), or pleth variability index (PVI). The dynamic preload parameters, SVV and PPV, are based on changes in the arterial pressure waveform due to changes in stroke volume in relation to positive pressure ventilation. The PVI is an algorithm that allows for the continuous and automatic estimation of respiratory variations in the pulse oximeter waveform amplitude to assess fluid responsiveness. To use these parameters for GDT, it is mandatory to continuously measure the blood pressure or the pulse oximeter waveform amplitude. Today there are a variety of technologies available that can measure this invasively and non-invasively. Various studies have shown that SVV and PPV are better predictors of fluid responsiveness than the static parameters CVP, PCWP, and mean arterial pressure (MAP). SVV (area under

the curve [AUC] 0.84) and PPV (AUC 0.94) are good predictors of fluid responsiveness with clinically acceptable levels of sensitivity (0.82 and 0.89) and specificity (0.86 and 0.88) [45]. The cut-off for SVV has been published to be between 10% and 12% [46]. Benes et al. [47] investigated the hemodynamic goal-directed protocol based on SVV in high-risk surgery patients undergoing an elective abdominal operation. The results showed that the GDT-group had better intraoperative hemodynamic stability, a decrease in serum lactate at the end of the surgery, and a lower incidence of postoperative organ complications in comparison with the control group.

Scheeren et al. [48] investigated a combination of SVV and stroke volume optimization in 64 high-risk surgery patients, which were divided into two groups. The primary outcome measure was the number of postoperative complications. The authors could show that an SVV and stroke volume optimization protocol is feasible and can decrease postoperative wound infections. The number of patients with at least one complication (46% vs. 62%) and the number of postoperative complications per patient tended to be lower in the study group.

Other studies investigated PPV as a goal for GDT. The best cut-off value for predicting fluid responsiveness has been published to be between 10% and 15% [49]. Salzwedel and colleagues [50] performed a multi-center study in 160 patients undergoing major abdominal surgery and showed that hemodynamic GDT using PPV, cardiac index trending, and MAP led to a significant decrease in postoperative complications.

Even though the dynamic parameters are better predictors of fluid responsiveness, they have some significant limitations. First, the patient needs to be mechanically ventilated without spontaneous breathing. The published cutoffs in fluid responsiveness for SVV and PVV were validated in patients with a tidal volume > 8 ml/kg. So, if the patient is ventilated with a lower tidal volume, the patient may be false negative for volume responsiveness. Another limitation is that it can display a slow heart rate/respiratory ratio. In patients with extreme bradycardia or high respiratory rate (e.g., high-frequency ventilation), the results may be falsely negative for predicting fluid responsiveness. Another special situation is patients undergoing open-chest procedures. In such situations, the PPV (AUC 0.55) and SVV (AUC 0.49) show a low predictive power and should also be used with caution because the results may be falsely negative [51]. In spontaneous breathing patients and patients with arrhythmia, dynamic parameters cannot be used as ventricular filling depends on the variation of diastolic filling in severe arrhythmia, and there is no controlled stimulus in spontaneous breathing patients.

Volume therapy based on physiologic testing

Passive leg raising (PLR)

Widely known for treating acute circulatory failure, passive leg raising (PLR) has gained increasing interest in the perioperative prediction of fluid responsiveness. PLR is a safe method for reversible and rapid autotransfusion of approximately 300 ml of blood without the need for further fluid boli [52,53]. Since the accuracy of PLR is not dependent on a sinus rhythm or high tidal volume, ventilation can also be applied when dynamic preload parameters are not viable. On the other hand, surgical procedures which are not compatible with the movement of legs or the Trendelenburg position (e.g., neurosurgery, orthopedic surgery of the lower limbs) represent relative contraindications for PLR.

Even though most studies investigating PLR derive from critically ill patients, the predictive value can also be assumed for perioperative patients. A meta-analysis that summarized 23 studies investigating the diagnostic accuracy of PLR (measured with flow-based hemodynamic monitoring tools) showed that the pooled sensitivity of PLR was 86% (95% CI: 79%–92%), while its specificity was 92% (95% CI: 88%–96%). This shows its high diagnostic performance in predicting fluid responsiveness [54]. A second systematic review of 991 patients was able to confirm these findings but emphasized the need to measure CO as a target parameter in order to achieve reliable results [55]. It must be highlighted that PLR can be used to decide whether fluid therapy is needed or not. However, even though its practical implementation appears to be simple, some pitfalls have to be thoroughly considered to increase its predictive accuracy. Monnet and Teboul [53] summarized these as; The measurement starts from the semi-recumbent and not from a supine position and should target CO or its indices as opposed to blood pressure. CO can be assessed with different devices (e.g., echocardiography, pulse contour analysis), but it is of high importance that the measurements can detect rapid changes (< 1 min). Furthermore, the procedure does not end by the patient´s reposition but with a postinterventional observation period until the hemodynamic situation has been normalized. The depth of anesthesia should be appropriate to avoid sympathetic activation, and adrenergic stimulation blurring the effect of PLR. If these factors are taken into consideration, PLR can be considered a powerful diagnostic tool for predicting perioperative fluid responsiveness and is recommended by several international guidelines [56–58].

End-expiratory occlusion test (EEOT)

A decade ago, Monnet et al. [59] introduced EEOT. The underlying principle of EEOT is based on the influence of deep inspira-

tion on cardiac preload. By carrying out a short (15–30 seconds) end-expiratory occlusion in mechanically ventilated patients, CO is impaired while the atrial filling is simultaneously facilitated, leading to an increase of ventricular stroke volume. To receive a reliable prediction of fluid responsiveness, continuous CO measurement is necessary during EEOT [60]. Furthermore, an EEOT-induced change of 5% of CO is generally accepted as proof of fluid responsiveness [60]. Pulse contour analysis superiorly performs to echocardiography in terms of the precise detection of CO changes during EEOT [61]. However, other devices such as echocardiography, non-invasive CO measurements, and Doppler-based methods are feasible but need more confirmative studies [20,62,63].

EEOT imitates a fluid challenge without the need for fluid application. In contrast to the PLR test, the patient does not need to be moved, making it an attractive solution for surgery. Its predictive value was confirmed in several studies for patients ventilated with tidal volumes ≥ 8 ml/kg. However, its accuracy in patients ventilated with smaller tidal volumes is still being debated [20,59,60,64–66]. Most studies investigating EEOT under low-tidal volume ventilation derive from an intensive care setting and cannot be directly transmitted to surgery [65,67]. Only one study involving neurosurgical patients directly compared the effects of low- to regular-tidal volume ventilation on the accuracy of EEOT, and showed a very low predictive value of EEOT under low-tidal volume ventilation (AUC of the change of cardiac index 0.53 [95% CI: 0.35–0.71]) [68]. Guinot et al. [66] published the only study showing a low predictive value of EEOT for fluid responsiveness under sufficient tidal volumes (of 8.2 ml/kg) in a heterogeneous study of surgical patients. The reason for these findings remains unclear, but differences between the perioperative and intensive care ventilation strategies might be a factor. However, it has been shown that the level of PEEP does not affect the reliability of EEOT [69].

Novel physiological tests for predicting fluid responsiveness

End-tidal carbon dioxide concentration ($P_{ET}CO_2$) is a surrogate for CO and is well-known for detecting successful cardiopulmonary resuscitation. It has been shown that $P_{ET}CO_2$ directly correlates to CO and can sufficiently predict fluid responsiveness when combined with PLR testing [70,71]. Tusman et al. [72] introduced a further method based on volumetric CO_2 measurements by quantifying the amount of exhaled CO_2 instead of the concentration. To detect a lack of intravascular fluids, the patient´s fluid responsiveness was provoked with an elevation of PEEP from 5 to 10 cmH$_2$O for one minute. During this, patients were monitored with volumetric capnography and pulse contour

analysis. Afterward, patients received 500 ml of crystalloids, and the measurements were repeated. A decrease of exhaled CO_2 volume during the PEEP challenge was predictive of fluid responsiveness. Furthermore, a ROC-analysis revealed a high predictive performance that was superior to the change of end-tidal CO_2 concentration and PPV. Even though this method is only available in ventilated patients, it offers a non-invasive and accurate approach for predicting fluid responsiveness that is worthy of further validation [72,73].

In 2005, the respiratory systolic variation test (RSVT) was introduced for predicting fluid responsiveness in surgical patients [74]. The RSVT is performed through the application of three consecutive inspiratory breaths with increasing peak inspiratory pressures (of 10, 20, and 30 cmH$_2$O) and the simultaneous detection of the three lowest systolic arterial pressures. Next, this blood pressure is correlated to the peak pressure of the inspiratory breath, resulting in an RSVT slope. The slope corresponds to the Frank-Starling curve-enhancing a physiologic comprehension of the fluid challenge and is comparable to PPV and SVV in predicting fluid responsiveness [74,75].

Deresuscitation strategies using monitoring of volume status

Originally, the term deresuscitation was used to describe a strategy that aimed to treat fluid overload following the resuscitation and stabilization of critically ill patients. Fluid therapy is used to restore the intravascular volume homeostasis to achieve sufficient tissue oxygenation and can be characterized by the Resuscitation, Optimization, Stabilization, and Evacuation (ROSE) concept [76,77]. First, patients must be resuscitated from circulatory shock (resuscitation). To avoid adverse outcomes associated with fluid overload, fluid responsiveness should be guided by validated tests such as PLR and EEOT (optimization and stabilization). After stabilization of the patient´s hemodynamic status, de-escalation should be considered early and monitored with tests for fluid responsiveness (evacuation) [76]. The goal of the evacuation, deresuscitation phase, is to restore the patient´s physiologic homeostatic intravascular balance and to eliminate superfluous fluids. In the intensive care setting, this can be performed with restrictive volume therapy, diuretics, and/or renal replacement therapy. It has been shown that a negative fluid balance over three days predicts an improved ICU survival rate [76–78]. However, two questions arise: First, what is the best approach to guide deresuscitation, and second, should we consider deresuscitation strategies in the perioperative setting?

To treat fluid overload, it first has to be accurately diagnosed. Assuming that fluid non-responders reflect patients with balanced or overloaded fluid status, a possible approach could be to identify

them using fluid responsiveness tests. Since these patients do not benefit from a volume challenge, it can be assumed that CO does not decrease through fluid removal. Besides, fluid removal can be performed until the fluid responsive tests return positive results. In addition to PLR and EEOT, dynamic preload parameters, body weight quantification, bioimpedance measurements, and respiratory variations of the diameter of the inferior cava vein have been evaluated as treatment goals for deresuscitation [73,76,79–83]. To reduce adverse outcomes after acute lung injury, Cordemans et al. [84] used intra-abdominal pressure and the extravascular lung water index to guide the treatment protocol. This consisted of high PEEP levels, small volume resuscitation with albumin, and fluid removal (PLA - treatment, [PEEP, albumin, and Lasix®]). However, the role of albumin in critically ill patients has to be further investigated because two prospective studies failed to show a beneficial effect of albumin therapy [85,86]. While the Furosemide and Albumin for Diuresis of Edema study failed to proof feasibility [86], the Albumin Italian Outcome Sepsis study showed no improvement in the 90-day survival rate after targeting an albumin plasma level ≥ 30 g/L over 28 days after septic shock [85].

Since the phases of ROSE do not generally apply to surgical patients, it cannot be directly adopted in the perioperative setting. However, it is well-known that perioperative fluid overload is associated with adverse outcomes and should be avoided [87–89]. Hence, smart perioperative volume therapy should prevent fluid overload and the need for perioperative deresuscitation. Over the last decade, several GDT protocols have been introduced and evaluated. A recent meta-analysis summarized 95 randomized-controlled trials and was able to show a GDT-induced reduction in mortality, morbidity, and length of hospital stay [90]. Contrastingly, the Optimisation of Cardiovascular Management to Improve Surgical Outcome (OPTIMISE) study was not able to prove the benefits of GDT [88]. The OPTIMISE study protocol aimed to maximize CO by optimizing it individually with fluids until no increase of stroke volume was detectable with further support from dopexamine. Since no reduction of mortality or morbidity was detected, it can be questioned if a maximized CO target is reasonable. Additionally, it is unclear as to whether patients should receive fluids until their preload capacity is completely exploited [73,91]. Hence, modern GDT protocols do not aim for a maximized CO but rather utilize personalized hemodynamic GDT management with multiple parameters for assessing blood flow and fluid responsiveness [91]. Even though the beneficial effects of personalized GDT are indisputable, only a small degree of patients receive this hemodynamic management. This highlights the need for its greater implementation in daily anesthetic routines [12].

Fluid overload is a common issue following the stabilization of critically ill patients. Deresuscitation strategies using tests for fluid responsiveness as well as hyperoncotic infusion combined with diuretics, and renal replacement therapy, might help to remove the extra fluids and increase survival. However, further high-quality studies are required to confirm these findings. Furthermore, the role of deresuscitation must be discussed in terms of intensifying actions for preventing perioperative fluid overload. To achieve this, personalized hemodynamic treatment goals combined with GDT protocols appear to be an effective approach.

Conclusions

Adverse outcomes after surgery are still common, with surgery considered one of the leading causes of death. Many prospective and retrospective studies have shown that volume management and fluid overload can have detrimental effects on postoperative outcomes. Therefore, strategies that help to prevent fluid overload and assess the individual need for volume during and after surgery should be implemented to increase patient safety. In many patients, no monitoring of volume therapy is performed, or inadequate static parameters are used, such as arterial blood pressure, venous filling pressures, or volumetric parameters that cannot assess fluid responsiveness. As a gold standard to assess whether patients can benefit from an increase of stroke volume and, therefore, potentially by an increase in oxygen delivery, dynamic testing should be performed. This can be done using dynamic parameters like SVV or PPV or by forced manipulation of the preload, e.g., by a volume challenge, PLR, or another physiological testing method.

In combination with a goal-directed hemodynamic monitoring protocol and the application of vasopressors and inotrope medication, a reduction of mortality by 58 was observed in a recent meta-analysis [90]. Whether goal-directed volume therapy can reduce perioperative mortality still needs to be demonstrated in larger multicenter studies. Whether the concept of using suitable parameters for goal-directed deresuscitation reduces complication rates, and mortality in the perioperative setting still awaits confirmation by larger trials. Nevertheless, this concept appears to be promising.

Conflicts of Interest

No potential conflict of interest relevant to this article was reported.

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