Volumetric Parameters: A Physiological Background

12

Vsevolod V. Kuzkov

Contents

12.1 Introduction

As has been stated in Chaps. 3 and 4, the diagnostic and prognostic values of filling pressures (central venous and pulmonary arterial occlusion pressures) are limited by presenting only one part of the "static" parameters of preload [1, 2]. Another part of preload, the volumetric variables, deals with quantification of volume of all the heart chambers, great vessels, and pulmonary vascular bed, as well as the extravascular compartment of the lungs [1, 3, 4]. Thus, the direct quantification of the volumetric parameters using transpulmonary thermodilution in parallel with assessment of fluid responsiveness has opened up new opportunities for the personalization of hemodynamic therapy in different categories of the critically ill [3, 5].

One of the key volumetric parameters is the *global end-diastolic volume index* (GEDVI), also referred as a current clinical "gold standard" of bedside invasive preload assessment (Chap. 13). The discrete evaluation of the end-diastolic volumes of the right- and left-sided heart chambers, as well as the ejection fractions, is also technically possible, but highly invasive as it requires both systemic and pulmonary arterial catheters and is mainly limited to the clinical research activity for invasive cardiology and organ transplantation [6–8]. The combination of the GEDVI and other variables can be helpful in assessment of heart contractility ("inotropism"). Therefore, a

V. V. Kuzkov (✉)
Department of Anesthesiology and Intensive Care Medicine, Northern State Medical University, Arkhangelsk, Russian Federation
e-mail: v_kuzkov@mail.ru

© Springer Nature Switzerland AG 2021

M. Y. Kirov et al. (eds.), *Advanced Hemodynamic Monitoring: Basics and New Horizons*,
https://doi.org/10.1007/978-3-030-71752-0_12

range of derived parameters based on the methodology of single transpulmonary thermodilution (Chap. 7) are of interest, including the *global ejection fraction* (GEF) and the *cardiac function index* (CFI).

The increased vascular permeability is a complex and multifactorial pathophysiological phenomenon that cannot currently be measured directly at the bedside [9, 10]. Indeed, all attempts to normalize the preload can be ineffective and even dangerous when the fluids leave the vascular bed and leak into the interstitial space. Thus, the interpretation of volumetric parameters and a safe clinical decision would be incomplete without information about the severity of pulmonary edema and capillary leak [1]. The volumetric monitoring gives us a clinical clue to the indirect assessment of these processes by means of the *extravascular lung water index* (EVLWI) and the *pulmonary vascular permeability index* (PVPI; Chap. 14).

Practical Advice

"Classic" volumetric monitoring includes the global end-diastolic volume index, the extravascular lung water index, and the global ejection fraction/cardiac function index. These parameters characterize preload, lung fluid balance, and heart contractility respectively.

Unfortunately, in many complex clinical scenarios, attempts to increase the circulating blood volume with fluid therapy do not result in a steady increase in cardiac output and oxygen delivery as fluids readily extravasate [10, 11]. Thus, in these situations the hemodynamic stabilization can eventually be achieved only at the price of progressing tissue edema, worsening organ function, and developing complications (Fig. 12.1). As these complications are the key features of the distributive shock (Chap. 25), monitoring the GEDVI, EVLWI, and PVPI in the critically ill reflects the dynamic fluid balance between the intra- and extravascular compartments [12–14].

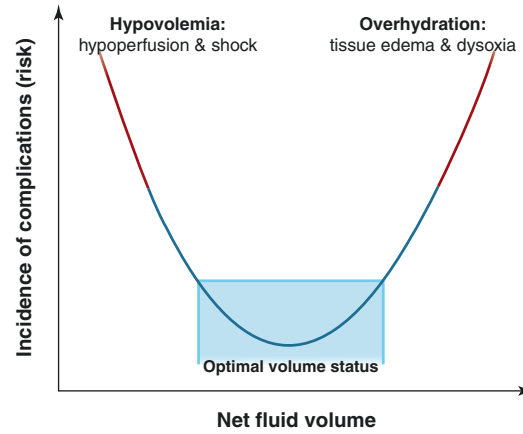

Fig. 12.1 Incidence of complications: dangers of hypovolemia and overhydration

Considered together, the volumetric variables can characterize both the efficacy and safety of the preload optimization, the response of cardiac contractility and the contribution of the "fluid sink" of the vascular bed, making volumetric monitoring an attractive approach for the bedside personalization of hemodynamic status. The normal clinical values of the volumetric parameters are presented in Table 12.1.

Of note, the *static volumetric* and the *dynamic fluid responsiveness parameters* are not interchangeable and have different practical applications [15, 16]. Dynamic parameters are often evaluated together with functional tests to predict the short-term response of cardiac output to fluid load; however, the fluid responsiveness cannot guarantee that an instantly increased preload will be associated with steady, prolonged and, last but not least, physiologically beneficial, hemodynamic changes (Chaps. 15–17).

The clinical area of application of volumetric monitoring includes many critical care and perioperative scenarios. The most promising indications are different subsets of circulatory shock associated with cardiovascular comorbidities and respiratory failure, as well as the perioperative period of high-risk and complicated interventions such as complex cardiothoracic surgery and organ transplantation (Table 12.2).

Table 12.1 The normal values and ranges of hemodynamic and volumetric variables[a]

Variable	Ranges
Flow	
Cardiac output, L/min	5.0–7.0
Cardiac index, L/min/m^2	3.0–5.0
Pulse contour cardiac index, L/min/m^2	3.0–5.0
Cardiac preload	
Global end-diastolic volume index, mL/m^2	680–800
Intrathoracic blood volume index, mL/m^2	850–1000
Central venous pressure, mmHg	5–7
Volume responsiveness	
Stroke volume variation, %	≤10
Pulse pressure variation, %	≤10
Afterload	
Systemic vascular resistance index, dyn × s × cm^{-5}/m^2	1700–2400
Cardiac contractility	
Cardiac function index, L/min	4.5–6.5
Global ejection fraction, %	25–35
Index of left ventricular contractility (dPmax), mmHg/s	1200–2000
Cardiac power index, W/m^2	0.5–0.7
Pulmonary edema	
Extravascular lung water index, mL/kg PBW	3–7
Pulmonary vascular permeability index	1–3

PBW predicted body weight
[a]The volumetric parameters discussed are presented in bold

Table 12.2 The areas for clinical application of volumetric hemodynamic monitoring

Critical care settings	Perioperative settings
• Sepsis and septic shock [2, 17, 18] • Nonseptic distributive shock [11] • Pulmonary edema [17, 19] • Cardiogenic shock and severe heart failure [20] • Severe acute respiratory distress syndrome [17, 21] • Severe burns [22] • Severe subarachnoid hemorrhage [23] • Severe necrotizing pancreatitis [24] • Overhydration [11]	• Complex cardiac surgery [25, 26] • Thoracic surgery (lung transplantation) [27, 28] • Complex neurosurgery [29] • Liver transplantation [30, 31]

Practical Advice

Personalized approach to "normal" values of the global end-diastolic volume index may be considered in some subsets of ICU patients, including "permissive hypovolemia" (GEDVI 500–650 mL/m^2) for those with severe global permeability syndrome and "permissive hypervolemia" (GEDVI 800–950 mL/m^2) for those with severe systolic heart failure.

12.2 Transpulmonary Thermodilution and Volumetric Parameters

Transpulmonary thermodilution (TPTD) for volumetric hemodynamic assessment is currently recommended for advanced monitoring in severe shock [3, 5, 32] and can be achieved in several commercially available systems of complex hemodynamic monitoring [33, 34].

The invasive quantification of volumetric hemodynamic parameters, characterizing heart filling and vascular permeability, is based on the dilution of a thermal indicator, injected into the systemic circulation.

The methodology of TPTD is described in detail in Chap. 7. In brief, the thermal indicator "keeps warm" (or loses the "negative heat") depending on multiple intrinsic factors (blood flow velocity, time of heat exchange, and tissue heat capacity) when passing by and mixing with blood of the heart chambers, limited portions of the great vessels (vena cava and aorta), and the pulmonary vascular bed [33]. The process of this thermal exchange depends on both the physical volume of distribution and the thermal capacity/conductivity of the pulmonary tissue, therefore allowing the quantification of the EVLWI. The physiology and mathematical background for the calculation of the volumetric parameters are depicted in Fig. 12.2.

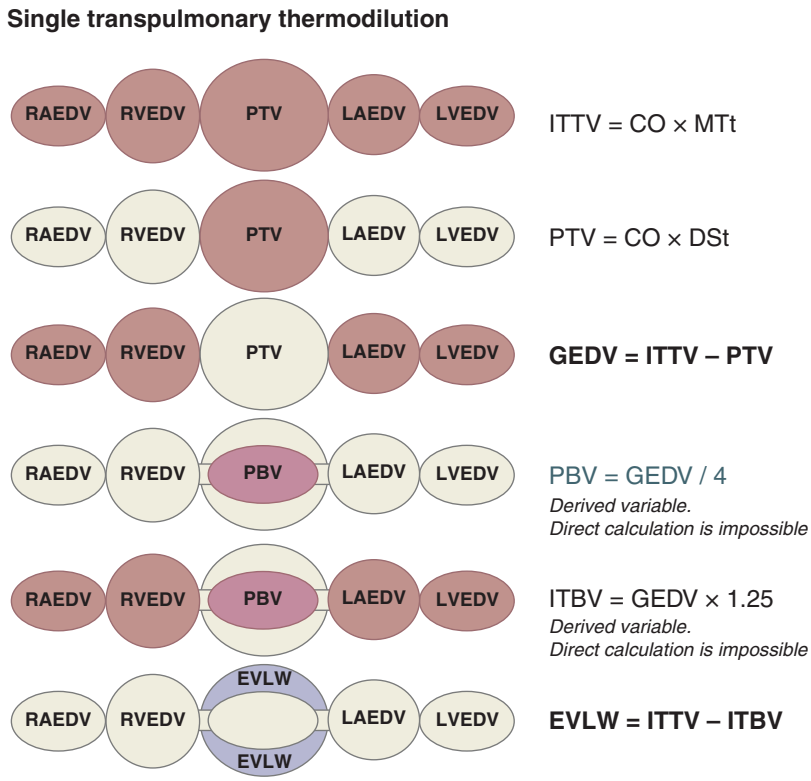

Fig. 12.2 Physiological layout and calculation of the volumetric parameters using single transpulmonary thermodilution. *EDV* end-diastolic volume, *RA* right atrium, *RV* right ventricle, *LA* left atrium, *LV* left ventricle, *MTt* mean transit time of the thermal indicator, *DSt* down-slope time of the thermodilution curve, *CO* cardiac output, *ITTV* intrathoracic thermal volume, *PTV* pulmonary thermal volume, *GEDV* global end-diastolic volume, *PBV* pulmonary blood volume, *ITBV* intrathoracic blood volume, *EVLW* extravascular lung water

12.3 Volumetric Parameters of Preload

12.3.1 Global End-Diastolic Volume Index

As has been stated already, the preload assessment with central venous and pulmonary artery occlusion pressures is limited by changing myocardial compliance, positive pressure mechanical ventilation, and, in some cases, valvular disturbances. The dynamic parameters and functional tests do predict instant heart response to the rapid increase in preload but cannot help us to assess the real-time kinetics of the fluid being administered. In addition, the use of functional parameters is restricted in overhydrated ICU patients and in the late phase of distributive shock. Therefore, according to the current phasic paradigm of shock management, the GEDVI can be one of the most promising variables for preload quantification.

Practical Advice

Among current volumetric parameters, the global end-diastolic volume index measured using single transpulmonary thermodilution represents a clinical "gold standard" for bedside preload assessment in the critically ill.

There are many reasons why the GEDVI is the most accurate preload marker. Of note, the GEDVI is the summarized value of the maximal volumes of all four heart chambers indexed to the calculated body surface area. Thus, the GEDVI is more accurate for preload assessment than central venous pressure (CVP), pulmonary artery occlusion pressure, the right ventricular end-diastolic volume, and the left ventricular end-diastolic area [35, 36]. In contrast to CVP, the GEDVI accurately quantifies the preload in septic shock and severe ARDS [17, 37]. The accu-

racy of this parameter has also been confirmed in children and neonates [38, 39]. In many cases, the GEDVI has been used as a reference parameter for echocardiographic variables [36, 40]. This parameter is plausible during normovolemia, moderate hypovolemia, pulmonary hypertension and inotropic support [41, 42]. Moreover, the GEDVI accurately characterizes preload in both controlled mechanical ventilation and spontaneous breathing. However, aortic aneurism and prominent dilatation of the left atrium can result in a falsely increased GEDVI. The plausibility of GEDVI interpretation can also be limited in severe heart failure [42, 43]. The interplay between the GEDVI and another important volumetric parameter, the EVLWI, during fluid resuscitation is of utmost clinical interest in many categories of ICU patients [44, 45]. The methodology of measurement and the clinical application of the GEDVI are discussed in detail in Chaps. 7 and 13 respectively.

12.3.2 Global Ejection Fraction

The GEF is another important volumetric variable allowing an assessment of the heart performance, particularly systolic function, in terms of its work (stroke volume) and preload. The calculation of the GEF using TPTD is based on the following formula: (4 × SV)/GEDV; thus, the normal value of GEF (25–35%; Table 12.1) differs from the echocardiographic ejection fraction. Most frequently, the decrease in GEF can result from the dilatation of the heart chambers, leading to increased GEDV. This parameter is a valuable key to revealing heart failure, whereas isolated right heart failure, pulmonary hypertension, and increased right heart afterload are known limitations decreasing the clinical plausibility of its measurement [46, 47]. In the case of systolic heart failure, both the GEF and the CFI (see below) are declining [46]. Nakwan et al. has shown that both the CFI and the GEF obtained using transpulmonary thermodilution are associated with the left ventricle ejection fraction measured using echocardiography in septic shock [48], whereas the GEF correlates closely with the results of transesophageal echocardiography in acute myocardial ischemia [49]. However, when assessment of cardiac output is unaffected by differences in ventricular size and outflow obstruction, the GEDVI, GEF, and CFI do not reflect the largely increased heart volumes and markedly impaired left ventricular function in dilated cardiomyopathy [50].

12.3.3 Cardiac Function Index

The CFI is a ratio of the cardiac index and the intrathoracic blood volume index (Fig. 12.2) and independently characterizes heart contractility under the current preload settings [51–53]. With normal values within the range 4.6–6.5 min^{-1}, this parameter is sensitive to the inotropic support and the position of the Frank–Starling curve [49]. It has also been proposed that assessment of cardiac function by the CFI using the transpulmonary thermodilution technique is a plausible alternative to the pulmonary catheter, and a low CFI identifies cardiac dysfunction in both acute heart failure and sepsis [54].

12.4 Other Volumetric Parameters

12.4.1 Extravascular Lung Water Index

The extravascular lung water index is a volumetric parameter quantifying pulmonary edema [3, 12, 14]. This parameter and the use of EVLW as a target for therapy are described in more detail in Chaps. 7, 14, and 26.

The close interplay between cardiac output and volumetric parameters can be integrated into clinical decision trees for the management of critically ill patients; the example of such an algorithm is presented in Fig. 12.3. The typical changes in volumetric hemodynamic parameters in the common critical care scenarios are presented in Table 12.3.

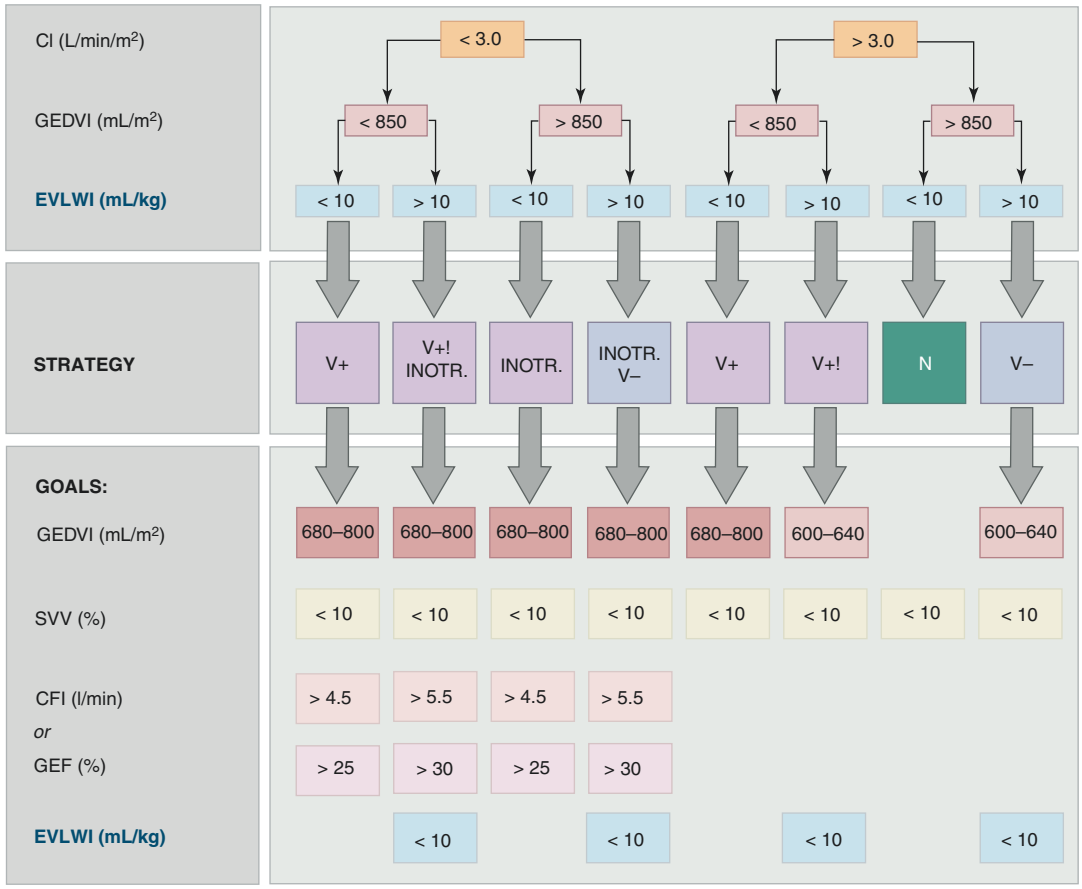

Fig. 12.3 A clinical decision tree for the personalization of hemodynamic management based on the volumetric parameters. *CI* cardiac index, *GEDVI* global end-diastolic volume index, *EVLWI* extravascular lung water index, *SVV* stroke volume variation, *CFI* cardiac function index, *GEF* global ejection fraction, *V+* fluid bolus (volume load), *V+!* titrated fluid (volume with caution!), *INOTR* inotropes, *V–*dehydration, *N* normal state

Table 12.3 The changes in volumetric parameters in the selected critical care scenarios

Condition	Etiology	Change in volumetric parameters
Severe hypovolemia (hemorrhagic shock)	Hemorrhage, severe burns, decreased preload in high intrapleural pressure, pneumothorax	Low GEDVI (usually <600 mL/m²), relatively low EVLWI (4–7 mL/kg), low CO, low CFI, low GEF. Increase in GEDVI leads to a rise in CO without the obvious risk of early EVLW accumulation
Overhydration	Volume overload, acute kidney injury, lymphatic blockade (sepsis, PEEP), ARDS	Normal to increased GEDVI. Increased EVLWI (usually above 10 mL/kg). No fluid responsiveness observed
Severe heart failure, cardiogenic shock	Structural changes leading to decreased myocardial contractility	Normal to increased GEDVI and "gray zone" EVLWI (7–10 mL/kg). In severe pulmonary edema, the EVLWI readily decreases after diuretics or positive pressure ventilation. Markedly decreased CO (below 1.8–2.0 L/min), decreased GEF (below 20%) and CFI

Table 12.3 (continued)

Condition	Etiology	Change in volumetric parameters
Pulmonary edema/ARDS	Direct and indirect causes of ARDS (pneumonia, sepsis, shock, pancreatitis, *etc.*)	Increased EVLWI (usually above 10 mL/kg) and PVPI (usually above 2.5–3.0). Low-to normal GEDVI during the early phase. Despite fluid responsiveness, attempts to increase the GEDVI by giving fluids lead to a rise in the EVLWI, therefore posing the question of a "permissive" hypovolemia
Distributive shock	Mostly sepsis	Increased EVLWI (sometimes even without ARDS criteria), normal-to-increased CO (hyperdynamic state), varying GEDVI (usually decreased during a capillary leak). Normal GEF and CFI do not exclude diastolic heart dysfunction

CO cardiac output, *GEDVI* global end-diastolic volume index, *EVLWI* extravascular lung water index, *PVPI* pulmonary vascular permeability index, *CFI* cardiac function index, *GEF* global ejection fraction, *PEEP* positive end-expiratory pressure, *ARDS* acute respiratory distress syndrome

12.5 Conclusion

Today, invasive volumetric monitoring is used in a variety of life-threatening critical care scenarios. The applicability and reproducibility of measurements for a wide range of hemodynamically unstable conditions are the key advantages of this technique. The accuracy of volumetric parameters for the quantification of preload, myocardial contractility, and pulmonary edema has been proven by numerous experimental and clinical studies. Because in critical care medicine volumetric monitoring co-exists with less-invasive ultrasound methods, we believe that both approaches will progress side-by-side. However, in contrast to echocardiography, transpulmonary thermodilution is less operator dependent and gives an all-in-one hemodynamic "bundle," facilitating a clinical decision. Thus, integration of personalized algorithms guided by volumetric parameters into the management of severe shock and acute respiratory distress syndrome would open up new horizons for the improvement of clinical outcomes and warrants further studies.

Keynotes

- The key target of volumetric monitoring is an accurate and versatile assessment of preload, heart function, and lung fluid balance.
- Volumetric monitoring can significantly improve our understanding of the kinetics of fluids used for resuscitation in the most severely ill ICU patients.
- In clinical practice, volumetric monitoring is interrelated with the real-time measurement of stroke volume and prediction of fluid responsiveness.
- The optimization of preload under the tight control of extravascular lung water may improve the safety of the phase management of shock and facilitate the personalization of hemodynamic therapy.
- Further studies are needed to investigate treatment protocols based on volumetric parameters.

Conflict of Interest None.

Funding None.

References

1. Rocca GD, Costa MG, Pietropaoli P. How to measure and interpret volumetric measures of preload. Curr Opin Crit Care. 2007;13:297–302.
2. Sakka SG, Bredle DL, Reinhart K, Meier-Hellmann A. Comparison between intrathoracic blood volume and cardiac filling pressures in the early phase of hemodynamic instability of patients with sepsis or septic shock. J Crit Care. 1999;14:78–83.
3. Teboul J-L, Saugel B, Cecconi M, De Backer D, Hofer CK, Monnet X, Perel A, Pinsky MR, Reuter DA, Rhodes A, Squara P, Vincent J-L, Scheeren TW. Less invasive hemodynamic monitoring in critically ill patients. Intensive Care Med. 2016;42:1350–9.
4. Akohov A, Barner C, Grimmer S, Francis RC, Wolf S. Aortic volume determines global end-diastolic volume measured by transpulmonary thermodilution. Intensive Care Med Exp. 2020;8(1):1.
5. Cecconi M, De Backer D, Antonelli M, Beale R, Bakker J, Hofer C, Jaeschke R, Mebazaa A, Pinsky MR, Teboul JL, Vincent JL, Rhodes A. Consensus on circulatory shock and hemodynamic monitoring. Task force of the European Society of Intensive Care Medicine. Intensive Care Med. 2014;40:1795–815.
6. Garcia Gigorro R, Renes Carreño E, Mayordomo S, et al. Evaluation of right ventricular function after cardiac surgery: the importance of tricuspid annular plane systolic excursion and right ventricular ejection fraction. J Thorac Cardiovasc Surg. 2016;152(2):613–20.
7. Wiesenack C, Fiegl C, Keyser A, et al. Continuously assessed right ventricular end-diastolic volume as a marker of cardiac preload and fluid responsiveness in mechanically ventilated cardiac surgical patients. Crit Care. 2005;9:R226–33.
8. Rocca GD, Costa MG, Feltracco P, et al. Continuous right ventricular end diastolic volume and right ventricular ejection fraction during liver transplantation: a multicenter study. Liver Transpl. 2008;14(3):327–32.
9. Fishel RS, Are C, Barbul A. Vessel injury and capillary leak. Crit Care Med. 2003;31:S502–11.
10. Marx G. Fluid therapy in sepsis with capillary leakage. Eur J Anaesthesiol. 2003;20:429–42.
11. Malbrain ML, Marik PE, Witters I, et al. Fluid overload, de-resuscitation, and outcomes in critically ill or injured patients: a systematic review with suggestions for clinical practice. Anaesthesiol Intensive Ther. 2014;46(5):361–80.
12. Isakow W, Schuster DP. Extravascular lung water measurements and hemodynamic monitoring in the critically ill: bedside alternatives to the pulmonary artery catheter. Am J Physiol Lung Cell Mol Physiol. 2006;291:L1118–31.
13. Khan S, Trof RJ, Groeneveld AJ. Transpulmonary dilution-derived extravascular lung water as a measure of lung edema. Curr Opin Crit Care. 2007;13:303–7.
14. Kirov MY, Kuzkov VV, Bjertnaes LJ. Extravascular lung water in sepsis. In: Vincent J-L, editor. Yearbook of intensive care and emergency medicine. Berlin, Heidelberg, New York: Springer; 2005. p. 449–61.
15. Cavallaro F, Sandroni C, Antonelli M. Functional hemodynamic monitoring and dynamic indices of fluid responsiveness. Minerva Anestesiol. 2008;74:123–35.
16. Monnet X, Marik PE, Teboul JL. Prediction of fluid responsiveness: an update. Ann Intensive Care. 2016;6(1):111.
17. Kuzkov VV, Kirov MY, Sovershaev MA, et al. Extravascular lung water determined with single transpulmonary thermodilution correlates with the severity of sepsis-induced acute lung injury. Crit Care Med. 2006;34:1647–53.
18. Yu J, Zheng R, Lin H, Chen Q, Shao J, Wang D. Global end-diastolic volume index vs CVP goal-directed fluid resuscitation for COPD patients with septic shock: a randomized controlled trial. Am J Emerg Med. 2017;35(1):101–5.
19. Tagami T, Ong MEH. Extravascular lung water measurements in acute respiratory distress syndrome: why, how, and when? Curr Opin Crit Care. 2018;24(3):209–15.
20. De Backer D. Is there a role for invasive hemodynamic monitoring in acute heart failure management? Curr Heart Fail Rep. 2015;12(3):197–204.
21. Vieillard-Baron A, Matthay M, Teboul JL, et al. Experts' opinion on management of hemodynamics in ARDS patients: focus on the effects of mechanical ventilation. Intensive Care Med. 2016;42(5):739–49.
22. Cartotto R, Greenhalgh DG, Cancio C. Burn state of the science: fluid resuscitation. J Burn Care Res. 2017;38(3):e596–604.
23. Okazaki T, Kuroda Y. Aneurysmal subarachnoid hemorrhage: intensive care for improving neurological outcome. J Intensive Care. 2018;6:28.
24. Huber W, Umgelter A, Reindl W, et al. Volume assessment in patients with necrotizing pancreatitis: a comparison of intrathoracic blood volume index, central venous pressure, and hematocrit, and their correlation to cardiac index and extravascular lung water index. Crit Care Med. 2008;36(8):2348–54.
25. Kirov MY, Lenkin AI, Kuzkov VV, et al. Single transpulmonary thermodilution in off-pump coronary artery bypass grafting: haemodynamic changes and effects of different anaesthetic techniques. Acta Anaesthesiol Scand. 2007;51:426–33.
26. Reuter DA, Felbinger TW, Schmidt C, et al. Trendelenburg positioning after cardiac surgery: effects on intrathoracic blood volume index and cardiac performance. Eur J Anaesthesiol. 2003;20:17–20.
27. Della Rocca G, Costa GM, et al. Preload index: pulmonary artery occlusion pressure versus intrathoracic blood volume monitoring during lung transplantation. Anesth Analg. 2002;95:835–43.

28. Della Rocca G, Brondani A, Costa MG. Intraoperative hemodynamic monitoring during organ transplantation: what is new? Curr Opin Organ Transplant. 2009;14(3):291–6.
29. Buhre W, Weyland A, Buhre K, et al. Effects of the sitting position on the distribution of blood volume in patients undergoing neurosurgical procedures. Br J Anaesth. 2000;84:354–7.
30. Costa MG, Chiarandini P, Della Rocca G. Hemodynamics during liver transplantation. Transplant Proc. 2007;39:1871–3.
31. Costa MG, Girardi L, Pompei L, et al. Perioperative intra- and extravascular volume in liver transplant recipients. Transplant Proc. 2011;43(4):1098–102.
32. Scheeren TWL, Ramsay MAE. New developments in hemodynamic monitoring. J Cardiothorac Vasc Anesth. 2019;33(Suppl 1):S67–72.
33. Sakka SG, Rühl CC, Pfeiffer UJ, et al. Assessment of cardiac preload and extravascular lung water by single transpulmonary thermodilution. Intensive Care Med. 2000;26:180–7.
34. Saugel B, Vincent JL. Cardiac output monitoring: how to choose the optimal method for the individual patient. Curr Opin Crit Care. 2018;24(3):165–72.
35. Lichtwarck-Aschoff M, Beale R, Pfeiffer UJ. Central venous pressure, pulmonary artery occlusion pressure, intrathoracic blood volume, and right ventricular end-diastolic volume as indicators of cardiac preload. J Crit Care. 1996;11:180–8.
36. Hofer CK, Furrer L, Matter-Ensner S, et al. Volumetric preload measurement by thermodilution: a comparison with transoesophageal echocardiography. Br J Anaesth. 2005;94:748–55.
37. Wan L, Naka T, Uchino S, Bellomo R. A pilot study of pulse contour cardiac output monitoring in patients with septic shock. Crit Care Resusc. 2005;7:165.
38. Cecchetti C, Stoppa F, Vanacore N, et al. Monitoring of intrathoracic volemia and cardiac output in critically ill children. Minerva Anestesiol. 2003;69:907–18.
39. Schiffmann H, Erdlenbruch B, Singer D, et al. Assessment of cardiac output, intravascular volume status, and extravascular lung water by transpulmonary indicator dilution in critically ill neonates and infants. J Cardiothorac Vasc Anesth. 2002;16:592–7.
40. Hofer CK, Ganter MT, Matter-Ensner S, et al. Volumetric assessment of left heart preload by thermodilution: comparing the PiCCO-VoLEF system with transoesophageal echocardiography. Anaesthesia. 2006;61:316–21.
41. Nirmalan M, Willard TM, Edwards DJ, et al. Estimation of errors in determining intrathoracic blood volume using the single transpulmonary thermal dilution technique in hypovolemic shock. Anesthesiology. 2005;103:805–12.
42. Renner J, Gruenewald M, Brand P, et al. Global end-diastolic volume as a variable of fluid responsiveness during acute changing loading conditions. J Cardiothorac Vasc Anesth. 2007;21:650–4.
43. Mundigler G, Heinze G, Zehetgruber M, et al. Limitations of the transpulmonary indicator dilution method for assessment of preload changes in critically ill patients with reduced left ventricular function. Crit Care Med. 2000;28:2231–7.
44. Marik PE, Lemson J. Fluid responsiveness: an evolution of our understanding. Br J Anaesth. 2014;112:620–2.
45. Marik P, Bellomo R. A rational approach to fluid therapy in sepsis. Br J Anaesth. 2016;116(3):339–49.
46. Combes A, Berneau JB, Luyt CE, Trouillet JL. Estimation of left ventricular systolic function by single transpulmonary thermodilution. Intensive Care Med. 2004;30(7):1377–83.
47. Kapoor PM, Bhardwaj V, Sharma A, Kiran U. Global end-diastolic volume: an emerging preload marker vis-a-vis other markers—have we reached our goal? Ann Card Anaesth. 2016;19(4):699–704.
48. Nakwan N, Chichareon P, Khwannimit B. A comparison of ventricular systolic function indices provided by VolumeView/EV1000™ and left ventricular ejection fraction by echocardiography among septic shock patients. J Clin Monit Comput. 2019;33(2):233–9.
49. Meybohm P, Gruenewald M, Renner J, Maracke M, Rossee S, Höcker J, Hagelstein S, Zacharowski K, Bein B. Assessment of left ventricular systolic function during acute myocardial ischemia: a comparison of transpulmonary thermodilution and transesophageal echocardiography. Minerva Anestesiol. 2011;77:132–41.
50. Hilty MP, Franzen DP, Wyss C, Biaggi P, Maggiorini M. Validation of transpulmonary thermodilution variables in hemodynamically stable patients with heart diseases. Ann Intensive Care. 2017;7(1):86.
51. Jabot J, Monnet X, Bouchra L, Chemla D, Richard C, Teboul J-L. Cardiac function index provided by transpulmonary thermodilution behaves as an indicator of left ventricular systolic function. Crit Care Med. 2009;37:2913–8.
52. Pfeiffer UJ, Wisner-Euteneier AJ, Lichtwarck-Aschoff M, Blumel G. Less invasive monitoring of cardiac performance using arterial thermodilution. Clin Intensive Care. 1994;5(Suppl):28.
53. Wisner-Euteneier AJ, Lichtwarck-Aschoff M, Zimmermann G, et al. Evaluation of the cardiac function index as a new bedside indicator of cardiac performance. Intensive Care Med. 1994;20:S21.
54. Ritter S, Rudiger A, Maggiorini M. Transpulmonary thermodilution-derived cardiac function index identifies cardiac dysfunction in acute heart failure and septic patients: an observational study. Crit Care. 2009;13(4):R133.