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Review Article

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Volumetric Monitoring and Extravascular Lung Water in Perioperative Setting and Critically Ill

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Abstract

In the new century, our diagnostic armamentarium has been significantly reinforced by the 'three-dimensional' volumetric haemodynamic monitoring currently available at the bedside in many perioperative and intensive care settings. The volumetric approach has improved our insight into the haemodynamic scenarios of many critical illnesses and surgical interventions, including sepsis, circulatory shock, acute respiratory distress syndrome as well as cardiothoracic and transplantation surgery. However, the influence of volumetric haemodynamic monitoring on clinical outcome is still a subject for debates. This review presents physiological background, technical details, aspects of bedside use, limitations and further perspectives of the volumetric approach to the cardiopulmonary monitoring.

Keywords: Volumetric monitoring, transpulmonary thermodilution, preload, global end-diastolic volume, pulmonary oedema, extravascular lung water

Introduction

Both diagnostic and prognostic significance of filling pressures (central venous and pulmonary arterial occlusion pressures) is limited by interaction with multiple extracardiac factors.[1] In contrast, volumetric variables allow quantification of physical volume of heart chambers, major vessels and pulmonary vasculature as well as thermal impact of the pulmonary extravascular compartment.[2,3] The 'classic' volumetric monitoring includes global end-diastolic volume, extravascular lung water, global ejection fraction (GEF) and cardiac function index (CFI).[3–5] These parameters characterise preload, lung fluid balance and heart contractility, respectively. Therefore, the volumetric approach to the assessment of the haemodynamics using transpulmonary thermodilution (TPTD) coupled with assessment of fluid responsiveness gives new opportunities for the personalisation of both haemodynamic and respiratory therapy in different subsets of perioperative and critically ill patients.[6]

Being one of the most important volumetric parameters, *global end-diastolic volume index* (GEDVI) has been referred recently as a 'gold standard' of the invasive assessment of the preload at the bedside.[7] The evaluation of the discrete end-diastolic volumes of right- and left-heart chambers as well as ejection fractions is also available; however, this approach requires both systemic arterial and pulmonary catheterisation and is mainly limited to the clinical research and selected cases in interventional cardiology and heart/lung transplantation.[8] The combination of GEDVI and other haemodynamic variables can be helpful in assessment of heart contractility. GEF and CFI are the most important derived contractility variables based on the technique of TPTD.[9]

All the attempts to optimise preload cannot be effective if the fluids escape the vasculature and leak into the interstitial space.[10] Being a complex pathophysiological phenomenon, the vascular permeability cannot be directly measured at the bedside.[11] Therefore, the interpretation of volumetric parameters and appropriate clinical decision require information about the severity of capillary leak and pulmonary oedema.[10,12,13] However, the volumetric approach gives a clinical clue to the indirect assessment of lung fluid balance by the quantification of *extravascular lung water index* (EVLWI) and *pulmonary vascular permeability index* (PVPI).[13]

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Table 1. The Normal Values and Ranges of Haemodynamic and Volumetric Variables*

Variable	Range
Flow	
Cardiac output (CO), $L\,min^{-1}$	4.5–6.5
Cardiac index (CI), $L\,min^{-1}\,m^{-2}$	3.5–5.5
Pulse contour cardiac index (PCCI), $L\,min^{-1}\,m^{-2}$	3.5–5.5
Cardiac preload	
Global end-diastolic volume index (GEDVI), $mL\,m^{-2}$	680–800†
Intrathoracic blood volume index (ITBVI), $mL\,m^{-2}$	850–1,000
Central venous pressure (CVP), mm Hg	5–7
Volume responsiveness	
Stroke volume variation (SVV), %	≤10
Pulse pressure variation (PPV), %	≤10
Afterload	
Systemic vascular resistance index (SVRI), $dyn\times s\times cm^{-5}\,m^{-2}$	1,700–2,400
Cardiac contractility	
Cardiac function index (CFI), $L\,min^{-1}$	4.5–6.5
Global ejection fraction (GEF), %	25–35
Index of left ventricular contractility (dPmax), mm $Hg\,s^{-1}$	1,200–2,000
Cardiac power index (CPI), $W\,m^{-2}$	0.5–0.7
Pulmonary oedema	
Extravascular lung water index (EVLWI), $mL\,kg^{-1}$ PBW	3–7
Pulmonary vascular permeability index (PVPI)	1–3

Abbreviation: PBW, predicted body weight.
*The volumetric parameters are presented in bold.
†Personalized approach to 'normal' values of global end-diastolic volume index may be considered in some subsets of ICU patients including 'permissive hypovolaemia' (GEDVI 500–650 $mL\,m^{-2}$) for those with severe global permeability syndrome and 'permissive hypervolaemia' (GEDVI 800–950 $mL\,m^{-2}$) for those with severe systolic heart failure.

In many complex perioperative and ICU scenarios, the attempts to restore the ventricular preload with fluids do not result in improvement of cardiac performance and oxygen transport since fluids instantly extravasate.[12,14] In these circumstances, the haemodynamic 'optimisation' can be eventually achieved only at a price of progressing tissue and pulmonary oedema, impending organ dysfunction and complications. The assessment of GEDVI, EVLWI and PVPI helps to avoid these side effects of therapy by reflecting the actual and dynamic balance between intra- and extravascular spaces and providing key targets for fluid management.[10,13] When considered together, these volumetric parameters can characterise both the efficacy and safety of the preload optimisation, the response of heart and the contribution of the fluid leakage, making volumetric monitoring a logical approach for the true personalisation of haemodynamic status. The normal ranges of the primary and derived volumetric variables are presented in Table 1.

Main Points

- The benefits of volumetric monitoring include an accurate and versatile assessment of preload, heart contractility and lung fluid balance.
- Volumetric monitoring can highlight the real kinetics of the resuscitation fluids used in the critically ill and high-risk perioperative patients.
- The optimisation of preload under the real-time monitoring of pulmonary oedema can improve the safety of the phasic shock management and open new perspectives in the personalisation of cardiopulmonary management.
- Further major studies are warranted to develop and explore goal-directed volumetric-based protocols.

Importantly, in clinical practice, the *static volumetric variables* and the *dynamic fluid responsiveness parameters* such as pulse pressure and stroke volume variations are not interchangeable.[14,15] Dynamic parameters are often evaluated together along with *functional tests* to predict the instant response of cardiac output to fluid resuscitation. However, the observed

Table 2. The Areas for Clinical Application of Volumetric Haemodynamic Monitoring

Perioperative settings	Critical care settings
Complex cardiac surgery	Sepsis and septic shock
Complex neurosurgery	Non-septic distributive shock
Thoracic and oesophageal surgery	Overhydration and pulmonary oedema
Transplantation	Cardiogenic shock and severe heart failure
	Acute respiratory distress syndrome and pneumonia (incl. viral)
	Severe burns
	Subarachnoid haemorrhage
	Necrotising pancreatitis

fluid responsiveness cannot guarantee that steadily increased preload will be associated with persisting and, finally, physiologically beneficial increase in oxygen transport.

The clinical applications of volumetric haemodynamic monitoring include multiple critical care and perioperative scenarios. The most important indications are various types of circulatory shock associated with cardiovascular and respiratory comorbidities as well as perioperative period of the high-risk and complicated interventions like complex cardiothoracic, liver and oncology surgery, as well as organ transplantation (Table 2).

Transpulmonary Thermodilution for Volumetric Monitoring

Single TPTD is a preferred invasive technique for volumetric haemodynamic assessment and is recommended currently for the advanced 'in-depth' cardiopulmonary monitoring in shock and complicated perioperative settings.[2,3,6] This technique is realised in several commercial systems for complex haemodynamic monitoring and is readily available at the bedside.[4,5]

The quantification of volumetric cardiopulmonary variables, characterising heart volume, extracardiac tissues and vascular permeability is based on the dilution of thermal indicator of known temperature and volume, injected into the systemic circulation (Figure 1).

Thermal indicator 'keeps warm' (or loses the 'negative heat') depending on the multiple intrinsic factors (blood flow velocity, rate of heat exchange and tissue heat capacity) when passing by and mixing with blood of the heart chambers, limited portions of great vessels (vena cava and aorta) and pulmonary vascular bed.[1,2,13] The process of this thermal exchange depends on both the physical volume of distribution and thermal capacity/conductivity of pulmonary tissue, therefore allowing the quantification of EVLWI. The physiology and underlying calculations are depicted in detail in Figure 2.

Volumetric Parameters of Preload

Global End-Diastolic Volume Index

Among current volumetric parameters, GEDVI measured with single TPTD represents a clinical 'gold standard' for the bedside preload assessment in critically ill and in perioperative settings.[1,7] According to the current phasic paradigm of the shock management, GEDVI can be one from the key variables for preload quantification.[12] As has been noticed yet, the assessment of the preload with central venous and pulmonary artery occlusion pressures is substantially limited by the changes in myocardial compliance, positive pressure mechanical ventilation and valvular disturbances.[1,7] The dynamic fluid responsiveness parameters and bedside functional tests reveal time-dependent heart response to the instant increase in preload but do not represent the real-time kinetics of the fluids administered. In addition, the diagnostic accuracy of functional parameters is limited during open-chest surgery, non-sinus rhythm, spontaneous breathing, low tidal volume, right heart failure and intra-abdominal hypertension.[14,15]

Currently, GEDVI is one from the most reliable preload markers.[7] Of note, GEDVI is the summarised value of maximal volumes of all four heart chambers indexed to the calculated body surface area. It has been shown convincingly that GEDVI is more accurate for bedside preload assessment compared with central venous pressure (CVP), pulmonary artery occlusion pressure, right ventricular end-diastolic volume and left ventricular end-diastolic area.[7,16,17] In contrast to CVP, GEDVI is able to quantify preload in septic shock and severe acute respiratory distress syndrome (ARDS).[13,18] In many studies, GEDVI has been used as a reference to validate echocardiographic variables.[17,19] The accuracy of this parameter has been also confirmed in children and neonates.[20,21] This parameter remains plausible in patients with normovolaemia, moderate hypovolaemia, pulmonary hypertension and in those requiring inotropic support.[13,22,23] Moreover, GEDVI accurately characterises preload in both controlled mechanical ventilation and spontaneous breathing that may be important in perioperative

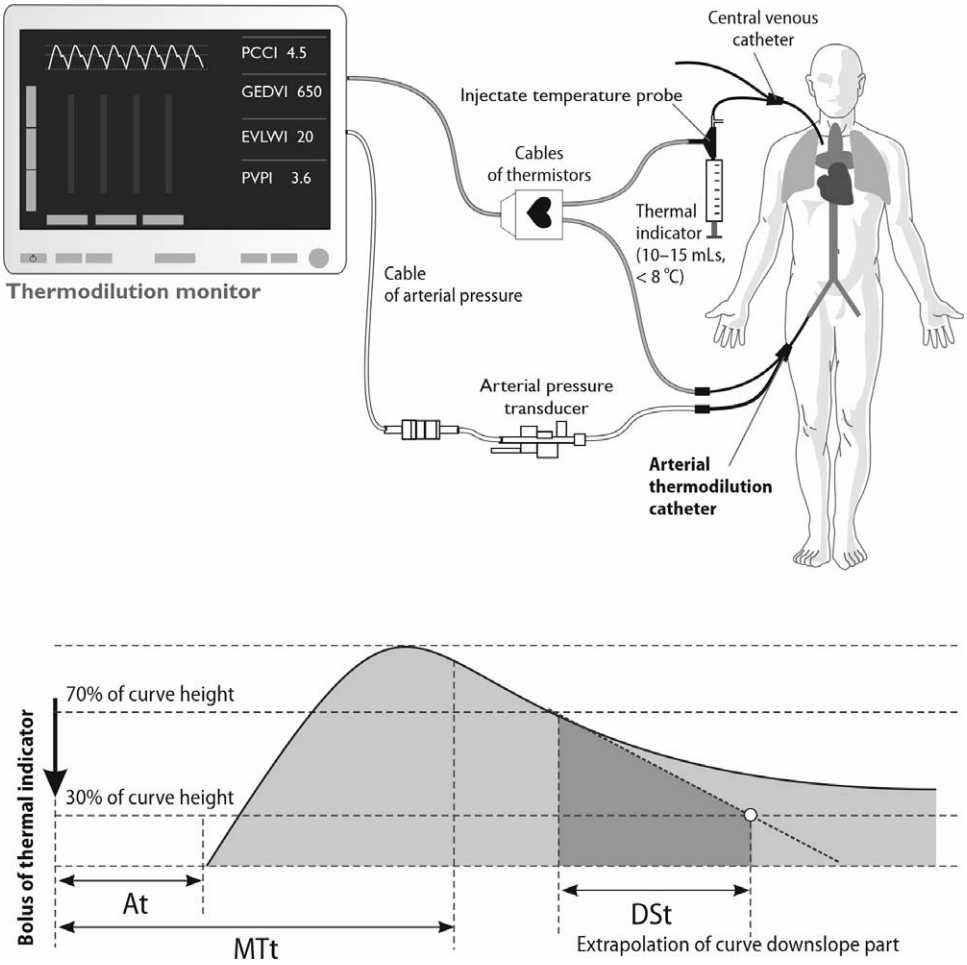

Figure 1. The layout of invasive volumetric monitoring and transpulmonary thermodilution curve. PCCI, pulse contour cardiac output; EVLWI, extravascular lung water index; GEDVI, global end-diastolic volume index; PVPI, pulmonary vascular permeability index; At, appearance time; MTt, mean transit time; DSt, down-slope time.

settings. The diagnostic value of GEDVI can be compromised in patients with aortic aneurism and prominent dilatation of left atrium; these factors can result in falsely increased GEDVI. The GEDVI interpretation can also be limited in severe left ventricular failure.[24] The interplay between GEDVI and another important volumetric parameter, EVLWI, during fluid resuscitation is of outmost clinical interest in many categories of ICU patients.[10,13] The methodology of GEDVI measurement is presented in Figure 1.

Global Ejection Fraction

GEF is important volumetric variable characterising heart performance, mainly, systolic function and myocardial work (stroke volume) under the actual preload condition. TPTD provides calculation of GEF as (4 × stroke volume)/GEDV, resulting in normal value of 25–35% (Table 1). Of note, the physiological meaning of GEF differs from the echocardiographic ejection fraction. Usually, the decrease in GEF results from the enlargement of heart chambers leading to increased GEDVI. In the case of systolic heart failure, both GEF and CFI are declining.[9] This volumetric parameter is useful to confirm clinically relevant heart failure. However, isolated right heart failure and pulmonary hypertension limit the diagnostic accuracy of GEF.[7,9] Despite these limitations, Nakwan et al.[25] have shown that in septic shock both GEF and CFI estimated by TPTD correlate with ejection fraction of left ventricle measured with echocardiography. In addition, GEF is strongly associated with the results of transoesophageal echocardiography in acute myocardial ischaemia.[19] When assessment of cardiac output is unaffected by differences in ventricular size and outflow obstruction, GEDVI, GEF and CFI do not reflect the largely increased heart volumes and markedly impaired left ventricular function in dilated cardiomyopathy.[26]

Cardiac Function Index

CFI can be calculated as a ratio of cardiac index and intrathoracic blood volume index (Figure 2). Thus, CFI independently characterises heart contractility under the current preload condition.[27] With normal values in the range of

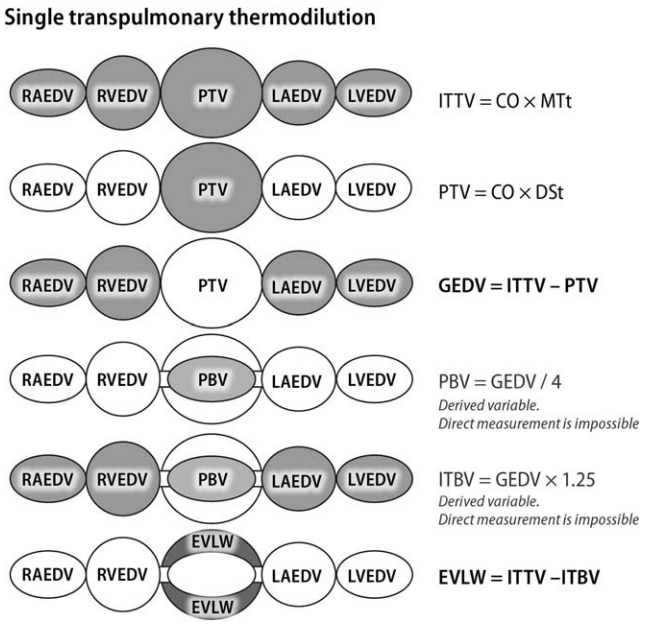

Figure 2. Physiological layout and calculation of the volumetric parameters using single transpulmonary thermodilution. EDV, end-diastolic volume; RA, right atrium; RV, right ventricle; LA, left atrium; LV, left ventricle; MTt, mean transit time of thermal indicator; DSt, down-slope time of thermodilution 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.

4.6–6.5 min^{-1}, this parameter is sensitive to inotropic support and the position of Frank–Starling curve.[19] It has been proposed that assessment of cardiac function by CFI using TPTD technique is a plausible alternative to the pulmonary catheter, and low CFI identifies cardiac dysfunction in both acute heart failure and sepsis.[28]

Pulmonary Oedema: Extravascular Lung Water Index and Pulmonary Vascular Permeability Index

EVLWI is a volumetric parameter quantifying pulmonary oedema.[2,5,29] This unique parameter can be of a great potential for the personalisation of intensive care and prevention of postoperative cardiopulmonary complications in the most complicated clinical scenarios (Figure 3). The complex patterns of the interplay of TPTD-derived volumetric parameters are proposed in Table 3. At this moment, TPTD is still referred to as a 'clinical gold standard' and a reference technique for EVLWI measurement despite strong competition from non-invasive methods, including lung ultrasound, and, possibly, computed tomography.[5,29,30]

EVLWI has been proved to be a useful guide to estimate pulmonary oedema and vascular permeability in sepsis, ARDS and heart failure.[5,31] Increased EVLWI can predict outcome and is strongly associated with the severity of ARDS.[32–34] In addition, EVLWI has a prognostic potential in shock, cardiothoracic surgery, transplantation, neurocritical care and other conditions (Table 2). Information about EVLWI and other volumetric variables might support decisions associated with decreasing duration of respiratory support and shortening ICU and hospital stays.[32,35,36] A personalised management based on EVLWI can reduce mortality in critically ill patients with increased EVLWI, as compared to treatment guided by Swan–Ganz catheter.[5,13,37] When integrated to treatment protocols, EVLWI has a potential to improve clinical outcome.[5,32]

Today, the best EVLWI cut-off value for discriminating diffuse alveolar damage is 10 mL kg^{-1},[32,38] and values exceeding 15 mL kg^{-1} correspond to severe ARDS with increased mortality.[32] The values between 8 and 10 mL kg^{-1} can be considered as belonging to 'a grey zone' (risk of ARDS).[38] The accuracy of TPTD and, therefore, EVLWI estimation can be influenced by the dynamic shifts in intrathoracic thermal conductivity and indicate 'heat leak' (myocardium and great vessels).[10,39] Inhomogeneous pulmonary oedema in ARDS, recirculation of indicator due to anatomical abnormalities and multiple other factors also might compromise the accuracy of readings[5,33] (Figure 3).

Pulmonary Oedema in the Perioperative Settings

The most suitable perioperative scenarios to implement volumetric monitoring include high-risk cardiothoracic,[37,40–42] transplantation,[43,44] neurosurgery[45] and major vascular interventions.[46] Off-pump coronary artery bypass grafting (CABG) has been shown to be associated with a decrease in

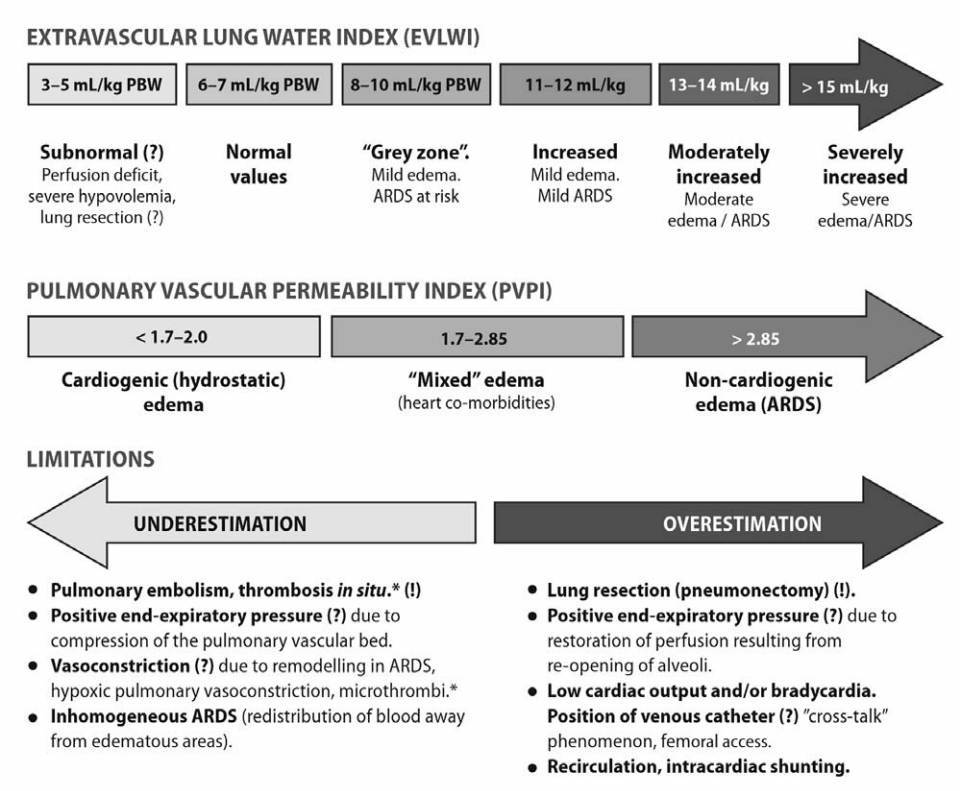

Figure 3. The interpretation and limitations of extravascular lung water and pulmonary vascular permeability index measurements. *Potentially, actual limitation in COVID-19-associated pulmonary embolism and/or pulmonary thrombosis in situ.

Table 3. The Volumetric Parameters in Different Critical Care and Perioperative Settings

Condition	Aetiology	Change in volumetric parameters
Severe hypovolaemia (haemorrhagic shock)	Haemorrhage, severe burns, decreased preload, high intrapleural pressure (pneumothorax)	Low GEDVI (usually $<600\,mL\,m^{-2}$), relatively low EVLWI ($4–7\,mL\,kg^{-1}$), low CO, low CFI, low GEF. Increase in GEDVI leads to rise in CO without early EVLWI accumulation
Pulmonary oedema/ARDS	Direct and indirect causes of ARDS (pneumonia, sepsis, shock, pancreatitis, etc.)	Increased EVLWI (usually above $10\,mL\,kg^{-1}$) and PVPI (usually above 2.5–3.0). Low-to normal GEDVI during early phase. Despite fluid responsiveness, attempts to increase GEDVI by giving fluids lead to rise in EVLWI, therefore posing the question about a 'permissive' hypovolaemia
Distributive shock	Septic shock, cardiopulmonary bypass	Increased EVLWI (sometimes even without ARDS criteria), normal-to-increased CO (hyperdynamic state), varying GEDVI (usually decreased during capillary leak). Normal GEF and CFI do not exclude diastolic heart dysfunction
Overhydration	Fluid overload or perioperative fluid accumulation, acute kidney injury, ARDS, lymphatic failure (sepsis, PEEP)	Normal or increased GEDVI. Increased EVLWI (usually above $10\,mL\,kg^{-1}$). No fluid responsiveness observed
Severe heart failure, cardiogenic shock	Structural changes leading to the decreased myocardial contractility, perioperative cardiac depression	Normal or increased GEDVI and 'grey zone' EVLWI ($7–10\,mL\,kg^{-1}$). In severe pulmonary oedema, EVLWI decreases after diuretics or positive pressure ventilation. Markedly decreased CO, CFI and GEF (below 20%)

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.

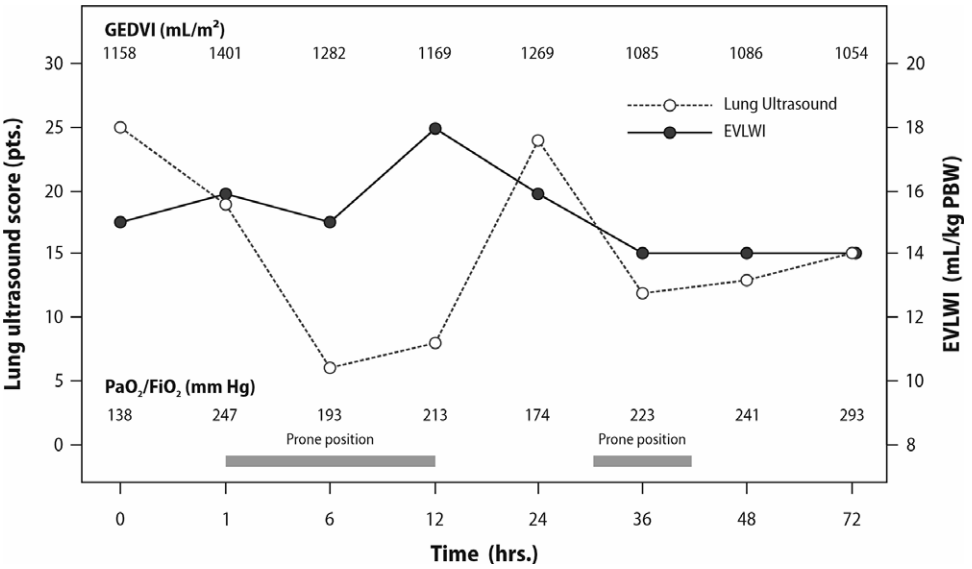

Figure 4. A clinical course of extravascular lung water index, lung ultrasound score, global end-diastolic volume index and oxygenation in a patient with severe COVID-19 associated with multiple organ failure, overhydration and severe heart failure. EVLWI, extravascular lung water index; GEDVI, global end-diastolic volume index.
An 80 years-old male with a history of arterial hypertension, myocardial ischaemia, chronic heart failure and chronic obstructive pulmonary disease has been transferred to the ICU for invasive mechanical ventilation on August 30, 2020 (3 days after the initial hospital admission) due to rapidly progressing bilateral viral pneumonia and respiratory failure related to the confirmed SARS-CoV-2. On the ICU admission, the increase in EVLWI (15 mL kg^{-1} PBW), corresponding to criteria of moderate ARDS and pulmonary oedema, has been registered. Of note, the increase in EVLWI and GEDVI was persistent despite fluid restriction, diuretics and negative cumulative fluid balance (–1100 mL) over the whole period of the ICU stay. EVLWI has reached peak of 18 mL kg^{-1} PBW at 12 hours after the ICU admission. Hypothetically, the actual EVLWI can be substantially underestimated under the conditions of perfusion deficit associated with the pulmonary embolism or COVID-19-related pulmonary microvascular thrombosis in situ. Phasic proning has resulted in a prominent yet transient increase in PaO_2/FiO_2 ratio, but not in any reproducible changes in the volumetric parameters tracked. Complex lung ultrasound assessment based on B-lines and consolidations has shown a somewhat delayed peak values that may reinforce an early warning role of EVLWI compared with 'beam' techniques. The observed refractory pulmonary oedema and overhydration can be explained by the severity of viral insult and concomitant heart failure with decreased cardiac output, associated with prominently increased GEDVI. Despite transient improvement of arterial oxygenation, on day 7 after the ICU admission the patient has died due to refractory shock and multiple organ failure.

EVLWI after revascularization.[40] In on-pump CABG, EVLWI >12 mL kg^{-1} requires diuretics to attenuate lung fluid accumulation; moreover, the haemodynamic optimisation based on CI and volumetric variables reduces requirements in vasopressors and inotropes and shortens the length of ICU stay.[41] Implementation of volumetric-based protocols targeted to oxygen transport can also be beneficial in high-risk cardiosurgical patients after complex valve surgery.[37]

Monitoring of EVLWI also might be useful in thoracic surgery including pulmonary and oesophageal resections and lung transplantation. The perioperative changes in EVLWI are helping to evaluate respiratory status of the patient and predict complications, including postpneumonectomy pulmonary oedema and hypoxaemia.[42,43]

Pulmonary Oedema in the Critically Ill

Pulmonary oedema is a hallmark of ARDS, thus bedside EVLWI quantification has a great potential to personalise and optimise both baseline and cumulative fluid balance as well as respiratory therapy.[5,29,34] The increase in EVLWI can be revealed before any substantial changes in blood gases and chest radiogram.[18] Kushimoto et al.[47] have demonstrated that ARDS grade based on the Berlin definition criteria was associated with EVLWI of 14.7, 16.2 and 20.0 mL kg^{-1} in mild, moderate and severe forms, respectively, while PVPI followed the same pattern with values of 2.6, 2.7 and 3.5. Being indexed to predicted body weight, EVLWI is increased in the vast majority of patients with ARDS,[18,48] and those with peak EVLWI exceeding 21 mL kg^{-1} and PVPI of more than 3.8 have a dismal survival below 30%.[33]

Of note, monitoring of EVLWI might be used in patients with cardiogenic pulmonary oedema and circulatory shock.[49,50] The values of EVLWI > 10 mL kg^{-1} associated with PVPI below 2.0 may indicate the hydrostatic mechanism of PE.[13,47] Currently, in severe COVID-19 accompanied by ARDS and myocardial dysfunction, the interpretation of EVLWI, PVPI and GEDVI has a great potential to distinguish between a predominant mechanism of lung oedema (non-cardiogenic, hydrostatic or mixed) and to tailor the personalised therapy[51] (Figure 4). Thus, Rasch

et al.[52] have shown that patients with severe coronavirus infection demonstrated significantly increased EVLWI and PVPI values (17 (11–38) mL kg^{-1} and 2.9 (1.0–5.2), respectively) compared with those with other ARDS aetiologies (11 (6–26) mL kg^{-1} and 1.9 (1.0–5.2), respectively), therefore reflecting progressing non-cardiogenic PE. In addition, we are awaiting the results of multicentre observational study on Haemodynamic Characteristics of Patients With SARS-CoV-2 (PiCCOVID study; NCT04337983).

Sepsis also requires a personalised approach to the fluid balance. Since Surviving Sepsis Campaign 2016[53] recommends invasive haemodynamic monitoring in septic shock, TPTD may increase the safety and efficacy of resuscitation and de-escalation phases of fluid management in these patients.[5,13,29] In sepsis, the rise in EVLWI predicts progression to ARDS approximately three days before the patients meet routine clinical criteria.[18,48] The increased EVLWI may be considered as an alert for avoiding unnecessary fluid load.[10] Thus, an increase in EVLWI in septic shock by more than 10% from baseline (or $\geq$10 mL kg^{-1}) can serve as a safety limit to stop fluid resuscitation[54,55] and apply protocols to reduce EVLWI.[13,55]

In addition, the combined monitoring of EVLWI and other volumetric parameters have been successfully studied in the ICU patients with severe burns,[56] subarachnoid haemorrhage,[57] necrotising pancreatitis,[58] multiple organ failure and renal replacement therapy.[59] It is important to note that the results of studies focussing on EVLWI, PVPI and other volumetric parameters are strongly dependent on protocol and personalised approach.

Conclusions

Today, the invasive volumetric monitoring has a clear potential to serve as an important haemodynamic puzzle in a wide range of ICU and perioperative scenarios. Assessment of volumetric parameters should be considered as an integral part of personalised phasic management of life-threatening organ failure and opens new perspectives to improve clinical outcome. The advantages of this technique include acceptable accuracy and reproducibility of measurements in the majority of critically ill patients. The bedside interpretation of volumetric cardiopulmonary parameters, including cardiac output, GEDVI, EVLWI and PVPI, provides reliable targets for the management of fluid balance and vascular permeability. This '3D' approach plays an invaluable diagnostic and prognostic role in ARDS, sepsis, circulatory shock and complicated perioperative period. Considering possible limitations, the volumetric monitoring has also a great potential in severe COVID-19.

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