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Intravascular volume monitoring and extravascular lung water in septic patients with pulmonary edema

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Abstract *Objective:* To evaluate whether different indicators using for guiding volume expansion are valuable tools to assess edematous lung injury in patients with septic shock. *Design and setting:* Prospective observational clinical study in a university intensive care unit. *Patients:* Sixteen consecutive mechanically ventilated patients developing septic shock with evidence of pulmonary edema on chest radiograph and severe hypoxemia (PaO_2/FIO_2 <250 mmHg). *Measurements and results:* A pulmonary artery catheter was used for the measurement of cardiac index (CI), central venous pressure (CVP), and pulmonary artery occlusion pressure (PAOP). A fiberoptic catheter was placed in the descending aorta. Measurements of extravascular lung water index (EVLWI), intrathoracic blood volume index (ITBVI), and total end-diastolic volume index (TEDVI) were obtained using the thermal dye dilution technique. Measurements were taken just after placement of catheters and 24 h later. Fluid balance was also estimated within the first 24 h. TEDVI and ITBVI were significantly correlated with EVLWI, but not CVP and PAOP. Analysis of 24-h changes showed that the changes in TEDVI and in ITBVI reflected the change in EVLWI, whereas PAOP, CVP, and fluid balance did not. *Conclusions:* Volume variables (TEDVI, ITBVI) are more useful indicators than pressure variables (CVP, PAOP) for assessment of EVLWI in septic patients with pulmonary edema.

Keywords Sepsis · Extravascular lung water · Total end-diastolic volume · Intrathoracic blood volume · Central venous pressure · Pulmonary artery occlusion pressure

Introduction

The cascade of events occurring during septic shock causes damage to endothelium, interstitium, and epithelium. Numerous studies focusing on the response of pulmonary microvessels to shock and resuscitation have shown marked structural changes. Alteration in microvascular permeability and marked interstitial edema occur, thickening the blood gas barrier and leading to disturbances of gas transport and subsequently to hypo-oxygenation of the tissues. Clinical data suggest that the intention to limit the amount of fluid accumulation within the lung positively influences the course of illness and improves outcome in patients suffering from permeability edema [1]. In hypotensive patients clinicians often try to balance the potential benefits of intravascular volume expansion on cardiac and renal function against the potentially negative effect of causing or worsening pulmonary edema. The primary goal is to correct deficits in tissue perfusion, reflected by alteration in oxygen consumption, by maintaining blood volume and cardiac output (CO). This can be achieved with volume loading or administration of vasoactive and inotropic drugs. Intravascular volume expansion in amounts large enough to

meet the goals of tissue perfusion may have deleterious effects on lung fluid balance. Appropriate monitoring of patients at risk of respiratory failure is essential to detect injury, to help prevent deterioration, and to follow the effect of therapy. This has been the rationale for assessing venous pressures in patients with cardiogenic edema and high permeability edema. Central venous pressure (CVP) and pulmonary artery occlusion pressure (PAOP) have been used as preload indicators to guide fluid therapy. Conceivably a reduction in hydrostatic pressures in the pulmonary circulation as judged by the measured PAOP could reduce the pressure gradient producing edema and promote clearing of lung water.

However, the pulmonary artery catheter has recently been criticized for lack of predictive value as guidance for fluid therapy. Numerous reports have demonstrated limitations of filling pressures as guides for monitoring of fluid volume expansion. In fact, in intensive care patients no correlation is found between PAOP and left ventricular end-diastolic volume (LVEDV) [2, 3, 4], which is usually defined as cardiac preload. Thus alternative methods for assessment of intravascular volume have been evaluated. Determination of circulatory blood volumes and CO with the less invasive transpulmonary double-indicator dilution (TDID) technique has gained increasing clinical acceptance. TDID estimates intrathoracic blood volume (ITBV) and total end-diastolic volume (TEDV); variables that have been shown to guide volume expansion more efficiently than CVP and PAOP. Extravascular lung water can also be determined, providing an estimate of the interstitial water in the lung.

This study tested whether CVP, PAPO, ITBV, and TEDV are useful for assessing edematous lung injury in patients with septic shock.

Materials and methods

Patients

Patients were recruited between November 1998 and December 1999. We prospectively studied 16 patients with septic shock (defined according to the criteria of the American College of Chest Physicians/Society of Critical Care Medicine consensus conference [5]) with evidence of pulmonary edema on chest radiography and severe hypoxemia (PaO_2/FIO_2 <250 mmHg). All patients were mechanically ventilated during the period of the study. Infusion of fentanyl and midazolam as sedative and analgesic drugs varied with the patient's current condition. Severity of illness at baseline was assessed using the Simplified Acute Physiology Score (SAPS) II [6]. The degree of organ dysfunction was assessed from clinical and laboratory parameters such as hemodynamic variables, dose of vasopressor therapy, ratio of PaO_2/FIO_2, bilirubin, creatinine platelet count, and Glasgow Coma Scale score. Sepsis-related Organ Failure Assessment (SOFA) score was calculated to assess the degree of organ dysfunction at the entry in the study [7]. The scoring system suggested by Murray et al. [8] was used to define the severity of lung injury. The Lung Injury Score (LIS) was calculated each day of the study. Chest radiography was performed with patients in the supine position within 2 h of an EVLW measurement using a routine mobile unit. At the same time PaO_2/FIO_2 ratios were recorded for all patients using arterial samples for determination of blood gas analysis. Fluid intake, gastric aspirates, diuresis, and insensible loss were recorded during the 24 h of the study.

For hemodynamic management and evaluation of lung edema a 7.5-F five-lumen pulmonary artery (pa) catheter was placed for the continuous measurement of cardiac index (CIpa), central venous pressure (CVP), mean pulmonary artery pressure (MPAP), and pulmonary artery occlusion pressure (PAOP). A 4-F fiberoptic catheter (Pulsiocath PV 2024, Pulsion Munich, Germany) was also placed in the descending aorta through a 6-F introducer in one of the femoral arteries. Mean arterial pressure (MAP) was recorded through the side port of the introducer sheath. Treatment of shock followed a standard protocol. All patients received fluid expansion starting before the pulmonary artery catheter was inserted. Once the pulmonary artery catheter was inserted, left ventricular preload was estimated from pulmonary artery occlusion pressure and was considered optimal when, at a given level, additional fluid infusion was no longer accompanied by an increase in cardiac index. If hypotension persisted after optimal fluid resuscitation, dopamine was started. The infusion rate was increased by 2–5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ until a maximum dose of 20 $\mu g \cdot kg^{-1} \cdot min^{-1}$. If shock persisted, hemodynamic status was assessed using the pulmonary artery catheter. For predominant cardiac failure, dopamine was combined with dobutamine. If the combination failed to reverse shock, epinephrine was infused alone. For predominant vascular failure, norepinephrine was combined with dobutamine or epinephrine was infused alone.

Measurements

Measurements of EVLWI, ITBVI, and TEDVI (total end-diastolic volume index (TEDVI) were obtained with the thermal dye dilution technique (COLD Z-021 system, Pulsion, Munich, Germany). Measurements were made by injecting 10–20 ml ice-cold indocyanine green solution (1 mg/ml) into the proximal port of the pulmonary artery catheter. The thermal dye dilution technique uses two indicators: ice-cold water and indocyanine green. Cold distributes to both intra- and extravascular volumes, whereas indocyanine green remains intravascular binding to plasma protein. Both indicators are injected into the right atrium and concentration changes over time are recorded in the descending aorta. All injections of indocyanine green were made manually after rapid transfer of the sirynge from the ice bath. Thus a dilution curve can be constructed for both indicators. Mean transit time and exponential downslope time of the thermal and dye indicators are recorded (Fig. 1). Cardiac index (CIfa) is determined from the thermodilution curve. The mean transit time of a suddenly injected indicator bolus between the site of injection and the site of detection multiplied by the cardiac output equals the total volume marked by the indicator. The ITBV calculation is based on the dye indicator curve, while the intrathoracic thermal volume (ITTV) is based on the thermal indicator curve. Multiplying the CIfa by the exponential downslope time of the thermodilution curve yields the pulmonary thermal volume, which is the largest single mixing volume. TEDV is obtained by subtracting the pulmonary thermal volume index from the intrathoracic thermal volume index (Fig. 1).

The EVLW and CO measurements were determined simultaneously. The average of at least three measurements that varied less than 10% were recorded. Hemodynamic and transpulmonary indicator dilution data were performed after placement of the catheters and again 24 h after the first measurement.

Statistical analysis

Characteristics of patients at H0 and H24 are given as mean ±SD. For comparison between the measurements of CIpa and CIfa we

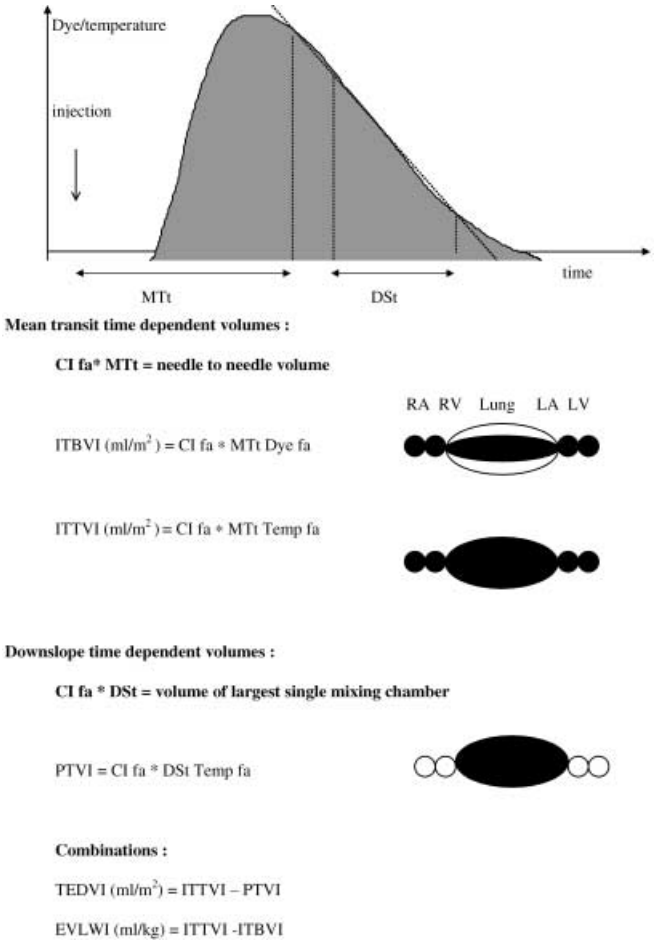

Fig. 1 Schematic femoral artery indicator dilution curve explaining mean transit time (*MTt*) and the exponential downslope time (*DSt*), which are used to calculate volumes by a femoral artery catheter in combination with the thermodilution cardiac output from the *dotted area under the curve*. *RA* Right atrium; *RV* right ventricle; *LA* left atrium; *LV* left ventricle; *CI fa* cardiac index based on femoral arterial thermodilution; *ITBVI* intrathoracic blood volume index; *ITTVI* intrathoracic thermal volume index; *PTVI* pulmonary thermal volume index; *Dye fa* dye dilution in femoral artery; *Temp fa* cold dilution in femoral artery

performed Student's *t* test. Changes in values at 24-h were reported with the prefix Δ. Linear regression analysis was used to evaluate the correlation between two variables. Differences at the level of $p<0.05$ were considered significant.

Results

Baseline characteristics

The 16 patients in the study included 13 men and 3 women, with a mean age of 59 years (range 34–75). Their SAPS II score was 56±18. All patients had clear evidence of sepsis: bacterial pneumonia in 14, abdominal sepsis in one, and septicemia due to catheter in one. They received vasopressor support (1 dopamine, 5 dobutamine, 8 norepinephrine, 7epinephrine). At H0 the mean SOFA score was 12.6±3.3 (Table 1). Three patients had two organ failures, nine patients had three, and four patients had four. Pulmonary edema was seen on radiography in all the patients. The mean LIS was 2.7±0.53 (range 1.3–3.6) corresponding to severe lung injury. At baseline the PaO_2/FIO_2 ratio was 128±46 mmHg. In the enrolled patients acute lung injury was secondary to pneumonia in 11 patients, gastric inhalation in 3, and sepsis in 2. The extravascular lung water index (EVLWI) was 17.3±8.8 ml/kg (normal range 5–10 ml/kg) and intrathoracic blood volume index (ITBVI) was 1115±330 ml/m² (normal range 800–1000 ml/m²; Table 1).

Table 1 Hemodynamic data and transpulmonary indicator dilution data from initial (H0) and 24 h (H24) measurements (mean ±SD) (*CI* cardiac index, *CVP* central venous pressure, *EVLWI* extravascular lung water index, *HR* heart rate, *ITBVI* intrathoracic blood volume index, *MAP* mean arterial pressure, *MPAP* mean pulmonary arterial pressure, *PAOP* pulmonary artery occlusion pressure, *TEDVI* total end-diastolic volume index)

Variable	H0	H24
HR (beats/min)	100±12	96±10
MAP (mmHg)	82±12	79±14
CIpa ($l\cdot min^{-1}\cdot m$)	3.5±1.2	3.4±1.1
CVP (mmHg)	12±6	11±5
PAOP (mmHg)	13±5	13±6
MPAP (mmHg)	31±7	30±6
TEDVI (ml/m²)	942±292	984±267
ITBVI (ml/m²)	1116±330	1193±301
EVLWI (ml/kg)	17.3±8	17±8.9
PaO_2/FIO_2 (mmHg)	128±46	125±18
Murray score	2.7±0.5	2.7±0.5

Correlation between cardiac output measurements and validation of measurements of ITBVI and TEDVI

Investigating ITBVI and TEDVI requires validation of CI by femoral arterial thermodilution (CIfa). To verify the reliability and reproducibility of CI fa, all simultaneous measurements of CIpa and CIfa were compared. The mean difference in CI was 0.003±0.4 ($p=0.97$).

Correlation between CVP, PAOP, TEDVI, ITBVI, and ELVWI

A total of 32 measurements were made during the 24-h study. No significant correlation between EVLWI and CVP or PAOP was found during the study (Figs. 2, 3). However, the linear correlation coefficient between EVLWI and the volumes variables TEDVI ($r=0.64$, $p<0.0001$) and ITBVI ($r=0.56$, $p<0.001$) was significant (Figs. 4, 5).

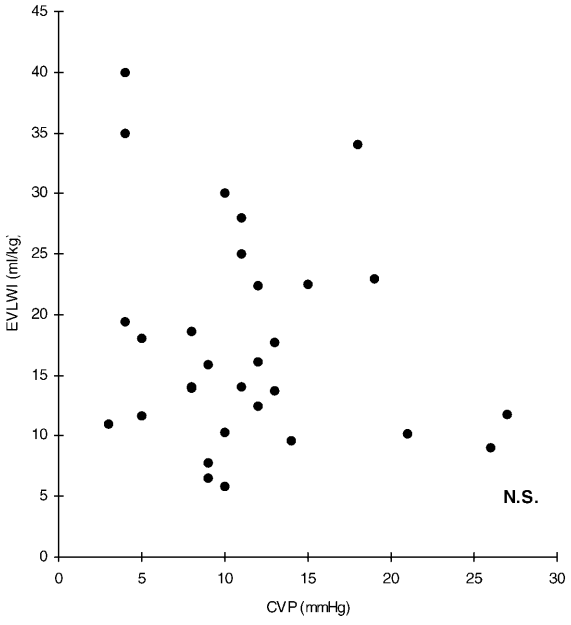

Fig. 2 Relation between CVP and EVLWI in 16 patients. *NS* non-significant

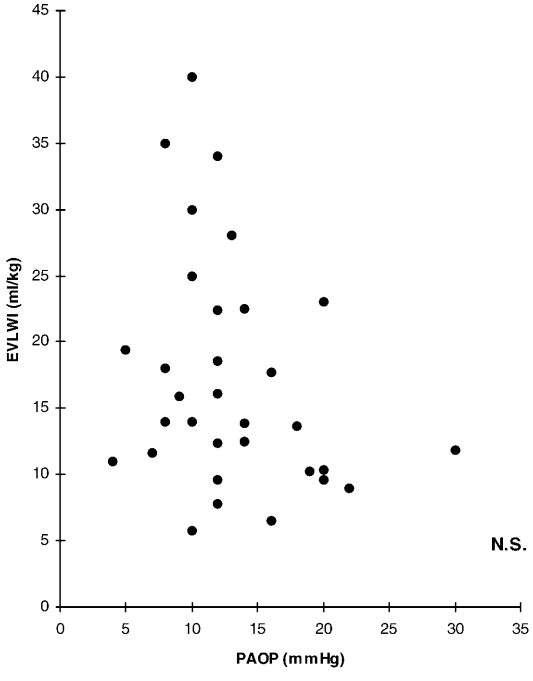

Fig. 3 Relation between PAOP and EVLWI in 16 patients. *NS* nonsignificant

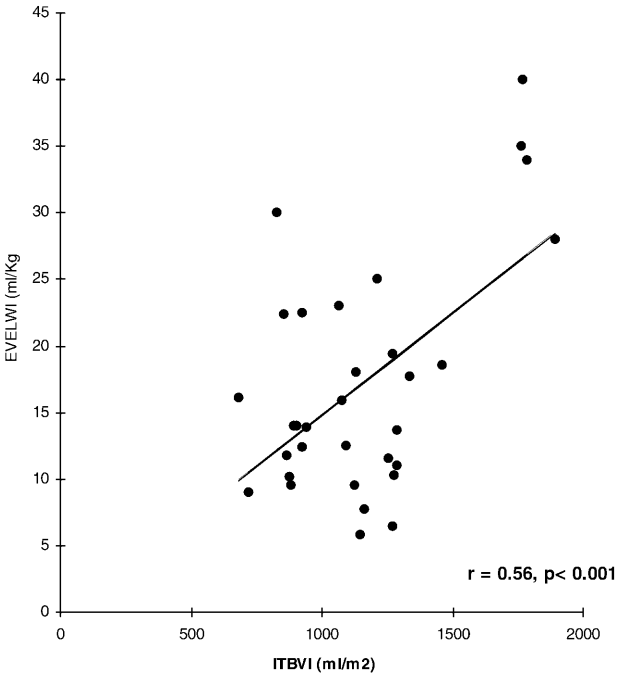

Fig. 4 The correlation between ITBVI and EVLWI in 16 patients

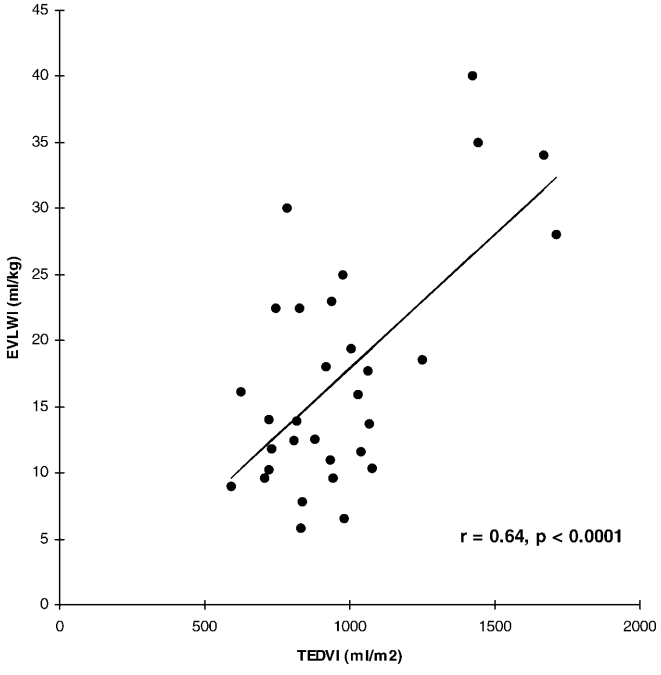

Fig. 5 The correlation between TEDVI and EVLWI in 16 patients

Correlation between fluid balance, ΔPAOP, ΔCVP, ΔTEDVI, ΔITBVI, and ΔEVLWI

For each patient the changes in EVLWI and pressure or volumes variables over the first 24 h are reported in Table 2. Table 3 shows the correlation coefficients of the regression analysis the 24-h differences in the variables. The mean fluid balance after 24 h was +1156±2320 ml. Three patients had negative balances and 13 positive balances. The change in fluid balance was not correlated

Table 2 Changes (prefix Δ) in pressure or volume variables and EVLWI over the first 24 h for each patient (*CVP* central venous pressure, *EVLWI* extravascular lung water index, *FB* fluid balance, *ITBVI* intrathoracic blood volume index, *PAOP* pulmonary artery occlusion pressure, *TEDVI* total end-diastolic volume index)

Patient no.	ΔCVP	ΔPAOP	ΔTEDVI	ΔITBVI	ΔFB	ΔEVLWI
1	–5	–4	38	146	–4640	–3.1
2	–4	–4	95	220	1750	–2.1
3	0	2	–19	2	–1810	5
4	1	–2	–4	–17	1900	–2
5	2	6	345	445	1940	3.7
6	–1	–1	–72	17	540	–8.4
7	–7	–6	36	37	1170	5.5
8	–7	–7	–13	5	1260	–0.6
9	1	8	143	146	2240	2.8
10	2	2	43	24	870	–7.6
11	3	1	461	431	–910	9.4
12	–4	–6	–193	–139	1370	–0.5
13	5	13	38	21	4550	–1.3
14	–3	–3	220	146	5460	3.5
15	5	4	253	341	510	–0.2
16	–7	–2	–689	–573	1150	–9

Table 3 Correlation between change in EVLWI and change in pressure or volume variables (prefix Δ) over the first 24 h (*CVP* central venous pressure, *EVLWI* extravascular lung water index, *FB* fluid balance, *ITBVI* intrathoracic blood volume index, *PAOP* pulmonary artery occlusion pressure, *TEDVI* total end-diastolic volume index, *NS* nonsignificant)

	r	*p*
ΔCVP	0.22	NS
ΔPAOP	0.1	NS
ΔITBVI	0.6	<0.01
ΔTEDVI	0.68	<0.005
ΔFB	0.3	NS

with ΔEVLWI during the 24 h. The 24-h differences in ITBVI (r=0.6, p<0.01) and TEDVI (r=0.68, p<0.005) were significantly correlated with ΔEVLWI. No significant correlation was found between ΔCVP, ΔPAOP, and ΔEVLWI.

Correlation between LIS and EVLWI

During the 2 days of study EVLWI values were not correlated with LIS (r=0.2).

Discussion

Our study tested whether various indicators using for monitoring volume expansion are useful in assessing edematous lung injury in patients with septic shock and pulmonary edema. The performance of various pressure (CVP, PAOP) and volume (ITBVI, TEDVI) variables was assessed by time correlation analysis. We found no correlation between PAOP, CVP, and EVLWI, but ITBVI and TEDVI were significantly correlated with EVLWI.

Measurements of EVLW, ITBVI, and TEDVI

We measured EVLWI using a commercially available thermal dye dilution technique. A close correlation has been shown between the thermal dye EVLW and EVLW measured by gravimetric techniques in both hydrostatic [9] and permeability pulmonary edema [10] in both human [11] and animal studies [12]. With the development of small bedside computers the measurement of EVLW by thermal dye dilution has become easy, and changes in lung water can be reliably detected. Currently the COLD Z-021 System, which employs a thermistor-tipped, fiberoptic femoral artery catheter to detect the thermal and optical signals in situ, allows more accurate measurements of EVLW. Before investigating ITBVI and TEDVI, the underlying measurement of CI by femoral arterial thermodilution is validated by a direct comparison to standard pulmonary artery thermodilution. Our results showed a close agreement between CIfa and CIpa.

ITBVI and TEDVI as indicators for intravascular volume status

Since the PAOP reflects the left ventricular end-diastolic pressure (LVEDP), it may reliably indicate cardiac preload in spontaneously breathing patients [13]. However, in mechanically ventilated patients PAOP and LVEDP are both affected by factors other than the LVEDV. In particular, changes in intrathoracic pressure or myocardial compliance may be responsible for the incorrect estimation of cardiac preload by affecting the relationship between PAOP and LVEDV, and between LVEDP and LVEDV. An alternative approach is to use ITBV and TEDV, which are less influenced by changes in intrathoracic pressures [14] and myocardial compliance [15] to guide fluid administration. Several studies [14, 16, 17,

18] have demonstrated the value of ITBV and TEDV comparing to the filling pressures CVP and PAOP in patients undergoing mechanical ventilation. Sakka et al. [19] compared the value of each variable in the early phase of hemodynamic stabilization in 57 critically ill patients with sepsis or septic shock who were receiving dobutamine and norepinephrine infusions. There was a close correlation between ITBV and stroke volume but no relationship between conventional cardiac filling pressures and stroke volume. Recently patients with life-threatening burns [20] were managed using ITBV to guide volume therapy. In these patients augmentation of ITBV was significantly correlated with changes in CI and oxygen transport. No such correlation was observed for the conventional preload parameters such as CVP and PAOP.

Correlation between pressure variables (CVP, PAOP), volume variables (ITBVI, TEDVI), and EVLWI

As shown by Mitchell et al. [21], management of pulmonary edema can improve outcome of ICU patients. A randomized trial randomized 52 patients requiring pulmonary artery catheterization to an EVLW management group and the other 49 patients PAOP management with fluid management decisions being guided by PAOP measurements. In the PAOP-guided group an upper limit of 18 mmHg was used and in the EVLW group an upper limit of 7 ml/kg. In the latter, EVLW decreased significantly, and ventilator-days and ICU days were significantly shorter than in the PAOP group.

Pulmonary artery catheter are used routinely in the management of septic patients with pulmonary edema. The PAOP is measured as a reflection of the adequacy of cardiac filling and the likelihood of the development of pulmonary edema. To our knowledge, our study is the first considering the relationship between PAOP or CVP and EVLWI in septic patients with pulmonary edema. We found no correlation between CVP or PAOP and EVLW index. This has been observed in a previous study in patients with acute cardiogenic pulmonary edema who required mechanical ventilation [22]. In this study Bindels et al. [22] reported the value of PAOP and the EVLW index. The pulmonary edema estimated from the EVLW index, resolved rapidly when cardiac performance improved despite no PAOP change. Similarly, and increased EVLWI was found in patients with acute myocardial infarction despite low PAOP [23]. Considering the results of these studies, we wonder whether the usual cardiac preload indices of PAOP and CVP are useful in the management of patients with pulmonary edema. First, it is clear from the Starling equation that the hydrostatic pressure is one among several factors influencing the development of pulmonary edema. Moreover, PAOP represents the pressure in the pulmonary venous circulation. Therefore venous dilatation or constriction affects the PAOP. Secondly, the sensitivity of PAOP as a measure of cardiac preload is increasingly controversial.

Correlation between LIS and EVLWI

Radiographic findings provided only belated and unreliable information about the fluid content of the lung in our patients. This is consistent with the findings of previous reports [24, 25].

Correlation between fluid balance and EVLWI

In our study changes in EVLW index were not correlated with fluid balance. Bindens et al. [22] also found patients with acute cardiogenic pulmonary edema to have positive fluid balances and weight gain in the first 24 h despite decreases in EVLW index. Clearly, net input-output represents the fluid balance from all body compartments and not only the lung. There is no a priori reason for instance why a given amount of diuresis in a patient with peripheral edema (indicating total body salt and water overload) should lead to the same change in EVLW as in a patient without peripheral edema. Similarly, in the injured lung it is not clear how changes in EVLW would follow fluid balance.

In conclusion, our study in septic patients with pulmonary edema showed that radiographic findings, fluid balance or pressure variables such as CVP and PAOP provide unreliable information about the contents of the lung. ITBV and TEDV were superior in the assessment of pulmonary edema. The measurement of ITBVI, TEDVI and lung water provided direct information about two of the major endpoints of hemodynamic management: intravascular volume status and lung edema. Additional studies are required to evaluate whether a therapeutic strategy taking into account ITBV, TEDV, and EVLW can improve supportive therapy and outcome of these patients.

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