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Originals

Intrathoracic blood volume accurately reflects circulatory volume status in critically ill patients with mechanical ventilation

M. Lichtwarck-Aschoff[1], J. Zeravik[1] and U. J. Pfeiffer[2]

[1] Institute of Anesthesiology and Surgical Intensive Care Medicine, Zentralklinikum Augsburg, Stenglinstrasse 2, W-8900 Augsburg, FRG
[2] Institute of Experimental Surgery, Technical University of Munich, Ismaningerstrasse 22, W-8000 Munich 80, FRG

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Abstract. Positive pressure ventilation in patients with acute respiratory failure (ARF) may render the interpretation of central venous pressure (CVP) or pulmonary wedge pressure (PCWP) difficult as indicators of circulating volume. The preload component of cardiac (CI) and stroke index (SI) is also influenced by the increased intrathoracic pressures of positive pressure ventilation. Moreover CI and SI do not indicate volume status exclusively but also contractility and afterload. We investigated whether intrathoracic blood volume (ITBV) more accurately reflects blood volume status and the resulting oxygen transport (DO_2). CVP, PCWP, cardiac (CI) and stroke index (SI) were measured, oxygen transport index (DO_2I) and oxygen consumption index (VO_2I) were calculated in 21 ARF-patients. Ventilatory patterns were adjusted as necessary. CI, SI and intrathoracic blood volume index (ITBVI) were derived from thermal dye dilution curves which were detected with a 5 F fiberoptic thermistor femoral artery catheter and fed into a thermal-dye-computer. All data were collected in intervals of 6 h. There were 224 data sets obtained. Linear regression analysis was performed between absolute values as well as between the 6 h changes (prefix Δ). The following correlation coefficients were determined: CVP/CI and PCWP/CI 0.01 and -0.142 ($p<0.05$); CVP/SI and PCWP/SI -0.108 and -0.228 ($p<0.01$); ITBVI/CI and ITBV/SI 0.488 ($p<0.01$) and 0.480 ($p<0.01$); ITBVI and DO_2I 0.460 ($p<0.01$); ΔCVP/ΔCI and ΔPCWP/ΔCI -0.069 and -0.018; ΔCVP/ΔSI and ΔPCWP/ΔSI -0.083 and -0.009; ΔITBVI/ΔCI and ΔITBVI/ΔSI 0.715 ($p<0.01$) and 0.646 ($p<0.01$); ΔITBVI and ΔDO_2I 0.707 ($p<0.01$). We conclude that in mechanically ventilated patients ITBV is a suitable indicator of circulating blood volume.

Key words: Double indicator dilution – Extravascular lung water – Intrathoracic blood volume – Mechanical ventilation – Cardiac filling pressures – Acute respiratory failure

The circulating blood volume is a major determinant of oxygen supply and can be influenced by therapeutic manoeuvres. The problem is how to measure it.

In clinical practice circulating blood volume status is most often estimated using cardiac output or the cardiac filling pressures CVP and PCWP as indicators.

Cardiac output can serve as a rough estimate because it reflects not only preload (i.e. adequate circulating blood volume) but also heart rate, contractility and afterload. Moreover as stressed in a recent editorial [1] thermodilution technique does not measure cardiac output (systemic blood flow) from the left ventricle (LV) but rather measures pulmonary arterial blood flow from the right ventricle (RV). The increased intrathoracic pressure (ITP) in positive-pressure ventilation in mechanically ventilated patients results in an inspiration-induced decrease in pulmonary arterial flow [2]. Thus estimates of cardiac output using thermodilution measurements of pulmonary arterial flow are very sensitive to ventilation-induced changes in flow and inaccurate as a parameter for systemic blood flows.

The cardiac filling pressures CVP and PCWP reliably indicate volume status in spontaneously breathing patients [3] but are additionally influenced, in mechanically ventilated patients, by the increased intrathoracic pressures. In spite of these difficulties we have to decide whether a patient needs volume or inotropic support. Looking for a "pure" volume indicator reflecting neither contractility nor increased intrathoracic pressure, we could demonstrate in experimental investigations, that the intrathoracic blood volume (ITBV) accurately reflects the volume status even if mechanical ventilation was used [4].

We now examine this relationship in critically ill patients with varying degrees of acute respiratory failure (ARF). We wanted to know, whether ITBV gave evidence of adequate volume status which CVP and PCWP could not give under these circumstances.

Patients and methods

The study was performed on 21 patients (mean age 36.2±13.4) with acute respiratory failure (ARF). Patients with pre-existing heart failure were excluded from the investigation. All patients were admitted to and cared for in the surgical intensive care unit. They had the following inju-

ries or diagnosis: multiple trauma ($n = 8$), neurologic trauma ($n = 4$), blunt chest trauma ($n = 2$), pneumonia ($n = 3$), peritonitis after abdominal surgery ($n = 4$).

The investigative protocol was approved by the institutional ethics committee, and consent was obtained from the patients of their next of kin.

Protocol

All patients had ARF of different extent and needed mechanical ventilation. Looking for the ventilatory pattern which was most adaequate for the patients actual condition 3 different modes of ventilation provided by the Servo Ventilator 900 C (Siemens-Elema, Solna, Sweden) were used in each patient: 1. Volume-cycled ventilation, 2. Combined high frequency ventilation (Servo 900 C and HF Unit; Siemens-Elema), 3. Spontaneous breathing with different levels of pressure support [4, 5]. Each ventilatory pattern was used for 6 h. Data were collected then and every 6 h after. These data were used to decide which ventilatory pattern was most helpful for the patient in terms of pulmonary gas exchange and oxygen transport. Due to the continuously changing situation of most patients this decision had to be re-evaluated several times. In that way one and the same patient was treated with at least 3 different ventilatory modes and in most cases one of these 3 modes had to be used repeatedly during the following 4 days. In 21 patients we got 224 data points for the absolute values and 199 for the Δ-values, i.e. the 6 h differences in the values. The use of sedative, analgesic or paralytic drugs was maintained unaltered. Also the use of positive inotropic drugs such as dopamine or dobutamine was not changed during the study period.

During the study the fluid balance including intravasular volume replacement was guided as usual, by CVP, PCWP and CI measurements using the "try and see" procedure.

Catheterization and measurements

A 7.5 F five-lumen Swan-Ganz pulmonary artery thermodilution catheter (Edwards Laboratories, Santa Ana, CA, USA) and a 5 F femoral artery catheter equiped with fiberoptics and thermistor (Pulsion Medizintechnik, München, FRG) were already placed in each patient for monitoring of CVP, MPAP, PCWP and MAP, for withdrawal of blood samples, and for the thermal dye recording as described below.

Thermal dye dilution: Both the pulmonary artery and arterial catheters were connected to an integrated fiberoptic monitoring system (COLD Z-02; Pulsion Medizintechnik) [6–10]. Glucose (10 ml, 5%) containing 0.75 mg/ml indocyanine green dye at 0°–4 °C was injected into the right atrium at a rate of 10 ml/s with a temperature-controlled pneumatic injector (ZI-03 Injector; Pulsion). The injection was started at end expiration, this moment being easily recognized by the ventilator's acoustic signals.

This system calculates the intrathoracic blood volume, ITBV and the extravascular thermal volume ETV besides other hemodynamic parameters. The dye and the thermal curve recorded in the femoral artery were used for the calculation of CO, ITBV, and ETV: The dye stays intravascularly during one passage through the cardiopulmonary system, whereas cold both diffuses and is convected into the extravascular space depending on time, vascular surface, heat conductivity and capacity. Thus an intravascular volume IVV_{MTT} and a thermal distribution volume TV_{MTT} can be calculated:

$$IVV_{MTT} = Q_D \times MTT_D \quad (1)$$

where Q_D is the dye dilution flow and MTT_D is the mean transit time of the dye indicator, and

$$TV_{MTT} = Q_T \times MTT_T \quad (2)$$

where Q_T is the thermodilution flow and MTT_T is the mean transit time of the thermal indicator. IVV_{MTT} corresponds to the intrathoracic blood volume ITBV, TV_{MTT} corresponds to the sum of the intravascular blood volume IVV_{MTT} and the extravascular heat accessible volume. Thus the extravascular thermal volume ETV_{MTT}, defined as

$$ETV_{MTT} = TV_{MTT} - IVV_{MTT} \quad (3)$$

can be calculated [8]. With the calculation of ETV the measurement of extravascular lung water (EVLW) as physical property for quantification of lung damage became possible.

The coefficient of variation of five successive ITBV determinations in these patients was determined to be 8.3%.

Hemodynamic and respiratory variables were calculated using the following formulas

$$CaO_2 = 1.39 \times Hb \times SaO_2 + PaO_2 \times 0.0031 \quad [ml\ O_2/100\ ml\ blood]$$

$$CvO_2 = 1.39 \times Hb \times SvO_2 + PvO_2 \times 0.0031 \quad [ml\ O_2/100\ ml\ blood]$$

$$SVRI = \frac{(MAP - CVP) \times 79.9}{CI} \quad [dyn \times s \times cm^{-5} \times m^{-2}]$$

$$PVRI = \frac{(MAP - PCWP) \times 79.9}{CI} \quad [dyn \times s \times cm^{-5} \times m^{-2}]$$

$$DO_2I = CaO_2 \times CI \times 10 \quad [ml/min \times m^2]$$

For the quantification of oxygenation PaO_2/FIO_2 was calculated.

Ventilatory variables

An estimate of the respiratory system total static compliance C_{st} was obtained by dividing V_T by the pressure difference ($P_{pause} - PEEP$). When the end-inspiratory and end-expiratory pressures were measured the hold functions of the Servo 900 C ventilator were used for 3 s i.e. no flow, pressure equilibrium in the lungs, airways and ventilator circuit was established.

With this method, C_{st} could only be determined during CPPV, or when switched to CPPV within the other ventilation modes at the same mean airway pressure (MPaw). MPaw and volume per minute were read from the ventilator display.

We did not measure esophageal pressure because of the methodological limitations of esophageal pressure as index of pressures surrounding the heart and because we wanted to get information with as little interference with clinical routine as possible.

Lung injury score (LIS)

A scoring system as suggested by Murray et al [11] was chosen in order to define the severity of lung injury. According to those authors LIS quantifies, albeit roughly, the presence, severity, and evolution of acute and chronic lung damage characterized by a chest radiograph (0–4 points), level of oxygenation (PaO_2/FIO_2, 0–4 points, PEEP (0–4 points) and compliance (0–4 points). The final value of LIS is obtained by dividing the aggregate sum by the number of components that were used. For determination of the chest radiograph score, the routine radiograph was used. A score of more than 2.5 implies ARDS, the most severe form of ARF.

The patients were placed in supine position at least 15 min before data collection was started. All pressure measurements were performed at end-expiration without removal of PEEP. The dye injections was started at the beginning of expiration. Thermal dye dilution derived variables were determined as mean of three successive measurements.

Statistical analysis

We compared the absolute values and the 6 h-differences of the variables (prefix Δ).

As the inotropic support was not changed during the study period it could be assumed that relevant changes of myocardial inotropic status as well as changes of afterload did not occur in these patients during the investigation. Validity of this assumption given, a change of a preload dependent variable would indicate a change of cardiac preload. Therefore linear regression analysis were performed between the

Table 1. Anthropometrie data of the patients (mean ± SEM)

LIS	2.25 ± 0.07
ETV (ml/kg)	16.7 ± 0.65
C_{stat} (ml/mbar)	49.7 ± 1.3
MP_{aw} (mbar)	20.4 ± 0.46
Min. Vol (l/min)	13.5 ± 1.2
$PaCO_2$ (mmHg)	40.8 ± 0.84
PaO_2/FiO_2 (mmHg)	215 ± 19.0
Hr (beats per min)	105 ± 1.0
CVP (mmHg)	12.7 ± 3.1
PCWP	16.1 ± 0.35
CI ($l/m^2 \times min$)	4.7 ± 0.9
SI ($ml/m^2 \times beat$)	44.7 ± 0.89
ITBVI (ml/m^2)	881 ± 14.0
DO_2I ($ml/m^2 \times min$)	812 ± 15.0

preload indicating variables CVP, PCWP, and ITBV versus the preload dependent variables SI, CI, DO_2, SvO_2, and SVRI, for absolute values as well as for 6 h-differences of the variables (prefix Δ). Results are presented as mean ± standard error of the mean (SEM).

Results

Table 1 gives the anthropometric data of the patients. Mean cardiac index was 4.7(±0.9) $l/min \times m^2$ and mean intrathoracic blood volume index 881 (±14.0) ml/m^2. This resulted in a mean oxygen delivery index of 812 (±15.0) $ml/m^2 \times m^2$.

Table 2 shows the correlation coefficients (R) of the regression analysis between the absolute values of CVP, PCWP, ITBVI and CI and SI. There was no correlation between CVP, PCWP and CI and SI, whereas ITBVI reveals correlations with CI ($r = 0.488$, $p < 0.01$) and SI ($r = 0.480$, $p < 0.01$).

Table 3 shows the correlation coefficients of the regression analysis of the 6 h-differences (Δ) of the above displayed variables plus the 6 h-differences of DO_2I and SvO_2. There exists neither correlation between ΔCVP and ΔCI and ΔSI, nor between ΔPCWP and ΔCI and ΔSI, but ΔITBVI correlates with ΔCI ($r = 0.714$, $P < 0.01$) and with ΔSI ($r = 0.646$, $P < 0.01$) as well as with ΔDO_2I ($r = 0.707$, $P < 0.01$). ΔSvO_2 shows correlation with none of them, ΔSVRI shows an $r = -0.49$, $P < 0.01$ to ΔITBV.

These findings are illustrated by the Figs. 1–3. Figure 1 shows the individual regression analysis for all patients for the relationship between the intrathoracic blood volume and $\Delta CI/\Delta DO_2I$. For the relationship ΔITBVI/ΔCI 70% of the patients show $r \geq 0.702$, for the $\Delta ITBVI/\Delta DO_2I$ 74% show $r \geq 0.705$. This confirms that the overall relationship which can be found when analyzing the complete data set of 199 data points is also true for the majority of the individual patients, the slope of the regression line varying from patient to patient with varying basic conditions.

Table 3. Correlation factor r for the relationship between the 6-h difference values (prefix Δ) ($n = 199$)

	ΔCVP	ΔPCWP	ΔITBVI
ΔCVP	–	0.267**	−0.086*
ΔPCWP	0.267**	–	−0.108
ΔITBVI	−0.086	0.108	–
ΔCI	−0.069	−0.018	0.714**
ΔSI	−0.083	−0.009	0.646**
ΔDO_2I	−0.047	−0.03	0.707**
ΔSVRI	−0.008	−0.074	−0.490**
ΔSvO_2	−0.079	−0.009	0.291**

*$p < 0.05$
**$p < 0.01$

Discussion

The validity and accuracy of the measurement of ITBV may be derived from the accuracy of the measurement of the extravascular lung water (EVLW), accurately estimated by extravascular thermal volume (ETV). ETV is calculated from the difference of total thoracic thermal volume and intrathoracic blood volume. In several experimental investigations ETV has been compared to post-mortem determined extravascular lung water and an excellent correlation has been found between EVLW and ETV. Others, and we ourselves, have shown that the measurement of ETV with the Pulsion Cold System was not dependent on flow, whilst a flow dependency has been shown for the Edwards Lung Water Computer [12–14]. From this we conclude that the determination of ITBV as a side product of the calculation of ETV must be as accurate as the ETV measurement.

Given the fact that in a recent investgation [15] the coefficient of variation for the thermodilution CO (six successive determinations) was found to be 3%–5% the

Table 2. Correlations factors r for the relationship between the absolute values ($n = 224$)

	CVP [mmHg]	PCWP [mmHg]	ITBVI [ml/m^2]	DO_2I [$ml/m^2 \times min$]	PAP [mmHg]
CVP [mmHg]	–	0.561**	−0.372**	−0.025	
PCWP [mmHg]	0.553**	–	−0.226**	−0.815**	
ITBVI [ml/m^2]	−0.372**	−0.226**	–	0.460**	−0.15*
CI [$l/m^2 \times min$]	−0.01	−0.412*	0.488**	0.935**	−0.024
SI [ml/m^2 beat]	−0.108	−0.228**	0.480**	0.865**	−0.191**
SvO_2 [%]			0.184*	0.47**	−0.366**

*$p < 0.05$
**$p < 0.01$

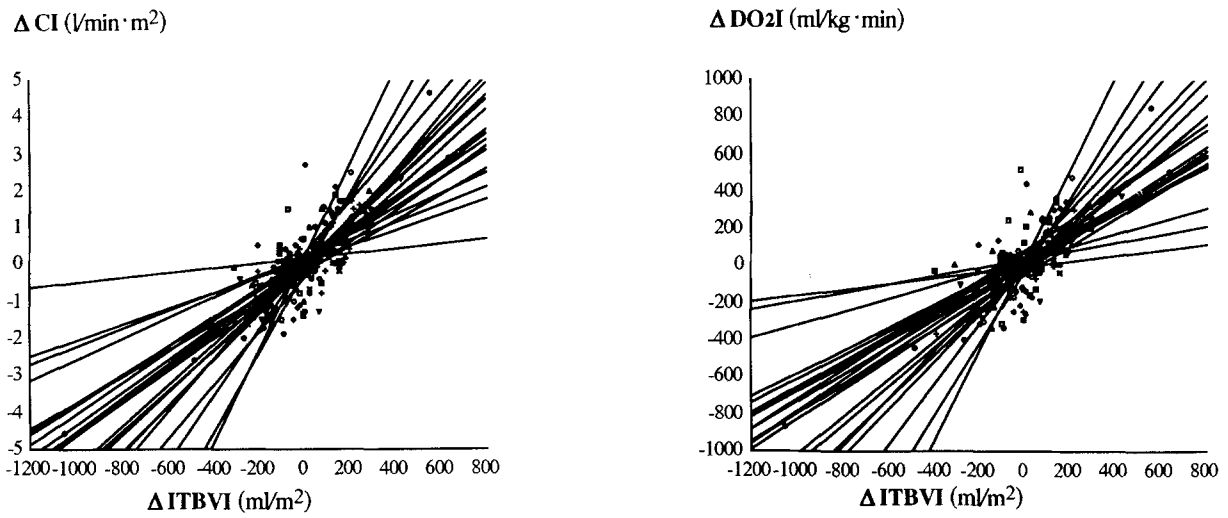

Fig. 1. Individual regression analyses for all patients for the relationship between intrathoracic blood volume index (ITBVI) given as the 6 h-differences of the values (prefixΔ) and the cardiac index (ΔCI) (*left*) as well as ΔITBVI to oxygen delivery index (ΔDO_2I) (*right*)

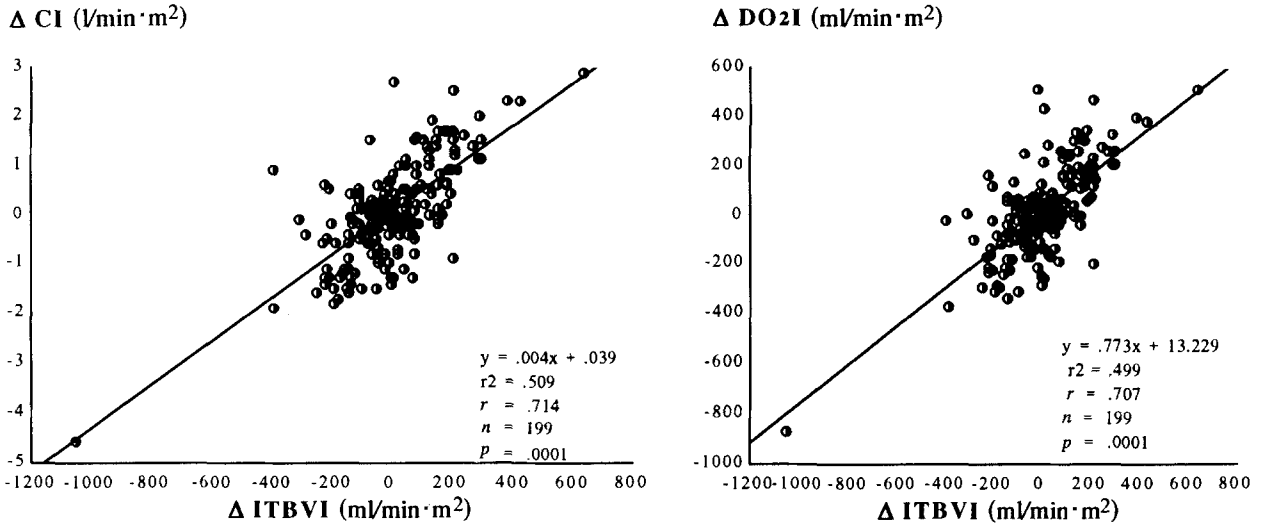

Fig. 2. Regression analyses for the relationship between Cardiac index given as the 6 h-differences of the values (ΔCI) and intrathoracic blood volume index (ITBVI) (*left*) and ΔITBVI and oxygen delivery index (ΔDO_2I) (*right*)

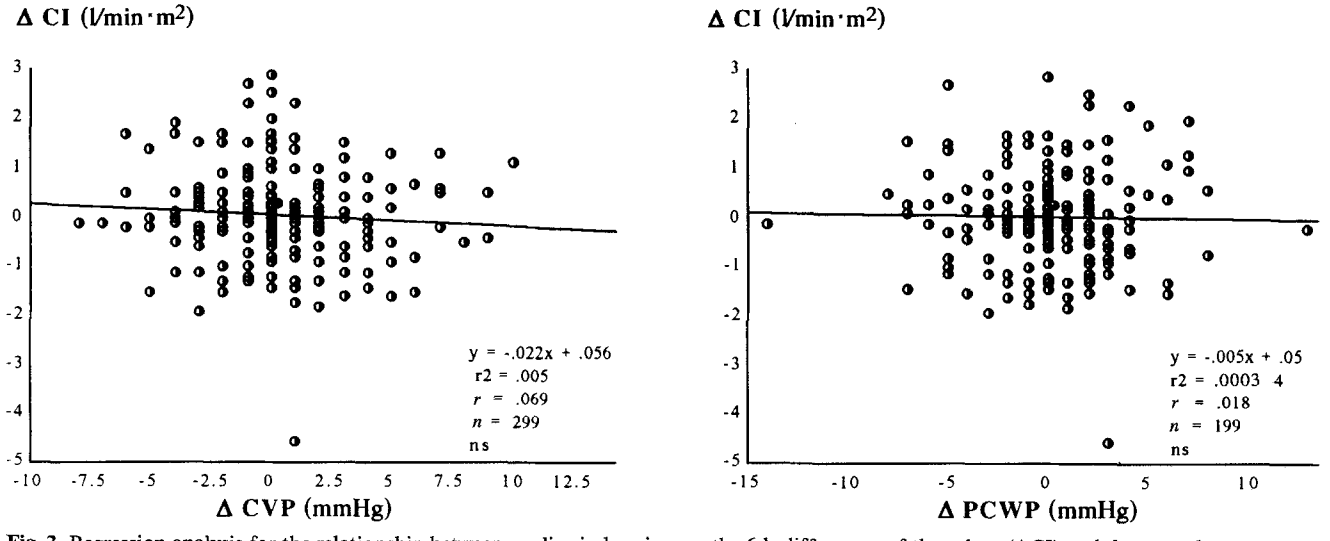

Fig. 3. Regression analysis for the relationship between cardiac index given as the 6 h-differences of the values (ΔCI) and the central venous pressure (ΔCVP) (*left*) and ΔCI and ΔPCWP (*right*)

8.3% variability of ITBV – measurement that we found (five successive determinations) is acceptable.

We should underline that determination of ITBV is not technically more difficult than a standard CO determination and that no additional catheter is needed because the femoral catheter that is usually already in place can be used.

The results obtained for ITBV agree quite well with results from Hedenstierna et al. [16] who, in ventilated patients without pulmonary edema, found 1410 (±120) ml ITBV which corresponds to estimated 700–750 ml ITBV per m^2 body surface area. Other results, although difficult to compare because obtained with different techniques and in spontaneously breathing subjects lie in a quite scattered range of 660–1000 ml/m^2 [17, 18].

Central venous pressure (CVP) and pulmonary capillary wedge pressure (PCWP) represent the intravascular pressure at a specific point in the circulation. In surgical cirtical care medicine both pressures are used as indicators of circulating volume status [3].

With the increase of intrathoracic pressure caused by mechanical ventilation with positive airway pressure a compression of the low pressure capacitance system occurs. CVP and PCWP measured against atmospheric pressure increase. Right ventricular filling is decreased because of reduced venous return, though an improvement of cardiac filling by the elevated CVP would be implied. Keeping this in mind clinicians often try to get an impression of circulatory filling with CVP and/or PCWP measurements during removal of PEEP ventilation [19–20]. It is of utmost importance to verify the changes of the cardic preload related to the changes of intrathoracic pressure. In fact exclusively the intrathoracic content of the total circulatory blood volume influenced by airway pressure contributes to oxygenation, hence the acutal intrathoracic blood volume is the major determinant of oxygen transport rather than the total circulating blood volume.

These findings based on theoretical as well as on experimental and clinical investigations emphazise more criticism on the appraisal of CVP and PCWP. In fact our results revealed neither correlations between CVP and CI nor between PCWP and CI and confirm the conclusions of other authors, who stated, that CVP and PCWP are convenient to measure, but have no predictive character as guiding variables for a sufficient volume supply [21–26].

In contrast we proved the good accordance of intrathoracic blood volume with CI and SI. This does not mean that for evaluation of the patients hemodynamic situation ITBV could or should be used instead of CI or SI. ITBV being a pure volume indicator correlates only with the preload component of CI and SI. In patients who maintain their CI mainly by high heart rate or high inotropic drive the correlation between ITBV and CI will of course be only loose. This is in agreement with the results of experimental studies claiming ITBV as useful guide for the management of circulatory filling [4].

Using a double-indicator method we obtained accurate values for cardiac index by basing the estimate of cardiac output on the computerized analysis of the arterial thermodilution curve. In contrast to the thermodilution in the pulmonary artery this technique measures both right and left ventricular output unaffected by the respirator induced cyclic variations in pulmonary blood flow [1].

Although we measured lactate we could not find peripheral perfusion deficits indicated by increasing lactate levels with decreasing ITBV. We feel this to be due to the quite different basic condition of our patients as far as sedation, temperature etc is concerned. Only SVRI showed a moderate correlation to ITBV, the systemic vascular resistance increasing when volume was low.

The different baseline conditions of our patients may also have blurred a presumably existing relationship between ITBV and SvO_2 and between ITBV and mean pulmonary artery pressure. ITBV did not correlate with mean pulmonary artery pressure and was only loosely correlated to SvO_2.

It is not only of theoretical interest, which indicators best reflect the cardiac preload and the circulating blood volume in mechanically ventilated patients. As preload is the main determinant of oxygen transport in many situations its exact determination is decisive in clinical practice. With the assessment of CVP and PCWP it is not possible to increase (and thus perhaps to optimize) DO_2, whereas this is possible with the measurement of ITBV as derived from the correlation between ITBV and DO_2.

We could demonstrate: (1) The cardiac filling pressures do not indicate the volume status, when patients are mechanically ventilated. CVP and PCWP mislead to an overestimation of circulatory volume status. (2) ITBV is a reliable tool for guiding cardiac ouput and reveals a significant correlation with DO_2.

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Dr. M. Lichtwarck-Aschoff
Institute of Anesthesiology and
Surgical Intensive Care Medicine
Zentralklinikum Augsburg
Stenglinstrasse 2
W-8900 Augsburg
FRG