Effect of pleural fluid on the measurement of extravascular lung water by single transpulmonary thermodilution

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Abstract

Objectives: To examine the reliability of the extravascular lung water value obtained by single transpulmonary thermodilution in the presence of a large amount of pleural fluid.

Design: Observational clinical study.

Setting: Medical intensive care unit of a tertiary teaching hospital.

Patients and participants: Patients in whom an evacuation of pleural fluid was performed and who had a central venous catheter and a thermistor-tipped arterial thermodilution catheter (PiCCOplus system, Pulsion Medical Systems, Munich, Germany) for haemodynamic management.

Interventions: None.

Measurements and results: We included eight patients (mean age 66 ± 7.9 years admitted to the medical intensive care unit with diagnoses of septic shock (n = 4, of which two patients had ARDS) and cardiogenic shock (n = 4). A total of 14 sets transpulmonary thermodilution measurements were obtained before and after evacuation of 765 ± 330 ml pleural fluid (some patients received more than one thoracocentesis). Data of the two samples were compared using the Wilcoxon matched pairs signed rank sum test. The extravascular lung water index increased from 11.3 ± 4.8 to 12.1 ± 4.9 ml/kg (p = 0.041) and the pulmonary vascular permeability index increased from 1.9 ± 0.8 to 2.1 ± 0.9 (p = 0.055).

Conclusions: Fluid in the pleural space does not contribute to the volume transversed by the thermal indicator (“cold”) in single transpulmonary thermodilution measurements and does not cause an overestimation of the extravascular lung water.

Introduction

The pulmonary artery occlusion pressure (PAOP) has been shown to be a poor indicator of oedema formation in both non-cardiogenic¹ and cardiogenic²,³ pulmonary oedema. A protocol using measurements of extravascular lung water (EVLW) instead of PAOP to guide fluid management in mixed patients requiring pulmonary artery catheterization was associated with reduced ventilator and intensive care unit (ICU) days and a lower cumulative fluid balance.⁴ Measurement of EVLW is more sensitive than chest X-ray for detecting pulmonary oedema.⁵–⁷ Furthermore, EVLW was found to be an independent predictor of survival (mortality increased with higher EVLW values).⁸ Therefore, an accurate measurement of EVLW is important.

Determination of EVLW by “single transpulmonary thermodilution” (meaning thermodilution with a single indicator) depends on measurement of the intrathoracic thermal volume (ITTV), which is the largest accessible volume transversed by the thermal indicator (“cold”). Theoretically, the distance from the thermal indicator in the pulmonary arterial vasculature and capillaries to pleural fluid is greater than the distance to the pulmonary oedema that we want to quantify. The aim of the present study was to exclude the possibility that large amounts of pleural fluid participate in this temperature exchange, and that they cause an overestimation of the EVLW. Only one study addressed this problem.
before: Blomqvist and colleagues showed that pleural fluid injection (up to 20 ml/kg) had no effect on EVLW measurement by transpulmonary double-indicator dilution in healthy dogs. To study this for the first time in critically ill humans, we performed transpulmonary thermal dilution determinations of EVLW before and after thoracocentesis and hypothesised that the EVLW value would not decrease. Part of this work was presented on the 33rd Congress of the Society of Critical Care Medicine in February 2004 in Orlando, Florida.

Materials and methods

Patients

Between September 2002 and July 2003, we included all patients (a) in whom an evacuation of pleural fluid was performed, and (b) who had a central venous catheter and a thermostar-tipped arterial thermodilution catheter (Pulsiocath 5F (femoral artery) or 4F (brachial artery) and PiCCO-plus system, Pulsion Medical Systems, Munich, Germany) for haemodynamic management. We merely analysed this existing situation and did nothing to influence events. The medical records were only accessed by the treating ICU physicians.

Thoracocentesis

A small chest tube (Arrow-Clarke™ Pleura-Seal® thoracocentesis set, 8 Fr, 12 cm length, Arrow International, Reading PA, USA) was placed in the fifth intercostal space just anterior to the mid-axillary line. The interval between two sets of thermodilution determinations (and hence the time for evacuation of pleural fluid) was two to four hours.

Thermodilution measurements

“Single transpulmonary thermodilution” measurements were obtained by central venous injection of 20 ml of “iced” (< 4°C) 5% dextrose in water and carried out by ICU nurses who were not aware of the study purpose. For each data measurement point, a set (of three injections) of thermodilution determinations was performed before, and after thoracocentesis, in the same body position. For each set of thermodilution determinations, the calculated mean values were used for haemodynamic management and statistical analysis. The cardiac output (CO) was determined by thermodilution using the Stewart-Hamilton method.11,12 Calculations were carried out with the following equations using a computer system (PiCCO plus, Pulsion Medical System, Munich, Germany).12 ITTV and pulmonary thermal volume (PTV) were respectively calculated from the mean transit time (MT) and the exponential downslope time (DS) of the thermodilution curve: ITTV = CO*MT and PTV = CO*DS. The ITTV consists of the PTV and the sum of the end-diastolic volumes of all cardiac chambers. Accordingly, the global end-diastolic volume (GEDV) was calculated as: GEDV = ITTV − PTV. Based on a linear relation between GEDV and intrathoracic blood volume (ITBV), ITBV = 1.25*GEDV. Since EVLW is the difference between ITTV and ITBV, EVLW = ITTV − ITBV. Pulmonary blood volume (PBV), pulmonary vascular permeability index (PVPI), stroke volume (SV) and global ejection fraction (GEF) were derived from these values: PBV = ITBV − GEDV; PVPI = EVLWI/PBV; SV = CO/heart rate and GEF = (4*SV)/GEDV. Absolute values for CO, GEDV, ITBV and SV were normalized as indexed by body surface area (CI, GEDVI, ITBVI and SVI) and for EVLW by body weight (EVLWI).

Statistical analysis

Because of the small sample size, it was not safe to assume that the sampling distribution of the population was normal. Therefore, we used the (non-parametric) Wilcoxon matched pairs signed rank sum test to compare the values before and after thoracocentesis.13 All values are given as mean ± standard deviation.

Results

Eight critically ill patients (five male, three female, mean age 66 ± 7.9 years) were included. Their APACHE-II score was 22.5 ± 12.5, SAPS-II score 50.1 ± 16.8, MODS score 4.6 ± 3.2 and SOFA score 7 ± 4.6. The patients were admitted to the medical intensive care unit with diagnoses of septic shock (n = 4, of which two patients had ARDS) and cardiogenic shock (n = 4). The amount of pleural fluid evacuated was 765 ± 330 ml. Since several patients received more than one thoracocentesis, in total 14 sets of measurements were obtained. The patient was invasively ventilated in 13 sets (fully sedated, no spontaneous breathing and continuous positive pressure ventilation in nine) and non-invasively in one.

The transpulmonary thermodilution and other haemodynamic measurements, and arterial blood gas values before and after thoracocentesis are listed in Table 1 and Table 2. The EVLWI increased after thoracocentesis from 11.3 ± 4.8 to 12.1 ± 4.9 (p = 0.041).
Discussion

The main finding of this study is that the EVLWI, obtained by single transpulmonary thermodilution, did not decrease after evacuation of a large amount of pleural fluid. This correlates well with the findings of Blomqvist and colleagues. 9

So, did thoracocenthesis cause any change in thermodilution measurements? Remarkably, we found slightly higher values for EVLWI (p = 0.041) and PVPI (p = 0.055) after thoracocenthesis. These findings are compatible with an increased PTV. Possibly this was caused by mild reexpansion pulmonary edema, or by the opening of areas of compression atelectasis after thoracocenthesis, or both. In dog models, Gray and Carlile et al., and Oppenheimer et al. showed that the double indicator (thermo-dye) dilution technique may underestimate EVLW in lung zones that are less perfused.14−16 Therefore it seems possible that in areas of compression atelectasis the EVLW is underestimated. On the other hand, the pCO2 and pO2/FiO2 ratio were not changed after thoracocenthesis.

We have no explanation for the significant increase in CVP. There was no change in body position and we did not administer fluids during thoracocenthesis.

This study, although the first to investigate the effect of thoracocenthesis on EVLW measurement in humans, is limited by its small sample size. Furthermore, we neither investigated the hemodynamic effects of thoracocenthesis over the following hours, nor the short-term or long-term benefits of thoracocenthesis in the critically ill, as this was beyond the scope of this study. A larger study that focuses on the ventilatory and haemodynamic effects of thoracocenthesis is needed.

In conclusion, although we can not say that pleural fluid has no effect on the measurement of EVLW, this study demonstrated that fluid in the pleural space does not contribute to the volume transversed by the thermal indicator (“cold”) in single transpulmonary thermodilution measurements and does not cause an overestimation of the EVLW.

Acknowledgements

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References


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Table 1. Transpulmonary thermodilution measurements before and after thoracocenthesis

<table>
<thead>
<tr>
<th></th>
<th>Before thoracocenthesis</th>
<th>After thoracocenthesis</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>EVLWI (ml/kg)</td>
<td>11.3±4.8</td>
<td>12.1±4.9</td>
<td>0.041</td>
</tr>
<tr>
<td>PVPI</td>
<td>1.9±0.8</td>
<td>2.1±0.9</td>
<td>0.055</td>
</tr>
<tr>
<td>ITBVI (ml/m²)</td>
<td>1196.9±253</td>
<td>1181.8±249.7</td>
<td>0.875</td>
</tr>
<tr>
<td>Cl (l/min/m²)</td>
<td>4±1.1</td>
<td>4±1.2</td>
<td>0.857</td>
</tr>
<tr>
<td>GEF (%)</td>
<td>21.3±5.4</td>
<td>20.9±3.7</td>
<td>0.752</td>
</tr>
<tr>
<td>SVI (ml/m²)</td>
<td>49.8±11.1</td>
<td>49.1±12.2</td>
<td>0.730</td>
</tr>
</tbody>
</table>

EVLWI, extravascular lung water index; PVPI, pulmonary vascular permeability index; ITBVI, intrathoracic blood volume index; Cl, cardiac index; GEF, global ejection fraction; SVI, stroke volume index.

Table 2. Vital parameters, mean arterial and central venous pressure, and arterial blood gas values before and after thoracocenthesis. MAP, mean arterial pressure; CVP, central venous pressure

<table>
<thead>
<tr>
<th></th>
<th>Before thoracocenthesis</th>
<th>After thoracocenthesis</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (/min)</td>
<td>82±18.3</td>
<td>82.1±19</td>
<td>0.972</td>
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<tr>
<td>MAP (mmHg)</td>
<td>86.2±17.8</td>
<td>85.2±14.9</td>
<td>0.950</td>
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<tr>
<td>CVP (mmHg)</td>
<td>9.9±4.1</td>
<td>12.2±4</td>
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<td>pCO2 (mmHg)</td>
<td>47.1±7.1</td>
<td>45.3±7.3</td>
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<tr>
<td>pO2/FiO2 (mmHg)</td>
<td>219.3±68.3</td>
<td>223.3±93.2</td>
<td>0.477</td>
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