Transoesophageal echocardiography in the management of whole lung lavage

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Summary
The effects of two lung ventilation, one lung ventilation and alveolar lavage on right ventricular mechanics in a patient have been observed using transoesophageal echocardiography (TOE). Alveolar lavage resulted in additional stresses to those of one lung ventilation (OLV) and the point when these were noted to be resulting in right ventricular changes was used as the signal to terminate the filling phase of a cycle of alveolar lavage. A significant increase in right ventricle afterload occurred on starting OLV. The response to lung lavage was biphasic, initially a further increase in afterload followed by a reduction in preload. Because there was rapid adaptation to these changes, it was felt appropriate to perform sequential lung lavage during the same anaesthetic. In similar circumstances where pulmonary vascular resistance is changed, information obtained from TOE may be used to guide therapy. (Br. J. Anaesth. 1998; 81: 262–264).

Keywords: measurement techniques, transoesophageal echocardiography; lung, lavage

Patients with increased pulmonary vascular resistance, secondary to chronic lung disease, may develop right heart dysfunction with disease progression or as a result of medical intervention, such as positive pressure ventilation and single lung ventilation. We have observed the right ventricular responses to the early effects of three interventions in a patient with a normal heart.

Case report
A 37-yr-old female with a history of pulmonary alveolar proteinosis (PAP), presented for a third session of whole lung lavage (WLL) because of deteriorating lung function. There was no clinical, electro- or echocardiographic evidence of associated right ventricular dysfunction. After her consent, we attempted to monitor the process and assess the risks of proceeding to a sequential single lung lavage during the same anaesthetic using transoesophageal echocardiography (TOE).

Induction of anaesthesia was with propofol, fentanyl and vecuronium, and a left-sided double lumen tube placed. A left radial artery cannula was inserted. Maintenance of anaesthesia was with propofol infusion and a minute ventilation of 100 ml kg⁻¹ during all stages. With the patient supine, a 5MHz multiplane transoesophageal probe, connected to a Hewlett Packard Sonos 1500 imaging system was passed into her oesophagus. The probe was advanced into the stomach until the standard short axis view of the left ventricle at the level of the papillary muscles was obtained. This was used as the mark for the short axis image of the right ventricle.

Observations were made when the patient was stable during two lung positive pressure ventilation, during left single lung ventilation (SLV), and throughout lavage of the right lung. Automated detection of the endocardial border (ABD) allowed real-time display of the data for right ventricular end systolic area (ESA), end diastolic area (EDA) and fractional area change (FAC), calculated on line as FAC = (EDA − ESA)/EDA. One representative ventilation cycle during each stage of ventilation was identified and the value of each variable was taken for every heart beat during the full ventilation cycle (from peak inspiration). Normality of data was evaluated using the Anderson-Darling test and the means, calculated for each cycle, were compared using the two-tailed Student t test (Minitab Inc., Release 11.2).

Results
The means of EDA, ESA, and FAC are tabulated (table 1) and reported as representative values for right ventricular function during baseline two lung ventilation, during change to OLV (transition period—on acute clamping of double lumen tube), and established on OLV before right lung lavage.

Figure 1 summarizes the changes that were observed during the first lavage cycle of 1 litre of saline. Again, the mean of each variable is shown, as calculated for one ventilation cycle from each 15 s as the lavage progressed, and for the first 30 s of drainage after filling the lung with warmed, buffered normal saline.

Systolic arterial pressure decreased from 136 mm Hg on OLV to 78 mm Hg at the end of the first filling phase, with recovery to 124 mm Hg on emptying the right lung. Peripheral oxygen saturation was 99% throughout both phases.

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Data at 60 s are missing because it is when physiotherapy at that time.

Table 1  Changes in right ventricular dynamics as left single lung ventilation is established. (**P<0.01 compared with two lung values)

<table>
<thead>
<tr>
<th>Stage</th>
<th>Mean EDA (cm²)</th>
<th>Mean ESA (cm²)</th>
<th>Mean FAC (% difference from two lung values)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Two lung IPPV</td>
<td>6.5</td>
<td>4.8</td>
<td>26.2%</td>
</tr>
<tr>
<td>Range</td>
<td>(5.8–7.0)</td>
<td>(4.0–5.4)</td>
<td>(22%–31%)</td>
</tr>
<tr>
<td>Transition period</td>
<td>6.4</td>
<td>5.4</td>
<td>21.5%</td>
</tr>
<tr>
<td>Range</td>
<td>(5.8–6.7)</td>
<td>(4.6–5.3)</td>
<td>(18%–26%)</td>
</tr>
<tr>
<td>(% difference from two lung values)</td>
<td>(–1.5)</td>
<td>(4)</td>
<td>(–18)</td>
</tr>
<tr>
<td>Single lung IPPV</td>
<td>7.5**</td>
<td>6.1**</td>
<td>18.3%**</td>
</tr>
<tr>
<td>Range</td>
<td>(7.4–7.7)</td>
<td>(5.9–6.3)</td>
<td>(15%–21%)</td>
</tr>
<tr>
<td>(% difference from two lung values)</td>
<td>(15)</td>
<td>(27)</td>
<td>(–30)</td>
</tr>
</tbody>
</table>

Figure 1  Changes in right ventricular dynamics during the first cycle of the whole lung lavage of the right lung. The lack of data at 60 s is because of physiotherapy at that time.

Discussion

The right ventricle is difficult to study using standard imaging techniques because of its complex structure. Animal studies have shown that right ventricular area assessed with ABD reflects changes in right ventricular volume and that the relationship between cross-sectional area and volume varied little with changes in left ventricular volume. Two dimensional echocardiography may be more sensitive to changes in volume and pressure loading than pulmonary artery flotation catheters. However, in patients whose lungs are ventilated, inflated lung can restrict the available acoustic windows and limits transthoracic echocardiographic examination. TOE is therefore more useful for studying right ventricular responses during positive pressure ventilation.

The most marked change seen on starting single lung ventilation is a 30% reduction in FAC, associated with a progressive dilatation of the right ventricle, initially at end-systole, but then also at end-diastole (table 1). It is not possible to determine the significance of this isolated observation in terms of normality though the predominance of systolic dilatation on transition suggests that the fall in FAC is primarily a reflection of a change in right ventricular afterload imposed by an increase in pulmonary vascular resistance when the total tidal ventilation is applied to one lung. The data confirm that this manoeuvre may represent a significant insult to a normal right ventricle and is of relevance to the effects of acute clamping of double lumen tubes in the conduct of anaesthesia for pulmonary surgery in those whose right ventricles are compromised.

PAP is a very rare condition in which there is accumulation of a PAS-positive substance in the alveoli resulting in deterioration in efficiency of gas exchange, the development of respiratory failure and ultimately cor pulmonale. WLL is the only treatment shown to be of therapeutic benefit. This procedure is conducted under general anaesthesia and involves the filling of one lung with 0.9% saline under pressure (15 cm H₂O) and then allowing it to drain passively to FRC, and repeated until the drained fluid is no longer turbid with proteinaceous material. It is common practice to wash out only one lung at a time, and repeat the procedure on the other lung at some time later. In animals and in humans, this controlled drowning process is associated with an acute and cyclical rise in pulmonary vascular resistance.

The echocardiographic experience is that WLL is initially associated with an additional increase in pulmonary vascular resistance, greater than that resulting from single lung ventilation, which caused the right ventricle (fig. 1) to dilate further. Initially, FAC remains unchanged. As lavage volume increases, EDA and ESA decrease (–45% and –58% respectively). This pattern is in keeping with a reduction in right ventricular preload and reflects the effect of intrathoracic pressure on systemic venous return. The increase in FAC (+75%) is suggestive of an increase in right ventricular systolic function but as the FAC is a function of a smaller EDA (by inference, end-diastolic volume), it is not possible to interpret the net effect on right ventricular stroke volume.

Data at 60 s are missing because it is when physiotherapy was used to improve the washout yield of proteinaceous material. An increase in heart rate was seen during the lavage (85 beat min⁻¹ during single lung ventilation to 115 during WLL) and it is possible that the tachycardia represents the stimulation of this manoeuvre, but as there was no other evidence of inadequate depth of anaesthesia we consider the commensurate increase in heart rate to be the result of the autonomic response to the lavage.

As the lung emptied, the effect on preload was reversed and the right ventricle dilated, initially to two lung ventilation values, but returning to OLV values as the afterload effect of ventilating one lung is again the predominant influence.

The initial 1 litre volume of washout had produced visible alterations in right ventricular function so it...
was decided that the observation of reduced heart areas would be the marker for the fluid volume limit for each subsequent lavage cycle. By setting the limits to lavage volume to the level where significant right ventricular changes were apparent (approximately 35–50% reduction in EDA), and by demonstrating the rapid adaptation and recovery of the right ventricle to the various stresses imposed, TOE aided the decision to proceed to lavage of the second lung during the same anaesthetic. The whole therapy with 500- to 750-ml aliquots of saline to a total of 15-litre washout to each lung was undertaken without incident and decreases in systolic arterial pressure were not seen during the filling phases. At follow-up, there was therapeutic benefit, subjectively and in pulmonary function tests (oxygen saturation on air from 90% to 96%; FEV1/FVC 1.71/2.19 to 2.12/2.67).

In detailing the responses of a normal right ventricle to inflationary loads, TOE proved useful in defining alterations in right ventricular afterload and preload, and assisting clinical judgement in deciding safety limits of therapy. Other clinically imposed stresses affecting the pulmonary vascular bed in which a window on right ventricular mechanics would be of great value to optimize therapy include the use of PEEP in acute lung injury, positive pressure ventilation in cor pulmonale and systemic vasconstrictors in pulmonary hypertensive disease. TOE of the right ventricle has great potential as a guide to the most appropriate form of ventilation for an individual with cardiopulmonary disease.

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References