EFFECTS OF LUNG SURGERY AND ONE-LUNG VENTILATION ON PULMONARY ARTERIAL PRESSURE, VENOUS ADMIXTURE AND IMMEDIATE POSTOPERATIVE LUNG FUNCTION

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During lung surgery, clinically important derangements in gas exchange or haemodynamics are not usually seen when a patient is placed in the lateral position, or when the pleura is opened [1, 2]. However, $PaO_2$ may be reduced significantly during one-lung ventilation (OLV) [3—5], and experimental evidence suggests that pulmonary function is impaired following collapse and re-expansion of the lung [6, 7].

The aims of the present study were to study further the effects on oxygenation and haemodynamics of lung surgery and OLV, and to evaluate lung function immediately after the combined trauma of surgery, OLV and re-expansion.

PATIENTS AND METHODS

We studied 17 patients with localized unilateral lesions in the lung or thoracic wall, undergoing thoracotomy. The study was approved by the local Human Studies Committee, and informed consent was obtained from each patient. A detailed description of patients and general methods has been given elsewhere [1].

Anaesthesia was induced with thiopentone and maintained with intermittent pethidine i.v. and 50% nitrous oxide in oxygen. Inspired oxygen fraction ($FiO_2$) was 0.5 throughout the procedure, including the period of OLV. A Carlens double-lumen endobronchial tube was inserted after administration of suxamethonium. Additional neuromuscular block was achieved with a single dose of pancuronium 6–8 mg. Volume preset, constant flow ventilation was given at a rate of 10 b.p.m. with a Siemens–Elema Servo Ventilator 900 B. Inspired volume was adjusted to produce an end-tidal $PCO_2$ ($P_{e\text{-}CO_2}$) of approximately 3 kPa with the patient in the supine position; inspired volume was not changed subsequently. Zero end-expiratory pressure was used throughout the study.

The inspiratory gas source was the same for both lungs and thus tidal volume was distributed according to the resistance and compliance prevailing on each side. Valves directed expired gas from one lung through the expiratory flowmeter of the insufflating ventilator, and from the other lung through an identical ventilator, synchronized...
with the first. Each ventilator was equipped with a Siemens–Elema CO₂ Analyzer 930 [8]. Thus expired minute volume (∆E), PEECO₂, and elimination rate of carbon dioxide (∆CO₂) of each lung could be measured separately. The quotients (∆E of operated lung)/(total ∆E) and (∆CO₂ of operated lung)/(total ∆CO₂) were calculated. These were termed ∆E fraction and ∆CO₂ fraction, respectively.

Surgery was discontinued for 1–2 min during measurements performed at the following stages: (I) with the patient in the lateral position immediately before pleurotomy; (II) 5–10 min after pleurotomy with the ribs parted by a retractor and the lung freely moving; (III) after 10–15 min of OLV and surgery with the Carlens tube lumen of the operated lung open to atmosphere; (IV) with both lungs ventilated after re-expansion of the operated lung, and the ribs still retracted; (V) after airtight closure of the pleural cavity and application of suction drainage. Re-expansion was achieved by manual hyperinflation until all visible lung collapse had disappeared. The surgical procedure allowed measurements during stage IV in only 11 of the 17 patients.

The techniques for measurement of compliance of the respiratory system (Crs), cardiac output, systemic and pulmonary arterial pressures and blood-gas tensions have been described elsewhere [1, 2]. Blood samples for measurement of haemoglobin concentration (Hb, g litre⁻¹), PCO₂ and PO₂ were obtained simultaneously with measurements of ∆E and ∆CO₂. Blood-gas tensions were derived with the standard two-compartment model of pulmonary blood flow distribution [10]:

\[
\frac{Qs}{Qt} = \frac{(Cc'_{O_2} - Ca_{O_2})}{(Cc'_{O_2} - Cv_{O_2})}
\]

Lung function before (stage I) and after surgery (stage V) was compared in nine patients in whom only minor or no resection of lung parenchyma had been performed (segmental resection, lung biopsy, extirpation of paravertebral neuroma). In addition, these patients had no significant gas leakage from the operated lung during stage V (∆E at least 95% of a supine control).

The significance of changes was assessed by Wilcoxon's two-sided rank sum test for paired data. Spearman's rank correlation was used to calculate correlation coefficients (r). Regression lines were obtained by the method of least squares. Values are presented as mean (SD).

RESULTS

Haemodynamic data and blood-gas tensions from stages I and II have been presented previously [2]. Results relevant to the present study are repeated below.

Haemodynamic data (table I)

Heart rate increased following pleurotomy and again during OLV; it decreased after re-expansion of the upper lung. Cardiac index increased after pleurotomy, without further significant changes between consecutive stages. Mean systemic arterial pressure decreased during OLV, but there were no other significant changes between consecutive stages. Mean pulmonary arterial pressure (MPAP) was unaltered following pleurotomy. It increased during OLV, and decreased when both lungs were ventilated again.

Venous admixture and blood-gas tensions

Following pleurotomy, Qs/Qt increased, with a further increase to 31 (11)% during OLV. Qs/Qt values were similar before and after OLV (fig. 1). PaO₂ was in the range 4.4–20.4 kPa during OLV, and 12.4–33.9 kPa after resuming ventilation of the upper lung. PVO₂, which was 5.1 (0.6) kPa after pleurotomy, decreased to 4.2 (0.6) kPa during OLV (P < 0.01), increased to 4.6 (0.4) kPa after resuming ventilation of the upper lung (P < 0.01), and did not change significantly after closure of the chest.

During OLV, Qs/Qt was related to MPAP. The regression equation was: Qs/Qt (%) =
Table I. Mean haemodynamic data (SD) during thoracic surgery. I = Before pleurotomy; II = after pleurotomy; III = during one-lung ventilation; IV = after re-expansion of the upper lung; V = after chest closure. n = 17 unless otherwise stated. Significance of difference from previous stage: *P < 0.05;

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<th>III</th>
<th>IV</th>
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<tbody>
<tr>
<td>Heart rate (beat min⁻¹)</td>
<td>68 (9)</td>
<td>77 (17)*</td>
<td>86 (18)**</td>
<td>77 (17)**</td>
<td>71 (15)</td>
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<td>(n = 17)</td>
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<tr>
<td>Cardiac index (litre min⁻¹ m⁻²)</td>
<td>2.3 (0.6)</td>
<td>2.6 (0.8)*</td>
<td>2.4 (0.5)</td>
<td>2.1 (0.4)</td>
<td>2.1 (0.4)</td>
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<td>(n = 15)</td>
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<td>(n = 15)</td>
<td>(n = 8)</td>
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<tr>
<td>Mean systemic arterial pressure (mm Hg)</td>
<td>114 (14)</td>
<td>114 (16)</td>
<td>90 (13)**</td>
<td>97 (15)</td>
<td>92 (16)</td>
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<td>(n = 15)</td>
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<td>(n = 11)</td>
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<tr>
<td>Mean pulmonary arterial pressure (mm Hg)</td>
<td>17 (5)</td>
<td>17 (6)</td>
<td>19 (6)**</td>
<td>16 (4)</td>
<td>16 (4)</td>
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<td>(n = 8)</td>
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Fig. 1. Venous admixture (Qs/Qt) and arterial oxygen tension (PaO₂) during the study (mean (SD)). *P < 0.05; **P < 0.01.

Fig. 2. Individual values for mean pulmonary arterial pressure (MPAP) and venous admixture (Qs/Qt) during one-lung ventilation. The line of regression, r and P values do not include the dot in parentheses, which represents a patient with endobronchial tube obstruction.

2.5 + 1.5 × MPAP (mm Hg) (fig. 2). This excludes one patient, who had a temporary obstruction by a mucus plug in the Carlens tube lumen of the ventilated lung during OLV. The correlation coefficient was 0.59; with this patient included it was 0.64 (P < 0.05 for both cases). Qs/Qt during OLV correlated also with MPAP as obtained both before OLV (stage II) (P < 0.05) and after it (stage IV) (P < 0.05).

PaCO₂ was 4.1 (0.3) kPa before pleurotomy (stage I), and remained virtually unchanged.

Lung function changes following surgery and OLV (table II)

PaCO₂ and MPAP were not significantly different before (stage I) and after (stage V) surgery.
but Crs and $Pe CO_2$ of the operated side decreased, as did $VE$ and $VCO_2$ fractions. Crs and $Pe CO_2$ of the non-operated side were unaltered.

**DISCUSSION**

After pleurotomy, $\dot{Q}S/\dot{Q}t$ increased from 9 (3) to 12 (5) %, probably because of the reduced volume of both the lower [11] and the upper lung. Pre-existing collapse in dependent lung regions [12] probably increased, and new atelectases may have formed in the upper lung; in the absence of positive end-expiratory pressure, the upper lung usually collapses partially. A similar increase in $\dot{Q}S/\dot{Q}t$ after thoracotomy was observed by Kerr and colleagues [4].

During OLV, arterial and mixed venous oxygenation decreased, MPAP increased and there was a correlation between MPAP and $\dot{Q}S/\dot{Q}t$. The correlation is probably a result of gravity-dependent distribution of pulmonary blood flow [13]; the fraction of blood flow perfusing the collapsed upper lung may be expected to increase with increasing pulmonary arterial pressure. Another possible explanation for the correlation is that reduced alveolar and mixed venous oxygen tension may increase MPAP [14]. However, the correlation between $PvO_2$ and MPAP during OLV was poor ($r = -0.34$), but we found that $\dot{Q}S/\dot{Q}t$ during OLV correlated significantly also with MPAP both before and after OLV, at which stages (II and IV) $PaO_2$ exceeded 9.8 kPa in all patients. These findings favour the first explanation. Total pulmonary blood flow did not influence MPAP during OLV in these patients; no significant correlation was found between cardiac index and MPAP.

Mean $\dot{Q}S/\dot{Q}t$ was 31 % after 10-15 min of OLV. This is less than the mean values (38-44 %) observed in several studies [15-19] which used halothane, enflurane, isoflurane or ketamine anaesthesia, without opioids. Potent inhalation agents inhibit hypoxic pulmonary vasoconstriction [20, 21] and may affect venous admixture during OLV [19]. Thus Peltola [22] reported a marked difference in $\dot{Q}S/\dot{Q}t$ during OLV between enflurane (49 %) and enflurane-fentanyl anaesthesia (30 %). Benumof, Augustine and Gibbons [19], replaced halothane with a combination of fentanyl and other i.v. anaesthetics during OLV, which reduced $\dot{Q}S/\dot{Q}t$ from 44 to 37 %. In patients not exposed to potent inhalation agents before the i.v. anaesthetic, the same centre reported even lower $\dot{Q}S/\dot{Q}t$ values (31 and 33 %) [23].

Our anaesthetic technique was based on opioid analgesia. No neuromuscular blocking drug was given after the lateral position had been established. This allowed us to assess if depth of anaesthesia was adequate. Movements, coughing and increases in arterial pressure and heart rate were considered indications for additional doses of pethidine. The total doses of 350-850 mg may have attenuated increases in pulmonary arterial pressure induced by painful stimuli [24]. The addition of 0.5 MAC nitrous oxide contributes substantially to the anaesthetic. The reduction in $FiO_2$ produced, although reducing mean $PaO_2$ [16], is not the main threat to adequate oxygenation during OLV; an increase in $FiO_2$ does not greatly improve $PaO_2$ in the presence of large right-left shunts [25]. Hypoxaemia during OLV may occur irrespective of the anaesthetic technique, and may be managed optimally [5] only if oxygenation is monitored continuously. Pulse oximetry, which was not available at the time of this study, is well established in this context [26]. The lowest $PaO_2$ of 4.4 kPa during OLV was the result of a tube obstruction, a problem which it is not always possible to avoid, and which would have caused hypoxaemia with any anaesthetic technique.

Patients who have a normal distribution of pulmonary perfusion before operation, tend to be more hypoxic during OLV than those who have a reduced flow in the diseased lung [27, 28]. Most of our patients probably had normal perfusion, as we selected individuals who had benign or thoracic wall lesions (13 of 17 patients), and who had only minor impairment of lung function [1]. In spite of this, mean $\dot{Q}S/\dot{Q}t$ during OLV was less than those noted in most other studies, perhaps because we used opioids rather than inhalation agents. However, as in other studies, there were large inter-individual variations in $PaO_2$, emphasizing that arterial oxygenation should be monitored continuously.

We compared lung function immediately after chest closure (stage V) with that just before pleurotomy (stage I) in patients with limited surgical removal of parenchyma and negligible air leak. As pancuronium was given only once, early in the procedure, conditions for measuring Crs may have been different at the two stages. However, even the first stage of this study was performed at least 1 h after administration of
pancuronium. This, and the unchanged Crs of the lower side, suggests that muscle tone did not bias measurements. The decrease in Crs and $V_e$ fraction of the operated side is consistent with increased lung water content [29] as shown in experimental studies of re-inflated atelectatic lungs [6, 7]. It also agrees with case reports of unilateral pulmonary oedema following positive pressure re-expansion of acutely [30] or chronically [31] collapsed lung. The reduction in $V_{CO_2}$ and $P_E CO_2$ on the operated side indicates reduced perfusion of the re-expanded lung, and is consistent with reduced blood flow of re-expanded lung in patients treated for spontaneous pneumothorax [32]. In the present study, surgical handling of the lung may have contributed to altered function.

In conclusion, high pulmonary arterial pressures were associated with increased venous admixture during OLV. Even when there was only minor resection of lung parenchyma, lung function on the operated side was impaired after surgery. However, this did not affect $Pa_o_2$ or $Pa_cO_2$ in these patients with good lung function before operation.

REFERENCES
27. Hurford WE, Kolker AC, Strauss HW. The use of


