air swallowing or gastric inflation by ventilation via a mask prior to intubation. No increase in vomiting was seen in tracheally intubated patients who would have received positive pressure mask ventilation prior to intubation. Nitrous oxide anesthesia does not affect bowel motility.

We found no association between a history of recent upper respiratory infection and negative postoperative ear pressure. However, all admission tympanograms were normal, suggesting that preoperative eustachian tube function in this group was normal, despite a recent upper respiratory tract infection. A recent study has questioned any association between the use of nitrous oxide and development of postoperative nausea and vomiting in adults. Female gender, a younger age, and a previous history of nausea and vomiting were found to be associated with vomiting. Although 60% of cases in this study were female, 41% were under age 12 yr and likely prepubertal. No association between female gender and vomiting was found.

In summary, we found no association between postoperative negative middle ear pressure and postoperative vomiting in outpatient pediatric patients after nitrous oxide and halothane anesthesia.

REFERENCES

Anesthesiology
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Improving Arterial Oxygenation during One-lung Ventilation

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Maintaining adequate oxygenation during one-lung ventilation (OLV) for thoracic surgery is often a problem. Studies have shown a significant incidence of $\text{PaO}_2$ values less than 70 mmHg in spite of high inspired oxygen concentrations ($\text{FiO}_2$) when the non-dependant lung (ND-lung) is allowed to collapse. Application of continuous positive airway pressure (CPAP) with an $\text{FiO}_2$ of 1.0 to the ND-lung has been reported to be an effective method of improving $\text{PaO}_2$ during OLV without interrupting surgery and re-inflating the ND-lung. However, subsequent human studies have not found application of CPAP to produce consistently satisfactory oxygenation. Published human studies on CPAP during OLV have not detailed the method by which the CPAP was applied to the ND-lung. Allowing the ND-lung airway pressure (Paw) to fall to atmospheric pressure (Patm) for even a short period may result in collapse of small airways which will not be re-opened by clinically useful levels of CPAP. However, application of CPAP 5 cm or more to the extremely compliant ND-lung after the thorax is open results in a lung that stays at a high volume. This high volume combined with...
the transmitted mediastinal motion from the ventilated (dependant) lung impedes surgery. Clinically, we have found that maintenance levels of CPAP less than 5 cmH2O are often not enough to prevent progressive intraoperative atelectasis in the ND-lung and deteriorating PaO2 values. This study was designed to determine if the method in which CPAP is applied affects the PaO2 during OLV, and if modifying the method of CPAP application can improve subsequent arterial oxygenation.

**Materials and Methods**

The study was approved by the Hospital Clinical Trials Committee, and informed consent was obtained from patients preoperatively. Twenty patients having elective thoracotomies were studied. FEV1 and VC were measured and an arterial blood sample was drawn while the patients were supine and breathing air the evening before surgery.

Preoperative medication consisted of promethazine 50 mg and glycopyrrolate 0.2 mg im 1 h pre-induction. Following insertion of intravenous and intra-arterial cannulae, anesthesia was induced with fentanyl (5 μg/kg), sodium thiopental (2–3 mg/kg), and pancuronium (0.08 mg/kg) intravenously.

Anesthesia was maintained by the inhalation of isoflurane (0.5–1.5% inspired concentration) in oxygen. The isoflurane dose was titrated to maintain the systolic blood pressure within 20% of the pre-induction value.

After adequate relaxation, the patients’ tracheas were intubated with a disposable, left endobronchial tube (Rusch®, Canada, Scarborough, Ontario). Position of the endobronchial tube was checked by auscultation and re-verified by fiberoptic bronchoscopy in cases of uncertainty. Isolation of the lung on the side of surgery was verified by observing for underwater leak through the exhaust limb of the CPAP circuit during ventilation of the non-operative side. The patients’ lungs were ventilated with a Nuffield 400 volume ventilator (Penlon®, Abingdon, England) using a tidal volume of 10 ml/kg for both one- and two-lung ventilation. The respiratory rate was adjusted to maintain an arterial CO2 tension of 36 ± 2 mmHg. After insertion of an esophageal temperature probe and stethoscope, the patients were turned to the lateral decubitus position and the position of the endobronchial tube was re-verified. The initial intraoperative arterial blood gas sample (2-Lung Pre) was obtained with the patient in the lateral position during two-lung ventilation 15 min after induction of anesthesia. All blood gas samples were placed on ice and analyzed within 5 min using a Corning 165 pH/blood gas analyzer (Corning® Scientific Instruments, Medfield, MA).

After the thorax on the side of operation was opened, OLV was commenced and CPAP was applied to the ND-lung via a modified Mapleson-D circuit using an FiO2 of 1.0 and a flow of 1 L/min. The level of CPAP in the circuit was controlled by varying the depth of the underwater exhaust. An aneroid manometer previously calibrated against a water column was incorporated into the circuit to verify the level of CPAP. The patients were randomly allocated to one of two treatment sequences. In group A (10 patients), after a tidal volume inflation to an airway pressure (Paw) of 20 cmH2O, the ND-lung was allowed to deflate to 0 cm CPAP (Ptnm) for 5 min. Then, the CPAP was increased to 5 cm for 20 min (Method 1). Arterial blood gases were drawn 5 and 20 min after application of CPAP 5 cm. After 20 min of CPAP 5 cm (25 min of OLV), surgery was halted and the ND-lung re-inflated to a Paw of 30 cmH2O for a minimum of 30 s or until all visible atelectatic areas were re-expanded. The lung was then given a tidal volume inflation to Paw 20 cm and allowed to deflate to a CPAP of 2 cmH2O for 5 min. Then, the CPAP was increased to 5 cmH2O for 20 min (Method 2). Again, arterial blood gases were drawn 5 and 20 min after the application of CPAP 5 cm.

Group B (10 patients) followed an identical protocol, except that the sequence of CPAP applications was reversed (i.e., Method 2 preceded Method 1).

Surgery to the ND-lung continued during the periods of CPAP application by both methods. The entire experimental sequence was completed before ligation of

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**TABLE 1. Patient Data Prior to One-lung Ventilation**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Group</th>
<th>Operation</th>
<th>Age</th>
<th>Side</th>
<th>FEV</th>
<th>FEV/VC</th>
<th>PaO2</th>
<th>2 Lung Pre</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>A</td>
<td>Hiatus hernia</td>
<td>61</td>
<td>L</td>
<td>1.4</td>
<td>.70</td>
<td>70</td>
<td>233</td>
</tr>
<tr>
<td>2</td>
<td>A</td>
<td>Upper lobectomy</td>
<td>58</td>
<td>R</td>
<td>1.25</td>
<td>.68</td>
<td>66</td>
<td>372</td>
</tr>
<tr>
<td>3</td>
<td>B</td>
<td>Middle wedge</td>
<td>68</td>
<td>L</td>
<td>2.4</td>
<td>.80</td>
<td>79</td>
<td>330</td>
</tr>
<tr>
<td>4</td>
<td>A</td>
<td>Upper lobectomy</td>
<td>75</td>
<td>L</td>
<td>1.7</td>
<td>.79</td>
<td>105</td>
<td>455</td>
</tr>
<tr>
<td>5</td>
<td>B</td>
<td>Pneumonectomy</td>
<td>62</td>
<td>R</td>
<td>2.0</td>
<td>.69</td>
<td>66</td>
<td>253</td>
</tr>
<tr>
<td>6</td>
<td>B</td>
<td>Lower lobectomy</td>
<td>49</td>
<td>R</td>
<td>2.95</td>
<td>.83</td>
<td>81</td>
<td>349</td>
</tr>
<tr>
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<td>Upper lobectomy</td>
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<td>L</td>
<td>2.45</td>
<td>.71</td>
<td>78</td>
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<td>Upper lobectomy</td>
<td>65</td>
<td>L</td>
<td>1.0</td>
<td>.47</td>
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<td>70</td>
<td>R</td>
<td>1.9</td>
<td>.73</td>
<td>75</td>
<td>386</td>
</tr>
<tr>
<td>10</td>
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<td>Lower lobectomy</td>
<td>71</td>
<td>R</td>
<td>2.0</td>
<td>.69</td>
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<td>480</td>
</tr>
<tr>
<td>11</td>
<td>A</td>
<td>Lower/middle</td>
<td>66</td>
<td>R</td>
<td>2.5</td>
<td>.77</td>
<td>73</td>
<td>386</td>
</tr>
<tr>
<td>12</td>
<td>A</td>
<td>Pneumonectomy</td>
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<td>R</td>
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<td>2.8</td>
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<td>2.5</td>
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<td>56</td>
<td>L</td>
<td>1.5</td>
<td>.75</td>
<td>76</td>
<td>315</td>
</tr>
<tr>
<td>16</td>
<td>B</td>
<td>Upper wedge</td>
<td>54</td>
<td>L</td>
<td>2.2</td>
<td>.76</td>
<td>70</td>
<td>376</td>
</tr>
<tr>
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<td>R</td>
<td>2.0</td>
<td>.81</td>
<td>80</td>
<td>392</td>
</tr>
<tr>
<td>18</td>
<td>B</td>
<td>Upper/middle</td>
<td>74</td>
<td>R</td>
<td>1.15</td>
<td>.62</td>
<td>83</td>
<td>398</td>
</tr>
<tr>
<td>19</td>
<td>A</td>
<td>Upper lobectomy</td>
<td>71</td>
<td>R</td>
<td>2.0</td>
<td>.74</td>
<td>76</td>
<td>262</td>
</tr>
<tr>
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<td>B</td>
<td>Pneumonectomy</td>
<td>47</td>
<td>L</td>
<td>3.6</td>
<td>.83</td>
<td>90</td>
<td>423</td>
</tr>
</tbody>
</table>

Side = side of operation; FEV1 = preoperative forced expiratory volume in 1 s; FEV/VC = FEV1 to vital capacity ratio; PaO2 = preoperative PaO2 breathing air; 2 Lung-Pre = initial intraoperative PaO2 in lateral position breathing 100% oxygen; wedge = pulmonary wedge resection.
any major pulmonary vessels. A further intraoperative arterial blood gas was drawn 15 min after resumption of two-lung ventilation (2-Lung Post). In the pneumonectomy cases, this control blood gas was drawn 15 min after ligation of the pulmonary artery. Heart rate, systolic blood pressure, and esophageal temperature were recorded every 5 min throughout the surgery.

The similarity of the numerical data for group A and group B were assessed using a two-tailed t-test for independent samples. The Shapiro-Wilk test was used to verify the normality of distribution of the PaO₂ results. The mean levels of PaO₂ during OLV were compared using a two-tailed t-test for paired samples. Significant differences in the mean PaO₂ values attained by the two methods were identified using repeated measures analysis of covariance while controlling for the potentially confounding effect of baseline variables, namely: side of operation, FEV₁/VC ratio and the initial intraoperative PaO₂. P < 0.05 was considered significant.

RESULTS

Demographic data are presented in table 1. The patients' ages ranged from 45–79 yr with a mean (±SD) of 62 ± 9 yr. The thoracotomy was on the left side in eight patients (5 group A, 3 group B) and on the right side in 12 patients (5 group A, 7 group B). There were no statistically significant differences between the means for group A and group B with respect to age, FEV₁, FEV₁/VC ratio, preoperative PaO₂, or any intraoperative PaO₂ levels.

The individual changes in intraoperative PaO₂ values after 20 min of CPAP 5 cm during OLV (i.e., 25 min of total OLV) are shown for group A and group B in figures 1 and 2, respectively. During OLV, only three of the study patients (1 group A, 2 group B) developed arterial hypoxemia to a PaO₂ level less than 70 mmHg. All of these hypoxemic episodes occurred during OLV with Method 1 CPAP.

Significant correlation coefficients were found between the FEV₁ and the FEV₁/VC ratio (r = .698), the FEV₁ and the patient's age (r = −.611), and the preoperative PaO₂ and the 2-Lung Pre PaO₂ (r = .605). The analyses based on all continuous covariates showed the redundancy of three of these covariates, and only the most explanatory ones were retained for further analysis, namely: FEV₁/VC ratio and 2-Lung Pre PaO₂. In addition, side of operation and group were retained in further statistical analyses.

The assumption of normality was verified for all 5- and 20-min PaO₂ results. The mean (±SD) PaO₂ values for groups A and B combined during OLV with CPAP 5 cm using Method 1 were: 202 ± 97 mmHg at 5 min and 187 ± 92 mmHg at 20 min. For Method 2, the 5- and 20-min PaO₂ results were: 329 ± 95 mmHg and 294
pm 106 mmHg, respectively (P < .001 for both 5- and 20-min values).

Repeated measures analysis of covariance demonstrated that Method 2 resulted in significantly better PaO₂ levels than Method 1 both at 5 min (mean difference = 127, P < .0001) and at 20 min (mean difference = 107, P < .0001) while adjusting for group, side of operation, FEV₁/VC ratio, and 2 Lung-Pre PaO₂.

Discussion

This study shows that the arterial oxygenation during OLV with CPAP to the ND-lung is dependant on the method by which the CPAP is applied. Since changes in oxygen consumption and cardiac output were unlikely in our patients, the difference in arterial oxygenation during OLV was due to a difference in intra-pulmonary shunt. Thus, the method of CPAP application is a major determinant of shunt during OLV with CPAP to the ND-lung.

Method 1 application of CPAP was designed to mimic a common clinical situation. The initiation of OLV is a time of rapid physiologic changes. The anesthesiologist must assure adequate isolation and ventilation of one lung, assess oxygenation and compliance, and react quickly to any deficiency. Application of CPAP may be delayed and the ND-lung collapses to such an extent that usual maintenance levels of CPAP (5–10 cm) do not give adequate improvement in oxygenation. However, application of CPAP 5 cm to the fully inflated, extremely compliant ND-lung results in a lung which stays well above its normal residual capacity and impedes surgery.

Method 2 was developed as a compromise between too much and too little collapse of the ND-lung. Pilot studies showed that a maintenance level of only 2 cm CPAP was often not enough to prevent gradual ongoing atelectasis in the ND-lung and subsequent deterioration in PaO₂ levels. In pilot studies, the combined use of CPAP 2 cm for 5 min followed by 5 cm had been found to allow the operative lung to deflate enough for easy surgical access and still result in adequate arterial oxygenation.

Facilitating surgery is the commonest reason for using OLV. There are other methods of treating or preventing hypoxemia during OLV, but all of these have inherent deficiencies. Intraoperative hypoxemia can be treated by re-expanding the ND-lung, but only at the cost of interrupting surgery. Application of positive end-expiratory pressure to the dependant lung is an unreliable treatment of hypoxemia in this situation. Some slight improvement in PaO₂ values may be achieved by manipulation of the tidal volume of the dependant lung. However, application of CPAP is recommended as the primary treatment of hypoxemia during OLV.

The time course of PaO₂ changes beyond 25 min of OLV with CPAP has not been studied. Bindslev et al., in a study not using volatile anesthetic agents, reported that hypoxic pulmonary vasoconstriction (HPV) was
maximum after 15 min of OLV, and did not increase with subsequent hypoxic challenge. Rogers and Benu- 
mof14 showed there was no significant difference in pa-
patients' PaO2 values during OLV with or without a vola-
tile agent. Merridew and Jones15 found a similar lack of 
difference in oxygenation between anesthetic tech-
niques with and without a volatile agent in a study of OLV with ND-lung CPAP. Benuhof16 feels that there 
would be only minimal redistribution of blood flow be-
tween the lungs after 30 min of OLV. For these rea-
sons, it was decided to limit the study time of OLV with 
each method to 25 min.

It has previously been shown that the arterial oxy-
genation during OLV has a positive correlation with the 
degree of obstructive lung disease.11 Since the patient 
population in this study had very little significant ob-
structive disease (mean FEV1/VC = 0.73 ± 0.09), the applicability of the results to patients with high de-
grees of obstructive disease is uncertain.

In conclusion, this study has shown that the method 
by which CPAP is applied to the ND-lung will effect the 
subsequent arterial oxygenation during OLV. It is pos-
able to apply the CPAP in a controlled fashion that will 
reduce the risk of intraoperative hypoxemia without 
compromising surgical conditions.

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REFERENCES

1. Read RC, Friday CD, Eason GN: Prospective study of the Robert-
shaw endobronchial catheter in thoracic surgery. Ann Thorac 
2. Tremper KK, Konchigeri HN, Cullen BF, Kapur PA, Thangath-
ural D, Percival C: Transcutaneous monitoring of oxygen ten-
sion during one-lung anesthesia. J Thorac Cardiovasc Surg 
88:22–25, 1984
3. Torda TA, McCullough CH, O'Brien HD, Wright JS, Horton DA:
Pulmonary venous admixture during one-lung anesthesia, the 
effect of inhaled oxygen tension and respiration rate. An-
aesthesia 29:272–279, 1974
Chanlen J: Optimization of arterial oxygenation during one-
5. Ohara H, Tanaka O, Hoshino Y, Kaetsu H, Muekawa N, Iwai S: 
One-lung ventilation, the effect of positive end expiratory 
pause to the nondependent and dependant lung. Anesthes-
ia 41:1007–1101, 1986
Vliars P: High frequency jet ventilation in patients undergoing 
resection of aortic aneurysm by thoracic incision (abstract). 
ANESTHESIOLOGY 65:A486, 1986
7. Benuhof JL: One-lung ventilation and hypoxic vasoconstriction: 
Implications for anesthetic management. Anesth Analg 
64:821–833, 1985
exchange and hemodynamics during thoractomy. Br J An-
esthesiol 56:1343–1348, 1984
9. Peltola K: Central haemodynamics and oxygenation during tho-
1983
10. Tarhan S, Lundborg RO: Effects of increased expiratory pressure 
on blood gas tensions and pulmonary shunting with the use of the 
11. Katz JA, Laverne RG, Fairley HB, Thomas AN: Pulmonary oxy-
gen exchange during endobronchial anesthesia: Effect of tidal 
volume and PEEP. ANESTHESIOLOGY 56:161–170, 1982
12. Benuhof JL, Alleny DD: Anesthesia for thoracic surgery, Anes-
thesia, 2nd edition. Edited by Miller RD. New York, Chur-
chill-Livingston, 1988, pp 1371–1462
Hypoxic pulmonary vasoconstriction in the human lung: Ef-
fect of repeated hypoxic challenges during anesthesia. ANES-
THESIOLOGY 62:621–625, 1985
14. Rogers SN, Benuhof JL: Halothane and isoflurane do not de-
crease PaO2 during one-lung ventilation in intravenously anes-
thetized patients. Anesth Analg 64:946–954, 1985
15. Merridew CG, Jones RDM: Nondependent lung CPAP (5 cm 
H2O) with oxygen during ketamine, halothane or isoflurane 
anaesthesia and one-lung ventilation (abstract). ANESTHESIO-
LOGY 63:A567, 1985
16. Benuhof JL: Isoflurane anesthesia and arterial oxygenation 
during one-lung ventilation (editorial). ANESTHESIOLOGY 
64:419–422, 1986