air swallowing or gastric inflation by ventilation *via* a mask prior to intubation.[13] 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.[14]

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.[1] 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.

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# Improving Arterial Oxygenation during One-lung Ventilation

P. SLINGER, M.D., F.R.C.P.(C.),* W. TRIOLET, M.D., F.R.C.P.(C.),* J. WILSON, M.D., F.R.C.S.(C.)†

Maintaining adequate oxygenation during one-lung ventilation (OLV) for thoracic surgery is often a problem.[1,2] Studies have shown a significant incidence of $Pa_{O_2}$ values less than 70 mmHg in spite of high inspired oxygen concentrations ($FI_{O_2}$) when the non-dependant

* Assistant Professor of Anaesthesia.

† Associate Professor of Surgery.

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Address reprint requests to Dr. Slinger: Department of Anaesthesia, Montreal General Hospital, 1650 Avenue Cedar, Montreal, Quebec, Canada H3G 1A4.

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lung (ND-lung) is allowed to collapse.[3] Application of continuous positive airway pressure (CPAP) with an $FI_{O_2}$ of 1.0 to the ND-lung has been reported to be an effective method of improving $Pa_{O_2}$ during OLV without interrupting surgery and re-inflating the ND-lung.[4] However, subsequent human studies have not found application of CPAP to produce consistantly satisfactory oxygenation.[5,6] 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.[7] However, application of CPAP 5 cm or more to the extremely compliant[8] ND-lung after the thorax is open results in a lung that stays at a high volume. This high volume combined with

TABLE 1. Patient Data Prior to One-lung Ventilation

| Patient | Group | Operation | Age | Side | FEV | FEV/VC | $Pa_{O_2}$ | 2 Lung-Pre |
|---|---|---|---|---|---|---|---|---|
| 1 | A | Hiatus hernia | 61 | L | 1.4 | .70 | 70 | 233 |
| 2 | A | Upper lobectomy | 58 | R | 1.25 | .68 | 66 | 372 |
| 3 | B | Middle wedge | 68 | R | 2.4 | .80 | 79 | 330 |
| 4 | A | Upper lobectomy | 75 | L | 1.7 | .79 | 105 | 453 |
| 5 | B | Pneumonectomy | 62 | R | 2.0 | .69 | 66 | 253 |
| 6 | B | Lower lobectomy | 49 | R | 2.95 | .83 | 81 | 349 |
| 7 | A | Upper lobectomy | 64 | L | 2.45 | .71 | 78 | 375 |
| 8 | B | Upper lobectomy | 65 | L | 1.0 | .47 | 64 | 408 |
| 9 | A | Pneumonectomy | 79 | R | 1.9 | .73 | 75 | 386 |
| 10 | B | Lower lobectomy | 71 | R | 2.0 | .69 | 100 | 480 |
| 11 | A | Lower/middle lobectomy | 66 | R | 2.3 | .77 | 73 | 386 |
| 12 | A | Pneumonectomy | 45 | R | 4.7 | .85 | 62 | 376 |
| 13 | A | Lower lobectomy | 62 | L | 2.8 | .81 | 86 | 397 |
| 14 | B | Lower lobectomy | 64 | R | 2.5 | .68 | 91 | 485 |
| 15 | A | Upper lobectomy | 56 | L | 1.5 | .75 | 76 | 315 |
| 16 | B | Upper wedge | 54 | L | 2.2 | .76 | 70 | 376 |
| 17 | B | Upper lobectomy | 63 | R | 2.0 | .81 | 80 | 392 |
| 18 | B | Upper/middle lobectomy | 74 | R | 1.15 | .62 | 83 | 398 |
| 19 | A | Upper lobectomy | 71 | R | 2.0 | .74 | 76 | 262 |
| 20 | B | Pneumonectomy | 47 | L | 3.6 | .83 | 90 | 423 |

Side = side of operation; $FEV_1$ = preoperative forced expiratory volume in 1 s; $FEV_1/VC$ = $FEV_1$ to vital capacity ratio; $Pa_{O_2}$ = preoperative $Pa_{O_2}$ breathing air; 2 Lung-Pre = initial intraoperative $Pa_{O_2}$ in lateral position breathing 100% oxygen; wedge = pulmonary wedge resection.

the transmitted mediastinal motion from the ventilated (dependant) lung impedes surgery. Clinically, we have found that maintenance levels of CPAP less than 5 cm are often not enough to prevent progressive intraoperative atelectasis in the ND-lung and deteriorating $Pa_{O_2}$ values. This study was designed to determine if the method in which CPAP is applied effects the $Pa_{O_2}$ 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. $FEV_1$ 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 $\mu$g/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 $CO_2$ tension of $36 \pm 2$ 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 $F_{I_{O_2}}$ 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 cm $H_2O$, the ND-lung was allowed to deflate to 0 cm CPAP (Patm) 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 cm $H_2O$ 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 cm $H_2O$ for 5 min. Then, the CPAP was increased to 5 cm $H_2O$ 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 preceeded 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

FIG. 1. Intraoperative $Pa_{O_2}$ changes in group A patients. Individual patient's values are connected by lines. 2 Lung-Pre = initial $Pa_{O_2}$; OLV = one-lung ventilation; 2 Lung-Post = $Pa_{O_2}$ 15 min after resuming two-lung ventilation (15 min after ligation of pulmonary artery in pneumonectomies).
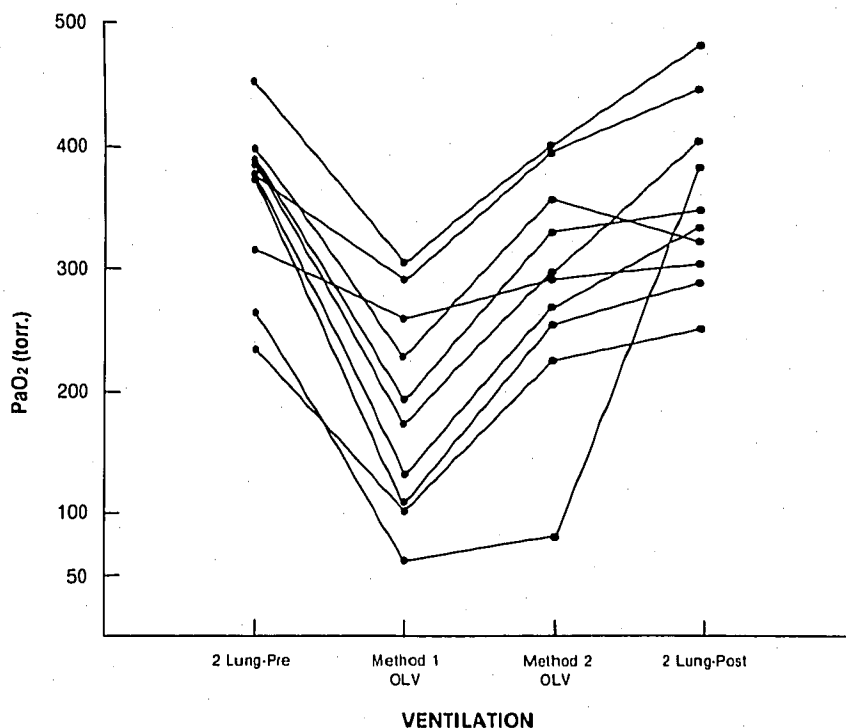
any major pulmonary vessels. A further intraoperative arterial blood gas was drawn 15 min after resumption of two-lung ventilation (2-Lung Post). In the pneumonectomy cases, this control blood gas was drawn 15 min after ligation of the pulmonary artery. Heart rate, systolic blood pressure, and esophageal 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 Sharpiro-Wilk test was used to verify the normality of distribution of the $Pa_{O_2}$ results. The mean levels of $Pa_{O_2}$ during OLV were compared using a two-tailed $t$ test for paired samples. Significant differences in the mean $Pa_{O_2}$ 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, $FEV_1/VC$ ratio and the initial intraoperative $Pa_{O_2}$. $P < 0.05$ was considered significant.

### RESULTS

Demographic data are presented in table 1. The patients' ages ranged from 45–79 yr with a mean ($\pm SD$) of $62 \pm 9$ 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, $FEV_1$, $FEV_1/VC$ ratio, preoperative $Pa_{O_2}$, or any intraoperative $Pa_{O_2}$ levels.

The individual changes in intraoperative $Pa_{O_2}$ 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 $Pa_{O_2}$ level less than 70 mmHg. All of these hypoxemic episodes occurred during OLV with Method 1 CPAP.

Significant correlation coefficients were found between the $FEV_1$ and the $FEV_1/VC$ ratio ($r = .698$), the $FEV_1$ and the patient's age ($r = -.611$), and the preoperative $Pa_{O_2}$ and the 2-Lung Pre $Pa_{O_2}$ ($r = .605$). 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: $FEV_1/VC$ ratio and 2-Lung Pre $Pa_{O_2}$. 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 $Pa_{O_2}$ results. The mean ($\pm SD$) $Pa_{O_2}$ values for groups A and B combined during OLV with CPAP 5 cm using Method 1 were: $202 \pm 97$ mmHg at 5 min and $187 \pm 92$ mmHg at 20 min. For Method 2, the 5- and 20-min $Pa_{O_2}$ results were: $329 \pm 95$ mmHg and 294
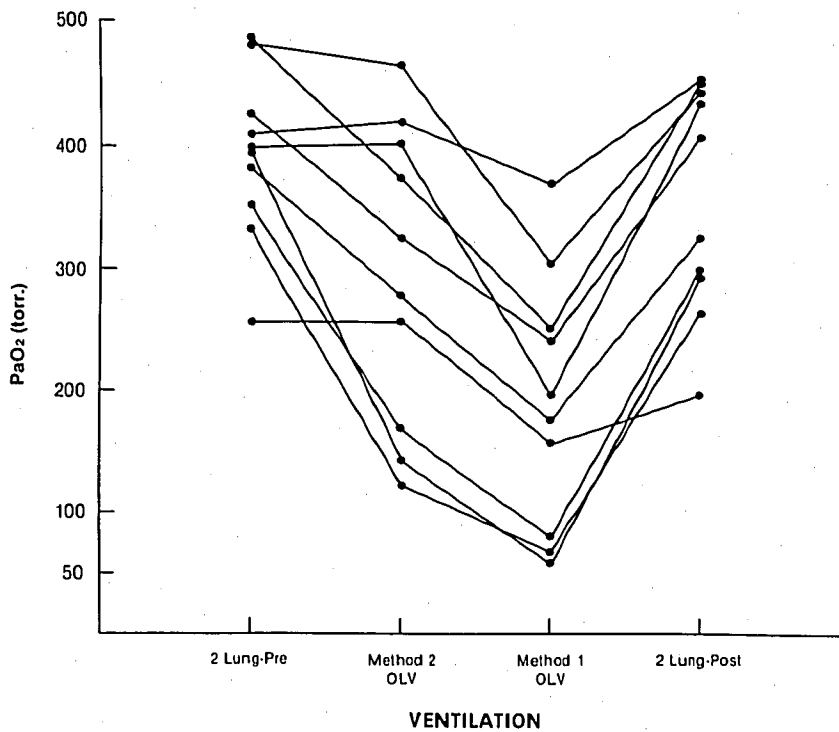
FIG. 2. Intraoperative $Pa_{O_2}$ changes for group B patients.

± 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 $Pa_{O_2}$ 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, $FEV_1/VC$ ratio, and 2 Lung-Pre $Pa_{O_2}$.

## 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,[9] 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 $Pa_{O_2}$ 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.[10] Some slight improvement in $Pa_{O_2}$ values may be achieved by manipulation of the tidal volume of the dependant lung.[11] However, application of CPAP is recommended as the primary treatment[12] of hypoxemia during OLV.

The time course of $Pa_{O_2}$ changes beyond 25 min of OLV with CPAP has not been studied. Bindslev et al.,[13] 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 Benumof[14] showed there was no significant difference in patients' $Pa_{O_2}$ values during OLV with or without a volatile agent. Merridew and Jones[15] found a similar lack of difference in oxygenation between anesthetic techniques with and without a volatile agent in a study of OLV with ND-lung CPAP. Benumof[16] feels that there would be only minimal redistribution of blood flow between the lungs after 30 min of OLV. For these reasons, it was decided to limit the study time of OLV with each method to 25 min.

It has previously been shown that the arterial oxygenation 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 obstructive disease (mean $FEV_1/VC = 0.73 \pm .09$), the applicability of the results to patients with high degrees 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 possible to apply the CPAP in a controlled fashion that will reduce the risk of intraoperative hypoxemia without compromising surgical conditions.

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