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# *Pulmonary Oxygen Exchange during Endobronchial Anesthesia:*

## *Effect of Tidal Volume and PEEP*

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**To determine the effects of tidal volume ($V_T$) and positive end-expiratory pressure (PEEP) on pulmonary oxygen exchange during endobronchial (one-lung) anesthesia, the authors studied the effects of $V_T$ at 8 and 16 per cent total lung capacity (TLC), at zero end-expiratory pressure (ZEEP), and at 10 cm$H_2O$ PEEP in 16 patients in the lateral position. Anesthesia was maintained with halothane and oxygen. During two-lung ventilation ($F_{I_{O_2}}$ 0.99), mean $Pa_{O_2}$ and physiologic shunt ($\dot{Q}_s/\dot{Q}_t$) were 421 ± 12 mmHg and 0.22 ± 0.02, respectively. During one-lung ventilation, $Pa_{O_2}$ decreased and venous admixture (or $\dot{Q}_s/\dot{Q}_t$) increased in every patient. The magnitude of this decrease correlated directly with preoperative forced expiratory volume in one second ($FEV_1$) ($r = 0.66$, $P < 0.005$). A $V_T$ of 16 per cent of TLC at ZEEP resulted in the highest mean $Pa_{O_2}$ (210 ± 30 mmHg) and lowest $\dot{Q}_s/\dot{Q}_t$ (0.35 ± 0.02), probably as a result of end-inspiratory alveolar recruitment with the least pulmonary blood flow redistribution. When 10 cm$H_2O$ PEEP was applied during 16 per cent TLC ventilation, mean $Pa_{O_2}$ decreased from 210 ± 35 to 162 ± 25 mmHg ($P < 0.05$). PEEP did not significantly affect $Pa_{O_2}$ during 8 per cent TLC ventilation. At both levels of $V_T$, PEEP reduced mean $\dot{Q}_t$ by approximately 10 per cent ($P < 0.01$) and increased compliance ($P < 0.01$). However, PEEP did not significantly affect mean $\dot{Q}_s/\dot{Q}_t$ or mean arterial or pulmonary arterial pressures at either level of $V_T$. There was considerable variation in $Pa_{O_2}$ and $\dot{Q}_s/\dot{Q}_t$ among patients. (Key words: Anesthesia, thoracic. Anesthetic techniques: endobronchial. Lung: compliance; function; shunting. Ventilation: mechanical; oxygen tension (gradients); positive end-expiratory pressure; tidal volume; zero end-expiratory pressure.)**

ONE-LUNG (ENDOBRONCHIAL) ANESTHESIA offers special advantages during certain types of thoracic surgery,[1,2] but has the disadvantage of causing defective pulmonary oxygen exchange and an increase in the alveolar-to-arterial oxygen pressure difference $P(A\text{-}a)_{O_2}$.[3–5] This increase in $P(A\text{-}a)_{O_2}$ is probably due to perfusion of collapsed nondependent and, possibly, dependent lung.[6,7] The pulmonary blood flow to the nondependent lung may be less when that lung is diseased diffusely.[7] During lateral thoracotomy, dependent lung atelectasis may be caused by the gravitational effects of the mediastinum and the abdominal contents,[8] as well as by denitrogenation by more soluble gases.[9] In addition, $P(A\text{-}a)_{O_2}$ may be increased further by anesthetic agents that reverse hypoxic pulmonary vasoconstriction.[10]

Techniques designed to minimize $P(A\text{-}a)_{O_2}$ during endobronchial anesthesia have included varying tidal volume ($V_T$)[11,12] and positive end-expiratory pressure (PEEP)[13–16] in the dependent lung. Insufflating oxygen into the nondependent, nonventilated lung has been suggested also.[16] Data from prior studies on varying $V_T$ have been inconsistent.[11,12] Also, although the use of PEEP generally has been shown to increase $P(A\text{-}a)_{O_2}$,[13–16] the interaction of $V_t$ and PEEP has not been examined. The effects of large $V_T$ and PEEP on atelectasis are directionally equivalent in that both may recruit atelectatic lung. PEEP may be more effective than a transient (intermittent) end-inspiratory pressure peak in recruiting atelectatic lung because of the longer time course required for recruitment.[17] However, PEEP may also de-

ABBREVIATIONS

| | |
|---|---|
| $\overline{BP}$ | = mean systemic blood pressure |
| $Ca_{O_2}$ | = arterial oxygen content |
| $C\acute{c}_{O_2}$ | = end-pulmonary capillary oxygen content |
| $C_{L,T}$ | = lung-thorax compliance |
| $C_{O_2}$ | = oxygen content |
| $C\bar{v}_{O_2}$ | = mixed venous oxygen content |
| $FEV_1$ | = forced expiratory volume in one second |
| $F_{I_{O_2}}$ | = fractional concentration of inspired oxygen |
| $\overline{FVC}$ | = forced vital capacity |
| $\overline{PA}$ | = mean pulmonary artery pressure |
| $P(A\text{-}a)_{O_2}$ | = alveolar-to-arterial oxygen pressure difference |
| $Pa_{CO_2}$ | = arterial carbon dioxide partial pressure |
| $Pa_{O_2}$ | = arterial oxygen partial pressure |
| $PA_{O_2}$ | = alveolar oxygen partial pressure |
| $P_{aw}\bar{I}$ | = airway plateau pressure |
| PEEP | = positive end-expiratory pressure |
| $P_{I_{O_2}}$ | = partial pressure of inspired oxygen |
| $P_{O_2}$ | = partial pressure of oxygen |
| $\dot{Q}_c$ | = blood flow through non-shunted pulmonary capillaries (per minute) |
| $\dot{Q}_s/\dot{Q}_t$ | = venous admixture or physiologic shunt |
| $\dot{Q}_t$ | = cardiac output |
| $Sa_{O_2}$ | = arterial oxygen saturation |
| $S_{O_2}$ | = oxygen saturation |
| $S\bar{v}_{O_2}$ | = mixed venous oxygen saturation |
| TLC | = total lung capacity |
| $V_T$ | = tidal volume |
| ZEEP | = zero end-expiratory pressure |

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TABLE 1. Preoperative Pulmonary Function

| | Measured | Percentage of Predicted |
|---|---|---|
| $FEV_1$ (1) | 2.1 ± 0.6 | 71 ± 20 |
| FVC (1) | 3.1 ± 0.7 | 80 ± 16 |
| $FEV_1/FVC \times 100$ | 69 ± 11 | — |
| TLC (1) | 6.4 ± 0.9 | 106 ± 13 |
| RV (1) | 3.3 ± 1.0 | 152 ± 42 |
| FRC (1) | 4.2 ± 1.0 | 121 ± 29 |

Values are means ± SD.

crease cardiac output ($\dot{Q}_t$) and mixed venous oxygen content ($C\bar{v}_{O_2}$), as well as cause a sustained redistribution of pulmonary blood flow from ventilated to atelectatic regions.[18]

This study evaluates the interaction between $V_T$ and PEEP as well as the importance of prior lung disease.

## Methods

The study was approved by the Committee on Human Research, and informed consent was obtained from each patient. We studied 17 adult patients (13 men and 4 women) undergoing elective thoracotomy. Their average age (±SE) was 56 ±2.4 years; mean height, 171 ±1.9 cm; and mean weight, 63 ±2.8 kg. Pulmonary function testing was performed preoperatively in the sitting position and included forced expiratory volume in one second ($FEV_1$), forced vital capacity (FVC), residual volume (RV), total lung capacity (TLC), and functional residual capacity (FRC; as determined by plethysmography) (table 1). No patient had cardiac disease. Premedication consisted of morphine sulfate, 0.1 mg/kg, im. An intravenous line was established and the radial artery cannulated preoperatively. Anesthesia was induced with thiopental and pancuronium and maintained with halothane and oxygen, delivered through a (left-sided) Robertshaw double-lumen tube. Tube position was confirmed by auscultation and visually at thoracotomy. In 13 patients it was possible, after induction of anesthesia, to insert a no. 7 triple-lumen pulmonary arterial catheter percutaneously via the right internal jugular vein. The catheter was floated into the wedge position, then withdrawn a few centimeters to make inadvertent measurement of pulmonary capillary wedge pressure impossible, and to ensure that sampled pulmonary artery blood was not arterialized. The location of the tip of the catheter (the main pulmonary artery) was documented from the chest radiograph taken in the early postoperative period.

## Measurements

All patients were ventilated with an Ohio Medical Products® anesthesia ventilator. Tidal volume ($V_T$) was adjusted to either 16 per cent (large $V_T$) or 8 per cent (small $V_T$) of total lung capacity (TLC) as predicted from height and age. (In our patients, large $V_T$ was 14 ±0.4 ml/kg; and small $V_T$, 7 ±0.2 ml/kg). Expired $V_T$ was measured by integration of flow through a heated Fleisch® pneumotachygraph precalibrated with oxygen. When adjustments of $V_T$ were made, respiratory frequency was changed to maintain minute ventilation at a constant level (±5 per cent). Airway pressure ($P_{aw}$) was measured using a Statham® P23DP pressure transducer. $P_{aw}$ and $V_T$ were recorded simultaneously on a Gould® recorder. Radial and pulmonary arterial pressures were measured using a Hewlett-Packard® 1280c pressure transducer. Cardiac output, which was determined using a thermodilution technique (Edwards Laboratory, model 9520A), was expressed as the mean of two consecutive measurements that differed by less than 10 per cent. Blood gas tensions ($P_{O_2}$ and $P_{CO_2}$) and $pH$ of arterial and mixed venous blood were measured using a Corning® 175 blood-gas analyzer and were corrected to the patient's temperature. Hemoglobin (arterial and mixed venous) concentration, oxygen (arterial and mixed venous) saturation, and carboxy hemoglobin saturation were measured using an Instrumentation Laboratory CO-Oximeter® 282. These measurements were taken at six points (table 2.)

Stage 1 began after induction of anesthesia prior to skin incision. The patient was in the lateral position receiving two-lung ventilation ($V_T$ was 16 per cent of TLC. After one-lung ventilation (pleural cavity open) was begun but prior to Stages 2–5, $Pa_{O_2}$ was measured every 5 min for 20 min (fig. 1). Stability of oxygen exchange during one-lung ventilation was demonstrated, as there was no statistical difference in $Pa_{O_2}$ measured at 10, 15, or 20 min (fig. 1). Stages 2 and 3 were assigned in random sequence, and then Stages 4 and 5 always followed in the same $V_T$ sequence. During Stage 6; the thorax was closed, and the patient was in the lateral position receiving two-lung ventilation ($V_T$ was 16 per cent of TLC at ZEEP). In two patients, measurements at Stages 4 and 5 could not be obtained. Measurements were made 8 min after each adjustment of the mechanical ventilator.

Surgical manipulation and compression of the nondependent lung were halted during the various cardiopulmonary measurements (Stages 2–5). All measurements (Stages 2–5) were concluded prior to any occlusion and division of the pulmonary artery.

Pulmonary variables were calculated as follows. Lung-thorax compliance ($C_{LT}$), $V_T/P_{aw}$, was derived from the difference between $P_{aw}$ and $V_T$ from the end of a period of zero end-inspiratory flow of 1.0 s to end-expiration. Data from five breaths were averaged to derive each value.

Arterial ($Ca_{O_2}$), mixed venous ($C\bar{v}_{O_2}$), end-pulmonary

TABLE 2. Cardiopulmonary Data before, during, and after Thoracotomy

| | Stage | | | | | | |
|---|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | 6 | $P < 0.05$* |
| PEEP ($cmH_2O$) | 0 | 0 | 0 | 10 | 10 | 0 | |
| $V_T$ (per cent pred. TLC) | 16 | 8 | 16 | 8 | 16 | 16 | |
| Lungs ventilated (n) | 2 | 1 | 1 | 1 | 1 | 2† | |
| Chest open | No | Yes | Yes | Yes | Yes | No | |
| $Pa_{O_2}$ (mmHg) | 421 ± 12 | 184 ± 28 | 210 ± 30 | 157 ± 27 | 162 ± 25 | 412 ± 15 | a, d, e |
| $Pa_{CO_2}$ (mmHg) | 33.6 ± 1 | 37.6 ± 1 | 35.4 ± 1 | 38.2 ± 2 | 35.1 ± 2 | 33.9 ± 2 | a, c, d, f |
| $\dot{Q}_s/\dot{Q}_t \times 100$ | 21.9 ± 1.5 | 39.3 ± 2.2 | 34.5 ± 2.0 | 36.9 ± 2.1 | 34.0 ± 1.9 | 21.8 ± 0.8 | a, c |
| $S\bar{v}_{O_2}$ (per cent) | 84 ± 1 | 80 ± 2 | 79 ± 2 | 78 ± 2 | 76 ± 1 | 84 ± 1 | c, e |
| $\dot{Q}_t$ (l/min) | 5.3 ± 0.4 | 6.5 ± 0.5 | 6.3 ± 0.5 | 5.8 ± 0.5 | 5.6 ± 0.6 | 5.9 ± 0.5 | b–e |
| $\dot{Q}_t$ (l/min) | 4.1 ± 0.3 | 3.9 ± 0.3 | 4.1 ± 0.4 | 3.6 ± 0.3 | 3.7 ± 0.4 | 4.6 ± 0.4 | d, e |
| $\dot{Q}_s$ (l/min) | 1.2 ± 0.2 | 2.6 ± 0.3 | 2.2 ± 0.2 | 2.2 ± 0.3 | 1.9 ± 0.2 | 1.3 ± 0.1 | a–c |
| $\overline{BP}$ (mmHg) | 81 ± 4 | 85 ± 4 | 82 ± 3 | 86 ± 5 | 79 ± 4 | 78 ± 4 | NS |
| $\overline{PA}$ (mmHg) | 17 ± 1 | 21 ± 2 | 21 ± 2 | 22 ± 2 | 21 ± 1 | 19 ± 2 | NS |
| $P_{aw}\bar{I}$ ($cmH_2O$) | 20 ± 1 | 17 ± 1 | 26 ± 1 | 23 ± 1 | 32 ± 1 | 20 ± 1 | a–f |
| $C_{LT}$ (ml/$cmH_2O$) | 52 ± 3 | 31 ± 2 | 37 ± 2 | 39 ± 2 | 41 ± 2 | 52 ± 3 | a, b, c, e |

Values are means ± SE.

* a = Stage 2 *vs.* 3; b = Stage 2 *vs.* 4; c = Stage 2 *vs.* 5; d = Stage 3 *vs.* 4; e = Stage 3 *vs.* 5; and f = Stage 4 *vs.* 5. Data were analyzed using two-way analysis of variance; differences were determined using the Newman-Keuls test. Stages 1 and 6 were not significantly different for any variable.

† Except for three patients after pneumonectomy.

capillary ($C\bar{c}_{O_2}$) oxygen contents, and physiological shunt ($\dot{Q}_s/\dot{Q}_t$) were calculated using standard equations (see Appendix for equations).

Data are expressed as means ±SE. Data were analyzed using Student's *t* test for paired data, two-way analysis of variance, method of least-squares (for correlation coefficients), and multiple linear-regression analysis, as indicated in the figures and tables.

## Results

Cardiopulmonary data for individual patients are available in archives.¶ Mean data for preoperative pulmonary function are listed in table 1. Mean (±SE) preoperative $Pa_{O_2}$ (supine) was 78 ± 2 mmHg [fractional concentration of inspired oxygen ($FI_{O_2}$) was 0.21].

During two-lung ventilation, mean (±SE) values were as follows (table 2). During Stage 1, $Pa_{O_2}$ was 421 ± 12 mmHg $FI_{O_2}$ 0.99), with a corresponding $\dot{Q}_s/\dot{Q}_t$ of 0.22 ± 0.02 (range was 0.15–0.33). During Stage 6, $Pa_{O_2}$ was 412 ± 15 mmHg, with a corresponding $\dot{Q}_s/\dot{Q}_t$ of 0.22 ± 0.01. These values were not significantly different from values for Stage 1.

During one-lung ventilation, $Pa_{O_2}$ was always lower than during two-lung ventilation (table 2). Mean $\dot{Q}_s/\dot{Q}_t$ increased from 0.22 ± 0.02 (Stage 1) to 0.39 ± 0.02 (Stage 2) and to 0.35 ± 0.02 (Stage 3). The lowest $Pa_{O_2}$ was 45 mmHg, which occurred in a patient undergoing an esophagectomy. This value was obtained 15 and 20 min after one-lung ventilation was begun ($V_t$ 16 per cent of TLC). Since this level represented significant arterial desaturation, study of this patient was discontinued and two-lung ventilation resumed, which increased $Pa_{O_2}$ to 211 mmHg. This patient did not have significant preoperative lung disease ($FEV_1$ was 2.8 1, 87 per cent of the predicted value).

$Pa_{O_2}$ during one-lung ventilation (Stage 3) correlated inversely with the percentage of predicted $FEV_1$ ($r = -0.66$, $P < 0.005$) (fig. 2) and the percentage of predicted FVC ($r = -0.51$, $P < 0.05$), but did not correlate significantly with $FEV_1$/FVC, preoperative $Pa_{O_2}$, or $Pa_{O_2}$ during two-lung ventilation (Stage 1).

### Effect of Tidal Volume on Cardiopulmonary Variables during One-Lung Ventilation

Although large $V_T$ ventilation (Stage 3) was associated with a larger $Pa_{O_2}$ and with a smaller $\dot{Q}_s/\dot{Q}_t$ than was observed with small $V_T$ ventilation (Stage 2) (fig. 3), it did not change $\dot{Q}_t$. The mean difference in $Pa_{O_2}$ between levels of $V_T$ was small (26 mmHg). However, the individual patient data showed wide variation. Five patients increased $Pa_{O_2}$ by more than 40 mmHg with large, as compared with small, $V_T$ ventilation. Only one patient had a significant decrease in $Pa_{O_2}$ with large $V_T$. $Pa_{CO_2}$ was greater during small $V_T$ than during large

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$V_T$ ventilation ($P < 0.05$) (table 2). However, the mean difference (2.2 mmHg) was small.

$P_{aw}$ and $C_{LT}$ were greater during large $V_T$ ventilation than during small $V_T$ ventilation ($P < 0.001$) (table 2).

There was no significant difference in mean systemic blood pressure or pulmonary arterial pressure between levels of $V_T$.

### Effect of PEEP (10 $cmH_2O$) on Cardiopulmonary Variables during One-Lung Ventilation

Mean $Pa_{O_2}$ decreased during large $V_T$ ventilation with 10 $cmH_2O$ PEEP ($P < 0.05$) (fig. 4). There was no significant effect on $\dot{Q}_s/\dot{Q}_t$ (fig. 4). Although mean $Pa_{O_2}$ fell with PEEP, this was not uniform; two patients had important improvements in oxygen exchange with PEEP, and other patients had little or no change.

PEEP reduced mean $\dot{Q}_t$ by an amount that was independent of the level of $V_T$ (fig. 4), and mean $S\bar{v}_{O_2}$ decreased during large $V_T$ ventilation with application of 10 $cmH_2O$ PEEP (table 2). Ventilation with PEEP increased $C_{LT}$ at each level of $V_T$, but there was no significant effect on mean arterial or pulmonary arterial pressures (table 2).

The change in $\dot{Q}_s/\dot{Q}_t$ with the application of 10 $cmH_2O$ PEEP during one-lung ventilation at both small and large $V_T$ ventilation correlated inversely with the initial $\dot{Q}_s/\dot{Q}_t$ during two-lung ventilation (Stage 1) ($r$ = −0.72, $P < 0.02$; and $r$ = −0.68, $P < 0.05$, respectively).

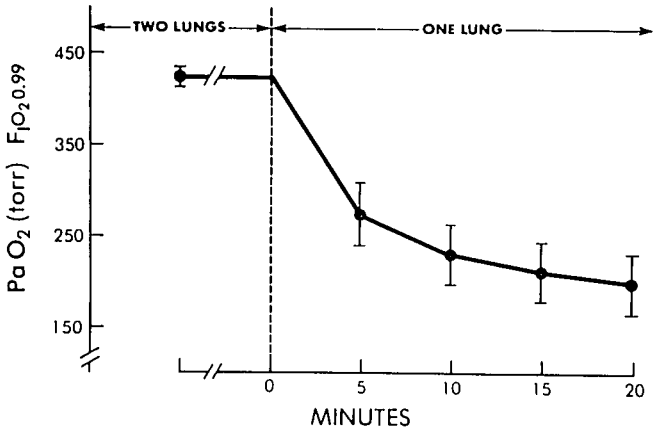


FIG. 1. Course of arterial oxygen partial pressure ($Pa_{O_2}$) during endobronchial (one-lung) anesthesia (lateral position, mean ± SE, n = 17). Vertical dashed line indicates the start of one-lung ventilation. Mean $Pa_{O_2}$ during two-lung ventilation was significantly different ($P < 0.001$) from all other measurements. Mean $Pa_{O_2}$ at 5 min (during one-lung ventilation) was significantly different ($P < 0.05$) from all other measurements. No statistically significant differences occurred in mean $Pa_{O_2}$ measured at 10, 15, and 20 min. At 15 min, two incomplete data points (two patients) were generated using the method suggested by Snedecor and Cochran.[27] Data were analyzed using two-way analysis of variance; differences were determined using the Newman-Keuls test.

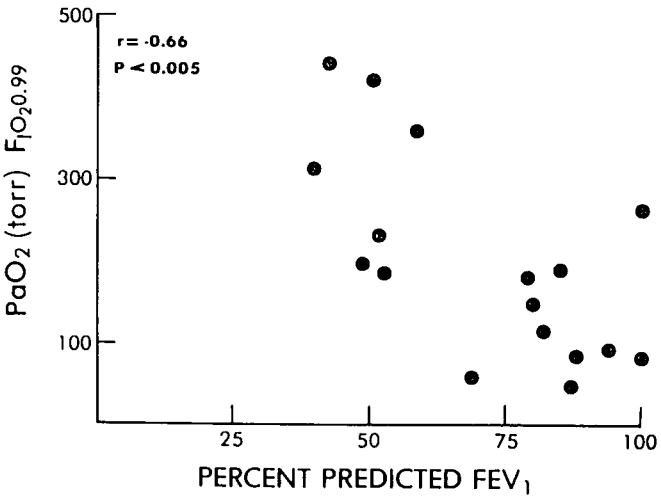


FIG. 2. Preoperative forced expired volume in one second ($FEV_1$) and arterial oxygen partial pressure ($Pa_{O_2}$) during one-lung ventilation (Stage 3).

Multiple linear-regression analysis of variables that might have changed $Ca_{O_2}$ ($\Delta Ca_{O_2}$) (with the application of PEEP) was performed. These variables included $\dot{Q}_s/\dot{Q}_t$, $\dot{Q}_t$, and $C\bar{v}_{O_2}$. The multiple correlation between (change in) $\dot{Q}_s/\dot{Q}_t$, $\dot{Q}_t$, $C\bar{v}_{O_2}$, and $\Delta Ca_{O_2}$ was 0.95 ($P < 0.001$); and a significant partial correlation occurred with $\dot{Q}_s/\dot{Q}_t$ ($r$ = −0.73, $P < 0.001$).

## Discussion

It is widely accepted that pulmonary oxygen exchange is impaired during endobronchial anesthesia. In this study, one-lung ventilation with an $FI_{O_2}$ of 0.99 resulted in a $Pa_{O_2}$ of less than 80 mmHg in five of 17 patients; in two patients, $Pa_{O_2}$ was less than 60 mmHg. Table 3 lists prior studies that have examined the effects of mechanical ventilation in the dependent lung on gas exchange during endobronchial anesthesia. Our data support the use of large $V_T$ (at ZEEP) when ventilation is confined to the dependent lung. This practice is supported by the finding of Kerr *et al.*[7] that a decrease in $V_T$ during one-lung ventilation resulted in an increase in $\dot{Q}_s/\dot{Q}_t$ from the beginning to the end of surgery.

In our study, 10 $cmH_2O$ PEEP decreased mean $Pa_{O_2}$, but only with large $V_T$. This finding agrees with that of Tarhan and Lundborg[13] and Capan *et al.*,[16] who also showed a decrease in $Pa_{O_2}$ with 10 $cmH_2O$ PEEP. Two of our patients showed important improvements in $Pa_{O_2}$ with PEEP. These patients may be distinguished from the majority by two variables: a relatively low $Pa_{O_2}$ (less than 80 mmHg) during one-lung ventilation

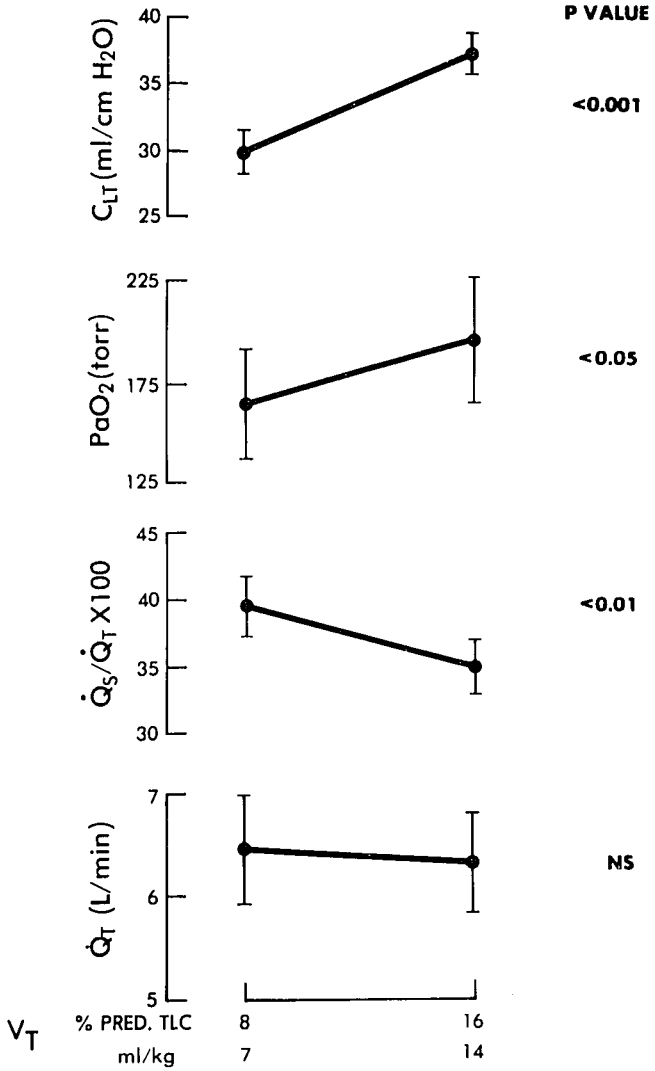


FIG. 3. Effect of tidal volume ($V_T$) during one-lung ventilation (mean ± SE, n = 13). Data were analyzed using Student's *t* test for paired data. $C_{LT}$ = lung-thorax compliance; $Pa_{O_2}$ = arterial oxygen partial pressure; $\dot{Q}_s/\dot{Q}_t$ = physiologic shunt; and $\dot{Q}_t$ = cardiac output.

before PEEP was applied, and a $\dot{Q}_s/\dot{Q}_t$ above 0.25 during two-lung ventilation. A similar circumstance can be found in the data from Tarhan and Lundborg.[13] They found that with the application of 10 $cmH_2O$ PEEP, $Pa_{O_2}$ decreased in ten patients, was unchanged in one, and increased in three. Of these three patients, two had $Pa_{O_2}$ values that prior to PEEP were less than 80 mmHg.

Evaluating our own data and those of other authors, we believe the major predisposing mechanisms to be as follows. In healthy volunteers in the lateral position (chest closed), there is a reduction in FRC that is most marked in the dependent lung.[19] In addition, with anesthesia, muscle paralysis, and controlled ventilation, a loss in diaphragmatic forces further contributes to the decrease in FRC and an abnormal distribution of ventilation.[20] These factors (dependent lung atelectasis and maldistribution of ventilation in relation to perfusion) result in an increase in venous admixture, and may explain our mean $\dot{Q}_s/\dot{Q}_t$ of 0.22 (Stage 1).

$Pa_{O_2}$ and $\dot{Q}_s/\dot{Q}_t$ did not change between Stage 1 and Stage 6 (before and after one-lung ventilation). In particular, patients who underwent pneumonectomy had no clinically significant changes in $Pa_{O_2}$ or $\dot{Q}_s/\dot{Q}_t$ between Stages 1 and 6. These data support the possibility that much of the $\dot{Q}_s/\dot{Q}_t$ during two-lung ventilation can be related to the dependent lung. During one-lung ventilation, $\dot{Q}_s/\dot{Q}_t$ increased greatly (table 2). This increase may have occurred not only by continued perfusion of the nondependent (collapsed) lung, but also in areas of dependent-lung atelectasis. The contribution of the latter is supported by the observed effect of changing tidal volume to the dependent lung. In addition, the direct correlation between $\dot{Q}_s/\dot{Q}_t$ during two-lung ventilation and the magnitude of improvement in $\dot{Q}_s/\dot{Q}_t$ following the application of PEEP further supports the hypothesis that dependent-lung atelectasis is an important variable. The exact division of $\dot{Q}_s/\dot{Q}_t$ between nondependent and de-

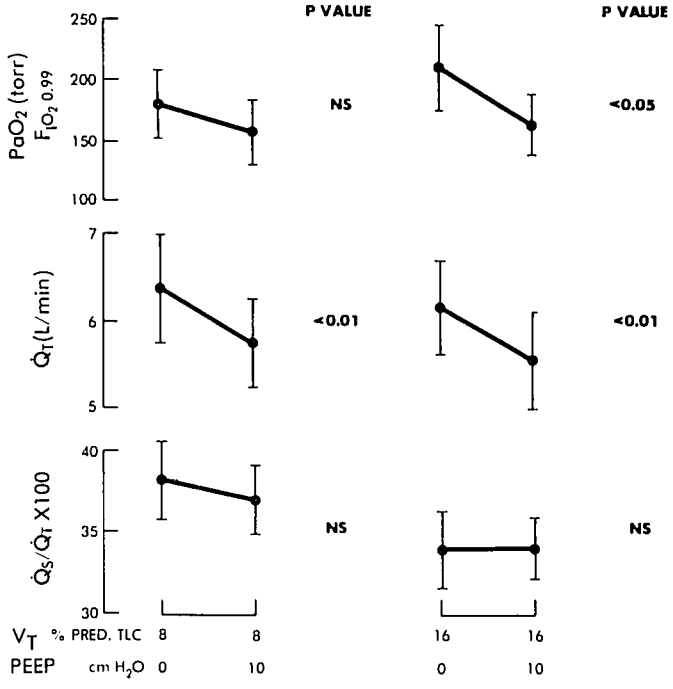


FIG. 4. Effect of 10 $cmH_2O$ of positive end-expiratory pressure (PEEP) at tidal volumes ($V_T$) of 8 and 16 per cent of total lung capacity (TLC) during one-lung ventilation (mean ±SEM, n = 11). Data were analyzed using two-way analysis of variance; differences were determined using the Newman-Keuls test. $Pa_{O_2}$ = arterial oxygen partial pressure; $F_{IO_2}$ = fractional concentration of inspired oxygen; $\dot{Q}_t$ = cardiac output; and $\dot{Q}_s/\dot{Q}_t$ = physiologic shunt.

TABLE 3. Effect of Tidal Volume ($V_T$) and Positive End-Expiratory Pressure (PEEP) on Pulmonary Oxygen Exchange during Endobronchial (One-lung) Anesthesia

| Investigator | $V_T$ (ml/kg)* | PEEP ($cmH_2O$) | $Pa_{O_2}$ | $\dot{Q}_s/\dot{Q}_t$ | $\dot{Q}_t$ |
|---|---|---|---|---|---|
| Khanam and Branthwaite[11] | 7–10† | 0 | NS | | |
| Flacke *et al.*[12] | 8–15 | 0 | NS | NS | |
| Katz *et al.*[17] | 7–14 | 0 | ↑ | ↓ | NS |
| Tarhan and Lundborg[13] | Manual ventilation | 0–10 | ↓ | | |
| Khanam and Branthwaite[14] | 7 | 0–10 | NS | | |
| Capan *et al.*[16] | Variable | 0–10 | ↓ | ↑ | NS |
| Katz *et al.*[17] | 7 | 0–10 | NS | NS | ↓ |
| Katz *et al.*[17] | 14 | 0–10 | ↓ | NS | ↓ |
| Aalto-Setälä *et al.*[15] | Not stated | 0–5 | NS | NS | NS |

*Mechanical ventilation.
† Not specifically stated, value approximated from data.

↑ = Significant ($P < 0.05$) increase. ↓ = Significant ($P < 0.05$) decrease. NS = no significant change.

pendent lung cannot be delineated using the data available in this study.

The multiple linear-regression analysis of the variables affecting a change in $Ca_{O_2}$ with the application of PEEP during one-lung ventilation strongly suggests that distribution of pulmonary blood flow between ventilated and nonventilated lung regions ($\dot{Q}_s/\dot{Q}_t$) is the major contributing factor, independent of $C\bar{v}_{O_2}$.

Two major factors interacted in the determination of $\dot{Q}_s/\dot{Q}_t$. First, the ventilatory maneuvers could have influenced the volume of ventilated dependent lung; and, second, the distribution of blood between ventilated ($\dot{Q}_c$) and nonventilated ($\dot{Q}_s$) lung could be influenced by the intra-alveolar pressure in the former. Increased intra-alveolar pressure would increase pulmonary vascular resistance in ventilated areas and could thereby reapportion $\dot{Q}_c$ and $\dot{Q}_s$. This redistribution of blood flow has been shown experimentally in the dog by Finley *et al.*[21] $\dot{Q}_s/\dot{Q}_t$ would ultimately be determined by the balance between dependent-lung recruitment and increased vascular resistance in that lung. The data show a significant effect of $V_T$ on $\dot{Q}_s/\dot{Q}_t$ (fig. 3) but no effect of PEEP (fig. 4). Large $V_T$ (16 per cent of TLC) ventilation to the dependent lung improved compliance (fig. 3). Furthermore, no significant change in $\dot{Q}_t$ occurred. The explanation for this improvement in compliance is probably a recruitment of atelectatic dependent lung at end inspiration, and this might be expected if the dependent lung had an initial volume that was very low. Since the application of PEEP also increased the volume of ventilated lung but did not improve $\dot{Q}_s/\dot{Q}_t$ or $Pa_{O_2}$, we must conclude that it also caused an offsetting effect by redistributing pulmonary blood flow. The beneficial effect of large tidal volumes on $\dot{Q}_s/\dot{Q}_t$, and therefore $Pa_{O_2}$, suggests an improvement in ventilated lung volume without this offsetting redistribution in blood flow to unventilated areas.

We found an inverse correlation between the preoperative percentage of predicted $FEV_1$ (an index of prior lung disease) and $Pa_{O_2}$ during one-lung ventilation (fig. 2). Our explanations for this inverse correlation are only speculative. It is possible that some patients with a low $FEV_1$ had this reduction as a consequence of unilateral (restrictive) pulmonary disease (mainly carcinoma), causing a redistribution of pulmonary perfusion preoperatively away from the operative lung. With acute atelectasis in the operative lung, there would be less perfusion of this lung, or redistribution to it, and therefore a higher $Pa_{O_2}$ during one-lung ventilation. Kerr *et al.*[7] found that patients with pulmonary lesions had smaller decreases in $Pa_{O_2}$ during one-lung ventilation than those undergoing thoracotomy for nonpulmonary procedures.

An alternative explanation that does not depend on an uneven distribution of pulmonary abnormality might be as follows. The increased static lung volumes (TLC, FRC, and RV) (table 1) indicate overdistension compatible with loss of lung elastic recoil and obstructive lung disease. During atelectasis, pulmonary vessels may collapse and kink.[22] This has been discounted in lungs of normal dogs by Benumof.[23] However, a loss of elastic recoil may permit a greater physical deformity of the pulmonary vasculature during atelectasis. The vascular resistance in such lungs would then be accentuated, and redistribution of pulmonary blood flow during one-lung anesthesia minimized. Our data do not permit further examination of these possibilities.

The wide variability of $Pa_{O_2}$ during one-lung ventilation may also be due in part to the variability of alveolar hypoxic pulmonary vasoconstriction.[24] Miller and Hales[25] described two populations of dogs, one with a strong initial response (within 7 min of alveolar hypoxia achieved by nitrogen ventilation of one lung), and the other with a weak initial response that became stronger with time (approximately 4–6 h). The strong responders had a 30 per cent decrease in perfusion to the hypoxic lung on the first hypoxic challenge, whereas the weak

responders had only a 5 per cent decrease. Thus, an inherent form of host variability may contribute to the wide range of $Pa_{O_2}$ seen during endobronchial anesthesia. The use of an $FI_{O_2}$ of 0.99 and halothane also may have contributed to the variability in $Pa_{O_2}$ during endobronchial anesthesia.[20,25]

We conclude that when employing the technique of endobronchial anesthesia, one must be aware that pulmonary oxygen exchange is impaired. Despite the use of an $FI_{O_2}$ of 0.99, $Pa_{O_2}$ may fall to low levels. The degree of impairment in $Pa_{O_2}$ correlates inversely with the preoperative percentage of predicted $FEV_1$. Therefore, patients with normal preoperative pulmonary function, as assessed by $FEV_1$, are not without risk of hypoxemia. Frequent blood-gas monitoring is imperative for early detection of this hypoxemia. In this study, a $V_T$ of 16 per cent of TLC at ZEEP most frequently resulted in the best oxygen exchange without a reduction in $\dot{Q}_t$ or evidence of over-distension of the dependent lung. In addition, the application of 10 cm $H_2O$ PEEP generally resulted in a decrease in $Pa_{O_2}$, probably secondary to a redistribution of pulmonary perfusion. These beneficial effects of large tidal volumes without PEEP are not consistent, and the high degree of variability is another reason for monitoring arterial oxygen tension during one-lung anesthesia.

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## Appendix

### *Equations*

$$C_{O_2} = (Hgb)(S_{O_2})(\gamma) + (P_{O_2})(\beta) \quad (1)$$

$$PA_{O_2} = PI_{O_2} - Pa_{CO_2}\left[FI_{O_2} + \frac{1 - FI_{O_2}}{R}\right] \quad (2)$$

When $Sa_{O_2} \geq 0.995$:

$$C\bar{c}_{O_2} = [P(A\text{-}a)_{O_2}](\beta) + Ca_{O_2} \quad (3)$$

When $Sa_{O_2} < 0.995$:

$$C\acute{c}_{O_2} = [P(A\text{-}a)_{O_2}](\beta) + Ca_{O_2} + [(S\acute{c}_{O_2})(Hbg)(1 - Sa_{CO})] - (Sa_{O_2})(Hgb)] \quad (1.34)$$

$$\dot{Q}_s/\dot{Q}_t = \frac{C\acute{c}_{O_2} - Ca_{O_2}}{C\acute{c}_{O_2} - C\bar{v}_{O_2}} \quad (4)$$

### *Abbreviations Used in Appendix*

$\beta$ = solubility of $O_2$ in plasma (0.003 ml $O_2$ per 100 ml of blood per mmHg of $O_2$ tension)
$Ca_{O_2}$ = arterial oxygen content
$C\acute{c}_{O_2}$ = end-pulmonary capillary oxygen content
$C\bar{v}_{O_2}$ = mixed venous oxygen content
$FI_{O_2}$ = fractional concentration of inspired oxygen
$\gamma$ = 1.34 ml of $O_2$ per gram of hemoglobin per 100 ml of blood
Hgb = hemoglobin (grams per 100 ml of blood)
$PA_{O_2}$ = alveolar oxygen partial pressure
$Pa_{O_2}$ = arterial oxygen partial pressure
$PI_{O_2}$ = partial pressure of inspired oxygen [$(P_{baro} - PA_{H_2O})$ $(FI_{O_2})$; $P_{baro}$, barometric pressure in mmHg; $PA_{H_2O}$, water vapor pressure]
$P\bar{v}_{O_2}$ = mixed venous oxygen partial pressure
$\dot{Q}_s/\dot{Q}_t$ = physiologic shunt
R = $\dot{V}_{CO_2}/\dot{V}_{O_2}$; assumed = 0.85
$Sa_{O_2}$ = arterial oxygen saturation
$Sa_{CO}$ = arterial carbon monoxide saturation
$S\acute{c}_{O_2}$ = calculated end-pulmonary capillary oxygen saturation (assumed to be 100 per cent)
$S\bar{v}_{O_2}$ = mixed venous oxygen saturation