Original contribution

The effect of increased apparatus dead space and tidal volumes on carbon dioxide elimination and oxygen saturations in a low-flow anesthesia system

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Received 20 October 2006; revised 4 September 2007; accepted 21 September 2007

Keywords:
Airway; Anesthesia; Anesthetics; Carbon dioxide; Gas exchange; Lung, dead space; Lung, volume; Ventilation; Ventilation-perfusion

Abstract

Study objective: To determine if a large tidal volume (VT), with an unchanged end-tidal carbon dioxide partial pressure (PETCO2), could improve arterial carbon dioxide elimination, oxygen saturation (SpO2), and arterial blood oxygenation.

Design: Prospective, randomized, clinical study.

Setting: Single university hospital.

Patients: 60 ASA physical status I and II patients scheduled for elective urologic or general surgery.

Interventions: Patients were randomly assigned to one of two treatments: patients in group 1, nondead space (NDS), received a fresh gas flow of 1 L/min without added apparatus dead space volume. Patients in group 2, dead space (DS), received ventilation using an added dead space volume between the Y-piece and tracheal tube. In both groups, patients’ lungs were ventilated to a fixed PETCO2 value of 33.8 mmHg. Patients in the DS group were ventilated with VT to maintain an airway plateau pressure (Pplateau) of 0.04 cm H2O/kg over initial plateau pressure. The corrugated tube was then adjusted to maintain a fixed PETCO2.

Measurements: Dead space volumes, PETCO2, arterial CO2 tension (PaCO2), SpO2, arterial O2 tension (PaO2), VT, and airway pressures were measured.

Main Results: Arterial CO2 tension was significantly lower in the DS group, 36 ± 2.3 mmHg, compared with the NDS group, 37.5 ± 2.3 mmHg (P < 0.05), and the difference between PETCO2 and PaCO2 was lower in the DS group than in the NDS group (P < 0.001). Oxygen saturation was 99% ± 1.0% in the DS group compared with 98.5% ± 1.5% in the NDS group (P < 0.05). Arterial O2 tension was 13.2 ± 25.5 mmHg in the DS group and 119.1 ± 30.2 mmHg in NDS group (not significant).

Conclusion: Larger VT, with an unchanged PETCO2 concentration created by an added apparatus dead space volume, improved arterial carbon dioxide elimination.

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1. Introduction

Pulmonary gas exchange is frequently reduced with mechanical ventilation during general anesthesia. During general anesthesia patients in the supine position often have reduced arterial blood oxygenation (PaO₂) because of decreased functional residual capacity (FRC). Other major causes of reduced PaO₂ are collapse of lung tissue (atelectasis) and airway closure [1]. Lung volume is reduced during general anesthesia, muscle paralysis, and mechanical ventilation, as a result of the cranial shift of the diaphragm and reduction in the thoracic transverse area. The decrease in thoracic volume is a result of a reduction in FRC and a displacement of blood from the thorax to the abdomen [2].

There is a relationship between airway closure and perfusion of poorly ventilated lung regions [3]. Hedenstierna and colleagues [1] concluded that airway closure and thereby the ventilation to perfusion mismatch, can only be prevented with increasing FRC with positive end-expiratory pressure (PEEP). With this background, we sought to determine if larger tidal volumes (V₁₅), with an unchanged partial pressure of end-tidal carbon dioxide (P₄CO₂) and inspired oxygen concentration (FiO₂), would improve arterial CO₂ elimination and PaO₂.

2. Materials and methods

The study was approved by the University of Lund Hospital (Lund, Sweden) regional ethics committee, and written, informed consent was obtained from all patients. We studied 60 ASA physical status I and II patients who were scheduled for elective general or urologic surgery with an expected anesthesia time of two hours or more. Patients with a history of pulmonary or significant cardiovascular disease were excluded from the study. The 60 patients were randomly assigned to one of two treatments (30 patients in each group) via sealed envelope assignment. The lungs of patients in group 1 (nondead space [NDS]) were ventilated without an extraneous dead space volume. In group 2 (dead space [DS]), patients received ventilation using an added dead space volume between the Y-piece and endotracheal tube.

All patients received premedication with midazolam, 7.5 mg, orally 30 minutes before admission to the operating theater. After preoxygenation with 100% oxygen for three to four minutes and a fresh gas flow of 4.5 L/min, anesthesia was induced with fentanyl, 2 μg/kg, and propofol, 1.5 to 2 mg/kg. Rocuronium, 0.6 mg/kg, was administered for muscle paralysis. Ventilation was manually assisted with 2 mg/kg. Rocuronium, 0.6 mg/kg, was administered for muscle paralysis, and thereafter with a ventilator (Dräger Primus; Dräger Medical, Lübeck, Germany). Fresh gas flow (FiO₂, 0.35 in nitrous oxide [N₂O]) was 4.5 L/min during the first 5 min and then adjusted to 1.0 L/min (anesthesia was maintained with sevoflurane, adjusted to an end-tidal concentration of 1.3%). No PEEP was used. The ventilator rate was 15 breaths per minute, inspiratory-expiratory ratio was 1:2 (including inspiratory pause of 10%). In the DS group, an adjustable corrugated tube (single-use plastic tube, Medcore AB, Uppsala, Sweden) was placed between the Y-piece and the heat and moisture exchanger. In the NDS group, no adjustable tube was used. Patients in the NDS group received ventilation with V₁₅ to achieve ventilator plateau pressure (Pplateau) of 0.04 cm H₂O/kg over initial measured plateau pressure. The corrugated tube was then adjusted to maintain PPECO₂ pressure of 33.8 mmHg. Before each anesthetic administration, fresh soda lime (Drägersorb, Dräger Medical, Lübeck, Germany) was used, and end-tidal inspired carbon dioxide (FiCO₂) was measured to detect rebreathing. Patients in the DS group were ventilated with V₁₅ to achieve ventilator plateau pressure (Pplateau) of 0.04 cm H₂O/kg over initial measured plateau pressure. The corrugated tube was then adjusted to maintain PPECO₂ pressure of 33.8 mmHg. We measured dead space volumes in the DS group by filling the tube with water, then measuring this volume of water.

Monitoring during the procedure included three-lead electrocardiography, heart rate, invasive arterial pressure (measured in the radial artery), and oxygen saturation by pulse oximetry (SpO₂). Inspired oxygen and end-tidal concentrations of sevoflurane, N₂O, and CO₂ were monitored at the distal end of the tracheal tube throughout the two-hour study time, and analyzed gases were returned to a port fitted into the CO₂ absorber. The PPECO₂ values were measured and analyzed by the Primus anesthetic machine using a sidestream technique with 150 mL flow and response time less than 500 ms (Dräger Primus, Dräger Medical, Lübeck, Germany). Arterial blood oxygenation was sampled from the arterial cannula, then measured every 15 minutes using an automated analyzer (ABL 725, Radiometer, Copenhagen, Denmark).

Additional doses of fentanyl were administered if mean blood pressure increased more than 20% from baseline. Decreases in blood pressure were treated with intravenous IV ephedrine, 5 to 10 mg. Neuromuscular block was maintained with rocuronium IV, and supplementary doses were given when two twitches were reached with a neuromuscular transmission analyzer (TOF-Watch; Organon Teknika BV, Boxtel, The Netherlands).

Table 1: Demographic data of the NDS and DS groups

<table>
<thead>
<tr>
<th></th>
<th>NDS (N = 29)</th>
<th>DS (N = 30)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>63 ± 14</td>
<td>61 ± 13</td>
</tr>
<tr>
<td>Men-Women ratio</td>
<td>23:6</td>
<td>21:9</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>82.6 ± 12.5</td>
<td>78.9 ± 15.2</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>27.6 ± 3.4</td>
<td>25.8 ± 3.3</td>
</tr>
<tr>
<td>Smoker</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

Values are means ± SD. There were no statistical differences between the groups in age, gender, body mass index (BMI), weight, or smoking status (smoker).
2.1. Statistical analysis

All data are reported as mean values ± SD. A power analysis showed that when assuming an arterial O₂ difference at 15 mmHg with a DS of 7.5 mmHg, 30 patients in each group would be needed for a power of 0.85 at \( P < 0.05 \). A Gaussian distribution test was done before the \( t \) test. Demographic data were analyzed using the unpaired, two-tailed \( t \) test (body mass index [BMI], weight, age), \( \chi^2 \) test (male-female), and Fisher’s exact test (smoking). Dead space volumes, ventilator plateau pressures, \( V_T \), \( S_pO_2, P_aO_2, \) and \( P_aCO_2 \) were analyzed using the unpaired, two-tailed \( t \) test. All statistical analysis was performed using SPSS statistical computing program (SPSS version 12.0, SPSS, Chicago, IL).

3. Results

All surgical procedures had an anesthesia time of more than 120 minutes. No intraoperative problems were noted during the study. All patients recovered uneventfully from anesthesia and were discharged from the hospital in accordance with normal practice for their respective surgical procedures. One patient from the NDS group was excluded from the study after 90 minutes because of a period of desaturation of \( S_pO_2 \) less than 90%. Demographic data showed no significant differences between the groups (Table 1).

All patients received ventilation with a \( FIO_2 \) of 0.35. Peak and plateau pressures and \( V_T \)s were significantly higher in the DS group than in the NDS group (Table 2). The adjustable dead space volume between the Y-piece and the heat and moisture exchanger was 239 ± 95 mL in the DS group. In the DS group, there was more rebreathing of CO₂; \( FICO_2 \) was 1.5 ± 0.8 mmHg in the DS group compared with 0.8 ± 0.0 mmHg in the NDS group (Table 2). End-tidal carbon dioxide partial pressure was 34.2 ± 1.5 mmHg in the DS group versus 33.7 ± 0.8 mmHg in the NDS group (not significant). Arterial carbon dioxide tension in the DS group was significantly lower (36.3 ± 2.1 mmHg) than that of the NDS group (37.5 ± 2.3 mmHg) (\( P < 0.05 \)) (Table 3). There was a significant arterial to \( P_ETCO_2 \) difference in both groups, though the difference was smaller in the DS group (2.2 ± 1.9 mmHg) than the NDS group (4.3 ± 2.2 mmHg) (\( P < 0.001 \)) (Table 4).

Oxygen saturation was 99% ± 1.0% in the DS group and 98.5% ± 1.5% in the NDS group (\( P < 0.05 \); Fig. 1, Table 3).

### Table 2
Comparison of tidal volumes (\( V_T \)), ventilator peak pressure (\( P_{peak} \)), ventilator plateau pressure (\( P_{plateau} \)), and inspired carbon dioxide concentration (\( FICO_2 \)) between the dead space (DS) and non dead space (NDS) groups

<table>
<thead>
<tr>
<th></th>
<th>15 min</th>
<th>60 min</th>
<th>120 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>( V_T ) (mL)</td>
<td>DS</td>
<td>742.20 ± 93.9 *</td>
<td>739.2 ± 110 *</td>
</tr>
<tr>
<td></td>
<td>NDS</td>
<td>456.60 ± 84.4</td>
<td>468.8 ± 80.9</td>
</tr>
<tr>
<td>( P_{peak} ) (cm H₂O)</td>
<td>DS</td>
<td>26.90 ± 4.1 *</td>
<td>28.23 ± 4.0 *</td>
</tr>
<tr>
<td></td>
<td>NDS</td>
<td>18.27 ± 6.0</td>
<td>19.17 ± 5.4</td>
</tr>
<tr>
<td>( P_{plateau} ) (cm H₂O)</td>
<td>DS</td>
<td>15.67 ± 3.0 **</td>
<td>16.97 ± 3.2 ***</td>
</tr>
<tr>
<td></td>
<td>NDS</td>
<td>13.70 ± 4.0</td>
<td>14.9 ± 3.3</td>
</tr>
<tr>
<td>( FICO_2 ) (mmHg)</td>
<td>DS</td>
<td>1.5 ± 0.8</td>
<td>1.5 ± 0.8</td>
</tr>
<tr>
<td></td>
<td>NDS</td>
<td>0.8 ± 0.0</td>
<td>0.8 ± 0.0</td>
</tr>
</tbody>
</table>

Values are means ± SD. 
* \( P < 0.05 \).
** \( P < 0.01 \).
*** \( P < 0.001 \).

### Table 3
Comparison of \( P_aO_2, S_pO_2, P_ETCO_2 \), and \( P_aCO_2 \) between the dead space (DS) and non dead space (NDS) groups

<table>
<thead>
<tr>
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<th>15 min</th>
<th>60 min</th>
<th>120 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>( S_pO_2 ) (%)</td>
<td>NDS</td>
<td>97.73 ± 2.36</td>
<td>97.30 ± 2.53</td>
</tr>
<tr>
<td></td>
<td>DS</td>
<td>98.70 ± 1.73 *</td>
<td>98.13 ± 1.74 **</td>
</tr>
<tr>
<td>( P_aO_2 ) (mmHg)</td>
<td>NDS</td>
<td>117.2 ± 34.7</td>
<td>113.3 ± 36.3</td>
</tr>
<tr>
<td></td>
<td>DS</td>
<td>132 ± 30.1</td>
<td>123.3 ± 30.6</td>
</tr>
<tr>
<td>( P_ETCO_2 ) (mmHg)</td>
<td>NDS</td>
<td>32.6 ± 2.3</td>
<td>33.8 ± 1.1</td>
</tr>
<tr>
<td></td>
<td>DS</td>
<td>33.8 ± 2.5</td>
<td>33.7 ± 1.1</td>
</tr>
<tr>
<td>( P_aCO_2 ) (mmHg)</td>
<td>NDS</td>
<td>37.5 ± 2.5</td>
<td>38.5 ± 1.8</td>
</tr>
<tr>
<td></td>
<td>DS</td>
<td>36 ± 2.8 *</td>
<td>36 ± 1.9 ***</td>
</tr>
</tbody>
</table>

Values means ± SD. There were no statistical differences between the groups in \( P_aO_2 \) or \( P_ETCO_2 \).
* \( P < 0.05 \).
** \( P < 0.01 \).
*** \( P < 0.001 \).
Arterial oxygen tension was 132 ± 25.5 mmHg in the DS group and 119 ± 30 mmHg in the NDS group (not significant) (Table 3).

### 4. Discussion

We tried an easy, practical way of increasing the $V_T$ ventilation, and thus FRC, without changing $P_{ET\ CO_2}$, so as to decrease ventilation-perfusion (VA/Q) mismatch. This was done by adding a dead space volume, an adjustable tube, between the Y-piece and the heat and moisture exchanger [4]. Scott and colleagues [5,6] described a variable apparatus dead space method designed to maintain normocapnia despite overventilation. However, in their study the mixing of gases was dependent on a number of factors. Our study is different in that the amount of functional dead space was not dependent on rate of fresh gas flows.

In this study, during low-flow anesthesia, the extra dead space volume was used for at least two hours of anesthesia. The main results show a significantly lower PaCO$_2$, with a slight improvement of SpO$_2$, values in the DS group, although the PaO$_2$ difference between the groups was statistically insignificant. Although there was a statistical difference in PaCO$_2$ between the groups, this difference may be accounted for by the measurement error in the blood gas analyzer alone and may be not clinically significant.

Fletcher and Jonson [7] found that the arterial CO$_2$ versus P$_{ET\ CO_2}$ difference was inversely related to $V_{Ts}$ in anesthetized patients whose lungs were ventilated with varying respiratory rates but also with constant alveolar ventilation. Their study showed a median arterial CO$_2$ versus P$_{ET\ CO_2}$ difference of 4.5 mmHg at small volumes versus 2.3 mmHg at larger $V_{Ts}$. Whiteley et al [8] confirmed these findings, that breathing patterns with longer inspiratory times yield lower values of arterial PCO$_2$. Based on the Enghoff modification of the Bohr equation, dead space volumes increased with increasing pulmonary shunt (Appendix) [9,10]. In our study, the DS group showed a smaller difference in PaCO$_2$ versus P$_{ET\ CO_2}$ compared with the NDS group. Therefore, in relation to the Enghoff modification equation and the findings of Fletcher and Jonson, our study supports the theory that the increased alveolar ventilation probably occurred because the mean arterial CO$_2$ versus P$_{ET\ CO_2}$ difference was lower in the DS group.

Hypercapnia increases cardiac output, decreases systemic vascular resistance and oxygen extraction, and increases oxygen availability to tissue [11]. Mild intraoperative hypercapnia is known to increase subcutaneous and cerebral oxygenation. By provoking local subcutaneous vasodilatation, Akca et al [12] showed that tissue Po$_2$ was greater in mildly hypercapnic patients. In our study, SpO$_2$ values in the DS group were significantly higher than in the NDS group. These findings may indicate an improved peripheral oxygenation, which could be ascribed to a smaller alveolar shunt. Our finding is consistent with those of Sykes et al [13] and Visick et al [14] that showed that larger $V_{Ts}$ improved PaO$_2$. However, our results must be interpreted with caution because the SpO$_2$ value was significantly increased without significantly improved PaO$_2$. A larger sample of patients might show a significant difference in PaO$_2$ between the groups.

The present method may be considered controversial insofar as increased $V_{Ts}$ may increase the risk of barotrauma and volutrauma. However, we believe that this method corresponds well to different patients’ thoracic compliance. The adjustable dead space volumes in the DS group were 239 ± 95 mL and reflect the variation of the different body weights and BMI. The method used in this study, with a $P_{plateau}$ of 0.04 cm H$_2$O/kg over the initial plateau pressure, yielded a maximum $P_{plateau}$ in the DS group of 18 to 21 cm H$_2$O and corresponded with a $P_{plateau}$ of 15 to 18 cm H$_2$O (NDS group) with a PEEP of 5 cm H$_2$O. The rationale for the present target of plateau pressure in this study is based on the study of Johansson et al [4], which determined that larger $V_{Ts}$ decreased CO$_2$ temperatures during low flow and minimal flow. Using their data, we calculated the plateau pressure that was used in our study.

It is true that alveolar recruitment can be achieved not only by PEEP or higher plateau pressures. In an animal study, Syring et al [15] recently described different ventilatory rates and high-frequency ventilation modes as an alternative to this issue of alveolar recruitment. However, their study was conducted in lung-injured animals, and the

### Table 4

Comparison of differences between PaCO$_2$ and P$_{ET\ CO_2}$ in the dead space (DS) and non dead space (NDS) groups

<table>
<thead>
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<th></th>
<th>15 min</th>
<th>60 min</th>
<th>120 min</th>
</tr>
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<tbody>
<tr>
<td>DS (mmHg)</td>
<td>2.3 ± 2.1 *</td>
<td>2.3 ± 1.7 *</td>
<td>2.1 ± 1.9 **</td>
</tr>
<tr>
<td>NDS (mmHg)</td>
<td>4.7 ± 2.1</td>
<td>4.7 ± 1.9</td>
<td>3.8 ± 2.3</td>
</tr>
</tbody>
</table>

Values are means ± SD.

* $P < 0.001$.

** $P < 0.01$.

Hypercapnia increases cardiac output, decreases systemic vascular resistance and oxygen extraction, and increases oxygen availability to tissue [11]. Mild intraoperative hypercapnia is known to increase subcutaneous and cerebral oxygenation.
lung mechanics are therefore completely different. Oscillatory mechanisms, for example, become more important in this setting; we think that there are lesser mechanisms in healthy lungs. The above-described method, P_{plateau} of 15 to 18 cm H\(_2\)O (NDS group) with a PEEP of 5 cm H\(_2\)O, is commonly used in general anesthesia. According to the V\(_T\)s used in the DS group, patients were not ventilated with V\(_T\)s above 10 mL/kg. We believe that all patients were ventilated within safe limits, both with regard to volume and pressures.

We were unable to detect a beneficial improvement in PaO\(_2\) in this study. However, we found that larger V\(_T\)s, created by increased apparatus dead space volumes, improved arterial CO\(_2\) elimination with a minor increase in SpO\(_2\).

**Appendix A**

The Enghoff modification of the Bohr equation:

\[ V_{Dphysiol}/V_T = (P_{acO_2} - P_{ECO_2})/P_{acO_2} \]

where \( V_{Dphysiol} \) = dead space, \( V_T \) = tidal volume, \( P_{acO_2} \) = arterial carbon dioxide tension, and \( P_{ECO_2} \) = mixed expired carbon dioxide.

**References**