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Carbon dioxide rebreathing with the anaesthetic conserving device, AnaConDa®. A laboratory study.

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Running head: Carbon dioxide rebreathing with AnaConDa®.
Summary

Background: The anaesthetic conserving device (ACD), AnaConDa®, was developed to allow the reduced use of inhaled agents by conserving exhaled agent and allowing rebreathing. Elevated $PaCO_2$ have been observed in patients when using ACD, despite tidal volume compensation for the larger apparatus dead space. The aim of the present study was to determine whether CO$_2$, like inhaled anaesthetics, adsorbs to the ACD during expiration and returns to the lung during the following inspiration.

Methods: The ACD was attached to an experimental test lung. Apparent dead space by the single-breath test for CO$_2$ and the amount of CO$_2$ adsorbed to the carbon filter of the ACD was measured with infrared spectrometry.

Results: Apparent dead space was 230 ml larger using the ACD compared with a conventional heat and moisture exchanger (internal volume 100 ml and 50 ml, respectively). Varying CO$_2$ flux to the test lung (85 – 375 ml min$^{-1}$) did not change the measured dead space, nor did varying respiratory rate (12 – 24 breaths min$^{-1}$). The ACD contained 3.3 times more CO$_2$ than the predicted amount present in its internal volume of 100 ml.

Conclusions: Our measurements show a CO$_2$ reservoir effect of 180 ml in excess of the ACD internal volume. This is due to adsorption of CO$_2$ in the ACD during expiration and the return of CO$_2$ during the following inspiration.

Key words: Airway - dead space; Anaesthetic techniques - inhalation; Carbon dioxide - rebreathing; Equipment - heat-moisture exchanger
**Background**

In the 1990s, an anaesthetic conserving device (ACD), AnaConDa® was developed to allow the reduced use of inhaled agents with open breathing circuits during anaesthesia; the ACD conserves exhaled agent and allows rebreathing.\(^1\)\(^-\)\(^3\) The efficient use of inhaled sedation with the ACD to intensive care patients has been demonstrated.\(^4\) The ACD is a modified heat and moisture exchanger (HME) with an internal volume of approximately 100 ml. Elevated levels of \(P_{aCO_2}\) have been observed in patients when using the ACD, despite tidal volume increase to compensate for the larger apparatus dead space of the ACD compared with a conventional HME.\(^5\)

The aim of this study was to test the hypothesis that the ACD retains \(CO_2\) in a way analogous to the inhaled agents, and is returned to the lungs in the next inspiration. Such effects were studied using established methods to measure dead space after adjustments of tidal volume (\(V_t\)) and/or respiratory rate (\(RR\)) in a laboratory set-up using an artificial test lung.
Methods

Fig 1 Experimental setup consisted of a ServoVentilator 900C (A), tubes to a Y-piece (B), a CO₂ transducer on ventilator side (CO₂\text{vent}, C), an HME/ACD (D), a CO₂ transducer on the lung side (CO₂\text{lung}, E), a connection tube to the experimental lung (F) and an experimental lung (G). CO₂ from a bottle (H) was fed through a rotameter (I), to the test lung. Signals for flow from the ventilator (J) and CO₂ signals from both CO₂ analyzers (K), were fed to a computer (L) and displayed on a screen (M).

The experimental set-up is shown in Fig. 1. A ServoVentilator 900C with two Analyzer 930 (Siemens-Elema AB, Solna, Sweden) mainstream CO₂ analysers ventilated a test lung in the form of a 20 litre plastic bottle with dry gas containing no anaesthetic agent. One of the CO₂ transducers (CO₂\text{vent}) was placed in the standard position between the Y-piece and the device tested, which in one single-run sequence was an HME (Vital Signs Inc., Totowa, NJ, USA), an in-active ACD in which there was no adsorbing filter and an active, off-the-shelf ACD. The second CO₂ transducer (CO₂\text{lung}) was placed between the device tested and the tube.
connecting to the test lung. The purpose of the $CO_{2\text{lung}}$ transducer was to quantify the volume of $CO_2$ returned to the test lung according to the hypothesis.

The test lung was fed with pure $CO_2$ close to its bottom. The $CO_2$ flux, modelling metabolic rate in terms of $CO_2$ production, was adjusted and controlled by a precision rotameter between the $CO_2$ bottle and the test lung. Inside the test lung, gas was mixed by a fan. Ventilator flow rate and $CO_2$ signals from both analysers were fed to a personal computer, which sampled the signals at 100 Hz. A measurement sequence comprised 10 consecutive breaths. Data from each breath in the sequence were exported to an Excel spread sheet for compilation covering a full experiment. The previously described data collection system and subsequent data analysisyielding the single breath test for $CO_2$ (SBT-CO$_2$) was applied for each of the $CO_2$ analysers.\(^6\)\(^7\)

**Procedure**

Starting with a conventional HME which has an internal volume of 50 ml, ventilator setting was: volume control 6.4 litre min$^{-1}$, RR 16 breaths min$^{-1}$ (i.e. $V_T$ 400 ml), inspiratory time 25\%, postinspiratory pause 10\% and PEEP zero; RR was not changed during this experiment. The baseline $CO_2$ flux was set to 180 ml min$^{-1}$ and was kept constant throughout the experiment. After approximately 30 minutes, steady state was observed with respect to $CO_2$ turnover as defined by the stable $CO_2$-values for endtidal concentration and elimination from the test lung. At this point, two SBT-CO$_2$ measurement sequences were performed. Then, during a prolonged post-inspiratory pause, the connecting tube was clamped, the HME was exchanged for a in-active ACD, and $V_T$ was increased by 50 ml to compensate for the volume difference between the HME and the ACD. SBT-CO$_2$ measurement sequences were performed when steady state again prevailed. Then, during a postinspiratory pause, the in-
active ACD was exchanged for an active ACD. When, as observed in pilot experiments, endtidal CO$_{2\text{vent}}$ approached the upper limit of the transducer range (10 %), $V_T$ was increased stepwise.

In a second and third series of experiments, SBT-CO$_2$ measurements with active ACD:s were performed at steady state with varying CO$_2$ flux (85 - 375 ml min$^{-1}$) and varying RR (12 - 24 breaths min$^{-1}$), respectively. In the latter series tidal volume was adjusted, aiming at a steady state endtidal CO$_2$ of 5.0 %. In a fourth set, CO$_2$ content of one active ACD at various endtidal CO$_2$-values was measured.

**Data analysis**

All recorded breaths were for each of the CO$_2$-analysers were analysed for SBT-CO$_2$ (Fig. 2). Please note that the computed SBT-CO$_2$ of the CO$_2$ fraction vs. expired volume differs from the CO$_2$ fraction vs. time graph displayed on conventional capnographs. In Figure 2, expired flow increases the accumulated expired volume rightwards. During the following inspiration, flow is plotted negative and CO$_2$ decreases towards zero and thus completes the loop of the SBT-CO$_2$ plot of the breathing cycle. We assessed airway dead space ($V_{Daw}$) by determining the volume expired at the steepest increase in the CO$_2$ fraction during expiration.$^7$ This method was chosen and validated for our test lung system, giving reproducible results as opposed to more commonly used alternatives designed for mammal lungs. The volume of CO$_2$ eliminated during a tidal breath ($V_I$CO$_2$) was determined from the area of the loop. The volume of CO$_2$ re-inspired at the start of inspiration ($V_I$CO$_2$) was determined from the area to the right of the loop.
Fig 2 The single breath test for CO₂ displays expired tidal volume on the X-axis and CO₂ fraction, %, on the Y-axis. During expiration, CO₂-free airway gas is expired first, followed by mixed airway and alveolar gas, and finally alveolar gas (alveolar plateau). Initially, during inspiration, CO₂-containing gas from the Y-piece and ventilator tubing is re-inspired. The volume of re-inspired CO₂ ($V_{ICO₂}$) is represented by the yellow area. The blue area represents the volume of CO₂ eliminated during the breathing cycle ($V_{TCO₂}$). The volume of CO₂ expired during the breath corresponds to blue plus yellow areas ($V_{TCO₂} + V_{ICO₂}$). Endtidal CO₂ ($E_{ICO₂}$) is indicated by the horizontal arrow and the airway dead space ($V_{Daw}$) by the vertical arrow.
Results

Endtidal CO₂

At baseline with the HME, endtidal CO₂vent was stable at 5.0 - 5.1 %. When the HME was replaced by the in-active ACD and Vₜ increased by 50 ml, endtidal CO₂vent remained stable (Fig. 3). When the in-active ACD was replaced by the active ACD without any further increase in Vₜ, endtidal CO₂vent increased gradually, indicating accumulation of CO₂ in the test lung. When endtidal CO₂vent approached 10 % and Vₜ was increased stepwise endtidal CO₂vent declined (Fig. 3). When Vₜ was increased by 250 ml compared with baseline Vₜ with an HME, endtidal CO₂ returned to the baseline level. With a Vₜ 300 ml higher than baseline, endtidal CO₂ decreased below the baseline level.
Fig 3 Endtidal CO$_2$ ($E_T$CO$_2$) measured at the ventilator CO$_2$ transducer using an HME, inactive ACD and active ACD (upper curve). Tidal volume was increased by 50 ml corresponding to the difference in the internal volume between HME and ACD:s, when the inactive ACD was introduced (lower curve). When the active ACD had been introduced and $E_T$CO$_2$ approached 10 %, $V_T$ was increased stepwise in order to restore the original value of $E_T$CO$_2$.

**Apparent V$_{Daw}$ and CO$_2$ volumes**

Figure 4 illustrates SBT-CO$_2$ from both CO$_2$ transducers with the HME and the active ACD, respectively. Tidal volume was adjusted to maintain isocapnia and steady state prevailed. SBT-CO$_2$ from CO$_2$vent (Fig. 4A) showed V$_{Daw}$ values comprising the volume of the HME and tubing to the test lung (142 ml) and the volume of re-inspired CO$_2$ ($V_I$CO$_2$) from the Y-piece and adjacent tubes was 1.6 ml. SBT-CO$_2$ from CO$_2$lung (Fig. 4B) showed V$_{Daw}$ values representing tubing only (101 ml), while $V_I$CO$_2$ included the volume of CO$_2$ in the HME and accordingly was higher (4.5 ml). When the ACD was studied with CO$_2$vent (Fig. 4C), no CO$_2$ appeared at the transducer until about 200 ml had been expired and V$_{Daw}$ was estimated to 372 ml. $V_I$CO$_2$ was similar to breaths with conventional HME, as in Fig. 4A. When the ACD was studied with CO$_2$lung, CO$_2$ arrived rapidly at the CO$_2$lung transducer (Fig. 4D) and V$_{Daw}$ was estimated to 113 ml. During inspiration, the fraction of inspired CO$_2$ remained high for about 200 ml and then gradually decreased towards zero as CO$_2$ washout from the ACD progressed. Of the total expired volume of CO$_2$ (13.3 + 15.5 ml), more than 50 % was re-inspired (15.5 ml). Varying CO$_2$ flux to the test lung between 85 and 375 ml min$^{-1}$ did not change the measured V$_{Daw}$. Varying RR between 12 and 24 breaths min$^{-1}$, with concomitant changes of tidal volume to maintain constant endtidal CO$_2$, also did not alter V$_{Daw}$ (Table 1). The use of higher respiratory rate allowed lower tidal volumes to maintain isocapnia.
Fig 4 Single breath tests for CO₂ (SBT-CO₂) with an HME (A and B) or active ACD (C and D) registered on ventilator (A and C) or lung (B and D) side of the device, respectively. All volumes in millilitres. The volume of CO₂ eliminated during the breath ($V_{TCO₂}$, blue area) and inspired ($V_{tCO₂}$, yellow area) is indicated. Apparent airway dead space ($V_{Daw}$) is indicated by the vertical arrow. SBT-CO₂ was studied after adjustment of tidal volume to maintain isocapnia and in steady state with respect to CO₂ elimination. As illustrated in panel C, the effect of the ACD is analogous to a large increase in $V_{Daw}$ observed on the ventilator side of the device. In panel D this is reflected by the yellow area, representing a large volume of re-inspired CO₂ from the ACD.
Table 1  Tidal volume ($V_T$), steady state endtidal CO$_2$, and apparent dead space ($V_{Daw}$) at different respiratory rates (RR) with an active ACD.

<table>
<thead>
<tr>
<th>Respiratory rate (min$^{-1}$)</th>
<th>Tidal volume (ml)</th>
<th>Endtidal CO$_2$ (%)</th>
<th>Apparent dead space (ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>12</td>
<td>760</td>
<td>5.1</td>
<td>372</td>
</tr>
<tr>
<td>16</td>
<td>676</td>
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<tr>
<td>20</td>
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<td>364</td>
</tr>
<tr>
<td>24</td>
<td>560</td>
<td>5.3</td>
<td>372</td>
</tr>
</tbody>
</table>

**ACD CO$_2$ content**

Steady state endtidal CO$_2$ values between 1 % and 8 % were achieved by modifying the CO$_2$ flux. The volume of CO$_2$ retained in the active ACD, $V_{CO_2ACD}$, was measured with the CO$_2$lung transducer after disconnection of the test lung during a post-expiratory pause. During the first breath 95 % of this volume was washed out and after four breaths all detectable CO$_2$ had been eliminated. A strict proportionality between the $V_{CO_2ACD}$ and $E_T$CO$_2$ was found: $V_{CO_2ACD} = 3.3 \times E_T$CO$_2$ ($R^2 = 0.99$, Fig. 5). Thus, the active ACD contained 3.3 times more CO$_2$ than the predicted amount present in its 100 ml of gas.
**Fig 5** Amount of CO₂ in millilitres eliminated from the active ACD at different CO₂-fractions.
Discussion

The present investigation was performed in a laboratory setting in order to allow studies which cannot be done in animal or man, by allowing wide ranges of $V_t$, respiratory rate and CO$_2$.

Our measurements consistently showed that the introduction of an active ACD was associated with a CO$_2$-free expire equivalent to an airway dead space increase exceeding the internal volume of the device by 180 ml regardless of the CO$_2$ flux or respiratory rate. Furthermore, we found a substantially increased volume of re-inspired CO$_2$ when using the active ACD. The hypothesis was thus confirmed; CO$_2$ is adsorbed by the ACD during expiration and returned to the lung during inspiration. The rapid washout of CO$_2$ adsorbed in the device shows that CO$_2$ is present only in a single fast compartment. The linearity of the relationship between end-tidal CO$_2$ and CO$_2$ volume retained in the active ACD implies that the device is not saturated even at a CO$_2$ concentration of 8 %. It can therefore be concluded that the active ACD has a high CO$_2$-binding capacity, the upper limit of which was not explored.

These results agree with our observations in patients$^5$ that isocapnia is not maintained by merely increasing $V_t$ with the extra internal volume of the active ACD. In that study, the mean $PaCO_2$ was 1.1-1.4 kPa higher in patients where the ACD was used, and we suggested caution whenever using the ACD in situations where tight $PaCO_2$ control is necessary. Thus, endtidal CO$_2$ was only moderately increased compared with the present results. In the present study, applying such tidal volume compensation, endtidal CO$_2$ increased from 5 % to at least 10 %. This data may indicate that the dead space effect of the ACD in patients is less than in the present laboratory setting. A steady state is not quite achieved in patients within 30 minutes due to the large volume of exchangeable CO$_2$ stored in the body.$^8$ This may to some
extent explain the difference between the degrees of CO₂ retention observed in the two studies. Tentative reasons for the seemingly different behaviour of the ACD in the present study are that in the present study humidity was zero, the temperature was that of the room and that no inhaled anaesthetic was used. This may also explain why CO₂ retention has not been addressed as a significant problem in previous reports on the clinical use of the ACD.²⁻⁴

We explored the effects of VT and RR on CO₂ elimination in this laboratory model. Increasing VT was effective in restoring endtidal CO₂ after introduction of the ACD. Somewhat shorter postoperative time to extubation when using smaller tidal volumes has recently been demonstrated in cardiac surgery patients suggesting a possibly negative effect of large volumes.⁹ If the findings in the present study are representative of effects in the clinical use of the ACD, it will be important to recognize risks associated with large tidal volumes. On the other hand, using higher respiratory rate allowed lower tidal volumes to maintain isocapnia in our test lung. If this also applies to the mammal respiratory system, increasing respiratory rate could be a possible strategy for compensating impaired CO₂ excretion induced by the use of an ACD.

In conclusion, we have demonstrated that the ACD adsorbs large amounts of exhaled CO₂ which is returned during the next inhalation creating a dead space effect. Further studies on factors which modify this effect are required before our results can be allowed to modify clinical practice.
**Acknowledgements**

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Declaration of interests

The authors wish to acknowledge the generous supply of in-active ACD:s by Sedana Medical AB, Uppsala, Sweden. There was no other conflict of interests.
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List of references


