Oxygen Supplementation in Noninvasive Home Mechanical Ventilation: The Crucial Roles of CO₂ Exhalation Systems and Leakages

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BACKGROUND: When supplemental oxygen is added to noninvasive ventilation using a non-ICU ventilator, it is usually introduced with a preset flow into the circuit near the ventilator; however, the impact of different CO₂ exhalation systems and leaks on the actual FIO2 and gas exchange has not been elucidated. METHODS: In a randomized, open-label, 4-treatment (2-by-2), 4-period crossover design, 4 daytime measurements (60 min each) were performed in 20 subjects receiving home mechanical noninvasive ventilation plus supplemental oxygen (≥ 2 L/min) inserted near the ventilator: active valve circuit or leak port circuit with or without artificial leakage (4 mm inner diameter). Oxygen concentration near the ventilator, oxygen concentration at the mask, and blood gases were measured. RESULTS: Overall, oxygen concentration at the mask (29 ± 5%) was lower than oxygen concentration at the ventilator (34 ± 4%), with a mean difference of 5.1% (95% CI 4.2–5.9%, P < .001%). With the leak port circuit, oxygen concentration at the mask decreased by 3.2% (95% CI 2.6 to 3.9%, P < .001), compared to the active valve circuit. When artificial leakage was introduced into the circuit, oxygen concentration at the mask decreased by 5.7% (95% CI 5.1 to 6.4%, P < .001%), PaO₂ by 10.4 mm Hg (95% CI 3.1 to 17.7 mm Hg, P = .006), and PaCO₂ increased by 1.8 mm Hg (95% CI 0.5 to 3.1 mm Hg, P = .008). CONCLUSIONS: The use of a leak port circuit and the occurrence of leak around the interface significantly reduced oxygen concentration at the mask and negatively impacted gas exchange in subjects receiving home noninvasive ventilation and supplemental oxygen. (German Clinical Trials Registry, www.drks.de, DRKS00000449). Key words: COPD, mechanical ventilation, chronic respiratory failure, long-term oxygen therapy. [Respir Care 2014;59(1):113–120. © 2014 Daedalus Enterprises]
suffer from both hypoxemic and hypercapnic respiratory failure.\textsuperscript{5,6}

When using an ICU ventilator, a fixed $\text{FiO}_2$ is set during ventilatory support. In contrast, in smaller and portable ventilators outside the ICU oxygen is typically placed directly into the circuit, using a constant flow. However, in the latter scenario the actual inspired oxygen concentration is unknown and, importantly, is dependent on several factors: oxygen flow, leakage, circuit, and interface.\textsuperscript{7-9} The effect of an altered oxygen flow on inspired oxygen concentration remains unclear in the clinical setting, as this is not regularly measured. Furthermore, conflicting results exist in the literature about whether $\text{FiO}_2$ is dependent on the location of oxygen insertion and the presence of leak\textsuperscript{7,10-13}; this is most likely attributable to the fact that the majority of relevant studies were performed in vitro, using test lungs.\textsuperscript{7-9,11-13} So far, no study has been performed in patients with hypoxemic and hypercapnic respiratory failure where the impact on gas exchange may differ substantially, depending on the underlying pathology. In addition, the impact of leak on $\text{FiO}_2$ has not been conclusively investigated.

Two different single-tube circuits are regularly used for NIV: the leak port circuit, and the active valve circuit.\textsuperscript{14} In the leak port circuit an intentional leak is integrated, into the circuit or the mask, to wash out CO\textsubscript{2}.\textsuperscript{15} In contrast, in an active valve circuit an exhalation port opens during expiration and closes during inspiration.

The present study investigated the effects of unintentional leak during NIV, and intentional leak in the circuit (to promote CO\textsubscript{2} elimination) on $\text{FiO}_2$, oxygenation, dyspnea, and alveolar ventilation. We hypothesized that both intentional and unintentional leak would reduce oxygenation and ventilation.

Methods

The study protocol was approved by the institutional review board for human studies at Albert-Ludwigs University, Freiburg, Germany, and was performed in accordance with the Declaration of Helsinki. Written informed consent was obtained from all subjects.

Dr Dreher has disclosed relationships with VitalAire, ResMed, Dräger, Respironics, Vivisol, and Linde. Dr Windisch has disclosed relationships with Maquet, Dräger, Heinen und Löwenstein, Respironics, Weinmann, ResMed, Cividien, Linde, Siare, and Breas. Dr Schmoor has disclosed no conflicts of interest.

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QUICK LOOK

Current knowledge

Oxygen supplementation during noninvasive ventilation is typically accomplished by adding a constant flow of oxygen into the ventilator circuit. The site of oxygen injection, leak at the interface, and the type of exhalation valve may impact the delivered oxygen concentration.

What this paper contributes to our knowledge

The oxygen concentration at the mask of subjects receiving noninvasive ventilation was lower with a fixed leak circuit than with a circuit with an active exhalation valve. The oxygen concentration was also diminished as leak increased, as a consequence of higher ventilator flow as part of the leak-compensation system.

Subjects

Stable NIV patients without evidence of acute respiratory failure, signs of respiratory infection (eg, fever, purulent sputum), or severe obesity (body mass index $>35\text{ kg}/\text{m}^2$) were included. Subjects were recruited during routine follow-up visits at University Hospital Freiburg, after previously being established on long-term NIV.\textsuperscript{16} Subjects had to have been on home NIV for at least 2 months prior to the study, with supplemental oxygen of $\geq 2\text{ L/min}$.

Measurements

Lung function parameters (Masterlab-Compact Labor, Erich Jaeger, Friedberg, Germany) were assessed in accordance with international guidelines.\textsuperscript{17,18} Arterial blood samples were taken from the arterialized earlobe and analyzed (AVL Omni, Roche Diagnostics, Basel, Switzerland).

Oxygen concentration was measured with 2 sensors (E-17/J, Nuova, Ratzeburg, Germany) connected to the ends of the ventilator circuit (Fig. 1).

Study Design

The study had a randomized, open-label, 4-treatment (2-by-2), 4-period, crossover design. It was performed as a single-center study, at the Department of Pneumology, University Medical Center Freiburg, Freiburg, Germany, between January and August 2010. The study setup is illustrated in Figure 1. Two ventilators capable of measuring oxygen concentration (Vivo 50, Breas Medical,
Mo¨lnlycke, Sweden) were used during all measurements. The first ventilator was used for ventilating the subject and measuring oxygen concentration at the mask. The second ventilator was connected to a test lung and exclusively measured the oxygen concentration near the ventilator. Ventilation parameters and oxygen flows were adopted from the subject’s last-used home NIV settings, and were not changed during any measurements. The oxygen was given from the hospital wall socket, via an oxygen adapter (Sauerstoff adapter 901963, Medisize Deutschland, Neunkirchen-Seelscheid, Germany), to the ventilator circuits proximal to the ventilator. During all measurements, all subjects used an oronasal mask (Ultra Mirage, ResMed, San Diego, California) that does not have integrated exhalation ports.

An artificial leak (Oxygen Adapter 1974, Medisize Deutschland, Neunkirchen-Seelscheid, Germany, inner diameter 4 mm) was inserted into the circuit between the exhalation port and the mask, to simulate unintentional leak, as described previously.16 The reusable patient circuit with exhalation valve (5055, Breas Medical, Mölnlycke, Sweden) was used for all the active valve circuit tests. For the leak port circuit setups, subjects were ventilated with a reusable circuit with leak (5065, Breas Medical, Mölnlycke, Sweden) connected to a passive exhalation port (Silentflow 2, Weinmann & Co, Hamburg, Germany).

The effects of the circuit type (active valve vs leak port) and leak type (artificial leak vs no leak) were both investigated. The subjects were ventilated during daytime with 4 different settings, in a 2-by-2 design; each setup was studied for 60 min (see Fig. 1).

• Active valve circuit without artificial leak (active-valve-circuit-without-leak)
• Leak port circuit without artificial leak (leak-port-circuit-without-leak)
• Active valve circuit with artificial leak (active-valve-circuit-with-leak)
• Leak port circuit with artificial leak (leak-port-circuit-with-leak)

Subjects were randomized to 4 test sequences:


Measurements were performed on 2 consecutive days (2 sequences/day). Between each sequence there was a wash-out period of 120 min during which the subject received oxygen at his or her usual home-treatment flow without NIV. After each measurement the subject was asked to state his or her level of dyspnea, according to the Borg dyspnea scale.19

Study End Points

The aim of the study was to assess the differences between active valve and leak port circuit, and the impact of an artificial leak. The primary end point was the difference in the mean oxygen concentration at the mask between the active valve and leak port circuits during 60 min of daytime NIV. Here we hypothesized that oxygen concentration at the mask (ie, the actual FIO2) would be 10% lower with the leak port circuit than with the active valve circuit. Further end points were oxygen concentration near the ventilator, Pao2, Paco2, and Borg dyspnea score.

Statistics

Sample size calculation was based on the primary end point: oxygen concentration at the mask. The study was designed to show at a 2-sided significance level of .05 with a power of .90 a difference between the active valve circuit and the leak port circuit, when the true absolute oxygen concentration difference was 2.5%. A standard deviation in the difference of 3% oxygen concentration was assumed, according to previous findings.8 Under these assumptions, recruitment of 20 subjects was required.
The effect of active valve circuit versus leak port circuit was analyzed by comparing the active-valve-circuit-without-leak and active-valve-circuit-with-leak setups to the leak-port-circuit-without-leak and leak-port-circuit-with-leak setups. The effect of artificial leak was analyzed by comparing the active-valve-circuit-without-leak and leak-port-circuit-without-leak setups to the active-valve-circuit-with-leak and leak-port-circuit-with-leak setups. Additionally, an analysis of interaction between type of circuit and an additional leak was performed.

All subjects who were ventilated with all 4 setups in the crossover setting were included in the analysis. Analysis of variance models were used, assuming normal distribution of data, with circuit, artificial leak, the interaction between circuit and artificial leak, period, and randomized sequence defined as fixed effects, and subject within sequence defined as a random effect. For analysis of oxygen concentration at the mask, the oxygen concentration near the ventilator measurement was included as a fixed effect in the model for adjustment. For analysis of PaO2 and PaCO2 after ventilation, the measurements at the start of ventilation were included as a fixed effect in the model for adjustment. Treatment effects and interactive effects were estimated with 95% CI and tested with a 2-sided level of .05.

Table 1. Demographic and Lung Function Data

<table>
<thead>
<tr>
<th></th>
<th>COPD n = 11</th>
<th>No COPD n = 9</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male/female, no</td>
<td>6/5</td>
<td>6/3</td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td>55.2 ± 8.3</td>
<td>72.1 ± 10.1</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>26.7 ± 8.7</td>
<td>27.9 ± 5.6</td>
<td>.29</td>
</tr>
<tr>
<td>Months on noninvasive ventilation</td>
<td>28.0 ± 34.8</td>
<td>62.3 ± 56.8</td>
<td>.32</td>
</tr>
<tr>
<td>Months on oxygen therapy</td>
<td>35.4 ± 36.6</td>
<td>52.2 ± 57.8</td>
<td>.76</td>
</tr>
<tr>
<td>FEV1, % predicted</td>
<td>24.7 ± 11.1</td>
<td>38.3 ± 8.3</td>
<td>.01</td>
</tr>
<tr>
<td>FVC, % predicted</td>
<td>48.4 ± 11.1</td>
<td>43.2 ± 17.7</td>
<td>.43</td>
</tr>
<tr>
<td>FEV1/FVC, %</td>
<td>43.6 ± 11.3</td>
<td>73.6 ± 10.6</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Residual volume, % predicted</td>
<td>261.4 ± 63.0 (n = 9)</td>
<td>84.8 ± 35.9</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Total lung capacity, % predicted</td>
<td>127.3 ± 33.0 (n = 9)</td>
<td>58.6 ± 19.3</td>
<td>&lt; .001</td>
</tr>
</tbody>
</table>

Values are mean ± SD.

The effect of active valve circuit versus leak port circuit was analyzed by comparing the active-valve-circuit-without-leak and active-valve-circuit-with-leak setups to the leak-port-circuit-without-leak and leak-port-circuit-with-leak setups. The effect of artificial leak was analyzed by comparing the active-valve-circuit-without-leak and leak-port-circuit-without-leak setups to the active-valve-circuit-with-leak and leak-port-circuit-with-leak setups. Additionally, an analysis of interaction between type of circuit and an additional leak was performed.

All subjects who were ventilated with all 4 setups in the crossover setting were included in the analysis. Analysis of variance models were used, assuming normal distribution of data, with circuit, artificial leak, the interaction between circuit and artificial leak, period, and randomized sequence defined as fixed effects, and subject within sequence defined as a random effect. For analysis of oxygen concentration at the mask, the oxygen concentration near the ventilator measurement was included as a fixed effect in the model for adjustment. For analysis of PaO2 and PaCO2 after ventilation, the measurements at the start of ventilation were included as a fixed effect in the model for adjustment. Treatment effects and interactive effects were estimated with 95% CI and tested with a 2-sided level of .05.

Table 2. NIV Settings and Oxygen Flow

<table>
<thead>
<tr>
<th></th>
<th>COPD n = 11</th>
<th>No COPD n = 9</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxygen flow, L/min</td>
<td>2.6 ± 0.2</td>
<td>2.7 ± 1.3</td>
<td>.86</td>
</tr>
<tr>
<td>Interface size</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Small</td>
<td>3</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Medium</td>
<td>7</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Large</td>
<td>1</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Ventilation mode</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pressure support ventilation</td>
<td>2</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Pressure controlled ventilation</td>
<td>0</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Assisted pressure controlled ventilation</td>
<td>9</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>Inspiratory pressure, cm H₂O</td>
<td>26.4 ± 5.2</td>
<td>23.4 ± 4.3</td>
<td>.17</td>
</tr>
<tr>
<td>Expiratory pressure, cm H₂O</td>
<td>4.8 ± 1.3</td>
<td>4.0 ± 1.8</td>
<td>.25</td>
</tr>
<tr>
<td>Set breathing frequency, breaths/min</td>
<td>17.0 ± 3.2</td>
<td>18.8 ± 2.7</td>
<td>.21</td>
</tr>
<tr>
<td>Inspiratory time, s*</td>
<td>1.1 ± 0.1 (n = 9)</td>
<td>1.2 ± 0.2 (n = 8)</td>
<td>.09</td>
</tr>
<tr>
<td>Inspiratory flow trigger setting†</td>
<td>3.7 ± 1.6</td>
<td>4.6 ± 1.8</td>
<td>.30</td>
</tr>
<tr>
<td>Expiratory trigger‡</td>
<td>3.5 ± 0.7 (n = 2)</td>
<td>5 (n = 1)</td>
<td></td>
</tr>
<tr>
<td>Rise time</td>
<td>1.6 ± 0.7</td>
<td>3.1 ± 2.7</td>
<td>.21</td>
</tr>
</tbody>
</table>

Values are mean ± SD.

* Set inspiratory time (in pressure support ventilation) range: 0.5–2.0 s.
† Inspiratory flow trigger setting range: 1 = most sensitive, 9 = least sensitive.
‡ Expiratory trigger setting (in pressure support ventilation) range: 1 = 90% of peak flow, 9 = 10% of peak flow.
For the Borg dyspnea scores the assumption of a normal distribution was not fulfilled, so non-parametric analysis was performed, using Wilcoxon tests and Hodges-Lehmann estimators of the crossover differences.

In addition, tests for period and carry-over effects (ie, treatment-period interactions) were performed, which showed no relevant effects.

Results

Twenty-three subjects were included in the study, and 20 subjects completed all measurements. Two subjects refused to be ventilated with the active-valve-circuit-with-leak setup, and one subject refused to be ventilated with the leak-port-circuit-with-leak setup, due to dyspnea. Demographic data and lung function parameters of the 20 subjects who completed the study are given in Table 1. Eleven subjects suffered from COPD. Nine subjects had restrictive ventilatory disorders (no COPD): obesity-hypoventilation syndrome ($n = 3$), kyphoscoliosis ($n = 3$), unclassified interstitial lung disease ($n = 1$), post-polio syndrome ($n = 1$), and phrenic nerve paralysis ($n = 1$). NIV settings and oxygen flows are shown in Table 2.

After summarizing all measurements from all 4 setups ($n = 80$), the mean ± SD oxygen concentration at the mask (29.1 ± 4.5%) was found to be lower than the oxygen concentration near the ventilator (34.2 ± 4.0%), with a mean difference of 5.1% (95% CI 4.2 to 5.9%, $P = .001$) during 60 min of NIV and supplemental oxygen. The drop in oxygen concentration along the circuit arose irrespective of the circuit being used or of the presence of artificial leak.

First, regarding the 2 circuits, the drop in oxygen concentration with the active valve circuit was 3.4% (95% CI 2.4 to 4.4%, $P = .001$), while using the leak port led to a 6.6% drop (95% CI 5.6 to 7.7%, $P = .001$); this revealed a treatment effect of 3.2% (95% CI 2.6 to 3.9%, $P < .001$) in the drop of oxygen concentration (active valve vs leak port).
Second, without the artificial leak the drop in oxygen concentration was 2.2% (95% CI 1.1 to 3.2%, \(P < .001\)), while inserting the leak led to a drop of 7.9% (95% CI 6.8 to 8.9, \(P < .001\)), thus representing a treatment effect of 5.7% (95% CI 5.1 to 6.4%, \(P < .001\)) in the drop of oxygen concentration (without vs with artificial leak).

The oxygen concentration measurements are given in Table 3, Figure 2, and Figure 3. Our hypothesis was confirmed, since oxygen concentration at the mask using the leak port circuit was about 10% lower than that with the active valve circuit. Table 3 also shows the effects on gas exchange after 60 min of NIV. \(P_{aO_2}\) tended to be lower after 60 min with the leak port circuit than with the active valve circuit. The addition of artificial leak substantially decreased \(P_{aO_2}\) (\(P = .006\)) and increased \(P_{aCO_2}\) (\(P = .008\)).

The median Borg dyspnea scores reached 0 (IQR 0–2.75) with the active-valve-circuit-without-leak setup, 0 (IQR 0–2.5) with the leak-port-circuit-without-leak setup, 0 (IQR 0–2) with the active-valve-circuit-with-leak setup, and 1 (IQR 0–3) with the leak-port-circuit-with-leak setup. There were no significant differences in Borg dyspnea score, either between the 2 circuits (active valve vs leak port \(P = .12\)), or with artificial leak (no vs yes 0, 95% CI –0.25 to 0.5, \(P = .83\)).

**Discussion**

Three major findings arise from this study. First, there is a substantial drop in oxygen concentration along the tubing from the ventilator to the interface, irrespective of the circuits used or the presence of unintentional leak.

Second, differences in the oxygen concentration measured at the mask are evident when different ventilatory circuits and their exhalation ports are compared. Here, the oxygen concentration at the mask was lower with the leak port circuit than with the active valve circuit, despite the fact that the same oxygen flow was fed into the ventilator circuit proximal to the ventilator.

Finally, adding artificial leak next to the mask to simulate unintentional leakage decreased oxygen concentration at the mask, which decreased \(P_{aO_2}\) and increased \(P_{aCO_2}\).

These findings have 2 clinically important implications. First, oxygen concentration at the mask was lower with a leak port circuit than with an active valve circuit. Furthermore, \(P_{aO_2}\) was approximately 10 mm Hg lower at end of the trial when an artificial leak was implemented. This drop in \(P_{aO_2}\) is very important, even though \(P_{aO_2}\) did not decrease to a critical value in the clinically stable subjects we investigated here. In a study using a test lung and healthy volunteers, obtaining oxygen concentrations higher than 50% was not feasible with the leak port system, despite oxygen flows up to 16 L/min.\(^7\) Preferably, patients who require supplemental oxygen should receive NIV via an active valve circuit, if a portable ventilator is used. In addition, the authors do not recommend using supplemental oxygen flow inserted into a T-piece proximal to the ventilator in patients suffering from acute hypoxemic respiratory failure, in which adequate oxygenation is the primary aim of acute NIV.\(^1,2,20\) In this scenario, an ICU ventilator, with which a fixed oxygen concentration can be set, is preferable for NIV in pure acute hypoxemic respiratory failure patients. As pointed out above regarding blood gas deterioration, a substantial drop in \(P_{aO_2}\) would
harm a patient with acute or more severe chronic hypoxic respiratory failure.

Second, leak during NIV should be kept to a minimum, since leak in the present study reduced oxygen concentration at the mask and $P_{\text{aO}_2}$, and increased $P_{\text{aCO}_2}$. In addition, the deleterious effects of leak on inspired oxygen concentration and gas exchange are exacerbated when a leak port circuit is used. Therefore, if leak is unavoidable, an active valve circuit would be preferable, to minimize the negative impact of leak on gas exchange. Today most patients receive pressure-preset NIV for home mechanical ventilation, since this is better tolerated, less expensive, and has leak-compensation capabilities. In contrast, volume-preset NIV produces more gastrointestinal side effects, is more expensive, and does not compensate for leak. Pressure-pre-set NIV is also primarily being used in acute respiratory failure patients. However, when the pressure-preset mode increases the inspiratory flow as a means of leak compensation, higher amounts of room air with oxygen concentration of 21% are mixed with the constant amount of oxygen delivered according to the preset oxygen flow. As a consequence, inspired oxygen concentration decreases in different etiologies of chronic ventilatory failure and their settings of ventilatory support, as clearly demonstrated in this study. A further drop of oxygen concentration in the circuit can be attributed to the presence of expiratory positive airway pressure, which maintains a flow during expiration. In line with previous findings the current results suggest that leaks cause a decrease in minute ventilation, as demonstrated by the increase of $P_{\text{aCO}_2}$, which negatively impacts on oxygenation. Thus, the advantage of pressure-preset NIV in terms of leak compensation can also serve as a disadvantage for the oxygenation of patients receiving supplemental oxygen in addition to NIV, and this seems to be valid for both acute and chronic respiratory failure patients.

Limitations

First, all measurements were performed during daytime, with one ventilator, and with an oronasal mask. Consequently, these results cannot directly be extrapolated to nocturnal circumstances, to different ventilators, or to other NIV interfaces. However, the amount of leak arising from the artificial leak we used was overall comparable to leak encountered during nocturnal NIV. The artificial leak we used remained constant throughout the entire ventilation time. In clinical practice the degree of leak changes over time, due to the individual circumstances of the patient. Thus, it could be speculated that a larger leak could have an even more deleterious impact on oxygenation and gas exchange. However, this needs further investigation.

Second, only 2 different exhalation ports and ventilatory circuits were tested. Other NIV systems are available, which might differ in dead space and resistance, thus potentially influencing oxygenation. In particular, exhalation ports directly integrated into the face mask were not investigated in this study. Interfaces with exhalation ports embedded into the mask were excluded, since their influence on $F_{\text{IO}_2}$ could not have been monitored with the oxygen sensor used in this study. However, in line with the current findings, a previous study showed a drop in $F_{\text{IO}_2}$ when using these interfaces. Furthermore, oxygen was integrated into the circuit near the ventilator, and hence was not in the vicinity of the patient’s interface. Indeed, there are conflicting results in the literature regarding the favored site of oxygen insertion. While some studies reported an increase in $F_{\text{IO}_2}$ when supplemental oxygen is inserted more proximal to the patient or the mask, others have found opposite results, with a lower $F_{\text{IO}_2}$ when oxygen is inserted next to the mask. One study found no effect of changing the position of oxygen insertion. However, the insertion of oxygen near the ventilator is recommended by many manufacturers as well as guidelines, and is suggested to be more convenient because the risk of disconnection is lower when oxygen is inserted close to the mask. This rationale therefore supported the decision in the present study to insert oxygen near the ventilator, and different results by inserting oxygen at other ports could not be excluded.

Third, we obtained arterial blood samples from the arterialized earlobe. This may have led to different results than analyzing arterial blood samples, especially focusing $P_{\text{aO}_2}$. However, our subjects were clinically stable, so insertion of an arterial line was avoided. Furthermore, a recently published meta-analysis demonstrated that capillary $pH$ and $P_{\text{aCO}_2}$ accurately reflect arterial values, and capillary $P_{\text{aO}_2}$ can be appropriate for replacing $P_{\text{aO}_2}$, respectively.

Conclusions

Oxygen concentration at the mask of subjects receiving NIV and supplemental oxygen was lower with a leak port circuit than with an active valve circuit. A further reduction in $F_{\text{IO}_2}$ occurs in the presence of air leak, and this directly translates into a deterioration of gas exchange. As a consequence, active leak valve circuits are preferable to leak port circuits when oxygen is administered in addition to NIV.

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