Effect of Supplemental Gases on End-tidal CO₂ and Oxygen Saturation in Patients Undergoing Fentanyl and Midazolam Outpatient Sedation

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Forty-six American Society of Anesthesiologists Class I and II adults were randomly assigned to one to two study groups. Each subject received 0.7 μg/kg of fentanyl and a titrated dose of midazolam. One group received 100% supplemental oxygen (O₂) while another group received 50% nitrous oxide (N₂O) and 50% O₂. End-tidal carbon dioxide (EtCO₂) and O₂ saturation (SpO₂) were measured at 5-min intervals throughout the procedure. We conclude that there was no significant difference in EtCO₂ or O₂ saturation between the two groups.

Key Words: Nitrous oxide; Outpatient sedation.

The administration of nitrous oxide (N₂O) and oxygen (O₂) for outpatient oral and maxillofacial surgical procedures is a well-recognized and accepted practice.1 Lytle and Yoon6 reported that N₂O was used more frequently than local anesthesia and was second only to O₂ as the most commonly used agent for dental outpatient anesthesia. Because of the widespread use of N₂O, it is not surprising that it has been used in conjunction with intravenous sedation.3

Conscious sedation in conjunction with regional anesthesia is an accepted method of rendering patients comfortable and cooperative for outpatient procedures.4,5 Inherent in the concept of conscious sedation is the fact that at no time is sedation deep enough to deprive the patient of protective reflexes. Intravenous opioid administration followed by a titrated dose of benzodiazepine is a frequently used conscious sedation technique. Stuebner8 has reported that adding N₂O and O₂ to intravenous sedatives is a popular modification of this technique.

Numerous studies3–10 have shown various degrees of respiratory depression following the intravenous administration of diazepam, fentanyl, or methohexital. Because of these findings, many authors6–13 recommend the addition of supplemental O₂ to the sedation regimen. Kraut14 showed significant differences in oxygen tensions in sedated patients receiving 100% O₂ compared with patients breathing room air. Even if the patient does not reach a depth of anesthesia that compromises protective reflexes, hypoxia is a potential danger. A conscious patient may be significantly hypoxic without clinical evidence of hypoxia.14

This study compared the end-tidal carbon dioxide (EtCO₂) tension and O₂ saturation (SpO₂) in patients receiving 100% O₂ to those receiving 50% N₂O and 50% O₂ during intravenous sedation for outpatient oral surgery.

METHODS

Forty-two American Society of Anesthesiologists Class I and II patients over the age of 18 who presented to the Oral and Maxillofacial Surgery Service of the Montefiore Medical Center, Bronx, NY, for removal of teeth requiring a minimum of 20 min of operating time were entered into this Institutional Review Board-approved study. A power analysis was performed to determine the number of patients required to detect a clinically no-

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tested for escort. For data computed were patients surgically removed. Onset of baseline cal anesthesia was initiated. Maximum administered. Each ter (preoperative placed, administration of 50% N2O and 50% O2. Treatment groups were unblinded.

Arterial blood pressures (Dinamap 18465x, Critikon, Tampa, FL), heart rate, SpO2, and EtCO2 were monitored (601 Poet, Criticare Systems, Inc., Milwaukee, WI). The EtCO2 monitor used nasal prongs. All monitored parameters were automatically recorded at 5-min intervals throughout the surgical period.

After a baseline determination of the above parameters (preoperative baseline), the nasal hood was placed, and O2 with or without N2O was administered for 3 min before administration of intravenous sedation. During this period, an indwelling intravenous cannula was placed, and the administration of 0.9% sodium chloride was initiated. After 3 min, fentanyl and midazolam were administered. Each patient received 0.7 μg/kg of fentanyl. Midazolam was then slowly titrated to effect, with a maximum allowable dose of 10 mg.

Once the patient was adequately sedated, start of surgery baseline measurements were obtained and locoal anesthesia (2% lidocaine hydrochloride with 1: 100,000 epinephrine) was administered. Following the onset of regional anesthesia, the indicated teeth were surgically removed. After the surgery was completed, the gaseous mixture was changed in Group B to 100% O2 for a 5-min period. When the patients were sufficiently alert and their vital signs stable on room air, the patients were discharged from the clinic in the care of their escort.

Changes from each baseline parameter were computed for data evaluated at each timepoint and were tested for significance using paired t-tests of Wilcoxon table difference between groups with 80% power, using a two-tailed test with a Type I error of 0.05. All patients were asked to sign an institutionally approved consent prior to participating in this study. The patients were instructed not to eat or drink for 6 hr before their surgical appointments and were accompanied by escorts to and from the clinic on the day of surgery.

A table of random numbers was used to assign patients to the study groups. Group A received a 6-L/min flow of 100% O2 via a nasal hood during the surgical procedure. Group B received a 6-L/min flow of 50% N2O and 50% O2. Treatment groups were unblinded.

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Changes from each baseline parameter were computed for data evaluated at each timepoint and were tested for significance using paired t-tests of Wilcoxon
between the groups derived from the repeated-measures analysis of variance were not significant (Table 3). Time trends for the two groups were significant for timepoint values, for changes from preoperative baseline, and for changes using the start of surgery baseline measure ($P < 0.05$).

Saturation values within the O$_2$-only group demonstrated significant differences at 10 min and at the end of surgery when compared to preoperative baseline ($P < 0.05$). There were no significant changes from the preoperative baseline in the N$_2$O group (Table 4). There were no significant changes from the preoperative baseline in the N$_2$O group (Table 4). Using a repeated-measures analysis of variance, there were no significant differences between the groups over time with regard to SpO$_2$ (Table 5).

There were no complications for patients in either treatment group, and all patients were hemodynamically stable throughout surgery.

**DISCUSSION**

The addition of 50% N$_2$O did not have a clinically notable effect on EtCO$_2$ or SpO$_2$. Patients in both groups of this study appeared well sedated and comfortable. The surgeons found no difference in operating conditions for either group. Previous studies have demonstrated decreased O$_2$ saturation during intravenous sedation when supplemental O$_2$ is not administered.$^{14,15}$ This study demonstrated significant differences in SpO$_2$ at 10 min and at the end of surgery when compared to baseline in the O$_2$ group. When reminded to breathe more deeply, the SpO$_2$ quickly returned to baseline. These findings reinforce the need for O$_2$ monitoring with pulse oximetry during conscious sedation. Administration of supplemental O$_2$ via nasal cannula or nasal mask are effective methods in maintaining O$_2$ saturation.

The addition of N$_2$O did not significantly decrease the amount of midazolam required to achieve sedation and did not confer any clinical advantages over the O$_2$-only group. Without an adequate scavenger system, the environmental hazards posed by N$_2$O outweigh any possible benefits obtained in using N$_2$O. However, those practitioners who choose to supplement intravenous sedation with N$_2$O/O$_2$, contrasted with those who administer O$_2$ only, do not appear to place their patients at increased risk of hypoxia or hypercapnia.

**REFERENCES**

10. Litchfield NB: Complications of intravenous diazepam

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**Table 4. Changes In Oxygen Saturation Over Time by Treatment Group**

<table>
<thead>
<tr>
<th>Variable (Change from)</th>
<th>Group A P Value</th>
<th>Group B P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>From Preoperative Baseline</td>
<td>From Start of Surgery Baseline</td>
</tr>
<tr>
<td>5</td>
<td>NS$^{a}$</td>
<td>NS</td>
</tr>
<tr>
<td>10</td>
<td>0.0098</td>
<td>NS</td>
</tr>
<tr>
<td>15</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>End</td>
<td>0.016</td>
<td>NS</td>
</tr>
<tr>
<td>Change</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

NS, not significant.

$^{a}$ Within-group changes were assessed for significance using a Wilcoxon Signed Rank test.

**Table 5. Oxygen Saturation Differences Between Treatment Groups**

<table>
<thead>
<tr>
<th>Oxygen (Between Groups)</th>
<th>P Value</th>
<th>Standard Deviation Mean</th>
<th>Standard Deviation Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 Min.</td>
<td>NS</td>
<td>98.55</td>
<td>1.57</td>
</tr>
<tr>
<td>10 Min.</td>
<td>NS</td>
<td>98.82</td>
<td>0.50</td>
</tr>
<tr>
<td>15 Min.</td>
<td>NS</td>
<td>98.77</td>
<td>0.53</td>
</tr>
<tr>
<td>End</td>
<td>NS</td>
<td>98.82</td>
<td>0.50</td>
</tr>
</tbody>
</table>

NS, not significant.


