Scientific Article

The effect of supplemental oxygen on apnea and oxygen saturation during pediatric conscious sedation

Gail K. Rohlfing, DDS Diane C. Dilley, DDS Warner J. Lucas, DDS, MD William F. Vann, Jr., DMD, MS, PhD

Abstract

Purpose: *This study compared the effect of supplemental oxygen (O_2) on pediatric patients' apnea status and oxyhemoglobin saturation during: 1) conscious sedation for dental procedures and 2) the recovery period following sedation.*

Methods: *Fourteen child patients (mean age 42 months) sedated with 50 mg/Kg chloral hydrate, 25 mg hydroxyzine pamoate, and 1.5 mg/Kg meperidine were treated for two separate appointments. The patients received supplemental O_2 via nasal cannulae at random at one of the two appointments. Following the operative period, all patients were monitored sitting upright for an additional 15 min.*

Results: *Intraoperative results showed that the risk of apnea was 39% (11/28), with apneic events distributed equally between O_2 and non-O_2 supplemented sedations. The overall risk of desaturation was 29% (8/28). Mean SpO_2 was always elevated with O_2 supplementation and the mean difference in O_2 versus non-O_2 was statistically significant. The risk of apnea in the postoperative period was 7% (1/14) for both the non-O_2 and O_2-supplemented patients. The risk of desaturation in the postoperative period was 11% (3/28) with one desaturation in a non-O_2 and two desaturations in O_2-supplemented patients.*

Conclusion: *We conclude that intraoperative O_2 supplementation prevents desaturations even in the presence of apnea during pediatric conscious sedation. (Pediatr Dent 20:1 8–16, 1998)*

Sedation of pediatric patients in the ambulatory care setting is important for several pediatric subspecialties, including pediatric cardiology, dentistry, emergency medicine, ophthalmology, radiology, and urology. Reliance on outpatient care has escalated in recent years and health care reform will most likely lead to further reliance upon sedation in this setting.

When sedating children in the dental office, conscious sedation is most often the desired outcome as opposed to deep sedation[1]; however, even during conscious sedation, patients are at risk for respiratory compromise that can lead to inadequate oxygenation or hypoxemia. Hypoxemia can result from: 1) a drug-induced respiratory depression from a decreased rate or depth of respiration or 2) airway obstruction due to loss of protective reflexes secondary to the sedative drug effect.[2–4]

Risk factors for hypoxemia in sedated children

Drug selection/dose of sedative

Several risk factors are associated with hypoxemia in consciously sedated children in the dental environment. Drug selection and drug dosages are fundamental risk considerations. Increasing the amount of drug has been implicated to increase respiratory compromise in children sedated for dental procedures.[5] This is true for narcotics,[4,6] probably because children are more sensitive to respiratory depressant effects of narcotics than are adults. Also, obstructive apnea is common when narcotics are used in conjunction with local anesthesia.[7]

Local anesthesia dosage

A side effect of all local anesthetics is dose-related, decreased ventilation. Even in acceptable dosages, local anesthetics can potentiate the effects of the sedative medicament.[7] The addition of a local anesthetic to a sedation regimen may increase the depth of sedation.[8]

Recently Verwest et al.[9] showed that the lidocaine dose was related to oxygen desaturation prevalence in children with increased tonsil size during restorative treatment with and without nitrous oxide/oxygen (N_2O/O_2) analgesia. They concluded that the amount of lidocaine is a risk factor for hypoxemia in the conscious sedation of children.

Tonsil size and upper airway obstruction

Clinicians and investigators agree that the most common cause of respiratory problems in children sedated for dental care is upper airway obstruction.[5] Soft tissue obstruction of the airway by the tongue is common, but increased tonsil size is also a risk factor. Decreased tone of the upper airway and the musculature of the tongue during sedation may be evident as in a sleeping child,[7] but supine patient position during sedation can magnify the effect of tonsil size. Verwest et al.[9] showed that patients with even mild tonsillar hypertrophy tended to desaturate more frequently than those without hypertrophy.

Use of N_2O/O_2 during conscious sedation

Oxygen supplementation via N_2O/O_2 analgesia has been theorized as one possible measure to increase the safety of sedation because the hyperoxygenated patient has an increased level of protection following an unanticipated drug-induced respiratory depression or airway obstruction.[2, 7] Because supplemental O_2 via N_2O/O_2 increases pulmonary O_2 reserve capacity, it has been postulated that desaturation of hemoglobin is delayed during periods of hypoventilation.

Some evidence supports this beneficial effect of N_2O/O_2 analgesia in adults. Everett and Allen[10] evaluated arterial O_2 concentrations of N_2O/O_2 analgesia in adults and found that the arterial O_2 concentration rose significantly to 350–500 mm Hg at concentrations of 10–40% N_2O. Cassidy et al.[11] showed that a continuous decline in transcutaneous oxygen levels ($TcPO_2$) resulted with increasing concentrations of N_2O/O_2 analgesia, although the $TcPO_2$ values remained higher than baseline throughout the study.

Available evidence does not support the concept that N_2O/O_2 analgesia carries this same beneficial effect for preventing airway obstruction in children. In a classic study by Moore et al.,[5] 27% of the pediatric patients aged from 2 to 5 experienced obstructed airways when sedated with 60 mg/Kg chloral hydrate and 40% N_2O/60% O_2. The same patients experienced no airway obstructions prior to administration of N_2O/O_2. There were also no airway obstructions with 20 mg/Kg and 40 mg/Kg chloral hydrate and N_2O/O_2.

Mathewson et al.[12] noted that N_2O/O_2 analgesia has a neuromuscular relaxation effect upon the tongue that can potentiate airway obstructions in children. It is plausible that, in children, the sedative properties of the N_2O in N_2O/O_2 analgesia may counterbalance any protective effects of supplemental O_2 via N_2O/O_2 during airway obstruction.

Effects of 100% O_2 on sedated children

When healthy patients are ventilating normally and supplemented with O_2, the SpO_2 is near 100%. The arterial oxygen tension (PaO_2) may increase from 90–100 mm Hg breathing room air to as high as 600 mm Hg depending on the actual inspired O_2 concentration. This phenomenon may provide protection for the patient on supplemental O_2. Due to the shape of the oxyhemoglobin dissociation curve, SpO_2 levels may not reflect decreasing O_2 availability in the apneic or hypoventilating patient until O_2 reserves in the patient's functional residual capacity are nearly depleted. This effect was found in adult patients receiving supplemental O_2 during conscious sedation for oral surgery procedures.[13] In this study, significantly fewer episodes of desaturation occurred during the intraoperative and postoperative periods for patients supplemented with O_2.

As for adults, the beneficial effects noted for O_2 supplementation may be applicable if O_2 alone is used for children. In studies by both Hasty et al.[4] and Croswell et al.,[14] 100% O_2 was used during conscious sedation for pediatric sedation and there was evidence that 100% supplemental O_2 may have added a beneficial effect against decreased SaO_2. For example, Croswell et al.[14] reported no true desaturations in 39 conscious sedation appointments using 1.5 mg/Kg of meperidine in children (24–48 mon), and speculated that lack of hypoxemic episodes was related to the fact that all patients were supplemented with 100% O_2.

Postsedation period

Intraoperative O_2 supplementation could mask the readiness for discharge of the pediatric patient following conscious sedation. Supplemental O_2 increases PaO_2, resulting in elevated SpO_2 levels throughout the intraoperative period. Upon discontinuation of O_2, the elevated PaO_2 may decrease below normal, resulting in a postoperative hypoxemia in a patient who may have had a residual drug-induced respiratory depression. Because the use of supplemental O_2 has not been investigated in any detail, this question needs to be examined.

Specific aims

This study has two specific aims:

1. To compare the effects of supplemental O_2 on patients' apnea and oxyhemoglobin saturation (SaO_2) status during conscious sedation for dental procedures
2. To compare the effects of supplemental O_2 on patients' apnea and oxyhemoglobin saturation

(SaO_2) status following completion of dental treatment and discontinuation of the supplemental O_2.

Methods

Sample

All children in this study were referred for sedation because of unmanageable behavior in the conventional dental environment, met requirements for American Society of Anesthesia Class I anesthesia risk, were 24–54 months of age at time of the first sedation appointment, and required two dental procedures, each lasting approximately 60 min from the time of injection of local anesthesia.

The sample consisted of 14 patients who were treated for two separate conscious sedation appointments. A double-blind, crossover study design allowed each patient to serve as his/her own control, with each patient receiving supplemental O_2 at random at one of the two appointments. The principal investigator was blinded as to the administration of supplemental O_2.

Sedation protocol and drug regimen

Patients were monitored by the principal investigator (PI) in the University of North Carolina at Chapel Hill (UNC–CH) Department of Pediatric Dentistry's Pediatric Sedation Clinic (PSC). The PSC's routine conscious sedation protocol was followed, which requires a presedation physical examination by the child's physician and informed consent for the dental treatment, physical restraint, and conscious sedation procedures. Additionally, parents or caregivers are required to make a commitment to adhere to written pre- and postoperative sedation instructions and written consent was obtained to participate in this study.

All children were sedated with only one regimen, the PSC's standard drug regimen for young children. This is an oral regimen of 50 mg/Kg chloral hydrate (Pharmaceutical Basics, Inc., Morton Grove, IL), 25 mg hydroxyzine pamoate (Vistaril®, Pfizer Laboratories, New York, NY), and 1.5 mg/Kg meperidine (Demerol®, Winthrop-Breon, New York, NY). The treatment procedures were carefully planned so that each appointment was standardized to approximately 60 min of operating time and the local anesthetic dose was approximately the same at both appointments for a given patient.

Prior to administration of the sedative medications, baseline SpO_2 and PR measurements were obtained using the Oxisensor II® and the Nellcor-1000® electronic monitors (Nellcor Inc., Hayward, CA). These readings were not revealed to the PI until the end of the procedure.

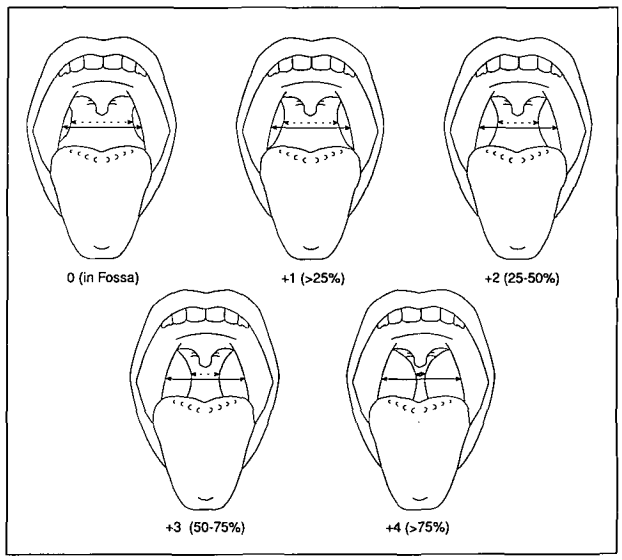


Fig 1. Tonsil classification system.

An evaluation of tonsil size was recorded using the Brodsky's 0–4 classification system.[15] This system classifies Type 0 tonsils as not present or atrophied, through a continuum to Type 4 tonsils, which are hypertrophied and in contact with each other. The PI and the dental operator reached a consensus for each patient's tonsil size at the beginning of each sedation appointment using a diagram of the 0–4 classification (Fig 1).

All patients were transferred to the dental operatory and placed in a Papoose Board® (Olympic Medical Group, Seattle, WA) 45 min after receiving sedation medications. Without the knowledge of the PI, a secondary investigator randomly assigned patients to receive supplemental O_2 at 100% or no supplemental O_2 at the first appointment. The large-tubing connector of the cannula system was attached to an oxygen outlet, a flow rate of 3.0 L/min was set for the supplemented patients and no flow was set for nonsupplemented patients. Because all patients had a nasal cannula (Salter Labs, Arvin, CA) in place for sampling of CO_2 during the procedure and the O_2 outlet was covered with a towel, the PI was blinded to O_2 supplementation.

Physiologic monitoring and data collection

All patients were monitored continuously and simultaneously by the PI using a capnograph, a pulse oximeter, and a precordial stethoscope. The pulse oximeter probe was placed on the great toe of the left

foot. Due to possible variations in pulse oximeter readings caused by skin temperature changes, the patient's temperature was monitored using a Mona-therm® temperature monitor (Mallinckrodt Medical Inc., Raleigh, NC). The temperature probe (Hi-Lo Temp Skin®, Mallinckrodt Medical Inc., Raleigh, NC) was placed on the dorsum of the left foot adjacent to the toes and the foot was covered by a towel to reduce ambient light.

It was easy to sample CO_2 continuously during the appointment via the nasal cannula. CO_2 was collected using the Nellcor-1000 combination pulse oximeter–capnograph. A precordial stethoscope was placed to the left of the sternum to monitor heart and breath sounds.

Physiologic data were collected continuously for each patient by the PI and recorded at 5-min intervals using a time-based anesthesia record for the entire operative and postoperative periods. A hard copy printout was generated each 30 min for each appointment using a miniprinter connected to the Nellcor-1000 unit.

The PI alerted the operator to respiratory compromise when the capnograph revealed apnea for longer than 60 s or the pulse oximeter read less than 90%. Once alerted, the operator repositioned the head to open the airway and assess ventilation. To distinguish true apneic episodes or desaturations and rule out false positive episodes, the PI recorded specific notes about the events (injection, rubber dam placement, crown placement, etc.) occurring during any episode of desaturation or apnea. True apnea was defined as capnograph reading of zero for respiratory rate and $EtCO_2$ with no visual signs of breathing and no breath sounds audible via the precordial stethoscope. True desaturation was defined as a drop in SpO_2 of 5.0 points from baseline in a child who was immobile and quiet.

Following the 60-min dental procedure, another investigator discontinued the supplemental O_2, or O_2 discontinuation was simulated for the nonsupplemented appointments. Following the intraoperative period, monitoring of SpO_2 and apnea continued postoperatively for 15 min with patients sitting upright in the dental chair. Following this 15-min period, if the SpO_2 was 96% or greater, the patient was assessed for discharge using the standard discharge protocol for the UNC–CH PSC.

Data analysis

The first aim of this study was to compare the effects of supplemental O_2 on patients' apnea and SpO_2 status during conscious sedation for dental procedures. This aim was addressed by comparing the number of true apneic episodes and true desaturations for appointments when patients received 100% O_2 versus appointments not supplemented. For analysis purposes, each patient served as his/her own control. The crossover research design was appropriate because the goal of the study was to examine the physiological parameters of the drugs and their effects on the patients, not the behavioral effects of the sedatives.

McNemar's test for paired data was used to determine if the event of apnea or desaturation was associated with O_2 supplementation. The data were analyzed further using Wilcoxon's signed rank test to compare the saturation mean differences within each patient. This nonparametric method was used because the distributions of these derived differences although relatively symmetric, were not distributed normally. A Fisher's exact test for two-by-two tables was used to evaluate whether the patients' tonsil size was associated with episodes of apnea or desaturation. An alpha of 0.05 was used as the level of significance for all statistical tests.

Our second aim was to determine if there were any differences during the postoperative period between those children supplemented with O_2 during the intraoperative period versus those not supplemented during the intraoperative period. To answer this question we compared the effects of supplemental O_2 on patients' apnea and SpO_2 status following the completion of dental treatment and discontinuation of the supplemental O_2, using each patient as his/her own control. We posed this question to examine for the presence of a rebound effect (negative) or protective effect (positive) from a patient's having received O_2 supplementation intraoperatively.

Results

Fourteen patients (five males, nine females, mean age 42 months, range 35–50 months) were enrolled in the study. The mean weight of the children was 15.2 Kg (range 12.7–18.6 Kg). The randomization of O_2 versus non-O_2 supplementation resulted in eight patients receiving supplemental O_2 at the first appointment and six patients not receiving O_2 at the first appointment. Wilcoxon's rank sum test was used to assess whether the sequence of treatment affected any variables of interest. There was no significant association between the order of treatment and the variables of pulse, respiratory rate, $P_{Et}CO_2$ or SpO_2. All *P* values were greater than 0.45, indicating no sequence effect.

Table 1. Apnea risk and events

	Overall Risk N = *28*	*With Oxygen* N = *14*	*Without Oxygen* N = *14*
Apnea *N* = 11	39% (11/28)	36% (5/14)*	43% (6/14)*
Total Apneic Events *N* = 22 (range 10–180 s)		45% (10/22)	55% (12/22)

*McNemar's test *P* value = 1.0

Because lidocaine has been suggested to affect SpO_2,[9] the same dose of 2% lidocaine with 1:100,000 epinephrine was used at both appointments for a given child with an average dose of 1.5 mg/Kg (range 1.0–2.2 mg/Kg). The age of the patients with episodes of apnea and desaturation ranged from 35 to 49 months.

Intraoperative data

Pulse, respiratory rate, and $EtCO_2$

The changes in pulse rate, respiratory rate, and $P_{Et}CO_2$ in the non-O_2 versus O_2-supplemented appointments were assessed for each individual because each patient served as his/her own control. The mean pulse in the non-supplemented group was 122 versus 118 for the supplemented group. The average change in pulse for an individual was 3.7 and this difference was not significant (Wilcoxon's signed rank test, $P = 0.90$). The mean respiratory rate for the nonsupplemented group was 20.4 versus 18.0 for the supplemented group. The average change in respiratory rate for an individual was 2.4 and this difference was significant (Wilcoxon' signed rank test, $P = 0.02$). The mean $P_{Et}CO_2$ for the nonsupplemented group was 35.4 versus 28.4 for the supplemented group. The average change in $P_{Et}CO_2$ for an individual was 7.0. This difference was significant (Wilcoxon's signed rank test, $P = 0.03$).

Apnea

We report only those apneic events that meet our definition of true apnea. Six children (two females, four males, age range 35–49 months) accounted for all apneic events, and five experienced apnea at both their nonsupplemented and supplemented visits. The sixth patient experienced apnea at only the nonsupplemented visit.

During the 28 appointments, apnea was recorded in 11 appointments, thus the overall risk of apnea for a given sedation appointment was 39% (11/28) for the study. As illustrated in Table 1, the risk of apnea without O_2 supplementation was 43% (6/14), while the risk of apnea with supplementation was 36% (5/14). This difference in proportions was not significant ($P = 1.0$) using the McNemar's test for matched paired data. The risk of apnea was distributed evenly between patients receiving O_2 at the first or second appointment.

Over the course of the 28 appointments, a total of 22 separate apneic events were recorded in the intraoperative period, with apnea ranging from 10 to 180 s. For all apneic episodes lasting longer than 60 s, the operator was instructed to reposition the head to open the airway; if normal ventilation was not resumed, the patient was verbally stimulated. Twelve apneic events occurred in patients not supplemented with O_2, while 10 occurred in supplemented patients (Table 1).

Desaturation

Foot temperature values remained constant for all patients with no more than a three-degree fluctuation in temperature (range 89–97°F). Accordingly we could not attribute desaturations to temperature.

We report only those events meeting our strict definition for true desaturation. Seven children (five females, two males, age range 35–49 months) accounted for all desaturation events, six of which occurred at only the nonsupplemented visit, while one patient experienced a desaturation event at both appointments.

For the 28 appointments, desaturations were recorded in eight visits during the intraoperative period; thus, the intraoperative risk for desaturation for the study was 29% (8/28). As illustrated in Table 2, the risk of desaturation without O_2 supplementation was 50% (7/14), while the risk of desaturation with supplementation was 7% (1/14). This difference in proportions was significant (McNemar's

Table 2. Desaturation Risk and Events

	Overall Risk N = *28*	*With Oxygen* N = *14*	*Without Oxygen* N = *14*
Desaturations $N = 8$	29% (8/28)	7% (1/14)*	50% (7/14)*
Total Desaturation Events $N = 13$ (range 78–93 s)		8% (1/13)	92% (12/13)

*McNemar's Test *P* value = 0.03

test, $P = 0.03$). The risk of desaturation was distributed evenly between patients receiving O_2 at the first or second appointment.

There was a total of 13 separate desaturation events in seven patients, ranging from 78 to 93%. Ninety-two percent (12/13) of the desaturations occurred in the nonsupplemented patients, while 7% (1/13) occurred in a supplemented patient (Table 2). The one true desaturation during O_2 supplementation was a drop of six points from a baseline of 97% .

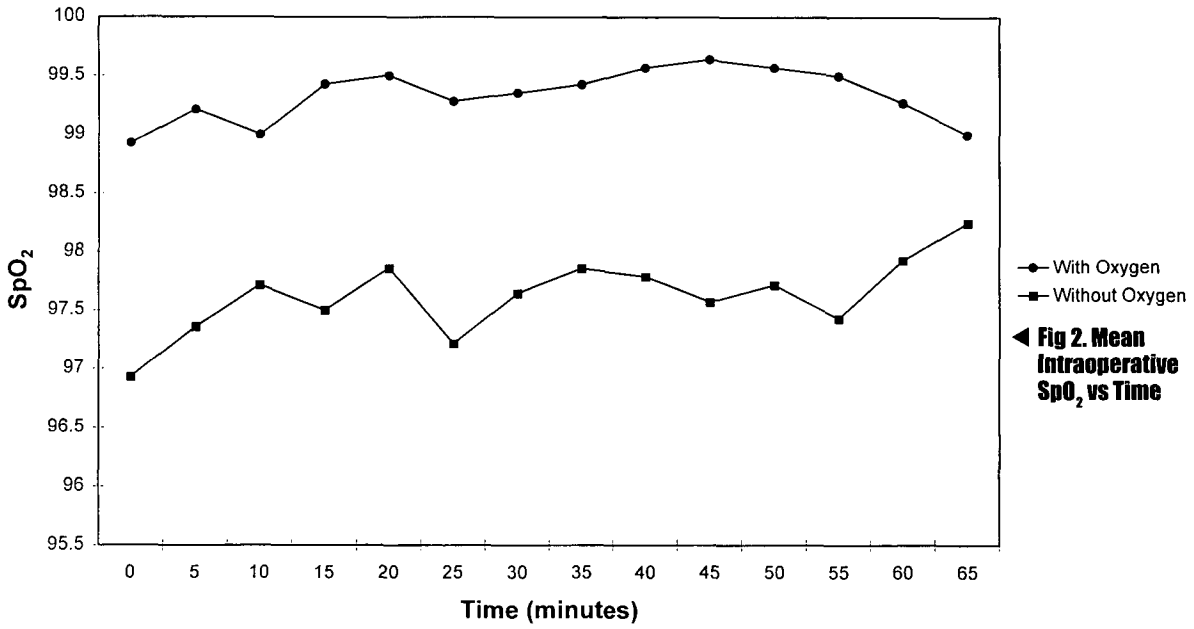
The overall effect of O_2 supplementation on SpO_2 is illustrated in Fig 2. Mean SpO_2 was consistently elevated with O_2 supplementation and the mean difference in O_2 versus non-O_2 was statistically significant at all time points when the means were compared using the Wilcoxon' signed rank test (*P* value range from 0.0001 to 0.006).

Desaturation following apnea

Desaturation events followed apneic episodes in four of the 11 appointments in which apnea occurred (Table 3). Without O_2 supplementation, desaturation followed apnea 67% (4/6) of the time. There were no desaturations associated with the five apneic events that occurred during O_2 supplementation.

Tonsil size

Five of the six patients who experienced apnea during the intraoperative period had tonsil size of 2 or greater, defined as tonsils seen readily in the airway with 25–50% of the airway obstructed.[15] All patients with desaturations had tonsil size of 2 or greater. No significant association was found between tonsil size and a true apnea event (Fisher's exact test, $P = 0.65$) or between tonsil size and a true desaturation event (Fisher's exact test, $P = 0.14$).



◀ Fig 2. Mean Intraoperative SpO_2 vs Time

TABLE 3. DESATURATION FOLLOWING APNEA

	Overall Risk N = *11*	*Apnea with Oxygen* N = *5*	*Apnea without Oxygen* N = *6*
Desaturations following Apnea *N* = 4	36% (4/11)	0% (0/5)	67% (4/6)

Postoperative data

Apnea

During the 28 postoperative periods, true apnea was recorded in two appointments, thus the risk of apnea in the postoperative period was 7% (2/28). For those children who were not supplemented intraoperatively, the risk of apnea in the postoperative period was 7% (1/14), as was the risk for those children who were supplemented intraoperatively. This difference in proportions was not significant (McNemar's test, $P = 1.0$). Both apneic events occurred immediately following sitting the children upright in the dental chair, at 1 and 3 min respectively, in the postoperative period. In both instances, we observed poor head control in which the patient's chin fell to the chest, occluding the airway. Desaturations were associated with both of these apneic events.

Desaturation

For the 28 appointments, true desaturations were recorded in the postoperative period at three visits; thus, the risk for desaturation in the postoperative period for this study was 11% (3/28). The risk of desaturation without O_2 supplementation was 14% (2/14), while the risk of desaturation with supplementation was 7% (1/14). This difference in proportions was not significant (McNemar's test, $P = 1.0$).

Over the course of the 28 appointments, three separate desaturation events were recorded during the postoperative period, ranging from 88–91%. Two desaturations occurred in patients who were not supplemented intraoperatively, while one occurred in a supplemented patient. After 15 min of monitoring in the postoperative period, all patients were alert, well-saturated, and ready for discharge.

Discussion

Pulse, respiratory rate, and $P_{Et}CO_2$

We did not anticipate differences in pulse, respiratory rate, and $P_{Et}CO_2$ in nonsupplemented versus supplemented patients. We do not know why the difference in respiratory rate was evident, but believe that the difference of 2.4 breaths per min and the 7.0 difference in $P_{Et}CO_2$ is probably due to the lack of air flow and the 50cc/min sampling rate through the nasal cannula in the nonsupplemented patients. We are not sure if these differences are clinically significant.

Apnea and desaturation associated with oxygen Supplementation

The results revealed that some pediatric patients will experience episodes of apnea whether they receive O_2 supplementation or not. This finding supports other studies reporting hypoxemia as a risk of conscious sedation in children. This should send a strong message to dental and medical clinicians who rely upon conscious sedation for pain and anxiety control. Except for one child, the patients who received O_2 supplementation did not desaturate, even during episodes of apnea. The logical explanation for this finding is that the O_2 supplementation elevated SpO_2 values during the dental procedures. The patients who did not receive O_2 supplementation were at greater risk for desaturations related to specific apneic events. Although it is not possible to prevent apneic episodes, based on these findings it appears that the number of desaturations is limited when O_2 supplementation is used adjunctively during the sedation.

We noted one episode of desaturation in a supplemented patient, a six-point drop to 91% from a baseline of 97%. This patient, not surprisingly, also experienced desaturation when no supplementation occurred. In a study by Hasty et al.,[4] two desaturations less than 90% SpO_2 occurred with 100% O_2 supplementation, but these could have been attributed to poor patient behavior. In data reported by Croswell et al.,[14] using the same sedation drug regimen as we used in this study, there were no episodes of desaturation when 100% O_2 supplementation was used. Taken together, these findings suggest that when children under conscious sedation are

supplemented with 100% O_2 there is little chance of desaturation if airway compromise is detected quickly and corrected.

Tonsil size

Tonsil size was difficult to associate with apnea or desaturation because our sample was confounded by a high percentage of children with large tonsils. Another confounding factor in this study was that some children had different size tonsils at the two appointments, even though the appointments were scheduled within a few weeks of each other. We can draw no conclusion about the association of tonsil size and sedation risk.

Oxygen supplementation

The 1992 *American Academy of Pediatrics Sedation Guidelines*[16] and the 1993 *American Academy of Pediatric Dentistry Sedation Guidelines*[17] are considered by many to set the standards of care for sedating children. Although both sets of guidelines are silent on the issue of O_2 supplementation, it is our opinion that all healthy sedated children should be supplemented with O_2. Hypoxemia is the greatest risk factor in pediatric conscious sedation and our results demonstrate unequivocally that O_2 supplementation can reduce hypoxemia as measured by SpO_2. Our study does not address the use of N_2O/O_2 as a vehicle to elevate SpO_2 to the same degree as 100% O_2 supplementation provides. This is a question that needs to be addressed in future studies.

Oxygen supplementation and its effects in the postoperative period

The postoperative period was of interest to us because we were aware of the potential for a rebound effect that might occur in a child whose PaO_2 fell below 100 after the discontinuation of O_2. There was no evidence of such a rebound effect in the patients who received O_2 supplementation.

Potential concerns with oxygen supplementation

One potential concern regarding the use of supplemental O_2 is the possibility of masking a drug-induced respiratory depression and a consequent decreased minute ventilation resulting in hypercarbia. While the risk of hypercarbia is less than that of hypoxemia, there still exists the need to verify adequate ventilatory function even in patients receiving supplemental O_2 who demonstrate acceptable SpO_2 levels.

Clinically detectable signs of significant hypercarbia may include cardiac dysrhythmias. Changes in pH secondary to respiratory acidosis and subsequent electrolyte abnormalities affect cardiac automaticity and irritability. While it is not standard practice to monitor patients receiving conscious sedation with an electrocardiogram (EKG), changes in cardiac rhythm can produce alterations in pulse amplitude and regularity that may be detected by the plethysmograph of the pulse oximeter. Episodes of suspected dysrhythmias which are not self-limiting or not corrected after restoration of adequate ventilation must be further investigated by EKG analysis.

Conclusions

Under the conditions of this investigation we conclude that

1. Oxygen supplementation had no effect on risk of apnea during the intraoperative period or postoperative periods
2. Intraoperative oxygen supplementation prevented desaturations from occurring during the intraoperative period
3. Oxygen supplementation prevented desaturations associated with apnea during the intraoperative period
4. The data suggest that oxygen supplementation during the intraoperative period had no effect on risk of apnea or desaturation during the 15-min recovery period.

This work was supported by a grant from USPHS Bureau of Maternal and Child Health (#MCJ-379494), which supports a Pediatric Dental Training Center at the University of North Carolina at Chapel Hill, NC.

Special thanks is given to Nellcor, Inc. for consignment of the Thinkjet™ miniprinter used in this study.

At time of this study Dr. Rohlfing was a Fellow, Department of Pediatric Dentistry, School of Dentistry; Dr. Dilley is Associate Professor, Department of Pediatric Dentistry, School of Dentistry; Dr. Lucas is Associate Professor, Department of Anesthesiology, School of Medicine; Dr. Vann is Professor and Graduate Program Director, Department of Pediatric Dentistry, School of Dentistry, all University of North Carolina at Chapel Hill, NC.

References

1. Wilson S: Review of monitors and monitoring during sedation with emphasis on clinical applications. Pediatr Dent 17:413–18, 1995.
2. Anderson JA, Vann WF Jr: Respiratory monitoring during pediatric sedation: pulse oximetry and capnography. Pediatr Dent 10:94–101, 1988.
3. Becker DE: The respiratory effects of drugs used for conscious sedation and general anesthesia. J Amer Dent Assoc 119:153–56, 1989.
4. Hasty MF, Vann WF Jr, Dilley DC, Anderson JA: Conscious sedation of pediatric dental patients: an investiga-

tion of chloral hydrate, hydroxyzine pamoate, and meperidine vs. chloral hydrate and hydroxyzine pamoate. Pediatr Dent 13:10–19, 1991.
5. Moore PA, Mickey EA, Hargreaves JA, Needleman HL: Sedation in pediatric dentistry: a practical assessment procedure. J Amer Dent Assoc 109:564–69, 1984.
6. Moore PA: Local anesthesia and narcotic drug interaction in pediatric dentistry. Anesth Prog 35:17, 1988.
7. Allen GD: Diagnosis and treatment of respiratory problems in sedation and anesthesia for dentistry. Anesth Prog 39:150–56, 1992.
8. Goodson JM, Moore PA: Life-threatening reactions after pedodontic sedation: an assessment of narcotic, local anesthetic, and antiemetic drug interaction. J Amer Dent Assoc 107:239–45, 1983.
9. Verwest TM, Primosch RE, Courts FJ: Variables influencing hemoglobin oxygen desaturation in children during routine restorative dentistry. Pediatr Dent 15:25–29, 1993.
10. Everett GB, Allen GD: Simultaneous evaluation of cardiorespiratory and analgesic effects of nitrous oxide–oxygen inhalation analgesia. J Amer Dent Assoc 83:129–33, 1971.
11. Cassidy DJ, Nazif MM, Zullo T, Ready MA: Transcutaneous oxygen monitoring of patients undergoing nitrous oxide–oxygen sedation. Pediatr Dent 8:29–31, 1986.
12. Mathewson RJ, Primosch RE, Robertson JD: Nitrous oxide inhalation for children. In Fundamentals of Pediatric Dentistry, 2nd Ed. Chicago: Quintessence Publishing Co., 1987, pp 155–65.
13. Papageorge MB, Hunter MJ, Norris LH, Rosenburg MB: Supplemental oxygen after outpatient oral and maxillofacial surgery. Anesth Prog 39:24–27, 1993.
14. Croswell RJ, Dilley DC, Lucas WJ, Vann WF Jr: A comparison of conventional versus electronic monitoring of sedated pediatric dental patients. Pediatr Dent 17:332–39, 1995.
15. Brodsky L: Modern assessment of tonsils and adenoids. Pediatr Clin North Am 36:1551–69, 1989.
16. American Academy of Pediatrics Committee on Drugs: Guidelines for monitoring and management of pediatric patients during and after sedation for diagnostic and therapeutic procedures. Pediatrics 89:1110–15, 1992.
17. Guidelines for the elective use of pharmacologic conscious sedation and deep sedation in pediatric dental patients. Pediatr Dent 15:297–301, 1993.

PEDIATRIC DENTISTRY
The Journal of the American Academy of Pediatric Dentistry

Publication Member of the American Association of Dental Editors