Abstract

Pulse oximetry to monitor oxygen saturation during pediatric dental sedations has revolutionized the early detection of hypoxemia. Previous sedation studies confirmed the occurrence of hemoglobin oxygen desaturation when sedative agents are administered in conjunction with nitrous oxide. The purpose of this study was to monitor the hemoglobin oxygen saturation levels of nonmedicated pediatric patients during routine restorative procedures to study the effect of nitrous oxide, chronologic age, tonsil size, and lidocaine dose. Two identical pulse oximeters and probes were attached to a patient's right and left feet to validate the occurrence of oxygen desaturations. The results revealed that 20.3% of the patients demonstrated at least one occurrence of oxygen desaturation during the routine dental procedure. Administration of nitrous oxide had no effect on the prevalence of oxygen desaturation. Although chronologic age, tonsil size, and lidocaine dose did not exert a statistically significant influence when analyzed independently, when combined they were related statistically ($P < 0.0001$) to the occurrence of oxygen desaturation as defined by the parameters of the study. These data suggest that some of the oxygen desaturations recorded during routine restorative appointments may result from probe error and a combination of patient-related variables. (Pediatr Dent 15:25–29, 1993)

Variables influencing hemoglobin oxygen desaturation in children during routine restorative dentistry

Tim M. Verwest, DMD Robert E. Primosch, DDS, MS, MEd
Frank J. Courts, DDS, PhD

Introduction

Health care has undergone a vast revolution in vital sign-monitoring technology over the last decade with the advent and continual improvement of pulse oximetry.[1,2] One of the major functions of pulse oximetry is to monitor the patient's per cent oxygen saturation of hemoglobin (oxyhemoglobin) as an estimate of arterial oxygen saturation (SaO_2). The SaO_2 levels are detected by a noninvasive photodetector probe placed over a pulsating arterial bed. Detectable plethysmographic pulsations are required, making fingers, toes, and ear lobes ideal sites for probe placement. The pulse oximeter provides continuous monitoring and a digital display of the SaO_2 levels. Large decreases in arterial oxygen tension (PaO_2) from 100 to 60 mm Hg can occur for a SaO_2 change from 96 to 90%. Small SaO_2 changes are more critical in children due to their decreased oxygen reserve, characteristic of their smaller body size, higher basal oxygen consumption, and lower functional residual lung capacity. The reduced oxygen reserve in children causes oxygen desaturations to be more rapid and profound than in adults.[3,4]

Numerous reports in the pediatric dental literature have demonstrated that, when monitored by pulse oximetry, sedated patients experience periods of oxygen desaturation or hypoxemia.[5–10] A recent conscious sedation study found that children receiving only 40% nitrous oxide in oxygen as a control, experienced no episodes of hypoxemia.[11] Although the actual effect of nitrous oxide-oxygen (N_2O/O_2) upon SaO_2 levels remains to be clarified, supplemental oxygen administration can elevate SaO_2 above resting (room air) baseline values.[12–14] Supplemental oxygen administered via N_2O/O_2 during conscious sedation in adults reduced the duration and number of desaturation episodes.[15] In children, it was speculated that supplemental oxygen reduced the occurrence of desaturations monitored during conscious sedation.[7,10]

The influence of lidocaine administration alone upon SaO_2 is unclear. During oral surgery procedures in nonsedated adults, the prevalence of oxygen desaturation was 43% when lidocaine was used as the sole agent.[15] Eighty-seven per cent of these reported desaturations may have been associated with patient movement of the photodetector probe. Erroneous SaO_2 levels associated with probe movement have been reported to be as high as 75-89% in young sedated children.[8,16] The occurrence of oxygen desaturations in sedated pediatric dental patients also has been related to chronologic age[17] and procedures involving rubber dam isolation[9] and the mandibular arch.[18]

In a recent study of 14 5- to 7-year-old nonsedated children receiving amalgam restorations on mandibular molars, no statistical correlation emerged between the routine restorative procedure and changes in the SaO2 levels monitored by a pulse oximeter.[19] The purpose of the present investigation was to study the effect of lidocaine and nitrous oxide/oxygen administration, dental arch, patient's chronologic age, and tonsil size on the SaO_2 levels of nonsedated pediatric dental patients during routine

restorative appointments.

Materials and Methods

Participants were selected randomly from patients being treated at the pediatric dental clinic of the University of Florida College of Dentistry. Patients were excluded from the study if they presented with moderate to severe tonsillar enlargement or uncooperative behavior that interfered with treatment. The procedures, risks, and benefits were explained fully to the patients and their parents and their informed consent was obtained before the investigation as approved by the University's Institutional Review Board. Sixty-four healthy (ASA I) patients (mean age = 7.2 ± 1.9 yrs., range = 3.5–10.8 years) were assigned to one of two groups. Group I comprised of 32 patients (mean age = 6.6 ± 1.9 years) selected to receive 40% $N2O/O_2$ and local anesthesia. Patients were administered N_2O/O_2 based upon standard selection criteria of previously established guidelines.[20] Group II contained 32 patients (mean age = 7.7 ± 1.8 years) receiving only local anesthesia. Analysis of group assignment by paired *t*-test found that chronologic age was not statistically different between the two groups.

All patients underwent routine dental restorative procedures with rubber dam isolation and lidocaine local anesthesia (2% Xylocaine®, Astra Pharmaceutical Products Inc., Westboro, MA). The dental procedures were performed by multiple operators, but the recorder remained constant and independent throughout the study. During the entire test period, all patients were placed in a supine position. The amount of lidocaine (mg/kg) administered and the treated dental arch were recorded. Tonsil size was recorded as either normal or minimally hypertrophic.

Two new and identical pulse oximeter units (CSI #503, Criticare System, Milwaukee, WI) and probes were used simultaneously with each subject. The probes were clipped to the great toes, one on each foot, and their positions adjusted until strong and continuous readings were obtained. Adhesive tape was placed across the bridge of each foot to help hold the probe's power cord in place, thus lessening the likelihood of dislodging the probe with movement.

The experiment proceeded over a 40-min period, divided into eight intervals of 5 min each. Group I patients received 40% N_2O at 4–5 L/min from a portable N_2O machine (MXR, Porter Instrument Co., Hatfield, PA) via a rapid induction technique.[20] At the end of the first 5-min interval, the baseline SaO2 was determined and set into the pulse oximeter using one of its programmed features. The baseline SaO_2 was obtained after N_2O/O_2 administration to neutralize any effect that the high oxygen supplementation had upon resting "room air" SaO_2 levels. Group II patients remained quiet and undisturbed in the dental chair for the first 5-min interval and then SaO2 baseline values were similarly determined as with Group I patients. Following determination of the baseline SaO_2, the patients were left undisturbed until completion of the second 5-min interval. The third 5-min interval was used to administer local anesthesia. The fourth interval was used to assess effectiveness of the local anesthesia and to place the rubber dam. An additional 5-min interval was allowed at this point to re-inject local anesthetic if the first injection was unsuccessful in obtaining profound anesthesia. During the remaining 20 min, the patients were monitored for oxygen desaturations occurring during the restorative procedures.

From the moment of baseline SaO2 determination (beginning of the second 5-min interval), both groups were observed for the occurrence of oxygen desaturation displayed on the units by either probe. The SaO2 was recorded manually on a data sheet under the corresponding time interval of its occurrence. The patient's behavior (resting, sleeping, crying, movement, etc.) also was recorded for each observed desaturation. Only oxygen desaturations judged to be clinically significant were selected for data analysis. Clinically significant desaturations were divided into major and minor categories. A major oxygen desaturation was defined as ≥ 5% decrease from baseline as denoted by activation of the unit's alarm system. A minor oxygen desaturation was defined as a > 2 but < 5% desaturation. Only bilateral (both probes) major desaturations or the simultaneous occurrence of a minor and major desaturation (one with each probe) were deemed clinically significant and suitable for statistical analysis. Neither bilateral minor desaturations nor unilateral desaturations were tabulated for analysis.

Results

The present study revealed several interesting findings in nonmedicated children's SaO2 levels during routine restorative dental appointments (Table). Overall, 13 of the 64 patients (20.3%) experienced at least one episode of major oxygen desaturation during the course of the dental appointment. The 13 patients accounted for the occurrence of 22 individual desaturation events for a frequency rate of 1.7 desaturations/subject. Of the major desaturations recorded, the majority (19/22, 86.4%) were major bilateral occurrences. Patient movement was associated with 45.5% (10/22) of the oxygen desaturations recorded. Two-thirds of the motion-associated desaturations occurred during the 20-min period of restorative procedures.

No statistical difference was shown for the prevalence of oxygen desaturation between the $N2O/O_2$ group and the room air group. The apparent lack of effect of N_2O/O_2 administration upon the occurrence of oxygen desaturation during routine restorative appointments permitted the pooling of data from both groups for the remaining statistical comparisons. Of the 64 patients examined, the mean prevalence of oxygen desaturation for the mandibular arch and maxillary arch was likewise not statistically different.

The comparison of tonsil size, lidocaine dose, and chronologic age to the prevalence of oxygen desaturation demonstrated significant interrelationships. Although not

Table. Prevalence of oxygen desaturation by group

	Group	Prevalence		P-value*	P-value†
N_2O/O_2	Yes	7/32	(21.9%)		
	No	6/32	(18.8)	0.76	
Dental arch	Mandibular	7/34	(20.6)		
	Maxillary	6/30	(20.0)	0.95	
Chronologic age	< 7 years	9/32	(28.1)		
	≥ 7 years	4/32	(12.5)	0.18	
Tonsil size	No hypertrophy	5/36	(13.9)		0.0001
	Mild hypertrophy	8/28	(28.6)	0.15	0.0001
Lidocaine dose	< 1.5 mg/kg	4/20	(13.3)		0.014
	≥ 1.5 mg/kg	9/34	(26.5)	0.19	
Total		13/64	(20.3%)		

* Chi-square analysis; † Two factor repeated measure ANOVA.

statistically significant when analyzed independently (Table), tonsil size and lidocaine dose were statistically relevant ($P = 0.014$) when paired and analyzed by the 2-factor repeated measures ANOVA. Increasing tonsil size and lidocaine dose was related to increasing prevalence of oxygen desaturations. Chronologic age, however, demonstrated an inverse relationship to the prevalence of desaturation for patients younger than 9 years old (Figure). When chronologic age was combined with tonsil size and lidocaine dose, 2-factor repeated measures ANOVA demonstrated significant relevance at $P < 0.0001$ (Table).

Discussion

In the dental literature, mild hypoxemia was judged to occur when SaO_2 levels decreased below 95%.[5] Therefore, a clinically significant desaturation was defined as any sustained SaO_2 level below 95%. This approach did not take into account the magnitude of the change, so that a patient with a baseline SaO_2 of 97% only has to drop 2% compared to another patient with a baseline SaO_2 of 100% who has to drop 5% to be classified as a desaturation. Average baseline room air SaO2 levels have been reported to be 99.1%.[9, 14] Recent studies by Clayton et al.[21, 22] found the pulse oximeter selected for use in this study was among the most reliable units available, providing readings within 4% of reference co-oximeter readings 95% of the time. One of the unique features of the CSI 503 was its programmed functions which automatically set each subject's baseline and signaled any drop in $SaO_2 \geq 5\%$. This adjustment provided a relative comparison among the patients. Recent studies have supported the definition of a clinically significant desaturation (≥ 5% decrease from baseline) as used in this study.[15, 23]

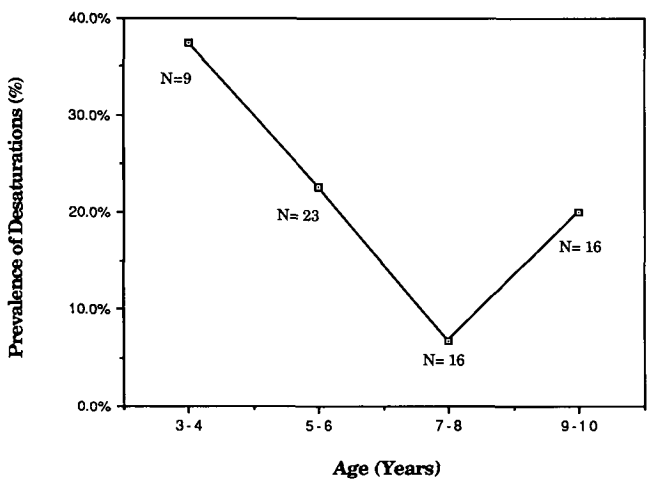

Fig. Effects of chronologic age (years) on the prevalence of oxygen desaturation.

Although pulse oximetry has revolutionized the monitoring of patients' vital signs, it is still susceptible to erroneous readings caused by patient movement[16] or ambient light.[24] Since the potential for erroneous SaO_2 levels associated with probe movement can be as high as 75–89%,[8, 15, 16] one must discriminate false readings (artifact) from true ones. This study was the first investigation to use two identical pulse oximeter digit probes attached to different feet of the same subject simultaneously to help validate the occurrence of oxygen desaturations. The use of two probes helped to identify and reduce the potential of a false positive recording of oxygen desaturation elicited from the use of only one probe. In clinical research, this methodology is a useful tool. In clinical practice, the occurrence of a false positive desaturation alerts the clinician to a potential problem that might require airway management (chin life maneuver). The response to a false positive desaturation would never compromise a patient's safety.

The results of this study demonstrated that oxygen desaturations occurred in non-medicated children during routine restorative dental appointments. Poiset et al.[19] found no significant change in SaO_2 levels in a small sample of nonmedicated dental patients during routine restorative appointments. The present study, however, found 20.3% of the pediatric dental patients experienced at least one episode of oxygen desaturaton. This is in agreement with others,[4, 15] who found that nonmedicated adults also experienced oxygen desaturation during surgical procedures. One conclusion that might be drawn from the re-

sults of this investigation is that perhaps some of the oxygen desaturations reported during pediatric dental sedations may have occurred normally in the absence of sedative medications. Wilson[16] found that some of his placebo patients experienced oxygen desaturations as did his sedated patients.

The results of this study found no significant difference, clinically or statistically, between the occurrence of desaturations and use of nitrous oxide-oxygen inhalation. This finding was in agreement with Whitehead et al.[11] who reported that nitrous oxide and oxygen alone did not cause any hypoxic episodes. It has been speculated that the high oxygen content delivered with N_2O/O_2 inhalation elevated the SaO_2 levels and thus potentially reduced the number of oxygen desaturations observed in sedation studies in adults[15] and children.[7, 10] Administration of supplemental oxygen might neutralize desaturation tendency resulting from airway obstruction due to the neuromuscular relaxation effects of nitrous oxide upon the tongue.[25] Since these data did not differentiate the effects of N_2O and supplemental O_2, a future area of investigation should be to repeat the present study with a third group supplemented with 100% oxygen only.

The patients in this study also were evaluated for any oxygen desaturation differences occurring between the maxillary and mandibular arch. No statistical significance was shown, in contrast to sedated patents, when manipulation of the mandibular dental arch created a greater prevalence of desaturations. It was suggested that manipulation of the mandibular arch was more likely to create airway obstruction, a cause for oxygen desaturation.[18]

Tonsil size also was considered a possible source of airway obstruction that could result in decreased SaO_2 levels. Children were only selected for this study if their tonsils were at most only mildly hypertrophic. Subjects with mild tonsillar hypertrophy tended to desaturate more than those without hypertrophy, but the difference was statistically insignificant. Younger children also tended to desaturate more than older children; this may be related to the amount of lidocaine per kg of body weight. Although the three factors of tonsil size, chronologic age, and lidocaine dose did not exert any influence on the occurrence of desaturations when analyzed independently, when combined in pairs there appeared to be statistical relevance.

Conclusion

This study found that oxygen desaturations commonly occurred when pediatric patients underwent routine restorative dental appointments. Some combined factors potentially associated with oxygen desaturations in children were tonsil size, chronologic age, and lidocaine dose. Thus, the dentist needs to be more aware of the possibility of oxygen desaturations occurring in young children with large tonsils who are given significant amounts of lidocaine. Two factors found not to be associated with oxygen desaturation were use of nitrous oxide and oxygen and dental arch manipulation. Pulse oximetry technology without concurrent blood gas determination is not infallible, as motion artifacts are reported widely in the dental literature to produce false positive readings. Pulse oximetry is only a tool to assist in the determination of an oxygen desaturation event. Appropriate intervention must be weighted by the operator's clinical judgment in the context of simultaneously occurring events related to patient behavior and activity.

Special thanks is given to Criticare Systems Inc. for donating the pulse oximeters used in this study.

Dr. Verwest is in private practice in Ft. Myers, FL. Dr. Primosch is professor and Dr. Courts is associate professor, Department of Pediatric Dentistry, University of Florida College of Dentistry, Gainesville, FL.

1. Wukitsch MW, Petterson MT, Tobler DR, Pologe JA: Pulse oximetry: analysis of theory, technology, and practice. J Clin Monit 4:290–301, 1988.
2. Alexander CM, Teller LE, Gross JB: Principles of pulse oximetry: theoretical and practical considerations. Anesth Analg 68:368–76, 1989.
3. Anderson J, Vann W: Respiratory monitoring during pediatric sedation: pulse oximetry and capnography. Pediatr Dent 10:94–101, 1988.
4. Hovagim AR, Vitkun S, Manecke G, Reiner R: Arterial oxygen desaturation in adult dental patients receiving conscious sedation. J Oral Maxillofac Surg 47:936–39, 1989.
5. Mueller W, Drummond JN, Pribisco TA, Kaplan RF: Pulse oximetry monitoring of sedated pediatric dental patients. Anesth Prog 32:237–40, 1985.
6. Houpt M, Manetas C, Joshi A, Desjardins P: Effects of chloral hydrate on nitrous oxide sedation of children. Pediatr Dent 11:26–29, 1989.
7. Iwasaki J, Vann WF, Dilley D, Anderson J: An investigation of capnography and pulse oximetry as monitors of pediatric patients sedated for dental treatment. Pediatr Dent 11:111–17, 1989.
8. Badalaty MM, Houpt MI, Koenigsberg SR, Maxwell KC, Desjardins PJ: A comparison of chloral hydrate and diazepam sedation in young children. Pediatr Dent 12:33–37, 1990.
9. Wilson S, Tafaro ST, Vieth RF: Electromyography: its potential as an adjunct to other monitored parameters during conscious sedation in children receiving dental treatment. Anesth Prog 37:11–15, 1990.
10. Hasty MF, Vann WF, Dilley DC, Anderson JA: Conscious sedation of pediatric dental patients: an investigation of chloral hydrate, hydroxyzine pamoate, and meperidine vs. chloral hydrate and hydroxyzine pamoate. Pediatr Dent 13:10–19, 1991.
11. Whitehead G, Durr D, Adair S, Proskin H: Monitoring sedated pediatric dental patients. ASDC J Dent Child 55:329–33, 1988.
12. Everett GB, Allen GD: Simultaneous evaluation of cardiorespiratory and analgesic effects of nitrous oxide-oxygen inhalation analgesia. J Am Dent Assoc 83: 129–33, 1971.
13. Cassidy DJ, Nazif MM, Zullo T, Ready MA: Transcutaneous oxygen monitoring of patients undergoing nitrous

oxide-oxygen sedation. Pediatr Dent 8:29–31, 1986.
14. Moody EH, Mourino AP, Campbell RL: The therapeutic effectiveness of nitrous oxide and chloral hydrate administered orally, rectally, and combined with hydroxyzine for pediatric dentistry. ASDC J Dent Child 53:425–29, 1986.
15. White CS, Dolwick MF, Gravenstein N, Paulus DA: Incidence of oxygen desaturation during oral surgery outpatient procedures. J Oral Maxillofac Surg 47:147–49, 1989.
16. Wilson S: Conscious sedation and pulse oximetry: false alarms? Pediatr Dent 12:228–32, 1990.
17. Meyer ML, Mourino AP, Farrington FH: Comparison of triazolam to a chloral hydrate/hydroxyzine combination in the sedation of pediatric dental patients. Pediatr Dent 12:283–87, 1990.
18. Malamed SF, Quinn CL, Hatch HG: Pediatric sedation with intramuscular and intravenous midazolam. Anesth Prog 36:155–57, 1989.
19. Poiset M, Johnson R, Nakamura R: Pulse rate and oxygen saturation in children during routine dental procedures. ASDC J Dent Child 57:279–83, 1990.
20. Simon JF, Vogelsberg RJ: Use of nitrous oxide-oxygen inhalation sedation for children, in Behavior Management in Dentistry for Children. Wright, GZW, ed. Philadelphia: WB Saunders Co, 1975, pp 178-96.
21. Clayton DG, Webb RK, Ralston AC, Duthie D, Runciman WB: A comparison of the performance of 20 pulse oximeters under conditions of poor perfusion. Anaesthesia 46:3–10, 1991.
22. Clayton DG, Webb RK, Ralston AC, Duthie D, Runciman WB: Pulse oximeter probes. A comparison between finger, nose, ear and forehead probes under conditions of poor perfusion. Anaesthesia 46:260–65, 1991.
23. Anderson JA, Lambert DM, Kafer ER, Dolan P: Pulse oximetry: evaluation of accuracy during outpatient general anesthesia for oral surgery. A comparison of four monitors. Anesth Prog 35:53–60, 1988.
24. Amar D, Neidzwski J, Wald A, Finck A: Fluorescent light interferes with pulse oximetry. J Clin Monit 5:135–36, 1989.
25. 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.