



Pulse oximetry evaluation of vitality in primary and immature permanent teeth

Curt Goho, DDS

Dr. Goho is a Lt. Col. in the United States Army Dental Corps, Werzburg Dental Activity in Germany.

Abstract

Present methods of assessing pulp vitality (electric and thermal testing) are of limited use with children, often resulting in false positive or false negative results. Pulse oximetry is a proven, atraumatic method of measuring vascular health by evaluating oxygen saturation (SaO₂). This pilot study explores the use of a modified pulse oximetry ear probe to assess pulp vascular oxygen saturation in primary and immature permanent teeth. Pulse oximetry readily differentiated between known vital and nonvital teeth. Vital teeth consistently provided SaO₂ values that were lower than the values recorded on the patients' fingers. Further study of the SaO₂ changes in traumatized teeth, with a probe designed specifically for teeth, is warranted by these initial results. Although additional research is indicated, pulse oximetry is already an objective, atraumatic clinical alternative to the present electrical and thermal methods of assessing pulp vitality in children's teeth. (Pediatr Dent 21:109-113,1999)

Evaluation of pulp vitality is an important diagnostic aspect of treating traumatized teeth. The methods currently used are thermal and electrical stimulation. However, thermal and electric testing have limitations in providing accurate diagnosis¹, and both methods are difficult to administer or inconclusive when used with children.

Since both electric and thermal pulp testing require subjective responses from the patient, their use often leads to inaccurate results. Children cannot always describe subjective symptoms or sensitivity to a stimulus. False positives and false negatives occur if the dentist asks the child a leading question². Furthermore, both thermal and electric pulp testing are perceived as unpleasant stimuli, which may result in behavior management/cooperation problems with the pediatric patient. As children adapt their behavior to avoid a painful stimulus, their ability to properly respond to pulp testing is limited^{3,4}.

Another problem with present pulp testing methods is that they only indirectly monitor pulp vitality by measuring neural responses and not circulation. Since pulp vitality is purely a function of vasculature health, a vital pulp with an intact vasculature may test nonvital if only its neural component is injured. This situation is common encountered with recently traumatized teeth⁵. On the other hand, pulp nerve fibers are more resistant to necrosis than vascular tissue⁶, and thermal or electric testing of only pulp neural response may also result in false positive results if only the pulp vasculature is damaged.

For electric and thermal testing to be effective, the pulp must have a sufficient number of mature neurons. However, both primary and immature permanent teeth are not fully innervated with alpha myelinated axons, the neural components

which are responsible for the pulpal pain response⁷. Permanent teeth may not exhibit full alpha myelinated axon innervation until 4-5 years after eruption⁸. This reduced number of pain receptors makes them less responsive to stimuli^{9,10} and therefore more susceptible to false negative results from thermal and electric pulp testing. Considering all these limitations, present pulp testing with thermal and electric methods cannot be considered reliable vitality tests for the pediatric patient.

A direct measurement of pulpal circulation is the only real measure of pulp vitality. Pulse oximetry is a completely objective test, requiring no subjective response from the patient, that directly measures blood oxygen saturation levels. It is proven effective and is routinely used in medical applications through the use of finger, toe, foot, and ear probes. Pulse oximetry is based on placing arterial blood vessels between a light source and a detector. The light source diode emits both infra-red and red light, which is received by a photo-detector diode. Blood pulsating through the vessels changes the light path, which modifies the amount of detected light. This determines the pulse rate. To determine oxygen saturation (SaO₂), the pulse oximeter measures and compares amplitudes of the ratios of transmitted infra-red with red light. This ratio varies with relative fractions of oxygen saturated to unsaturated hemoglobin, and is used to calculate SaO₂. Skin, bone, and venous blood do not interfere with measurements¹¹. These characteristics infer that pulse oximetry is also capable of evaluating the blood vasculature status within a tooth, and therefore pulp vitality.

An earlier pilot study indicated the efficacy of using pulse oximetry to test pulp vitality on mature permanent teeth¹². Studies with in vivo permanent tooth models clearly demonstrated the strong correlation between pulse oximetry evaluation of pulp oxygen saturation and arterial blood gas analysis¹³. The purpose of this pilot study was to evaluate the efficacy of pulse oximetry for testing pulp vitality of primary and immature permanent teeth.

Materials and Methods

The sample population consisted of primary and permanent maxillary central and lateral incisors of children age 4 to 10. Sample criteria required teeth to be free of caries, restorations, developmental defects, or mobility. Roots, evaluated by panoramic or occlusal radiographs, had to be free of pathology or root resorption. The patient's dental history had to be negative for any history of trauma to the face, mouth, or teeth. Immature permanent incisors with root formation from Nolla stage 6 to 9 were included in the sample. Forty-five primary



Fig 1. Placement of a modified pulse oximetry ear probe partially erupted central incisor.

and forty-eight permanent incisors met the criteria and were used as the sample population.

As a control population to confirm the pulse oximeter's evaluation of pulp vascularity, ten known nonvital primary teeth with complete endodontic fillings were tested. Pulse oximeter readings from the patient's fingers served as the control sample for comparison of pulp oxygen saturation values with the patient's systemic oxygen saturation values.

Testing equipment consisted of the Lifestat 1600 pulse oximeter (Physio-Control, Redmond, Washington). The standard finger probe was used to measure finger oxygen saturation values. An earprobe with some of its plastic housing removed to reduce the probe's bulk, and therefore facilitate adaptation to small teeth, was used to measure tooth oxygen saturation.

Procedure

The patient's SaO₂ was first measured on the index finger using a finger probe. The patient's incisors were then evaluated by placing the modified ear probe onto the tooth (Fig 1). The probe was placed on the erupted crown so that light would travel from the facial to the lingual through the middle of the crown. The probe was positioned to create a light axis that was perpendicular to the axis of the erupted crown. Values were recorded after thirty seconds of monitoring each tooth. SaO₂ results were recorded and analyzed using the Spearman correlation analysis to evaluate the relationship between the finger and tooth pulse oximeter readings.

Results

The ten known non-vital teeth (control) recorded SaO₂ values of 0%. The immature permanent incisors SaO₂ values averaged 94% (SD=2.25). Their control values, measured on the patients' index fingers, averaged 98% (SD=1.74). Primary incisor SaO₂ values averaged 93% (SD=3.23). Their control values, also measured on the patients' index fingers, averaged 97% (SD=1.67). A Spearman correlation analysis of each individual's finger and tooth SaO₂ values showed a 0.22 correlation for the primary teeth subjects, and an 0.15 correlation for the permanent teeth subjects.

Discussion

This study confirmed the ability of the pulse oximeter to differentiate between vital and non-vital teeth. The average SaO₂

values for primary teeth (93%, SD=3.23) and immature permanent teeth (94%, SD=2.25) reflect similar readings found on an earlier study¹² of permanent teeth in which average SaO₂ values were 94%.

Both primary and immature permanent incisors had SaO₂ values that were lower than the SaO₂ values recorded on the patients' fingers. Primary incisor SaO₂ values were 4.12% lower than their associated finger readings, and immature permanent teeth had SaO₂ readings 3.41% lower than their associated finger readings. This also reflects an earlier study¹² on mature permanent teeth in which tooth and finger readings varied by 3%.

The lower tooth SaO₂ values may be attributable to several causes. Diffraction of infrared light by enamel prisms and dentin may cause decreased SaO₂ readings¹⁴. Fein, et al¹⁵ suggested that lower SaO₂ values for pulpal circulation may also be attributed to light ray scatter through the gingiva. Finally, the ear probe, although modified to make it smaller, was still too large to ideally position it on many of the smaller primary and partially erupted permanent incisors. This may have resulted in less than perpendicular direction of the light source through the crown, resulting in lower SaO₂ recordings.

Statistical analysis of the difference between finger and tooth readings, using the Spearman correlation analysis, showed a low correlation between finger and tooth SaO₂ readings. This finding did not conform to the original hypothesis that the lower tooth SaO₂ values would vary from the control (finger) readings by a consistent percentage. Although this initially appears to question the qualitative value of SaO₂ readings obtained from the teeth, it must be remembered that all vital teeth provided consistent SaO₂ readings, and all non-vital teeth recorded no SaO₂ values. This confirms that pulse oximetry is capable of detecting pulp vitality through enamel and dentin. The lack of statistical correlation between tooth and finger values may reflect inherent optical properties of teeth, or it may be due to inaccuracies from using a probe that was not specifically designed to fit on teeth. However, since a reproducible SaO₂ level is obtainable on vital teeth, pulse oximetry has immediate clinical value by providing baseline vitality data for traumatized teeth. Future readings can then be compared to the baseline SaO₂ values to determine any trends toward nonvitality. Further research, using a longitudinal study of pulse oximetry evaluation of traumatized teeth, is indicated.

Conclusion

This pilot study shows that pulse oximetry is an effective, objective method of evaluating dental pulp vitality. It is especially applicable to primary and immature permanent teeth where patient cooperation and incomplete pulp innervation reduces the effectiveness and reliability of thermal and electric pulp testing methods. Consistent pulse oximeter readings in this and other studies confirm that pulpal circulation and blood SaO₂ can be detected by the probe. The SaO₂ values from teeth routinely register lower than the readings from the patient's finger, however, no significant statistical correlation was found between the tooth and finger SaO₂ values. This may be due to the limitations of using a probe designed for other body parts, and not specifically for the anatomy of a tooth. To assess the effect of a smaller probe, further research is indicated using a smaller probe designed specifically for use on teeth.

Longitudinal studies using pulse oximetry to evaluate changes in traumatized teeth is also indicated. Until then, pulse oximetry does provide an objective, atraumatic method for the clinician to document pulp vitality in primary and immature permanent teeth.

References

1. Peters D, Baumgartner J, Lorton L. Adult pulpal diagnosis. Evaluation of the positive and negative responses of cold and electric pulp tests. *J Endo* 20:506-511, 1994.
2. Cash R. Bruxism in children. *J Pedod* 10: 105-126, 1986.
3. McDonald I- Avery D. *Dentistry for the child and adolescent*, 5th Edition. Phila, CV Mosby, 516-517, 1987.
4. Kennedy D, Kiely M, Keating P. Efficacy of pulp testing. *J Irish Dent Assoc* 33, 41-46, 1987.
5. Gazellus B, Olgart L, Edwall B. Restored vitality in luxated teeth assessed by laser Doppler flowmeter. *Endod Dent Traumatol* 4:265-268, 1988.
6. Fuss Z, Trowbridge H, Bender I. Assessment of reliability of electrical and thermal pulp testing agents. *J Endo* 12:7, 3011-305, 1986.
7. Johnson D, Harschbarger J, Rymer H. Quantitative assessment of neural development in human premolars. *Anat Rec* 205:421-429, 1983.
8. Fearnhead R. The histological demonstration of nerve fibers in human dentine. Sensory mechanisms of dentine. Oxford, Pergamon Press, 15-24, 1968.
9. Klein H. Pulp responses to an electric pulp stimulator in the developing permanent anterior dentition. *J Dent Child* 45:199-202, 1978.
10. Fulling H, Anderson J. Influence of maturation status and tooth type of permanent teeth upon electrometric and thermal pulp testing. *Scand J Dent Res* 84:286-290, 1976.
11. Operating Manual, Lifestat 1600 Pulse Oximeter, Physiocontrol Corporation, Redmond, Washington, Oct 1986.
12. Schnettler J, Wallace J. Pulse oximetry as a diagnostic tool of pulp vitality. *J Endo* 17:488-490, 1991.
13. Noblett W, Wilcot L, Franklin S, Johnson W. Detection of pulpal circulation in vitro by pulse oximetry. *J Endo* 22:1, 1-5, 1996.
14. Schmitt J, Webber R, Walker E. Optical determination of dental pulp vitality. *Trans Biomed Eng* 38(4), 346-352, 1991.
15. Fein M, Gluskin A, Goon W. Evaluation of optical methods of detecting pulp vitality. *J Biomed Optics* 2(1), 58-73, 1997.

ABSTRACT OF THE SCIENTIFIC LITERATURE



RETENTION OF FLUORIDE/TRICLOSAN IN PLAQUE FOLLOWING ADMINISTRATION.

Triclosan is a bisphenol which is used in both dentifrice and mouthrinse solutions, either combined with zinc citrate or with a copolymer (polyvinylmethyl ether maleic acid copolymer; GantrezÆ). In long term (> 6 months) clinical studies, a 12-59% plaque reduction was observed in individuals who used a NaF/triclosan/GantrezÆ dentifrice. Approximately 25% less plaque were found in a group of subjects who used a NaF/triclosan/GantrezÆ pre-brush mouthrinse solution. Little is known about the retention of triclosan in dental plaque in vivo after various modes of administration, i.e. after using dentifrice or rinsing solution. Since triclosan-containing products also contain fluoride, the aim of this study was to compare: (1) de novo plaque formation and (2) fluoride and triclosan concentration in approximal plaque when a NaF/triclosan/GantrezÆ-containing dentifrice slurry or a mouthrinse were administered during a 2-week period of no mechanical plaque control. 10 healthy dental students, without signs of destructive periodontitis and active caries lesions rinsed for 60 seconds, 2x daily, for a 14-day period with one of the following: a- a dentifrice slurry including 1 ml NaF/triclosan/GantrezÆ dentifrice mixed with 10 ml of tap water, b- 10 ml of a NaF/triclosan/GantrezÆ mouthrinse, or c- 10 ml of a NaF mouthrinse. De novo plaque formation was assessed on days 4, 7 and 14 using the Turesky's modification of the Quigley and Hein index. Samples of approximal plaque were obtained immediately after clinical examination on day 14, and were analyzed for fluoride and triclosan concentration using an ion-specific electrode and a high performance liquid chromatography (HPLC) system. The results show that: I- significantly more fluoride was retained in approximal plaque when delivered together with triclosan/GantrezÆ than without; II- less plaque was formed after the NaF/triclosan/GantrezÆ mouthrinse than the NaF/triclosan/GantrezÆ dentifrice slurry and III- the two different modes of administration of NaF/triclosan/GantrezÆ, i.e. a mouthrinse solution or a dentifrice did not differ in the concentration of either fluoride or triclosan in plaque.

Comment: The article demonstrates the new trend in dentistry to find agents that would assist in producing less plaque. With regard to pediatric patients, such agents must be obviously used cautiously in the very young ones. However specific high-risk patients like patients undergoing orthodontic treatment or compromised patients may benefit a great deal from these new products in the constant effort to reduce plaque formation and to prevent oral and dental diseases.

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Reprint Information: Downen Birkhed, Department of Cariology, Institute of Odontology, P.O. Box 450, SE-405 30 Goteborg, Sweden. Fax: +46 31 41 05 70, E-mail: birkhed@odontologi.gu.se.