Assessing Inflammatory Status of Pulp in Irreversible Pulpitis Cases with Pulse oximeter and Dental Hemogram

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Objective: *This study evaluated pulse oximetry and dental hemogram in teeth with the clinical diagnosis of irreversible pulpitis (IP) to assess the inflammatory status of the pulp.* ***Study design:*** *The study and control groups (30n each) had teeth with IP and sound teeth respectively. Patients in the study group had night pain with or without pain on mastication (NM, N). Blood oxygen saturation (%SpO2) was recorded with a custom made pulse oximeter (CPO). For dental and peripheral hemogram, smears were made for each patient from the first drop of blood while entering the pulp and finger blood respectively.* ***Results:*** *Control group had mean %SpO2 in finger 91% (86-97); and in teeth 84% (80-91), while the study group had mean %SpO2 in finger 92% (88-98) and in teeth 83% (71-94). Fifty percent of IP cases were vital while no tooth showed necrosis according to CPO which was further confirmed by bleeding status from the pulp. Based on the findings of the clinical diagnosis, %SpO2 and bleeding status of IP and normal cases, the terminology as coronal or total pulpitis seems more appropriate. The statistical difference was significant in fingers while non-significant in teeth of IP and normal pulp cases. Dental hemogram of IP cases showed an overall significant fall of neutrophil, lymphocyte, eosinophil and monocyte counts compared to normal.* ***Conclusion****: Pulse oximetry was the most accurate pulp test to diagnose vitality in normal as well as inflamed pulps while hemogram was inconclusive for the same.*

Keywords: Dental hemogram, Pulp vitality, Pulp test, Pulse oximeter

INTRODUCTION

Diagnosis of irreversible pulpitis (IP) has always been challenging to the clinicians both for permanent and primary teeth. The unique nature of the pulp to respond to an insult itself proves the vitality of pulp. Despite exhibiting vitality, it is conventionally believed that pulp will not be able to return to the normal status and therefore is recommended for radical treatment. Moreover, histological findings of the extent of inflammation have not been found correlating with a clinical diagnosis which says that the inflammation of pulp in response to dentinal caries might not reach to the point to be considered as irreversible.[1]

Moreover, evidence of successful, vital pulp therapies in the cases diagnosed as IP, prove the healing potential of inflamed pulp.[2] With the advent of new materials like MTA, CEM, Biodentine, PRF etc., it becomes increasingly important to review the traditional radicular treatment for irreversible pulpitis. Therefore the diagnosis of pulp status based on subjective clinical symptoms and nerve response,by electrical (EPT) or thermal pulp tests (TPT) need to be re-evaluated.The evidence has provoked the need of investigation of inflammatory pulp status which can aid in the case selection for vital pulp therapy in irreversible pulpitis cases and likewise improve the success rate[3, 4] for what can be a better obturating material than a vital pulp itself.

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Nonetheless, the capability of fighting an insult is related to immunity, i.e. white blood cells (WBC) which again is the function of the pulp vascularity. Prader[5] suggested that dental hemogram from the first drop of blood of an exposed pulp would indicate the actual status of pulp, and might represent the histological picture of that particular tooth. Later, Guthrie *et al*.[6] correlated dental pulp hemogram with pain history and histologic picture of pulp and found that the teeth having inflammatory cells near exposure were "good risk" and those having inflammatory cells in root pulp were "poor risk" for VPT. Although no specific correlation with pulp hemogram and histology could be established, haemorrhage from an exposed pulp was not recommended as a contraindication for the VPT.

Moreover, the immunity carried by WBC and the vitality, which indicates oxygenation of tissue carried by red blood cells (RBC) is a matter of vascular health of pulp. Recently evolved 'pulse oximeter' has been successful in diagnosing the vascular status of pulp including vital, non vital, obturated, traumatic or inflamed pulp.[7-10] Setzer *et al* had found the pulse oximeter successful in diagnosing different inflammatory diseases of pulp against the clinical diagnosis. In our previous validation study of the CPO, %SpO2 between 79%-85% in permanent and 80%-85% in primary teeth was not found while testing on extreme conditions of pulp, *i.e.* vital, non-vital and non-vital obturated primary or permanent teeth. Setzer *et al* [10]found this range (79%-86%) for IP. Nonetheless, the success of their study in detecting the different inflammatory status of the pulp led us to further confirm our CPO in IP cases and then validating it by entering the pulp, which is a 'gold standard' for any pulp testing apparatus.[7,11]

As Guthrie[6] stated that the bleeding from exposed pulp might not be a contraindication for the VPT, rather it would be interesting to explore the diagnosis of IP based on the history of pain with the hemogram and the pulse oximeter. The objective of this study was therefore, to correlate pulse oximetry and dental hemogram in teeth with clinical diagnosis of IP.

MATERIALS AND METHOD

The study was conducted after getting clearance from the Institutional Ethical Committee. Validation of CPO was done on intact vital, non-vital obturated and non-vital untreated primary or permanent teeth to check it's accuracy and sensitivity.[9] As the CPO showed 100% accuracy in previous study, we expanded the use of same CPO to detect inflammatory status of pulp in the cases with clinical diagnosis of IP.

Children from 4-12 yrs. of age, who visited the Department of Pediatric Dentistry of the institute, were included in this study. In total, 60 teeth were divided into the study and control teeth having 30 teeth in each according to the criteria below:

Sample selection

The inclusion criteria for teeth of study group were- patients with written consent from their parents; history of pain suggestive of symptomatic irreversible pulpitis *i.e.* night pain with or without pain on mastication (NM and N respectively) for not more than a week in relation to the carious primary or permanent molar teeth, having intact buccal and lingual walls; carious primary tooth with atleast 2/3rd of the root remaining in radiograph. The exclusion criteria were- teeth affected with a dental anomaly, trauma, signs of necrosis, periodontal pocket >3mm, mobility, gingival edema, external/internal resorption in IOPA x-ray, orthodontic braces, prosthetic crowns or patient having any blood disorder. Teeth in the control group were- without any history of pain and indicated for extraction, *e.g.* serial extraction cases, corrective orthodontic cases, supernumerary teeth, and over-retained intact primary teeth.

Radiographs of all teeth were taken using a parallel technique (Medico 10D model dental X-ray system, Meditronics Asia PVT LTD operating at 70 kVp and 8Ma). Teeth with internal or external resorption and periapical bone radiolucency were excluded. Teeth with or without widened periodontal ligament were included.[12]

Study group (30n) comprised of 10 primary teeth- 6 mandibular molars (4 first, 2 second), 4 maxillary molars (1first, 3 second); and 20 permanent-17 mandibular molars (16first, 1 second), maxillary molars (3 first). All 30 teeth had night pain (N). Among these 23 teeth were with pain on mastication (NM) too.

Pulse oximetry

CPO for dental application was made by modifying a commercial finger Pulse Oximeter (PO) by an electrical engineer. Ranges of oxygen saturation (%SpO2) of regular vital, non-vital and non-vital obturated teeth were established with CPO.[9] Method of CPO application was similar to our previous study.[9]

On the basis of our previous findings (%SpO2 >85% was vital for both primary and permanent teeth), study cases were divided into two groups, *i.e*. cases with %SpO2 above 85% and below 85% in order to investigate further, the correlation between%SpO2 and inflammatory cells in hemogram.

Because of the 100% accuracy of our CPO[9] compared to EPT or TPT, these pulp testing were not performed, and thus the bias of these variables was removed. This endeavour may, therefore, establish the clinical use of pulse oximeter.

Dental hemogram

After anaesthetising the concerned tooth with the nerve block, it was isolated with a rubber dam. Gross caries was then removed carefully with a spoon excavator and was followed by peripheral caries removal with a large round bur. For in-depth caries removal near the pulp, micro motor with water irrigation and high suction was used slowly to avoid heat generation. As soon as the first drop of blood appeared out of the carious exposure, it was transferred with the help of disposable syringe to a sterilized, clean glass slide for making the smear. Similarly, a peripheral blood smear was made from a finger of the same patient, cleaned with isopropyl alcohol to serve as control to investigate any deviation from peripheral blood of IP cases. Both blood smears were sent to the Department of Oral Pathology of the same institute for differential WBC count. Similar procedure was done for normal pulp cases, where pulp was entered directly for collecting blood for hemogram, before extraction.

The first investigator SM, the postgraduate student, did the preliminary diagnosis, radiographic investigation and all other clinical procedures under continuous visionary of DSS, the senior professor. All clinical procedures were guided and assisted by CB. Individual file for each patient was maintained.

The collected data were analyzed using SPSS software (SPSSVERSION 21.0, IBM Corporation, Armonk, NY, USA). P-Value <0.5 was considered statistically significant.

RESULTS

The study correlated pulse oximetry and dental hemogram in teeth with clinical diagnosis of IP. Chi-square test showed subjects with pain (N or NM) were significantly higher in irreversible pulpitis than regular group (table-1).

Table 1: Comparison of clinical sign and symptoms between irreversible pulpitis and normal pulp groups

Clinical sign and symptoms		Groups		Chi-square test
		Irreversible pulpitis n (%)	Normal pulp n (%)	
Night pain	Yes	30 (100.00)	00 (0.00)	Yates' χ^2 = 56.067, df = 1, P = 0.000 (<0.001), Very high Sig.
	No	00 (0.00)	30 (100.00)	
Pain on mastication	Yes	23 (76.67)	00 (0.00)	χ^2 = 37.297, df = 1, P = 0.000 (<0.001), Very high Sig.
	No	07 (23.33)	30 (100.00)	

Table 2: Oxygen saturation values in finger and tooth in irreversible pulpitis and normal pulp cases

Groups	Oxygen saturation value			
	In Finger		In Tooth	
	Mean ± SD	Min- Max	Mean ± SD	Min- Max
Irreversible pulpitis	92.90 ± 2.48	88.00-98.00	83.54 ± 5.80	71.33-94.00
Normal pulp	91.37± 2.50	86.00-97.00	84.73 ± 2.66	80.00-91.00
Unpaired t-test	t = 2.385, P = 0.020 (<0.05), Significant		t = -1.025, P = 0.310 (>0.05), Not Significant	

Cases of IP had mean %$SpO_2$92% (88-98) in finger and 83% (71-94) in teeth. Normal pulp cases had mean%SpO_2 91% (86-97) in finger and 84% (80-91) in teeth. Unpaired t-test showed significant (p= 0.020, t= 2.385) difference in %SpO_2 in fingers; while non-significant (p= .310, t= -1.025) in teeth of irreversible and normal pulp cases.

Table-3 shows means, unpaired t-test (lymphocyte, neutrophil) and Mann-Whiney test (eosinophil, monocyte) for differential WBC counts in peripheral blood and dental pulps (dental hemogram) of IP and normal pulp cases respectively. Significant fall of neutrophil, lymphocyte, eosinophil and monocyte count (t= -3.577, -3.693; p= 0.001, 0.000 respectively and MW= 59.000, 158.000; p= 0.000, 0.000 respectively) was found in IP pulp cases compared to normal pulps. There was no difference in lymphocytes and neutrophil count in peripheral blood of IP or normal pulp cases, while eosinophil and monocyte were low in peripheral blood of IP cases compared to normal pulp cases.

DISCUSSION

Any diagnostic test is perfect, only when it is positive or negative in the presence or absence of the disease. The accuracy of a test is related to the extent it correctly classifies a disease. Pediatric patients are often unable to give the proper history, firstly due to the pain being a subjective response and secondly because of the presence of more than one carious teeth. Sensibility tests (EPT, TPT) rely on the sensory response by nervous tissues which may remain active even after full necrosis of pulp, and thus may give false positive result.[13]. Nonetheless,the vitality of any tissue is directly related to the blood supply, *i.e.* oxygen and inflammatory cells carrying capacity. The present study investigated whether the pulse oximetry (blood oxygenation of the pulp) might be considered as diagnostic method for the clinical inflammatory condition of the dental pulp.

The study consisted of 60 samples divided into two groups- 30 vital, *i.e.* sound teeth and 30 molars with the clinical diagnosis of irreversible pulpitis. In the study group, both the primary and permanent teeth were taken. This is because the preservation of the vitality of the pulp is equally essential in the young permanent tooth or in the complex root canal system in primary molars.

Table 3: Means and Inter-comparison of Differential WBC counts in peripheral and dental pulp smear in IP and normal pulps

	Neutrophils		Lymphocytes		Eosinophils		Monocytes	
	Peripheral	Dental Pulp	Peripheral	Dental Pulp	Peripheral	Dental Pulp	Peripheral	Dental Pulp
	Mean ± SD (Min- Max)	Mean ± SD (Min- Max)	Mean ± SD (Min- Max)	Mean ± SD (Min- Max)	Mean ± SD (Min- Max)	Mean ± SD (Min- Max)	Mean ± SD (Min- Max)	Mean ± SD (Min- Max)
IP	58.10 ± 5.82 (40.00-69.00)	55.70 ± 7.24 (40.00-69.00)	46.90 ±11.96 (26.00-67.00)	40.17 ± 10.23 (20.00-68.00)	3.73 ± 2.46 (1.00-9.00)	2.87 ± 2.18 (0.00-8.00)	0.73 ± 0.79 (0.00-2.00)	0.00 ± 2.00 (0.00-3.00)
NP	56.30 ± 2.93 (51.00-62.00)	60.93 ± 3.44 (53.00-68.00)	46.90 ± 3.95 (38.00-53.00)	47.17 ± 1.78 (42.00-49.00)	6.23 ± 1.52 (2.00-9.00)	7.50 ± 1.57 (3.00-9.00)	2.27 ± 1.70 (0.00-7.00)	3.20 ± 2.27 (0.00-8.00)
	t = 1.514, P = 0.135 (>0.05), NS*	t = -3.577, P = 0.001 <0.01), HS+	t = 0.000, P = 1.000 (>0.05), NS	t = -3.693, P = 0.000 (<0.001), VHS#	MW = 205.500, P = 0.000 (<0.001), VHS	MW = 59.000, P = 0.000 (<0.001), VHS	MW = 189.000, P = 0.000 (<0.001), VHS	MW = 158.500, P = 0.000 (<0.001), VHS

NS*= non significant, HS+= highly significant, VHS#= very high significant

Table-4: Relation of %SpO2, pain history, bleeding status and proposed inflammatory status of pulp

	Pulse oximeter %SpO2		Pain history	Number of teeth	Bleeding Status (gold standard)	Inflammatory status of Pulp (proposed terminology)
Control	>85% (86-97)		Not any	30	Yes	Vital (Not inflamed)
Irreversible Pulpitis (Clinical Diagnosis)	>85%		N#	5	Yes	Vital (Coronal pulpitis)
			NM*	10	Yes	
	<85% (78-81)	Primary	N	0	Yes	Coronal Ischemia Or (Total Pulpitis)
			NM	5	Yes	
					Yes	
		Permanent	N	3	Yes	
			NM	7		
Non Vital[51]	<80	Primary	Discolor and/or Sinus (no pain)	10	No	Non-vital (Necrosis)
	<78%	Permanent		10	No	

*NM= Night pain with pain on mastication, #N= Night pain only

Table 5: Correlation of Oxygen Saturation with Pain History and Hemogram

		CORONAL PULPITIS (%SpO2>85)	TOTAL PULPITIS (%SpO2<85)
HISTORY OF PAIN	NM*	**33.33%**	**80%**
	N#	**100%**	**100%**
NEUTROPHILS (53%-68%)	Below normal range	**26.66%**	**26.66%**
	Within normal range	**66.66%**	**60%**
	Above normal range	**6.66%**	**0%**
LYMPHOCYTES (42%-49%)	Below normal range	**60%**	**86.66%**
	Within normal range	**26.66%**	**13.34%**
	Above normal range	**13.33%**	**0%**

*NM= Night pain with pain on mastication, #N= Night pain only

According to the available literature, the treatment of choice for irreversible pulpitis is root canal treatment or extraction.[14,15] However in pediatric patients, pulpectomies are often painful and fearful requiring multiple visits, making it costly procedure. VPT might be the better choice of treatment for irreversible pulpitis cases.[2] Previously Schroder *et al* (1978)[16], Heilig *et.al* (1984)[17]and Fishman *et.al* (1996)[18] performed vital pulpotomy with $Ca(OH)_2$ on primary teeth with coronal chronic pulpitis and got 59%, 88% and 81% success respectively. Many authors also found VPT to be successful as an emergency treatment for relieving pain.[19-21]Moreover, the radical treatment always remains a choice if previous vital treatments have failed. The reason for the failure of pulpotomy was related to the pulpotomy material and clot formation over the root pulp stumps.[22, 16-18] Mejare[22] therefore, tried formocresol over pulp with tested vehicles to minimize the inflammation in root pulp so as to increase the effectiveness of formocresol for it does not work on inflamed tissue. It can be therefore said that the success or failure of VPT was more dependent on material and extent of inflammation in the pulp. In view of successful pulpotomies with newer materials like MTA[2], NEC[23], PRF[24] and CEM[25], choice of VPT should never be omitted provided, the actual status of pulp inflammation can be judged before the endodontic procedure.

Patients with irreversible pulpitis not having the pain more than one week were chosen. Whenever bacterial invasion is present in dentin, the inflammatory process starts in the pulp near the exposure site. Pulp has a capacity of localizing the inflammation, while the tissue adjacent to the inflammatory region may be completely normal.[26] However, if the bacteria or irritant is eliminated, inflammation subsides, and the reparative process starts over the injury site. Alternatively, if the irritant overwhelms the pulp defence ability or the stimulus is present for the long duration, the blood flow ceases, and the injured tissue undergoes necrosis slowly.[27,28,29] Necrosed part gets infected and inflammation proceeds in deeper pulp tissues in the root. This process of infection and inflammation progression explains the reason of failure of VPT in total pulpitis cases of Mejare.[22] Teeth with night pain with or without pain on mastication (NM or N) of more than a week had possibilities of partial/full necrosis of coronal pulp and thus inflammation extension towards and/or beyond root pulp.[6]

Results (table 2) of this study further confirmed our previous study[9] with CPO that the mean %SpO2 (standard cases) in finger was 91.37$\pm$2.50 and in teeth, was 84.73$\pm$3.50. For irreversible pulpitis cases, mean %SpO2in finger was 92.90$\pm$2.48,and in teeth, was 83.54$\pm$5.80. No correlation was found between teeth and finger

%SpO2% values in irreversible pulpitis cases while there was a strong positive relationship between tooth and finger in standard pulp cases. The same correlation was found in our previous study as well.[9]

Clinically diagnosed irreversible pulpitis cases showed >85%SpO2 in some cases while others had <85%SpO2. Previously we found %SpO2 >85% for all deciduous and permanent vital teeth; and %SpO2 < 78% in permanent and %SpO2<80.33% in primary for non-vital teeth.Table-4 shows the relation of the history of pain, a whole range of pulse oximetry readings, gold standard pulp status and a number of teeth as per our previous and current study. On the basis of findings proposed inflammatory status of pulp also has been laid down. Because of 100% accuracy of CPO, we took vital teeth only as a control to compare with an experimental group of teeth with a clinical diagnosis of IP.

Some of the irreversible pulpitis cases showed a range of %SpO2 78-81*i.e.* <85%, that meant teeth were either ischemic or necrotic. Nonetheless, when pulp was entered for hemogram, they bled. This was probably because even if pulps bled, overall oxygenation was less, i.e. pulp was severely ischemic and was in irreversible condition to be treated with VPT. Probably coronal pulpitis had extended to root pulp, i.e. total pulpitis. Mejare[22] had discussed their pulpotomy cases as coronal pulpitis and total pulpitis. Nonetheless, the division was arbitrary for the extent of inflammation of pulp. Our CPO showed a clear division for the extent of pulp inflammation. Irrevesible pulpitis cases, with >85 %SpO2 were expected to have inflammation in coronal pulp only; while cases with <85 %SpO2 to have ischemia/ partial necrosis in coronal part with the inflammation extendidng towards root pulp. This study confirmed the classification of pulp inflammation by Shroader *et al* [30]and Mejare.[22]Important to note that both the authors came to know the pulp status after entering, while with pulse oximeter it could be diagnosed without entering. As there is no established protocol for the relation of a pulseoximeter, inflammatory status and endodontic treatment strategy, we entered the pulp for hemogram.

In the second group of IP with <85%SpO2, maximum patients had a history of NM (table 4). Therefore it can be assumed that these teeth were more ischemic in the coronal portion of the pulp. Our previous study showed %SpO2 <78 for permanent teeth and <80.33 for primary teeth were non-vital.[9] In this study, 2 teeth having 80.33% and 80.66 %SpO2 were of particular interest for being at borderline readings. These teeth had pain history of NM and N respectively together with the bleeding. Contrary to our gold standard findings, Setzer[10] found this range of %SpO2 in teeth clinically diagnosed as pulp necrosis but important to note is the fact that they did not enter the pulp. To combine the results of this study and that of the Setzer's[10] it can be said that those teeth which was found to be severely ischemic were not the suitable for VPT.

When inflammatory cells in hemogram were investigated, no significant difference in eosinophils, monocytes and basophils between pulps of normal and irreversible pulpitis cases was found (Table-3). Neutrophils and lymphocytes showed an overall significant difference between average and irreversible pulpitis cases (Table3).

Tables-3 shows the relation of irreversible pulpitis and normal pulps with hemogram. Neutrophils and lymphocytes in irreversible pulps were significantly less compared to the normal pulps. Nonetheless, validation of CPO study clearly found the difference of %SpO2 of vital and non-vital teeth.[9] Overall %SpO2 in irreversible pulpitis cases showed a range of %SpO2 of non-vital as well as of vital range too. Therefore overall cases were further investigated according to %SpO2 found in irreversible pulpitis cases according to the proposed classification, correlated with a history of pain (N or NM) and hemogram (table-5). %SpO2> 85% had only 33.33% patients with NM. It can be taken either as an error of CPO or probably because the pulp had not reached to that ischemic condition to fall below 85% of %SpO2 or because the history of pain is subjective and thus is non-specific. It is important to note that maximum teeth >85% of %SpO2 were only with night pain history and were vital which probably made them the fine candidate for VPT (table-3 & 5). Gutherie[6] *et al.* found that the teeth with night pain were in "poor risk" and with the pain during mastication were in "good risk" for necessary treatment. Our results were in contrast to their study that maximum teeth with NM were total pulpitis cases according to %SpO2. Teeth with N only had better oxygenation as that of vital. Based on these results, history could not produce a true condition of vitality status of the pulp.

As stated above (table 5), IP cases were further divided on the basis of %SpO2. Lymphocyte seems more correlated compared to neutrophils. In cases of "above normal," it was only marginally increased, *i.e.* only 1 point (69%) from the normal range of neutrophils (53%-68%). Likewise in cases of total pulpitis, there was no significant difference in neutrophils counts, but more cases were there with increased lymphocytes counts compared to normal range.

Although the literature suggests that the inflammatory cells are always high in its reversible or irreversible pulpitis[15], this study could not show this. The difference may be due to the histological nature of those studies while this study investigated the first drop of blood, *i.e.* the peripheral region of pulp cavity and probable pulp near the long-standing carious exposure was ischemic and progressed towards necrosis. Results of this study were in contrast to that of Gutherie *et al* [6]Their results showed increased lymphocytes and extent of these cells in pulp indicated poor risk for VPT, while decreased lymphocytes were found in total pulpitis cases in this study. Nonetheless, Gutherie[6] recommended hemogram as not conclusive to diagnose the extent of pulpitis. Therefore, it can be concluded that hemogram did not give an overall picture of pulp inflammation but only near direct exposure. Moreover, it is a time consuming and invasive procedure. For these reasons, hemogram cannot be recommended as a predictor for VPT.

Design of CPO seems of utmost importance as there was decrease in %SpO2 saturation in supernumerary vital teeth compared to normal sound teeth in arch which was always above 85%. This decreased overall average of %SpO2 in sound teeth compared to our previous study, although. CPO design needs further improvements therefore.

CONCLUSION

Considering overall relation of pain history, pulse oximetry and hemogram, it can be said that although the diagnosis of irreversible pulpitis was made with history of pain, it could not show true status of vitality of the pulp, As CPO showed 50% of teeth pulp were vital and thus probably are good candidates for VPT. Therefore these teeth should be re-designated as coronal pulpitis. Other 50% of teeth were quite ischemic in coronal portion as per CPO. Those teeth with a history of night pain with or without pain on mastication with %SpO2 below vital range should be re-designated as total pulpitis, i.e. poor risk for VPT.

Likewise, hemogram was inconclusive that no inflammatory cells except only lymphocytes counts were found decreased in most of the total pulpitis cases.

Pulse oximetry was the most accurate vital pulp test to diagnose vitality status of the pulp in normal and inflamed pulps. This test should be further investigated for their accuracy by entering the proper pulp space followed by treatment done and with the follow-up. Therefore, further study is needed before executing the pulse oximeter for diagnosis of pulp inflammatory status in clinics.

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