Formocresol pulpotomy of primary teeth and occurrence of enamel defects on the permanent successors

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Fifty-two permanent tooth pairs of 37 children aged 8 to 16 years were evaluated for opacities and hypoplasia of the enamel. Formocresol pulpotomy had been performed in one of the primary teeth preceeding each tooth pair. The study comprised teeth with clinically and radiographically successful and unsuccessful pulpotomies. None of primary control teeth had a history of pulp exposure. 57 permanent teeth with caries-free predecessors were included for further control.

In the formocresol treated group the prevalence of opacities according to localization was: occlusal surface 25 %, buccal surface 37 % and palatal surface 14 %. In the control group, the corresponding prevalence was 21 %, 35 % and 14 %. The prevalence of hypoplasia was 2 %, 8 % and 0 % respectively, in the formocresol group and 2 %, 14 % and 4 % in the control group. In the 57 permanent teeth opacities were found in 16 % of the occlusal and buccal surfaces and in 18 % of the palatal surfaces. The prevalence of hypoplasia was 12 %, 5 % and 0 % respectively.

No differences in prevalence of enamel defects on permanent teeth could be demonstrated in relation to child age when formocresol pulpotomy was performed.

The conclusion of the study was that no relationship between formocresol pulpotomy of primary teeth and enamel defects on their permanent successors could be demonstrated.

Keywords: Endodontics; hypoplasia

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Developmental disturbances of enamel formation can be attributed to either systemic or local causes. Systemic causes such as a high fluoride ingestion resulted in changes usually observed clinically as mottling, while local causes most frequently would result in opacities or discoloration of the enamel (4). One of the most frequently described local causes to developmental disturbances in the permanent bicuspids is infection of the pulp and/or apical periodontitis of the primary tooth (1, 2, 9). Since one of the possible explanations of this effect may be the presence of accessory root canals in primary molars (14), it seems reasonable to suspect that any drug employed in the endodontic treatment of primary molars might result in the same effect. One recent study by Pruhs, Olen & Sharma (10) seems to indicate that the use of formocresol in the treatment of the pulp in a primary molar did in fact increase the

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prevalence of enamel changes in the corresponding permanent tooth. The purpose of the present study was to further evaluate the possible relationship between formocresol treatment of a primary tooth and enamel changes in the permanent successor.

MATERIAL AND METHODS

52 permanent tooth pairs of 37 children aged 8 to 16 years were examined for opacities and hypoplasia of the enamel on the occlusal, the buccal and palatal surfaces. In each tooth pair one of the corresponding primary teeth had been formocresol pulpotomized, while no pulp exposure had occurred in the contralateral primary tooth.

Both clinically and radiographically successful and unsuccessful pulpotomies were included.

All teeth were treated at the department of pedodontics or by the Child Dental Service in Aarhus. The age of the children when treatment was performed ranged from 2 years and 6 months to 9 years. Fluoride content of local drinking water was 0.1–0.3 parts/10^6 F. In no case was fluoride tablets administered.

Of the 52 formocresol pulpotomized primary teeth, 5 teeth had pathological periradicular conditions, 36 pulpotomized teeth were clinically and radiographically successfully observed to the time of exfoliation or extraction 3 to 5 years after treatment for histological investigation (11). Information could not be obtained of eleven cases.

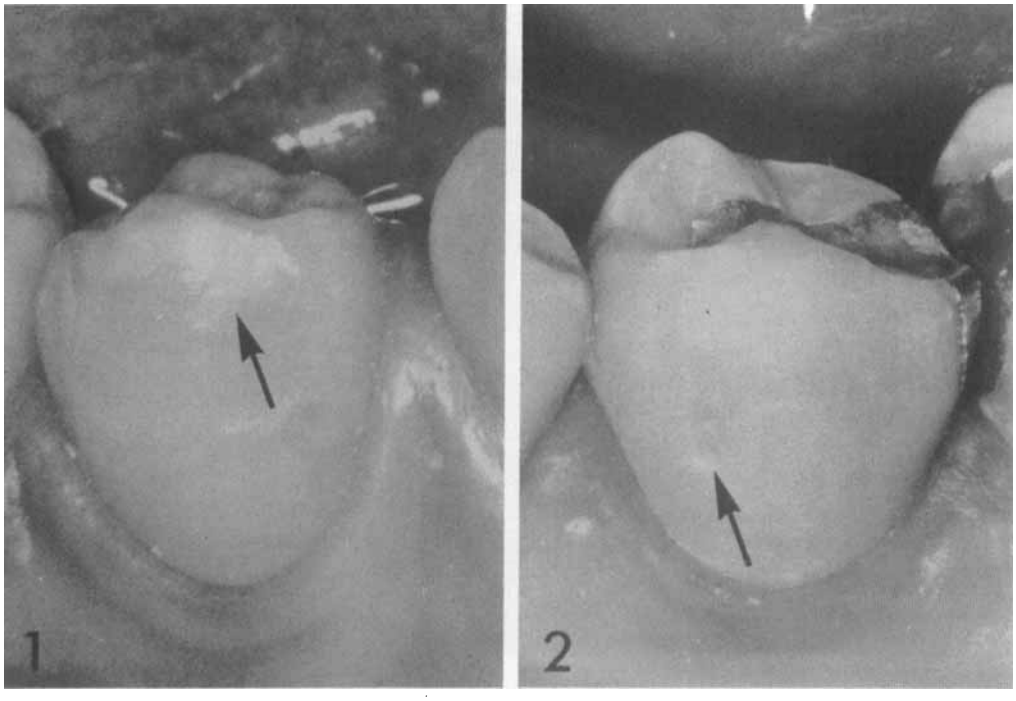
Enamel defects were defined as any abnormality in the surface morphology or colour. The diagnostic criteria and the criteria for distinction between opacities and hypoplasia were the same as those used by Niswander & Sujaku (9). Fig. 1 shows a *typical opacity* and Fig. 2 a typical hypoplasia of enamel. The number, type and location of defects were recorded clinically by translumination using fiber light (Dürr-Dental, Beitighing, Germany). Before the examination, the teeth were dried with compressed air for one minute. The examination was undertaken by two persons not knowing which tooth had been treated by pulpotomy.

The 52 tooth pairs were tabulated in 2 x 2 tables according to presence or absence of enamel defects on the tooth in the formocresol treated side and on the tooth in the control side. The resulting distributions were tested statistically using McNemars test (3) at the 5% significance level. Furthermore, to avoid bias due to inflammation of the pulp of the control teeth related to deep carious lesions or fillings, 57 permanent teeth with corresponding cariesfree teeth were examined. The age of these children ranged from 12 to 14 years.

RESULTS

Of the 52 tooth pairs, 53.8 % and 51.9 % had 1 to 4 opacities of the enamel respectively in the formocresol side and in the control side (Table 1). The size of the opacities varied, but in most instances the opacity was small and well-defined. No differences could be demonstrated between the two sides concerning size and number of opacities.

The prevalence of opacities of the formocresol side for specific surfaces was respectively 25.0 %, 36.5 % and 13.5 % on the occlusal, the buccal and the palatal surfaces. In the control group, the prevalence of opacities was 21.2 %, 34.6 % and 13.5 %, respectively on the corresponding surfaces (Table 1). No statistically significant differences were demonstrated. Hypoplasia of the enamel was observed in 9.6 % of the formocresol side and in 19.2 % on the control side (Table 2). One to 4 hypoplasia could be demonstrated in both groups. The variation in size and number of defects was



Figs. 1–2. Opacity of enemal (Fig. 1) and hypoplasia of enamel (Fig. 2) of the buccal surface (arrows).

similar in the two sides. The prevalence of hypoplasia of the formocresol side on specific surfaces was respecticely 1.9 %, 7.7 % and 0.0 % on the occlusal, the buccal and the palatal surfaces. In the control side the prevalence of hypoplasia was 1.9 %, 13.5 % and 3.8 %, respectively (Table 2).

Enamel opacities of the teeth with caries-free corresponding primary teeth were observed in 36.8 %. The prevalence of opacities was respectively 15.8 %, 15.8 % and 17.5 % on the occlusal, the buccal and the palatal surfaces. Only for the buccal surfaces could a statistically significant difference be demonstrated ($\chi^2 = 5.79$; d.f. = 1; $P < 0.05$). This could, however, also be found, when these teeth were compared with the teeth in the control side ($\chi^2 = 5.13$; d.f. = 1; $P < 0.05$). Hypoplasia of the enamel was observed in 14.0% of the teeth with caries-free predecessors, with a prevalence of 12.3 %, 5.3 % and 0.0 % for the occlusal, the buccal and the palatal surfaces respectively.

Table 1. *Prevalence of enamel opacities of permanent teeth in relation to formocresol pulpotomy of the corresponding primary teeth (52 tooth pairs)*

	Occlusal	Buccal	Palatal	Total
	(in percentage)			
Formocresol treated teeth	25.0	36.5	13.5	53.8
Control teeth	21.2	34.6	13.5	51.9

Table 2. *Prevalence of enamel hypoplasia of permanent teeth in relation to formocresol pulpotomy of the corresponding primary teeth (52 tooth pairs)*

	Occlusal	Buccal	Palatal	Total
	(in percentage)			
Formocresol treated teeth	1.9	7.7	0.0	9.6
Control teeth	1.9	13.5	3.8	19.2

Only when the total prevalence was considered was the frequency large enough to allow for a χ^2-test, which demonstrated no statistically significant difference.

DISCUSSION

The present material consists of 52 permanent tooth pairs where one of the predecessors in each pair had been pulpotomized according to the formocresol method.

The results from this study showed no relationship between opacities and hypoplasia of the enamel on permanent teeth and formocresol pulpotomy of the primary teeth. Only minor and inconsistent differences were observed when comparison was made with permanent teeth with caries-free predecessors. Thus, the findings of Pruhs et al. (10), were not corroborated. In a survey (7) of the occurence of non-fluoride opacities and nonfluoride hypoplasias of enamel in 588 children aged 9 to 14 years, it was reported that the prevalence of opacities or hypoplasias was 14.6 %. This seems considerably lower than the prevalence found in the present study, but could be due to differences in examination procedure.

The buccal surfaces of the teeth in the present study had the highest percentage for enamel defects. This is in agreement with the study by Pruhs et al. (10). A surprising finding was the more frequent occurrence of hypoplasia of the control teeth, to which no explanation can be offered. Due to relatively wide variation in age of the children when the formocresol treatment was performed, it may be argued that development of the corresponding permanent tooth was too advanced for the drug to have any harmful effect on the permanent tooth germs. It has been shown recently (8) that at the age of 6 years most bicuspids have reached a stage where the crowns radiographically are completely formed.

In the present study, 30 of the primary teeth were formocresol treated before the age of 6 years, while 22 of the primary teeth were treated after the age of 6 years. No difference was demonstrated in prevalence of opacities in the two groups of teeth. The number of hypoplasia was too small to allow a similar analysis.

In conclusion, the results seem to show that application of formocresol on the pulp tissue in primary teeth has no effect on the mineralization of the succedaneous permanent tooth germs.

This finding corroborates with the histological evaluation of pulp tissue of formocresol pulpotomized primary teeth, showing that inflammatory reactions normally occur in the pulp. However, in the apical part of the root canal, the tissue in most instances is free or nearly free of inflammation in clinically and radiographically successful treatments (11, 12, 13). Furthermore it has been shown in earlier studies (5, 6) that there is an attempt to wall off inflammation in the periapical area of primary teeth and a strong attempt at resistance and repair.

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