Histopathology of the pulp in primary incisors with deep dentinal caries

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Abstract

The purpose of this study was to assess the histological appearance of the pulp of human primary incisors extracted because of deep, unrestorable caries, and to determine how clinical pulp exposures affected the histological status of the pulp compared to nonexposures. Caries was removed carefully from all teeth after fixation; 24 incisors had pulp exposures, and 29 teeth had no pulp exposures. Histological examination showed normal pulps in 69% of the teeth without pulp exposures, compared to 33% of teeth with exposed pulps ($P < 0.05$). Microabscesses were observed in 33% of cases with pulp exposures, compared to 10% of cases without pulp exposures. In this study, 46 of 53 pulps remained vital in spite of the multiple and deep carious lesions. Teeth without pulp exposures were diagnosed in the treatable category in 20 of 26 cases. (Pediatr Dent 14:1372–75, 1992)

Introduction

The treatment of infants with nursing bottle caries frequently is performed under general anesthesia and the type of pulp therapy is based primarily on absence of clinical and/or radiographic findings and the presence of pulp exposure, including the quality and quantity of pulp bleeding.

As reported by Brännstrom and Lind,[1] pulp inflammation as a response to caries may be noticed even at the stages of early enamel carious lesions.

On the other hand, Langeland and Langeland[2] have shown that deep carious lesions may be found with relatively normal pulps or with a slight increase in chronic inflammatory cells. Reeves and Stanley[3] found that if the distance of the penetrating bacteria and the pulp averaged 1.1 mm or more, pathological lesions in the pulp were insignificant; they also observed that when the carious lesions reached to within 0.5 mm of the pulp, the degree of pathosis increased. It was not until the reparative dentin was invaded by caries that evidence of nontreatable pathosis was observed, such as abscess formation. Up to 79% of permanent teeth with clinical pulp exposures were considered to be in a nontreatable category as confirmed histologically by Seltzer et al.[4] The diagnosis of pulp exposure in that study was based on clinical examination and did not follow complete caries excavation. Trowbridge,[5] in a comprehensive review of the response of the pulp to dental caries, strongly suggested that the pattern of inflammation is determined by the permeability of dentin and the proximity of the carious lesion to the pulp. He further stated that the pulpal dentinal complex reacts to the carious process by forming dentinal sclerosis, resulting in occlusion of the dentinal tubules by mineralization and decreasing dentin permeability. Another defensive reaction is the formation of reparative dentin, isolating the carious process from the pulp. Mjor[6] reviewed the protective effect of reparative dentin; he described the lack of dentinal tubule communication between primary and reparative dentin, decreasing dentin permeability and pulpal damage.

Trowbridge[5] further describes the early inflammatory changes in the pulp, including deposition of collagen and proliferation of small vessels, and accumulation of chronic inflammatory cells which is followed by an acute inflammatory reaction as the lesion approximates the pulp. Progressive accumulation of large numbers of neutrophils results in suppuration that may be diffuse or localized, forming an abscess.

The pulpal dentinal complex response to dental caries in human primary teeth was described by Cohen and Massler[7] as being similar to that seen in permanent teeth; they describe pulpal dentinal reactions to dental caries in primary teeth including reparative dentin formation, a reduction in number and size of odontoblasts with a change of shape, an invasion of the cell-free zone of Weil by round cells, and capillaries under deeper lesions. The body of the pulp showed an increased number of inflammatory cells only under very deep lesions.

Rayner and Southam,[8] on the other hand, concluded that primary tooth pulp responds more rapidly to dental caries than pulp in permanent teeth; they showed that inflammatory changes in the pulp of carious primary teeth can develop when bacteria have penetrated to within 1.8 mm of the pulp.

Agreement between clinical and histological findings in chronic coronal pulpitis in primary teeth with exposed pulps was found in 30 of 37 teeth by Schröder.[9]

None of these studies were related to primary incisors with nursing bottle caries, a rapidly progressing

type of dental caries. The purpose of this study was to assess the histological appearance of the pulp of human primary incisors with deep dentinal caries, and to determine whether a clinical pulp exposure affects the histological status of the pulp.

Materials and Methods

The material consisted of 53 primary incisor teeth extracted under general anesthesia from 22 children ages 20 to 42 months; the teeth were extracted because of advanced rampant caries that was considered unrestorable. Teeth selected for this study were free of clinical and radiographic evidence of pulp pathology. The extracted teeth were placed in formalin-containing bottles following the extraction; apices were not removed so that the histological response of the whole length of the pulp could be assessed.

After formalin fixation, caries was removed carefully from all teeth with a round bur at low speeds; complete caries removal was confirmed by tactile examination with an explorer. During the tactile probing of the dentin, minute pulp exposures were detected in some of the teeth; these teeth were placed in separate vials and formed the group with pulp exposure. A total of 24 incisors had pulp exposures following caries removal, and 29 teeth were included in the group without pulp exposure.

The teeth were prepared for histological examination using 10% EDTA for demineralization. After demineralization, the teeth were trimmed, embedded in paraplast and cut longitudinally to obtain 6 μm-thick sections. The sections were stained with hematoxylin-eosin, and examined with a light microscope.

The histological diagnosis was based on the classification used by Seltzer et al.[4] with slight modifications as follows:

Normal pulp: intact, uninflamed pulp. **Transitional stage:** intact pulp with scattered inflammatory cells. Chronic partial pulpitis: excludes chronic inflammation of the radicular pulp as evaluated by the absence of any accumulation of inflammatory cells. **Chronic partial pulpitis with partial necrosis or with abscess formation:** inflammation limited to the coronal pulp, with necrosis or abscess formation in a small area near the excavated carious lesion or exposed pulp. **Chronic total pulpitis:** the inflammatory process affects the coronal and radicular pulp. **Chronic total pulpitis with partial necrosis or abscess formation:** as in chronic partial pulpitis, but the radicular pulp also is affected. **Total pulp necrosis:** all pulp tissue is necrotic.

These histological categories were classified further in three subgroups, based on the healing potential of the pulp, as follows:

1. **Treatable:** includes the histological diagnosis normal pulp and transitional stage, and is based on the ability of the pulp to heal following caries therapy.
2. **Untreatable:** includes chronic total pulpitis with or without partial necrosis and total necrosis, and requiring radical pulp therapy — either extraction or pulpectomy.
3. **Questionable:** includes teeth histologically diagnosed as chronic partial pulpitis with partial necrosis or micro-abscess, that could be treated with pulpotomy techniques.

A Chi-square test was used for statistical analysis, as described by McCarthy.[10]

Results

The results are presented in the table, which shows the prevalence of the various histological pulp conditions in teeth with and without pulp exposure. Normal pulp or transitional stage (treatable category) was diagnosed in 69% of teeth without pulp exposure, compared to 33% of teeth with exposed pulps.

Chronic partial pulpitis with partial necrosis or microabscess (questionable category) was diagnosed in eight of 24 cases (33%) with pulp exposures compared to three of 29 cases (10%) without pulp exposures. Chronic total pulpitis with or without partial necrosis and total pulp necrosis (nontreatable category) was diagnosed in six teeth without pulp exposure and eight teeth with exposed pulps. As shown in the table, the percentage of teeth with treatable pulp conditions was greater in teeth without pulp exposure. The observed

Table. Distribution of teeth according to histological diagnosis and clinical pulp exposure

Histological Diagnosis and Treatment Category	*Clinical Pulp Exposure*	
	Yes	*No*
Treatable		
Normal pulp	0	6
Transitional stage	8	14
Questionable		
Chronic partial pulpitis with partial necrosis or microabscess	8	3
Nontreatable		
Chronic total pulpitis	0	1
Chronic total pulpitis with partial necrosis	4	2
Total pulp necrosis	4	3
Total	24	29

$X^2 = 7.29$; $P < 0.05$.

frequencies of histological categories (treatable, questionable, nontreatable) between exposed and nonexposed pulps were statistically significant ($P < 0.05$); Chi-square with two degrees of freedom = 7.29.

Figs 1 to 3 are representative examples of the different pulp conditions found. Fig 4 (page 375) shows the only case of iatrogenic pulp exposure as demonstrated histologically.

Discussion

In this study the vast majority of the pulps, 46 of 53 were vital, in spite of the multiple and deep carious lesions present in most of the teeth, including 24 teeth with clinically detectable pulp exposures. This finding must be considered with caution, since teeth with obvious radiographic or clinical findings suggestive of pulp pathology were excluded from the study. The fact that 46 of 53 teeth showed vital pulps without evidence of vacuolization, and the seven teeth diagnosed as pulp necrosis showed histological features of dead cells — and not tissue disintegration — suggests that penetration of fixative was adequate. Seltzer and Bender[11] have suggested poor fixation as a reason for artifact production (such as vacuoles) that could be misinterpreted as microabscesses. The adequate fixation obtained without apex removal could be explained by the extension of the carious process and the thin layer of remaining dentin in most of the teeth, which allowed penetration of the fixative agent. Apices were not removed to enhance fixation because we wanted a view of the whole length of the pulps. The diagnosis of total pulp necrosis in three teeth without clinically detectable pulp exposures was an unexpected finding that can be explained by the high frequency of trauma in primary maxillary incisors. This often results in pulp necrosis, especially in displacement injuries as reported by Andreasen.[12] It is also possible that microscopic pulp exposures were present that could not be detected with the explorer; the method used to detect pulp exposures has its limitations, although it is clinically relevant and is utilized universally. Moreover, Pashley and Pashley[13] have described deep dentin as being more permeable than superficial dentin, because there are a greater number of tubules and increased tubular diameter in dentin closer to the pulp. However, dentin under a carious lesion is less permeable due to the production of dentinal sclerosis and reparative dentin as reported by Mjor.[6]

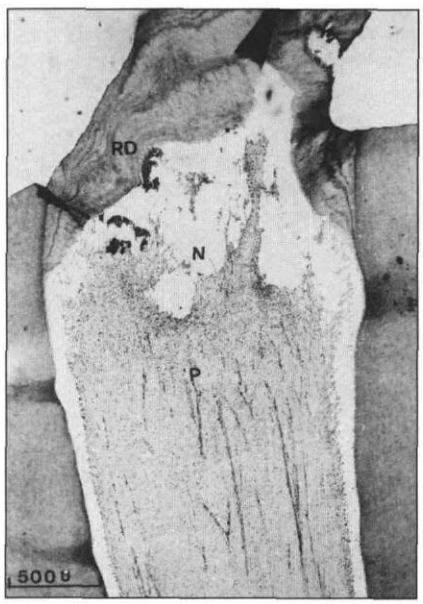

Fig 1. Mesiodistal section of coronal portion of pulp demonstrating incremental layers of reparative dentin (RD) that have been formed under the carious process. The pulp (P) has an area of abscess formation and necrosis (N); inflammatory cells are scattered apically in the pulp (P, Nontreatable category).

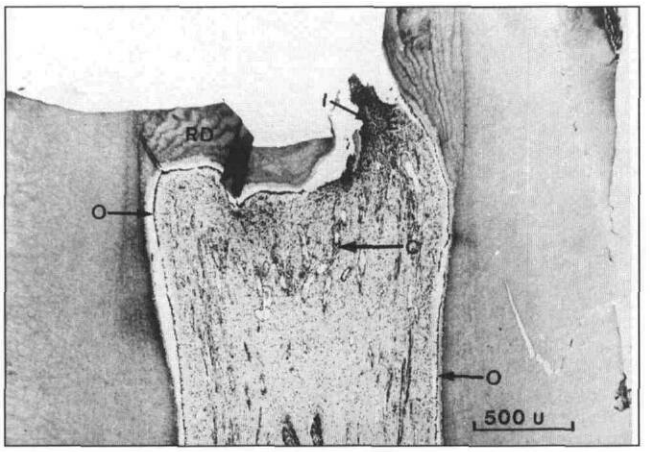

Fig 2. Buccal view of crown showing a very thin (30 µm) primary dentin (PRD) layer remaining following caries removal: a thick reparative dentin (RD) layer has been formed; the predentin (PD) is evident. The pulp (P) has a focus of scattered inflammatory cells in region (X, Treatable category).

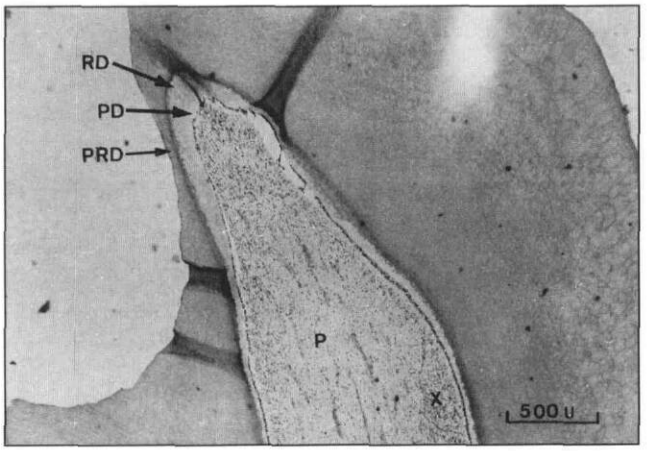

Fig 3. Coronal view showing remaining reparative dentin (RD) and pulp exposure (E) showing inflammatory reaction (I) and many capillaries (C) formed under pulp exposure. The odontoblastic layer (O) is intact (Questionable category).

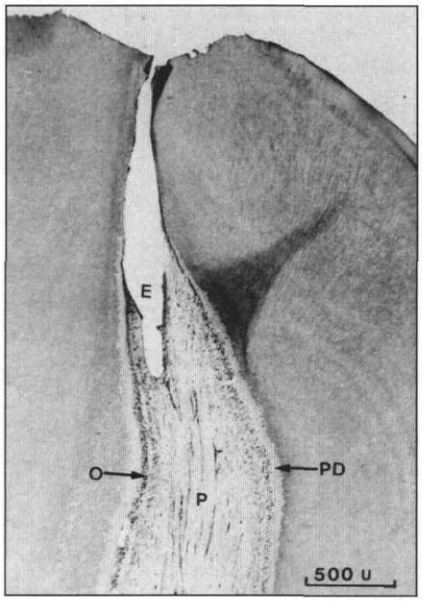

Fig 4. Incisal area showing an iatrogenic pulp exposure (E) caused during tactile examination with the probe. The pulp (P) is relatively normal. Odontoblasts (O) and predentin (PD) are evident.

In this study, caries was removed and pulp exposure was diagnosed following tooth extraction. Since the pulp was fixed, no bleeding was elicited; in the clinical situation, an exposed, nonbleeding pulp would be diagnosed as necrotic; moreover, since the pulps were fixed before caries excavation, the possible effect of the operative procedure on the pulp was eliminated.

Only 69% of the pulps in the nonexposed group were normal or almost intact histologically; this means that 30% of the pulps were in a questionable or nontreatable category. Lin and Langeland[14] state that "whereas the repair capacity of pulp tissue following removal of all carious dentin without pulp exposure is excellent, after carious exposure it is questionable and unpredictable." In our study, 29 teeth had no pulp exposures following caries removal; if we exclude the three teeth that had total pulp necrosis (which can be attributed to trauma inflicted to these teeth), there would remain only three of 26 cases in the nontreatable category, or 11.5% of cases. It is possible that pulps with micro-abscesses could heal, or the small affected area could be walled off and the pulp remain asymptomatic and vital. However, there is no scientific proof that microabscesses can heal following conservative caries therapy.

On the other hand, 33% (eight of 24) of the teeth with clinical pulp exposures were diagnosed as having pulps in the transitional stage (normal pulps with a few scattered inflammatory cells); this finding was surprising, since previous reports by Schröder[9] have shown chronic coronal pulpitis in 30 of 37 teeth with pulp exposures. The difference may be explained by the fact that in our study, all pulp exposures were minimal in size and were detected only by tactile examination with the tip of the probe. The clinician must take every precaution to minimize further trauma during the operative procedures in the presence of pulp pathology due to deep carious lesions. The additional trauma could lead to the establishment of nontreatable pulp conditions.

The results of this study indicate that absence of pulp exposure following caries removal is a good indicator of a normal histological status of the pulp. On the other hand, two-thirds of the teeth with pulp exposures had pulp pathology limited to inflammation of the coronal pulp; therefore, they were good candidates for pulpotomy therapy.

Conclusions

1. Pulps without pulp exposure following caries removal were more likely to be either treatable or questionable as defined in the methodology than were pulps with clinically evident pulp exposures.
2. Two-thirds of cases with pulp exposures (16 of 24) had inflammation limited to the coronal pulp.

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