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Exposed human pulp caused by trauma and/or caries in primary dentition: a histological evaluation

Raslan N, Wetzel WE. Exposed human pulp caused by trauma and/or caries in primary dentition: a histological evaluation. © Blackwell Munksgaard, 2006.

Abstract – The aim of this study was to compare the pulpal reactions after exposure due to trauma and/or caries and to suggest the appropriate treatment options for the management of pulp, in the light of the histological findings in the primary teeth. Fifteen primary maxillary incisors with pulpal exposure were examined with light microscope in a blind study. After the histological evaluation, teeth were divided into three groups for the etiological factors due to the exposure according to the patient's files: trauma group, caries group and caries and trauma group. The inflammatory infiltration was diminished from the pulp chamber towards the apical third of the root canal in all of the examined teeth. Teeth in the trauma group presented fewer inflammatory cells in the root canal in comparison with other groups. It was concluded that the teeth with traumatic pulp exposure were considered as more likely to respond positively to pulpotomy technique. Pulpectomy or extraction seemed to be indicated for the decayed or decayed and traumatically injured teeth.

Nabih Raslan, Willi-Eckhard Wetzel

Department of Pediatric Dentistry, University of Giessen, Giessen, Germany

Key words: dental pulp exposure; tooth, deciduous; trauma

Nabih Raslan, Department of Pediatric Dentistry, Center of Dentistry and Orthodontics, Schlangenzahl 14, D-35392 Giessen Germany
Tel.: +49 641 99 46241
Fax: +49 641 99 46239
e-mail: Nabih.Raslan@dentist.med.uni-giessen.de

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The dental pulp may be exposed due to caries, trauma or both. Its response to the exposure may vary depending on the etiological factor and the depth of the lesions or fracture.

The pulpal dentinal complex response to dental caries in human primary teeth was described by Cohen and Massler (1) as being similar to that seen in permanent teeth. They reported pulpal dentinal reaction to dental caries in primary teeth including reparative dentin formation, a reduction in number and size of odontoblasts with a change of shape. The body of the pulp displayed an increased number of inflammatory cells only under very deep lesions.

Di Nicolo et al. (2) observed that the inflammatory reaction of the pulp of primary molars to the dentinal caries was localized, being more intense beneath carious lesions and less intense at more distant regions, being almost absent in the radicular apical pulp.

In the study of Eidelman et al. (3) of the pulp of human primary maxillary incisors with deep dentinal caries, minute pulp exposures in 24 teeth of 53 tested were detected after careful removal of the caries. They found total pulp necrosis in three teeth without clinically detectable pulp exposures. They suggested that these primary incisors probably had been traumatized in the past several times. Furthermore, pulp inflammation as a response to caries may be found even at the stages of early enamel carious lesion (4).

The pulpal reaction after exposure due to caries is also described by Çalşkan et al. (5). Four permanent mandibular first molars with pulp polyps after complete coronal destruction by caries were histologically examined. The surface of the polypoid outgrowth in all four cases showed evidence of epithelialization. A dense infiltration was seen in the coronal pulps of all teeth. The middle and apical

third of radicular pulp tissue beneath a calcified barrier in three teeth was generally less vascular and more fibrotic, with absence of inflammatory cells.

On the other hand, injury resulting in an exposure of the pulp in preschool children is probably less prevalent than a pulp exposed by caries.

According to the recent study by Kramer et al. (6) of the prevalence of dental trauma in preschool children, the most common type of injury was enamel fracture (found in 75.5% of children); the enamel- dentin fracture with pulp exposure was uncommon (0.3%). Another study reported complicated crown fracture in 9.6% of children aged 0–3 years (7).

In a study of 138 primary incisors that had been exposed by trauma, one tooth, which sustained midcoronal fracture (involved exposing dental pulp), showed no correlation between clinical/radiographical observations and the histopathological changes in the pulp and periapical tissue (8).

Recent histological study of pulps exposed by induced crown fracture in monkeys by Cvek et al. (9) has reported a high frequency of pulpal healing after partial pulpotomy (PP). They found that the depth of inflammatory infiltration in the pulp ranged from 0.8 to 2.2 mm (mean 1.5 mm) 168 h after exposure.

In the study of Croll et al. (8) one tooth exhibited traumatic pulpal exposure in which there was a dense concentration of neutrophilic leukocytes in the pulpal horn extending through the coronal third; but no inflammatory cells were present in the radicular pulp and none in the foramen area.

Harrán-Ponce et al. (10) produced crown fracture with pulpal exposure in the teeth of adult mongrel dogs under general anesthesia after grinding small grooves at the middle third of the crown of these teeth. They showed that the accumulation of impacted remnants and polymorphonuclear leukocytes (PMN) on the surface of the exposed pulp favors the formation of abscess and pus. This situation is enhanced by the presence of bacteria on the surface of the dentin due to plaque. The odontoblastic layer, located close to the exposed pulp was totally destroyed or reduced to just a few cells. The inflammatory infiltration consisted mainly of chronic cells. Nevertheless, some neutrophils were still observed. The amount of inflammatory infiltrate was reduced towards the apex, the subsequent area being free of inflammatory cells.

Çalşkan et al. (5) examined the pulp tissue of one young permanent incisor with complicated crown-root fracture. The inflammation was in the coronal part, but the cervical radicular pulp tissue appeared normal with dilated functioning blood vessels.

Except the one case in the study by Croll et al. no histological report of human pulp reaction to exposure after complicated crown fracture in primary teeth has been published.

Among the various options for an injured primary incisor with complicated crown fracture PP has the greatest advantage. The procedure is quick and easy to perform. PP maintains the natural tooth color, preserves tooth structure for better retention of restoration, and is not expected to affect the permanent successor. Furthermore, when a tooth has undergone PP, it is expected to maintain its vitality and continue its root development. This mode of treatment is widely used in injured and decayed permanent dentition (11–13) and less so in primary teeth. Only two reports of PP have been published. These include reports by Ram and Holan (14) and by Kupietzky and Holan (15) with follow-up of 3 months and 2 years, respectively.

Our study was designed to examine the histological changes and its sequelae on the healing potential of dental pulp of complicated crown-fractured primary teeth and pulps exposed due to early childhood caries.

Materials and methods

The sample was composed of 15 primary maxillary incisors from 15 children between the ages of 2 and 4 years with clinical evidence of pulp exposure and without findings of mobility or displacement of the teeth. Extraction took place because of lack of cooperation from the children to perform other treatments successfully (16, 17).

Indicated radiographs were taken when possible according to the guidelines, which are designed to avoid unnecessary exposure to X-ray radiation. Following extraction, five teeth were fixed in 4% neutral buffered formalin. After fixation, the teeth were decalcified in (Shandon Decalcifier, Frankfurt/Main, Germany) a rapid hydrochloric acid decalcifier for approximately 12 h. It also decalcified thin pieces of teeth without damage to soft tissues or loss of staining properties. After decalcification the endpoint was using X-ray tested.

These five teeth were embedded in paraffin and thereafter sectioned serially. Sections were cut parallel to the long axis of the tooth to a thickness of 5–10 μm and stained with Hematoxilin & Eosin, Masson–Goldner and PAS-Reaction.

Because of further plans of additional application of electron microscope in another study, it was necessary to prepare the sections from the remaining 10 teeth in the following appropriate manner that is suitable for the two microscopes; teeth were fixed in Karnovsky's Glutaraldehyde, decalcified in

0.5 mol l^{-1} EDTA and sectioned to four parts (crown, coronal, middle, and apical third of the root) and embedded in a Spurr's Low Viscosity embedding mixture (18). This is recommended because of its excellent penetration qualities, and provides good and rapid infiltration of tissues. Sections were cut parallel to the long axis of the tooth to a thickness of 1 μm and stained with toluidine blue O/Pyronin.

Sections of all 15 teeth were examined and photographed with a light microscope (Olympus Vanox T/AH-2, Tokio, Japan). Each tooth was studied in the four parts (Pulp chamber, coronal, middle, and apical third of the root canal). In each part the inflammatory infiltration, odontoblastic layer, nerve fibers bundles and presence of epithelia in the superficial area of the tissue was assessed.

The degree of pulpal inflammation was graded on a 0–3 scale: (0) = normal, (1) = intact pulp with scattered inflammatory cells, (2) = moderate inflammatory infiltration, (3) = intense, highly inflammatory infiltration.

The quality of the odontoblastic layer was based on a 0–3 scale: 0 = no odontoblasts, 1 = odontoblast cells, 2 = partial odontoblastic layer, 3 = odontoblastic layer.

The appearance of the nerve fibers was rated as follows: 0 = no nerve fibers and 1 = nerve fibers.

Finally, the histological diagnostic classification used by Seltzer et al. (19) and by Eidelman et al. (3) and modified by Di Nicolo et al. (2) with some modification is as follows:

Normal pulp: intact, uninflammed pulp

Transitional stage: intact pulp with scattered inflammatory cells

Partial pulpitis: inflammation limited to the coronal pulp

Total pulpitis: the inflammatory process affects the coronal radicular pulp

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 partial polpotomy therapy.
2. Untreatable: includes total pulpitis and total necrosis.
3. Questionable: includes partial pulpitis that could be treated with pulpotomy techniques.

The sections were studied separately by two observers, a dentist with experience in histological examination and a histologist, under blind conditions. Following the histological examination, the teeth have been distinguished in the following groups according to the patient's files (Table 1).

Table 1. Classification of human primary teeth

Group	*n*
Trauma	5
Caries	6
Trauma and caries	4
Total	15

Results

The results are presented in Tables 2–5.

Pulps exposed by crown fractures

The changes in the pulp, which extend to the middle and/or apical third from the exposed surface, were characterized by hemorrhage and damage to the odontoblastic layer (Fig. 1, Tables 2 and 3). Polymorphonuclear leukocytes were seen in

Table 2. Depth of inflammatory reactions: 0 = normal, 1 = intact pulp with scattered inflammatory cells, 2 = moderate inflammatory infiltration, 3 = intense, highly inflammatory infiltration

	Trauma group					Caries group						Caries and trauma			
Case	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Pulp chamber	3	2	2	3	2	3	3	3	3	3	3	3	2	3	3
Coronal third	1	2	1	2	2	3	2	2	3	2	3	3	3	3	2
Middle third	1	2	1	2	2	2	2	2	1	1	3	2	3	1	2
Apical third	0	0	1	1	1	0	1	3	1	1	3	1	2	0	1
Interval (days)	1	2	2	60	7							28	7	2	4

Table 3. Presence of odontoblastic cells/layer: 0 = no cells or layer, 1 = few cells, 2 = layer and cells, 3 = normal layer

	Trauma group					Caries group						Caries and trauma			
Case	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Pulp chamber	1	1	1	0	0	0	0	1	0	2	0	0	1	2	1
Coronal third	3	2	2	1	2	1	1	1	1	2	0	1	3	2	3
Middle third	1	2	2	1	3	1	1	1	1	3	0	1	3	2	2
Apical third	1	1	1	2	1	1	1	1	1	2	0	1	1	1	2

Table 4. Nerves fibers bundles: 0 = no, 1 = yes

	Trauma group					Caries group						Caries and trauma			
Case	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Pulp chamber	1	0	1	0	0	0	1	1	0	1	0	0	0	0	1
Coronal third	1	1	1	0	1	0	1	1	1	1	0	1	1	1	1
Middle third	1	1	1	1	1	1	1	1	1	1	0	1	1	1	1
Apical third	1	1	1	1	1	1	1	1	1	1	0	1	1	1	1

Table 5. Epithelium: 0: no epithelium, 1 = some epithelia cells, 2 = epithelium

	Trauma group					Caries group					Caries and trauma			
Case	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Pulp chamber	0	0	0	2	0	2	0	0	0	0	2	0	0	1	0
Coronal third	0	0	0	0	0	0	0	0	0	0	2	0	0	0	0
Middle third	0	0	0	0	0	0	0	0	0	0	2	0	0	0	0
Apical third	0	0	0	0	0	0	0	0	0	0	2	0	0	0	0

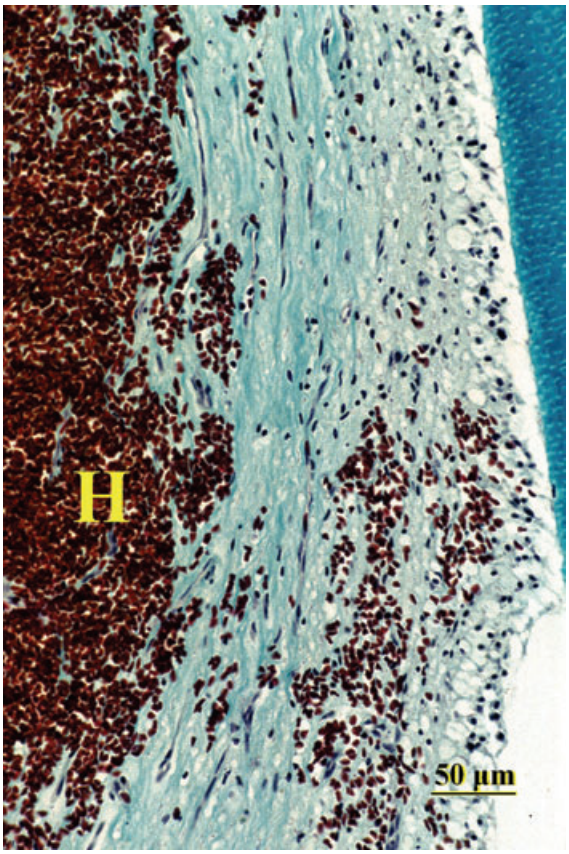

Fig. 1. Hemorrage (H) in the coronal third of the pulp due to trauma (Masson–Goldner).

the four pulps, and, in one of these, a few such cells were observed in the apical third.

In two of the teeth, the pulpal surface was entirely covered with fibrin. One tooth, which was extracted 2 months after trauma, exhibited an epithelium-covered-surface.

In the remaining two teeth, longitudinal fractures were associated with severe infiltration of inflammatory cells and necrotic tissue in the adjacent pulp; whereas on the opposite side of the pulp, there were less inflammatory cells. The pulpal surface was entirely covered with necrotic tissue; a barrier of plasma cells was in the middle third of the root canal separating the necrotic part of the pulp from the inflamed part (Fig. 2).

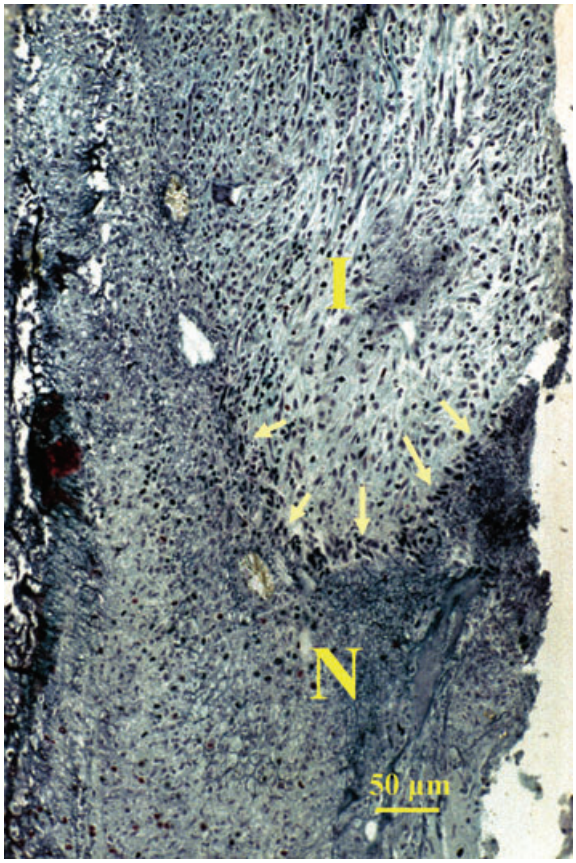

Fig. 2. Plasma cells (arrows) separating the necrotic part (N) of the pulp from the inflamed part (I) in the middle third of root canal (Masson–Goldner).

A comparison between the extent of inflammatory changes in the pulp after exposure of one or 2 days and then after 2 months showed structural differences; odontoblastic layer/cells were not found in the pulp tissue two months after traumatic injury but a stratified epithelium was covering the exposed pulp (Tables 4 and 5).

Pulps exposed due to caries

Between impacted debris and the surface of the exposed pulp, accumulations of polymorphonuclear leukocytes were found. Of the six polyps, two were epithelized (Fig. 3). The non-epithelized polyps were covered with a fibrin layer containing polymorphs. In the presence of insufficient epithelial-coverage of underlying pulp, bacteria were found invading the pulpal tissue (Fig. 4). In five cases, the depth of inflammatory reactions extend to the apical third of the radicular pulp tissue (Table 2), only one case showed no inflammatory reaction in the apical third. Retrogressive changes such as dentikel and fibrosis were found in this group (Fig. 5). One tooth has shown granulation tissue with a mix of chronic

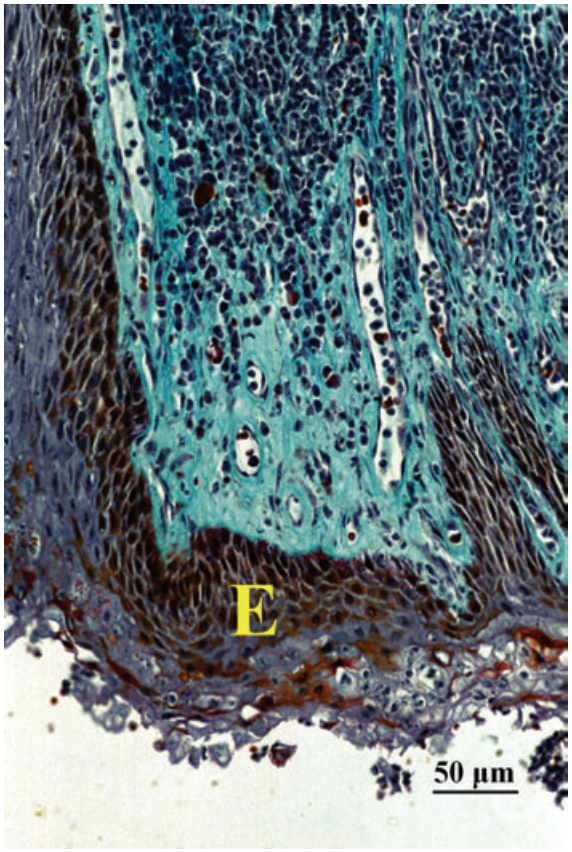

Fig. 3. Parakeratinized stratified squamous epithelium (E) on the surface of a pulp polyp with slight inflammatory cells directly underneath (Masson–Goldner).

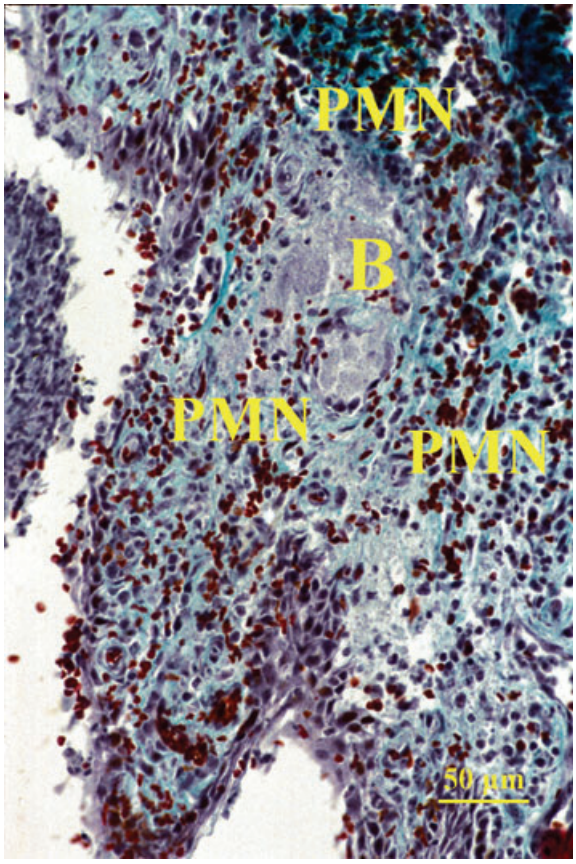

Fig. 4. Accumulation of PMN Leukocytes surrounds the Bacteria (B), which has invaded the pulpal tissue in a non-covered area with Epithelia (Masson–Goldner).

inflammatory cells, containing mainly plasma cells and lymphocytes, also covered with parakeratinized stratified squamous epithelium (Fig. 6).

Pulps exposed by crown fractures with caries involvement

In this group, polyps were covered with granulation tissue or with a fibrin layer containing polymorphs (Figs 7–10). In one case, individual epithelium cells are on and at the surface of the polyp tissue (Fig. 11). Mast cells were seen in three of the four pulps, and one tooth showed a denticle in the apical area. Nerve fiber bundles and odontoblasts cells were found in all cases with some damage (Tables 3 and 4).

According to the histological diagnostics, teeth in both the Caries and the Caries & Trauma groups were seen as 'Untreatable'. However two teeth with complicated crown fractures were considered as 'Questionable' and two as 'Untreatable' (Table 6). The healing potential of the pulp of these two teeth in the trauma group is 'Questionable' but by better compliance they could had been treated with pulpotomy techniques.

Discussion

In the evaluation of complicated crown fractured teeth in primary dentition, no distinctions were made between carious and none carious teeth. This may lead to false prognosis and treatment because of the possible pulpal alterations due to caries. In this study four of nine complicated fractured teeth were already carious, which may reflect too on the etiological factors of the crown fracture of primary incisors.

Moreover, in the trauma situation, there is often the risk that the most obvious trauma entity will be diagnosed (e.g. crown fracture), while a less obvious diagnosis (e.g. luxation injury) might be overlooked. In the case of crown fracture, this may lead to a prognosis after injury based exclusively on the analysis of crown fracture alone rather than a crown fracture in combination with a luxation injury. Robertson et al. (20) found that luxation injuries concomitant to crown fracture have a significant deleterious effect on pulpal prognosis with respect to both pulpal necrosis and pulp canal obliteration. In our study there were neither clinical

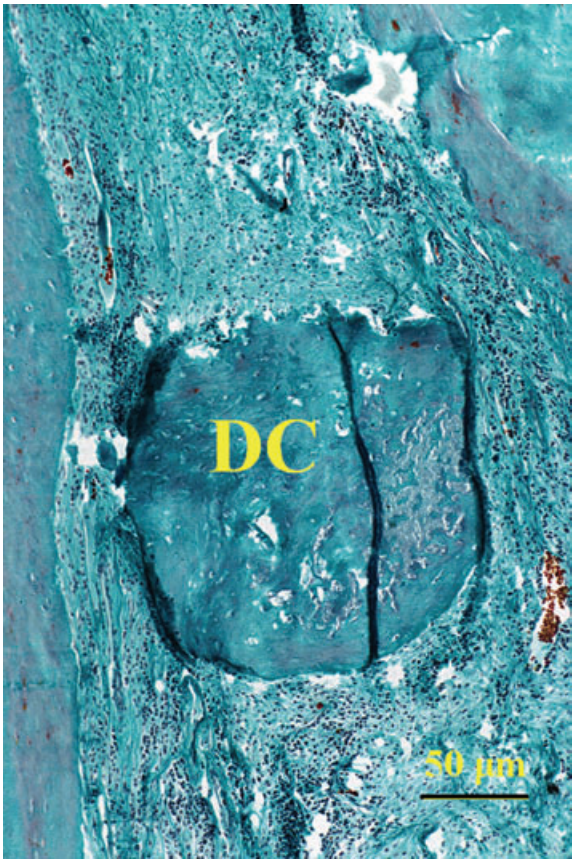

Fig. 5. Denticle (DC) in the coronal third of the root canal (Masson–Goldner).

nor radiographic signs of luxation injuries in the five crown fractured teeth.

In spite of the individual responses of the pulps to exposure, Trauma group showed fewer inflammatory reactions and no retrogressive pulp changes in comparison to the Caries Group and Caries & Trauma Group. In two teeth of the Trauma Group, the inflammation was limited to the pulp chamber with scattered inflammatory cells in the radicular pulp 1 and 2 days after trauma. Similar findings were found after 7 days in experimentally exposed primate pulps (21, 22). On the other hand, the case with complicated crown fracture 60 days after trauma showed moderate inflammatory infiltration deeper in the root canal. In this case the elapsed-time after injury was long enough that a thick layer of epithelium could grow over pulp polyp. The time here appears to be an important factor, as similar tissue reactions were found, since the pulp tissue exposed to the oral environment and the accumulation of impacted remains and bacteria on the surface of polyp and dentin (10).

In the case of complicated crown- root fracture, the depth of inflammatory reaction increased to the apical third of the root canal. Similar findings were seen in the presence of longitudinal fractures in the study by Cvek et al. (21).

In our study, inflammatory infiltration in the caries group occurred relatively more than that in the other two groups. Also occurring in one tooth was granulation tissue with epithelium replacing the pulpal tissue, in which no more nerve fibers or odontoblast cells were found.

Inflammatory infiltration in the middle third of the root canal in all of the teeth was present in the Caries and Caries & Trauma groups. The time elapsed after injury in the traumatized carious teeth was not important because of the early inflammatory infiltration due to the caries. These finding supports the results reported in previous studies on possibility of pulp inflammation as a response to caries that may be found even at the early stages of carious lesion (3, 4). This supports the presence of the retrogressive changes such as denticles and/or fibrosis in the pulp tissue of two cases of these groups.

Lin et al. (23) have observed structurally intact nerve fibers in the remaining inflamed root pulp despite the presence of the extensive periapical

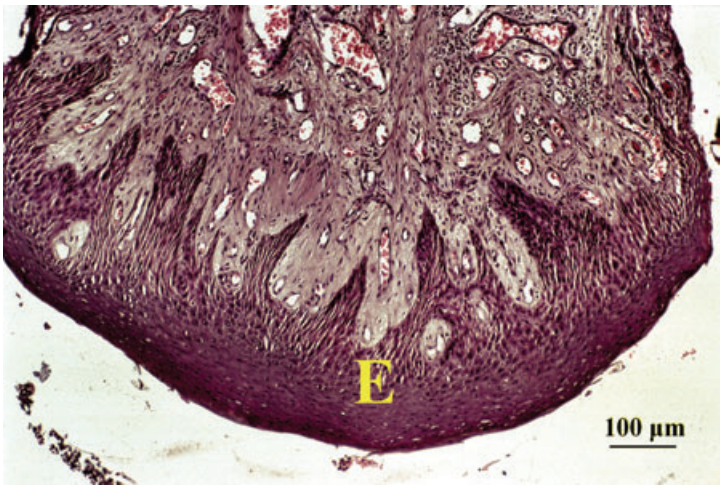

Fig. 6. Parakeratinized stratified squamous epithelium (E) covered the granulation tissue in a pseudo polyp (H & E).

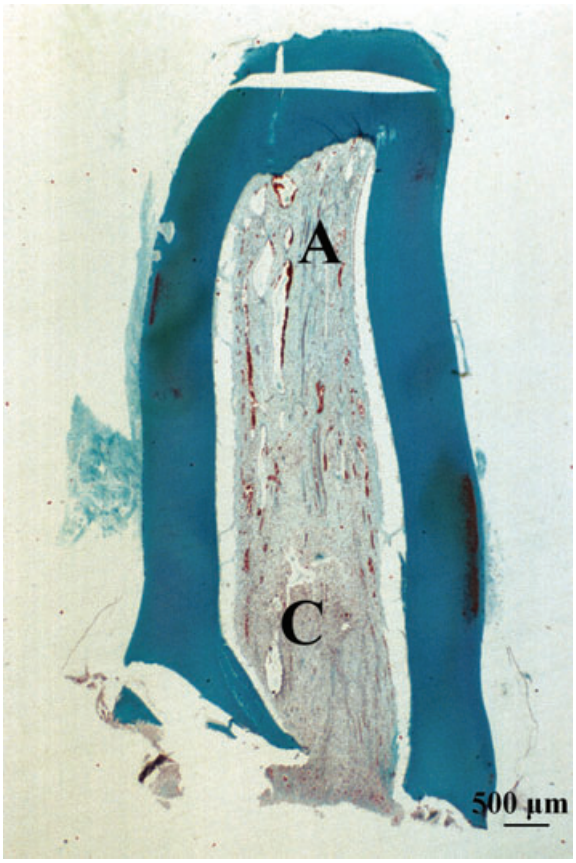

Fig. 7. Pulpal reaction following complicated crown fracture in a carious maxillary primary incisor: moderate inflammatory changes are present in the coronal part of the pulp while apical part shows only a minor inflammatory infiltration (Masson–Goldner).

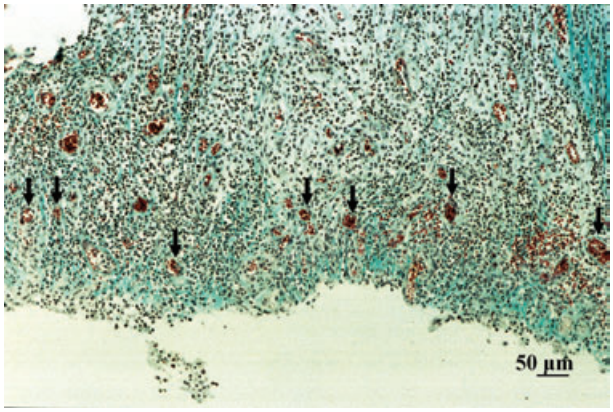

Fig. 8. A higher power of Fig. 7, several thin-walled capillary blood vessels (arrows) growing into damaged tissue associated with a perforation of the granulation tissue (Masson–Goldner).

lesion and necrosis in the coronal part of the root canal. On another hand ÖzÇelik et al. (24) have found prominence of degenerating myelinated nerve fibers 20 days after trauma in one case

Fig. 9. One week after trauna shows the pulp proliferation (Toluidine blue).

examined by light microscope. In our study nerve fibers were present in most of the teeth.

The pulp polyps of deciduous teeth are more likely to be covered with epithelium than those of permanent teeth (25). Southam and Hodson (26) have observed that 82% of the studied polyps were epithelized. The results of our study show only two teeth in which polyps were covered with epithelium. Çalşkan et al. (27) have suggested that the epithelial layer over the surface of the polyp protects the underlying granulation tissue from the harmful effects that will disturb wound healing in the oral cavity. These defensive reactions probably contribute to the inherent healing potential of young dental pulp in which hyperplastic pulpitis develops (5). This explains the relatively slight inflammatory infiltration in a narrow layer directly under the covered epithelia observed in our study. The epithelium protection was not absolutely optimal but rather loose in the marginal areas of the polyp, consequently allowing bacteria to repeatedly invade through these free areas inside the polyp. This resulted in acute inflammatory reaction with PMN cells. In the one case in Caries group that was diagnosed as pseudo- pulp- polyp, it was a presence of epithelium in combination with the

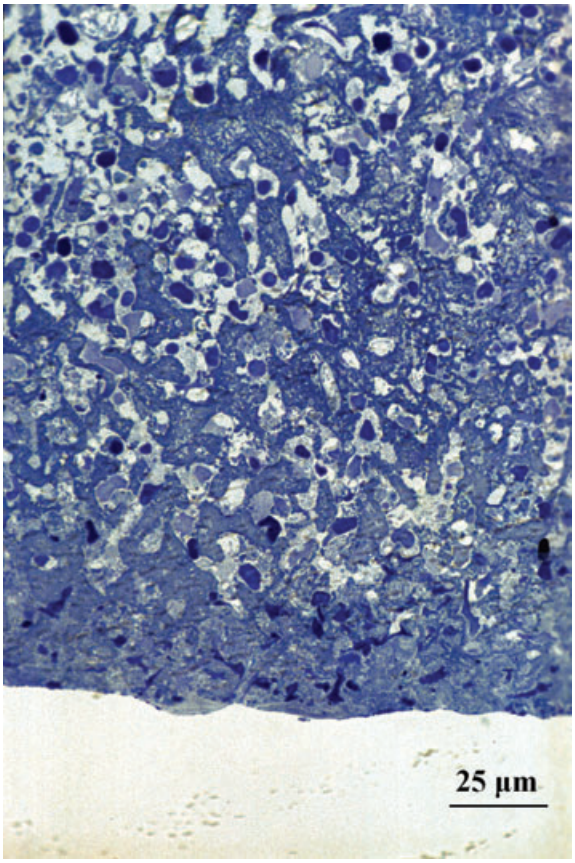

Fig. 10. A higher power of Fig. 9, layer of fibrin on the surface of hyperplastic pulpa granulation tissue (Toluidine blue).

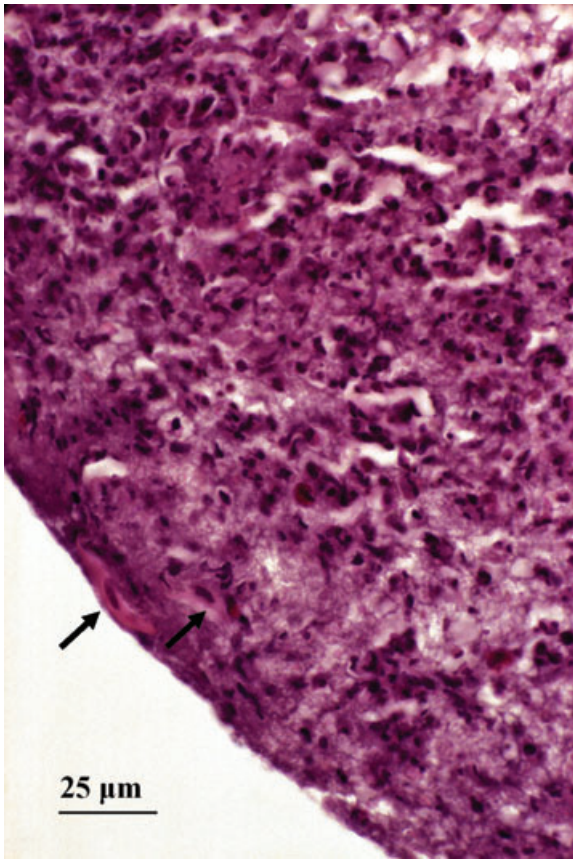

Fig. 11. A part from the surface of the polyp: epithelial cells (arrows) were found in and on the fibrin exudation (H & E).

granulation tissue in the root canal up to the apex replacing the pulpal tissue.

Çalşkan et al. (5) have suggested that the irregular calcification and reactive fibrosis frequently tended to separate the grossly inflamed area in the polyp from the middle and/or apical portion of the pulp, which remained apparently normal. It was likely that this process was promoting intrinsic defense of the pulp. We found that the spread of the inflammatory infiltration was no more to be hindered in the two teeth with denticles or fibrosis in comparison to the other teeth. These denticles and fibrosis count as retrogressive changes of the pulp due to the carious lesions affecting the teeth over a long period of time (28).

In the examined teeth in our study, pulps were largely exposed and the remaining length from the pulp chamber is indeed shorter than 2 mm. Therefore a PP is by no means possible here. On the other hand, we have found neither normal pulp nor transitional stage in all examined teeth. Chronic partial pulpitis with scattered inflammatory cells in the root canal was histological diagnosed in two of the five cases from the trauma group. In such cases cervical pulpotomy- if possible- as conservative treatment will be recommended.

Table 6. Distribution of teeth according to the histological diagnostic and treatment category

Histological diagnostic and treatment category	Trauma group	Caries group	Caries and trauma
Treatable normal pulp transitional stage	0	0	0
Questionable partial pulpitis	2	0	0
Untreatable total pulpitis total necrosis	3	6	4
Total	5	6	4

The treatment of choice for the remaining teeth may be pulpectomy or the extraction of the tooth.

Conclusions

1. The inflammatory infiltration as a response to the traumatically exposed primary teeth may be localized in the pulp chamber with scattered inflammatory cells in the root canal.
2. Caries not only gives rise to a weakening in primary teeth, it heightens the susceptibility to the occurrence of complicated crown fracture, induces chronic pulpal inflammation and produces retrospective changes. Conservative treatment, such as pulpotomy is therefore not indicated.

3. Primary teeth with pulp exposure due to the caries exhibit advanced inflammation and retrospective changes may result in pulp necrosis.
4. A cervical pulpotomy of primary incisors with exclusively large traumatic pulp exposure will be recommended.
5. Pulpectomy or extraction may be indicated for the carious primary incisors with pulpal exposure.

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