Reaction of the exposed pulp to Dycal treatment

Leif Tronstad, cand.odont., lic.odont., dr.odont., Oslo, Norway

DENTAL INSTITUTE OF EXPERIMENTAL RESEARCH, UNIVERSITY OF OSLO

The reaction of the exposed pulp in monkey teeth to treatment with Dycal or a paste of a dentin bridge at the border zone between the necrotic and vital tissue, and calcium hydroxide, the well-known reaction was observed; i.e., necrosis of a limited area of the pulp and a mild inflammation in the adjacent vital tissue, formation of a dentin bridge at the border zone between the necrotic and vital tissue, and healing of the inflammation. In the teeth treated with Dycal, an area of the pulp subjacent to the capping material initially contained only few and altered cells. However, the tissue of this area did not become necrotic, but was infiltrated by macrophages and giant cells, and after some 30 days, it was reorganized. Formation of a dentin bridge usually occurred in the Dycal-treated pulps, not at some distance from the exposure site as in the teeth treated with calcium hydroxide, but directly at the Dycal-pulp interface.

A number of hard-setting, calcium hydroxide–containing materials have become commercially available. These products have more desirable physical properties than a paste of pure calcium hydroxide mixed with water, and, as a rule, they are also biologically acceptable when used as base materials.[1] One such material which also appears to be suitable as a pulp-capping agent is Dycal (L. D. Caulk Co., Milford, Delaware).[2-5] It is known that the alkaline effect of this material is lower than that of pure calcium hydroxide,[6] and preliminary investigations have indicated that the reaction of the exposed pulp to Dycal in some respect differs from the well-known and characteristic reaction of the pulp to calcium hydroxide.[4] Because of the favorable physical properties of the Dycal paste, a detailed study of the healing events in the exposed pulp after treatment with this material was considered to be of interest. For comparison, the exposed pulp of some teeth was capped with a paste of pure calcium hydroxide and water.

MATERIALS AND METHODS

The following materials, bought on the open market, were included in the study: (1) Dycal, which is a two-paste compound consisting of a base (titanium

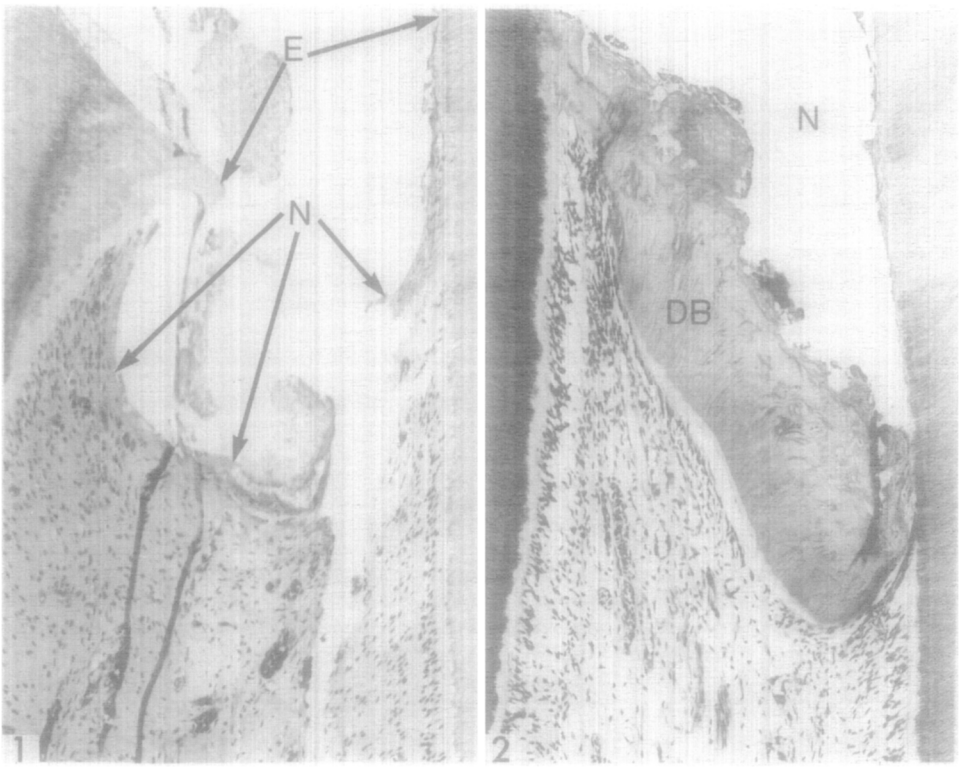


Fig. 1. Pulp capping with calcium hydroxide, 8 days. A well-delimited area of the pulp subjacent to the exposure site *(E)* has become necrotic *(N)*. A few scattered inflammatory cells are present in the vital tissue next to the necrotic area. (Hematoxylin and eosin stain. Magnification, ×100.)

Fig. 2. Pulp capping with calcium hydroxide, 82 days. A dentin bridge *(DB)* has formed at the border zone between necrotic *(N)* and vital pulp tissue. Cellular inclusions are seen in the bridge. The pulp is free of inflammation. (Hematoxylin and eosin stain. Magnification, ×100.)

dioxide 56.7 per cent in a glycol salicylate with a pigment) and a catalyst (calcium hydroxide 53.5 per cent, zinc oxide 9.7 per cent in ethyltoluene sulfonamide).[2] (2) Calcium hydroxide G. R. (E. Merck A. G., Darmstadt, Germany).

Sixty-four teeth in young monkeys were used in the experiments. Class I or V cavities were prepared in the teeth with the use of an air turbine with water spray. The pulp was exposed with a No. 2 round bur in a conventional engine at low speed. Bleeding from the pulp was controlled by means of cotton pellets. In forty-six teeth, Dycal was used as the pulp-capping agent, and in eighteen teeth a paste of calcium hydroxide and water was applied. The cavities were then filled with amalgam.

After observation periods of 48 hours, 8 days, 14 days, 30 days, and 82 days the animals were killed. Table I shows the detailed distribution of the material. The jaws were dissected free, and the teeth were separated from the jaws by means of a Gillings-Hamco thin-sectioning machine and immediately placed in 10 per cent neutral-buffered formalin for 48 to 72 hours. The teeth were de-

Table I. Distribution of the experimental material

Capping material	*No. of teeth*	*Observation periods*				
		48 hr.	*8 days*	*14 days*	*30 days*	*82 days*
Calcium hydroxide	18		9			9
Dycal	46	7	8	7	7	17

Table II. State of the pulp 82 days after capping

Capping material	*Hard-tissue formation*	*No. of teeth*	*Prognosis favorable**	*Prognosis uncertain†*	*Pulpal necrosis*
Calcium hydroxide	Complete bridge at a distance from exposure site	7	7		
	Incomplete bridge at a distance from exposure site	2		1	1
		9	7	1	1
Dycal	Complete bridge at the exposure site	11	11		
	Incomplete bridge at the exposure site	4	2	1	1
	Incomplete bridge at a distance from exposure site	2		2	
		17	13	3	1

*The pulp is free of inflammation, or there are only a few lymphocytes in the area of the lesion.

†A chronic inflammation persists in the lesion area of the pulp.

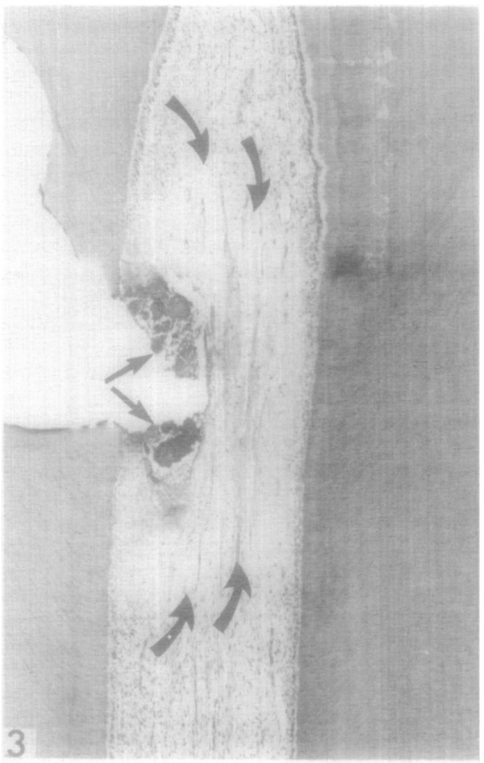
mineralized in 5.2 per cent nitric acid, and the end point of demineralization was controlled radiographically.[7] The teeth were then embedded in paraffin and serially sectioned at 4 microns. The sections were stained with hematoxylin and eosin or Van Gieson's stain.

RESULTS

Treatment with calcium hydroxide/water

Eight days. A well-delimited area of the pulp subjacent to the exposure site had become necrotic (Fig. 1). The rest of the pulp was vital, and in most teeth only a few inflammatory cells were seen in the tissue next to the necrotic area. In the pulp of two teeth, however, a severe inflammatory reaction with abscess formation had occurred. A narrow dense zone was often seen at the interface between the necrotic and vital tissue.

Eighty-two days. An apparent complete dentin bridge had formed at the interface between the necrotic area of the pulp and the vital pulp tissue in most teeth (Fig. 2; Table II). Inclusions of cells and occasionally of capillaries were seen in the newly formed hard tissue. The pulp of these teeth was free of inflammation, or only a few scattered lymphocytes were seen. Of the remaining two teeth, one was characterized by a moderate chronic inflammation in an area of the pulp subjacent to a defective dentin bridge. The pulp of the other tooth was totally necrotic, but with evidence of hard-tissue formation at some distance from the exposure site.



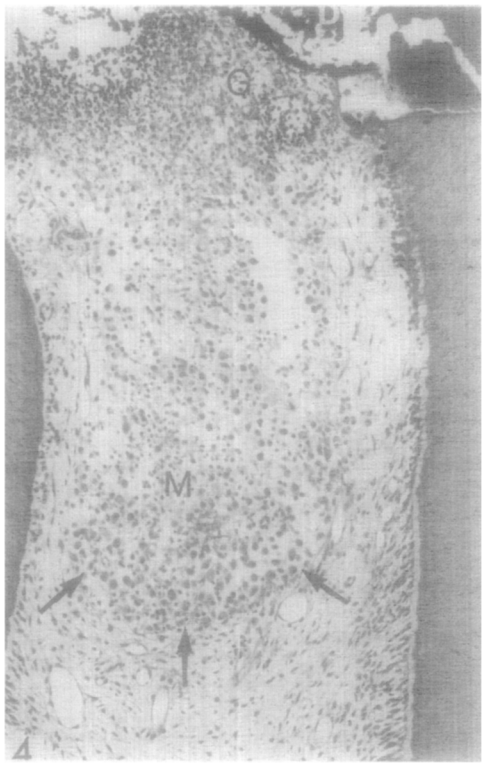


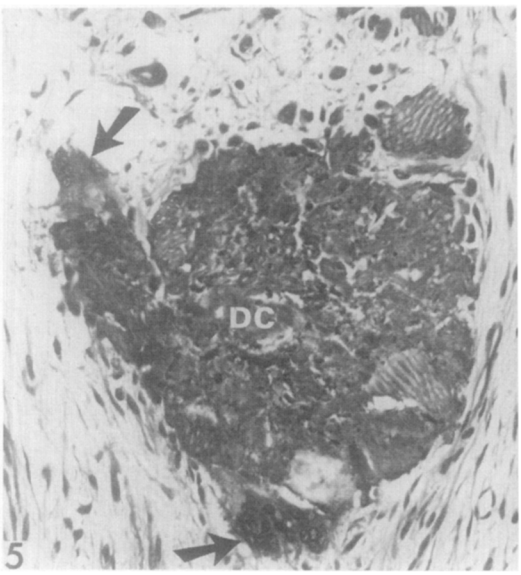
Fig. 3. Pulp capping with Dycal, 48 hours. An accumulation of granulocytes *(straight arrows)* is observed subjacent to the exposure site. In an area of the pulp next to the inflammatory focus, the number of cells is strongly reduced *(curved arrows)*. (Hematoxylin and eosin stain. Magnification, ×100).

Fig. 4. Pulp capping with Dycal, 8 days. The tissue in contact with the capping material *(D)* is vital, but infiltrated with granulocytes *(G)*. In the subjacent tissue, macrophages *(M)* in great number are present. The area of reaction is well delimited *(arrows)*, and in the rest of the pulp no changes are evident. (Hematoxylin and eosin stain. Magnification, ×100.)

Treatment with Dycal

Forty-eight hours. A uniform reaction had occurred in an area of the pulp subjacent to the exposure site in all teeth (Fig. 3). A small accumulation of granulocytes was seen in the tissue contacting the capping material. In the tissue adjacent to the inflammatory focus, the number of cells was strongly reduced as compared to the rest of the pulp tissue. The remaining cells in this area seemed to be altered, and usually only their nuclei were discernible. Other tissue components, such as capillaries and fibers, could be recognized. The size of the area of reaction varied in the different teeth, but it was always well delimited. In the adjoining pulp tissue no changes were evident.

Eight days. A characteristic reaction was observed in an area of the pulp subjacent to the exposure (Fig. 4). The tissue in contact with Dycal was vital but, to a varying degree, infiltrated by granulocytes. In the adjacent tissue, macrophages in great numbers were observed. In addition, giant cells were present, often in contact with dentin chips which had been introduced into the pulp



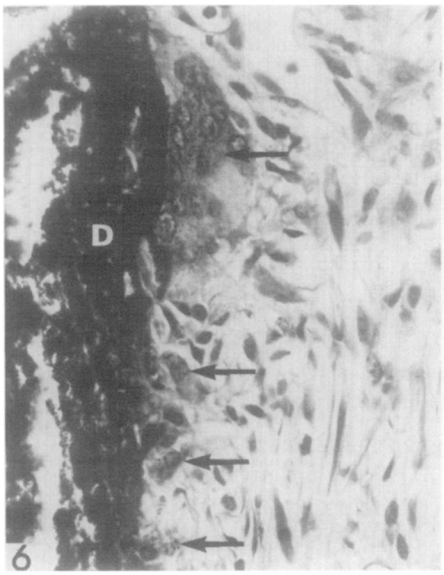
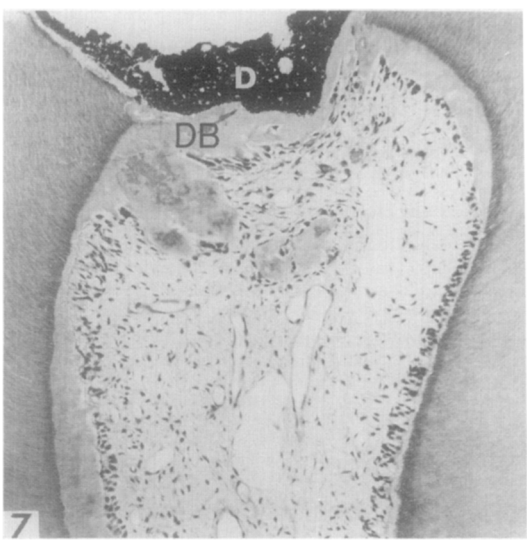


Fig. 5. Pulp capping with Dycal, 8 days. Many dentin chips *(DC)*, which appear to be "cemented" together, are seen. Giant cells *(arrows)* adhere to the chips, and macrophages are present in the tissue. (Hematoxylin and eosin stain. Magnification, ×250.)

Fig. 6. Pulp capping with Dycal, 14 days. The tissue in contact with the Dycal paste *(D)* is vital. Macrophages and giant cells, some containing Dycal particles *(arrows)*, are seen in the tissue. (Hematoxylin and eosin stain. Magnification, ×250.)



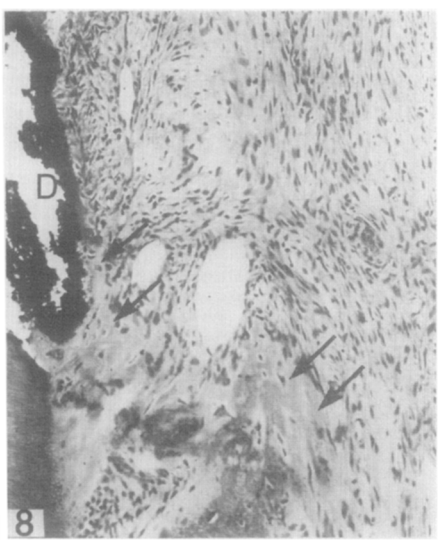


Fig. 7. Pulp capping with Dycal, 30 days. A fragile, but apparently complete dentin bridge *(DB)* has formed at the interface between the capping material *(D)* and the pulp. The pulp is free of inflammation. (Hematoxylin and eosin stain. Magnification, ×100.)

Fig. 8. Pulp capping with Dycal, 30 days. Hard-tissue formation *(arrows)* is evident at the Dycal *(D)*-pulp interface and at some distance from the exposure site. However, a complete bridge has not formed. A slight chronic inflammation persists in the lesion area of the pulp. (Hematoxylin and eosin stain. Magnification, ×100.)

tissue (Fig. 5). The area of reaction was well delimited, and in the surrounding tissue no microscopic changes were evident.

Fourteen days. At this time, almost no granulocytes were seen in the pulp. The common observation was a slight to moderate inflammation dominated by macrophages and giant cells in the affected area of the pulp (Fig. 6). Many macrophages contained Dycal paste, and giant cells were seen next to the capping material at the exposure site (Fig. 6). Scant formation of hard tissue had occurred in one instance at the Dycal-pulp interface. In one tooth, the pulp was totally necrotic, and Dycal particles were seen in remnants of necrotic pulp tissue, in the periapical tissue, and in the periodontium as far coronally as halfway between the apex and the gingival margin.

Thirty days. In three teeth the pulp was free of inflammation (Fig. 7), whereas in four teeth a slight chronic inflammation was observed in the affected area of the pulp (Fig. 8). Dentin formation was evident at the Dycal-pulp interface in all teeth, but a complete bridge had formed in only two instances (Fig. 7). Irregular hard-tissue formation was occasionally observed in the pulp at a distance from the exposure site (Fig. 8).

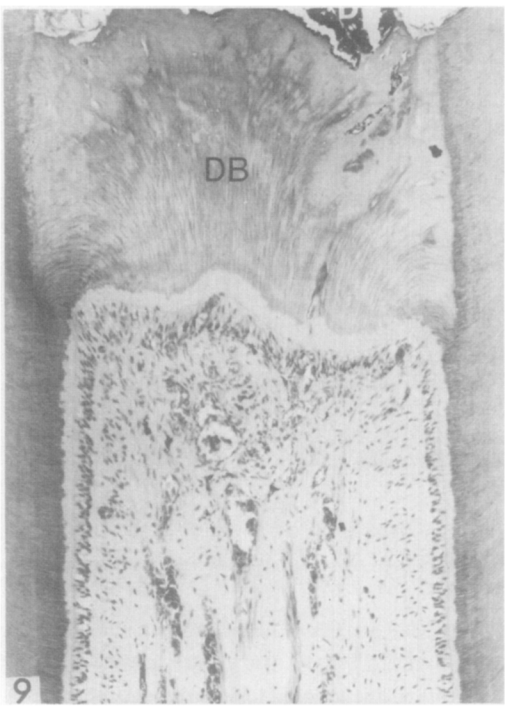
Eighty-two days. A dentin bridge had formed at the Dycal-pulp interface in eleven teeth (Fig. 9; Table II). Inclusions of soft tissue were sometimes seen in the new dentin (Fig. 10). The pulps of these teeth either were free of inflammation or only a few lymphocytes were present in the area of the lesion. Dycal particles were often observed in the tissue subjacent to the bridge (Figs. 9 and 10).

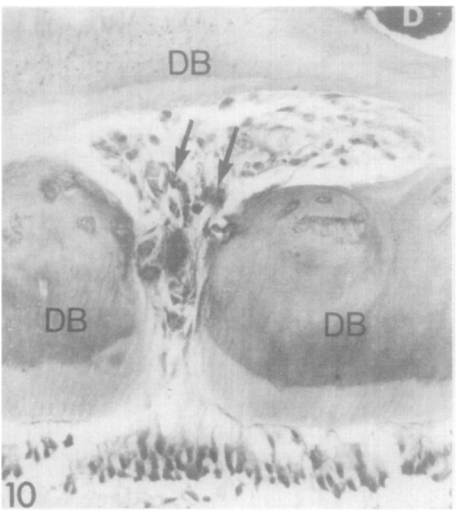
In four teeth the bridge formation at the Dycal-pulp interface was incomplete (Fig. 11). Vital pulp tissue was seen in contact with the capping material in three of these teeth. Relatively large amounts of Dycal paste were present in the superficial area of the pulp in these instances (Fig. 11). In two of the teeth the pulp was free of inflammation; in the third tooth there was a moderate chronic inflammation dominated by lymphocytes in the area of the lesion in the pulp. In the fourth tooth the greater part of the pulp was necrotic.

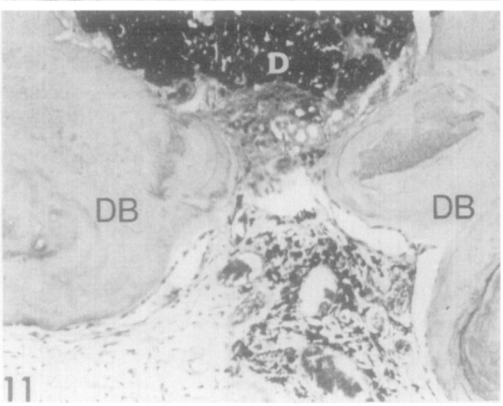
In the remaining two teeth of this series no dentin formation was evident at the Dycal-pulp interface. An area of reaction characterized by irregular strands of necrotic, inflamed, fiber-rich, or calcified tissue was observed subjacent to the exposure site (Fig. 12). At the interface between this area and the adjacent pulp tissue, which appeared unchanged, new dentin formation was evident, but a complete bridge had not formed in these instances.

DISCUSSION

The characteristic reaction of the exposed healthy pulp to calcium hydroxide was again observed in the present study, i.e., necrosis of a well-delimited area of the pulp and a mild inflammation in the adjacent vital tissue, formation of a dentin bridge at the border zone between the necrotic and vital tissues, and healing of the inflammation.[4, 8-11] In principle, the outcome of a successful capping with Dycal was similar. The exposure was closed by the formation of new dentin, and the pulpal inflammation had subsided. However, striking differences in the reaction of the pulp to the two materials were evident.







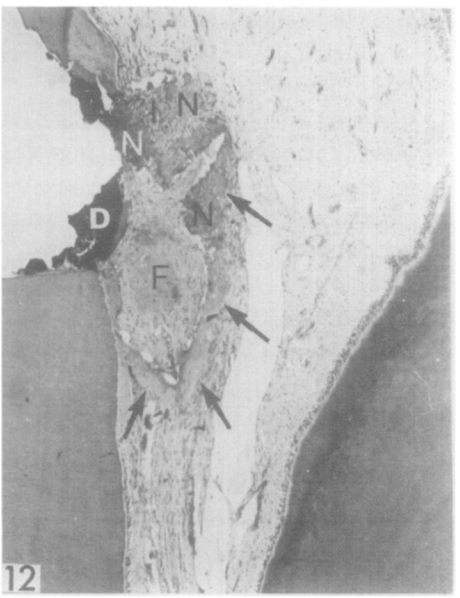


Fig. 9. Pulp capping with Dycal, 82 days. A complete dentin bridge *(DB)* has formed at the Dycal *(D)*-pulp interface. The pulp is free of inflammation. (Hematoxylin and eosin stain. Magnification, ×100.)

Fig. 10. Pulp capping with Dycal, 82 days. An inclusion of soft tissue is present in a complete dentin bridge *(DB)* at the Dycal *(D)*-pulp interface. Cells containing Dycal particles are seen *(arrows)*. (Hematoxylin and eosin stain. Magnification, ×250.)

Fig. 11. Pulp capping with Dycal, 82 days. The bridge *(DB)* at the Dycal *(D)*-pulp interface is incomplete. Relatively large amounts of the capping material are present in the superficial area of the pulp which is free of inflammation. (Hematoxylin and eosin stain. Magnification, ×250.)

Fig. 12. Pulp capping with Dycal, 82 days. The area of reaction subjacent to the capping material *(D)* has not been fully reorganized. An incomplete dentin bridge has formed, not at the Dycal-pulp interface, but at a distance from the exposure site *(arrows)*. Areas with fiber-rich *(F)*, inflamed *(I)*, or necrotic *(N)* tissue are found. (Hematoxylin and eosin stain. Magnification, ×40.)

Thus, the typical necrosis seen with calcium hydroxide was not induced by Dycal. Cells, although few and apparently altered, as well as other tissue components such as capillaries and fibers, were always present in the area of reaction subjacent to this material. To a great extent, the appearance of the pulp tissue of this area reminds one of the so-called cell-free or hyalinized zones observed in the periodontal membrane during orthodontic treatment.[12, 13] This similarity is underlined by the apparent analogy in the repair processes. In the periodontal membrane, macrophages, and, to some extent, giant cells, are said to play an important role in the reorganization of the cell-free zone.[13] Also the area of reaction in the pulps capped with Dycal became heavily infiltrated with macrophages and giant cells, and after a period of some 30 days the repair was completed, and the tissue of the area of reaction could no longer be distinguished from the rest of the pulp tissue.

An interesting observation during the period of repair was the uptake of Dycal by macrophages and giant cells. In unsuccessful cases, such particles had been moved far away from the exposure site, and were even observed in the periodontal membrane. However, Dycal particles were also seen in the pulp in successful cases. It seems, therefore, that the phagocytes are not able to digest the material, and, subsequent to repair of the pulp, it may remain in the tissue, apparently well tolerated.

Like pure calcium hydroxide, Dycal has the ability to induce differentiation of odontoblasts and dentin bridge formation. It is worth noticing, however, that since the area of reaction in the pulps capped with Dycal does not become necrotic, but is fully reorganized, the bridging in these teeth occurs at the Dycal-pulp interface and not at some distance from the exposure site as is the case when calcium hydroxide is used.[3-5] Langeland and his co-workers[14] have emphasized that hard-tissue barriers formed in the pulps after treatment with calcium hydroxide are often incomplete and contain inclusions of soft tissue. This was observed in the present study as well. The quality of the bridges induced by Dycal treatment was, however, generally good, and apparently not inferior to the quality of bridges in pulps capped with calcium hydroxide.

In two teeth of the present experimental series, the tissue of the area of reaction subjacent to the Dycal paste had not been fully reorganized after 82 days. In these instances, there was evidence of a walling off of the rest of the pulp from the area of reaction by hard tissue. The bridging was, however, extremely defective, and the prognosis of the teeth was rated as uncertain. Still it is interesting to note the similarity between the reaction of these teeth and the typical reaction of the pulp to pure calcium hydroxide when the bridge is formed at some distance from the exposure site.

The number of teeth in the present study is limited, but the results are in agreement with those of previous studies, so that Dycal may be considered to be an acceptable medium in the treatment of the exposed pulp.[2-5] However, the fact should be emphasized that, so far, Dycal has been used only in the treatment of exposed, healthy pulps, and, conceivably, its effect on an exposed, inflamed pulp may be less favorable.[15]

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Reprint requests to:
Dr. Leif Tronstad
Dental Institute of Experimental Research
University of Oslo, Blindern
Oslo 3, Norway