CHEMICAL GINGIVECTOMY

EFFECT OF POTASSIUM HYDROXIDE ON PERIODONTAL TISSUES

by

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Chemical cauterization has been and still is a rationale in periodontal therapy. The purpose of applying escharotic chemical substances on gingival tissues is to cauterize the marginal gingiva. As a consequence, the latter will exfoliate and the depth of the gingival pocket will be reduced.

The most effective cauterizing agents are found among strong inorganic acids and alkalis. It is evident that the use of acids is less suitable since these are liable to cause a demineralization of the hard tissues of the teeth. The application of alkaline caustics is motivated by their ability to cauterize without affecting the mineralized tooth structures.

Different concentrations of potassium hydroxide in watery solutions (from 10—40 per cent) as well as in the form of paste have been advocated (*Harndt* 1950, *Aas* 1960). As far as can be seen from the literature, the results after treatment with potassium hydroxide are exclusively based on clinical evidence. In view of the fact that potassium hydroxide is a strong alkali, it seemed justifiable to study:

(1) its initial effect on the periodontal tissue,
(2) the healing process following such treatment, and
(3) the long term effects of cauterization.

MATERIAL AND METHOD

Twenty-eight teeth in five normal dogs were used, their gingiva showing inconspicuous or no inflammation. Young dogs were preferred as in such animals the epithelial cuff has its apical

Table 1

Survey of the experiments and the tissue reactions.

Specimens	Tooth	KOH %	Obs. period	Depth of necrosis from CEJ (mm)	Formation of plaque	Distance CEJ to deepest point of epith.prolif. (mm)	Depth of pocket (mm)	Distance bottom of pocket to alv. crest (mm)	Root resorption
Dog 28	LL 4	10	15 min.	1.7			2.3	1.5	
„ „	LR 4	„	„ „	1.1			2.7	1.1	
„ „	UL 4	„	„ „	1.8			2.2	1.6	
„ „	UR 4	„	„ „	2.0			2.4	1.4	
E.S.*	UL 3	40	„ „	2.1			1.6	1.0	
E.S.*	UL 1	„	„ „	2.5			2.4	1.1	
Dog 24	UR 9	„	30 „	2.2			2.0	1.3	
„ 22	LL 9	„	45 „	2.1			1.9	1.4	
„ „	UR 8	25	„ „	2.2			1.8	0.8	
„ 29	LR 4	10	5 days		+	1.5	0.5	0.8	—
„ „	UR 9	„	„ „		—	2.0	0.5	0.0	+
„ „	UR 4	„	„ „		+	0.5	1.1	1.2	—
Dog 24	LR 4	25	9 „		+	1.2	1.1	1.2	+
„ 22	LR 4	„	12 „		+	0.9	1.3	1.2	—
„ 24	LL 4	„	15 „		+	0.8	1.3	1.2	—
„ „	UR 6	40	22 „		+	2.0	0.5	1.4	+
„ 22	UL 7	25	„ „		+	0.5	2.0	0.5	—
„ „	UR 9	„	„ „		+	1.3	1.9	2.0	—
„ „	UR 7	„	„ „		+	1.2	2.0	1.2	—
„ „	UL 4	„	„ „		+	0.6	1.5	1.3	—
„ „	UR 4	„	„ „		+	0.0	1.5	1.9	—
Dog 24	UL 9	„	23 „		+	1.2	1.5	0.7	—
„ „	UL 5	40	„ „		+	1.5	1.0	1.4	+
„ „	UR 9	25	42 „		—	1.1	1.5	2.5	—
„ „	UR 5	„	49 „		+	0.3	1.2	1.3	—
„ „	UR 4	40	„ „		+	1.1	1.5	1.5	—
„ „	UR 7	„	„ „		+	2.2	1.6	1.0	+
Dog 26	UR 8	10	968 „		—	0.7	2.2	1.8	—
„ „	UR 4	„	„ „		+	2.5	2.2	1.3	+
„ „	LR 4	„	„ „		+	2.1	2.3	2.6	—

* Human biopsy material.

limit at the cemento-enamel junction (CEJ). Any downgrowth of epithelium beyond this landmark is, consequently, easily registered.

After intravenous injection of Nembutal, thin cotton strings moistened in a 10, 25, or 40 per cent watery solution of potassium hydroxide were inserted into the buccal pockets. The drug was allowed to act for two minutes, after which the operation field was washed with water. Observation periods varied from 15 minutes to 968 days.

Thirteen teeth in five adult human beings were also part of the series. Since in such cases the bottom of the gingival pocket usually is located apical to the cemento-enamel junction, a fixed point of reference is lacking. For this reason these teeth were less suited for measurements and most of them were left out of Table 1. Only two teeth were included, as in these cases the deepest point of the epithelial cuff before treatment could be determined. The human teeth were extracted with special forceps which allowed simultaneous resection of approximately 10 mm of adjacent periodontal tissues.

Immediately after removal, all specimens were fixed in 10 per cent neutral formalin and demineralized in 5.2 per cent nitric acid. After embedding in paraffin, the teeth with the adjacent tissues were cut in sections 5 microns thick and stained in hematoxylin and eosin and in *Mallory*'s connective tissue stain.

OBSERVATIONS

Clinical observations immediately following the application of the escharotic revealed increased secretion from the gingival pockets and exfoliation of structureless tissue. The treated gingiva tended to bleed, and plaque formation was common. Patients whose gingiva had been cauterized, complained of pain in connection with, and for some time subsequent to, the treatment.

The histologic investigations showed that the cotton strings could be brought down to the cemento-enamel junction (Fig. 1). When potassium hydroxide was brought in contact with the cuff epithelium, the entire epithelial layer as well as the adjacent mesodermal tissues necrotized. The cells of the sub-epithelial and the supra-alveolar connective tissue in all cases lost their stainability (Fig. 2). The apical limit of the destruction was found either at the entrance of the alveolus, or more frequently, within

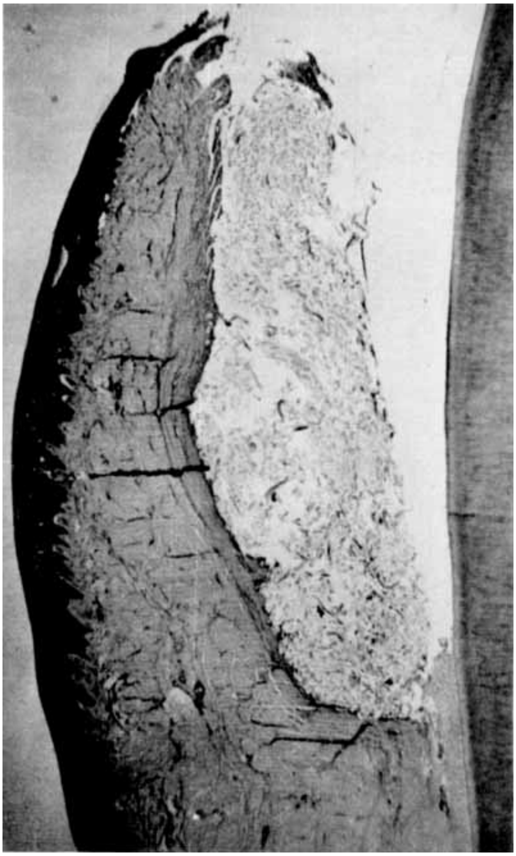

Fig. 1.
Dog 28, UL 4. Hematoxylin-eosin. Cotton string, moistened in 10 % KOH, pressed to cemento-enamel junction (CEJ). 15 minutes observation time. *Pocket epithelium completely necrotized.*

the alveolar periodontal membrane (Fig. 2) at a distance up to 2.5 mm beyond CEJ. It appears from Table 1 that the penetrating effect to some extent was enhanced with an increase of the con-

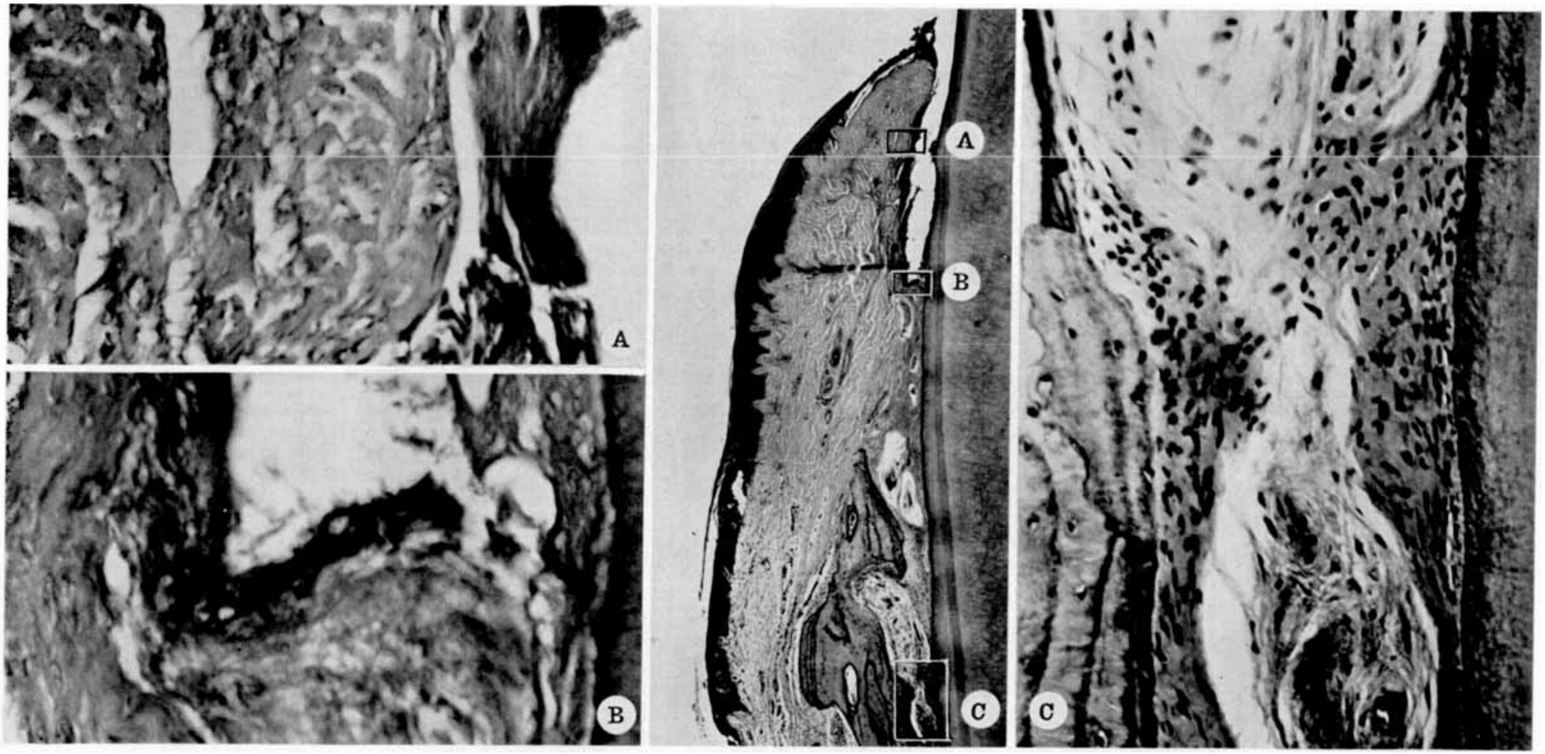

Fig. 2.

E. S., UL 3. Hematoxylin-eosin. Cotton string moistened in 40 % KOH pressed to the bottom of the gingival pocket 15 minutes before extraction of the tooth with the adjacent tissues. Necroses of the pocket epithelium, of the sub-epithelial (A) and the supra-alveolar (B) and intra-alveolar (C) connective tissue.

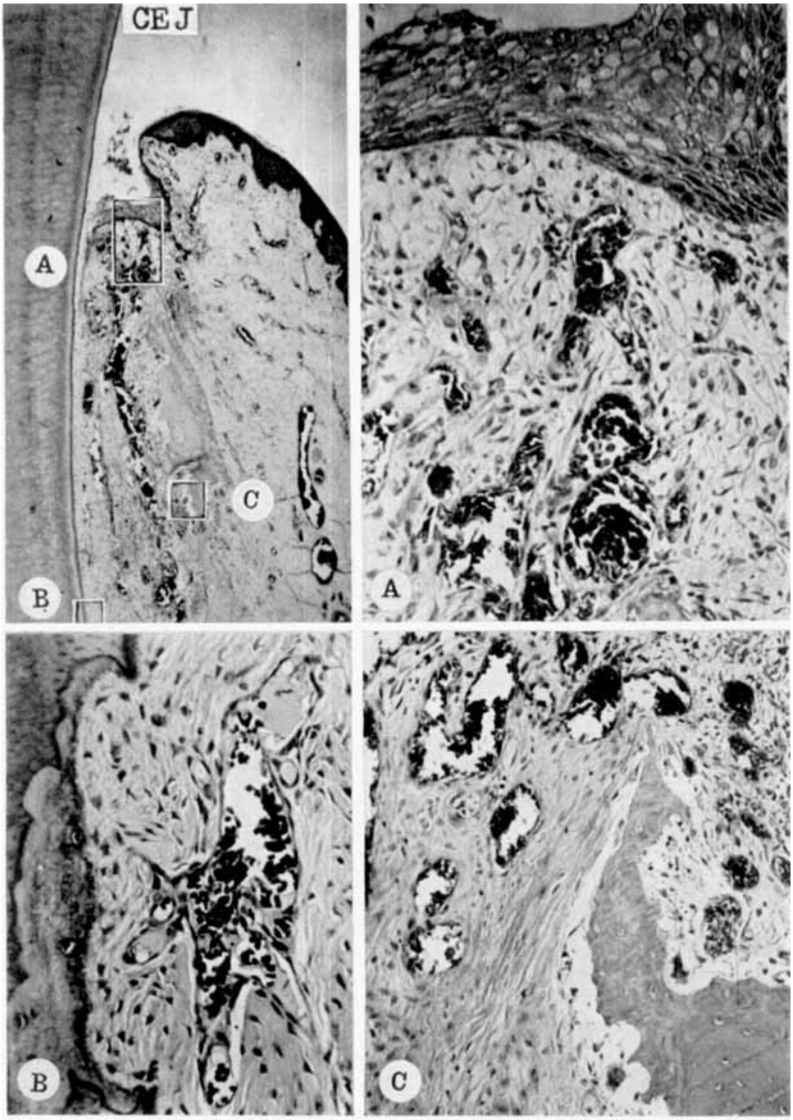

Fig. 3.

Dog 29, UR 9. Hematoxylin-eosin. Buccal pocket treated with 10 % KOH. Obs. period 5 days. Epithelial proliferation from the gingival margin. Numerous blood vessels in sub-epithelial connective tissue (A). Resorption of bone at margo alveolaris and from the periodontal membrane side (C). Area of resorption at the root being filled in by new hard tissue formation (B).

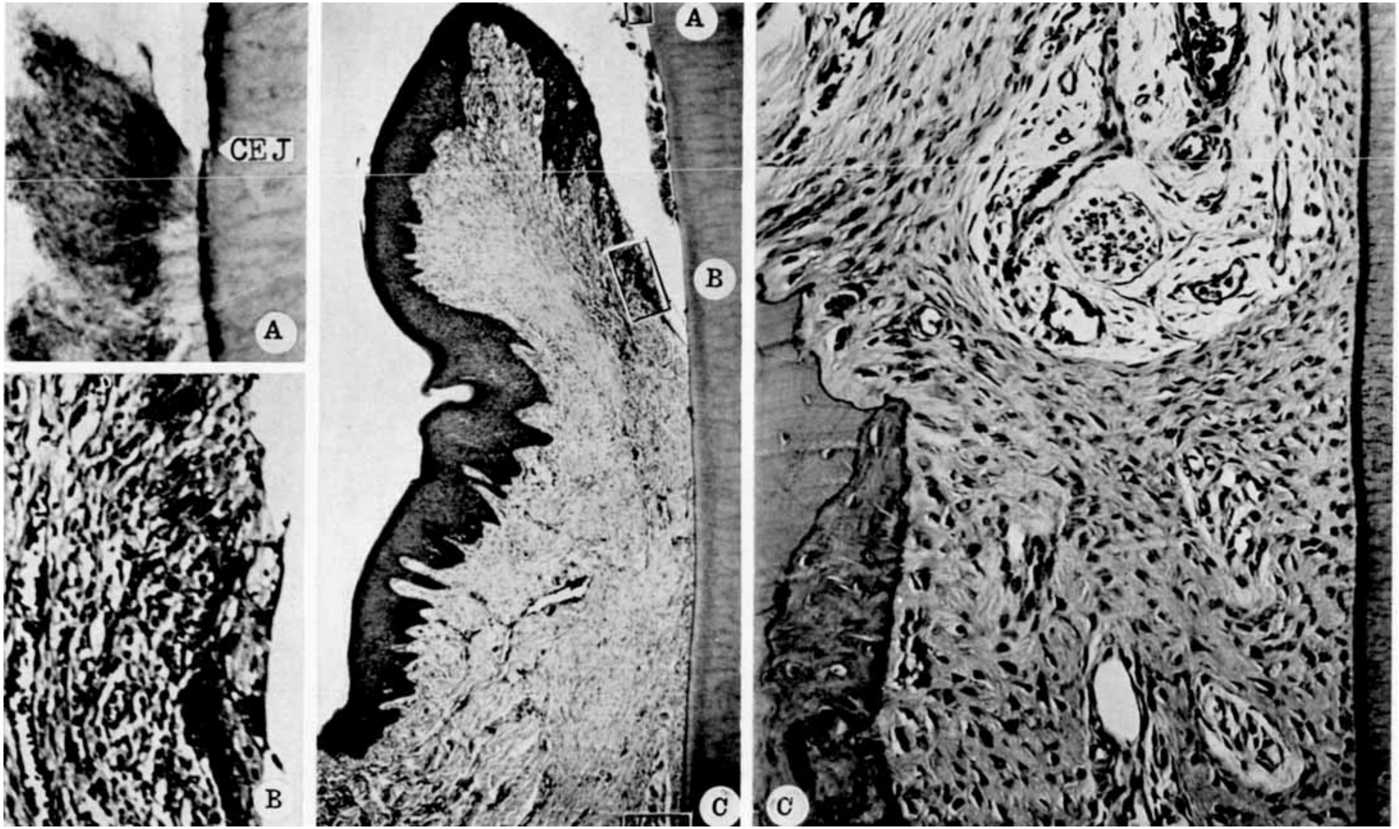

Fig. 4.

Dog 24, UL 9. Hematoxylin-eosin. Treatment with 25 % KOH. Obs. period 23 days. The apical limit of the epithelial cuff 1.2 mm apical to the CEJ. Plaque formation (A). Degenerative changes in the sub-epithelial connective tissue (B). Dilated vessels. bone tissue formation and usual fibre direction of the periodontal membrane (C).

centration from 10 to 25 per cent. On the other hand, no difference could be seen when the concentration was raised from 25 to 40 per cent.

After an observation period of five days, all necrotic tissue was exfoliated and the healing processes were well advanced (Fig. 3). Epithelial cells proliferated from the oral epithelium and formed the cover of the soft tissue against the tooth surfaces (Figs. 3—7). The epithelium itself showed degenerative changes (Figs. 3 A, 4 B, 5 A and 7 A). In nearly all cases, supra-gingival and sub-gingival plaque were seen bordering on the epithelium (Figs. 3—7).

At this stage of healing, the sub-epithelial connective tissue was characterized by inflammation. Numerous capillaries surrounded by neutrophilic leukocytes bordered on the cuff epithelium (Figs. 3 A and 4 B). Few collagen fibre bundles were seen in these areas; their number increasing further apically. The bulk of the fibre bundles was oriented more or less parallel to the rooth surface (Fig. 7 C). The deeper parts of the periodontal membrane also showed dilated vessels and presence of inflammatory cells. Resorption of the alveolar bone had taken place from the alveolar crest to an extent of 7—8 mm in the apical direction (Fig. 3 B). Fig. 5 shows a fragment of the alveolar bone in the sub-epithelial connective tissue. Most likely, it would have been exfoliated through the gingiva.

After more than fifteen days, bone resorption seemed to have ceased and bone formation processes started. Root resorption (Fig. 6) and deposition of hard tissue onto resorption lines (Fig. 8) were seen in six cases. In five of these teeth, the resorption areas were located just apical to the cemento-enamel junction, whereas in one case the resorption was localized in the intra-alveolar part of the root (Fig. 3 B). In the cases where the observation period exceeded twenty-two days, the deepest point of the epithelial cuff was situated apical to the resorption lacunae. A downgrowth of epithelium beyond the cemento-enamel junction took place in all but one case. Measurements showed that the length of the epithelial cuff ranged from 0.5 to 2.0 mm.

In the long term experiments, the downgrowth appeared to be somewhat increased (up to 2.5 mm), and the width of the epithelial cuff showed greater dimensions (up to 2.3 mm). Nearly

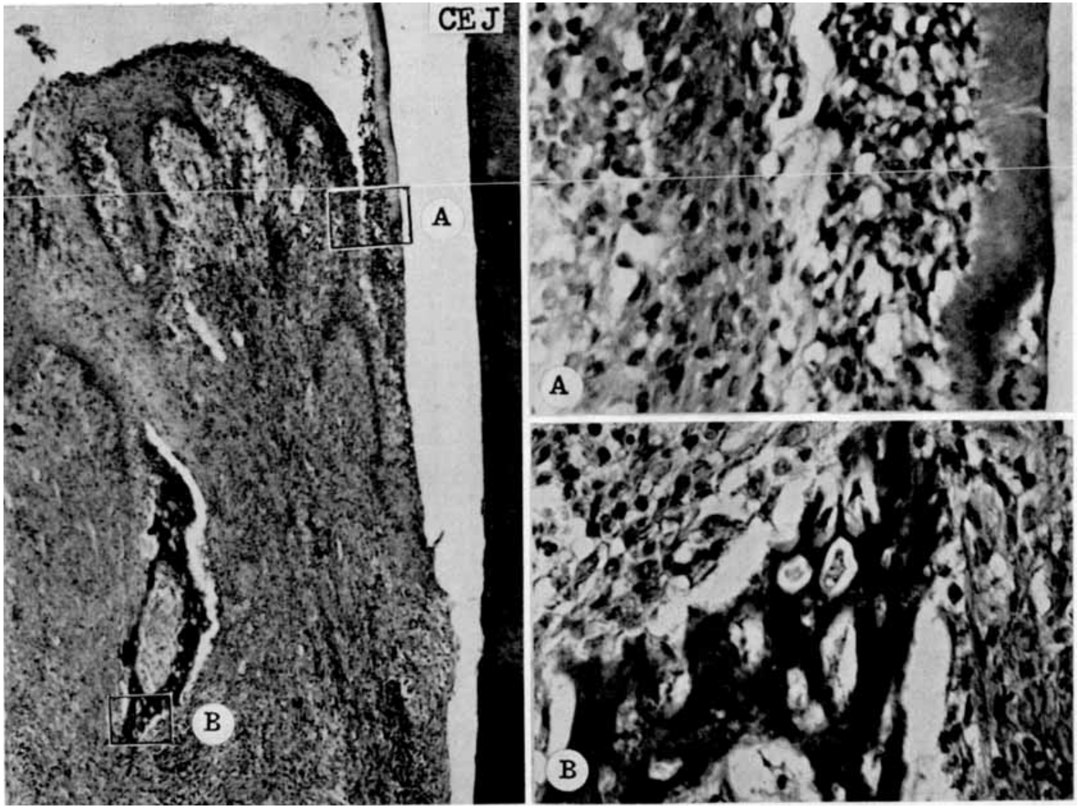

Fig. 5.

Dog 24, LL 4. *Mallory's* connective tissue stain. Buccal pocket treated with 25 % KOH. Obs. period 15 days. Epithelial downgrowth beyond the CEJ. Plaque formation, degenerated epithelium and inflammation in the subjacent connective tissue (A). Necrotized bone tissue being exfoliated through the gingiva (B).

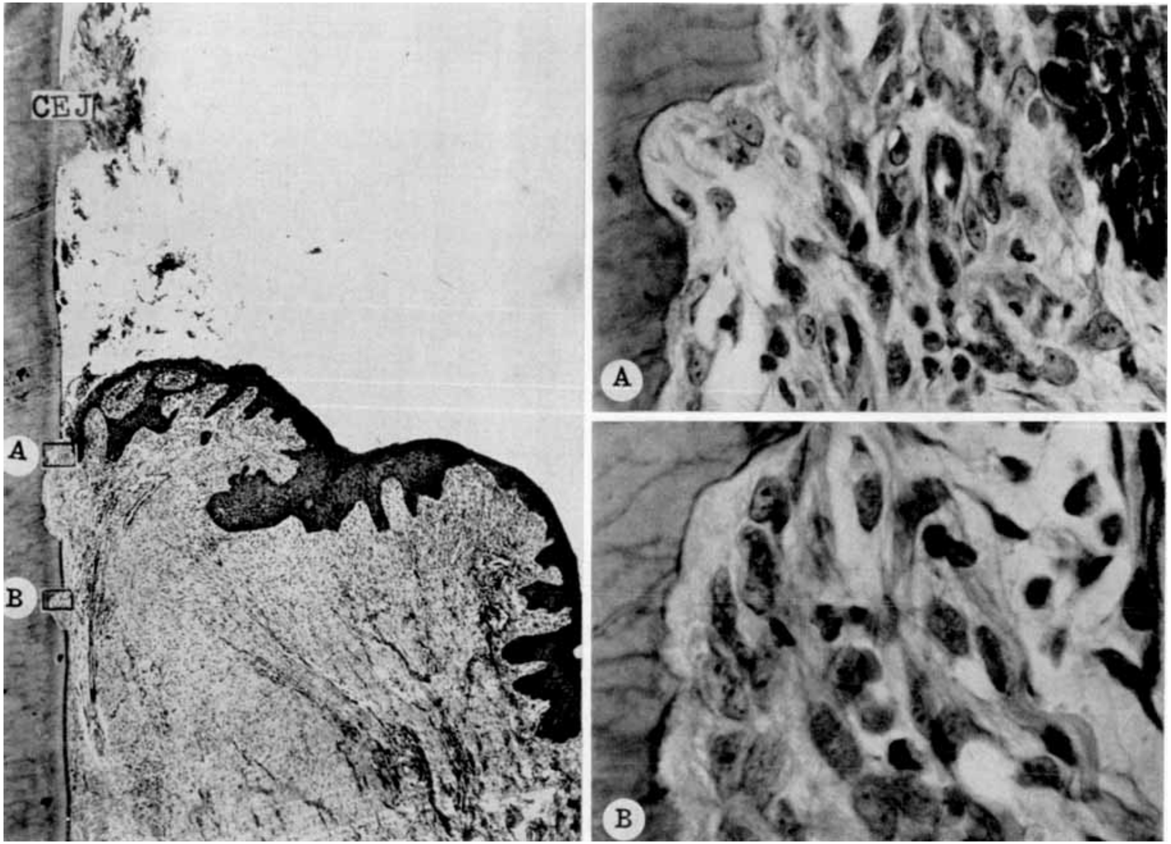

Fig. 6.

Dog 24, UR 6. Hematoxylin-eosin. 40 % KOH. Obs. period 22 days. Apical migration of epithelium 2.0 mm beyond CEJ. Root resorption (A). New hard tissue formation in the resorption lacunae (B).

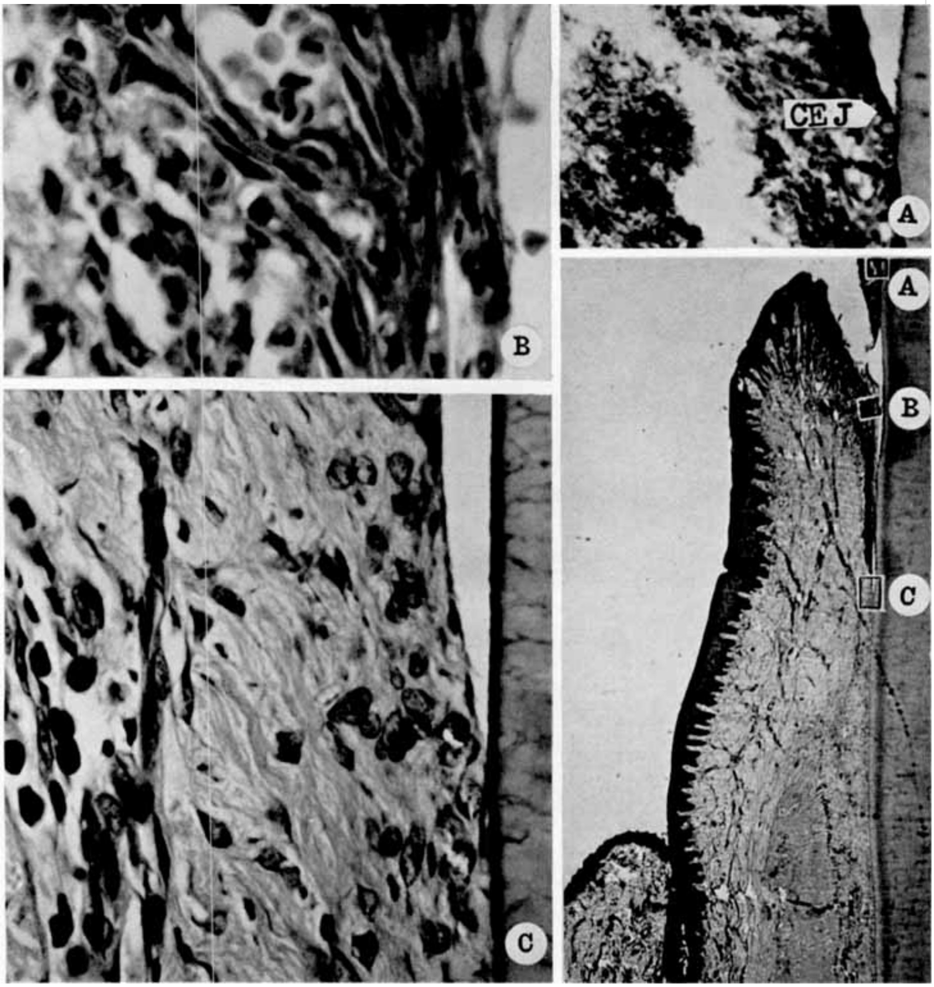

Fig. 7.

Dog 24, UR 4. Hematoxylin-eosin. 40 % KOH. Obs. period 49 days. Downgrowth of epithelium 1.1 mm apical to the CEJ. Plaque formation (A). Degenerated cuff epithelium (B) and collagen fibers running parallel to the root surface (C).

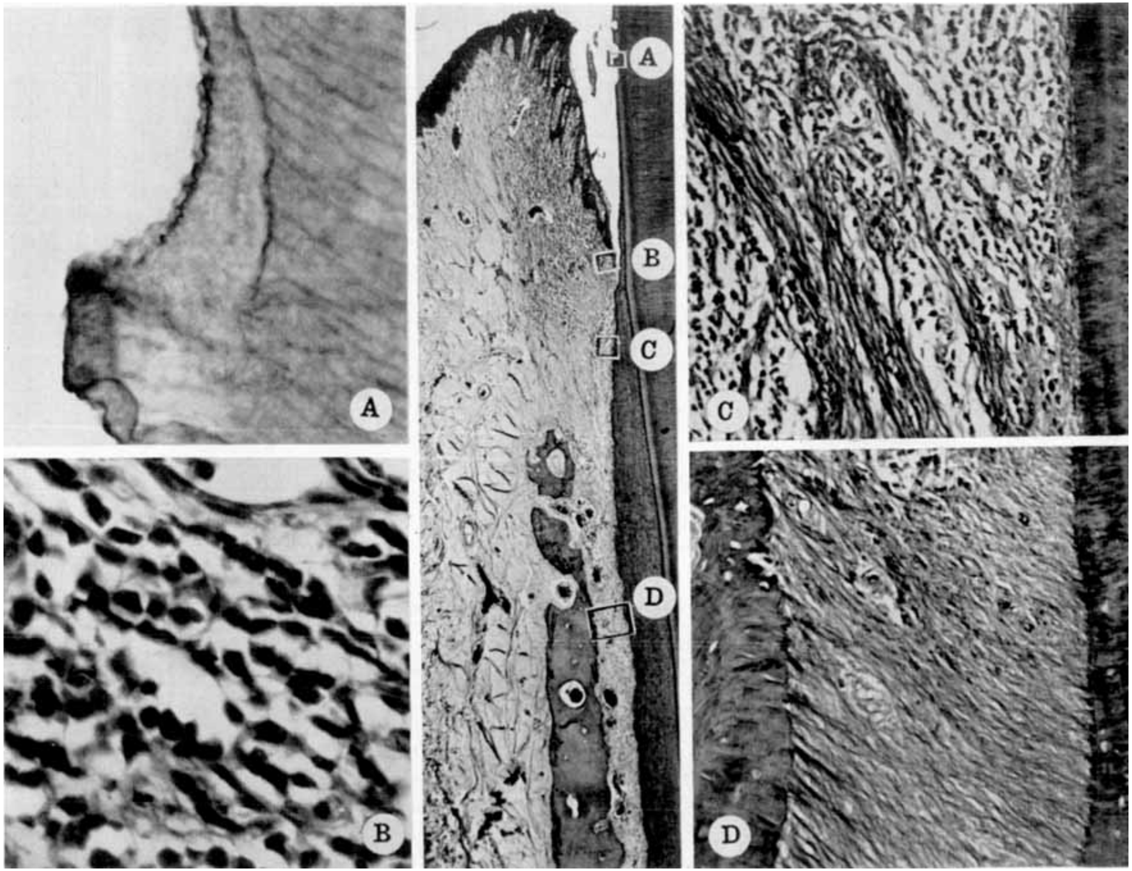

Fig. 8.

Dog 26, UR 4. Hematoxylin-eosin. *Mallory's* connective tissue stain. 10 % KOH. Obs. period 968 days. Epithelial proliferation 2.5 mm below the CEJ. Resorption lacunae just below CEJ (A). Aggregation of mononuclear inflammatory cells in the sub-epithelial connective tissue (B). Few collagen fibres in the supra-alveolar connective tissue (C). The intra-alveolar periodontal membrane is normal (D).

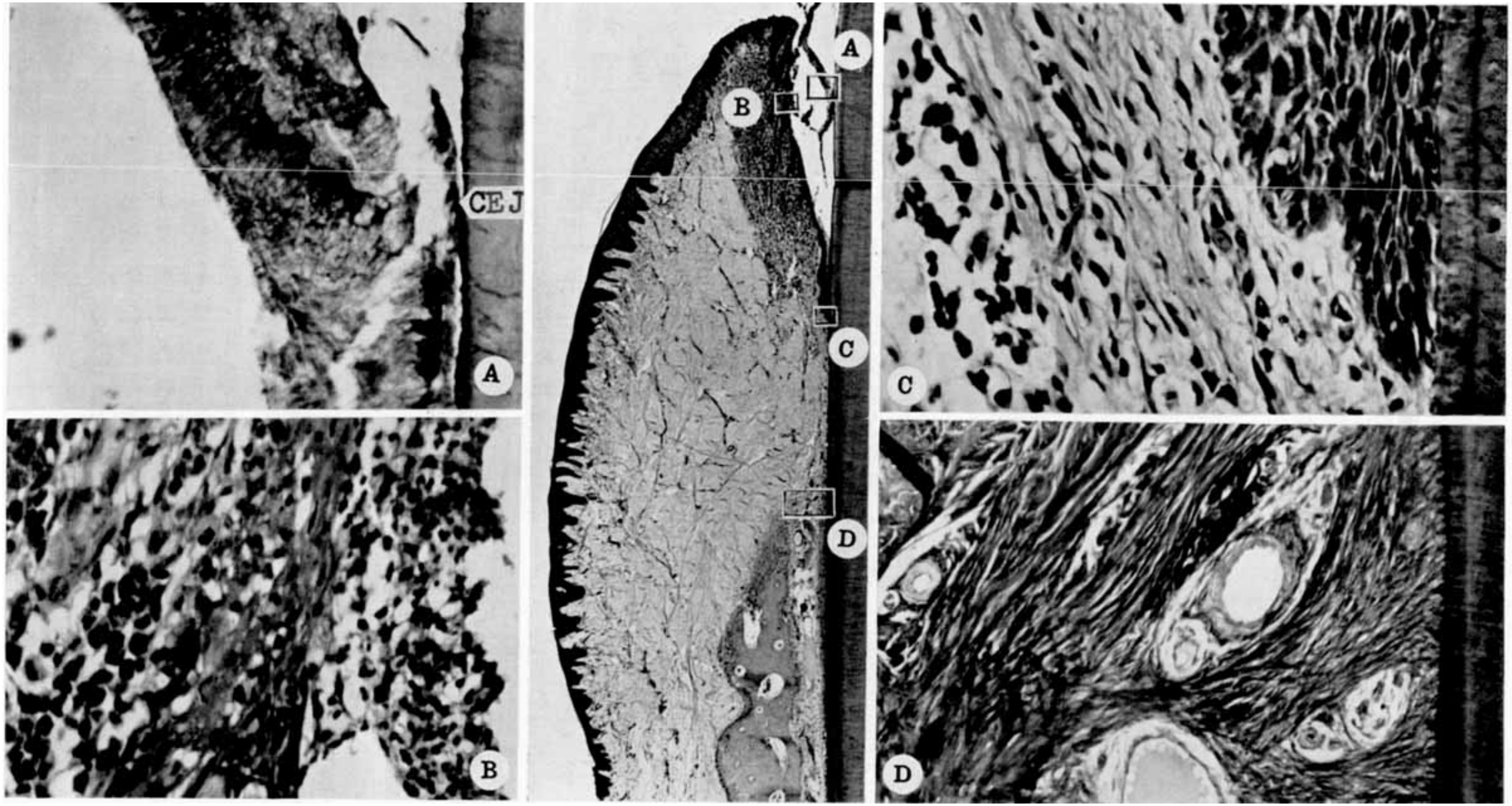

Fig. 9.

Dog 26. LR 4. Hematoxylin-eosin. *Mallory's* connective tissue stain. 10 % KOH. Obs. period 968 days. Apical limit of epithelial cuff 2,1 mm below the CEJ. Supra- and sub-gingival plaque (A). Leukocytes in the degenerated epithelium and subjacent connective tissue (B). Supra-alveolar (C) and intra-alveolar connective tissue normal (D).

three years after treatment a considerable inflammation and few collagen fibres could be seen in the sub-epithelial connective tissue. On the other hand, these cases presented normal periodontal membranes (Figs. 8 and 9).

DISCUSSION

Potassium hydroxide in gingival pockets produces necrosis of the epithelium, and it has a necrotizing effect on the sub-epithelial and supra-alveolar connective tissue, as well as on the coronal part of the periodontal membrane and the alveolar bone. This initial destruction extends to 2.5 mm beyond the bottom of the gingival pocket. Some difference seems to exist between the effect of a 10 per cent solution of potassium hydroxide and that of concentrations of 25 and 40 per cent. This difference, however, appears to be one of degree, as all three solutions cause necrosis of the soft tissue between the gingival margin and the entrance of the alveolus.

The first stage of healing is clinically characterized by exudation and exfoliation of necrotic tissue, and the gingiva generally tends to bleed. Five days after cauterization the gingiva is re-epithelized, although at this stage of healing the width of the epithelial cuff is small. Frequently, the epithelium of only a few cells thickness meets with the tooth surface in a linear contact (Fig. 6). Clinically, this relationship is characterized as "pockets of zero depth". It has been shown earlier (*Waerhaug* 1955, *Waerhaug & Löe* 1957), and it is confirmed in this investigation that such a relationship between gingiva and the tooth does not last very long. In cases where the apical limit of the epithelial line is stabilized, a new gingival cuff will develop coronally and reach normal width. In the present material, the growth in width was not completed within 49 days (Table 1).

During the first six to seven weeks the cuff epithelium and the subjacent connective tissue show inflammatory changes. It seems likely that the persistent inflammation is caused by the supragingival and sub-gingival bacterial plaque (*Waerhaug* 1955). Consequently, the wound surfaces should be kept clean during the healing period. The findings in the long term experiments emphasize the significance of this aspect of the treatment. In these cases a chronic inflammation persisted nearly three years after treatment. There seems to be little doubt that this inflammation is caused by the bacterial plaque (Fig. 9), and that it is not directly related to the cauterization.

Apical downgrowth of epithelium was a common finding. Only in one case the deepest point of the epithelial cuff was still at the cemento-enamel junction. As seen from Table 1, a full correlation does not exist between the depth of the initial necrosis and the downgrowth of epithelium beyond the CEJ. In eight teeth the proliferation was less than the smallest measurements of the depth of necrosis. At least three causes for this discrepancy are conceivable:

(1) The fact that the cotton string was left in the pocket until autopsy, viz., more than the usual two minutes, may have enhanced the effect of the agent.
(2) The cotton string has not reached the bottom of the pocket in all cases. This may happen in cases where the alveolar crest is located coronal to the apical limit of the epithelial cuff (*Waerhaug & Löe* 1958).
(3) A certain degree of reattachment may have been obtained during the healing.

The dilated vessels in the periodontal membrane, and the osteoclastic activity in the alveolar bone at the same level, suggest that potassium hydroxide has an initial remote effect beyond the area of necrotic cells. The long term experiments, however, show that these reactions do not cause permanent injury.

The present study gives no clue as to the effect produced by potassium hydroxide on the dental hard tissues. On the other hand, the possibility cannot be ignored that the organic constituents of the superficial cementum is affected to some extent by the contact with the alkaline fluid. Presumably, this may be a contributory cause of the root resorption which occurred in the gingival areas, although there are reasons to believe that the inflammation in the sub-epithelial connective tissue itself may initiate such activities.

The investigation shows that potassium hydroxide has a strong escharotic effect on the periodontal tissue. This observation is in accordance with the known effect of strong bases upon other animal tissues, and is explained by the physico-chemical characteristics of these fluids (*Anderson* 1953). The use of potassium hydroxide for removal of the epithelium in pathological pockets is therefore contra-indicated. Even more so, as other drugs which may be as effective in removing the epithelium, definitely show less injuries of the subjacent mesodermal tissues. (*Johnson & Waerhaug* 1956, *Waerhaug & Löe* 1958). Considering the uncontrollable damaging of the tissues, the use of potassium hydroxide in the treatment of hypertrophic gingiva (chemical gingivectomy) should be discouraged.

Repeated use of potassium hydroxide (*Wade* 1960) must also be deprecated as every repeated application of the agent will result in new tissue injuries and a healing according to the descriptions already given. In other words, every treatment with potassium hydroxide will cause an additional movement of the apical limit of the epithelial cuff and a corresponding loss of attachment.

SUMMARY AND CONCLUSIONS

Buccal pockets of twenty-eight teeth in normal dogs and thirteen human teeth were treated with 10 per cent, 25 per cent, and 40 per cent watery solution of potassium hydroxide. After observation periods of 15 minutes to 968 days, the following conclusions may be drawn:

(1) The treatment induces pain and causes gingival bleeding and increased secretion. Plaque formation is common.

(2) The histologic examination shows that potassium hydroxide necrotizes the eptihelium of the gingival pocket, the sub-epithelial and the supra-alveolar connective tissue, and the marginal part of the alveolar bone.

(3) Five days after treatment all necrotic soft tissue may be exfoliated and the pockets re-epithelized. The pocket epithelium frequently adjoins the tooth in a linear contact ("pocket of zero depth"). This situation is not permanent. Somehow a normal pocket depth will develop in all cases.

(4) In all teeth, with one exception, an apical downgrowth of the pocket epithelium (maximum 2.5 mm) took place with a corresponding loss of fiber attachment and reduction of the alveolar bone.

(5) The present series shows that the use of potassium hydroxide in periodontal therapy is contra-indicated.

RÉSUMÉ ET CONCLUSIONS

GINGIVECTOMIE CHIMIQUE. ACTION DE L'HYDROXYDE DE POTASSIUM SUR LES TISSUS PARODONTAUX

Les culs-de-sac vestibulaires de vingt-huit dents chez des chiens normaux et de treize dents humaines ont été traités à l'hydroxyde de potassium en solution aqueuse à 10 p.cent, à 25 p.cent et à 40 p.cent. Après une période d'observation de 15 minutes à 968 jours il est possible de conclure:

1) Le traitement est douloureux et provoque des hémorragies gingivales et une augmentation de la secrétion. La formation de plaque est fréquente.
2) L'examen histologique montre que l'hydroxyde de potassium nécrose l'épithélium du cul-de-sac gingivo-dentaire, le tissu conjonctif sous-épithélial et supra-alvéolaire et la partie marginale de l'os alvéolaire.
3) Cinq jours après le traitement, tous les tissus mous nécrosés peuvent être exfoliés et l'épithélium des culs-de-sac être régénéré. L'épithélium du cul-de-sac forme souvent avec la dent un contact ponctuel ("cul-de-sac de profondeur zéro"). Cette situation n'est pas permanente. D'une manière ou d'une autre, il s'est dans tous les cas développé un cul-de-sac de profondeur normale.
4) Pour toutes les dents, à une seule exception près, une prolifération de l'épithélium du cul-de-sac en direction de l'apex a eu lieu (maximum 2,5 mm), avec perte correspondante de l'attachement fibreux et réduction de l'os alvéolaire.
5) Cette série d'expériences montre que l'usage de l'hydroxyde de potassium pour le traitement des lésions parodontales est contre-indiqué.

ZUSAMMENFASSUNG UND SCHLUSSFOLGERUNGEN

CHEMISCHE GINGIVEKTOMIE. DIE WIRKUNG VON KALIUMHYDROXYD AUF DAS PERIODONTALE GEWEBE

Buccale Zahnfleischtaschen von 28 Hunde- und 13 menschlichen Zähnen wurden mit 10%iger, 25%iger und 40%iger wässeriger Kaliumhydroxyd-Lösung behandelt.

Nach Beobachtungszeiträumen von 15 Minuten bis zu 968 Tagen können folgende Schlussfolgerungen gezogen werden:

1. Die Behandlung ist schmerzhaft und verursacht gingivale Blutung und verstärkte Sekretion. Plaquebildung tritt regelmässig auf.
2. Die histologische Beobachtung zeigt, dass Kaliumhydroxyd eine Nekrose des gingivalen Taschenepithels, des subepithelialen und supraalveolären Bindegewebes und des marginalen Teils des Alveolarknochens verursacht.
3. Fünf Tage nach der Behandlung ist das gesamte nekrotische Weichgewebe abgestossen und die Taschen sind wieder epithelisiert. Das Taschenepithel liegt häufig dem Zahn in engem Kontakt an ("Tasche von 0-Tiefe"). Diese Situation ist nicht von Dauer. Irgendwie wird sich in allen Fällen eine normale Taschentiefe entwickeln.
4. Bei allen Zähnen — mit einer Ausnahme — fand ein apikales Tiefenwachstum des Taschenepithels (Maximum 2,5 mm) statt, verbunden mit einem entsprechenden Verlust parodontalen Bindegewebes und einem Abbau des Alveolarknochens.
5. Die vorliegenden Untersuchungen zeigen, dass die Verwendung von Kaliumhydroxyd in der parodontalen Therapie kontraindiziert ist.

RESUMEN Y CONCLUSIONES

GINGIVECTOMÍA QUÍMICA. EFECTO DEL HIDRÓXIDO DE POTASIO SOBRE LOS TEJIDOS PERIODONTALES

Se trataron 28 bolsas vestibulares de perros normales y 13 de dientes humanos con una solución acuosa de hidróxido de potasio al 10%, 25% y 40%. — Después de periodos de observación de 15 minutos a 968 días, se arribó a las siguientes conclusiones:

1) El tratamiento produce dolor, hemorragia gingival y aumento de secreción. Es común la formación de placas.
2) El examen microscópico muestra que el hidróxido de potasio necrosa el epitelio de la bolsa gingival, el tejido conectivo subepitelial y supraalveolar y la parte marginal del hueso alveolar.
3) Cinco días después del tratamiento se elimina todo el tejido blando necrótico y la bolsa se re-epitaliza. El epitelio de la bolsa frecuentemente se une al diente en un punto ("bolsa de profundidad cero"). Esta situación no es permanente. De algún modo reaparece en todos los casos la profundidad normal de la bolsa.
4) En todos los dientes, con una excepción, el movimiento apical del fondo de la bolsa (máximo 2.5 mm) produjo la correspondiente pérdida de inserción fibrosa y reducción del hueso alveolar.
5) Los casos presentados muestran que el uso de hidróxido de potasio en la terapía periodontal está contraindicado.

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