From:
The Department of Periodontology,
and the Department of General
Experimental Research,
Dental Faculty,
University of Oslo, Norway

REPAIR OF THE POST-EXTRACTION ALVEOLUS IN THE WISTAR RAT

A HISTOLOGIC AND AUTORADIOGRAPHIC STUDY

by

JAN R. JOHANSEN

INTRODUCTION

In an earlier report concerning the repair of the post-extraction alveolus in the guinea pig (*Johansen & Gilhuus-Moe*, 1969) it was noted that proliferation of osteogenic cells of the periosteum of the mandible around the extraction site took place before bone formation started within the extraction alveolus itself. These autoradiographic findings supported previous observations in dogs by *Boyne* and *Kruger* (1962) and in humans by *Boyne* (1966) with fluorescence microscopy. Even though the rat has been used to study post-extraction wounds by several authors (*Glickman et al*, 1947, *Huebsch et al*, 1952; *Smith*, 1958; *Frandsen*, 1963; *Pietrokovski & Massler*, 1967; *Pietrokovski*, 1967) little attention was focused on the extra-alveolar changes. While the present investigation was in progress, however, *Åstrand* and *Carlsson* (1969) used fluorescence microscopy to study maxillary extraction wounds in rats and reported observations similar to those of *Boyne* (1962).

The introduction of isotopes in experimental biology has considerably increased our knowledge about the processes at the molecular level. For the study of cellular proliferation, the use of tritiated (3H) thymidine, one of the precursors of desoxyribonucleic acid, has become a widely used tool during the last decade. The dipping technique (*Joftes*, 1963) has simplified the autoradiographic procedure whereby a large number of slides can be coated with the necessary photographic emulsion in a short time.

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By a combination of regular histologic staining methods and autoradiography it is possible to obtain more information about the dynamic changes taking place in a repairing tissue such as in the post-extraction alveolus than by other light microscopic techniques.

The purpose of this investigation was to further clarify the cellular proliferation in the repairing extraction alveolus and the surrounding structures in the rat. Furthermore, a comparison of the observations from this study with the observations from a previous study seemed desirable, since the anatomy of the guinea pig incisor and its periodontium are quite different from that of the rat molar.

MATERIAL AND METHODS

Eighteen white male Wistar rats were used in this study. Their body weights are presented in Table I. The animals were placed in separate cages 7 days prior to the experimental procedures to adjust to the new environment. The purpose of choosing such young animals was twofold. Firstly, the extraction of molars in rats is difficult and becomes even more so with increasing age due to the cementum apposition that takes place on the apices of the roots. Secondly, the high cost of the isotope makes small animals preferable for economical reasons.

The first lower left mandibular molar was extracted in each animal, after initial loosening of the tooth by using a small dental chisel as an elevator. The extractions were performed under Nembutal anesthesia (Nembutal®, 30 mg/kg body weight intraperitoneally). In some instances additional anesthesia with inhaled ether proved necessary to carry out the extractions. Fractures of one or more roots occurred, but no further attempt was made to take the fragments out in order to avoid additional surgical trauma that might influence the repair of the sockets. The animals were fed laboratory animal pellets (*Erichsen*, 1966) and water *ad libitum* during the experiment.

Two animals were killed by exsanguination under Nembutal® anesthesia at each observation interval (Table 1) and at the same hour of the day to avoid differences in the mitotic rate. The postoperative intervals were based on the experience about cellular proliferation gained in a previous experiment in guinea pigs (*Johansen & Gilhuus-Moe*, 1969a). One hour prior to sacrifice each animal was injected intraperitoneally with 1 μc 3H thymidine per g body weight, specific activity 2.0 curies/mM.* The heads were skinned and immediately placed in Lavdowsky's solution (*Williams & Frantz* 1948) for fixa-

* Radiochemical Centre, Amersham, England.

Table I

Animal Weights in Grams and Postoperative Observation Periods

Animal Code	Observation Periods, Days 0	2	9	14	15	16	17	18	20
R 20 A	130	134	182	214					250
R 20 B	136	122	150	178					204
R 17 A	140	132	156	190			213		
R 17 B	150	152	188	220			238		
R 11 A			220	228					250
R 11 B			214	240					270
R 6 A			205	228	232				
R 6 B			186	190	190				
R 5 A			195	198					
R 5 B			200	154					
R 4 A				228				238	
R 4 B				220				216	
R 3 A				244			250		
R 3 B				240			248		
R 2 A				208		206			
R 2 B				220		214			
R 1 A						250	250		
R 1 B						230	232		

tion. The specimens were decalcified in 10 % formic acid and Win 3000®,** embedded in paraffin and serially sectioned at 5 μ in a frontal plane. Thereby the extraction alveolus and the intact first molar on the control sides of the mandible and the maxilla could be examined in one section. The sections were stained with hematoxylin-eosin and Alcian blue — PAS (*Mowry*, 1963). Others were prepared for autoradiography, dipped in Kodak NTB2*** emulsion at 49°C, exposed in the refrigerator at 4°C for 21 days and developed and stained through the emulsion with Harris hematoxylin. The coating and developing procedures were done in complete darkness.

** Winthrop Laboratory, New York, N.Y.

*** New England Nuclear Corporation, Boston, Massachusetts, U.S.A.

Initially a blind approach was used in the evaluation of the slides. However, after a quick scanning of the slides the chronological order of the healing pattern was easily distinguished whereafter the blind approach became unnecessary.

OBSERVATIONS

The animals tolerated the experimental procedure well and no deaths occurred in the postoperative periods. In all animals there seemed to be a lag in the normal weight gain for approximately 2—4 days after the operation (Table I). The table shows great variation in body weights and one of the rats even showed a loss of weight after a five day postoperative interval.

Fractures of one or more roots or parts of the bony alveolus occurred and obviously these complications interfered with the optimal repair of the extraction alveolus as reported by *Glickman et al* (1947) and *Pietrokovsky* (1967). However, these complications gave interesting information about the reactions in the periodontium of the remaining root in these instances.

In the following a presentation of the uncomplicated repair of the extraction alveolus is presented. The observations on the reactions around the fractured roots will be considered separately.

One day after extraction the alveolus was filled with a blood clot containing fibrin strands and blood cells (Fig. 1). Remnants of the periodontal fibers were easily distinguished along the normal alveolar wall. The autoradiographs showed labelled connective tissue cells along the alveolar wall (Fig. 2). The pocket epithelium and the oral epithelium did not show any more labelling than that on the control side.

The periosteum covering the (lateral) side of the mandible at the extraction side showed dense labelling of the cells (Fig. 3). On the control side no such increase in the number of labelled cells was seen (Fig. 4).

Two days after extraction the oral epithelium and the cuff epithelium on the extraction side were labelled more heavily than on the control side (Fig. 5). The periosteal labelling over the peripheral cortical plate of the mandible was higher than after one day and labelled cells were also seen between the fibers of the masseter muscle (Fig. 6). The coagulum showed a more advanced pattern of organization into granulation tissue with an increased number of labelled cells. Both bone apposition and bone resorption had started within the extraction alveolus.

After 3 days the extraction wound was filled with granulation tissue with many macrophages containing hemosiderin. Labelled fibroblasts were abundant. Bone apposition and bone resorption took place concomitantly along

PLATE I

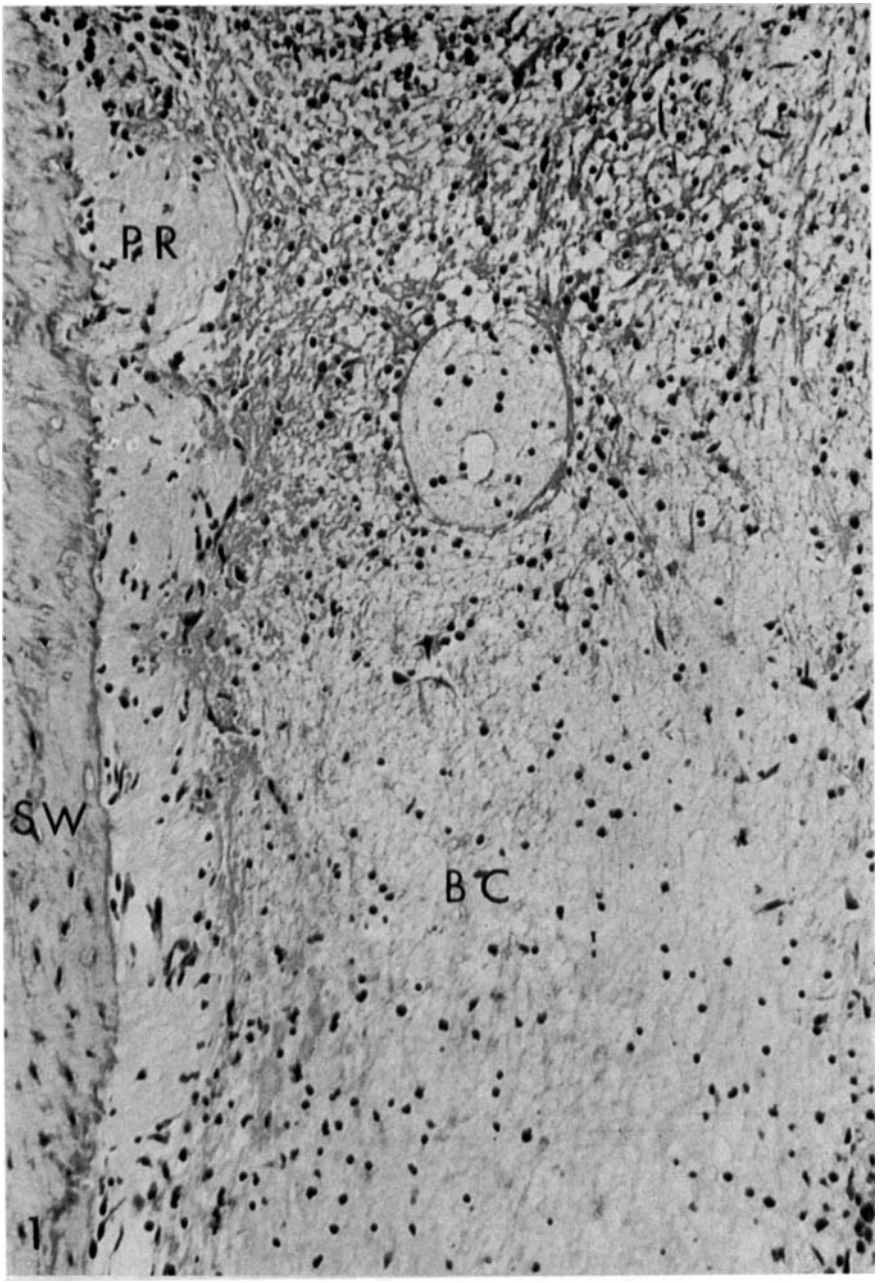
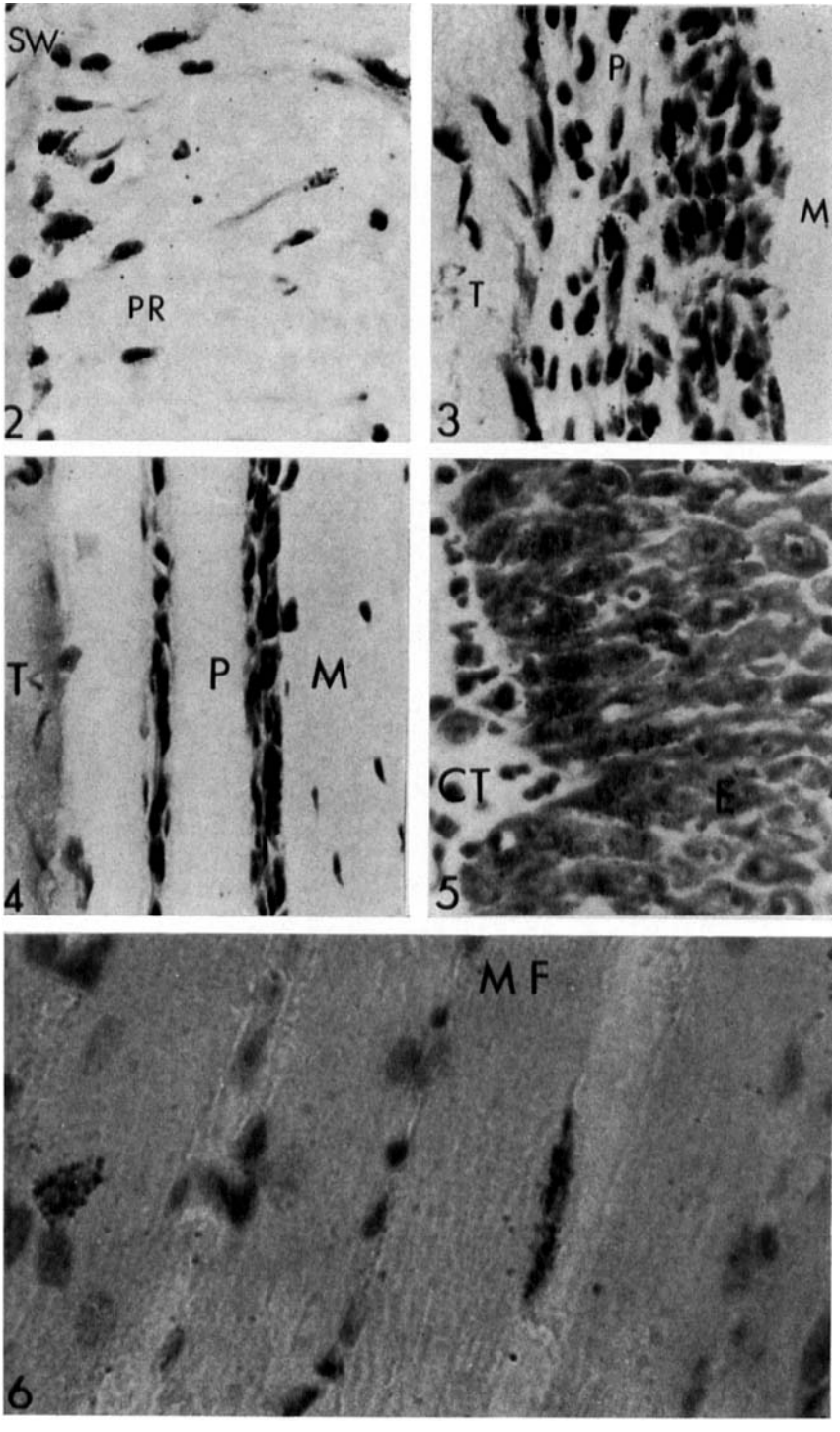


Plate I

Fig. 1. The alveolus one day after extraction. A blood clot (BC) fills the alveolus. Periodontal membrane remnants (PR) are seen attached to the socket wall (SW). Hematoxylin-eosin. Original magnification $\times 80$.

PLATE II



the alveolar wall of the socket (Fig. 7). Extensive bone apposition and labelling of the periosteal cells (Fig. 8) were also seen around the entire mandible outside the socket at the extraction site. The surface epithelium almost covered the wound.

After 4 and 5 days the periosteal labelling on the extraction side seemed to decrease. Even though this labelling was less intensive than after 3 days' observation time it was considerably higher than on the control side where only an occasional labelled cell was found. New bone was abundant towards the periphery (Fig. 9) as compared with the control side (Fig. 10). There was an intense bone formation with labelled osteoprogenitor cells in the alveolus. The surface epithelium now covered the wound and had started to keratinize. This epithelium showed more labelled cells than the epithelium adjacent to the wound.

At 6 days the alveolus was filled with new bone (Fig. 11). The epithelium was covering the wound completely, but the surface keratin layer was still thinner than normal. The periosteal labelling on the extraction side was now only slightly higher than on the control side. At this time bone apposition and labelled osteoprogenitor cells on the crest of the extraction alveolus were noticed. There were more labelled cells here than in the alveolus itself.

At 11 days the cellular labelling had decreased even more in the periosteum and the covering epithelium of the wound. Almost normal thickness of the keratin layer was present (Fig. 12). Bone apposition at the alveolar crest and on the trabeculae in the extraction alveolus was noticed but still the outline of the socket wall was clearly distinguishable (Fig. 13).

Plate II

Fig. 2. Labelled connective tissue cells in the periodontal membrane remnants (PR) after one day. The socket wall (SW) is seen to the left. Autoradiograph, hematoxylin. Original magnification ×320.

Fig. 3. From the same autoradiograph as Fig. 2. Many labelled connective tissue cells in the thickened periosteum (P) around the mandible (M) at the extraction site. (T) Attachment of the masseter muscle. Autoradiograph, hematoxylin. Original magnification ×320.

Fig. 4. From the same autoradiograph as Fig. 3. There is no labelling of the periosteum (P) at the control side. Mandible (M). Attachment of the masseter muscle T(). Autoradiograph, hematoxylin. Original magnification ×320.

Fig. 5. Two days after extraction. The gingival epithelium (E) close to the wound is densely labelled. Connective tissue (CT). Autoradiograph, hematoxylin. Original magnification ×320.

Fig. 6. The same specimen as Fig. 5. Labelled sarcolemma cells between the fibers (MF) of the masseter muscle at the extraction side. Autoradiograph, hematoxylin. Original magnification ×800.

PLATE III

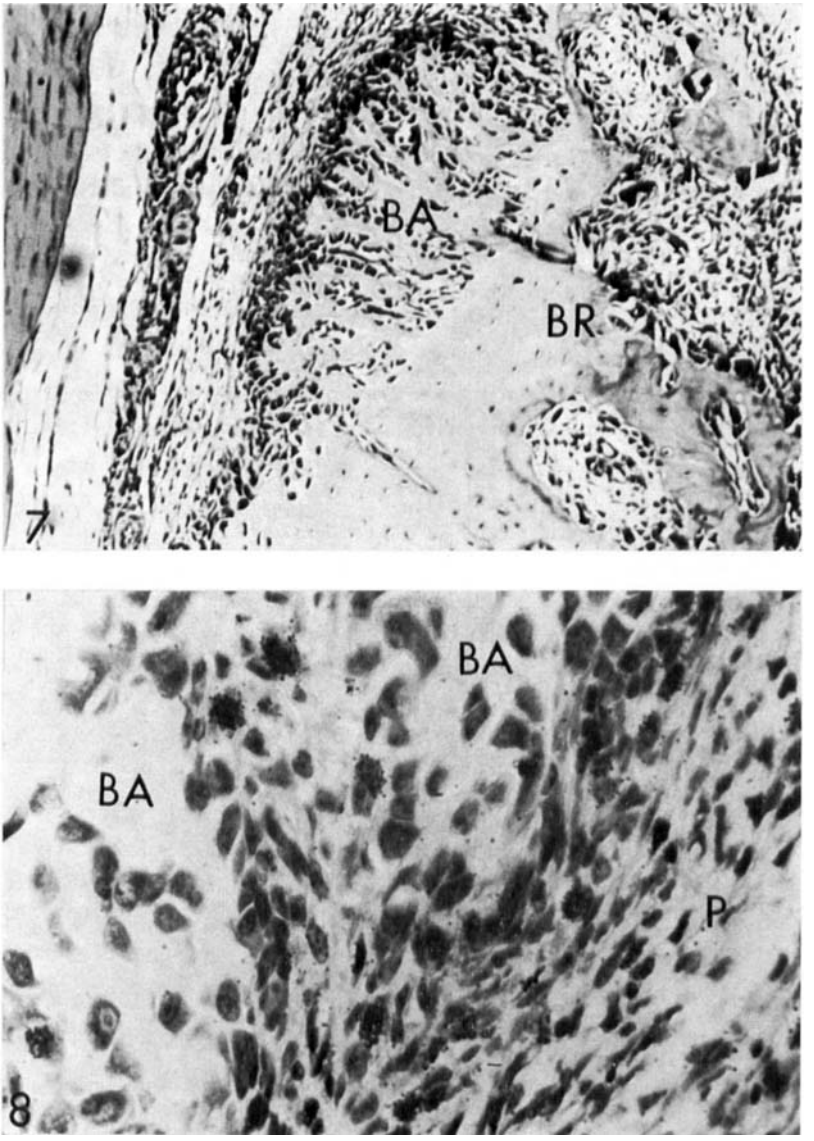
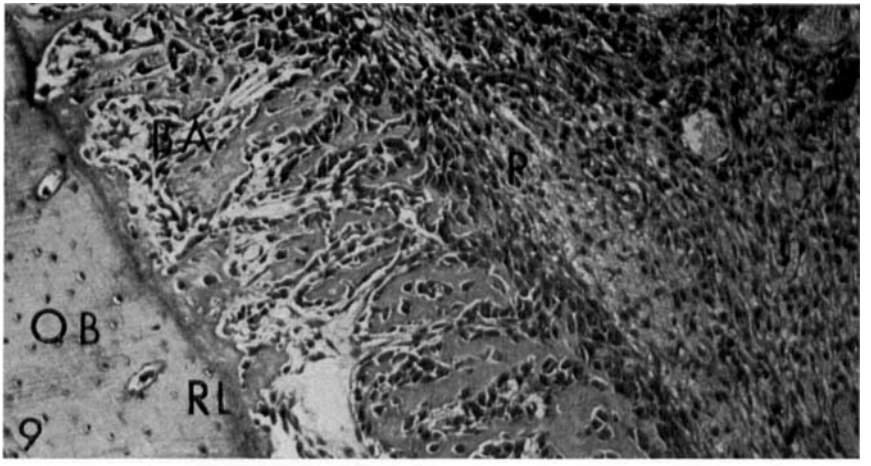
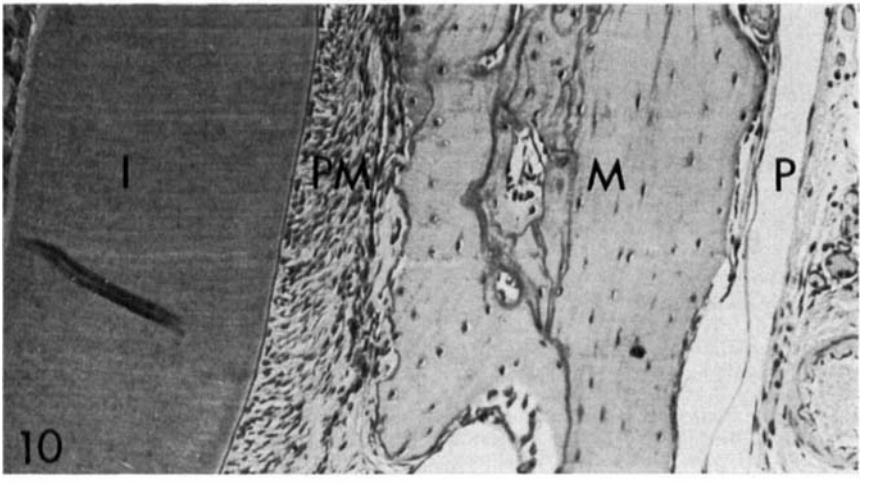


PLATE IV





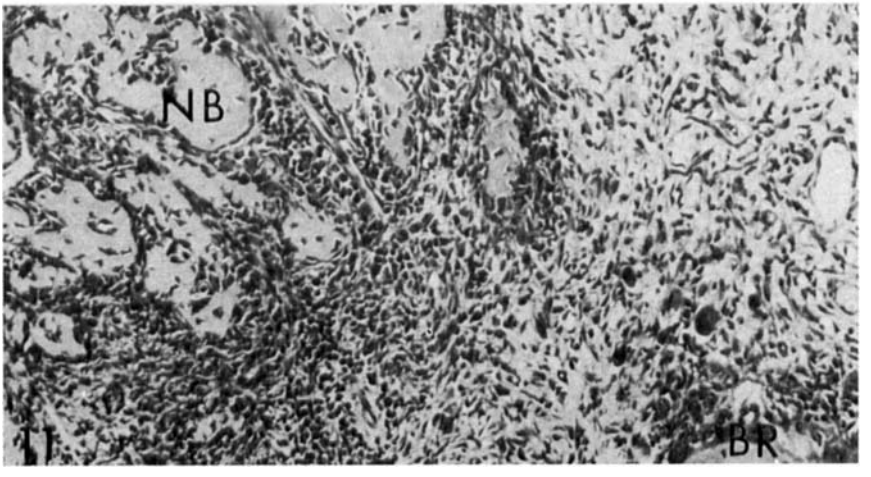


Plate III

Fig. 7. Three days after extraction. Bone apposition (BA) and bone resorption (BR) at the orifice of the postextraction socket. Hematoxylin-eosin. Original magnification ×80.

Fig. 8. From the same specimen as Fig. 7. Intense labelling of the periosteum (P) together with bone apposition (BA) on the buccal side near the base of the mandible at the extraction site. Autoradiograph, hematoxylin. Original magnification ×320.

Plate IV

Fig. 9. Four days after extraction. Abundant periosteal apposition of bone (BA) on the lingual side of the mandible on the operated side. The newly formed bone (BA) is as thick as the original mandibular bone (OB). »Resting line» of the mandibular bone (RL). Periosteum (P). Hematoxylin-eosin. Original magnification ×80.

Fig. 10. The same section as Fig. 9. No active bone formation is seen along the lingual side of the mandible on the control side. Compare the thickness of the mandible (M) in this illustration with the new bone (BA) in Fig. 9. Periosteum (P), periodontal membrane of the incisor (PM), incisor (I). Hematoxylin-eosin. Original magnification ×80.

Fig. 11. Six days after extraction. New bone is filling the alveolus. Bone formation (NB) and bone resorption (BR) are evident. Hematoxylin-eosin. Original magnification ×80.

Plate V

Fig. 12. Eleven days after extraction. The epithelium is keratinized (E). New bone (NB) is filling the postextraction alveolus. Hematoxylin-eosin. Original magnification ×20.

Fig. 13. A higher magnification of the same section as in Fig. 12. New bone (NB) is laid down on the original alveolar wall (AW) both on the tooth socket side (T) and the outside (OS). The original alveolar walls are easily distinguished by the resting lines (RL).

Plate VI

Fig. 14. Seventeen days after extraction. The frontal section of the specimen shows the increased thickness of the buccal part of the mandible on the extraction side. Compare the distance from the incisor (I) to the buccal surface of the mandible (BS) in this illustration to the same distance in Fig. 15. The new bone in the post-extraction alveolus (NB) is seen at the top of the picture. The hematopoetic marrow (HM) of the mandible is decreased. Mandibular nerve (MN). Hematoxylin-eosin. Original magnification ×20.

Fig. 15. From the control side of the same section as Fig. 14. Note the extent of hematopoetic marrow of the mandible as compared to Fig. 14. The distance from the incisor (I) to the buccal surface of the mandible (BS) is approximately one half of that at the operated side (Fig. 14). One root (R) of the first mandibular molar is seen at the top of the picture. Hematoxylin-eosin. Original magnification ×20.

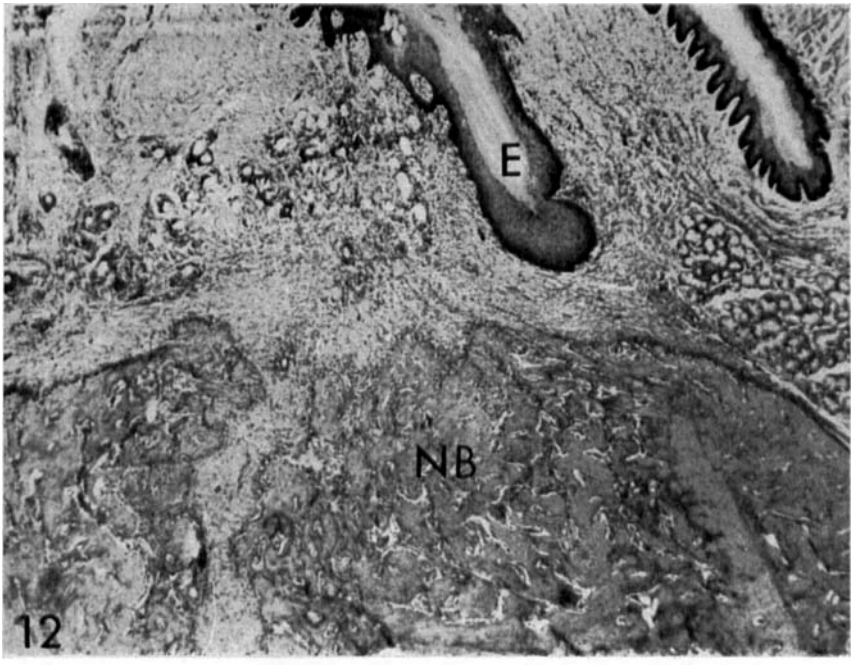
Plate VII

Fig. 16. Three days after extraction. Labelled endothelial and connective tissue cells mainly in the center of the periodontal membrane (PM) of a remaining root after attempted extraction. Cementum of fractured root (C). Alveolar bone (B). Autoradiograph, hematoxylin. Original magnification ×320.

Fig. 17. The periodontal membrane around a fractured root after 6 days. Labelling of connective cells in the center of the periodontal membrane (PM) is evident. Alveolar bone (B), cementum (C). Autoradiograph, hematoxylin. Original magnification ×320.

Fig. 18. From the control side of the same section as Fig. 17. There are less labelled connective tissue cells in the periodontal membrane (PM). Alveolar bone (B), cementum (C). Autoradiograph, hematoxylin. Original magnification ×320.

PLATE V



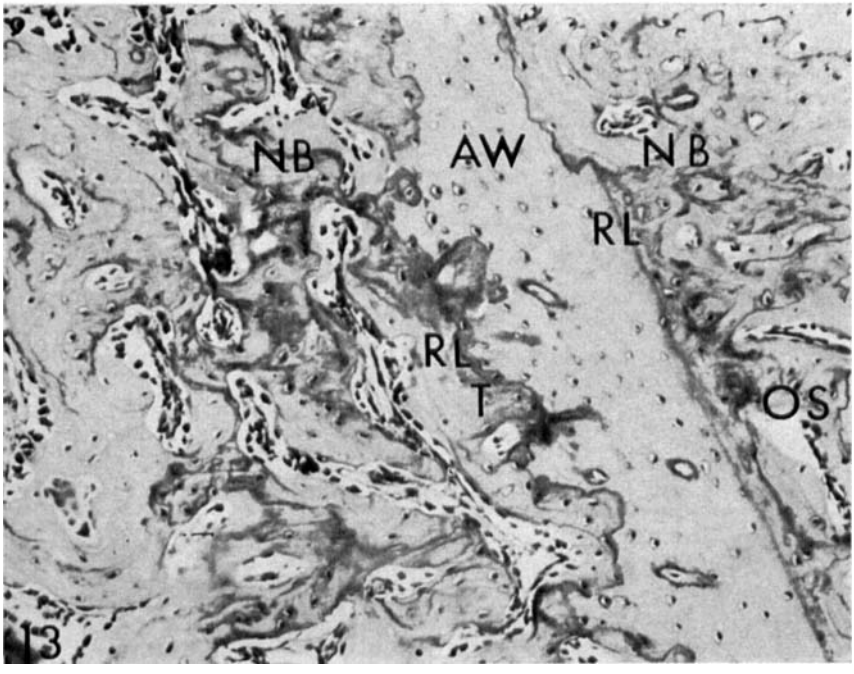


PLATE VI

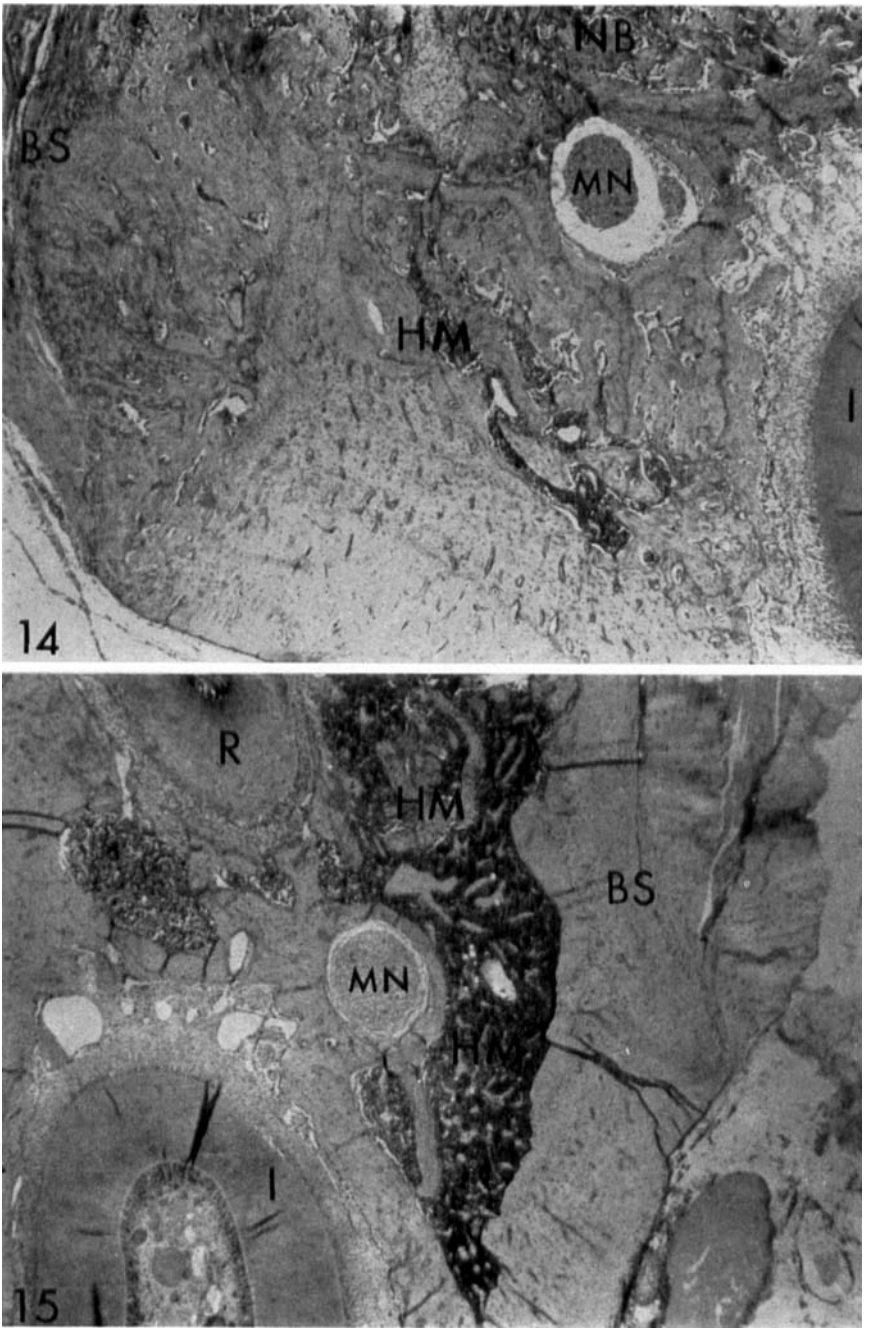
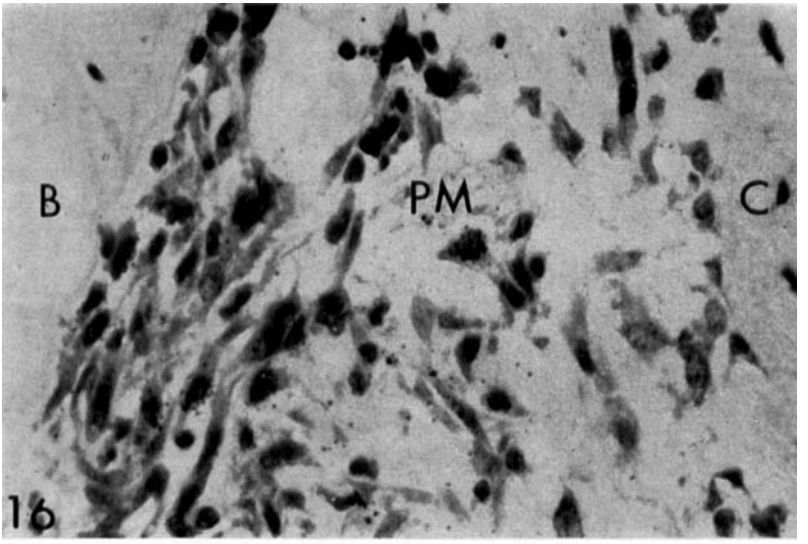
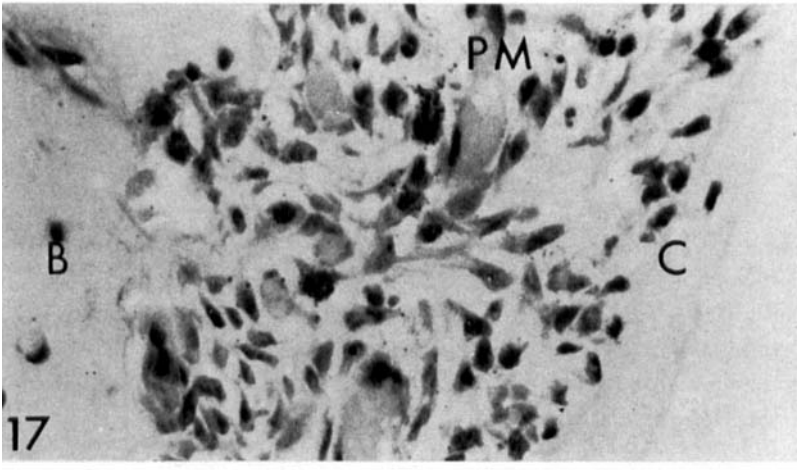
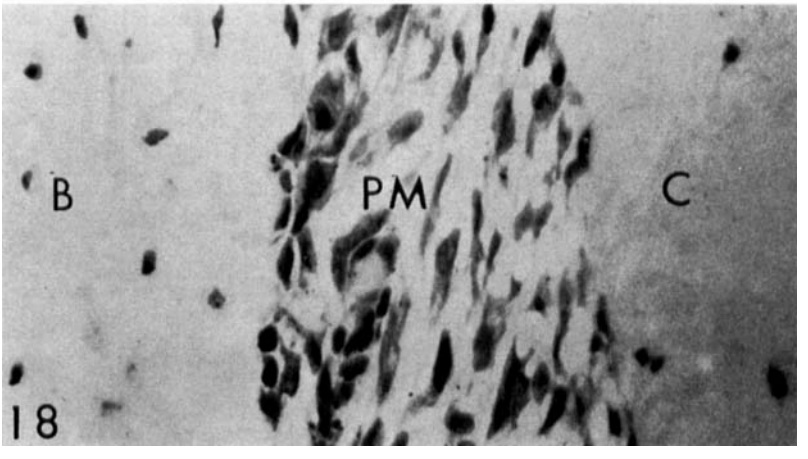


PLATE VII







At 17 and 20 postoperative days the overall situation was the same. Normal thickness and keratinization with a slightly increased number of labelled cells in the basal layers of the epithelium together with reorganization of the bone and apposition at the crest of the extraction alveolus were the general features. The mandible at the extraction site (Fig. 14) was twice as thick as that on the control side (Fig. 15) and a decrease in hematopoetic marrow was seen.

The presence of fractured roots in the alveolus delayed the migration of the epithelium over the wound. In some cases proliferating epithelium was seen to envelope bony sequestra of the marginal crest. The center of the periodontal membrane around the fractured roots showed marked changes. In the 3 day specimen connective tissue and endothelial cells were intensely labelled (Fig. 16), and after 6 days this labelling was even more pronounced (Fig. 17). Labelled epithelial rests of Malassez were also seen. The control side (Fig. 18) in the same section as Fig. 17 showed normal width and less labelling of the connective tissue cells of the periodontal membrane. The observation periods in this study were too short to observe the ultimate fate of the fractured roots.

DISCUSSION

A lag in the normal gain of body weight in the young rats was observed after the extractions. It is surprising to note that no comment has been made about this lag in weight gain postoperatively in previous investigations on extraction wound repair (*Huebsch et al*, 1952; *Smith*, 1958; *Pietrokovski*, 1967 and *Åstrand & Carlsson*, 1969). As previously reported (*Johansen & Gilhuus-Moe*, 1969a) the extraction of one mandibular incisor in he guinea pig affected the normal weight gain more than did the experimental fracture of the mandibular condyle (*Gilhuus-Moe*, 1969). In extraction experiments in small animals like rats (*Pietrokovski*, 1967) and mice (*Grewe & Felts*, 1969) the reduced gain in weight may be caused by interference with the chewing capability of the animals which may lead to inadequate nutrition and thereby influence the repair of the experimental wound.

Both the cuff epithelium and the oral epithelium of the gingiva were proliferating to cover the extraction wound by the second day. This confirms the observations made in a previous study in guinea pigs (*Johansen Gilhuus-Moe*, 1969a). The proliferation of both these parts of the gingival epithelium has not been mentioned by any investigator (*Huebsch et al*, 1952; *Smith*, 1958; *Pietrokovski*, 1967; *Åstrand & Carlsson*, 1969). The

cuff epithelium has been shown to have a high renewal rate under normal conditions (*Skougaard & Beagrie,* 1963; *McHugh & Zander,* 1965). Therefore, it is not surprising that this epithelium proliferated when stimulated. The fact that the cuff epithelium proliferated agrees with the modern concept of the cuff epithelium as a dynamic and constantly renewing epithelium quite different from the static tissue described by *Gottlieb* (1921) and *Orban* (1962). The autoradiographic techniques applied to this study facilitated the interpretation of cellular proliferation capacity in the repair of the post-extraction alveolus.

At 4 days after the experiment the epithelium covered the extraction wound and had started to keratinize, and at 11 days keratinization was completed. This was the normal sequence where no interferences by fractured roots or foreign bodies were present. In the cases of root fractures, the migration of the epithelium was delayed. The above-mentioned time intervals are somewhat different from other reports (*Huebsch et al,* 1952; *Smith,* 1958; *Åstrand & Carlsson,* 1969). It seems that a more qualified expression than »epithelium covered the wound,» would be desirable. The appearance of keratin may perhaps be a better criterion. Obviously, the presence of roots, bony fragments, and food impaction may be influencing variables in the migration of the epithelium. Therefore, this should be considered when reparative processes are studied.

The origin of the fibroblasts in the granulation tissue of repairing wounds is a controversial topic. Some authors state that preexisting fibrocytes in the wound take no part in the formation of new connective tissue components (*Gilman et al,* 1955; *Jackson,* 1961), and that cells of the mononuclear series of the blood have the ability to differentiate into fibroblasts (*Allgöwer,* 1956). On the other hand, evidence has been presented that migration of 3H thymidine labelled adventitial cells from the walls of the blood vessels takes place immediately after the appearance of leukocytes in the wound. Later these undifferentiated adventitial cells wer converted to fibroblasts and proceeded to proliferate and produce connective tissue components (*MacDonald,* 1959). In this study the alveolus was filled with a blood clot containing fibrin strands and blood cells after one day. Along the bony alveolus labelled connective tissue and endothelial cells were found amongst the remaining tufts of the periodontal membrane fibers after one day. In a previous study in guinea pigs (*Johansen & Gilhuus-Moe,* 1969a) the same pattern of labelling was noticed after 2 days. In both rats and guinea pigs the initial uptake of 3H thymidine appeared in cells along the bony wall of the extraction alveolus. Later labelled cells were found in the central portion of the clot. These labelled cells in the center of the clot were not found

in the vicinity of sprouting vessels. Their origin might therefore be cells of the monocytic series from the extravasated blood, as suggested by *Allgöver* (1956). It seems possible that proliferating endothelial cells, adventitial cells and mononuclear blood cells take part in the formation of the granulation tissue in the socket. In addition the undifferentiated connective tissue cells of the periodontal membranes of both the guinea pig incisor and the rat molar showed the initial uptake of 3H thymidine after extraction of the tooth. Whether this proliferation was stimulated by the appearance of leucocytes in the socket or the mechanical trauma connected with the extraction cannot be ascertained in this study. However, it seems safe to conclude that cells of the periodontal membrane remnants initially take part in the formation of the granulation tissue in the socket after extraction. These periodontal membrane remnants were gradually changed into granulation tissue and hyalinization as reported by *Euler* (1923) could not be verified.

After 3—4 days bone formation had started along the socket wall while resorption was seen in other parts. It appeared as if the transition from periodontal membrane remnants into granulation tissue and more mature connective tissue which was subsequently calcified took place in a fairly rapid course without resorption of the old socket wall as a prerequisite. This is somewhat different from the report by *Frandsen* (1963) who found that osteoclasts were found at the surface of the bony alveolus. This finding demonstrates that bone of the periodontium in rats is a dynamic tissue and that the resorption reported by *Frandsen* (1963) also occurs under normal conditions. In the present study bone formation took place directly on the original alveolar wall and no previous osteoclastic activity appeared essential for this process. Also, at the top of the alveolar crest noticeable bone formation occurred in the 6—21 days specimens. In contrast to the findings of *Åstrand* and *Carlsson* (1969) the outline of the original alveolar wall could easily be discerned after 21 days by the »resting line» in the bone.

In the guinea pig increased labelling of the periosteum at the extraction side compared to the control side was noted after 24 hours (*Johansen & Gilhuus-Moe*, 1969a). This feature was even more conspicuous in the present study in rats. *Åstrand* and *Carlsson* (1969) found increased activity of cells of the »scleroblastic type» along the bone margin opposite the distal socket of the first maxillary molar 48 hours after extraction in the rat. In the present study the labelling of osteoprogenitor cells of the periosteum was noticed in the 24 hour specimens. This increased activity on the extraction side seemed to reach its peak after 4—5 days, whereafter a gradual decline of the number of labelled cells was noticed. *Åstrand* and *Carlsson* (1969) reported that the »tetracycline activity» continued for about 7 days after ex-

traction. Differences in the examination techniques might very well explain this difference. Also it might be well to remember that definite conclusions about the exact sequence of reparative events should be proposed with caution in experiments with a small number of animals at different postoperative time intervals.

The observation of labelled cells in the sarcolemma of the masticatory muscles adjacent to the extracting wound was noteworthy. No such labelling was seen at the control side. This is in agreement with *Jackson* (1961), who found that labelled cells of the sarcolemma migrated and were a source of fibroblasts in the repair of the experimentally produced wound. A comparison of the results from experimentation in the rat and clinical experience obtained in the management of postoperative complications in humans after extractions seems farfetched. However, the possibility exists that a part of the postoperative swelling in humans in some cases might be attributable to cellular proliferation in the sarcolemma of the masticatory muscles after the surgical procedure.

The periosteal proliferation of osteoprogenitor cells and bone formation at a distance from the extraction wound are not easy to explain. It might be a compensatory mechanical reinforcement of bone to tolerate the stresses of mastication (*Arwill* & *Ågren*, 1968; *Åstrand* & *Carlsson*, 1969). But the same changes take place even after reimplantation (*Johansen*, 1970a). Cell proliferation is a common response in inflammation. It may be that the observed increase in the uptake of 3H thymidine in the periosteum, in the neighboring periodontium (*Johansen* & *Gilhuus-Moe*, 1969b), and in the epithelial rests of Malassez (*Johansen*, 1970b) is a response to certain stimuli of biochemical nature rather than to mechanical stress alone. Of course, speculations in many directions are possible. The autoradiographic methods might further contribute to clarify this hypothesis.

The unintentional tooth fractures following the extractions gave additional information about the reactions of the periodontal membrane. The increased number of labelled cells especially in the center of the periodontal membrane of fractured teeth at various observation times seems to justify some comments. The periodontal membrane is a dynamic tissue which responds to stimuli in different ways according to the nature of the stimulus. From these and previous autoradiographic observations it is clear that an increase in cellular proliferation (determined by the uptake of 3H thymidine) of the center of the periodontal membrane is a reproducible effect that follows the experimentation. Whether or not an intermediate plexus exists is still controversial, but the observed increase in labelled cells of the middle portion of the periodontal membrane (the intermediate plexus) in the neigh-

boring tooth after extraction (*Johansen & Gilhuus-Moe*, 1969b) indicates that this part of the periodontal membrane is reacting rapidly to changes in the normal homeostatic state. The same reactive capacity was observed in the present experiment as shown by the increased labelling in the periodontia of the fractured teeth and also by the labelling of the cells along the alveolar wall after extraction as the initial cellular response. Theories about different races of fibroblasts have been forwarded (*Godman*, 1963; *Hsu*, 1963). If such races exist, they might be present in the periodontal membrane and may include the connective tissue cells that react so rapidly when the normal situation is interfered with by experimentation.

The repair of the extraction wound is a widely used experimental model because the extraction of teeth is the most common surgical procedure in the dental office. The introduction of autoradiographic techniques has given additional information about the initial cellular proliferation in the repair of the socket. It seems, however, that this experimental model may also be suitable for research in the field of wound healing in general, and that information gained from this model may further clarify the controversial opinions about the source and origin of the cells that take part in the repair of wounds.

SUMMARY

The first mandibular molars in 18 young rats were extracted and the repair of the sockets was studied by histology and autoradiography with 3H thymidine. The following observations were made:

Cells of the periodontal membrane remnants in the alveolus and of the periosteum of the mandible at the extraction site showed uptake of 3H thymidine before any other cells.

The sources of the proliferating fibroblasts in the clot seemed to be cells of the periodontal membrane remnants and vessels along the alveolar wall. Bone formation started on the surface of the mandible at the extraction site before bone was formed in the alveolus itself.

Bone formation in the alveolus took place directly on the original alveolar wall without prior resorption.

Cells of the middle portion of the periodontal membrane of fractured teeth had a high uptake of 3H thymidine which suggested that this zone responded rapidly to changes in the homeostatic state.

The use of the post-extraction alveolus as an experimental model in the study of the source of fibroblasts in wound repair is suggested.

RÉSUMÉ

CICATRISATION DE L'ALVÉOLE APRÉS EXTRACTION CHEZ LE RAT WISTAR
ÉTUDE HISTOLOGIQUE ET AUTORADIOGRAPHIQUE

Les premières molaires inférieures de 18 jeunes rats ont été extraites et la cicatrisation de leurs alvéoles a fait l'objet d'une étude histologique et autoradiographique en utilisant la thymidine-^{3}H. Les observations suivantes ont été faites:

Les cellules des restes du desmodonte dans l'alvéole et les cellules du périoste mandibulaire au niveau de l'extraction présentaient une absorption de thymidine-^{3}H avant toutes autres cellules.

L'origine des fibroblastes qui proliféraient dans le caillot sanguin semblait être des cellules des restes du desmodonte et des vaisseaux accolés à la paroi alvéolaire. La formation osseuse débutait à la surface de la mandibule au niveau de l'extraction avant qu'il ne se forme de tissu osseux dans l'alvéole lui-même.

La formation osseuse dans l'alvéole se faisait directement sur la paroi alvéolaire originelle, sans résorption préalable.

Les cellules de la partie moyenne du desmodonte des dents fracturées présentaient une absorption élevée de thymidine-^{3}H, ce qui semblait indiquer que ces zones répondaient rapidement aux modifications de l'état homæostatique.

L'auteur indique la possibilité d'utiliser l'alvéole après extraction comme modèle expérimental pour l'étude de l'origine des fibroblastes dans la cicatrisation des plaies.

ZUSAMMENFASSUNG

HEILUNG DES ALVEOLUS NACH ZAHNEXTRAKTION BEI DER WISTAR-RATTE
EINE HISTOLOGISCHE UND AUTORADIOGRAPHISCHE UNTERSUCHUNG

Die ersten Unterkiefermolarzähne von 18 jungen Ratten wurden ausgezogen, und die Heilung der Alveoli mit 3H Thymidin histologisch und autoradiographisch untersucht. Folgende Beobachtungen wurden gemacht:

Zellen der verbliebenen periodontalen Membrane des Alveolus und Zellen des Unterkieferperiosteums an der Extraktionsstelle zeigten Aufnahme von 3H Thymidin vor allen anderen Zellen.

Die Quelle der wuchernden Fibroblasten im Blutgerinnsel schienen Zellen der verbliebenen periodontalen Membrane und der Gefässe entlang der Alveolarwand zu sein.

Knochenformation begann an der Oberfläche des Unterkiefers an der Extraktionsstelle, bevor Knochen im Alveolus selbst geformt wurde.

Knochenformation im Alveolus fand direkt an der ursprunglichen Alveolarwand statt, ohne vorausgehende Resorption.

Zellen des mittleren Teiles des periodontale Membrane von frakturierten Zähnen hatten eine hohe Aufnahme von 3H Thymidin, welches vermuten lässt, dass diese Zone schnell auf Veränderungen im homoostatischen Zustand reagiert.

Der Gebrauch vom Postextraktionsalveolus ist als ein experimentelles Model in der Untersuchung der Quelle von Fibroblasten in Wundheilung vorgeschlagen.

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Address:
The Department of Periodontology,
Dental Faculty,
University of Oslo,
Norway