From the Surgical Department of the Dental University Clinic, Helsinki.
(Head: Prof. JUUSO KIVIMÄKI, M. D.)

Penicillin for Treatment of Inflamed Dental Pulp.[1]

By

MAURI POHTO.

616.314.1800208

Introduction.

During the 19th century and the beginning of the 20th the question of healing inflamed dental pulp has been extensively studied. Attempts were made with a variety of antiseptic substances to destroy micro-organisms in the dentin and in the pulp without damaging the latter.

The studies in which almost every dental scientist of that time participated, finally led to a result which can be briefly expressed by PECKERT's (1913) classical sentence: Inflamed pulp is a lost organ.

Subsequently a root therapy based on devitalization became prevalent in the treatment of pulpitis and it is still in general use.

However, vital amputation is an exception. Its practical value has so far been slight, yet the life of the root pulp may be preserved by this method at least in cases of acute serous partial pulpitis and open chronic pulpitis, provided the patient is young and the power of resistance good.

ENTIN's method may be regarded as a unique experiment to retain the life of dental pulp. He first exposed the inflamed pulp, but did not undertake pulpal amputation. He then continued his treatment by covering the open surface of the pulp for 48 hours with acidophilic rods insulated from the mouth and capping the surface with dentin powder or sterile gypsum.

[1] Paper read at the Meeting of the Finnish Association of Dentists, October 25, 1946.

His method is based on the fact that the mentioned microorganisms are antagonistic to pathogenic streptococci. His material comprised 135 cases of pulpitis; in 111 of these clinical and roentgenological follow-up examination revealed that the treatment had been successful. A microscopic specimen was made only of one tooth, and thus the results of treatment cannot be conclusively proved.

FLOHR reported one case of pulpitis in which pain had been present for the duration of about two days. He capped the exposed pulp with vitapulp, a slightly alkaline substance containing calcium salts and dentin powder. Microphotographs revealed that healing had taken place and that new dentin had formed over the pulp chamber.

Neither ENTIN's nor FLOHR's method was of any practical importance. They deserve mention, however, as root treatment was replaced by pulp treatment, and both these investigators have been able to demonstrate by one microscpical specimen that healing of inflamed dental pulp is possible.

Cases of spontaneous healing of inflamed dental pulp have been reported in the literature. They are exceptions, however. Inflamed pulp always becomes destroyed if the cause of the inflammation cannot be eliminated.

Pulpitis is caused by bacteria and their toxins and their route to the pulp is mostly by a carious focus and the dentin canals.

According to HARNDT, the bacteria have not generally reached the pulp in acute serous inflammation, but the disease is caused by their toxins. In cases of chronic pulpitis there are bacteria only on the exposed pulpal surface, but in cases of purulent pulpitis great numbers of bacteria are already present in the pulp.

If the action of bacteria in the carious focus, in the dentin canals, and in the pulp itself can be effectively prevented, without damage to the pulp, the necessary conditions are created for healing. The subsequent course of healing depends on the tissue injuries which have arisen during the inflammation.

According to HARNDT, the histologic picture of serous pulpitis shows degenerative changes in the odontoblast layer in the area of the carious focus. In the partial phase serous exudate accompanied by leukocyte infiltrations is found in the coronal part of the pulp. The total phase differs from the partial phase only as regards the extent of the inflammatory changes, and histologic differentiation between these two phases is not always easy.

When the pulp has been invaded by great numbers of bacteria, the histologic picture of the pulp changes considerably. The odontoblasts have been completely destroyed, at least in the area of the carious focus. Pus cells predominate in the cell infiltration. Their ferments rapidly destroy tissue, either in a limited area, an abscess then being formed, or diffusely, in which case a phlegmon develops. Depending on the extent of the inflammatory process a partial and a total phase may be distinguished.

Destructive changes in the tissue are not noted in the ulcerative or in the granulating stage of chronic inflammation elsewhere than in the superficial layers of the pulp, provided that the cleaning of the open pulpal surface has not been prevented in some way.

Elimination of inflammatory products and replacement of necrosed parts of tissue are dependent on the vitality of the pulp. In this respect the patient's age and general power of resistance is important, as are the individual local conditions in the apical part of the roots of each tooth. The blood vessels' route to and from the pulp leads through a narrow and unyielding passage in the apical part of the root canal where considerable variations may be observed in the individual cases. In hyperemia the dilatation of arterial vessels leads directly to a decrease in the dilatation of veins, which results in retarded circulation, occasionally even in complete stasis.

Retardation of the blood flow easily causes metabolic disturbances which in turn may impede or even completely prevent healing of the pulp.

In case bacterial action in a tooth affected with pulpitis is actually prevented without damaging the pulp, the prognosis thus seems good in cases of acute serous and open chronic pulpitis on the basis of the histo-pathological picture. Consequently the vitality of the pulp is the determining factor as regards the result of treatment.

In purulent pulpitis greater vitality of the pulp is required for the repair of tissue injuries. In the partial phase the prognosis is thus much graver than in the cases mentioned above, and in total pulpitis cure is scarcely possible.

The Author's Investigations.

Since the spring of 1945 I have carried out experiments on patients in an effort to find out whether healing of inflamed dental pulp is possible.

For the purpose of preventing bacterial action in the dentin and the pulp a prontosil derivative soluble in water was initially used. This therapy was successful in a case of chronic ulcerative pulpitis. When the temporary filling was opened one year later the tooth proved to be vital and asymptomatic. The second case treated with prontosil, a serous total pulpitis, failed to respond to treatment.

Experiments were continued with marfanil. Three cases of serous partial pulpitis were treated, and they were all found to be symptom-free when clinically examined one year later. Of 3 cases of serosa totalis one was a treatment failure, the others were still asymptomatic when clinically examined after one year.

At the end of August 1945 Prof. P. E. SIMOLA, the Head of the Department of Medical Chemistry of the Helsinki University, made arrangements to provide me with penicillin for my experiments. In this connection I beg to offer my best thanks for this assistance and the support which he has given me during the progress of my work.

With penicillin I treated 6 cases of serous partial pulpitis, 5 cases of serous total pulpitis, one purulent partial pulpitis, and one case of chronic ulcerative pulpitis. Of these, one case of serous total and one of purulent partial pulpitis were treatment failures. The others were asymptomatic at the clinical follow-up examination carried out 2 to 8 months later.

By means of clinical examination it is not, however, possible to determine conclusively whether the treatment has been a success, and without the aid of histologic specimens we cannot get an idea of how the process of healing takes place.

Consequently I turned to the Chief of the Surgical Department of the Dental University Clinic at Helsinki. Prof. JUUSO KIVIMÄKI, asking for permission to collect a series of patients from his department who were willing to have a pulpitic tooth extracted.

Prof. KIVIMÄKI very kindly placed the required material at my disposal. He also followed the progress of my work with interest and gave me valuable assistance in word and deed. My sincere gratitude is due to Prof. KIVIMÄKI.

I take the opportunity of thanking also Prof. ARNO SAXÉN, Chief of the Department of Pathologic Anatomy at the University of Helsinki, who has kindly gone through the histo-pathological part of my investigation. I also thank the Council of Suomen Kulttuurirahasto (the Finnish Cultural Foundation) for the grant which enables me to carry out my investigation.

Material and Technique.

My material comprises a total of 10 cases; in each case the patient was willing to have a pulpitic tooth removed on account of pain or with a view to prosthetic replacement. The collection of the material was very difficult as the number of persons now applying for treatment at the dental clinic has decreased considerably owing to the present wage level. Furthermore, the majority of the patients wished to have their tooth removed immediately. Several of them probably went to other dentists, as only 14 of the 22 patients treated returned after the control period to have their tooth removed. Of these 14 teeth histologic specimens were prepared; in 4 cases, however, they resulted in complete failure on account of the laboratory assistant's lack of skill.

As regards the clinical diagnosis my 10 cases were distributed as follows:

Pulpitis ac. serosa partialis	2
» » » totalis	5
» chr. ulcerosa	3

The distribution of the cases was as follows with respect to the different teeth: 8+, 7+, 5+, +8 (3 cases), 6— (2 cases), —7, and —8.

The age of the patients varied from 17 to 35 years.

My experiments were carried out exclusively for the purpose of orientation. Therefore, I have not aimed at a careful treatment of each individual case with a view to obtaining as good end-results as possible, but the cases have been treated schematically.

In treating cases of pulpitis relief from pain should generally be considered in the first place. For this purpose I have used alypinum nitricum, which is an easily soluble substance of low toxicity and causes relaxation of the nerve endings.

To prevent the growth of bacteria in the dentin and in the pulp I have used penicillin which is highly bacteriostatic against gram-positive cocci, even if used in weak dilution.

According to SIEBERTH, MILLER, ENTIN etc., the deep layers of carious dentin contain chiefly streptococci. HARNDT designates the bacteria he has found in inflamed pulp as micrococci in most instances. It is probable, however, that at least the greater part of the bacteria observed in cases of pulpitis are penicillin-sensitive gram-positive cocci.

The drawback of penicillin is its exceptionally unstable chemical structure. It is destroyed fairly rapidly at a temperature above 10° C., also in an aquaeous dilution, and due to the action of acids and alkalis. At present four varieties of penicillin with a different antibiotic effect and stability are known. The sodium salt of penicillin which I have used is an Abbott manufacture; neither its quality nor its stability is stated, however.

For my experiments I dissolved 100,000 Oxford units of penicillin into 1 cc. of physiological saline. Thus I obtained a solution which was 400 times stronger than that which, according to the instructions of the manufacturer, is used in local treatment of infected wounds.

I used this high dilution, as the decrease of the potency of the solution during the control period at a temperature of less than 10° C. could not be established with certainty. Neither was it possible to estimate the amount of penicillin destroyed in the tooth before reaching the pulp. The penicillin solution used was 2 to 53 days old.

In local treatment of open wounds about 24 hours are required for the penicillin to become resorbed. It is scarcely probable that resorption would be more rapid in an organ which is as isolated as the tooth.

If bacteriostasis is produced in a tooth affected with pulpitis, the pulp must be protected from new infection. At the same time endeavours may be made to promote the activity of the healed pulp into forming hard tissue to cover the open pulp or to strengthen the roof of the closed pulp.

Consequently I have used a paste containing sulfathiazole and calcium glycerophosphate mixed with the penicillin solution mentioned above and with vigantol.

Sulfathiazole is substance of low solubility and thus remains long in the cavity. Its bacterostatic activity is considerable. The rôle of penicillin is partly the same as in the previous stage of treatment, although it does not penetrate into the dentin canals and into the pulp as easily when dissolved in vigantol as in saline solution. Because vigantol retards resorption the penicillin has partly the same task as sulfathiazole, viz. to prevent reinfection.

Calcium glycerophosphate and vigantol are added to promote the activity of the pulp itself in forming hard tissue to cover the exposed pulp or to strengthen the roof of the closed pulp.

Calcium glycerophosphate is an organic compound easily soluble in water. If the pulp can utilize a surplus of calcium and phosphorus, they are thus available.

Vigantol manufactured by the firm Merck u. Bayer is crystallized vitamin D_2 dissolved in vegetable oil. The rôle of vitamin D in the calcium metabolism is remarkable. In addition, vitamin D_1 when placed in the cavity, stimulates odontoblast activity, according to WEINMANN.

After treatment I have sealed the cavity with phosphate cement to fix the temporary filling as firmly as possible, yet at the same time avoiding pressure. Because of its high acid content the use of phosphate cement together with penicillin is not suitable. Phosphate cement is difficult to replace, however, as durability and adhesion are of great value in a moist cavity. In my experiments the high dilution of penicillin is partly accounted for by the simultaneous use of phosphate cement.

I fixed the control period at two months. Nevertheless, it often happened that the patient wished to have the tooth extracted earlier. Sometimes the patient did not appear for follow-up examination until more than 3 months had elapsed. On this account the control period varied from 43 to 100 days.

The inflamed pulp was treated as follows:

The lateral surfaces of the cavity were cleared of carious dentin; from the floor only the chips of loosening dentin were removed.

The cavity was swabbed with cotton and a small cotton ball immersed in penicillin solution was placed on the bottom. For pain relief a slight amount of alypin was added to the penicillin solution in each separate case. The cavity was closed with phosphate cementum, pressure being avoided.

On the following day the cavity was opened, the cotton ball removed, and a paste, for the purpose of protecting from reinfection, was lightly applied to the floor of the cavity. The cavity was then sealed with phosphate cementum; pressure was avoided.

In cases of chronic pulpitis the first stage of the treatment was abandoned and the paste was applied directly onto the surface of the exposed pulp.

As data on the use of penicillin in dentistry were not available at that time, I used the simplest possible therapeutical method: Penicillin treatment only once, followed by application of a paste protecting from reinfection; in cases of chronic pulpitis the paste was applied immediately.

After extraction the teeth were fixed in 10 % formalin and the paraffin method, according to WESTIN, was used for preparing the microscopic specimens.

The specimens were stained with hematoxylin-eosin.

Twenty-two microphotographs illustrate the results.

Results.

For the purpose of giving a clear picture of the results of my investigations, my cases are reported in two groups.

1: the cases in which microscopical examination revealed only local changes in the coronal part of the pulpal tissue.

2: the cases in which the changes in the pulpal tissue were diffuse and extended over the whole area of the pulp.

Group 1.

C a s e 1. Age 28 years. *Subjective symptoms:* Pain earlier on several occasions. At present tenderness felt only when masticating.

Clinical examination: Large carious focus in 8+, open pulp on bottom of focus.

Clinical diagnosis: Pulp. chr. ulc. dent. 8+.

After treatment a control period of 79 days, no symptoms during that time.

Histologic findings: The section did not run through the plane of the opening in the pulpal roof. The protective dentin next to the carious dentin is fairly thick. Figs. 1 a and b. In this area the odontoblasts are degenerated and have partly disappeared completely. In other parts they are regular. The connective tissue of the coronal pulp is slightly increased. The greater part of the coronal pulp is formed by a cavity the lateral surfaces of which are ragged. The fissures continue as far as to the dentin. The pulpal tissue is normal in other places. Fig. 1 c. No inflammatory cells are visible.

Conclusions: The cavity in the coronal pulp and the fissures in the dentin are seen also in the paraffin block. They may, however, have arisen earlier in the course of preparation of the specimen. If not, the cavity is probably an emptied abscess which has developed just before the roof of the pulp was exposed, as the process was becoming chronic. Accordingly, the patient has evidently come under treatment at this time, as the granulation tissue, which regularly develops in an open wound, or the connective tissue scar replacing it are not visible. The complete absence of inflammatory cells in the area of the cavity can be due only to a successful therapy and to the long (79 days) control period.

The slight increase of connective tissue, in the area of the coronal pulp only, indicates that the changes taking place at the time of the inflammation — with the exception of a potential abscess — have been slight and limited to the area of the coronal pulp. Microscopic examination reveals that healing has taken place.

C a s e 2. Age 25 years. *Subjective symptoms:* Pain one week earlier. Later no symptoms.

Clinical examination: Large carious focus in +8, open pulp on its bottom.

Clinical diagnosis: Pulp. chr. ulc. dent. +8.

A control period of 44 days followed the treatment. No symptoms during that time.

Histologic findings: The section did not run through the plane of the opening in the pulpal roof. Next to the carious focus the protective dentin is fairly thick. In this area the odontoblasts have completely disappeared or have become degenerated. They are regular in other parts. Granulation tissue with numerous blood vessels is observed adjoining the carious focus in the pulp. Fig. 2. The connective tissue is here markedly increased and delimits the granulation tissue fairly sharply from the regular connective tissue of the pulp. Slight, scattered lymphocytic and plasma cell infiltration is seen in the region delimited by connective tissue.

Conclusions: The section was made quite close to the opening in the pulpal roof, as the granulation tissue of the open wound in the pulp is visible. The increase of connective tissue and the small number of inflammatory cells show that the granulation tissue is beginning to form into scar tissue. The microscopic examination shows that healing is in progress.

C a s e 3. Age 28 years. *Subjective symptoms:* Pain earlier on several occasions. Now tenderness only when masticating.

Clinical examinations: Large carious focus in +8. Pulp exposed on bottom of focus.

Clinical diagnosis: Pulp. chr. ulc. dent. +8.

A control period of 79 days followed the treatment. No symptoms during that time.

Histologic findings: The section was oblique to the extent that the opening in the roof of the pulp is not seen. Next to the carious focus the protective dentin is fairly thick. At that point vacuolization is observed in the odontoblast layer or odontoblasts are completely absent. Elsewhere they are regular. The connective tissue of the pulp is slightly increased on the side of the carious focus. A strong inflammatory cell infiltration is seen in the pulp; in addition to lymphocytes and plasma cells, pus cells are observed. Figs. 3 a and b. Excepting hyperemia, the pulp tissue is regular in other parts.

Conclusions: The slight increase of connective tissue and the strong inflammatory cell infiltration accompanied by pus cells indicates failure of treatment.

Case 4. Age 22 years. *Subjective symptoms:* Pain for a fortnight, occasionally also at night. Cold or heat causing pain was not noted. Tenderness on mastication for about two days.

Clinical examination: Large carious focus in 6—. Sharp "shooting" pain caused by cold, considerably slighter by heat. Tenderness to percussion in the longitudinal direction.

Clinical diagnosis: Pulp. ac. ser. tot. dent. 6—.

After treatment a control period of 91 days. No symptoms during that time.

Histological findings: A comparatively thick layer of predentin is seen in the pulpal horn in the area of the carious focus. At this point of the odontoblast layer vacuolization is present, elsewhere the odontoblast layer is fairly regular. Figs. 4 a and b. The connective tissue of the pulp is increased in the region of the horn on the side of the carious focus. In the centre of the coronal pulp there is a small concentration of connective tissue and a few calcified portions are seen. Fig. 4 c. In other parts the pulp tissue looks regular. No inflammatory cells are visible.

Conclusions: In the area of the horn, in the immediate neighbourhood of the carious focus, an inflammatory process has been in progress. A connective tissue scar has formed in this area. The connective tissue concentration, as well as the calcified points in the centre of the coronal pulp, may be connective tissue scars or calcified scars resulting from metastatic foci at the time of inflammation. Histologic study shows that healing has taken place. The odontoblast layer being preserved, although degenerated, points to bacteria not having penetrated into the pulp. Accordingly, the case should be regarded as a serous partial pulpitis.

Case 5. Age 17 years. *Subjective symptoms:* Pain during one week, caused by cold and heat. Slight tenderness on mastication.

Clinical examination: Large carious focus in —7. Both cold and heat cause moderate pain. Slight tenderness to percussion in the longitudinal direction.

Clinical diagnosis: Pulp. ac. ser. tot. dent. —7.

A control period of 68 days followed the treatment. No symptoms during this time.

Histologic findings: In the entire area of the pulpal roof there is a remarkably thick layer of protective dentin which is more regularly calcified in the area adjoining the odontoblast layer than farther out. Figs. 5 a and b. The odontoblasts next to the carious focus are degener-

ated and partly completely destroyed; in other parts they seem to be fairly regular. In the centre of the pulp there is a large cavity the margins of which are partly deeply ragged. Granulation tissue has grown into the cavity at one point. In the pulpal horn, on the side of the carious focus, the connective tissue is considerably increased. Fig. 5 c. It is coarse and hyalinized. Around the blood vessels in this area there is a slight infiltration of lymphocytes and plasma cells. Fig. 5 d. Excepting the changes described, the pulpal tissue is regular.

Conclusions: The cavity in the coronal part of the pulp is seen also in the paraffin block, but may nevertheless be an artefact, as in Case 1. The granulation tissue in a marginal part of the cavity points to an emptied abscess. In the pulpal horn, close to the carious focus, a strong inflammatory process has been present. The horn is now filled by a connective tissue scar and the granulation tissue at its base with its small number of inflammatory cells shows that healing is still in progress. The partial destruction of the odontoblast layer in the area of the horn indicates that bacteria have penetrated into the pulp. Accordingly, the case is a purulent partial pulpitis?

Group 2.

Case 6. Age 31 years. *Subjective symptoms:* Slight pain occasionally during one week. Sharp "shooting" pain caused by cold. No tenderness on mastication.

Clinical examination: Carious focus in +8. Cold gives moderate pain, heat none at all. No tenderness to percussion.

Clinical diagnosis: Pulp. ac. ser. part. dent. +8.

A control period of 83 days followed upon the treatment. No symptoms during that time.

Histologic findings: The layer of protective dentin is thin in the whole area of the pulpal roof. The odontoblast layer appears to be fairly regular everywhere. Fig. 6 a. In the hyperemic pulp the connective tissue is increased throughout, especially in the area of the pulpal horns and around the opening of the root canal. In the latter area, Fig. 6 b, richly vascular granulation tissue is observed in which the connective tissue is greatly increased and hyalinized. Inflammatory cells are not seen.

Conclusions: The increase of the connective tissue throughout the pulp, shows that a diffuse inflammation has been in progress. The granulation tissue appearing in the coronal pulp on the side of the root canal and containing much connective tissue which has undergone hyaline degeneration, is a sign of a progressing scar formation in a region where there has been a stronger inflammatory process. Histologic study shows that healing has taken place.

The preservation of odontoblasts in the area of the pulpal roof indicates that bacteria have not penetrated as far as the pulp. Accordingly, this diffuse inflammatory process may be regarded as a case of serous total pulpitis.

Case 7. Age 25 years. *Subjective symptoms:* Pain occasionally for about a fortnight, caused by cold but not by heat. No tenderness on mastication.

Clinical examination: Carious focus in 7+. Cold causes sharp "shooting" pain, heat no particular pain. No tenderness to percussion.

Clinical diagnosis: Pulp. ac. ser. part. dent. 7+.

Control period of 84 days after treatment, no symptoms during that time.

Histologic findings: The layer of protective dentin is thin in the whole area of the pulp. The odontoblast layer seems to be fairly regular. In the hyperemic pulp the connective tissue is increased almost throughout. In the root pulp the odontoblasts are entirely absent at one point and the connective tissue has formed a scar. Figs. 7 a and b. No inflammatory cells are seen.

Conclusions: Inflammation has been in progress in the coronal pulp and the root pulp. At one point in the root pulp marked changes have been present and are replaced by scar tissue. The section did not run through the carious focus. For that reason scar tissue is not visible in the same measure as in the root pulp. The microscopic examination shows that healing has taken place. The fact that the odontoblast layer has been preserved in the region of the pulpal roof indicates that bacteria have not invaded the pulp. Accordingly, the case is a serous total pulpitis.

Case 8. Age 35 years. *Subjective symptoms:* Pain for one month, especially caused by cold. Tenderness on mastication during a few days.

Clinical diagnosis: Deep carious focus in 5+. Cold causes sharp "shooting" pain, heat no appreciable pain. Tenderness to percussion in the longitudinal direction.

Clinical diagnosis: Pulp. ac. ser. tot. dent. 5+.

Control period of 52 days after treatment, no symptoms during that time.

Histologic findings: Next to the carious focus the layer of protective dentin is very thick. The odontoblast layer has disappeared almost completely from the pulpal roof. Fig. 8 a. The connective tissue is increased and hyalinized throughout. Richly vascular granulation tissue abounds in the pulp. Fig. 8 b. The nerve fibres are plainly distinguished, Fig. 8 c. A slight amount of scattered infiltration of lymphocytes and plasma cells is seen in the area of the coronal pulp.

Conclusions: An inflammation has been in progress in the whole pulp. The pulpal tissue undergoes a change into a connective

tissue scar; the hyaline degeneration which is present shows that the scar tissue is already old (52 days after treatment). Although the inflammatory process has been strong and has extended over the whole pulp, the nervous tissue is preserved. The slight infiltration of inflammatory cells observed at the microscopical examination is a sign that healing is still in progress. The almost complete disappearance of odontoblasts in the area of the pulpal roof indicates that the pulp has been invaded by bacteria. The inflammatory process was thus a case of purulent total pulpitis?

Case 9. Age 20 years. *Subjective symptoms:* Pain for about two months, occasionally at first, later all day, also at night. Both cold and heat caused pain. Tenderness on mastication for about two weeks.

Clinical examination: Deep carious focus in 6—. Intense pain from cold and heat. Tenderness to percussion in the longitudinal direction.

Clinical diagnosis: Pulp. ac. ser. tot. dent. 6—.

Control period of 100 days after treatment; no symptoms during that time.

Histologic findings: The coronal pulp and the root pulp next to it are necrosed throughout. In this area connective tissue may be distinguished only close to the tooth wall. The odontoblasts have disappeared from the entire pulpal area. Fig. 9 a. In the root pulp the connective tissue is greatly increased. Fig. 9 b. Inflammatory cell infiltration, with lymphocytes, plasma and pus cells, is seen in the whole pulp, but especially in the coronal pulp.

Conclusions: The microscopical examination does not show whether the inflammation has extended over the whole pulp already before treatment, or whether the inflammatory changes in the root pulp have arisen during the control period. It shows, however, that the case was a treatment failure.

Case 10. Age 28 years. *Subjective symptoms:* Occasional pain for about two months. The day before the examination pain continued all day. Cold and heat caused pain. Tenderness on mastication for about a week.

Clinical examination: Deep carious focus in —8. Cold causes sharp and heat slighter pain. Tenderness to percussion in the longitudinal direction.

Clinical diagnosis: Pulp. ac. ser. tot. dent. —8.

Histologic findings: The coronal pulp and the root pulp next to it are filled with a large infiltration of inflammatory cells including lymphocytes, plasma and pus cells. Connective tissue is still discernible, however. The odontoblast layer is preserved in a state of degeneration here and there in the root pulp. The connective tissue is increased n the root pulp where infiltration of inflammatory cells is slight and scattered.

Conclusions: The microscopic examination does not show conclusively whether the inflammation has extended to the root pulp before treatment. The slight infiltration of inflammatory cells which is visible in this area, and the odontoblasts being preserved here and there indicate, however, that the inflammation has spread to the root pulp during the control period. In any case the tooth failed to respond to treatment.

Discussion.

Before proceeding to evaluate the results in the present series the writer will go through the method of treatment for the purpose of checking whether any errors have occurred.

Alypin was used for relief of pain and proved to be suitable. In any case no patients complained of pain after treatment. One of the treatment failures (Case 10) was an exception, but in this case the pain was due to the continuing inflammation.

A high dilution of penicillin was used. However, its potency decreased considerably while stored.

According to KIRBY, penicillin solution containing 500 to 1,000 units per cc. can be used for instillation. He is of the opinion that a solution of this kind retains its potency for three weeks, especially if kept in an ice-box.

My solution contained 100 to 200 times more penicillin per cc.; it was kept at a temperature below 10° C. It is, however, possible that the effectiveness of my solution decreased to the extent of affecting the results in the two cases which were treatment failures. One of them, the case of open pulpitis, was treated with paste containing penicillin which had been in the solution for 36 days. The second, a case of closed pulpitis, was treated with a 53-day old solution which was perhaps already quite ineffective.

To some extent the failures may have been caused by penicillin treatment having been applied only once, and also by the cases of open pulpitis not having been treated with the solution.

It should be mentioned in this connection, however, that a good result has been obtained with a 36-day old solution, even by treatment with paste alone.

The table below shows the time of storage of the penicillin solution used in each case, and the results of treatment:

Case	Time of storage of the penicillin solution (below 10° C.)	Result of treatment
4	2 days	+
9	8 »	—
5	18 »	+
8	23 »	+
7	26 »	+
2	28 »	+ paste treatment only
6	29 »	+
1	36 »	+ paste treatment only
3	36 »	— paste treatment only
10	53 »	—

I did not observe in my tests that penicillin would have injured the odontoblasts and the pulpal connective tissue, even if a 2-day old solution was used, although, according to GRÜNINGER, the potency of such a solution has not yet decreased.

In the paste protecting from reinfection the effect of penicillin and sulfathiazole seems to have been to the purpose. As regards sulfathiazole, it may be added that this drug has probably also played a rôle in healing the inflammation.

Calcium glycerophosphate and vigantol was added to the paste to promote the formation of hard tissue for covering the open pulp and reinforcing the roof of the closed pulp.

By the use of the vital amputation method new hard tissue has been brought to develop on the amputation surface, and even dentin, formed by the odontoblasts differentiated from the connective tissue cells of the pulp.

For capping the amputation wound HELLNER, FELDMAN, and NEUWIRT used dentin powder, MÜNCH applied a calcium-vitamin paste, FLOHR a mixture of calcium salts and dentin powder, and HERMANN, HOFFMANN etc. calxyl.

However, no connective tissue scar has developed over the wound in cases of vital amputation, as in the healed cases of open pulpitis included in my material.

It appears that new dentin may develop over a fresh pulpal wound if the proper capping is used, whereas in chronic ulcerative pulpitis the wound which is superficially necrotic will heal in the form of a connective tissue scar which prevents the differentiation of new odontoblasts and consequently also the growth of new dentin.

Accordingly, calcium glycerophosphate and vigantol, or any

other substances applied for the same purpose, are not beneficial in the treatment of chronic ulcerative pulpitis.

As regards chronic granulating pulpitis the case may be reversed, as a fresh incision surface must be made in order to remove the pulpal polyp. Unfortunately my material does not add to the knowledge of this question.

In cases of closed pulpitis, deposition of fairly regular dentin over irregularly calcified protective dentin could be noticed only in one case. I see no reason to ascribe this to the effect of calcium glycerophosphate and vigantol, more particularly so as these drugs had no effect in the other cases of the material.

The value of vigantol in retarding resorption of penicillin seems remarkable, on the other hand. As its rôle was not important in the formation of hard tissue, it may, however, be replaced by some indifferent oil.

The importance of oil for the preservation of sulfathiazole and its activity, cannot be determined by my investigations.

When carrying out the experiments I assumed that the use of phosphate cement for sealing the cavity might cause dissolution of the penicillin to an extent influencing the results of treatment.

Fixation of the phosphoric acid apparently takes place so rapidly, however, that its deleterious effect does not come to the fore in a disturbing degree. Yet it should be noted that the cement mixture should be fairly thick and pressure should be avoided.

The changes arisen in the tissues during the course of healing do not show definitely the situation at the time of the inflammation.

Serous exudate may disappear without leaving any trace. It often is organized, however, as connective tissue develops from the surrounding tissue, scar tissue being formed.

Purulent exudate does not disappear as a rule, it has to find an exit. Under favourable conditions the pus can be resorbed. In such cases the pus cells disappear and granulation tissue develops to replace the tissue defect; as the connective tissue increases the granulation tissue changes into scar tissue.

On the basis of the increase of connective tissue seen in the picture of healing in cases of closed pulpitis, it is possible to determine whether a partial or a total pulpitis has been present in the pulp. In the former the increase is local in the area of the coronal pulp, in the latter the connective tissue is increased also in the root pulp.

On the other hand, it is not possible to differentiate between serous and purulent inflammation on the basis of connective tissue changes arisen in the course of healing.

According to HARNDT's histo-bacteriological studies, it is possible to conclude from the state of the odontoblasts whether bacteria have already invaded the pulp; if this has happened he considers the pulp affected by purulent inflammation. In serous inflammation the odontoblasts degenerate and may even disappear partly. In purulent inflammation they have entirely disappeared at the point of invasion of bacteria. Accordingly, it would be possible to determine what type of inflammation has been present in each case in my series.

It should be taken into consideration that in the course of healing the inflammatory process first becomes chronic in the closed space of the pulp, and during this period changes may still arise in the odontoblast layer.

On the basis of the microscopic examination, I have designated two cases as purulent pulpitis, as the odontoblast layer was partly or completely destroyed in the area of the pulpal roof. On account of the potential secondary disappearance of odontoblasts mentioned above, I have added a question-mark to the diagnosis in these cases (Nos 5 and 8).

Histologic study showed in most cases that the clinical diagnosis had been wrong. This supports HARNDT's observation made on the basis of comparative clinical and histologic study, viz. that a differentiation between partial and total pulpitis may be possible by clinical examination, but not between serous and purulent pulpitis.

My results justify the following conclusions regarding the healing of inflamed pulp:

Open pulp with a chronic ulcerative inflammation heals in the same way as do infected wounds on the whole. The necrotic incision surface disappears and it is replaced by granulation tissue which forms into scar tissue owing to the increase of connective tissue. The state of the pulp after healing may be termed fibrosis pulpae partialis.

Closed pulp with an acute partial inflammation heals either as the serous exudate is organized into connective tissue, or the serous and perhaps also the purulent exudate is resorbed and replaced by granulation tissue which, due to the increase in connective tissue, develops into scar tissue. The designation fibrosis pulpae partialis may, similarly, be used in this case.

Closed pulp with acute total inflammation heals as the connective tissue of the pulp increases throughout. The exudate, the type of which cannot be definitely determined from the picture of healing, is resorbed and replaced by granulation tissue which gradually develops into a connective tissue scar. Healing has brought the pulp to a state which may be termed fibrosis pulpae totalis.

The reticular connective tissue of the pulp is thus replaced by scar tissue. It is noteworthy that nerve fibres can be noted in it; they have thus been preserved during the course of the inflammation.

The destruction of odontoblasts is followed by growth of connective tissue which replaces them and prevents the growth of new odontoblasts.

When odontoblasts are absent in the area of the roof of the pulp, protective dentine cannot develop when a new infection is threatening. The power of resistance of the healed pulp is thus dependent on the preservation of the odontoblast layer.

Except the statements made above regarding the healing of inflamed pulp, other conclusions may be drawn on the basis of my examinations.

As in my series penicillin was bacteriostatic in the dentin canals, it is now possible to leave carious dentin on the bottom of a deep cavity in non-infected teeth, if exposure of pulp cannot be avoided by any other means.

The life of a healthy pulp which has been accidentally exposed can also be preserved with penicillin. There should be no reason for the pulp to be destroyed as no inflammation is in progress, and infection can be prevented by means of penicillin.

The use of vital amputation will also be extended as penicillin can prevent infection also in this measure. In cases of chronic open pulpitis the crown of the tooth is often so severely destroyed that filling without the support of the pulp chamber cannot be considered. In such a case vital amputation may be selected. In the treatment of purulent pulpitis vital amputation may also prove necessary. As healing of inflamed pulp in general, this is a question which should be fully clarified before practical application may be thought of.

The introduction of penicillin has revolutionized the treatment of diseases of inflammatory origin. Its value in dentistry cannot yet be fully estimated, as experiments in this branch of science

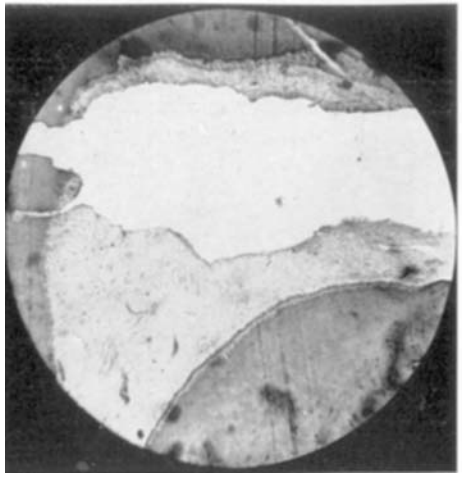

Fig. 1 a. Clinical and histological diagnosis: pulp. chr. ulc. dent. 8+.

Healing has taken place. The connective tissue is slightly increased. The cavity is probably an artefact.

Fig. 1 b. The same case. A thick layer of protective dentin adjoins the carious focus.

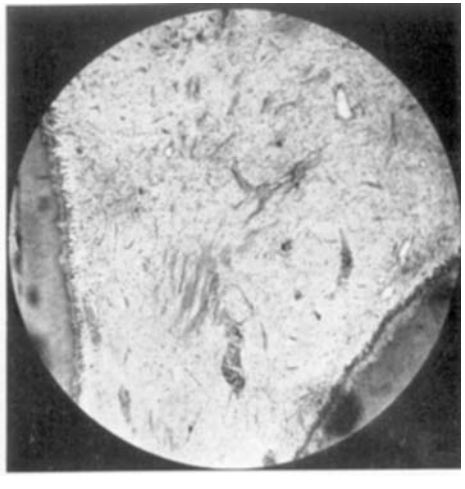

Fig. 1 c. The same case. Regular pulpal tissue.

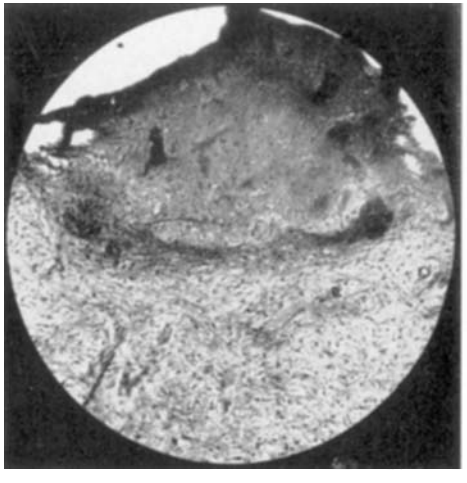

Fig. 2. Clinical and histologic diagnosis: pulp. chr. ulc. dent. +8.

Healing in process. Granulation tissue is forming into scar tissue. Slight infiltration of inflammatory cells.

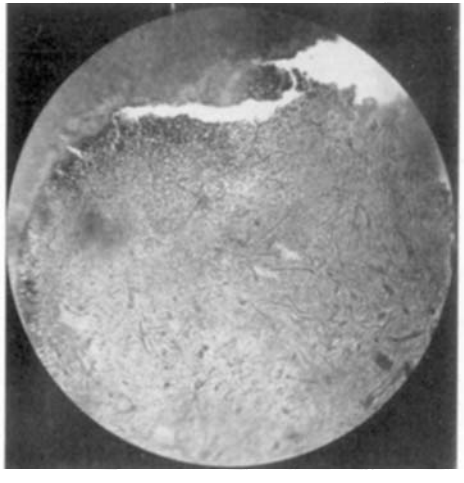

Fig. 3 a. Clinical and histologic diagnosis: pulp. chr. ulc. dent. +8.

Treatment failure. Strong infiltration of inflammatory cells in the pulp.

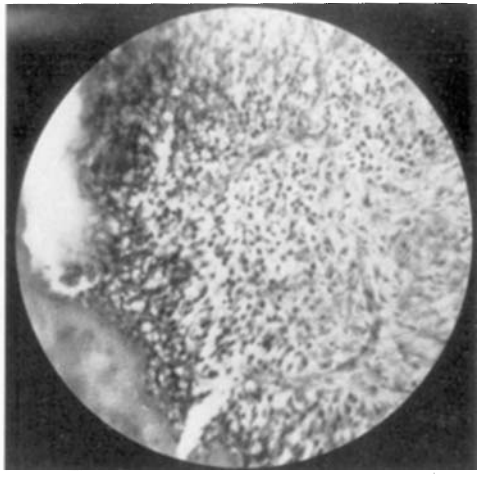

Fig. 3 b. A high power field of the same case.

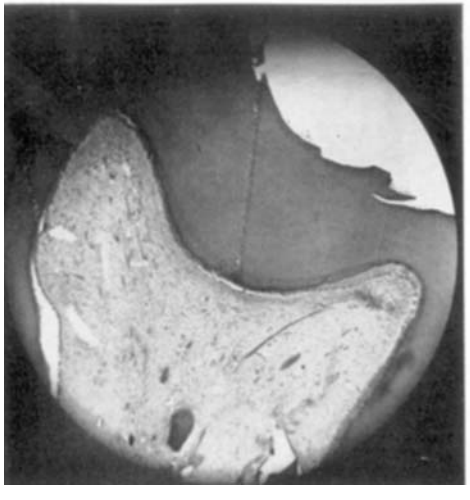

Fig. 4 a. Clinical diagnosis: pulp. ac. tot. dent. 6—.

Histologic diagnosis: pulp. ac. ser. part. dent. 6—.

Healing has taken place. Scar tissue has formed in the area of the inflammatory process in one horn of the pulp. In the central part of the coronal pulp there are connective tissue scars and calcified scars.

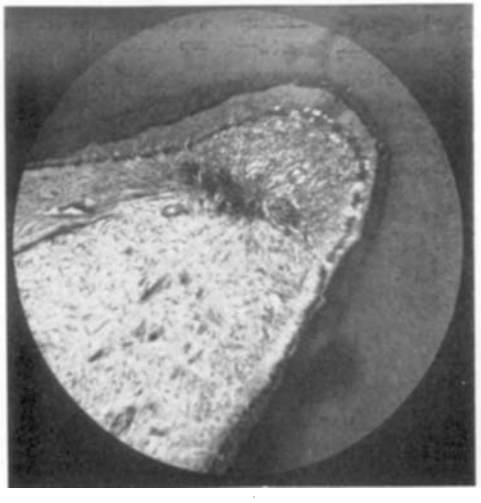

Fig. 4 b. A high power field of the pulpal horn. Vacuolization in the odontoblasts. Scar tissue in the pulp.

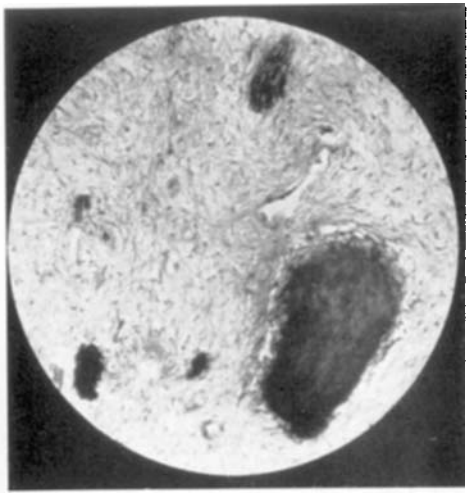

Fig. 4 c. A higher magnification of the connective tissue scars and the calcified scars.

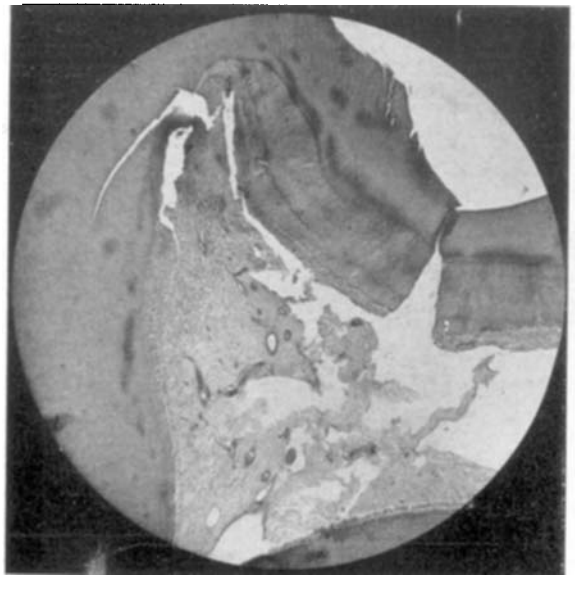

Fig. 5 a. Clinical diagnosis: pulp. ac. ser. tot. dent. —7.

Histologic diagnosis: pulp. ac. our. part. dent. —7?

Healing in process. The layer of protective dentin very thick. The odontoblast layer is completely destroyed in places in the pulpal horn, the reticular connective tissue is replaced by scar tissue.

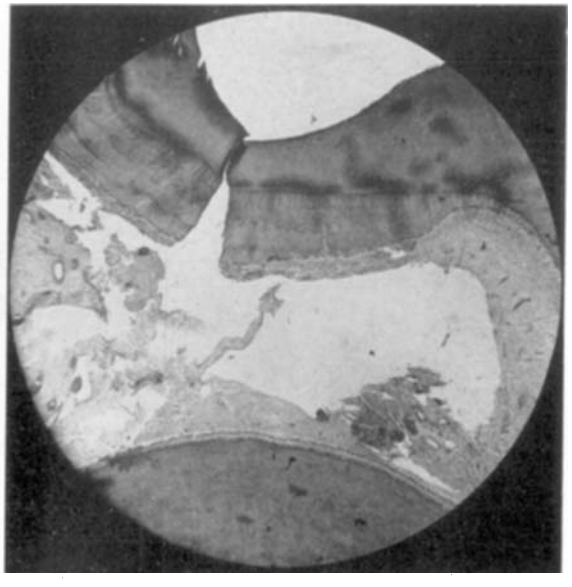

Fig. 5 b. The cavity in the pulp may be an emptied abscess or an artefact. A slight amount of granulation tissue at the edge of the cavity.

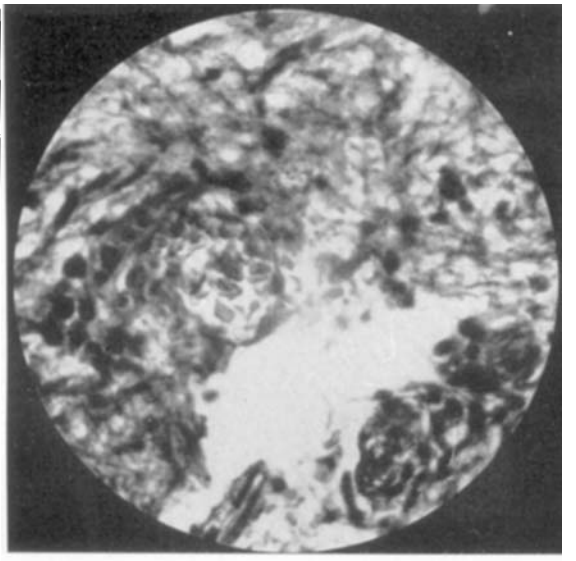

Fig. 5 d. Slight infiltration of inflammatory cells in the granulation tissue.

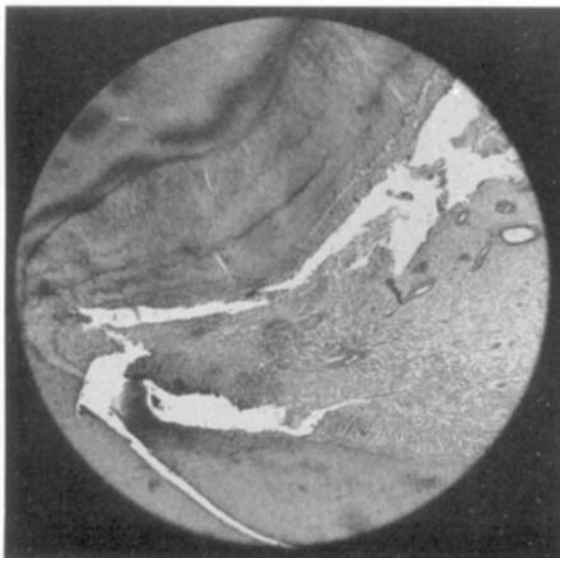

Fig. 5 c. A high power field of the area of the horn.

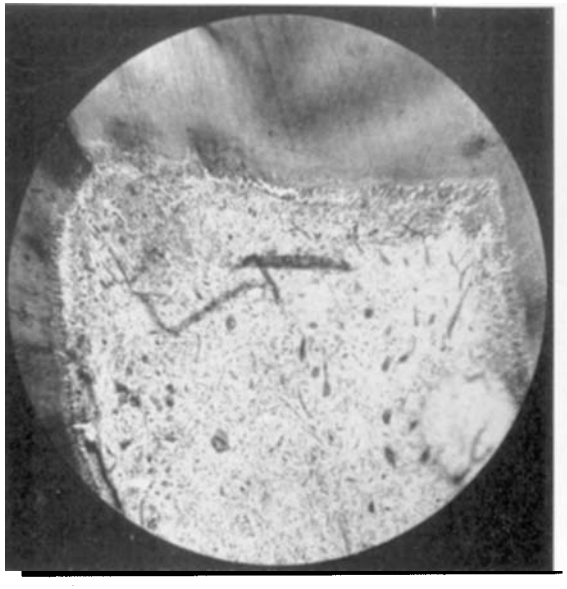

Fig. 6 a. Clinical diagnosis: pulp. ac. ser. part. dent. +8.

Histologic diagnosis: pulp. ac. ser. tot. dent. +8.

The odontoblast layer is fairly well preserved. The connective tissue increased throughout.

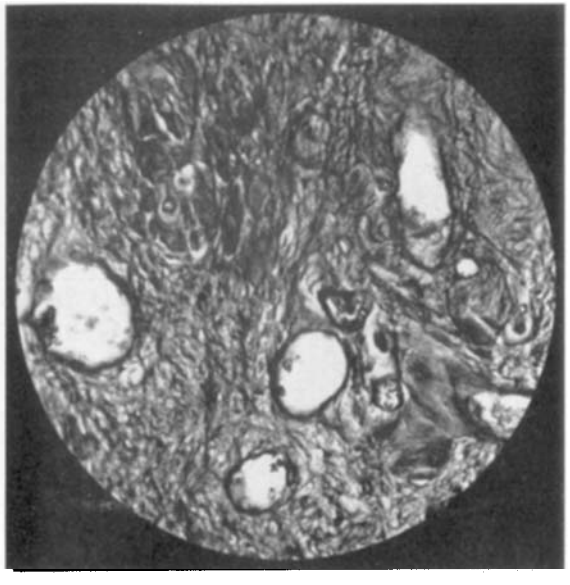

Fig. 6 b. The richly vascular granulation tissue of the pulp: the connective tissue is greatly increased and hyalinized.

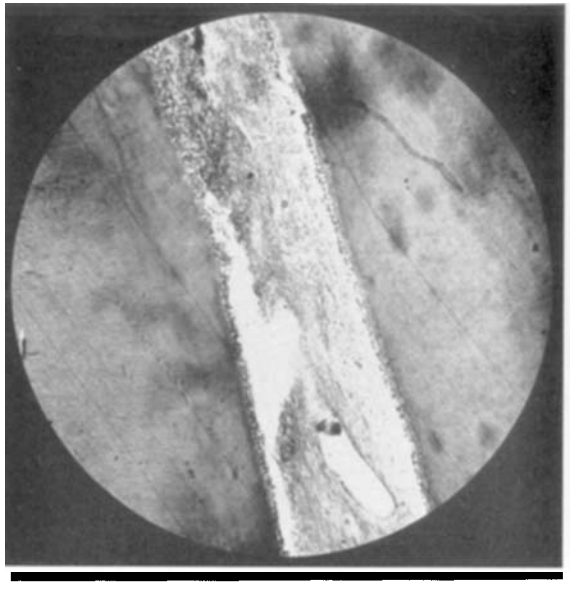

Fig. 7 a. Clinical diagnosis: pulp. ac. ser. part. dent. 7+.
Histologic diagnosis: pulp. ac. ser. tot. dent. 7 + .
In the root canal the connective tissue is increased particularly at a point where the odontoblasts are completely destroyed.

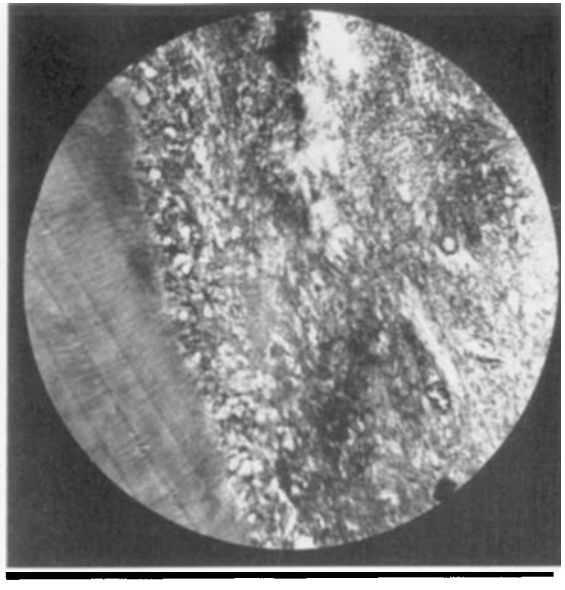

Fig. 7 b. A high power field of the increased, coarse and hyalinized connective tissue of the root canal.

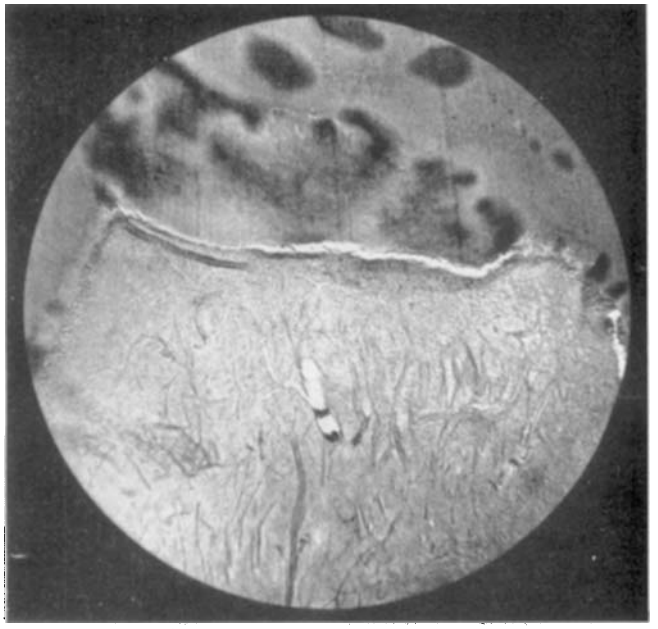

Fig. 8 a. Clinical diagnosis: pulp. ac. ser. tot. dent. 5+.

Histologic diagnosis: pulp. ac. pur. tot. dent. 5+?

The layer of protective dentin is very thick. The odontoblast layer is almost completely destroyed in the area of the pulpal roof. In the pulp the connective tissue is increased throughout.

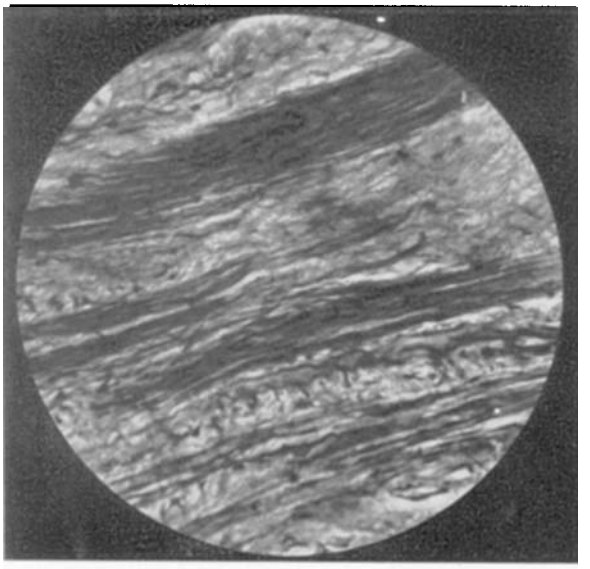

Fig. 8 c. Nerve fibres in the pulp.

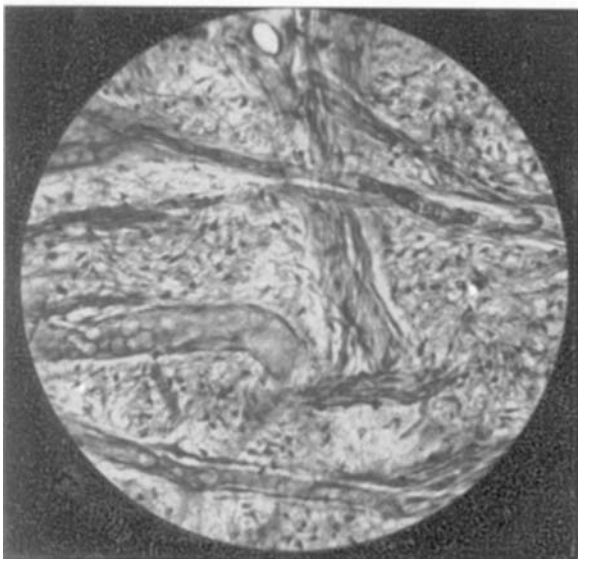

Fig. 8 b. Richly vascular granulation tissue in the pulp.

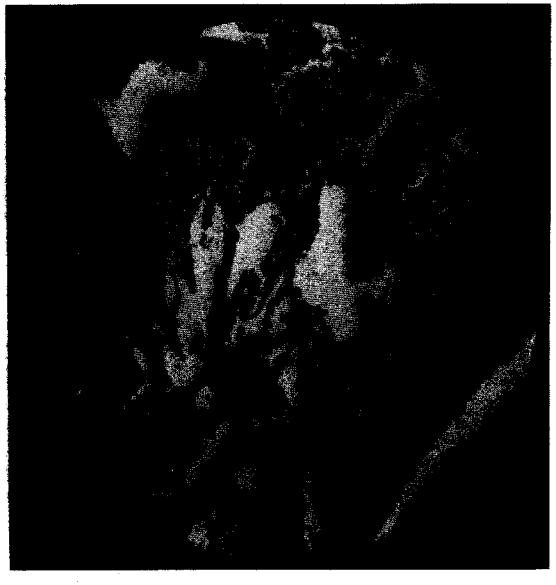

Fig. 9 a. Clinical diagnosis: pulp. ac. ser. tot. dent. 6—.
Histologic diagnosis: pulp. necrotic. dent. 6—.
The coronal pulp is necrosed almost throughout.

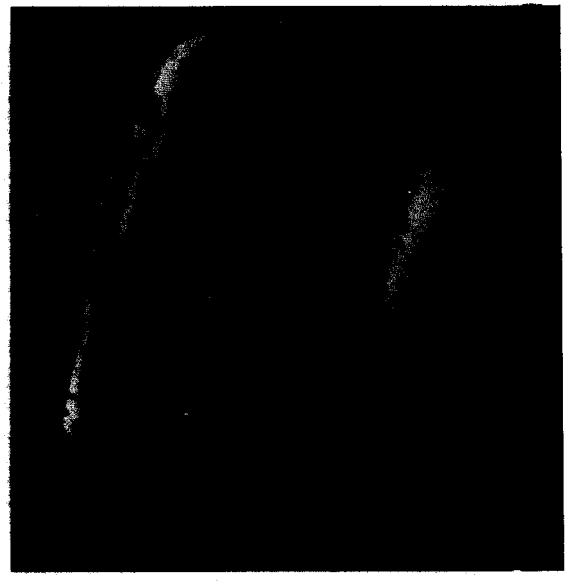

Fig. 9 b. The connective tissue is considerably increased in the root pulp. The odontoblast layer is completely destroyed.

have not been extensive so far. In addition, new antibiotic substances resembling penicillin are continually being discovered. It is possible that one of the new drugs, less sensitive to chemical and thermic influences, will be more useful in dentistry than penicillin.

In any case it seems certain that the antibiotic substances will greatly change our present-day methods of treatment.

Summary.

Local penicillin treatment has been found suitable for the purpose of healing inflamed dental pulp.

The process of healing is as follows: The exudate, the character of which cannot be established with certainty from the picture, is replaced by scar tissue. In chronic ulcerative and in acute partial pulpitis the process is limited to the area of the coronal pulp and may thus be termed fibrosis pulpae partialis. In total pulpitis the reticular connective tissue of the pulp is completely replaced by scar tissue; the result is fibrosis pulpae totalis. In the healed pulp nerve fibres are visible.

New odontoblasts do not develop in place of those which have been destroyed. If odontoblasts are lacking in the region of the roof of the pulp, protective dentin does not form when a new infection is threatening.

Zusammenfassung.

Örtliche Penizillinbehandlung hat sich zur Heilung von Entzündungen der Zahnpulpa bewährt.

Der Heilungsvorgang ist folgender: Das Exsudat, dessen Natur sich aus dem Bilde nicht sicher erkennen lässt, wird durch Narbengewebe ersetzt. Bei chronisch-ulzerativer und bei akuter partieller Pulpitis ist der Prozess auf das Gebiet der Kronenpulpa beschränkt und kann also als Fibrosis pulpae partialis bezeichnet werden. Bei totaler Pulpitis wird das gesamte retikulare Bindegewebe der Pulpa durch Narbengewebe ersetzt; das Resultat ist Fibrosis pulpae totalis. In der ausgeheilten Pulpa sind Nervenfasern sichtbar.

Neue Odontoblasten an Stelle der zerstörten kommen nicht zur Entwicklung. Wenn im Gebiete des Pulpadaches Odontoblasten fehlen, wird bei drohender erneuter Infektion kein schützendes Dentin gebildet.

Résumé.

Le traitement local par la Pénicilline s'est montré efficace contre les inflammations de la pulpe dentaire.

Le processus de la guérison est le suivant: L'exsudat, dont le caractère ne peut pas être déterminé avec certitude par son aspect, est remplacé par un tissu cicatriciel. Dans la pulpite ulcéreuse chronique et dans la pulpite aigue partielle le processus est limité à la région de la pulpe de la couronne et l'on peut dès lors lui donner le nom de fibrose pulpaire partielle. Dans la pulpite totale le tissu conjonctif réticulé de la pulpe est complètement remplacé par du tissu cicatriciel; il en résulte une fibrose totale de la pulpe. Des fibres nerveuses sont visible dans la pulpe guérie.

Il ne se dévéloppe pas de nouveaux odontoblastes à la place de ceux qui ont été détruits. S'il manque des odontoblastes dans la région du toit de la pulpe, la dentine protectrice ne se forme pas quand une nouvelle infection menace.

References.

ENTIN, D. A.: Neue Wege in der Therapie der Pulpaentzündung. Z. f. St. 1935.

FELDMANN, GR.: Neue Wege in der Therapie von Zähnen mit entzündeter Pulpa. V. f. Z. 1932.

FLOHR, W.: Biologische Wurzelbehandlung. Z. R. 1936.

—, Praktisches zur biologischen Wurzelbehandlung. Z. R. 1937.

GRÜNINGER, W.: Penicillin. Bern 1946. 2. Aufl.

HARNDT, E.: Histo-Bakteriologische Untersuchungen der erkrankten Zahnpulpa. D. Z. M. K. 1938.

—, Die klinische Diagnostik der Pulpaerkrankungen. D. Z. M. K. 1938.

HELLNER, E.: Die chirurgische Behandlung der Pulpitis. Z. f. St. 1933.

HERMANN, B. W.: Die biologische Wurzelbehandlung. Z. R. 1935.

HOFFMAN, M.: Die Vitalamputation mit Calxyl bei entzündeten Pulpen. Schw. M. f. Z. 1939.

KIRBY, W. M. M.: Quoted by KOLMER, J.: Penicillin Therapy. New York-London. 1945.

MILLER, W. D.: Die Bakterio-Pathologie der Zahnpulpa. K. bl. Z. 1894.

MÜNCH, J.: Biologische Wurzelfüllung. D. z. W. 1936.

NEUWIRT, F.: Die reparatieven Fähigkeiten der Pulpa. Z. f. St. 1933.

PECKERT, H.: Einführung in die konservierende Zahnheilkunde. Leipzig 1913.

SIEBERTH: Quoted by HARNDT: D. Z. M. K. 1938.

WEINMANN, J.: Dentinveränderungen durch lokale Einwirkung von Vitamin D. Z. f. St. 1933.

Address:
Museokatu 21 A,
Helsingfors,
Finland.

Further Studies of Aviation Dentistry.

Dental Symptoms Reported by Fighterpilots Exposed to Prolonged High Altitude Operations.

By

REIDAR FAUSKE SOGNNAES,

M. S., Ph. D.[1]

616.314.

In previous work a review of earlier literature (1) was followed up by a study of the dental condition in a group of 1000 aviators (2) and their various oral symptoms associated with simulated altitudes (3, 4) and accelerations (5, 6). To the extent permitted at a training centre, these experimental findings were correlated with dental problems encountered under actual flying conditions (7, 8). But not until the latter part of the war in Europe was it possible to contact the surviving pilots in the theatre of combat operations with a view to supplement past observations with this final report on wartime studies.

Material.

From several hundred replies to the questionnaire shown in chart 1 and personal interviews with the R. N. A. F. air-crews in the European theatre of war it soon became apparent that the majority of complaints related to the teeth was reported by the fighterpilots exposed to prolonged operational flying. Only scattered cases could be traced to bombercrews (not including stratosspherebombers) while nearly all of those fighterpilots who for some time had been involved in high altitude combat had had one or

[1] Now at Harvard School of Dental Medicine, Boston, Mass.

Table 1.

Flying Experience of the Positive Cases.
(R. N. A. F. Fighter pilots.)

Case No.	Age	Hours of flying experience			
		Pre-operational	High altitude operations		
			25,000 ft. 30,000 ft.	Above 35,000 ft.	In all
1.....	23	230	175	25	200
2.....	24	600	150	25	175
3.....	25	250	150	10	160
4.....	21	500	100	50	150
5.....	26	400	130	5	135
6.....	32	500	80	30	110
7.....	22	300	75	15	90
8.....	29	585	60	4	64
9.....	25	395	50	7	57
10.....	22	250	40	15	55
11.....	26	358	50	0	50
12.....	26	1300	50	0	50
13.....	27	250	40	2	42
14.....	25	400	25	10	35
15.....	23	300	30	4	34
16.....	27	400	25	5	30
17.....	24	800	25	0	25
18.....	22	300	15	5	20
19.....	24	370	17	2	18
20.....	24	360	15	0	15
Average	24.8	442	65	17	75

more experiences of dental interest. While this was a relatively rare observation during training (7) 10 % of the pilots on active operations reported such symptoms as tooth-ache and loss of fillings. The flying experience of 20 positive cases is listed in table 1 and shows that their operations have occupied from 15 to 200 hours at altitudes above 25,000 feet and from 0—25 hours above 35,000 feet, with a total average of 75 hours per pilot above 25,000 feet. Since past observations had more or less eliminated the effect of pre-operational low altitude flying (3, 4, 7), they have been listed in relation to the number of high altitude flying hours, regardless of their varying total flying experience.

Table 2.

Dental Symptoms Related to High Altitude Operations.

Symptoms from Teeth and Jaws	Case Number																				Total
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	
Breaks of fillings		×		×			×							×				×	×		6
Loosening of fillings								×					×			×	×	×			5
Loss of fillings	×							×		×						×			×	×	6
Sensitiveness to cold	×							×			×	×									4
Dull Tooth Ache		×							×	×							×			×	5
Dental pain					×						×	×			×						4
Bleeding from gingiva			×			×		×			×	×					×			×	7

Results.

The questionnaire shown in chart 1 includes the type of symptoms which — judging by past observations — (7) were thought to be encountered during flying. When listed in table 2 it is seen that the individual pilots reported from one to four of the symptoms in question. If we group the type of complaints it may be seen that altogether 11 of the 20 pilots reported subjective discomfort (»pain» symptoms) such as sensitivity, dull tooth ache or real pain in the teeth. Thirteen reported breaks, loosening and loss of fillings, six of them in conjunction with »pain» symptoms, three in conjunction with bleeding gums as well. Two reported bleeding gums as their sole dental trouble, and finally there were two who had experienced pain symptoms from the teeth as well as bleeding gums. Because of such combinations of different types of symptoms it becomes difficult to list in table form the flying conditions under which the various discomfort arose. But taken one by one the cause of the dental symptoms were given as follows.

All the 6 cases of breaks in fillings were attributed to high altitudes and barometric pressure changes, two combined with the simultaneous exposure to cold and the use of the oxygen supply.

Of the 5 cases complaining of loosening of fillings, four again gave the high altitude and the barometric pressure changes as the cause, while one suspected the accelerations.

Loss of fillings was reported by 6, of whom four held the high

Chart 1.

Name Rank Age Date

Serving as: Fighter-pilot ... Bomberpilot ... Observer ... Gunner ... Instructor

No. of flying hours: a) Previous to operational flying .....
b) Operational flying .....

No. of flying hours: a) Between 25,000 and 35,000 feet altitude .....
b) Over 35,000 feet altitude .....

Have you had any of the below mentioned experiences that you think may be attributed to any of the causes listed? if so, mark off below.

Symptoms from teeth and jaw	High altitudes	Great changes in pressure	Large accele-rations	Low temper-atures	Oxygen Supply	Other Causes
Breaks of fillings ...						
Loosening » ...						
Loss of » ...						
Sensitiveness to cold in the teeth						
Dull tooth-ache						
Pain in the teeth...						
Bleeding from the gums						
Other symptoms ...						

Remarks: .....
Have you — excepting periods of flying — had any discomfort in your jaw-joint such as the jaw out of joint (dislocated), feeling of tiredness or snapping sound in your joint of the jaw? Yes ..... No ..... Described further .....

Have you — excepting periods of cold — had any trouble with the ventilation of the middle ear? Does it take unreasonably long time to equalize the air pressure? Do you have temporary deafness or pain in the ears during or following rapid changes in altitude? Yes ..... No .....
Describe further .....

If you have anything to add to the above, or if you have any other experiences from the time you have been flying, which suggest that the teeth may suffer or give rise to discomfort from flying, please describe further on the other side of this form.

altitude responsible, and two the combination of low barometric pressure and low temperature.

Of the above complaints there are thus 17 reports of detrimental effects of flying upon the maintenance of dental restorations, and in 14 of these cases the high altitude and barometric pressure changes are held responsible. In only one case is the simultaneous experience of accelerations connected with the damage to the fillings.

With regard to the subjective sensation of discomfort and pain we again find the majority suspecting the high altitudes as its cause. Of the 4 who reported sensitivity to cold two added the oxygen supply to the above cause. Of the 5 who experienced dull ache from their teeth, three held the high altitude responsible, one accelerations, and one gave an irrelevant cause, equally applicable at ground level.

Finally the 4 cases of pain were by three pilots attributed to high altitude exposure, by one to rapid changes in barometric pressure. This adds up to 13 cases of subjective »pain» symptoms, 10 of whom associated their experience with their simultaneous exposure to very high altitudes. Again only one could associate such symptoms with the frequent exposures to extreme accelerative forces encountered during air battles.

In contrast to the determined association of the above troubles with high altitude flying, only two out of the 7 cases of bleeding gums, were similarly associated. Another pilot gave barometric pressure changes as a possible cause, while the four remaining described other causes not connected with flying.

The last questions on the questionnaire were intended to trace any possible difficulties with ventilation of the middle ear resulting from a malposition of the jaws. There were two who answered affirmatively to the question of discomfort in connection with the mandibular joint, and four thought that they needed unreasonably long time in order to equalize the air pressure in the middle ear. But in no case was there a relationship between the two.

Discussion and Conclusion.

Since the first few cases were observed in which chronic (9) and acute (10) dental defects gave rise to discomfort when flying, most of the reports regarding the effect of flying upon the teeth

have been based upon speculations (11, 12), isolated cases (13, 14) tests in decompression chambers (3, 4, 15, 16, 17, 18) and in vitro experiments (19, 20, 21). Besides flying experiences at training stations (7, 8, 22) little is known from actual studies in the theatre of war. Yet the final test of our conclusions from isolated experimental evidence rests on just this reaction of the pilot himself to actual combat exposure. It is therefore of some interest to know under which circumstances of air battle the pilot experienced his specific symptoms, and to determine to what extent these reactions can be correlated with experimental evidence on the isolated effect of the different factors studied singly.

In such an analysis this material confirms previous experiments on the effect of simulated altitude (3, 4) and accelerations (5), in which it was demonstrated that the former factor is more frequently associated with dental discomfort than the latter and that barometric pressure changes at high altitudes is most significant. With regard to the damage to fillings, however, there is a discrepancy between the actual reports of the pilots, on the one hand, and experimental evidence from in vitro tests (19) and tests in decompression chambers (3, 4, 15, 16, 17, 18) on the other. This either suggests that such damage is not associated with any factors encountered during flying, or is a result of a combination of factors characteristic to operation of fighter aircrafts. The latter explanation is supported by the fact that other air crews as well as ground crews gave negative answers to similar inquiries, and the fact that previous examinations of the fighterpilots in question had given nothing to suggest similar damage to fillings previous to their operational flying at higher altitudes.

It seems therefore that some other factor than those usually suspected, such as air traps under fillings (11) low temperatures (19, 20) and barometric pressure changes (21) must be responsible. By way of speculation two possibilities may be suggested; firstly that the damage is caused by unconscious occlusal strain in the heat of battle, secondly by increased caries frequency and recurrent caries along the margins of fillings. The latter observation is supported by several detailed case histories (7), and has been associated with a decreased salivary secretion at high altitudes (23). The first possibility will probably remain as a mere contention because it would be hard to test experimentally.

The expansion of air trapped under fillings has in addition been given as a possible cause of pain while flying at high altitudes (11).

This was not supported by previous observations (3) where it was concluded that »it may have been due to some pulpal disturbances rather than air traps under the fillings». This view has been confirmed by subsequent histological findings (24). Consequently a minor pulpal lesion, which does not give rise to symptoms at ground level may make itself known by acute discomfort at higher altitudes.

But what still remains to be determined is chronic damage to oral structures following past flying experience.

As far as is known this question has not been investigated. A few experiments related to salivary secretion under low oxygen tension have indeed raised the suggestion of lasting if not permanent damage to the salivary glands (25, 26, 27). But no delayed action on the dental structures themselves is known to have been studied or reported as a sequel to actual or simulated flying conditions. For that matter it may be too early to find cases.

But the circulatory conditions of the teeth and supporting structures represent characteristics which to the author warrant such a study. If negative it would seem that the peace time problems of aviation dentistry by and large may be limited to scattered cases of acute discomfort from minor pulpal lesions. These it should be possible to control by exacting diagnostic and therapeutic procedures, and because all available evidence relates aerodontalgia to high altitudes, i. e. low barometric pressure,even this limited problem may be eliminated by the general introduction of constant-pressure cabins.

As far as our flying patients are concerned it is still well to be familiar with the effect of flying upon the teeth. But there is at present nothing to suggest that problems of aviation dentistry are going to be of major significance in peace time air travel. In wartime its importance will be recognized and scrutinized as long as aircrafts are piloted by man.

Bibliography.

1. SOGNNAES, R. F.: Studies on aviation dentistry. National Research Council, Ottawa, May 1944. Review of the literature — (Part I).
2. — Dental conditions in a group of 1,000 airmen. Ibid. (Part II).
3. — Symptomatology of various dental conditions during changes in simulated altitude. Ibid. (Part III).
4. — The effect of malocclusion on the ventilation of the middle ear during changes in simulated altitude. Ibid. (Part IV).

5. — Symptomatology of various dental conditions during positive accelerations produced in the human centrifuge. Ibid. (Part V).
6. — A method of testing the retention of artificial teeth and its significance in the prosthetic treatment of aviators exposed to high accelerations. Ibid. (Part VI).
7. — General survey on the prevalence of dental trouble in aviators as based on questionnaires and case histories. Ibid. (Part VII).
8. — Practical considerations in the dental care of aviators as based on treated cases. Ibid. (Part VIII).
9. NEBLETT, H. C.: Chronic infection of the teeth and gums as a cause of staleness in air service pilots. Military Surgeon, 53: 224—229, 1923.
10. DREYFUS, H.: Les dents des aviateurs. *L'Odontologie*, 75: 612—613, 1937.
11. KOELSCH, FRANZ: Handbuch der Berufskrankheiten. Jena, Gustav Fischer, 1: 39—41, 1935.
12. LIPSON, H. J., and WEISS, S. G.: The biological approach to problems in aviation dentistry. J.A.D.A. 29: 1660—1663, 1942.
13. WILLHELMY, G. E.: Aerodontia. Dental Digest, 49: 311, 1943.
14. COONS, D. S.: Aeronautical Dentistry. J. Can. Dent. Assoc. 9: 320—323, 1943.
15. JOSEPH, T. V., GELL, C. F., CARR, R. M., and SHELESNYAK, M. C.: Toothache and the aviator. A study of tooth pain provoked by simulated high altitude runs in a low pressure chamber. U. S. Naval Med. Bull. No. 3: 643—645, 1943.
16. MITCHELL, D. F.: Aerodontalgia. Bull. U. S. Army Med. Dept., No. 73: 63—67, 1944.
17. PIGOTT, J. P.: Dental pain at high altitudes, origin and treatment. C. A. M. Report No. 383, National Research Council, Washington, 1944.
18. KENNON, R. H., and OSBORNE, C. M.: Dental problem concerning flying personnel. J.A.D.A. 31: 662, 1944.
19. ARMSTRONG, H. G., and HUBER, R. E.: Effect of high altitude flying on human teeth and restorations. Dental Digest, 43: 132—134, 1937.
20. HARVEY, W.: Tooth temperature with reference to dental pain while flying. Brit. Dent. J. 75: 221—228, 1943.
21. RETARSKI, J. S.: Effect of changes in barometric pressure upon dental fillings. U. S. Naval Med. Bull. 42: 155, 1944.
22. AAF Dental Surgeons: Dental Questionnaire. Office of Flying Safety, Washington, 1944.
23. STRELTSOV, V. V.: Digestion and metabolism in high altitude flights. Am. Review of Soviet Medicine, 1: 130, 1943.
24. ORBAN, G., and RITCHEY, B. T.: Toothache under conditions simulating high altitude flight. J.A.D.A. 32: 145, 1945.
25. FILLIPPOVICH, S. I.: Changes of salivation under the influence of reduced barometric pressure. Bull. de Biologie et de Médécine Expérimentale, 9: 338—340, 1940.

26. EDDY, N. B.: The effect upon salivary secretion of varying the carbon dioxide and oxygen content of the inspired air. Am. J. of Physiology, 88: 534—545, 1929.
27. BRASSFIELD, C. R.: Comparison of changes in pH of arterial blood and saliva during variations of pulmonary ventilation. Am.J. of Physiology, 116: 174—181, 1936.

Address:
Harvard School of Dental Medicine,
Boston, Mass.,
U. S. A.