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FUNDAMENTALS SUGGESTED BY RECENT RESEARCHES FOR DIAGNOSIS, PROGNOSIS, AND TREATMENT OF DENTAL FOCAL INFECTIONS

BY WESTON A. PRICE, D.D.S., M.S., F.A.C.D.
CLEVELAND, OHIO

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FUNDAMENTALS SUGGESTED BY RECENT RESEARCHES FOR DIAGNOSIS, PROGNOSIS, AND TREATMENT OF DENTAL FOCAL INFECTIONS

By WESTON A. PRICE, D.D.S., M.S., F.A.C.D., Cleveland, Ohio

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THE new light that has been thrown on dental infections and their relation to health and disease as a result of recent researches in many laboratories, including our own, has demonstrated the dire need for a revision of the fundamentals for diagnosis, prognosis, treatment and prevention of dental infections. This paper will present additional data; and because of the lack of time and the great bulk of new data, it will be necessary that they be abbreviated in the presentation of details. I shall endeavor in the time allotted to present new information on the following important phases of the dental infection problem: 1. Limitations of the roentgen ray. 2. Bacterial accommodation. 3. Grouping of individuals upon a basis of dental pathologic conditions. 4. Grouping of individuals upon a basis of presence or absence of streptococcal susceptibility. 5. Chemical changes in the blood. 6. Calcium metabolism. 7. Relationship of type of degenerative disease to type of bone reaction.

In order to orient ourselves, let us review the present situation. The current belief and basis of practice is, in my judgment, about as follows: That the roentgen ray will reveal the presence or absence of dental infection because dental infections produce changes in the supporting structures about an infected tooth in the form of a zone or chamber about the apex of the tooth. This absorption is, in volume, in direct proportion to the amount of infection present, and therefore the danger to the individual is expressed quantitatively by the extent of the zone rarefaction. The precise measure of this extent is believed to be revealed by roentgenograms. I shall present further evidence, which I find myself unable to interpret in any other way than as contradicting the validity of much of the foregoing premise and as indicating an entirely new basis for judgment.

In my recently published text,[1] I furnished a quantity of evidence which I shall assume that my readers are, in general, familiar with. I have shown, for example, that the presence of an extended zone of pus in the joints and muscles of the limbs of a rabbit is not revealed by the roentgenogram, and have presented much other data to demonstrate that dental infections may be present without being revealed.

In Figure 1, there will be seen the two central incisors of each of four individuals. One central incisor in each case is putrescent, with little or no indication roentgenographically of either the identity of the tooth or the extent of the infection. Three of these individuals are seriously ill and one gravely so, with

1. Price, W. A.: I Dental Infections, Oral and Systemic; II, Dental Infections and the Degenerative Diseases, Cleveland, Penton Publishing Company, 1924.

heart involvement. This raises the question at once: Are we correct in the premise as previously stated; and, if not, why not?

As a further introduction to the consideration of this problem, in Figure 2 we have a comparison of the apparent and actual appearance of a practical case in which, in *A* and *B*, above, will be seen the roentgenographic appearance of the first and second molars. In *A* there is distinct roentgenographic evidence of periapical radiolucence, which is but slightly, if at all, evident about the roots of *B*, the second molar; and, in *A* and *B*, below, we see that the granulomas on these roots are larger on *B* than *A*. Why has not the roentgenogram told us the whole truth? (There is no question that it tells the truth.) Is it not because we do not understand its message?

In order to determine the explanation for this phenomenon, I have made a series of determinations, some of which are as follows: By taking a piece of beef bone, and for this the sternum (the butchers refer to it as the brisket bone) was particularly well suited because it has a considerable portion of spongy bone in proportion to dense, and by cutting it in half and then placing a metal bar, approximately one-fourth inch square but with teeth on one side, between the two pieces, and placing these pieces in a vise and forcing the metal into the spongy bone, an indentation is made the size of the metal bar. This metal bar is shown roentgenographically in position in Figure 3*A*. In Figure 3*B*, we see the photographic appearance of these two indentations, and it will readily be observed that the teeth have registered their imprints in detail in the half in which they were embedded, while the opposite surface is relatively smooth. In *C* we have the roentgenographic appearance of these two pieces put in juxtaposition, and several impor-

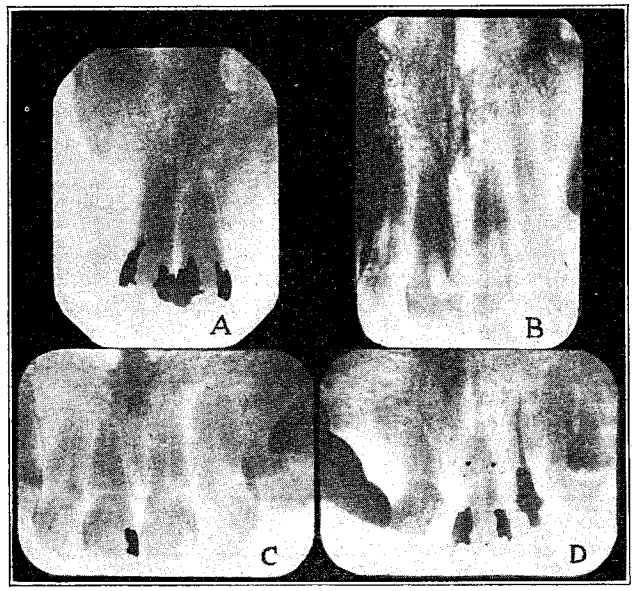

Fig. 1.—Four pairs of incisors. One of each pair is putrescent. Three of the patients were seriously ill. The true condition is not revealed.

tant things are at once discernible. The bone looks entirely different on the side to the left where the metal bar had teeth, but since the tooth prints are in alinement, there is a shading of the density, which, if we did not know what had caused it, would not be suggestive. On the other, or right hand side, where the metal bar was smooth and flat, the hole has a clear-cut limited margin, but the bone below it or beside it is more dense than the surrounding bone, because of its condensation. In *D*, we have rotated the two pieces approximately 10 degrees, and the line that separated the two pieces in the middle of the chamber now appears to be to one side of the chamber, and the chamber instead of appearing to be square with a diffused margin at one side where the teeth made their impressions is now oblong and would give one the impression of being only a fraction of the size that it appears in *C*, while it is actually the same. In *E*, we have the roentgenographic appearance at 20 degrees from *C*, and the margins are lost and the chamber is indistinct. In *F*, we have a view at 30 degrees, and the chamber is nearly lost, as also at 60 degrees in *G*. In *H*, we see it at 90 degrees, and the chamber does not show, although there is a faint suggestion of the tooth prints. In *I*, we have the roentgenographic appearance of the two halves laid side by side and corresponding with *B* in position; and it will be noted that while the bone was compressed in each half about the same amount, the final distribution of material due to displacement is a little different on account of the tapering shape of the teeth; and the half of the bone, which received the plain surface of metal, shows practically no impression or record of the chamber, shown photographically in the lower part of *B*. The bent wires show the approximate angles.

What I am desiring to demonstrate here is the effect of a zone of condensation when it occurs in the same path as an area of rarefaction. This is quite clearly demonstrated in Figure 4. In this case, a square-ended punch was driven into the bone, as shown in *A*, and the bone was then roentgenographed without the tool in position in *B*, in the same direction in which the presence of the chamber is but slightly recorded, and in *C*, at right angles to the chamber which not only shows the chamber distinctly but the condensed bone, which was nearly equivalent in total absorption quality to the same bone before the tool was driven.

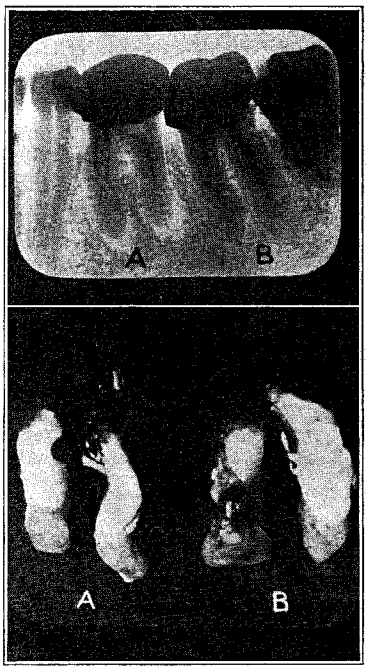

Fig. 2.—First and second lower molars shown in *A* and *B*, above, and their roots, with granulomas, after extraction, as shown in *A* and *B*, below. The granulomas are larger on the second molar than on the first molar, though their presence is not revealed roentgenographically.

We have here a duplicate of the physical condition that is found in dental practice, though it is produced by a different process. In Nature, the zone of condensation around the chamber of rare-

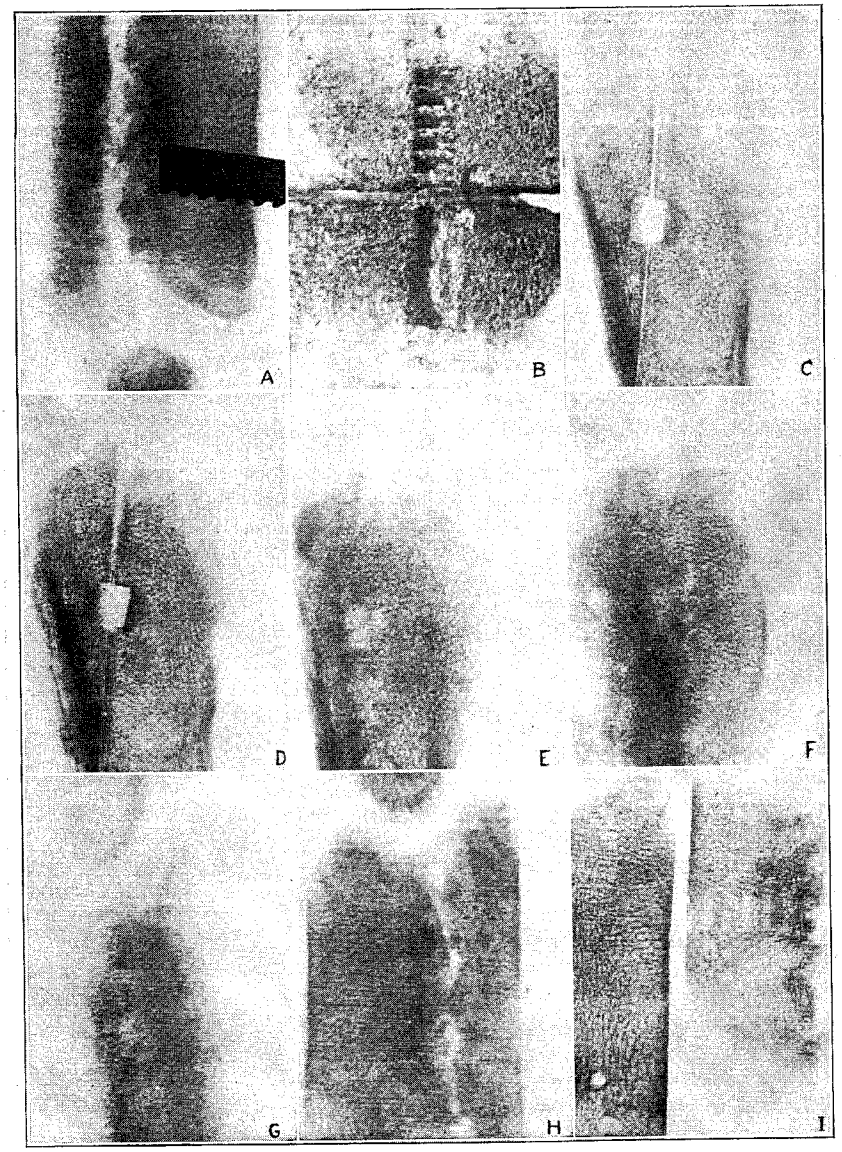

Fig. 3.—Chamber in bone at different angles: *A*, as made by a metal bar, producing a chamber one-fourth by one-fourth by one-half inch; *B*, appearance of chambers in separated bones; *C*, appearance with rays in line with the long axis of the chamber; *D* (10 degrees), *E* (20 degrees), *F* (30 degrees), *G* (60 degrees), and *H* (90 degrees) at different angles; *I* (90 degrees), the two pieces separated and roentgenographed at right angles to the long axis of the chamber. The bent wires indicate the angles. There is practically complete disappearance of this large chamber.

faction is produced as part of the reaction process, and, when superimposed above the zone of rarefaction, more or less completely disguises or obliterates its presence. We shall discuss later when and why Nature builds this type of bone.

By using a section of rib, we have more nearly the relative amount of spongy and dense bone that will be found in the mandible. In Figure 5, there will be seen two tests using sections of ribs. In *A1,* the tool, which is a cold chisel, has been driven through the section of bone in the direction of the long axis of the rib; and, while most of the material has been moved laterally, there is shown roentgenographically, in *A2,* a distinct outline of the chamber, which is about five-sixteenths by seven-sixteenths inch. In *A3,* we have the roentgenographic appearance when this chamber is viewed from the side; and is it not disquieting that there is relatively so little evidence as to its exact size? This is also illustrated in *B1, B2* and *B3* of the same figure. In *B1,* we have a screwdriver driven into a section of bone. In *B2,* we have the roentgenographic appearance in the direction that the screwdriver was driven, and, in *B3,* the relatively slight record of the presence of this chamber.

That this is just what is happening when teeth are roentgenographed for evidence of infection is abundantly demonstrated by the clinical findings. In Figure 6 will be seen the photographic appearance alongside the roentgenographic appearance of a number of extracted teeth in cases in which the granuloma remained attached to the root and by its presence could demonstrate the size and shape of the chamber from which it had been removed. The men who have been doing surgical work in these conditions are all familiar with the frequency with which they find the physical condition very different from that suggested by the roentgenogram. Since we are studying here only the question of the limitations of the roentgen ray, I am not justified, because of the large number of problems we wish to cover, in taking more time for this particular phase.

I find great difficulty in selecting typical cases to illustrate the tragic mistakes that are being made because of confi-

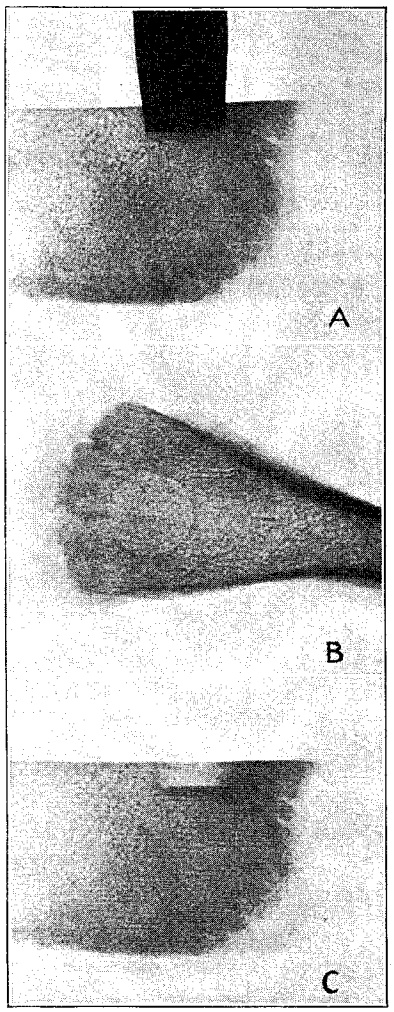

Fig. 4.—Limitations of roentgen-ray to disclose chamber in bone: *A*, square end punch in position; *B*, perpendicular roentgenographic appearance of chamber; *C*, right angle appearance of chamber. A zone of condensed bone obstructs the view in *B*.

dence in the old, and what I believe to be mistaken, basis of judgment as to what constitutes evidence of dental infection. I see many cases that would splendidly illustrate this situation. A recent case is presented herewith.

REPORT OF CASE

A woman physician, aged 65, had had removed three teeth that I had condemned in May, a diagnosis for which there did not at the time seem to her, to be justification either from the clinical symptoms or from the roentgenograms. The teeth had just previously been roentgenographed and the film interpreted by the dentist to whom her physician had sent her. When the teeth were extracted, she had a feeling that the evidence, on the basis of her training, had not justified their removal; but she had consented, because of the seriousness of her condition; and she wished now to apologize for her lack of confidence that the removal might be helpful, because of the great improvement that has been occasioned.

Her history briefly is this: She was operated on in 1917 for duodenal ulcer, for which a gastro-enterostomy was performed, and she had relief for seven years. The trouble returned with marked severity last January, and she had lost approximately 1 pound a week, going to the lowest weight since childhood, with much distress in the duodenal region. With the removal of the three teeth, shown in Figure 7 (marked x), she experienced a rapid improvement, with complete relief of the local symptoms and a return to normal weight and health in a short time. She states that she has no doubt that the removal of the teeth in question was the important factor in bringing about the change. Her physician interpreted her eosinophilia of 10 per cent as indicating an infection process. After the removal of the teeth, this was reduced to 2 per cent.

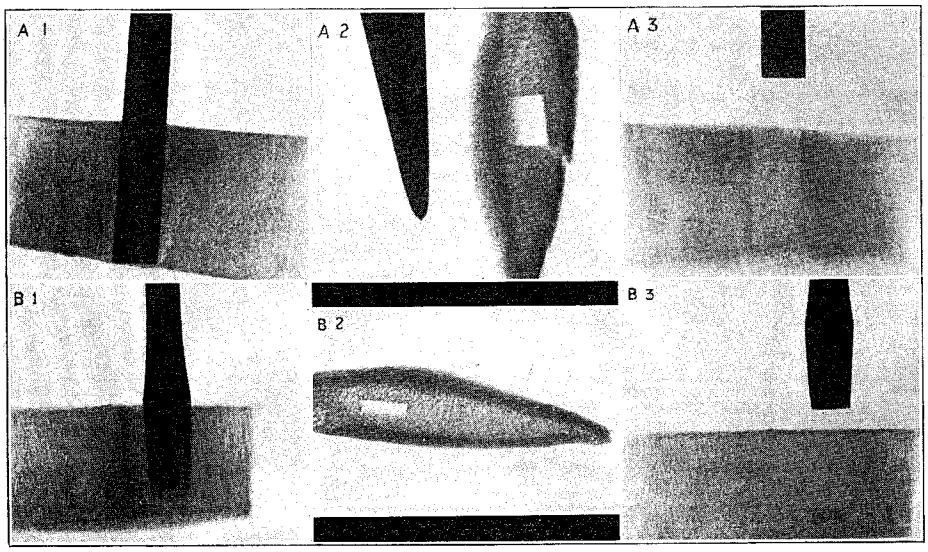

Fig. 5.—Studies of chamber in bone: *A-1*, chisel driven through section of rib; *A-2*, chisel, side view, and chamber, end view; *A-3*, right angle view of same chamber; *B-1*, screwdriver driven into section of rib; *B-2*, chamber, end view; *B-3*, chamber, at right angles.

By review of the roentgenograms, (Fig. 7), it will be noted that the upper right first and second bicuspids carried root-fillings, but were, on the basis of usual methods of interpretation, so-called roentgen-ray negative. The upper left first molar, which appears to be normal roentgenographically, did not respond normally to temperature and electric current, and contained a degenerating pulp. It, probably, with its three roots and pulp infection furnished more

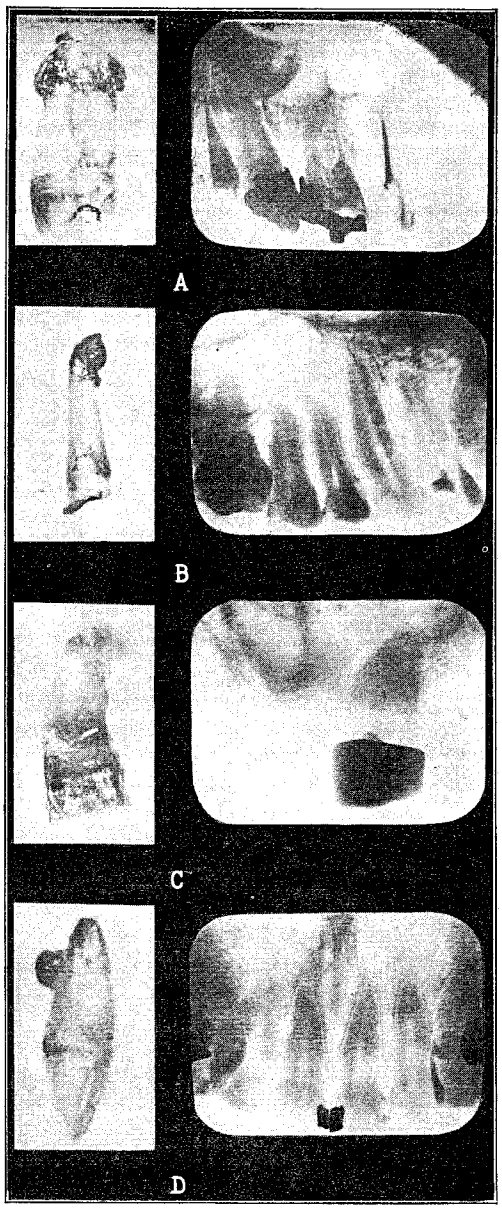

Fig. 6.—Comparison of roentgenographic appearances with the actual, as revealed by photographs of extracted teeth with granulomas. The absence of roentgenographic evidence of periapical involvement may be noted.

infection and toxic material than the two root-filled teeth. When cultured, it gave no growth in nine days aerobically, but grew a culture next day after covering with petrolatum.

This case illustrates not only the inadequacy of our accepted standards for roentgen-ray interpretation but also, splendidly, the presence of an eosinophilia in an individual with normally a high defense, carrying a chronic focal infection for a considerable time, during which the resistance has gone down. This type of person we find making up the group of individuals who develop sensitization reactions from the antigen from infected teeth. The evidence is accumulating that stomach and peptic ulcers are, in many cases, in part sensitization reactions.

BACTERIAL ACCOMMODATION TO AVAILABLE SOIL

We have thought of the type or strain of organism as being the chief and the determining factor in infection. We now have evidence that the soil is the most important factor. In other words, our chief attention is being changed from the biologic qualities of the organism to the biologic qualities of the host. I have previously presented data indicating that, when organisms are submitted to increasing strengths of various drugs, they develop a state of tolerance; for example, a strain of streptococcus, which required a dilution of 1:10,000 of liquor formaldehydi for growth came finally by successive stages and transfers to grow in a concentration of 1:320, approximately thirty times the original concentration. Likewise, the organisms changed their tolerance for alcohol through a range from 1:400 to 1:20; phenol, from 1:25,600 to 1:800; thalium sulphate, from 1:3,200 to 1:200. We have repeated these tests, applying them to several additional drugs, now sold for that purpose, and to date have found no exception to what seems to be a constant biologic quality of these strains of streptococci tending to develop a condition of endurance or tolerance for concentrations that formerly not only inhibited their growth but also actually devitalized them. These reports will be made later.

This quality of adaptability is not restricted to organisms of the streptococcus group. It has been shown, for example, that *Treponema pallidum,* when growing in the body, can acquire a state of toleration for the arsenic compounds, such as arsphenamin, if too small doses are used at first. The very principle of therapeutic procedures in infected pulpless teeth presupposes not only that the infected tooth can be sterilized but that

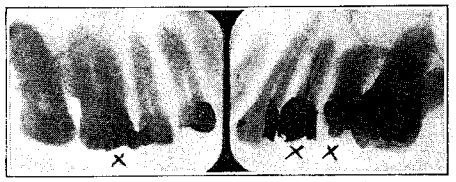

Fig. 7.—Three infected teeth, two root-filled (at right), one molar not root-filled but putrescent (first molar at left), apparently seriously injuring the patient.

a condition of intolerance for organisms can be maintained. That this state of intolerance is not readily established was early demonstrated to my satisfaction by the following test. I invested the teeth in plaster of Paris, and autoclaved the blocks containing the teeth and sent them to a series of dentists for placing of root-fillings by their methods. They did not have to sterilize the teeth for there was no question that they were sterile and free from living organisms. When the teeth were received, they were placed with their root-fillings in infected culture mediums. In two weeks, cultures were made from different parts of the teeth, and it was found that the majority of these teeth were already infected in the dentin and several grew cultures from sections of the root-filling material that

had been placed not only in a presumably sterile manner but also in teeth that had been saturated in medicaments to the liking of the operator. This also suggests why it is that such a large proportion of extracted pulpless teeth show infection when they are cultured in suitable mediums with suitable gradations of oxygen tension.

In my recent text, I have shown that when iodoform is saturated in alcohol and the alcoholic saturated solution is added to the culture medium, the organisms will grow in a 1:80 dilution of the saturated solution of iodoform and alcohol at the beginning of the test and that they grew finally in a 1:10 dilution. This seems to explain why it is that when teeth that have had their pulp canals packed with iodoform preparations in such great concentration are opened years afterwards, the odor of the iodoform will penetrate the room, and streptococci can frequently be grown from this iodoform paste.

In this connection, it is of interest to note that the biologic differentiating characteristics of organisms that are expressed in sugar fermentations are changed by these processes. In other words, we have found that organisms can be changed from a classification such as *S. salivarius* to a classification such as *S. mitis* and later to *S. faecalis.* It now seems probable that the environment in the different parts of the tooth and in different teeth of different individuals may change the ordinary mouth strains, usually found as *S. salivarius,* and relatively harmless with regard to disease-producing qualities, to other strains, such as *S. mitis* and *S. faecalis;* and indeed there is reason to believe that even the quality or extent and type of hemolysis can likewise be changed. In other words, the viridans or green-producing group seem now to have had their characteristic qualities, which have given them that classification, established by the fact that they have grown in the presence of charged oxygen and carbon dioxid tension, and changed serum proteins; in other words, the accumulating evidence compels the serious consideration, if not acceptance, of the belief that the host determines largely the biologic qualities of the organisms growing in infected teeth. An important additional light will be thrown on this theory by a consideration of the difference in systemic susceptibility of different individuals.

DIFFERENCES IN TYPE OF LOCAL REACTION AS A BASIS FOR CLASSIFICATION OF INDIVIDUALS

Another of our accepted fundamentals presupposes that an infection, if present in a tooth, will produce a destruction of bone about its root apex, which of necessity must appear as a zone of radiolucence. This assumes that individuals will behave similarly in the presence of an infection or rather as a result of the presence of an infected tooth structure within their tissues. I previously presented extended detailed evidence indicating that individuals do not react similarly, as regards the supporting structures, to a given dental infection. Our further detailed studies add much evidence that, in general, individuals do not present similar reactions but that they can be divided into three main groups on the basis of the type of reaction or tissue change produced in the supporting structures by a given dental infection. I probably should repeat frequently the words "given dental infection," for I am continually misunderstood because people apparently do not understand what I mean by a given dental infection. If a single-rooted tooth has a putrescent pulp, it presumably has more infection with both the capacity of the pulp chamber and dentin available than it would with the dentinal tubuli alone. Similarly, a molar has more capacity for holding infection than a lateral, assuming each to have a putrescent pulp. In the presence of a given dental infection, some indi-

viduals will produce a very large zone, some a large zone of radiolucence surrounded by a zone of radiopacity, and others very slight evidence of rarefaction at the apex, with or without considerable evidence of increased density; the alveolar bone about it. There is all the infection of a putrescent pulp. You will note particularly the absence of a zone of condensation about this zone of rarefaction. You will also note that there is distinct evidence of absorption of the

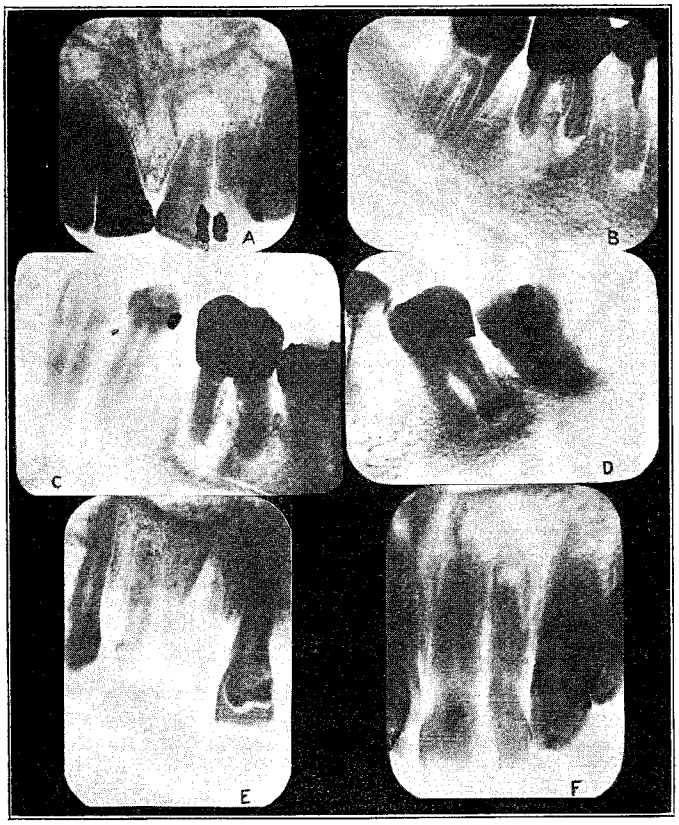

Fig. 8.—Six cases representing three distinct types: *A* and *B*, extensive apical decalcifications with root end absorptions, in individuals with a high defense and susceptibility group absent; *C* and *D*, individuals with condensations surrounding zones of rarefaction, acquired susceptibility group; *E* and *F*, teeth with slight apical absorption with tendency to condensation, in sick patients, inherited susceptibility group.

and a given dental infection may produce changes within this wide range in different individuals.

In Figure 8, we have six teeth, two of which represent one of three types. In *A*, we have a central incisor with an extensive zone of absorption of root substance itself. In *B*, we have a similar condition about the molar. In this type, on surgically opening the bone tissue, it is found that there is a marked tendency for the organized tissue that occupies this space to be in direct contact with the medulla of the bone. The

trabeculi tend to be relatively small and the intratrabecular spaces relatively large. *A* and *B* are typical of Group I.

In *C*, we have a molar that shows a marked zone of radiopacity about a large zone of radiolucence. The zone of dense bone tends to shut off quite completely the direct communication between the organized granulomatous tissue and the medulla. In *D*, we see a

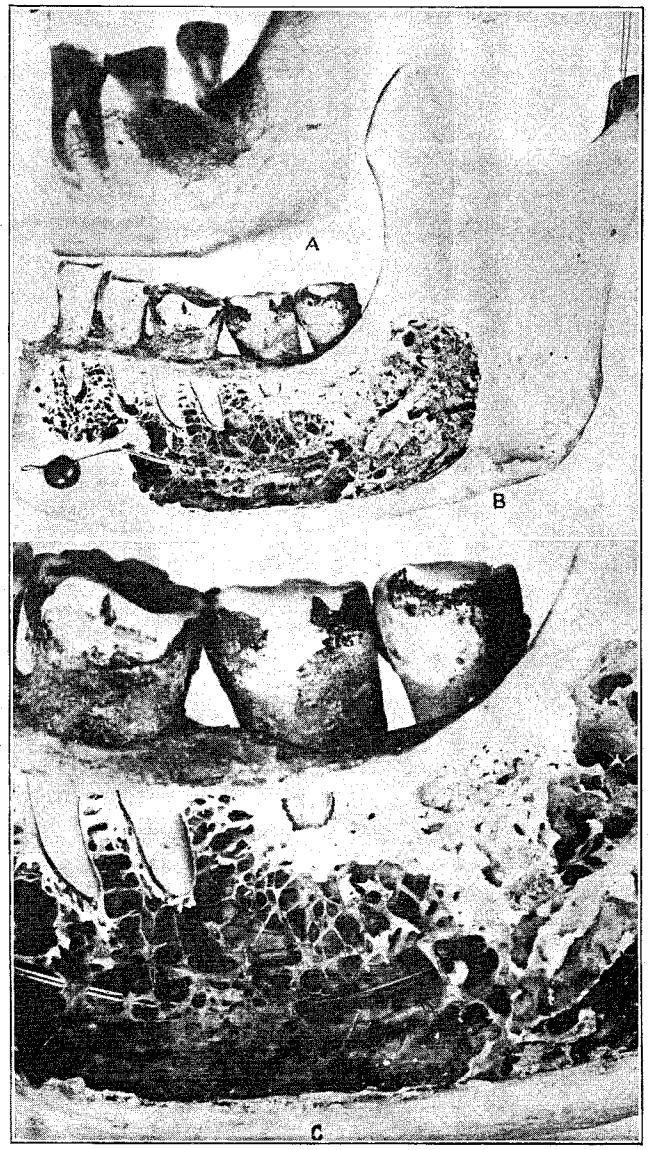

Fig. 9.—Condensed bone produced by long continued food pack between the second and third molars. The normal spaces between the trabeculi are obliterated.

similar condition, though more marked. *C* and *D* are typical of Group II.

In *E* and *F*, we have the quantity of infection of putrescent pulps and laterals, and in each case relatively a much smaller zone of radiolucence; particularly in *E*, it will be noticed that the intratrabecular spaces are smaller than in *A*. *E* and *F* are typical of the third group so far as they show the small zones, though in many cases we find more condensation than is shown here. These are both in young people, however. It is strange that phenomena that are so far-reaching in importance have received so little attention when such a vast throng has been in such close contact with this problem. This is probably largely due to the fact that we do not see the bone tissue free from its included organized or disorganized softer structures.

In Figure 9 will be seen, in the roentgenogram *A* and in the section *B*, the difference between the normal porous bone structure and the condensed bone resulting from long continued chronic irritation, in this case from a food pack between the second and third molars. *A* shows the roentgenographic appearance of this dense area. *B* and *C* show the photographic appearance of these structures, and it will be noted that the intratrabecular spaces in the vicinity of the irritation have been quite completely filled in, in such a manner as to make the bone practically continuous. In passing, this is the principal reason for the so-called dry socket, for this tissue with its poor vascularization and its frequent inherent infection has both poor repairing power and an internal infection, which bring about quite serious interference with the healing process.

DEGREE OF SUSCEPTIBILITY TO RHEUMATIC GROUP DISEASES AS A BASIS FOR GROUPING INDIVIDUALS

The diagnosis, prognosis and treatment of dental infections, as almost universally conceived and practiced, have, as the fundamental tenet, that various individuals are comparable to one another because they will, in the presence of similar infections, react with uniformity and similarly. Internists and surgeons have long observed that many individuals are found who, notwithstanding extensive dental infections, are in excellent health and give a history of always having been so. Several years ago, I began recording evidences of a familial aspect both for the type of systemic involvements that tended to develop and for the type of dental pathologic process that tended to exist in such individuals, classifying in detail the family characteristics in cases in which individuals suffered from acute and chronic infections associated with dental lesions. These data are now very extensive, including a study of more than 1,500 families, in which the individual in question, his or her brothers and sisters, the father and mother, the brothers and sisters of, each, the father and mother, and the four grandparents have all been considered. These studies have shown, as I have reported in detail, that individuals may be divided into three main groups on the basis of the presence or absence of a susceptibility to the rheumatic group diseases. These groups are, first, those with an absence of susceptibility, which has characterized the past and present of the individual and in general the members of the family; second, those with an acquired susceptibility; these, in the main, having been free from rheumatic group disturbances but having recently developed them under physical overload; and, third, those who have frequently been affected as have the members of their families. This is readily demonstrated by such analysis of the extensive data as the following:

Ten individuals were selected (and on the foregoing basis there is an average of approximately sixteen members for each of their families) having each of the following affections: rheumatism or arthritis, heart involvement, break in the nervous system, lesions of internal

organs. This study is made to compare the number of lesions in the various members of these families with those of the members of each of the other groups. This study shows that in the group of ten families, or approximately 160 individuals, when the patient's chief lesion was rheumatism or arthritis, there were fifty-nine suffering from this affection; whereas, in the other three groups, the number affected with this type of lesion was twenty-four, fifteen and thirteen, respectively. In the group with heart involvement, the figures are fifty-seven as compared with seven, nine and nine; in the nervous affections, 142 as compared with nineteen, twenty-five and thirty; and in lesions of an internal organ, ninety as compared with nineteen, thirteen and twenty-eight. As important as this observation is, it has a vastly enlarged interest and use in the light of the next important discovery, which was that individuals, when grouped on the basis of the type of dental pathologic condition and the presence or absence of susceptibility, tend to have the same individuals in unit groups, for it proves to be the case that those individuals who have the large zones of rarefaction, as illustrated in Figure 8, *A* and *B*, are the individuals in the group with the absence of rheumatic group susceptibility. By rheumatic group, we mean lesions of joints, muscles, hearts, kidneys, nervous system, etc., which tend largely to develop in susceptible tissues or to be aggravated by streptococcal focal infections. The accumulating data make the mass of evidence so overwhelming that there seems no other possible way to account for the close association between the type of dental pathologic condition and the type of systemic reaction.

Referring to the individuals whose teeth are shown in Figure 8, *A* represents an individual and family in whom the examination shows there was a total of four rheumatic group lesions. In my recently published data, I have shown that, in this group, the number of severe and mild lesions range from two to seven instances of attack for all tissues and for all members of the family in the different figures representing groups, the members of which are more or less strikingly characterized by an absence of susceptibility. Another characteristic of this group is also shown in *A* in the absorption of the apex of the root to which I have previously referred, and which is also shown in *B* on both roots of the molar and the root of the bicuspid.

B also shows an extensive zone of rarefaction and, as stated, is a typical illustration of the individuals of this group. A study of the family and of this individual shows that, in the patient and all the various members of the family, there have been only two instances of rheumatic group lesions. Neither of these two patients have ever suffered from any of the rheumatic group lesions. *A* had a sensitization disturbance, which was completely relieved by the elimination of a dental infection. We will refer to this characteristic of this group presently.

In *C* and *D*, we have illustrations from two individuals who represent the second group; namely, the one with the acquired susceptibility, and, on the basis of dental pathology, the group having large zones of rarefaction surrounded by definite zones of condensation around the zone of decalcification. When we make a study of the patients represented and the members of their families, we find that the individual whose teeth are shown in *C*, aged 32, had splendid health until recently but suffered from neuritis and sense of weakness. Four years ago, she had been overwhelmed with a series of illnesses, including malaria, smallpox and pneumonia, followed by tuberculosis, all occurring while she was carrying this and other infected teeth. A study of her case shows that there are, in all, seventeen cases of acute involvement of the rheumatic group recorded in the family. I shall refer later to the significance of an important factor here in the discussion of tuberculosis and dental infections.

The individual represented in *D*, aged 50, had acute digestive involvement with symptoms of ulcer and neck involvement. Her health had been good until the last few years, when the acute digestive disturbance began. Incidentally, it is four years since her teeth were removed, and she has had complete and continued relief from the distressing distention and pain in her stomach. When we make a study of the patient and the other members of the family, we find seventeen instances of rheumatic group lesions. This woman represents a great many of the individuals that make up a modern civilized community where the epidemic infectious diseases are under control, and people are now spared to develop the degenerative diseases. She had a sister suffering from severe stomach trouble. There is therefore this much suggestion of a family weakness in that tissue.

In *E* and *F*, we have roentgenograms showing the type of dental pathologic condition that is likely to be met with in those individuals who have not had an adequately high capacity for reaction against even a severe dental infection. In each of these cases, we have a putrescent pulp in a lateral incisor, and yet it will be seen that there is relatively little structural change revealed roentgenographically compared with the total large amount of infecting material.

E is taken from the case of a boy, aged 16, bedridden for years with deforming arthritis and so incapacitated that he could scarcely

move his hands or feet. He had been crying by the hour for months when I was called to the hospital. The multiple proliferative arthritis was so severe in practically every joint of his body that he was almost helpless, and we had to use force even to open his ankylosed jaw one-sixteenth inch to permit films to be placed in edgewise for making roentgenographic studies of his teeth. The very slight structural changes about the teeth in this patient should be noted particularly, none of which were sore or gave local discomfort. This, as we now know, is because of a lack of capacity for active reaction close to the source of infection. Photographs of this boy illustrate better than any words can the change in his life. In Figure 10, he will be seen lying in the hospital bed with his hands and feet so swollen and painful that his mother stated to me that she would be the happiest mother in the world if she could see her boy die to cease his suffering, which had been going on almost continuously since he was 12 years old. With the removal of these apparently roentgen-ray negative but definitely infected teeth, his improvement was remarkable and rapid. It was apparently assisted by the use of a vaccine. This was six years ago, and except for a recurrence of his trouble, which was again relieved by the removal of a newly infected tooth, when he returned to the city, he has been progressively improving since. Within a few months, the mandible, as well as his hands, had regained nearly normal movements, and he now does most beautiful carving. In Figure 11, he will be seen as he appears in a recent picture.

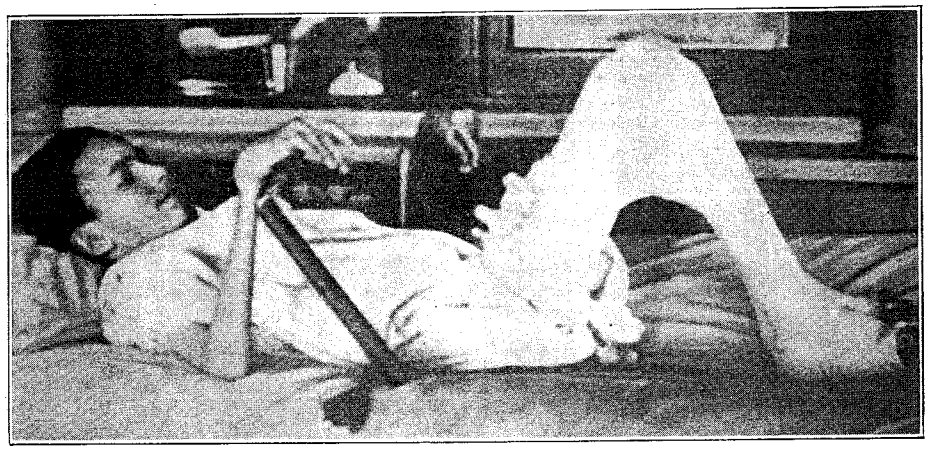

Fig. 10.—Boy suffering and bedridden for years from arthritis, whose tooth is shown in *E* of Figure 8. He has marked hereditary susceptibility.

From the standpoint of this present discussion, it is of interest to note that his father died at 57, having suffered severely with rheumatism, and his father's brother died with deforming arthritis. His mother's father suffered severely with arthritis, as did also his mother's brother. We find in the study of this case thirty instances of breaks with rheumatic group diseases. This boy is just one of the many that have had the misfortune to be born from a parentage that did not have a normal and adequate defense against strep-



Fig. 11.—Appearance, a few months later, of boy shown in Figure 10. Many joints stiffened for years have limbered up. Relief from pain is complete. The airplane and carvings were made by his hands, previously helpless for years.

tococcal infection and did have a very severe susceptibility to dental caries, which is so often characteristic of the individuals of this group.

In *F*, (Fig. 8) we have a lateral tooth of a young lady, aged 23, incapacitated from work with rheumatism and heart involvement. The slight reaction about a large area of infection, such as obtains in a tooth with a putrescent pulp, may be noted. The patient was very much underweight. With removal of her teeth, she gained 22 pounds, and in six years, she has had no return of rheumatism and heart trouble. In her family, we find that two brothers, three sisters, father, father's mother, father's sister, mother, and mother's mother, all (eleven), suffered from rheumatism, and eight suffered from heart trouble, with which four have died. In other words, in this family, we have thirty-eight breaks as rheumatic group diseases. The patient was doomed before she was born. Her mother died at 50 with acute heart involvement; her father's mother at 31, of acute heart involvement; her father at 55, of acute heart involvement.

Individuals apparently are not similar and our duty clearly is not to judge them all by the same standards with regard to their factors of safety but to adapt the load to each as conditions may justify.

CHEMICAL CHANGES IN THE BLOOD, UNDERLYING DEFENSE AND SUSCEPTIBILITY

On account of the largeness of the subject in proportion to the time available, we must pass over with but a word, the important finding, as previously published, that dental caries develops directly in proportion to our susceptibility to breaks in the form of rheumatic group diseases; which has tremendous significance since it means the direct exposure of all those individuals to a source of streptococcal infection who would probably be seriously injured by it, if such did exist as a focal infection. We must also pass over our findings that a tendency to periodontoclasia not only parallels the capacity for making a large chamber at the apices of teeth having dental infections but also develops in inverse ratio to the susceptibility to caries and in proportion to the existence of an adequate and high defense against rheumatic group disturbances. This throws tremendous light on the etiology of these diseases, but does not justify the claims of those who interpret periodontoclasia to be purely a systemic problem; for, just as periapical involvements do not develop except in the presence of an adequate irritant, in that case the infection within the pulp chamber, just so gingival reactions of a similar type do not of necessity develop except in the presence of adequate irritants, which take many forms, such as food packs, gingival deposits and, perhaps more important than either, bacterial invasion, all of which are subject to influence and in many cases to control. I am purposely inviting discussion at this point.

I have previously noted that generally patients with diabetes have, in its early stages, both high total calcium and a high ionic calcium. It is of particular interest to us also that the carbohydrate metabolism seems directly associated with the etiology of periodontoclasia, or so-called pyorrhea, and there is much evidence that one of the factors involved is a vicious cycle in which the gingival infection plays an important part. Some authorities give as the threshold of danger of blood sugar 120 mg. per hundred cubic centimeters, of resting blood; for there will usually be found several milligrams difference before and after breakfast. Evidence of the influence of dental infections on the blood sugar level will be illustrated in the following cases.

The first is one of severe diabetes, sufficiently grave that the patient has been receiving insulin three times a day. By its constant use, the urine has been kept free and the blood sugar has been kept down to 208 mg. After the removal of the pyorrhetic teeth, the blood sugar dropped to 153, which was more than half of the total pathologic sugar, with marked improvement in the patient's general feeling. In this case, there were seven teeth extracted. It is important to note that, in these diabetic

cases, we are always dealing with poor surgical risks; and since epinephrin lowers carbohydrate metabolism, it should be reduced or eliminated from the local anesthetic. This is exceedingly important.

Another typical and illustrative case is the following:

The patient's chief trouble was neuritis and digestive disturbance, associated with which, as an apparently contributing factor, he had ten infected teeth, without marked tendency to periodontoclasia. The blood sugar was 125 before the removal of the teeth, and fell to 91 thereafter. He also had complete relief from the neuritis and digestive disturbance. A letter just received from him states that he has gained in weight, and is now able to eat fruit, which he had not been able to eat before for more than a year.

Patients with diabetes develop, as part of the group of symptoms, a distinct acidosis, and it is particularly of interest to us that many of the patients presenting chronic dental infections have, as a part of the blood picture, a marked reduction in the alkalinity index. In the first of the two cases just cited the patient had an alkalinity index of 29.08, about 45 being normal. This is one of the most important factors involved in surgical shock and is one of the principal reasons why diabetic patients have such severe physical reactions following surgical procedures of all kinds, including extractions.

Since the normal hydrogen-ion concentration of the blood is distinctly on the alkaline side of neutrality, a p_H of 7 being neutral and normal blood being from 7.34 to 7.4, we have by this means an important index as to the danger of shock reaction following surgical procedure. As the p_H, or hydrogen-ion concentration of the blood, tends largely to be paralleled by the alkalinity index in the early stages of physical disturbances or as the result of acute processes, either or both of these factors give a direct indication of the factor of safety of the patient. While it is true that, in many diseased conditions, there is a compensated acidosis, in which condition the patient's body adjusts itself to a new level, we find a quite remarkable parallelism between the hydrogen-ion determination of the blood made by a potentiometer method and the alkalinity index, as is shown in Table 1. In this group, it will be seen that when the hydrogen-ion concentrations are arranged in a progressive order from 7.1 to 7.4, the alkalinity index of the blood made at the same time shows parallel depression, ranging from 25.7 to 46.46.

TABLE 1.—RELATION OF HYDROGEN ION TO ALKALINITY INDEX

Case	PH	Alkalinity Index
1008	7.12	25.70
1489	7.11	26.52
1495	7.12	29.08
1501	7.21	31.24
1491	7.28	31.08
1500	7.29	34.04
1508	7.32	38.04
1433	7.37	46.46
1503	7.41	40.30

It must be remembered that the science of blood chemistry is still in its infancy, and while much has been determined, there remains very much to be determined. In dental problems, we are concerned intimately with calcium metabolism and, it is of utmost importance that new light be thrown on this phase of our problems as rapidly as possible. Unfortunately, this work is exceedingly difficult and complicated and but few individuals have been trained for this field. It is unfortunate, indeed, that we have now no institution in a position to concentrate on this special dental problem, or probably we should say "this problem that is of special interest to the dental profession." Large quantities of material are always necessary before deductions can be made. What constitutes the difference between diffusible and nondiffusible calcium is not yet established. The former has been pre-

sumed to be in the ionic state. The question of terminology, while important, may be misleading. It is not entirely essential that we shall know the full physical chemistry involved or the exact physical state of the calcium in its different forms since we can get much information whether we think of the calcium as fixed or unfixed, combined or uncombined, diffusible or ionic, or active.

By determining the total calcium in the blood and the total calcium in the blood serum, and again in ascitic and thoracic fluid, it is shown that there is a difference in the calcium in that all of the calcium is not diffusible, since the calcium in the ascitic fluid of edema is distinctly lower than that of the blood.

In these studies, I have undertaken to accumulate extensive data, and, for want of better names, will speak of the calcium as total, ionic, or active, physiologically fixed, and pathologically fixed, with the full realization that the terminology will probably be modified, but it is the most nearly suggestive that we know at the present time. It is quite probable that some such term as diffusible or active calcium is a safer one for the present, so I have added it after the word ionic. Since the calcium of the blood is largely lightly linked to a protein molecule, it has seemed wise to work out the total albumin, and total globulin, as well as the total protein, and also the various euglobulins. A group of these are considered with the calcium and phosphorus, in Table 2. Many things of interest are shown, one of the most important of which is the difference between the total calcium and the ionic or active, a difference which varies from 0 to 4.12, in the last case of the list. Two sets of figures will be noted for Case 1514; indicating determinations before and after extractions. This is the case in which the patient's blood sugar was reduced from 125 to 91 (referred to previously) as is shown in this chart; and it will be noted that the total globulin increased from 1.76 to 3.35 and the albumin:globulin ratio was restored much more nearly to normal. It is also of interest to note that the ionic or diffusible calcium increased from 9.48 to 10.06, approximately a half milligram, in ten days, during the early part of which the extractions were made. It is of particular interest that the patients with typical periodontoclasia, with tendency to suppuration, as in so-called pyorrhea alveolaris, tend, if not breaking physically, to have an ionic calcium above 10.5 and frequently over 11. These patients also have large apical areas about infected teeth. It is also significant that the individuals with an ionic or diffusible calcium below 9.0 are nearly always below par physically, and those below 8.5 quite seriously so.

We have in our pens a rabbit, now in splendid health, that, on two occasions, has been brought almost to the point of death by placing an ordinary infected tooth under its skin and removing it just before the crisis has come. In each instance, there has been a loss in weight and also a lowering of the ionic or diffusible calcium, which has returned to approximately normal with removal of the tooth.

Another important factor in this connection is the change in the blood morphology, in that the polymorphonuclears, which may increase at first, tend quite rapidly to decrease in percentage with a corresponding increase in lymphocytes. In a group of nine rabbits so studied, the average depression of polymorphonuclears was 17 per cent and the increase in lymphocytes was 17 per cent. I have previously shown that this progressive change in the number of lymphocytes is attended by a progressive depression in the calcium. For example, in one rabbit so studied, while the polymorphonuclears decreased from 57 to 31 per cent, a depression of 26, the lymphocytes increased from 34 to 62, an increase of 26 per cent. While this was occurring, the calcium varied as follows: The ionic or diffusible calcium increased at first from

TABLE 2.—CHEMICAL ANALYSES OF BLOOD

Case	Alkalinity Index	Blood Sugar	Nonprotein Nitrogen	Inorganic Phosphorus	Calcium-Phosphorus Ratio	Total Calcium	Calcium, Active or Ionic	Calcium Physiologically Fixed	Calcium Pathologically Fixed	Total Protein	Total Albumin	Total Globulin	Euglobulin A.	Euglobulin B.	Eublogulin C.	Albumin-Globulin Ratio
1008	25.70	.....	.....	3.65	3.21	11.702	10.33	0.54	0.833	7.95	4.41	3.23	1.612	1.361	0.262	1.37
1510	28.60	125.0	28.00	2.14	5.00	10.686	9.92	0.54	0.22	8.03	4.37	3.43	0.275	2.60	0.562	1.27
1511	29.75	118.0	27.00	2.72	4.57	12.462	9.92	0.88	1.662	8.60	5.13	3.293	0.075	2.318	0.275	1.56
1513	27.76	102.0	23.00	2.00	5.86	11.702	10.40	0.84	0.568	8.57	4.575	3.799	1.668	1.431	0.700	1.345
1495	38.26	136.0	.....	5.20	2.36	12.24	.....	.....	.....	1.212	0.576	3.610	0.256	0.469	2.870	.....
1516	29.31	84.0	36.20	2.12	5.32	11.27	.....	.....	.....	8.025	5.138	2.498	0.281	1.174	1.043	2.05
1514	37.20	125.0	28.00	2.87	4.08	11.71	9.48	1.65	0.58	6.96	5.08	1.76	0.068	0.418	1.280	2.88
1390	36.12	105.0	24.00	3.51	3.11	10.92	9.12	0.96	0.84	6.62	4.119	2.350	0.290	0.800	1.260	1.75
1515	.....	117.2	36.00	4.83	2.33	11.25	9.62	0.52	1.11	5.65	.....	.....	.....	.....	.....	.....
1376	.....	92.0	24.00	4.45	2.11	9.412	9.02	0.04	0.036	6.65	3.37	3.16	0.032	1.418	1.718	10.66
1519	34.54	121.0	31.00	3.73	2.74	9.40	10.22	0.0	0.24	6.55	3.985	2.375	0.319	0.644	1.412	1.675
1527	31.16	113.0	33.00	2.14	5.67	12.142	.....	.....	.....	8.35	.....	.....	.....	.....	.....	.....
1514	36.70	91.0	27.50	3.78	3.32	12.54	10.06	1.14	1.342	6.638	3.10	3.352	0.406	0.187	2.668	1.00
1487	29.50	113.0	33.00	5.63	2.00	11.25	8.92	2.03	0.30	7.00	4.16	2.57	0.069	1.757	0.744	1.62
1521	39.10	83.0	26.56	5.45	2.12	10.54	9.52	0.10	0.81	6.72	4.14	2.410	1.075	0.700	0.627	1.71
1528	38.56	96.0	21.00	7.71	1.35	10.42	9.56	0.58	0.28	6.15	3.06	2.97	0.487	1.805	0.675	1.04
1520	32.12	113.0	28.00	3.29	3.62	11.92	8.96	2.7	0.26	6.95	3.51	3.26	0.418	1.272	1.600	1.08
1522	37.08	83.0	24.00	3.32	3.12	10.36	10.06	0.0	0.50	5.91	3.35	2.41	0.316	0.738	1.362	1.39
1267	34.12	90.2	.....	4.13	2.91	12.07	9.04	0.93	2.094	5.705	.....	.....	.....	.....	.....	.....
1527	32.21	108.0	27.00	3.94	2.97	11.69	9.64	1.43	0.62	6.67	4.269	2.225	0.118	1.256	0.850	1.92
1526	30.15	84.0	33.20	3.28	3.25	10.70	7.00	2.34	1.78	6.92	4.651	1.993	0.650	0.600	0.743	2.33
1523	27.31	97.0	26.00	4.12	2.70	11.10	8.78	2.32	1.38	5.90	4.342	2.208	0.813	0.732	0.663	1.95
1525	42.20	103.0	22.50	3.14	4.00	12.56	7.84	3.78	0.94	7.21	5.63	1.400	0.219	0.806	0.219	4.04
1529	31.78	104.0	37.00	3.21	4.12	13.24	9.40	2.68	1.16	2.28	1.61	0.506	0.181	0.112	0.212	3.18

11.53 to 11.80, then dropped progressively on successive days to 9.45, 8.46 and 8.05. Meanwhile, the total calcium increased from 13.0 to 13.22, then to 13.66 and then dropped to 8.71 and 9.80. On the third day of the experiment, the total calcium had increased 0.66 gm. or from 13 to 13.66, during which time the ionic calcium had decreased 2.08 mg. or from 11.53 to 9.45. If, now, we had been studying total calcium only, the picture would have been very different; and whether this calcium factor is properly named as ionic or active calcium, while important in that it is most desirable that we understand its physical state, is not so important as the fact that we are dealing here with calcium in different forms, or at least with different bonds, and the evidence strongly suggests that the difference between the total calcium and the ionic or diffusible is, to a definite degree, both an expression and a measure of the divergence from normal of both the well-being and factor of safety of the person or animal.

Probably no phase of this whole problem of calcium metabolism is receiving so much attention today as are the factors that disturb or control disturbances in the formation and growth of bone. This involves not only rickets as a pathologic entity but also calcification of the roots of teeth and the functioning during pregnancy. It has been shown that rickets can be cured in some instances by a restoration to the diet of absent vitamins; that rickets may be cured by sunlight and ultraviolet light, and, just recently, that even the exposure of food to ultraviolet light will impart to it the quality that will enable the same dietary under which rickets developed to cure it. I have been making studies for some time, tending to throw light on the mechanisms involved in calcium metabolism. Among the important new data developed are the following: Since we can produce in rabbits a typical ionic or diffusible calcium depression similar to that which exists in our sick patients by the simple procedure of placing under the skin of a rabbit an infected tooth, we have used such rabbits as the experimental material for again increasing the calcium. This we have been able to accomplish by different means, but the effect is practically always temporary. Calcium in its various forms, whether as phosphate, lactate or chlorid, will have little effect when introduced into the stomach unless there is administered at the same time an internal secretion hormone. For example, by the addition of a small quantity of thyroid extract, the ionic or diffusible calcium is definitely increased but tends to fall again quite soon. A continuation of administration of the thyroid with the calcium does not hold it up but instead produces a lowering of the threshold for calcium in the small intestine, and the final level is lower than before. This effect seems to be largely due to the use of too large a quantity of thyroid. The administration of parathyroid extract with the calcium lactate has a distinct stabilizing effect, for its action seems definitely to be to assist the body in neutralizing the toxins produced by the infection.

In order to secure information bearing on the problem of how light acts on the body, we have made studies of the effect of placing animals, in which we have produced metabolic disturbances, out in the sunlight. One rat, for example, that had been kept in the dark and was placed on a diet deficient in vitamins, was in a state of complete collapse, lying on its side and gasping as though in the terminal stages of the vitamin deficiency disease. In this condition, it was placed out in the bright and direct sunlight and in two hours was up and running around and eating; and though kept on the same diet, but placed out in the sunshine frequently, it made rapid improvement. This and other such tests suggested that in some way the sunlight and similarly the ultraviolet light must act on some

TABLE 3.—BLOOD CALCIUM CHANGES AFTER INJECTION OF SERUM EXPOSED TO ULTRAVIOLET

Rabbit	Serum Exposed Minutes	Before	After	Time After Injection	Active Calcium (Ionic)	Ca Ionic Plus Combined	Calcium In Combination
				Hours			
1318	15	*			8.74	9.23	0.49
			*	2	9.64	10.20	0.56
1312	15	*		1	8.38	9.92	1.54
			*		8.94	10.73	1.79
1340	60	*			9.18	9.90	0.72
			*	2	8.89	10.13	1.24
1340	20	*		2	8.76	9.64	0.88
			*		9.28	9.92	0.64
				AFTER CALCIUM LACTATE AND THYROID BY STOMACH			
1340	0	*		2	8.64	9.22	0.58
			*		9.52	11.14	1.62

elements that are carried through the blood stream and probably on some of its constituents.

In order to determine somewhat of the mechanism of the radiation, we have made an extended series of exposures of blood serum and defibrinated blood to the ultraviolet light from a mercury vapor lamp, and we have found that, when the ionic or diffusible calcium of an animal has been lowered by the means indicated above of placing an infected tooth beneath the skin, the ionic calcium of such an animal could be materially raised in even one hour by the return to its circulation of some of the serum exposed for from ten to twenty minutes to ultraviolet light. Too long an exposure, as for example sixty minutes, made a depression in the ionic calcium from 9.18 to 8.89; whereas a twenty minute exposure of the same animal (Rabbit 1340), on another day, increased it from 8.76 to 9.28. The ionic calcium of Rabbit 1318 was raised from 8.74 to 9.64 two hours after the injection of the radiated serum, and that of Rabbit 1312 increased from 8.36 to 8.94 in one hour after fifteen minutes' exposure. These various factors are shown in Table 3.

In order further to study the effect of the light, we have used the blood of animals in different states of health, as well as that of patients, and have tested the capacity of the blood to produce impressions on photographic emulsions after being irradiated. For this purpose we have used, each, the ultraviolet light, the sun's rays concentrated with quartz lens (since glass obstructs the rays of the order with which we are concerned) and also radium radiation, from radium enclosed in glass. (No radium emanation was in contact with the blood.) A typical illustration of these is shown in Figure 12. This is the first publishing of this discovery although I have reported it in dental lectures, first in February, 1924.

There is probably much significance in our finding that the blood of an individual with high defense and in good health makes a stronger photographic impression than does the blood of an individual having marked symptoms of chronic focal infection.

These findings, that the blood was

directly capable of receiving energy, which it would give off as radiation, and that the efficiency of the blood seemed in a definite way to be related to this quality, led us to study various other sub-

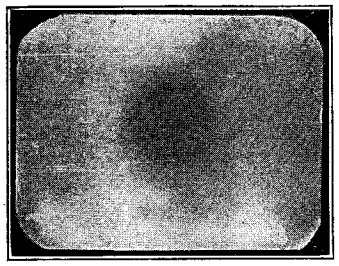

Fig. 12.—Fresh rabbit serum exposed to ultraviolet light, then used to expose film.

stances, including oils, fats, proteins and aqueous chemical solutions, to ascertain what other substances possessed this quality on exposure to ultraviolet radiation or that from the sun and radium. Olive oil is but slightly affected in comparison with cod liver oil, which is very actively affected, and persists in throwing off radiations for an extended period. We have been able to make an extract from cod liver oil that is many times more sensitive than the raw oil.

I proceeded to use these products in connection with the calcium lactate to ascertain the influence that they would exert on its metabolism. These results have been very marked and satisfactory in certain cases of delayed calcification in children and in benefiting expectant mothers. This can best be illustrated by a detailed reference to individual cases.

A boy with delayed calcification has grown eleven-sixteenths inch in about two months, although he had grown very little the preceding two years. The remarkable change in the calcification of his teeth is shown in Figure 13, which shows the roentgenographic appearance at 7 years and 4 months, and again at 11 years and 3 months, when the treatment was begun. It will be noted that the deciduous molars are in position over the forming bicuspids, and the roots of the latter are greatly delayed in calcification. These deciduous teeth were not loose, but, in five weeks after starting medication, these deciduous teeth were so uncomfortably loose that it was necessary to remove them; and, in the third view, showing the appearance at 11 years and 6 months, or three months later, it will be seen that there is a distinct change in the structure of the supporting bone and in the progress of root calcification. The boy's appearance, appetite and mental attitude have all changed. In two months, both his diffusible or ionic calcium and his inorganic phosphorus showed marked increase. This case is presented in Table 3.

The improvement in a typical case of calcium stress of pregnancy is presented in Table 4, in which it will be seen that fatigue was changed to exhilaration, depression to animation, poor appetite to an excellent appetite; and in twenty-four hours the level of the diffusible or ionic calcium was distinctly raised, as were also the levels of inorganic phosphorus, total protein and total globulin of the blood.

Another expectant mother, also in a second pregnancy, in five weeks gained 11¾ pounds, and lassitude and weariness, which only allowed her to be up an hour or two a day, changed to such physical well-being that she got up in the morning and remained up all day without even resting.

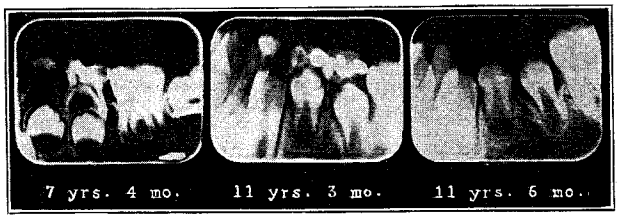

Fig. 13.—Calcification changes produced in three months by medication.

In these cases, I am recommending at present for children one 5-grain tablet

TABLE 4.—DISTURBED CALCIFICATION OF CHILDHOOD*

Conditions or Symptons	Before Treatment	After Treatment (2 Months)
Total weight	70 lbs.	74 lbs.
Height	4 ft. 5½ in.	4 ft. 6 3/16 in. (Gain 11/16 in.)
Physical state	Poorly nourished	Looks better nourished
Nervous state	Listless, irritable	Very anxious to go to school
Digestive condition	Poor appetite, shunning fruits and vegetables	Good appetite, now likes vegetables
Blood active calcium	9.06	9.56
Inorganic phosphorus	2.65	7.71
Calcium balance	—24	+73

*Case 1528; patient, aged 11½ years.

of calcium lactate, three times a day and a 0.5 c.c. capsule of cod liver oil that has been exposed in an open dish to bright noonday sunshine, both given with meals and preferably five days a week, stopping two, and to adults about twice this quantity. It is easy to overdose with activated cod liver oil, and sunshine is proving better than ultraviolet rays from a quartz mercury art lamp.

In our cases of pregnancy, we have noticed a complete change in the viscosity of the saliva, and an absence of caries. In children with marked tendency to caries, we have found this treatment exceedingly beneficial.

This important advance is the direct result of the intensive chemical researches on the blood of individuals in these various abnormal states and their comparison with the normals, and is a splendid indication of how the intelligent application of pure science will rapidly change dentistry from the maintenance of a system of repair shops, which amounts to furnishing substitutes and crutches, and must in a sense be considered a failure as compared with that newer type of dental service that is coming out of this work, by which teeth will be kept from the ravages of caries and thereby not only will the necessity for the fillings be avoided, but, more important, the possibility of apical involvement due to pulp infection and devitalization be avoided, with its tragic sequelae reducing one's efficiency and comfort and even the duration of life, by the development of the degenerative diseases in susceptible and overloaded individuals (which we all at times of stress become).

If time and space permitted we should report in detail on several other problems on which our researches have furnished important new data. We shall only briefly refer to one. In a recent paper,[2] I presented data that reveal an important relationship between the types of disease from which individuals suffer and the types of dental pathologic proc-

2. Price, W. A.: Some Structural and Biochemical Factors Involved in Dental Infections and Related Degenerative Diseases, J.A.M.A., 84: 254 (Jan. 24) 1925.

TABLE 5.—CALCIUM STRESS OF PREGNANCY*

Conditions or Symptons	Before Treatment	Time Elapsed	After Treatment
Disturbance	Many boils	6 weeks	Complete absence
Physical state	Fatigue		Exhiliaration
Nervous state	Depression		Animation
Digestive condition	Poor appetite and nausea		Excellent appetite, no nausea
Blood active calcium	10.06	24 days	10.28
Inorganic phosphorus	3.32		3.87
Total proteins	5.91		7.12
Total globulin	2.41		4.02

*Case 1522, second pregnancy in a woman, aged 30; treatment begun at third month.

esses which tend to develop in those individuals. Our accumulating data reinforce that general finding, which may be briefly expressed as follows:

Calcification and decalcification processes are related to fundamental systemic factors that determine qualities of defense and susceptibility to certain types of diseases on one hand and the nature and type of reaction about a zone of infection on the other. Thus, patients with a marked tendency to periodontoclasia, or pyorrhea, do not tend to develop proliferative arthritis but do tend to be susceptible to diabetes, tuberculosis, anemia and cancer; and, conversely, patients with a marked tendency to calcification about dental infections tend to have a much smaller amount of rarefaction about the apices of infected teeth than do patients with pyorrhea. These are the group in which are found the cases of proliferative arthritis and as a rule are individuals that do not develop cancer or tuberculosis of the miliary pulmonary type. In other words, the lesions tend to heal, probably by calcification of the tubercle; whereas, in the former group there is a tendency to make a poor defense, probably by the reverse process of decalcification or lack of calcification of a tubercle. Any person may check this by finding for me, if they can, one single instance of a patient with proliferative arthritis who is suffering from either cancer or progressive tuberculosis. I have not been able to locate one single case. (We might expect to find a case of degenerative arthritis although I have not yet definitely done so.)

Table 5 presents a consolidation of these general data. In Column 1 will be seen the three fundamental types of reaction in bone, which are used as the basis for this observation. The first group is the one with extensive decalcification, as large apical areas or extensive gingival absorptions, recorded as "rarefying osteitis with extensive decalcification." The other two groups show, progressively, tendencies to absence of decalcification in the presence of an irritant, and tendency to calcification. The first group is conspicuously absent in susceptibility, as shown in the second column, to the rheumatic group disturbances, though under sufficient overload they

TABLE 6.—SUMMARY OF DATA

Relation of Calcification and decalcification to type of systemic disease				Systemic Disturbances streptococcal and nonstreptococcal																				
Type of Local Reaction In Bone	Susceptibility Group	Blood (Ionic) Calcium Active	Bactericidal Efficiency of Blood for Streptococci	Heart	Proliferative Arthritis	Acute Rheumatism	Caries	Kidneys	Stroke	Nervous Breakdown	Degenerative Arthritis	Neuritis	Skin Sensitizations	Hypertrophic Rhinitis sen's.	Asthma Sensitization	Periodontoclasia (Pyorrhea)	Hay Fever	Extensive Salivary Calculus	Diabetes	Tuberculosis	Cancer
Rarefying osteitis, with extensive decalcification	Absent	Normal or high	High													*	*	*	*	*	*
Condensing osteitis about extensive rarefying osteitis	Acquired	High or subnormal for individual	Subnormal				*	*	*	*	*	*	*	*	*	*	*	*	*		
Condensing osteitis, or very slight rarefying osteitis, or both	Inherited	Subnormal or low	Low	*	*	*	*	*	*	*											

tend to break and then enter the next group, the acquired; whereas, the third group tends by inheritance to be chronically susceptible. The active diffusible or ionic calcium tends to be higher in proportion to the defense, as does also the bactericidal efficiency of the blood, both of which are shown in Columns 3 and 4. In Column 5, "Systemic disturbances, streptococcal and nonstreptococcal," we see, as pointed out in the text above, the marked difference in the type of systemic involvement that tends to develop in these different groups. Individuals with a marked inherited susceptibility to the rheumatic group diseases do not tend to have cancer, tuberculosis or diabetes, diseases which tend to involve the individuals whose natural defense is high in this regard. The sensitization reactions appear chiefly also, in this and the group with an acquired susceptibility, owing largely to overload plus infection. On this basis of tendency to calcification or to decalcification is expressed, apparently, a strong factor regarding capacity to resist tuberculosis. Those in whom decalcification takes place make the

poorer fight against this disease. Is it a question, in part of calcification or decalcification of tubercles? I have not, as yet, been able to find a patient with typical multiple proliferative arthritis with either tuberculosis or cancer. Can a reader refer me to one? (Not the degenerative type.) Why do diabetics tend so strongly to have periodontoclasia? Our newer data suggest that the anemias tend to develop in the groups showing a tendency to decalcification. This is discussed at greater length in the article referred to.[2]

Time does not permit a more complete discussion of this phase in this paper, nor does it permit the presentation of new data relating to the causes of pulp degeneration and newly discovered factors involved in the repair of sockets after extractions. These will be presented in a later communication.

When we contemplate the suggested significance of many of these findings herewith reported and, further, the fact that we have simply pried open some additional windows, I find myself depressed with a sense of profound regret that we do not have an institution equipped, endowed and manned, and pushing forward with all possible rapidity researches in these various lines. Perhaps the saddest phase of our situation is that we have so few men under special training and preparation for attacking these problems. If we had, the time might be moved forward by decades when suffering humanity might be expected to be relieved. I find myself continually asking whether this generation could not do more for the next. If, as seems now clearly demonstrated, dental infections are very important and often determining factors in the etiology of degenerative diseases, an opportunity for human service lies at the door of this generation such as probably has never been possible to any other. Just as Lister, probably more than any other man, had a mind prepared for the great truths regarding the relation of infections to disease, which Pasteur discovered, and therefore was able to apply those great principles to surgery, with results that revolutionized that science, just so had we prepared minds today, the newer knowledge of biology would, by its application to dental problems, bring a period of human health betterment that would go into history as one of the brightest of medical progress.