BACTERIAL INVASION AND DECALCIFICATION OF THE DENTAL TISSUES: A PRELIMINARY COMMUNICATION ON RECENT INVESTIGATIONS OF CARIES

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THE acid produced by the fermentation of carbohydrate food particles in the oral cavity, has, since the researches of W. D. Miller, been regarded as the chief factor in the phenomenon of caries. All subsequent investigations have been based upon this assumption, and have, therefore, been confined to the conditions of the mouth which favor fermentation. In the light of our present knowledge, there can be no doubt that the acid responsible for the decalcification in caries is produced by micro-organisms. The fermentation theory, as at present accepted, does not, to my mind, give a satisfactory explanation as to the place and manner of the formation of the acid that actually dissolves the lime salts of the dental tissues.

In discussing this subject, I wish to confine myself entirely to the phenomenon of caries, and to leave the question of etiology, vital resistance, etc., for future consideration.

According to the fermentation theory, particles of carbohydrate food retained in the mouth undergo fermentation which results in the formation of acid. This acid is supposed to attack the enamel, and in the later stages of the disease to

diffuse into the dentin to the point where the solution of the lime salts takes place; the organic structures, after decalcification, are destroyed by saprophytic micro-organisms. This theory cannot, however, give a satisfactory explanation for all the phenomena observed by the practitioner or by the microscopist.

The first questions that arise in considering this theory are, how strong an acid can be produced under the most favorable conditions, and what is its radius of action? In regard to the first point, Miller says: "The greatest amount of acid which I have as yet observed in lactic acid fermentation is 75 per cent." To account for the decalcification which takes place in caries, this amount appears remarkably small, if we consider the fact that the acid is expected to diffuse some distance thru other solutions, or thru substances containing moisture. Long before reaching the point where decalcification is supposed to take place, the strength of the acid would have fallen to a small fraction of 1 per cent, that is, to such an extent as to have become practically inert.

To more clearly illustrate the objections that can be raised against the acid

diffusion theory, I will consider several phenomena of dental caries with which every practitioner is well acquainted.

Leathery dentin.—This tissue, if we may still call it so, is composed of the greater part of the organic structures of the dentin. It contains large quantities of moisture and, according to Miller, 26 per cent of lime salts. In order to reach the undecalcified dentin, the acid must diffuse thru the entire layer of this leathery dentin. In passing thru the outer zone, it encounters the products resulting from the decomposition of the basis substance by the saprophytic micro-organisms. These products are invariably basic in nature and therefore must neutralize at least part of the acid. In the deeper layers the acid is further diluted by the moisture contained in the leathery dentin. However, the question most difficult to explain from a physico-chemical standpoint is, Why should the acid pass thru a tissue containing 26 per cent of lime salts without combining with these, in order to reach the lime salts contained in the more remote, newly infected dentin? To accomplish this, the acid would have to leave a tissue still containing a comparatively large amount of lime salts, to pass along the dentinal canaliculi, and to penetrate the acid resisting Neumann's sheath, in order to attack the same salts that it had refused to dissolve in the beginning.

Penetrating caries also proves to be difficult of explanation, as the acid would have to remain inert until it reached the deeper layers of the dentin. The same is true of secondary caries under leaky fillings. We should naturally expect the acid to decalcify the walls of the channel along which it penetrates from the surface, between the filling and the dentin. In practice everyone has seen numerous cases in which the dentin along the margin was healthy, while the secondary de-

cay was confined to the deeper parts of the cavity, often directly over the pulp.

Fissure caries, also, is an inexplicable phenomenon, if we assume that the acid is formed in the sulci of the tooth. The acid would have to act in the unnatural manner of refusing to unite with the salts lying in its immediate neighborhood in the enamel and in the outer layer of the dentin, in order to reach remote points along the dentin-enamel border.

These facts alone would be sufficient to shake our confidence in the acid diffusion theory, even if observations of recent investigators did not tend in the same direction. Eckermann was unable to affect carious dentin with a 2 per cent solution of lactic acid, and says further: "As a matter of fact, lactic acid does very little harm to living teeth, altho in vitro it produces a superficial erosion of dental enamel free from saliva." This would prove that the lactic acid formed in the mouth, which can only reach the area of advancing caries in a greatly diluted state, would have but slight power of decalcification.

In writing upon the etiology of caries, Pickerill states: "There is a popular saying that 'nature abhors a vacuum'; it might also be said: 'Nature abhors a free acid,' for neither can exist *naturally*. The acid seeks a base to combine with *immediately* after it is formed." In regard to the action of lactic acid the same author says: "Both Head's and the writer's experiments show that lactic acid will sooner combine with a salivary salt than with the calcium of the enamel, and that when the former combination has occurred—even tho there still exists a slight acid reaction (to litmus)—no effect will be produced on a normal enamel surface. It is also equally obvious that if acid is formed in proximity to a tooth and a sufficiency of alkaline saliva is not at hand to satisfy it, it

must *at once* combine with whatever other base is available, and this, as a rule, is found in the lime salts of the enamel surface, and a cavity is initiated."

In applying the above to the acid diffusion theory, the question arises, Why should the acid in the beginning of caries "*at once* combine with whatever base is available," while later during the process the same acid is supposed to wander long distances before combining with a base?

From the foregoing it becomes evident that the weak acid formed by the fermentation of food particles in the oral cavity, on reaching the advanced zone of caries by diffusion, (1) would have become so dilute as to be ineffectual as a decalcifying agent; (2) would have to travel a long distance from its place of production, thereby decreasing its power of reaction, which is greatest in *statu nascenti*; (3) would have acted contrarily to its usual chemical behavior by not combining with the bases in its immediate neighborhood.

If the fact that the decalcifying acid cannot be produced upon the surface of the tooth is admitted, it might still be maintained that the acid is produced by fermentation of food particles, which are by some means brought from the surface to the place where the decalcification is taking place. It soon becomes evident, however, that this theory cannot be accepted, for food particles, or even single starch grains, cannot pass thru a layer of leathery dentin, or along the dentin-enamel border, or into the tubuli of the dentin to the seat of carious activity.

A consideration of all the above-mentioned facts has led me to abandon the theory that decalcification is due to acid formed by the fermentation of carbohydrate foods or to acid saliva. The theory I wish to offer is based upon clinical experience as well as upon observa-

tions and experiments of different investigators. Even tho some points must still be substantiated by experimental evidence, I feel confident that the theory is of sufficient interest to warrant my presenting it to your notice in the form of a preliminary communication. I wish again to emphasize the fact that not the etiology but the phenomena of caries as such is to be the subject of my consideration.

As the microscopic conditions presented by caries are familiar to everyone, I need not go into detail. The essential features are the presence of micro-organisms in the organic structures of the tissues attacked and the solution of the lime salts of the infected area. It is self-evident that only micro-organisms can have produced the acid necessary to decalcify the tissues. This acid we may consider, as the result of different investigations, to be, in the main, lactic acid. As already stated, the highest percentage of this acid found by W. D. Miller in his cultures was 75 per cent. An aqueous solution of lactic acid of this strength has, as observed by my brother in his experiments, only a very slight decalcifying power. Eckermann found that *in vitro* lactic acid (strength of acid solution not stated) in presence of saliva has no effect on dental enamel. This behavior is attributable to the mucin which is present in every saliva and which covers the surfaces of all the tissues in the oral cavity. This film of mucin is present at all points protected from mechanical disturbance. But not only *in vitro* does this film protect the underlying structures from the action of the acid. Every practitioner knows that the cement lute of an inlay dissolves out only to a slight extent. The explanation given by Grieves, and I believe this to be the correct one, is that the solution of the cement stops as soon as the crevice

is deep enough to retain a film of mucin. As this mucin film is deposited upon every protected surface in the mouth, it must be specially well developed in every cavity and upon the surfaces on which caries is beginning. Is it not contradictory to assume that in caries the acid of fermentation passes freely thru a thick film of mucin to the underlying dentin, while a thinner film of mucin protects the cement lute?

The fact remains, however, that the dental tissues are decalcified by the lactic acid produced by the micro-organisms; and if we examine the conditions under which the acid acts in natural caries, we will perceive that the acid is far more active and more concentrated than that which plays a part in the experiments in vitro. Generated at the point where the decalcification takes place, the acid is in its *statu nascendi*, a condition in which it exhibits its highest power. Owing to the fact that the dental tissues contain but a small amount of fluid, the acid remains concentrated. Since the mucin of the saliva is not able to reach the point of decalcification, the diffusion of the acid is not interfered with. There can be no doubt that the lactic acid produced by the micro-organisms *at the seat of the disease* can and does decalcify the tissues in dental caries. If we reject the fermentation theory on the ground that acids formed upon the surface of a tooth cannot reach the deeper points where decalcification is taking place, and that fermentation of carbohydrate foodstuffs cannot occur at these points, as they cannot penetrate the tissues, there can be only one explanation for the formation of the acid, namely, that the formation of lactic acid by the micro-organisms can take place only in the presence of *soluble* carbohydrates. Soluble carbohydrates, however, do not result from fermentation, but are the products of normal salivary digestion.

This thought may at first appear very radical, as we have been accustomed to consider fermentation and acid conditions of the mouth almost synonymous with caries. I hope, however, that the facts I have already stated and the explanations I will offer of the phenomena that could not be satisfactorily explained by the fermentation theory, will go far toward proving the truth of my arguments.

In order to explain my theory more easily, I will divide the process of caries into two phenomena, which in the disease occur simultaneously: (1) the invasion of the micro-organisms; (2) the decalcification of the dental tissue.

As the formation of the acid has taken up our attention so far, I will first consider the question of decalcification. At the focus of the disease, the micro-organisms act upon the *predigested* carbohydrates, i. e., the dissolved sugars, and change them into lactic acid. This acid, in *statu nascendi*, combines with whatever lime salts it finds in its immediate neighborhood, thereby forming a solution of high osmotic tension. This solution, following the laws of osmosis, will diffuse thru the leathery dentin toward the surface. As the acid has been neutralized at the point of its formation, it will in passing outward, not disturb the alkalinity of the putrefactive process taking place in the outer zone of the dentin. On the contrary, the dissolved phosphates may even (according to W. D. Miller) stimulate the growth of micro-organisms. The lime salt solution, in passing thru the leathery dentin, takes up soluble substances which increase the osmotic tension; this increase is, however, slight in comparison to the osmotic tension produced by the mineral salts. Upon reaching the mucin film, which in this case acts as a dialyzing membrane, an osmotic exchange takes place, the lime salts passing out into the oral cavity and the

water of the saliva passing into the infected dentin. This water, however, contains dialyzable products of salivary digestion in the form of sugars of low osmotic tension. This dilute solution of sugars diffuses thru the leathery dentin, being steadily drawn onward, owing to its low osmotic tension, to the points wherever solutions of lime salts with high osmotic tension are being formed. On arriving at the seat of bacterial activity, the sugars serve as nourishment for the micro-organisms, while the water takes up the newly formed lime salts, thereby being increased in osmotic tension and, in its turn, passing out to the surface.

I have previously mentioned several conditions which cannot be satisfactorily accounted for by assuming that dental caries is caused by acids formed in the oral cavity. Can they be explained by the theory that only such acid as is formed at the focus of the disease, plays an active part in the phenomenon of caries?

Under "leaky fillings" we have the following conditions. The narrow opening of the crevice will most certainly be obstructed by a film of mucin, which, as has been already stated, will prevent acid and food particles from entering. I will go even further and state that I doubt whether the micro-organisms can penetrate this mucin film, if it is of any thickness. Predigested carbohydrates, however, can and do pass thru and furnish nourishment to whatever micro-organisms are *already present* under the filling. The position of these micro-organisms determines the point at which secondary caries begins. If they are present in the dentin at the margin, decay will begin there; if present only at the deepest point of the cavity—and the dentinal tubules of even a well-excavated cavity often contain micro-organisms at this place—caries will begin at this

point. It may even penetrate to the pulp without attacking the dentin lying nearer the margin. On the other hand, if there are no micro-organisms present in the dentin, it is possible for a filling to become "leaky" without secondary decay occurring.

Before considering penetrating caries and fissure caries, I wish to express my views regarding the invasion of the micro-organisms, as this is an important factor in the explanation of these phenomena. So far, I have considered only two factors in the process of caries: the chemical, i. e., formation and action of acid; and the physical, i. e., diffusion and osmotic tension. These factors alone do not, however, suffice to explain the advance of the disease along certain lines within the tissues. A third factor in the process of caries, which, in my opinion, is the most important, and which so far has not received sufficient attention, is the *vital factor*.

In invading any tissue, micro-organisms which have no movement of their own—and this is the case in carious infection—can progress only by proliferation, or thru the movement of the fluid in which they are suspended. The latter condition does not apply to caries. If the theory of diffusion is correct, large numbers of micro-organisms cannot be carried into the unaffected tissue, as they are situated at the point of highest osmotic tension and the movement of the fluid is consequently from this point to the surface, i. e., to the point of lowest osmotic tension.

Wherever micro-organisms advance by proliferation, they will naturally grow along the path that offers the most suitable nourishment. The micro-organisms of caries require a *mixed nutrient medium*, one containing proteids as well as carbohydrates. This is proved by the cultures in the laboratory as well as by conditions in the mouth.

Undoubtedly there are micro-organisms present under the mucin film covering a cement lute, and soluble carbohydrates can certainly pass thru this film. Still, the micro-organisms do not grow, and no acid is produced, because the cement contains no digestible proteid substance. On the other hand, the dentinal canaliculi under many fillings contain micro-organisms, as has been repeatedly proved. They, too, do not proliferate or produce acid, altho easily digestible proteids are present. They, in their turn, lack that slight amount of soluble carbohydrate nourishment necessary for their growth.

As the result of our microscopical examinations of the dental tissues, my brother and I have found that the micro-organisms of caries advance most rapidly in the organic structures containing protoplasm. The contents of Tomes's fibers—which we consider to be the tubular prolongations of the odontoblasts, having the same contents as these cells, and consisting, therefore, of almost pure protoplasm—offer the richest nutrient medium to the micro-organisms. For this reason, the invasion of Tomes's fiber and the interglobular spaces is more rapid than that of any other part of the dental tissues. The next most favorable medium seems to be present at certain areas of the dentin-enamel border. Neumann's sheaths, the outer layer of Tomes's fibers, i.e., the walls of the odontoblastic processes as well as the interprosmatic structures of the enamel not being protoplasmic in character are but slowly affected.

If we keep in mind the facts just stated, we can readily explain the phenomenon of penetrating caries. If, at the point where the infection of the dentin occurs, the canaliculi are larger than those in the neighborhood, and the organic matter in the dentin-enamel border

is comparatively slight, the micro-organisms will proliferate into these larger canaliculi, as they offer more protoplasmic material than the other parts of the tissue affected. More acid will be produced and therefore a more rapid solution of lime salts will take place at this point. The consequent higher osmotic tension will draw more soluble carbohydrates to this point and furnish new nourishment to the micro-organisms. This process will go on until the pulp is reached, *if these canaliculi continue to offer more rich albuminous material than other places in the tissue affected.* Should, however, a new supply of protoplasmic material be invaded nearer the surface—perhaps an interglobular space—and thus a new point of high osmotic tension be formed, the invasion of the canaliculi originally attacked will be retarded or even brought to a standstill; thereby, changing the so-called penetrating caries into the form usually met with.

In this connection, I wish to mention the so-called chronic caries, which is characterized by a slow rate of progress. In my opinion, this phenomenon is due solely to the fact that the tissues attacked contain but a small amount of easily digestible organic matter. Relatively few micro-organisms can, therefore, develop; the amount of acid formed will consequently be small, and decalcification will advance but very slowly. If the advancing caries encounters an area in which there is practically no protoplasm present, the micro-organisms can no longer develop, and as a result the formation of acid, and with it the decalcification of the dental tissue, whether it be enamel or dentin, ceases. The condition necessary for the development of such arrested or self-limited caries, are most often found in the enamel, as this tissue contains but a slight amount of easily digestible proteid matter, especi-

ally in its outer layer. In dentin this phenomenon is met with almost exclusively in that variety of this tissue, known as senile dentin. Every practitioner has observed such cases. They have been variously designated as black caries, healed caries, or, as W. D. Miller names them, decay marks.

The zone of invasion in carious dentin, examined under the microscope, shows a very irregular margin. If decalcification were due to acid penetrating from the surface—as assumed by the fermentation theory—the line of advance would be quite regular. This is, however, not the case, even under conditions most favorable for the action of the free acid in the mouth. Sections made of the carious dentin of a freely exposed shallow, cervical cavity, show irregularly distributed funnel-shaped areas of infection, varying greatly in width and in the depth to which they penetrate the dentin. The point of the funnel is invariably formed by one or more infected odontoblastic processes or so-called Tomes's fibers. Each funnel is surrounded by a zone of decalcified dentin, the line of demarcation running symmetrically to the outline of the infected areas. Between these and extending to the free surface exposed to the fluids of the mouth, there are areas of undecalcified dentin with uninfected dentinal canaliculi present. Such conditions cannot be accounted for by the fermentation theory. Furthermore, if we assume that the free acid of the mouth attacks the surfaces of the dentin, the disease would advance by a solution of the lime salts of all of the exposed basis substance, with a subsequent invasion of micro-organisms. Microscopic examination, however, shows the reverse to be true, an invasion of the dentinal canaliculi and a subsequent decalcification of the intermediate basis substance.

From what has been said above, the

phenomenon of fissure caries not only becomes explicable, but, in my opinion, it also helps to prove that caries is primarily an infection of the organic structures of the dental tissues. The course of the infection corresponds exactly to the distribution of the uncalcified organic matter as determined by histological examination.

In this connection, I wish to emphasize the necessity of completing our knowledge of the histology of the enamel and dentin, as without this the true explanation of dental caries will never be found. Is it not contrary to reason to try to explain the pathological conditions of a tissue whose histology is not known in detail? This is the case with enamel, the tissue primarily affected by caries. Recent investigations have gone far toward clearing up this question; the outer zone of the enamel, however, has not been, as yet, sufficiently studied in regard to the distribution and nature of the organic matter which it contains. For it is, in my opinion, just this uncalcified organic matter which is most intimately connected with the initial lesion of caries.

The last point to which I wish to call attention briefly is the parallelism existing between dental caries and the infectious diseases. In both cases micro-organisms gain an entrance into the tissues and attack the cellular protoplasm; in caries, the contents of the odontoblastic processes, the interglobular spaces, and the structures along the dentin-enamel margin. The bacterial activity in both cases results in the production of substances deleterious to the tissues infected. In one case, toxins are formed, in the other, acids. Furthermore, the tissue destroyed by the primary disease is subject to reinfection by a different micro-organism, for instance, the mixed infection of tuberculosis, in which the tis-

sue destroyed by the tubercle bacilli is reinfected by pyogenic micro-organisms. To this condition dental caries also presents a parallel, the decalcified tissue being subsequently attacked by saprophytic micro-organisms.

In conclusion, I wish again to call attention to the following facts based upon the investigation of my brother and myself:

1. It is physically and chemically impossible for the acid formed by fermentation of food particles in the mouth to reach the zone of advancing caries in a sufficiently active state to decalcify the tissue.

2. The acid, which dissolves the lime salts, can only be formed by the action of micro-organisms at the point where the decalcification takes place.

3. The growth and activity of these micro-organisms are dependent upon the presence of a mixed nutrient medium, i.e., one containing proteids as well as carbohydrates.

4. The necessary carbohydrates are brought to the seat of infection by osmosis in the form of dissolved sugars. These cannot be produced by fermentation but are the products of normal salivary digestion.

5. The micro-organisms producing caries, not having any movement of their own, must advance by proliferation; the rate of growth being in direct proportion to the richness of the nutrient medium.

6. In dental caries, the micro-organisms always invade the tissues along certain definite lines. Microscopic examination of the dentin has proved that these lines are identical with that part of the uncalcified organic matter which is richest in albumin, i.e., the contents of the odontoblastic processes, the contents of the interglobular spaces and the structures along the dentin-enamel border.

7. What is true of the dentin and of the inner zone of the enamel must also

be true of the outer portions of the enamel. Not, however, before the distribution of the uncalcified organic matter in the outer zone has been definitely determined can the initial lesion of caries and with it the etiology of this disease be explained.

8. The many similarities between caries and infectious diseases in general seem to justify the assumption that further investigation will prove dental caries to be an infectious disease.

Further histological research and further bacteriological investigation will undoubtedly furnish an explanation of the initial lesion of dental caries, which will lead to a true and simple solution of the question of etiology. The futility of trying to determine the etiology of a disease before its pathology is known, is proved by a statement made by a well-known author on dental caries, to the effect that after estimating the combinations and permutations of all the factors of dental caries, he finds that in about 3,628,000 cases of caries, no two might have exactly the same combination of causes. I believe, however, that when the true explanation of the etiology is forthcoming, we will be satisfied with less.

DISCUSSION

Weston A. Price, Cleveland, Ohio: I would like to ask if Dr. Bodecker has, with this new information, been able to produce artificial areas of caries?

C. F. Bodecker, Berlin: My brother and I have worked on this subject for only a short time. My brother was led to this conclusion by examining the specimens that I had made for the purpose of studying the dentinal tubules, and we have not as yet made any bacteriological examinations or conducted any experiments along this line, and therefore have not been able to demonstrate that we can produce artificial caries in this manner. But I am absolutely certain that we will throw some light on this subject by producing artificial caries in the laboratory.

Question: Will you kindly explain Tomes's fiber as you understand it, the circulation in the dentin, etc.?

Dr. Bodecker: My idea of the formation of Tomes's fiber is that it is not in the form of a solid fiber but in the form of a tube.

Neumann's sheath incloses the protoplasm of the fiber, which is the prolongation of the odontoblast. After finding that Tomes's fiber was a tube and not a solid fiber, I came to the conclusion that if we would treat the dentin by my special histological method, pyridine under vacuum, to dissolve out all of the protoplasmic fluid in Tomes's fiber, we would have a tube, of which you have seen specimens in cross-sections of dentin, one ring representing Neumann's sheath, and in this ring a second, which represents the tubular process of the odontoblast.

In regard to the circulation in the dentin, I consider that the circulatory fluid passes out from the odontoblast, thru this tubular process of the odontoblast (Tomes's fiber) and carries the nutrient fluid to the basal substance in the dentin. That explains how we can have changes in the dentin. We note that in age the canaliculi become smaller; how would the calcium salts be transported except by these channels? If we admit that a circulation goes out from the pulp to the surface, we have to find some channel which will take these fluids back to the pulp tissue, and as there is a space around the odontoblastic tube, inside of Neumann's sheath, my brother and I have considered this to be the return channel. Up until the present time, nobody has given to Neumann's sheath its full physiological significance. It has simply been seen, demonstrated and recorded, but no one ever considered that it had any function. Neumann's sheath is a condensed basal substance, but chemically it must be somewhat different, because it acts differently under the stains.

Question: Then is it by that process that the enamel of the tooth becomes hard after its first development in a child?

Dr. Bodecker: There are two theories which are advocated as to the hardening of the enamel after eruption. As stated, I have not seen the literature for five years, so I am not able to consider the work of the later authors, but in Germany it is considered (and I do not adhere to this view) that the enamel is hardened by the salivary salts, thru osmosis, by passing thru Nasmith's membrane. I do not see why we have to find some different mode of metabolism for every tissue of the body. We have circulation in the dentin, and we have organic matter in the enamel, in connection with the pulp. Why should the tooth not continue to calcify and harden thru the original channel of the pulp? What is the function of the pulp? Why should it remain? It is a large organ, it occupies quite an area in a temporary tooth. When the

tissues of the tooth have absolutely hardened, in forty to fifty years, the pulp diminishes in size, until it is like a thread.

William Hopkinson, Milwaukee, Wisconsin: I would like a little more clearness in regard to the circulating fluid passing in and out of the dentin. If the so-called odontoblastic processes pass into the dentinal tubules not as processes at all, but as tubular extensions of the odontoblastic cells, what can be the character of this circulating fluid; is it blood or lymph or is it simply hyaloplasm from the odontoblastic cells?

Dr. Bodecker: Probably it is lymph. It contains albumin and calcium salts. The albumin in it is responsible for our regarding Tomes's fiber as solid, because this albuminous fluid is coagulated by the fixing agents we use, therefore in many microscopic specimens, as ordinarily treated, the fiber appears to be solid. Of course, it is not blood, and as Dr. Noyes, in his investigations on the lymphatics of the pulp, has again proved their presence, it is probable that these furnish the nutrient fluid in the dentin.

J. M. Prime, Omaha, Nebraska: How do you view the result to the central artery or nutrient supply of the tooth in the loss of the pulp? What has been your observation of the effect upon the tissues normally nourished thereby?

Dr. Bodecker: I undoubtedly believe that the dentin of the crown and enamel of a pulpless tooth are dead and have no further circulation. The root, as high as the gingiva, receives circulation from the periodontal membrane. As yet I have done no experimental work on the cementum. I cannot show you the channels of circulation between the cementum and the dentin, but from clinical experience I believe they exist. A pulpless tooth is decidedly more liable to caries; the crown will break off, the enamel is more friable, and it will soon, in an neglected mouth, crumble down to the edge of the gum. And in that condition, the roots can stay in the mouth for many years, caries advancing slowly from the gingiva, but the sides and walls will stand longest as these are attached to the pericementum and still receive nutrition.

I believe that the change of nutrition of the tooth is one cause of caries. That is the vital factor. Take for instance a case of proximal caries in an upper molar and bicuspid. Why should one proximal surface have a large cavity and the other have only a brown spot? If primary caries is due to local formation of acid from food particles, lodged between the teeth, it should act in all directions, and the acid should therefore attack the enamel of both teeth at the point of contact equally. And it would do so, unless there were a vital

factor which causes the acid to dissolve the more predisposed tooth more easily than the other.

I think you have all known patients that have had little predisposition to caries, and then within, say, six months they have come back with several freshly decayed areas. And in nine cases out of ten examination of the general condition will show that either just previous to or right after such a predisposition to caries, the patient has developed some systemic disturbance.

Question: Do the fibers running thru the dentin convey sensation? Otherwise why is the dentin sensitive?

Dr. Bodecker: We used to think that Tomes's fiber conveyed sensation and nutrition, but in the last ten or twelve years quite a few authors have proved the presence of nerve filaments in the dentin. The reason they have not been proved before is that the microscopical technical methods have not been adequate for demonstration. Now the function of sensation is attributed to the nerve filaments above, and nutrition to the tubular processes of the odontoblasts.

Russel W. Bunting, Ann Arbor, Michigan: I presume Dr. Bodecker knows of the work of Pickerill, which has had a profound bearing on this subject. In some of the work that we have done we have followed Pickerill, and have noticed this: That the enamels of various teeth differ, and they differ especially on their external surface when first erupted and during the subsequent years. We find that the condensation and the perfecting of these enamels is a process taking place after eruption, and that the defects that exist in this structure are on the periphery of the enamel—not seemingly in the deeper or dental portions. We have also noted that even in adult life the teeth vary as to their porosity on the periphery, in one mouth they may be very pervious and in another exceedingly impervious. We also find that these differences in the enamel structure are in direct relation to the amount of calcium in the saliva. This conclusion is very similar to that arrived at in the work of Broderick of England several years ago. And we have been able to work this out to a point where we feel certain that there is a relationship, whether coincidental or actual, between the amount of calcium in the saliva and the perfection of the condensation of the enamel.

We have added to this the direct proof that it is possible for salts to permeate the enamel and dentin to the pulp from the saliva inward or from the pulp outward. We have done this in the test-tube, under every precaution, to prove that this is possible. From this viewpoint the condensation of the enamel

appears to be a physicochemical phenomenon depending on the salt concentrations of the blood within and the saliva without the tooth.

Dr. E. C. Kirk said many years ago: "It is a strange thing that the organisms which cause dental caries, and which we believe feed upon the carbohydrates of the mouth, should burrow deep in the tissues and grow away from their source nourishment, when all other forms of organic life grow toward their nourishment." Therefore Dr. Bodecker's expression of the correlation between proteins and carbohydrates is a very interesting statement. So also is his suggestion that the bacteria of dental caries may be carried into the dentin by osmotic flow.

I was greatly interested in what Dr. Bodecker said regarding the production of artificial caries. Miller said he produced artificial caries, but I am sure that the more one has worked with this problem the more one doubts that Miller ever did it. He produced artificial decalcification of the tooth, but true caries is something we have never been able to produce except when he had the pull of osmosis. We put a tooth into saliva that was fermenting, covering the whole tooth and leaving a little hole to see if we could produce a cone-shaped entrance into it; and invariably we found that we could only produce a parallel decalcification, which is not characteristic of true caries.

I wish to ask if pulpless teeth are more prone to carious attack than are vital teeth; whether or not caries in pulpless teeth is the same as it is in vital teeth, and whether it does spread more rapidly in the pulpless tooth than in one that has a vital pulp. In such cases we, of course, have entirely eliminated all possibility of true osmosis as a factor in the case. We have been watching for clinical evidence regarding the progress of dental caries in pulpless teeth for some time but as yet have not enough data to be conclusive.

One other factor I wish to mention in regard to the reason why caries is deep-seated rather than superficial. We have, and I know Dr. Bodecker has time and again, made decalcifying solutions and put certain teeth into them, and they would remain there days and months and never be affected, but another group of teeth subjected to the same conditions would decalcify rapidly. So we cannot get away from the type of the individual tooth structure we are working with. Do we not find that deep, penetrating carious lesions are cases in which the enamel is not readily affected by the acids we are dealing with in caries? But if by osmosis that process can be carried into the dentin, the lesion is then operating upon a structure which is more soluble, and it is our experience that those

deep-seated cases are confined to certain types of teeth.

All this brings us back to the question, What is a normal tooth?

Dr. Bodecker: It is my conviction that a pulpless tooth is more liable to caries than a vital one. I do not base my conviction upon laboratory, but upon clinical experience, and I have considered this point for over fifteen years. The pulpless tooth is, to my mind, considerably more affected by caries, in spite of the fact that after it has been well excavated, and the pulp removed and the approximal cavities well extended so that only the labial and lingual walls stand; nevertheless these walls will often fail long before those of adjacent vital teeth.

The structural differences of enamel are undoubtedly caused by a varying percentage of organic and inorganic matter. Several years ago this was absolutely denied, but Pickerill has proved the existence of malacotic and sclerotic enamel. I believe that three factors are concerned in the predisposition to caries: first the vital factor, second the structural, and third, the ratio of organic to inorganic matter.

Vida A. Latham, Chicago: As regards pulpless teeth Dr. Bodecker is right in his statement that the lower third of the tooth, the "root," if we may so term it, to the gingival border, has a better circulation than the enamel ever can have. We must not forget the cause of the loss of pulpless teeth. The loss of that tooth depends a great deal on the difficulty of preventing a chronic inflammation. Those who have extracted the roots of teeth, whether they have been crowned or not, know that there may be a great deal of thickened membrane around them. That membrane, of course, is in a chronic inflammatory condition, which can become sub-acute. For instance, a pulpless tooth is very comfortable; it has been crowned very nicely, and the patient pays no attention to it, but often, if the immunity declines a little, swelling forms, with a little tenderness, and the patient visits the dentist because the tooth is a little sore to bite on. The dentist applies something, the tooth is comfortable again, and the patient will go along for a time. Histologically what happens? If you take a root like that out, cut it and make a microscopical section, you will find there is an osteoclastic formation going on just as bone is reabsorbed in the process of development. As fast as the osteoblasts build up bone the osteoclasts pull it down. That is why a pulpless tooth becomes so loose that you have to take it out. Sometimes there is a granulomatous tendency or one to malignancy.

As regards circulation, I have had a speci-

men which showed the circulation of the blood absolutely to the outer layer of the enamel. I also had specimens which demonstrated the nerve filaments passing into the enamel, and I reported a case where one could demonstrate the nerve filaments coming on the outside of the cuspid.

The method of growth of the enamel is another interesting point. Some have had cases of cuspids with an extra point on the distal surface, and patients have had this point ground off because of irritation to the tongue. There must be a proliferation of enamel to produce it. The question arises as to whether enamel grows or not. Miller said it did.

The essayist stated that teeth vary, and they do vary tremendously. I have injected teeth in which I have had beautiful illustrations of circulation right thru into the dentin in loops and into the enamel, but I believe the best injection in every case is at the apex and gingival border. I have slides in which the dentin and the granular layer show the circulation right thru to the alveolar surface, and in some cases the nerve supply runs with it.

Dr. Prime: I want to know if it is true, and if true why we frequently find caries arrested just as it approaches the pulp. It does not appear to me to be a calcific protection from the pulp, but there seems to be some effort in nature to arrest the process at that point, and we find a good, substantial protection between the carious cavity and the dental pulp.

Dr. Bodecker: I believe that in all cases of arrested caries, some condensation of the basal substance of the dentin will be found, building up a wall between the cavity and the pulp and also that the amount of organic matter in the protoplasmic bodies has been reduced and almost eradicated. Therefore, in this case, one of the two factors needed by the micro-organisms of caries for their proliferation (proteid nourishment) is lacking. They may have plenty of carbohydrates from the mouth, but they will not have the proteid material; therefore the process of caries comes to a standstill.

In regard to Nasmyth's membrane, it is true that very little work has been done on this tissue. On the cusps and grinding surfaces it does wear off, but on the labial and buccal surfaces I believe that Nasmyth's membrane may persist for many years or thru life. I think it is a very important subject because it has something to do with the initial lesion of caries.

A. T. Rasmussen, La Crosse, Wisconsin: In connection with the statement that pulpless teeth are destroyed more rapidly by caries

than are teeth with vital pulps, I would like to ask what Dr. Bodecker's experience has been as to just how caries attacks the tooth in these cases. In pulpless teeth there are usually large restorations of metal or some other material. We know that the most vulnerable point of any tooth, whether pulpless or not, is at the junction of the tooth substance and the artificial restoration—in other words, the cavity margin. How have these teeth been attacked in your experience? Has the caries which ultimately destroyed such crowns attacked them at that point, viz., the cavity margin or have they been liable to attack at other points not usually so attacked in vital teeth?

I might say that from my own clinical experience, I do not feel justified in saying that pulpless teeth are more readily attacked by caries than other teeth, in the same mouths, having vital pulps, other conditions being the same. It is possible, of course, that observations made in private practice would be different from those made in a free clinic or similar institution. It would seem, however, that the private patients who are seen regularly for a term of years would be better cases for observation than those who are seen only a few times.

Dr. Bodecker: The most vulnerable part of a tooth, pulpless or not, is at the point of junction of tooth tissue and restoration. But pulpless teeth are usually filled with due regard to that. We have in our textbooks the special warning to extend our fillings up over the cusps to prevent these from being

injured by mastication. And even if we do that, they frequently decay. These teeth are more brittle, and the reason lies in the fact that the organic matrix in the enamel of a pulpless tooth has not the binding power; therefore the prisms seem to cleave more easily under the stress of mastication.

A. E. Webster, Toronto: If we accept the theory that the dentin is dead in a pulpless tooth, then there must be some different scheme of decay taking place in the enamel of a tooth that is dead. I cannot agree with men who say that the root of a pulpless tooth is in the same condition as that of a vital tooth. To come back to the crown of this tooth, we heard it stated that when the dentin in the crown is dead the dentin in the root may not be dead, and that this is the reason why the root does not decay. I think it has been shown by some observers that there is a certain anastomosis in dentin which perhaps carries nourishment to the crown, and yet caries would because of vitality the more readily attack it. I do not agree at all that the nonvital tooth does not decay any more rapidly than the vital one. Dr. Bodecker refers to the increased friability of the enamel in pulpless teeth, and everybody here must be familiar with that fact. Why should it be more friable in a pulpless tooth than in the vital tooth, unless there were some degenerative change present?

Dr. Bodecker: We place great weight on the idea that the micro-organisms of caries require a mixed nutrient medium for their proliferation.