THE PRESENT STATUS OF CARIES CONTROL MEASURES*

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It is my assignment to summarize the vast amount of material which has been presented at this Workshop. The task is an easy one, if not actually superfluous. I have been familiar with the studies of all the essayists as I have listened to most of them discuss their problems many times, but I am compelled to remark, at the risk of breaking the long-established taboo against dental caries research workers complimenting each other, that they certainly carried the mail this past week.

All of the speakers appreciate the rigid requirements for effective research as stipulated by Dr. Wallace at the opening of this meeting. You have listened to the various phases of dental caries research presented dispassionately and very simply by lecturers whose observations were supported by data which were presented to you for inspection. Your conclusions may differ, even as there are differences of opinion among the investigators who have gathered these data. That is quite natural since all of us view this material from vantage points based upon backgrounds of varied personal experiences.

This conference, however, was not characterized by confusion. After listening to and participating in these discussions those of you whose job it is to disseminate dental health material should have little patience with the oft-repeated phrase, "Little is known about the etiology and control of dental caries." A great deal is known about both. The mechanism by which the carious lesion is produced has been clarified by studies which were described during this Conference. It is perfectly obvious, from the data which have been presented, that the onset of caries activity can be predicted and that the development of the lesion can be prevented. That, it seems to me, is knowing a great deal about a disease. It is true that the known control measures are not practical in every case, but the fact that they are workable at all, even under very special conditions, is evidence that dental caries research has been fruitful and that the future is not as bleak as it appeared not so many years ago.

In order to appreciate fully the significance of this Conference it is well to remind you that the lecturers were not selected because of any uniformity of opinion. Nevertheless, it was apparent that there was agreement on some points. That should be a comfort to those of you whose job it is to transfer information from the laboratory to the field.

Writers on this subject have shown a tendency of late to refer to dental caries research workers as constitutionalists or environmentalists, depending upon whether they think that the disease is influenced by systemic or environmental factors. We in Ann Arbor, for instance, have been cited as horrible examples of the environmentalists because so much of our attention has been focused upon the relation of lactobacilli to dental caries activity, and also because we were never able to relate caries to the state of nutrition. As a matter of fact, it would be ridiculous to assume that conditions surrounding the teeth, which

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exist in a body cavity, are not influenced by systemic conditions. Consequently, it is of interest to determine which systemic conditions might regulate such dental environmental factors as the oral flora and conditions which permit the production of acid on tooth surfaces.

We have heard a lot about both during this Conference. All of the speakers agreed that caries control measures are effective through their influence upon the tooth's environment. It was also apparent from these discussions that acid production in the mouth is of major importance.

From Dr. Chase's scholarly dissertation, it is evident that the dental tissues are vulnerable to the action of acid. Dr. Stephan demonstrated differences in the acid potential of dental plaques. He pointed out that the pH of the plaque drops immediately after the ingestion of sugar and that the extent of the drop varies directly with the degree of caries activity on each particular tooth surface. From this it may be assumed that the nature of the bacteriological flora of the dental plaques may vary from tooth to tooth. This should suffice to relieve anyone of the delusion that the caries process is a simple one. However, two facts are definitely known: a quantitative relationship must exist between acid production and the carbohydrate substrate from which the acid is produced. It has also been shown that the enzyme system which is responsible for the acid production on the tooth is bacterial in origin. Inasmuch as the flora associated with caries is characterized by the presence of lactobacilli, most attention is being centered upon the fermentation of carbohydrate in the mouth and upon the growth characteristics of lactobacilli.

A relationship has been shown to exist between the presence of carbohydrate in the mouth and the growth of lactobacilli. The carbohydrate then may have a double-barreled action in that it provides both the substrate for acid production and a growth factor for the bacterial which brings about the acid production. Dr. Becks has described the clinical application of this knowledge. By restricting the amount of sugar in the diets of a large number of patients, he has observed a drop in the number of oral lactobacilli and a reduction in caries activity. While he was describing the diet which he employed so successfully in California I could catch the gleam in Genevieve Stearns' shining eyes because it was apparent that the Becks' diet is nutritionally adequate and in good balance and certainly Dr. Stearns and her associates in Iowa have for years maintained that an adequate diet which meets the nutritional requirements of the body will serve to prevent dental caries as well. We have been impressed with the evidence presented by Dr. Stearns relative to the importance of nutrition in the maintenance of good growth and health. Many of us are wondering, though, if caries wouldn't have been arrested in the Iowa studies on diets which were less perfect nutritionally but which contained the same low level of sugar intake. We think we made just that observation here at Michigan when we demonstrated an extremely low incidence of dental caries in a group of children on a low-sugar diet which was not optimum in a nutritional sense. Although I can't imagine Dr. Stearns ever experimenting with children on inadequate diets, I have an idea that some day the Iowa and Michigan studies will come out at the same place.

Dr. Robinson was well supplied with the ammunition necessary to demonstrate how completely dental caries is disassociated from general disease as well as with perfectly normal states of which pregnancy is a classic example. Certainly Dr. Armstrong presented sufficient evidence for an understanding of why dental caries is not associated with disturbances of mineral metabolism.

There are dentists in this room who have observed patients free of dental caries even though they consume large amounts of sugar. They haven't seen many such patients it is true, but they exist, nevertheless. That is why Dr. Williams is interested in the serological characteristics of lactobacilli. There are times when conditions in the mouth are not compatible with the growth requirements of lactobacilli so that the immunological implications should be explored. He has found, as have others, that lactobacillus agglutinins may be produced in the blood of human beings. He has also learned that before much more can be learned about immunization against lactobacilli, it will be necessary to untangle a complex serological relationship which exists between many types of lactobacilli. It is also apparent that Dr. Williams is well on the way to clarifying this difficult problem.

There are other possibilities in the caries prevention field. The role of silver nitrate in caries control has been discussed for years. Among us here, Olin Hoffman of Iowa thinks it works and John Knutson of Washington thinks it doesn't. Dr. Zander, whose job it was to render a verdict, "isn't saying." He is of the opinion that silver nitrate could be given a fairer trial. If I have misconstrued his deductions, I'm sure he'll let me know about it.

Dr. Hine was unable to tell us that prophylaxis and tooth brushing are very effective means of combatting dental caries. His assignment was most difficult since practically no statistical data are available on this subject. I think we can deduce from Dr. Hine's presentation and the discussion which followed it that the prevalence of dental caries will not be reduced by tooth brushing but that the lesions themselves are less extensive when they occur on teeth which are regularly brushed.

Remarkable results are not to be expected from tooth brushing unless of course you brush your teeth with Dr. Kesel's tooth powder. He has found that lactobacillus counts are reduced following the use of a tooth powder which contains dibasic ammonium phosphate and urea. He has gathered sufficient data to theorize that the ammonia ion inhibits the lactobacillus, thus allowing the entrenchment of Bacterium aerogenes. This organism produces an enzyme which deaminizes the amino acids, thus liberating ammonia making it difficult for the lactobacillus to re-establish itself. A cycle is thus created which to the lactobacillus is "vicious." Dr. Kesel and his associates are conducting a study in several Illinois cities in which school children use the preparation under strict supervision. The outcome of this extensive program will be awaited with great interest. Certainly I should prefer a tooth brushing regimen to one which requires the diligent selection of low-carbohydrate foods.

In a sense, antibiotics play an important part in the inhibition of lactobacilli through the use of the ammonium urea compounds by favoring the growth of B. aerogenes. According to Dr. Hill's studies, lactobacillus counts can be reduced by the use of penicillin dentifrice. Neither Dr. Kesel nor Dr. Hill has presented clinical data which indicate the extent to which caries is controlled by these means, but the bacteriological evidence is good. Some allergists frown upon the regular use of penicillin, and there is the problem of maintaining the potency of penicillin at room temperature. To date, Dr. Hill has seen no contraindication for its use because of sensitization. The fact that he is not advocating the use of this drug for the control of caries at this time does not minimize the importance of these studies. The discovery of a means of altering the oral flora contributes enormously to the knowledge of the etiology and control of this disease.

One of Dr. Hill's famous protégés was fortunate to make his debut in a problem about which there has been no controversy. I am referring, of course, to Dr. Arnold and his interest in the epidemiology of caries with reference to fluorine in domestic water supplies. Everyone agrees that low caries attack rates occur in populations supplied by waters which contain fluorine. One reason for the lack of argument about this fact is due largely to the degree of perfection of the epidemiological techniques employed by Trendley Dean, Arnold, and their They had incontrovertible evidence before they made known their You have noticed that Dr. Arnold is unwilling to state that the addition of sodium fluoride to water will reduce dental caries activity. I have enjoyed witnessing several episodes during which some pretty insistent inquisitors have attempted to extract such a positive statment from him. Of course I've had similar experiences myself, but it's much more fun watching Arnold's reaction to these traumata. The fact remains that it is not yet known whether or not the addition of fluoride will reproduce the conditions which we have observed in areas where fluorine occurs naturally. Our a priori reasoning tells us that positive results will be obtained in experiments now in progress, but it certainly is dangerous for research workers to draw conclusions from data which have not yet been obtained. Even if we should find that water treatment is effective, it is apparent from Dr. Arnold's review of this subject that calcium fluoride and bone meal in the products now on the market are not effective caries control agents. At any rate, it is not conceivable how fluorine taken internally would affect the susceptibility of teeth already calcified. Dr. Knutson has presented very impressive evidence which indicates that sodium fluoride applied topically to teeth reduces the incidence of caries. His studies confirm the observations of others that multiple applications of fluoride to the teeth of children up to 15 years of age are effective. When Arnold, Dean, and Singleton applied sodium fluoride to the teeth of 19-year-old men, no beneficial results were observed. Their study differed from those yielding positive results in that they treated an older age group and, although they made three applications, the treatments were given at the same sitting. This would indicate that either the age of the patients or the interval of time which elapses between the applications is very important. Dr. Knutson's findings indicate that four treatments given within a two- or three-week period produce optimum results in children up to fifteen years of age. It is possible that four treatments applied to adults during the same time interval would be equally effective. At present, there is no statistical evidence indicating whether or not adults may benefit by this treatment. It is interesting to speculate upon the mode of action of fluorine. Several possibilities have been considered. Fluorine is an enzyme poison and could interfere with the fermentation of sugars into acid. It could reduce acid activity by lowering the solubility of the teeth, or by inhibiting the growth of the bacteria themselves. Perhaps it works in all three ways. In this connection it is a curious fact that when applied topically fluorine does not reduce the oral lactobacillus count despite the fact that lactobacillus counts are unusually low in individuals whose teeth were calcified in fluoride areas. It is quite obvious that we have here a fascinating problem. Fosdick will very likely come up with the answer some day.

His studies have thrown a great deal of light on the mechanism by which acid is produced on the teeth from carbohydrate. Dr. Fosdick has experimented with enzyme inhibitors in the hope of finding a method of treating sugar so that it may be used with impunity. Two-methyl-1, 4-naphthoquinone is such a substance. He has presented evidence which indicates that the use of chewing gum containing it has a caries-inhibiting effect. Although it is theoretically sound, I do not think that the practicability of this procedure has been established. I have the distinct feeling that Dr. Fosdick will find a way, but I hope it won't require the regular use of chewing gum.

At any rate, Bibby has shown that chewing gum isn't a very good vehicle for fluorine. He was one of the first to obtain satisfactory results with sodium fluoride used topically but has not been able to find that mouthwashes or other salts of fluorine are practical.

In closing these rather rambling remarks, I hope that the workers in public health who have attended this Conference agree with me that they can go home with some definitely established facts about dental caries control. First of all, dental caries activity can be greatly reduced by the adoption of low-sugar diets. That these diets should meet all of the nutritional requirements for good health is just plain common sense. It is also clearly evident that it is possible to maintain earies-free teeth in a poorly nourished body. That, of course, is merely of academic importance.

Dental caries activity can be reduced by the topical application of sodium fluoride. Mineral and fluoride preparations now being marketed are not effective. Of the measures in the immediate offing, I am most encouraged by the possibilities of fluorination of public water supplies, the use of dentifrices utilizing the principle of dibasic ammonium phosphate and urea, and the treatment of fermentable sugars with enzyme-inhibiting substances. It has been a long, hot week, but I hope you have benefited by these discussions as much as I have.

DR. EASLICK.—You are well fed, relaxed, and ready for more. I know that the next speaker is relaxed because he doesn't know that he is the next speaker. You may be sure that none of these proceedings have escaped his attention. Dean Bunting, will you please tell us what you are thinking about?

DR. BUNTING.—I am very pleased to have the opportunity of expressing my personal appreciation of this significant conference on dental caries. As I have sat through the sessions of the week and have listened to the presentations made, I have been impressed with the scope and the quality of study that has been devoted to this problem. It recalls to me the early days of dental caries research and by comparison shows so clearly how much progress has been made.

It has been my good fortune to live through and to be intimately associated with a long period of evolution in the study of dental caries. I was privileged to know W. D. Miller personally and, had he lived, I was to have been his assistant in the new laboratories which were built for him in the new dental building here. That was in 1907, just forty years ago.

Following his untimely death, I was left alone to carry on in this school the investigations in dental caries and pathology to which Miller had contributed so largely. For this, I was wholly unprepared and it was many years before any substantial progress was made. In these early years of development, I was associated with Faith Hadley, U. G. Rickert, and Mary Crowley. Our progress was slow and halting until in 1930 when we obtained a substantial grant from the Children's Fund of Michigan to undertake a concentrated and coordinated study of the problem in which trained workers in the fields of bacteriology, nutrition, chemistry, and dentistry were employed. In the organization of this Michigan Dental Caries Research Group and the conduct of the studies, Dr. Philip Jay has always played a very important role.

If, as the result of our studies here, we have been able to make some significant contribution to the knowledge of this subject, we are indeed very glad. Since the termination of our group studies, many new developments have been brought forward, and previous observations have been given greater substantiation. In this school Dr. Jay has carried on notable studies of the carbohydrate factors and, in conjunction with the National Institute of Health, has taken an active part in the experimental fluorination of community water supplies. Major research projects are also being conducted in other centers and the investigators are delving into many new and untilled fields of study related to the problem of dental caries. It is with great satisfaction that we view these advancements in knowledge and the gradual extension of the frontiers of understanding of this intricate problem.

Looking back in reminiscence over the years of active study of dental caries, we have seen many alluring theories come and go; panaceas and methods of prevention have been proposed and widely accepted, only to be found that they were but will-o'-the-wisps that led nowhere. Today they are gone and forgotten, except for their record in the annals of our profession, but one day they were broadcasted throughout the land as great discoveries which would forever abolish the scourge of dental caries. I have often thought, in reviewing the stages by which our present knowledge has been evolved, that if I had the pen of a Paul de Kruif, I would write a book outlining the interesting trends of thought through which we have passed and I would call it "The Romance of Dental Caries." To me it has been a glamorous quest that has been at times almost an obsession. Now that I can see so much substantial progress as has been reported at this conference and the zeal and true scientific approach with which the investigators are attacking the problem, I have real hope that even in my lifetime, this great question may be solved, and that the boon of freedom from dental caries may be vouchsafed to mankind through dental research.

May I wish you Godspeed.