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AMERICAN COLLEGE OF DENTISTS

Objects: The American College of Dentists "was established to promote the ideals of the dental profession; to advance the standards and efficiency of dentistry; to stimulate graduate study and effort by dentists; to confer Fellowship in recognition of meritorious achievement, especially in dental science, art, education and literature; and to improve public understanding and appreciation of oral health-service." — *Constitution, Article I.*

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Fellowships and awards in dental research. The American College of Dentists, at its annual meeting in 1937 [*J. Am. Col. Den.*, *4*, 100; Sep. and 256, Dec., 1937] inaugurated plans to promote research in dentistry. These plans include grants of funds (The William John Gies Fellowships) to applicants, in support of projected investigations; and also the formal recognition, through annual awards (The William John Gies Awards), of distinguished achievement in dental research. A standing committee of the International Association for Dental Research will actively cooperate with the College in the furtherance of these plans. Applications for grants in aid of projected researches, and requests for information, may be sent to the Chairman of the Committee on Dental Research of the American College of Dentists, Dr. Albert L. Midgley, 1108 Union Trust Bldg., Providence, R. I. [See "The Gies Dental Research Fellowships and Awards for Achievement in Research," *J. Am. Col. Den.*, *5*, 115; 1938, Sep.]

Journal

AMERICAN COLLEGE OF DENTISTS

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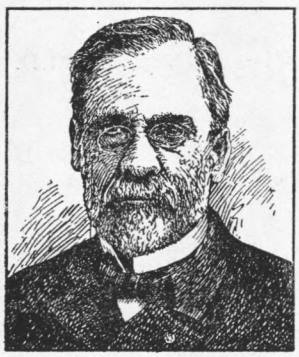
American College of Dentists

PASTEUR said: "Young men, have confidence in those powerful and safe methods, of which we do not yet know all the secrets. And, whatever your career may be, do not let yourselves become tainted by a deprecating and barren scepticism, do not let yourselves be discouraged by the sadness of certain hours which pass over nations. Live in the serene peace of laboratories and libraries. Say to yourselves first: 'What have I done for my instruction?' and, as you gradually advance, 'What have I done for my country?' until the time comes when you may have the immense happiness of thinking that you have contributed in some way to the progress and the good of humanity. But, whether our efforts are or (are) not favoured by life, let us be able to say, when we come near the great goal, 'I have done what I could'."



LOUIS PASTEUR

DISTRIBUTION OF THE ORGANIC MATRIX OF THE ENAMEL IN THE HUMAN TOOTH AND ITS RELATION TO THE HISTOPATHOLOGY OF CARIES[1]

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INTRODUCTION

Caries of the enamel is more than a simple process of acid erosion or solution of its inorganic constituents and it has long been recognized that the pathology of the disease is intimately related to the nature and extent of the organic elements comprising its matrix. In the past decade many theories have been advanced as to the role of this matrix and as to its final disposition in the adult fully-calcified tooth. Opinion in this regard has been somewhat uncertain, halting and contradictory. Increasing evidence has, however, been forthcoming that an appreciable amount of the matrix is present in the fully calcified enamel. This view is held by many investigators but it is generally thought that the matrix persists only as remnants in isolated portions of the calcified enamel and with few exceptions only the most casual consideration has been given to the possibility of a continuous matrix being present in the adult structure. The organic remnants are assumed to undergo a continued change, possibly in the nature of desiccation, and in the view of some, this change may proceed to complete or almost complete substitution by inorganic salts. There is likewise little certainty as to the function of the organic material. It is contended that it permits the distribution of

[1]Supported in part by a grant from the American College of Dentists to the University of California, College of Dentistry, Operative Division, and conducted in the Division of Operative Dentistry.

dental lymph[12, 14, 19, 20, 27] within the enamel and allows the transport of mineral salts associated with the increasing hardness of the enamel observed with progressive age.

Much of the conflict in opinion is due perhaps to the difficulties encountered in recovering the enamel matrix in any quantity for histological examination. As is well known, the effect of the strong mineral acids used for decalcification is to produce almost total dissolution of the enamel, rendering it impossible to trace pathological processes. For our understanding of this important aspect of the caries problem, investigators have been compelled to utilize for the most part, ground sections with all their limitations. The first important step taken for the solution of these difficulties was the introduction of the acid celloidin decalcification of Bodecker,[9, 10] which with its subsequent modifications,[1, 18, 22, 28] allowed the recovery, in varying degrees, of the organic constituents and the direct examination of some of the changes occurring in them. The existing methods are, however, exacting and difficult, and from the point of view of completeness of decalcification and preservation of the matrix, not entirely successful, nor can they easily be used for routine purposes.

In previous communications,[39, 45] the precise relationships of the ameloblast, Tomes' process and the detailed elements of the enamel rod and developing matrix, were discussed. It was concluded that the product of the ameloblast was the enamel matrix in which calcification occurred by a secondary humoral process and the necessity of divorcing the process of matrix formation from that of its calcification was emphasized. The recovery of a complete matrix structure in undecalcified thin sections of the enamel of the molar and incisor tooth of the rat, pig and other animals, observed just prior to eruption and secondarily decalcified under the microscope, suggested the probability that this material persisted in its entirety. We were further encouraged by observations on the recovery of the matrix and behavior of fixatives in the routine preparation of adult teeth of both man and animals for histological sectioning.

The purpose of the present paper is to demonstrate the existence of a complete matrix in the adult human tooth, to describe a method

for its recovery and the factors which influence its preservation after the exhibition of decalcifying agents; to show that the structure of the adult matrix retains the essential morphology of that laid down during development; and to illustrate the relationship which this matrix bears to the histopathology of caries and the effect upon it of bacterial invasion.

MATERIAL AND METHODS

In order to recover the full width of the organic matrix of the enamel, the closest attention must be paid to several factors, namely, fixation, rate of penetration of fixatives, decalcification and the relative rates of removal of the calcium salts from the dentin and enamel.

As already mentioned, Bodecker[9] as early as 1905, introduced a method of decalcifying in acid celloidin based on the conception "that as soon as the acid dissolves the lime salts, the celloidin fills up the space occupied by these, and in this manner sustains the fine organic matrix of the enamel."

He drew attention to the importance of immediate fixation and recommended absolute alcohol or a saturated solution of bichloride of mercury for this purpose. He observed the differences in rate of decalcification between dentin and enamel and advised the removal of some of the dentin to prevent undue loss of enamel structure. Malleson[35, 36] appears to have been one of the first to appreciate the importance and need for a more complete fixation of the enamel prior to decalcification and by combining the decalcifying agent with a fixative carried on these processes simultaneously. He was able to recover appreciable amounts of the matrix. Other investigators using either the Bodecker[1, 6, 7, 18, 23, 28, 38] or Malleson[25] techniques or direct decalcification[2, 3, 4, 5, 26, 31] have been similarly successful to varying degrees.

Fixation: The successful recovery of the enamel matrix primarily depends upon the complete denaturation of the protein, thus rendering it less soluble to the action of acids used for decalcification. *In a material as dense as enamel, time and penetration are important factors.* Observe *Fig. 1* where surface penetration has resulted in

the fixation of the peripheral units, central areas having undergone dissolution. This suggests that by preliminary removal of the dentin and exposure of a second surface to the fixative more complete penetration would be assured.

In initial experiments preparations were fixed in 10 per cent neutral formol, alcoholic formol or formol saline. It was found that best results were obtained from specimens fixed in aqueous formol for considerable periods of time, up to six months. The rate and completeness of penetration was studied by the addition of light green to the fixative which proved doubly advantageous in revealing the presence of dentin remnants and their amount. In employing the dye to check the different penetration levels it is realized that the rate of penetration of the dye and the fixative are co-incidental and that this criterion might not be applicable to other tissues.

It was observed that in contrast to the aqueous formalin where fixation is relatively slow and continuous for long periods of time, alcoholic formalin penetrates rapidly at first but after superficial penetration further progress is arrested. We have no rational explanation for the varying behavior of fixatives but it seems permissible to consider that dehydration occasioned by alcohol prevents further diffusion of the fixative.

Penetration and fixation is more rapid in young enamel, in areas where calcification is less complete and in the peripheral portions of carious lesions where the inorganic salts have undergone partial dissociation from the organic matrix. The rate and degree of penetration was observed to be greater from the dentino-enamel junction than from the outer surface.

Decalcification: The time required after fixation for decalcification of the enamel is considerably less than that necessary for the dentin. If appreciable amounts of dentin are left in the preparation, the time of exposure to the acid necessary for its decalcification is so extended as to result in solution of the less acid resistant enamel matrix. The latter element is consequently lost long before the dentin has been sufficiently decalcified to allow of histological sectioning. During the removal of the inorganic constitutents, the process must be carefully controlled, otherwise parts of the matrix

throughout the structure are digested by the acid which in turn results in collapse of the remaining portion of the matrix and subsequent confusion of morphologic detail.

Digestion of the enamel matrix by the acid agent can be determined and controlled to a certain extent by the periodic examination of the preparation under the microscope. Nitration of the organic matrix yields the characteristic yellow color of the well known xanthoproteic reaction and the onset of this reaction is indicative of the end point of declacification of the enamel. *This end point must be precisely and carefully observed if the matrix is to be saved with any degree of completeness.*

Material: The material employed in this investigation consists for the most part of fully erupted normal and carious human teeth from different age groups.

Procedure: The specimens recently removed from human beings are *immediately* fixed in 10 per cent neutral formol. After preliminary fixation for six months, sections five mm. thick, of normal and carious enamel are freed from dentin by cutting with dental burs until only remnants of dentin remain at the dentino-enamel junction. Fixation in 10 per cent neutral formol together with light green is continued for varying lengths of time depending on the rapidity of penetration of the fixative.

The completely fixed enamel is then decalcified by placing the specimen on gauze stretched over a frame constructed of platinum wire and then immersed in 5 per cent nitric acid in 80 per cent alcohol. Decalcification is complete in 24 to 48 hours in contrast to the decalcification of dentin which requires a considerably longer period of time. Because of the delicate structure of the decalcified organic matrix the most careful handling is necessary. After decalcification of the matrix, dehydration without preliminary washing is commenced with 70 per cent ethyl alcohol and continued at 5 per cent steps to absolute alcohol and ether. From the alcohol-ether, the specimen is infiltrated at 56° C. for two weeks successively in 10 per cent, 25 per cent and 50 per cent low viscosity, nitrocellulose solution (Koneff and Lyons).[32] The nitrocellulose is allowed to harden slowly in a covered embedding dish at room temperature for

several days, loosening the margins from time to time as the nitrocellulose contracts. The block is trimmed and allowed to harden further for an extremely hard block is essential if thin sections are to be obtained. Sectioning is carried out on a sliding microtome and cut to the order of 3 and 4μ.

The principal stains employed in these studies are hematoxylin, hematoxylin and eosin and Masson's trichrome, supplemented in the case of the carious material by MacCallum's Gram and Gram-Weigert methods, Ziehl Neelsen and Murray's modification of Van Giesen. A modified Von Kossa's[33] silver nitrate stain is also employed to check for the presence of calcific material and completeness of decalcification.

THE NORMAL ENAMEL MATRIX OF THE ADULT TOOTH

The organic matrix is distributed throughout the entire structure of the adult enamel of both man and animals, e.g., the rat, pig and monkey, and may be recovered from teeth of any age group if the proper procedures are employed. In man, the enamel matrix is in its essentials similar in structure and morphological arrangement to the matrix of the pig and rat and parallelisms can be drawn between all three. In these forms the unit of the matrix is the hyalinized rod together with the inter-rod or interprismatic matrix. As has been previously described[39, 45] it is possible to distinguish during development, several elements in the structure of the rod matrix and to differentiate different stages in their hyalinization. Each rod presents a cortex, core and sheath. The peripheral or cortical portion is the most completely hyalinized element, is first to calcify, and remains the most highly calcified portion of the rod. The center or rod core is less completely hyalinized, less heavily calcified and may contain areas which are immature and which have failed to undergo complete transformation. Surrounding the hyaline rod and therefore external to the cortex is the rod sheath which can be selectively stained and which appears to be in continuity with the cell membrane. It is therefore assumed to be a thickened remnant of the wall of the initial ameloblast from which the rod arose. The rods are connected by an inter-rod matrix which possesses staining reactions similar to, although less

intense than, those of the hyaline rod itself. This substance is calcified at a slightly later period and may remain as the least fully calcified portion of the enamel.

In the adult tooth all these early structural characteristics of the matrix can be identified and are identical with those of the developing stages. The staining reactions of the various elements, *Fig. 2*, are similar but less precise.

In specimens in which the calcium has been totally removed and subsequently stained with Masson's trichrome combination, the matrix shows a continued change in reaction with progressive age. In the young but fully erupted tooth, the matrix accepts the acidophilic dye strongly and is quite red; in the older, staining is less intense, the color is grey and suggests neutrality. These changes indicate progressive maturation of the matrix after eruption.

The course of the rod matrix from the dentino-enamel junction to the surface of the tooth varies considerably with the area from which the section is taken. In sections from the axial surfaces the rods are seen to be almost parallel with one another while in the grooves and fissures the rods turn and follow diversified courses. In all instances where the sections are cut to the order of 3u and not more than 4μ, an almost constant relation between rod and inter-rod matrix is apparent. The rod matrix when cut precisely at right angles to its length, is always approximately hexagonal in shape and it is only when the rods are cut obliquely that there is any deviation from these appearances.

The various elements of the matrix show varying degrees of resistance to acid decalcifying agents. In incompletely fixed material or when the exposure to the acid has been prolonged, solution of the inter-rod substance isolates the rods, or loss of both inter-rod substance and the rod core leaves behind the cortical portion of the rod alone as a ghost of the true structure.

The Enamel Lamellae: The enamel lamellae[9, 30, 40] are generally considered to be tracts of organic material extending from the dentino-enamel junction to the surface of the tooth. Their origin and significance has given rise to considerable controversy.

On observing the progress of fixation under the microscope, a

more rapid penetration of the fixative and dye is found to occur along definite channels far in advance of that in other parts. In preparations in which the exposure of the fixative is brief, these tracts alone are preserved against the macerating action of the decalcifying fluid, the rest of the enamel matrix going into solution. In other specimens, fixed for longer periods of time, penetration may be sufficient to retain not only these tracts but in addition varying amounts of the surface matrix. (Fig. 1). Where penetration and fixation are complete, a full matrix is observed through its entire extent. (Fig. 2). Preparations of the latter varieties are the most useful in determining their nature and relations.

By these means two types of tracts are exposed. The first consists of organic material running from the dentino-enamel junction in a relatively straight course to the surface of the tooth which approximately parallels the direction of the rods. This type is that seen in ground sections and they are assumed to be the classical enamel lamellae of the literature.

The second type is found in the carious tooth but in areas which otherwise appear to be normal enamel. These tracts pursue a variable and irregular course and in those preparations where fixation is incomplete they are readily mistaken for the classical lamellae, their irregularity being ascribed to distortion due to loss of support following the solution of the surrounding matrix. As these tracts are essentially pathological they will be discussed more fully under the appropriate heading.

THE PATHOLOGY OF CARIES OF THE ENAMEL

The Plaque: With proper fixation and careful handling it is possible to retain the bacterial plaque *in situ* overlying the area of superficial carious enamel. The plaque varies in thickness and when special bacteriological stains are used (such as MacCallum's Gram, Gram-Weigert, or Murray's modification of Van Gieson), a vast variety of Gram positive and Gram negative organisms of diverse morphology can be demonstrated occupying the detritus. Threadlike forms appear to predominate and together with myriads of other organisms weave a complex pattern. The arrangement of the morpho-

logical types is however relatively constant. The thread forms appear to preponderate in the superficial layers, *Fig. 3*, but spheroidal organisms are found in greater abundance in immediate juxtaposition to the deeper zone of active enamel erosion.

The background of the plaque is amorphous and strongly eosinophilic in reaction. In the earliest stages of the pathologic process, there is little more than surface erosion of the enamel matrix. In later stages as penetration increases, the enamel matrix is highly irregular and the extremities of eroded rods project into the plaque. At this time small islands of enamel matrix in varying stages of dissolution may be found in the deeper layers of the plaque. The majority of these islands are more strongly eosinophilic than the rest of the matrix and as they break up they contribute to the general amorphous detritus. With greater penetration and a fully developed carious cavity these islands become a conspicuous feature and their appearance will be described later.

Superficial Caries: The matrix immediately underlying the surface plaque early exhibits an increasingly intense acidophilic reaction to stains when compared with the deeper placed and normal matrix. *This acidophilic alteration of the matrix is the earliest pathological change observed and may extend into the substance of the enamel for a considerable distance ahead of its solution and bacterial disintegration.* Following closely and concomitant with this change there is a progressive loss of structural detail in enamel elements and when this alteration is intense and advanced in development, the matrix becomes almost homogeneous. The area affected eventually undergoes solution, *Fig. 4*. The process may on the one hand be fairly extensive and massive or on the other, be confined to limited areas or to individual rods. In the earlier stages these areas are devoid of organisms. The process is apparently one of proteolysis presumably produced by extracellular enzymes.

Continued proteolysis results in the exposure of individual rods which have not as yet undergone so complete a change. These rods or groups of rods therefore project beyond the area of liquefaction, *Fig. 5*, rendering the invaded surface increasingly irregular. In this manner the rod core and inter-rod intervals are opened up estab-

lishing portals of entry for further bacterial invasion. Organisms by this time have penetrated the lysed surface matrix and are found advancing deep into the matrix well beyond the general area of decomposition and occupying the core in the interior of individual rods or lying within the inter-rod matrix. In such areas of penetration the organisms are progressively fewer in number. *The heterogeneous admixture of morphological types seen in the surface cavity, is no longer found and without exception in these deeper areas of penetration, the organisms are uniformly spheroidal in shape and Gram positive in reaction.* In longitudinal sections, *Fig. 6*, these spheroidal organisms are found within the core of isolated or single rods extending for some distance into the healthy matrix and the proteolytic reaction is limited to the infected rod. In some areas where penetration has occurred via the inter-rod matrix, the proteolytic changes are confined to the several adjacent rods. This mode of penetration is however usually less extensive than that by way of the rod core. The regions of active advance present a striking histological picture in that with routine stains the infected rods stand out owing to their intense acidophilic reaction in contrast to the relatively neutral reaction of the surrounding healthy matrix.

Cross sections through zones of advance such as immediately beneath the carious cavity, *Fig. 7*, are highly instructive. Enamel rods in all stages of invasion and dissolution may be observed. In some, the rod is entirely filled with organisms; in others the core alone, the rod cortex remaining in part or entirely intact. When the core is invaded the proteolytic changes are most intense in this region, the cortex staining most lightly and appearing to be resistant to the proteolytic process. In other regions invasion of the interrod substance can be followed isolating rods or groups of rods which remain relatively well preserved. With invasion of the inter-rod matrix it is observed that the direction of proteolysis differs, the staining reaction diminishing in intensity from the periphery toward the center of the adjacent rod. Areas of more complete liquefaction disclose that the cortex of the rod is most resistant to the process, the inter-rod matrix less so and the rod core, the least. Eventually the rods are completely liquefied, little more than ghosts of their former

selves remaining, or groups of rods, in varying degrees of proteolysis, are isolated to contribute to the amorphous detritus of the plaque or to form small islands of matrix substance within it.

The enamel matrix lining the superficial cavity in areas other than those of active penetration, appear to be remarkably resistant to any further degree of solution. The matrix may show little or no change in staining reaction and appear entirely healthy. Expansion of the cavity in this region is purely a surface phenomenon. It has been regularly observed that such portions of the cavity lie always in relationship to areas where the threadlike and mixed organisms predominate. In these regions deep penetration is entirely absent.

The Pathway of Deep Penetration (the pseudo-lamellae): As described above extension of the surface caries occurs following the opening up of the rod core and inter-rod intervals to organismal invasion by Gram-positive spheroidal shaped organisms. The margin of advance in superficial caries extends but for a little distance beyond the floor of the cavity. A similar process extending through the full thickness of the enamel is found in some fields leading to the establishment of a focus at the dentino-enamel junction, *Fig. 8. These pathways of deep penetration are narrow, irregular tracts which on superficial examination of routine stained preparations closely resemble the classically described enamel lamellae and for reasons already mentioned, may readily be mistaken for them in preparations in which the preservation of the enamel matrix is incomplete or imperfect.* Under the higher powers of the microscope and with bacteriological stains these tracts are found to consist of one or two isolated rods in thickness whose cores are packed with organisms. The infected rod matrix undergoes moderate proteolytic changes which produce the distinctive differential staining reaction seen in the routine preparations. In some instances, infection is confined to a single rod or groups of rods in almost their entire extent thus producing a pseudo-lamella which is comparatively straight and linear. In others the invasion proceeds down a single rod for some distance, spreading laterally to the inter-rod interval and implicating neighboring rods for some little distance, thence down the core of one of these partially infected rods to deeper levels and substantially

in the same maner, to the dentino-enamel junction (Fig. 9). This mode of spread is responsible for the great irregularity and typical wavy course which many of these tracts pursue. From an examination of serial sections it has been observed that usually only one and occasionally two or more of these local pathways of extension are present.

In the earlier stages of their formation, the organisms occupying these tracts are invariably of similar morphological type; Gram-positive, spheroidal forms. As the tract widens from extension of the process to neighboring rods and with increasing proteolytic changes, the spheroidal organisms are followed and almost wholly replaced by numerous and various Gram-positive and -negative thread-like and bacillary forms. The initial tract on the other hand may not widen to any appreciable extent or it may expand in some local portion of its extent establishing minute foci in the depths of the enamel. These intermediate foci tend to remain of limited extent in contrast to the foci eventually produced at the dentino-enamel junction.

THE DENTINO-ENAMEL FOCUS AND RETROGRADE SPREAD

The pseudo-lamellae having reached the dentino-enamel junction, establish there a secondary focus of the carious process. Proteolytic changes extend laterally along the junction, *Fig. 8*, opening up the dentinal extremities of the rods and inter-rod intervals. In the earlier stages these foci are small and the organisms found are exclusively Gram-positive and spheroidal in shape. As the initial tract expands in size, secondary organisms of diverse morphology make their appearance and cavitation with lysis of the matrix elements now becomes extensive. Lateral spread of the focus seems to be confined at first to the enamel matrix and only in advanced disintegration does the dentin become involved.

As continued breakdown and liquefaction of the enamel elements progresses, islands of healthy matrix, the centers of which are as yet unaffected by the proteolytic change, are isolated. At this stage widening of the initial tract and the presence of secondary organisms other than the spheroidal type is the rule.

From such secondary dentino-enamel foci, retrograde spread into the overlying enamel matrix proceeds. The frontal areas of attack present the same picture as that observed in superficial caries. Retrograde invasion of the rod core, opening up the inter-rod intervals, and proteolytic changes, are typical. Once again the predominating organism in the zone of advance is the spheroidal form. Although secondary contaminants are observed in the body of the cavity, these soon replace the spheroidal type, giving rise to an irregular filiform appearance of the advancing margins of the cavity.

Extensive Caries: A different appearance of pathological process is associated with the production of extensive caries of the matrix. In this type the development of multiple irregular channels of dissemination is characteristic. The process has its inception as a surface lesion, the pathological features of which nowise differ from those described under superficial caries. From this focus, numerous intricate and lateral branching pseudo-lamellae extend through the substance of the healthy matrix. These channels present all the features present in the isolated pathways discussed in the spread to the dentino-enamel junction, except that instead of following a single rod or a group of rods, for any distance into the matrix, infection of the inter-rod substance preponderates and lateral extension, for considerable distances, becomes the characteristic feature. The multiple tracts often present an extremely complex pattern and as progressive proteolytic changes occur wide areas of the matrix become involved. The channels contain Gram-positive, spheroid organisms at their advancing extensions, *Fig. 10*, but show a tendency to early widening followed by the early invasion of other heterogeneous bacteria.

Islands of healthy matrix become isolated by the fusion of these spreading and anastomosing channels until the whole matrix is broken up. The periphery of these islands display early proteolytic changes while their centers long remain unaffected, *Fig. 11*. The channels extend in width at the expense of the isolated material until eventually a massive cavity is established which is occupied by numerous completely separated portions of the matrix, presenting all types of dissolution. When the breakdown is nearing completion a

variety of heterogeneous organisms are present in great profusion.

At the deeper levels, mixed types predominate in the channels and the spheroid forms are found in the periphery of the islands. The newly invaded areas at the periphery of the lesion show relatively few organisms, all of the spheroid type, and an acidophilic reaction of the matrix along the pathways of invasion. When advanced this process leads to rapid and extensive cavitation.

DISCUSSION

An essential preliminary to the consideration of caries as a pathological process necessitates a brief examination of the histological structure of the organic content of the enamel in which this process occurs. In previous communications[39, 45] we have attempted to define these elements from investigations on their development and concluded that enamel is established by the transformation of the ameloblast into an enamel matrix, which undergoing further histo-chemical differentiation and maturation becomes calcified by a secondary humeral process. There has been considerable difference of opinion as to whether this organic matrix persists. Many authors hold the view that in the adult tooth there is an eventual substitution[46, 47] or replacement of the organic elements by the inorganic complex and would regard the organic content as constituting but an insignificant if any part of the mature enamel. Such an opinion is enhanced by the daily observation of the almost invariable absence of any enamel structure in routine decalcified histologic sections of the tooth. The replacement or substitution theory must necessarily postulate as a corollary the existence of an active circulation within the enamel for the removal of these elements. On the other hand, several observers, Bodecker,[9, 10, 13, 15, 18] Malleson[35, 36] etc.[1, 2, 3, 4, 5, 6, 7, 18, 22, 23, 26, 28, 31, 38] have succeeded in recovering various amounts of the enamel matrix from the fully developed human tooth but their findings have been generally interpreted as demonstrating that no more than remnants of a matrix exist.

The problem has been approached chemically and although ash analysis demonstrates the presence of organic constituents, the difficulties inherent in the method and lack of knowledge as to the precise

chemical nature of the matrix, tend to create the impression that the content is not of much significance. Our observations show that the organic matrix persists throughout the full thickness of the enamel and remains throughout life. The matrix exhibits the same essential structure as that observed during its development. Its histological demonstration in the adult tooth is dependent upon full denaturation of its substance and selective decalcification if its total destruction by the acids necessarily employed, is to be avoided.

We have previously demonstrated[39, 45] that during development, the enamel matrix is composed of hyalinized rods and inter-rod substance. The rod was shown to be composed of three parts, the cortex, the core and the rod membrane. The cortex constitutes the lateral wall of the rod, is homogeneous, dense and the first part of the rod to undergo complete maturation and calcification. The core is the less dense center of the rod which in certain areas may show incomplete hyalinization. This delay or lack of maturation may involve the full length of isolated rods or segments of it. Surrounding the rod elements and therefore external to its cortex is a delicate rod membrane which is regarded as a remnant of the initial ameloblastic membrane. The inter-rod matrix or inter-prismatic substance is composed of similar material to that of the rod but it is less dense, and occupies the intervals between the rods and its tinctorial responses are less pronounced than that of the rod. It is the last portion of the enamel to accept the calcium complex.

The adult matrix possesses these same components except that the various elements do not stain with the same precision as during development and particularly with advancing age. The rod membrane can no longer be identified with any clarity for it blends so intimately with the cortex as to lose its identity. "The Rod Sheath" of Bodecker[9, 10, 11, 12, 15] and others[6, 27, 31, 35] would seem to be identical with the cortex as described by us[39].

The markings of the enamel, dependent upon the pattern of calcification, such as incremental and striation lines, have never been observed when the calcium salts have been totally removed from the matrix as indicated by Von Kossa's stain. The description of these

features by Malleson[35, 36] and Berke[2, 3] leads us to believe that their material was insufficiently decalcified.

It has been observed that during decalcification the inter-rod substance is the first to give up its organic constituents, to be followed a little later by the core and finally the cortex, a process which is in inverse relationship to that of its calcification in the developmental period.

The enamel matrix would seem to undergo progressive maturation with increasing age, a process which is but a continuation of that observed in the developmental period, until eventually its various elements can be distinguished only with difficulty and its substance becomes relatively chromophobe. We suggest that this continuing maturation is one of progressive dehydration or desiccation and that it is possibly related to the increasing resistance of the enamel to the carious process with increasing age. The greater degree of desiccation of the cortex quite probably explains the resistance of this element to proteolysis. There is possibly sufficient water for the diffusion of the enzyme but an amount insufficient to permit catalytic action. The process of enamel maturation is not only related to an increase of the inorganic complex but also involves changes in the underlying matrix.

Bodecker and Bodecker[17] have deduced that the organic rod is composed of two kinds of protein, an albumin-like material within the rod and a keratin within its sheath. We believe that the evidence is at present inadequate to substantiate such a claim in histologic preparations, nor do we know of any histo-chemical method which would permit differentiation between the two proteins mentioned. However, there is little doubt that differential methods could be developed since the proteins in question are of a different classification and show different characteristics. Depending upon tinctorial reactions, which are recognized as by no means conclusive, there is some evidence that agrees in a general way with Chase[24], Rosebury and Gies[43], Rosebury[44] and Pincus[41, 42] that the matrix is protein in nature and may be closely allied to the keratins.

Caries of the enamel is a far more complicated problem than simple decalcification and removal of the inorganic salts. The underlying

organic structure is fundamentally related to the development of the pathological process; a feature which has not been sufficiently appreciated due to the technical difficulties related to its preservation for microscopic examination. The initial lesion is associated with the formation of a bacterial plaque and the breakdown of the enamel cuticle. This cuticle, the final product of the ameloblast, is relatively structureless and homogeneous and establishes a surface covering over the ends of the enamel rods and inter-rod intervals. The importance of this layer in protecting the tooth has been appreciated by many observers, Miller[37], Box[21], Deitz[25], etc., and it is possible that penetration of this layer is due to the action of acidogenic bacteria removing the more readily soluble calcium carbonates. Gore[29] has produced evidence to indicate that while some question exists as to the exact solubility product constant for the tri-basic calcium phosphate and although its solubility increases with increased hydrogen-ion concentration, the accumulation of calcium and phosphate-ions, presumably by buffer action, retards and finally stops its action. This is an important consideration and probably explains why experimental lesions *in vitro* are always surface phenomena without deep penetration. (Compare the recent experiments of Deitz[25].) Acid action may therefore account for penetration and dissociation of the surface cuticle but that it establishes more than the surface lesion is not borne out by our observations on the pathological progress of the disease.

On the other hand it may well be that the protection offered by the fluorides[34] is related to the substitution of the more readily diffusible calcium carbonate of the surface layer by the harder and relatively insoluble calcium fluorides together with alterations in permeability produced thereby (compare Lukomski[34], Bibby[8], Gore[29].) The high susceptibility in rampant caries is possibly related to faulty development and calcification of the surface cuticle and there are numerous clinical and experimental observations which emphasize the significance of this layer.

Once integrity of the surface layer has been destroyed, exposing the rods and inter-rod intervals, the further penetration of the disease occurs by invasion of these elements by Gram-positive bacteria

of uniform spheroidal shape. We have advisedly avoided the use of the term "coccus," because of the impossibility of establishing their identity by morphology alone, and it may be unwarrantably assumed that strepto- or staphylo-cocci are inferred. The penetration occurs in the region of the irregular advancing edge of the lesion and proceeds for considerable distances into the enamel substance where secondary foci may arise. The various types of spread have been described and the danger of confusing these lines of penetration with the classical lamellae has been indicated. It has been mentioned that penetration occurs generally via the core of a single rod or by cross contamination of the inter-rod substance to collateral rods, pursuing often, in this wise an irregular course. In previous observations on enamel development[38] it was shown that the core of either entire rods or segments of rods may remain immature. *It is suggested that rods in which segments remain immature are more susceptible to invasion, immaturity accounting for the irregular penetration and establishment of the diffuse type.*

Several investigators, Baumgartner[1], Fleischmann[28], Malleson[35, 36], Bodecker[15, 16], Bodecker and Bodecker[17], Bibby[6] and Berke[2, 3], have associated penetration of bacteria with caries of enamel and have also recognized that the disease is not entirely one of decalcification but a two-phase process in which proteolysis is a significant factor. These investigators fall into two general groups. The first are those[2, 3, 6, 15, 16, 17, 35, 36] who consider that the decalcification by acidogenic bacteria allows the subsequent ingress of organisms which destroy the remaining matrix by proteolytic action. On the other hand Baumgartner[1] and Fleischmann[28] are of the opinion that having formed portals of entry, the bacteria produce the acid *in situ* resulting in decalcification.

Our observations indicate that once the lesion is established the essential and primary pathologic lesion is proteolysis of the matrix. In view of the basic reaction of the invaded protein matrix, it is difficult to conceive that this is due to the action of acid products alone but rather to the characteristic action of bacterial enzymes. Extension of the proteolytic process and liquefaction of the matrix is envisaged as freeing the less soluble basic tricalcium phosphate from its organic

bond and thus favoring its solution by the products of acidogenic bacteria which secondarily penetrate along the widening pathways of ingress.

Several authors[6, 7, 15, 36] have described a reaction of the matrix which they have termed condensation and which is considered by them as being due to either a reactive defensive mechanism of the tooth or to the accumulation of debris. A similar appearance has been observed by us associated with the development of the initial carious lesion. This process when viewed in relationship to the full width of the enamel matrix can be shown to be but an early stage in proteolysis in which the loss of matrix structure allows the altered matrix to accept the stain more strongly.

Apart from the description of the pathological process we believe that our experimental data demonstrate for the first time the precise mechanism of caries of the enamel. We advance the hypothesis that this process is primarily a proteolysis of the organic matrix resulting from the enzymic action of micro-organisms followed by the subsequent dissociation of the inorganic constituents. We believe that questions of immaturity of the matrix are important in providing pathways of further dissemination of the disease, and that full development and maturation of the matrix together with the formation of the enamel cuticle[39] and its calcification undoubtedly play a significant role in rendering the individual caries immune. Our experimental work further supports the hypothesis that defective formation of organic structure due to developmental, dietetic or other causes, in more ways than one, favors the establishment of the disease.

SUMMARY

By the development of a more precise technic which insures a complete fixation of the enamel, and its subsequent decalcification, the authors have been able to demonstrate the existence of a complete matrix in the adult human tooth, to show that the structure of the adult matrix retains the essential morphology of that laid down during development, and to illustrate the relationship which this

matrix bears to the histopathology of caries and the effect upon it of bacterial invasion. The following observations were made:

The Normal Enamel Matrix

1. An organic matrix is distributed throughout the entire structure of the adult enamel of both man and other animals and may be recovered from teeth of any age group if the proper procedures are employed.

2. In the adult tooth the early structural characteristics of the matrix can be identified and they are identical with those of the developing stages. The staining reactions of the various elements are similar but less precise than those of the developing structure. The histologic demonstration of the matrix substance is dependent on full denaturation and selective decalcification if its total destruction by acids necessarily employed is to be avoided.

3. As has been previously described each rod presents a cortex, core and sheath. The rods are connected by the inter-rod matrix which possesses staining reactions similar though less intense than those of the rod itself.

4. The course of the enamel rod matrix from the dentino-enamel junction to the surface of the tooth varies considerably with the area from which the section is taken. In sections from axial surfaces, the rods are seen to be almost parallel with one another while in areas of grooves and fissures the rods will turn and follow diversified courses.

5. In precise cross sections, the rod matrix appears to be roughly hexagonal in shape and there is a constant relationship between inter-rod and the rod matrix.

6. The markings of the enamel dependent upon the pattern of calcification, such as incremental and striation lines, have never been observed when the calcium salts are totally removed and the description of these features by other authors leads us to believe that their material was insufficiently decalcified.

7. The enamel matrix appears to undergo progressive maturation with increasing age. This process, we believe, is but a continuation of that observed in the developmental period.

8. We suggest that this continuing maturation is one of progressive dehydration or desiccation and that it may be related to the increasing resistance of the enamel to the carious process with age.

9. Based on tinctorial reactions which are recognized as by no means conclusive, there is some evidence that agrees in a general way with other investigators that the matrix is protein in nature and may be closely related to the keratins.

10. We believe that the immaturity of the matrix is important in providing pathways for dissemination of the pathologic process of caries and that full development and maturation of this matrix together with the formation of the enamel cuticle and its calcification, play a significant role in rendering the individual caries immune.

11. During the process of fixation a more rapid penetration of the fixative occurs along definite channels far in advance of that in other parts of the structure. Two distinct types of penetration are observed. The first tract consists of organic material running from the dentino-enamel junction in a relatively straight course to the surface of the tooth. The second type is more clearly demonstrated in the carious tooth and pursues a variable and irregular course through the enamel.

The Pathology of Caries of the Enamel

The Plaque: 12. With proper fixation and careful handling it is possible to retain a bacterial plaque *in situ* overlying the area of superficial carious enamel. The arrangement of the morphological types of organisms in the plaque are observed to be relatively constant. The thread forms appear to predominate in the superficial layers. The spheroid forms are found in greater abundance in juxtaposition to the deeper zone of active enamel erosion.

13. The background of the plaque is amorphous and eosinophilic in reaction and small islands of enamel matrix are found in the deeper layers which continue to break up and by liquefaction contribute to its appearance.

Superficial Caries: 14. The initial carious lesion on the surface of the enamel is probably associated with the formation of the bacterial plaque and the breakdown of the enamel cuticle; thereby uncovering the ends of the enamel rods and inter-rod matrix.

15. The penetration of this calcified homogeneous cuticle may be due to acidogenic bacteria removing the more readily soluble inorganic constituents from this layer. Acid action may therefore account for the opening up of the rods and inter-rod matrix for the invasion by spheroidal Gram-positive micro-organisms.

16. Once the primary cuticle is destroyed our observations indicate that the essential and primary pathologic lesion is one of proteolysis of the matrix. We envisage the extension of this proteolytic process and liquefaction of the matrix as freeing the less soluble basic tricalcium phosphate from its organic bond, thus favoring its solution by products of acidogenic bacteria which secondarily penetrate along widening pathways of ingress.

17. The earliest pathologic change observed in the matrix is an increasingly intense acidophilic reaction to stains immediately underlying the surface plaque.

18. Closely following the acidophilic reaction in the matrix there is a progressive loss of structural detail in the enamel elements and when advanced in development the matrix becomes almost homogenous, the affected area eventually undergoing solution. In the early stages these areas are devoid of organisms. This process is one of proteolysis and is probably produced by extra cellular enzymes.

19. Continued proteolysis results in the exposure of individual rods or groups of rods which have not as yet undergone so complete a change and project beyond the area of liquefaction. In this manner the rod core and inter-rod matrix are opened up, thus establishing portals of entry for invasion by other organisms.

20. Gram-positive organisms spheroidal in form having penetrated the lysed surface matrix are found advancing deep into the matrix well beyond the general area of decomposition.

21. Depending on the mode of ingress the organisms are found either within the core of the rod or the inter-rod matrix and changes in the staining reaction may be observed in the immediately surrounding matrix.

22. Superficial carious areas other than those of active penetration appear to be remarkably resistant to further solution, expansion of the cavity being purely a surface phenomenon. It has been regu-

larly observed that such portions of the cavity lie always in relationship to areas where the threadlike and mixed organisms predominate.

23. Following the opening up of the rod core and the inter-rod matrix there is an invasion of Gram-positive spheroid shaped organisms. The process is observed to extend through the full thickness of the enamel leading to the establishment of foci at the dentino-enamel junction. The pathways of penetration are narrow, irregular tracts and may resemble the classically described lamellae.

24. Under the higher powers of the microscope and with the use of bacteriological stains these tracts are found to consist of one or several isolated rods whose cores are packed with organisms.

25. The organisms found occupying these tracts are invariably of similar morphological type, Gram-positive spheroidal forms.

26. Invasion along the so-called lamellar tracts having reached the dentino-enamel junction, establishes a secondary focus of the carious process with proteolytic changes extending laterally along the junction and opening up the dentinal extremities of the rod and inter-rod matrix.

27. In the earlier stages the foci are small and the organisms found are exclusively Gram-positive and spheroidal in form. With expansion of the tract secondary organisms of diverse morphology make their appearance with resulting cavitation and lysis of the matrix elements.

28. From such dentino-enamel foci retrograde spread into the overlying enamel matrix proceeds. The frontal areas of attack present the same picture as that observed in superficial caries.

Extensive Caries: 29. A different penetration of the pathologic process is the production of extensive caries of the matrix through the development of multiple irregular channels of dissemination. This process has its inception as a surface lesion, the pathologic features of which in nowise differ from those described under superficial caries.

30. Islands of healthy matrix become isolated by fusion of the spreading channels and the entire matrix is broken up.

31. When the breakdown nears completion the process leads to rapid and extensive cavitation. The newly invaded periphery of

these areas shows relatively few organisms, all of the spheroidal type, and the margins of the lesion are acidophilic in reaction. Secondary invaders present a variety of organisms of heterogenous morphology and in great profusion.

32. We have been able to demonstrate by histo-pathologic procedures that caries is more than a simple decalcification and the removal of the inorganic salts. The underlying organic structure is fundamentally related to the pathological process.

33. We offer as a working hypothesis that with the establishment of the lesion, caries of the enamel is primarily a proteolysis of the organic matrix resulting from the enzymatic action of micro-organisms followed by the subsequent dissociation of the inorganic constituents.

34. We believe that our laboratory data demonstrates for the first time the mechanism involved in the pathology of caries of the enamel and places the problem on a sound experimental basis.

We should like to express our thanks and indebtedness to Miss Barbara Killian for technical supervision, to Dr. John Eiler for his help in the biochemical considerations, to Dr. Max Marshall and Miss Janet Gunnison for their part in the interpretation of the bacteriology of the problem and to Mrs. Helen M. Capon for secretarial assistance.

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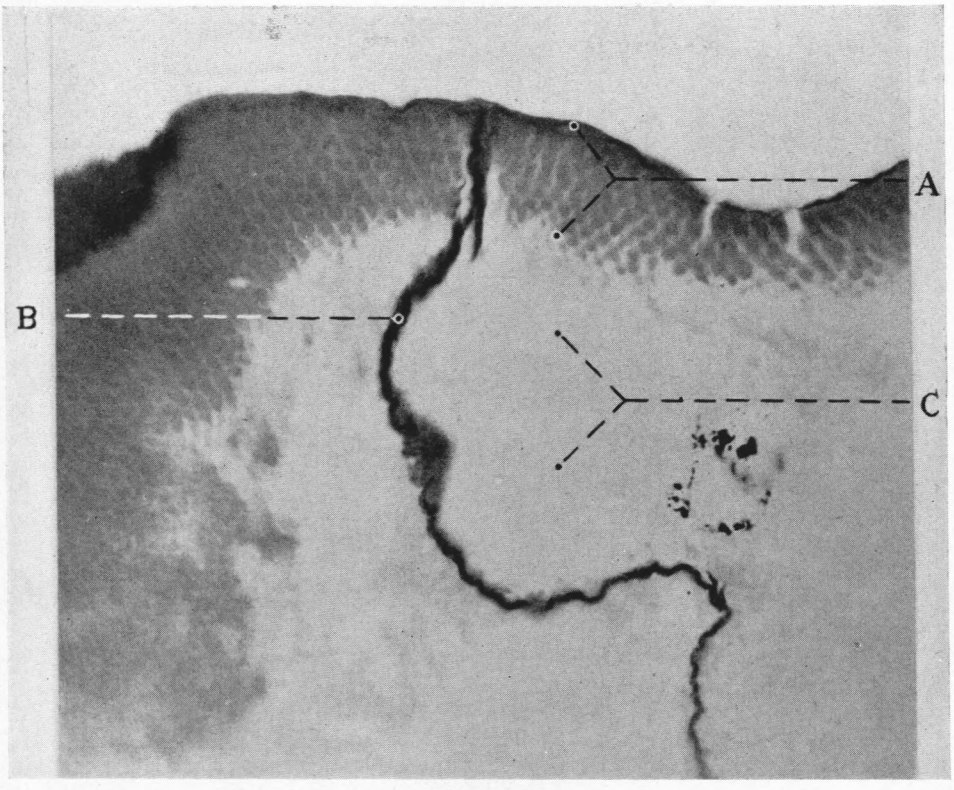

FIG. 1. ORGANIC ENAMEL MATRIX FROM THE ADULT HUMAN TOOTH 6μ NEUTRAL FORMOL FIXATION. HEMATOXYLIN AND EOSIN × 1000. ILLUSTRATING INCOMPLETE FIXATION RESULTING IN THE RECOVERY OF PERIPHERAL UNITS AND "LAMELLA."

A. AREA OF SURFACE FIXATION WITH ROD MATRIX IN TRANSVERSE SECTIONS.

B. ORGANIC TRACT WHERE THE PENETRATION OF THE FIXATIVE IS RAPID OR "PSEUDO-LAMELLA."

C. AREA SHOWING LOSS OF MATRIX RESULTING FROM INCOMPLETE FIXATION AND ACID ACTION.

NOTE THE CONSTANT RELATIONSHIP BETWEEN THE INTER-ROD AND ROD MATRIX IN THE AREA OF SURFACE FIXATION.

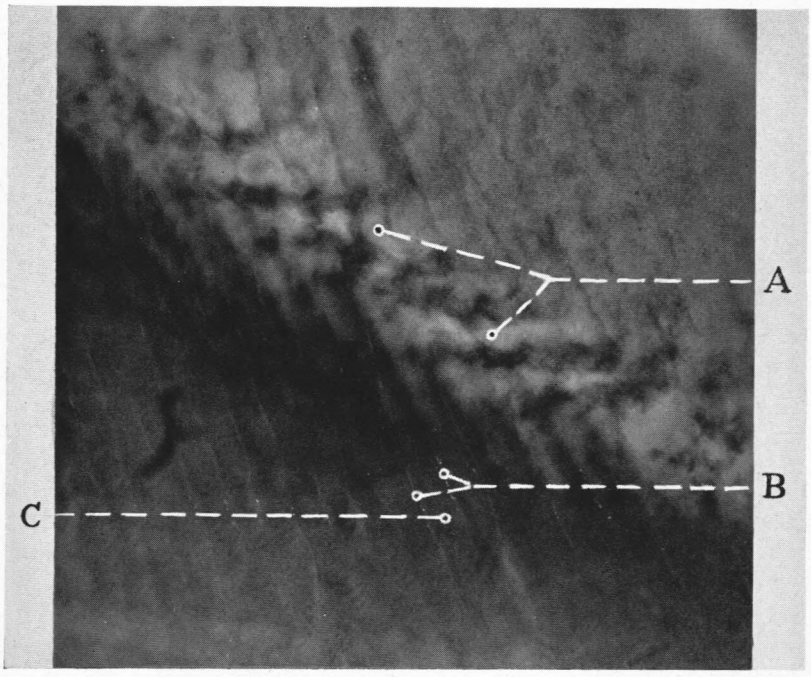

FIG. 2. ORGANIC ENAMEL MATRIX FROM THE ADULT HUMAN TOOTH 6μ NEUTRAL FORMOL FIXATION. HEMATOXYLIN AND EOSIN $\times$ 1000. ILLUSTRATING NORMAL ROD AND INTER-ROD MATRIX.

A. AREA OF PARTIAL RECOVERY OF MATRIX.

B. INTER-ROD MATRIX.

C. ROD MATRIX.

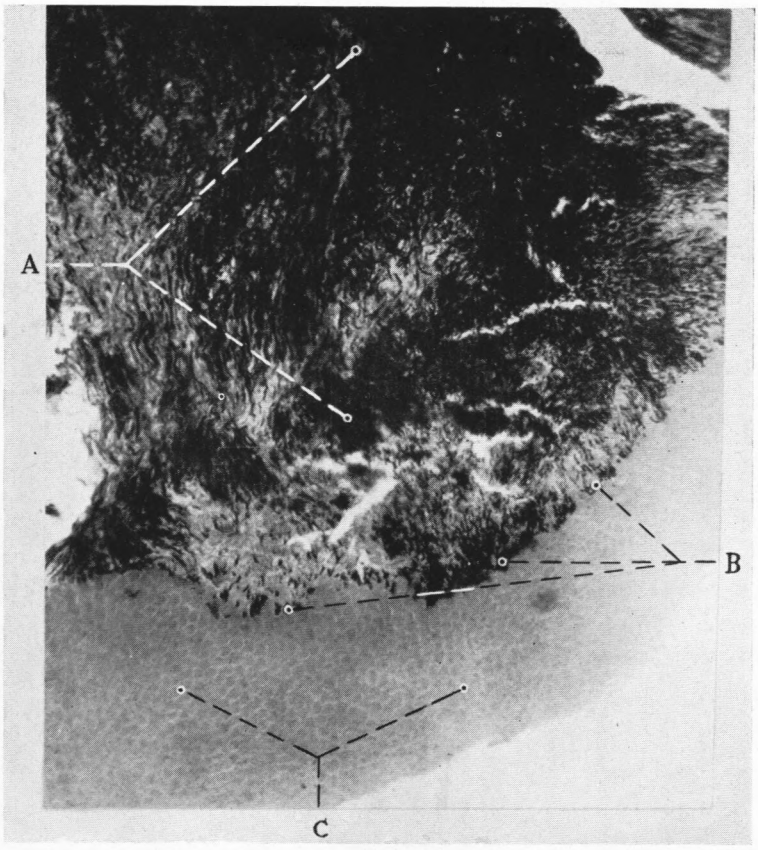

FIG. 3. ORGANIC ENAMEL MATRIX FROM THE ADULT HUMAN TOOTH 4μ NEUTRAL FORMOL FIXATION. MACCALLUM'S GRAM $\times$ 500. ILLUSTRATING SURFACE PROTEOLYSIS AND MICRO-ORGANISMS IN CLOSE PROXIMITY TO THE ENAMEL MATRIX.

A. SURFACE PLAQUE.

B. MICRO-ORGANISMS IN PROXIMITY TO THE ENAMEL MATRIX.

C. ENAMEL RODS IN CROSS SECTIONS.

NOTE THE CONSTANT RELATIONSHIP OF THE INTER-ROD MATRIX TO THE ROD MATRIX IN THE NORMAL STRUCTURE.

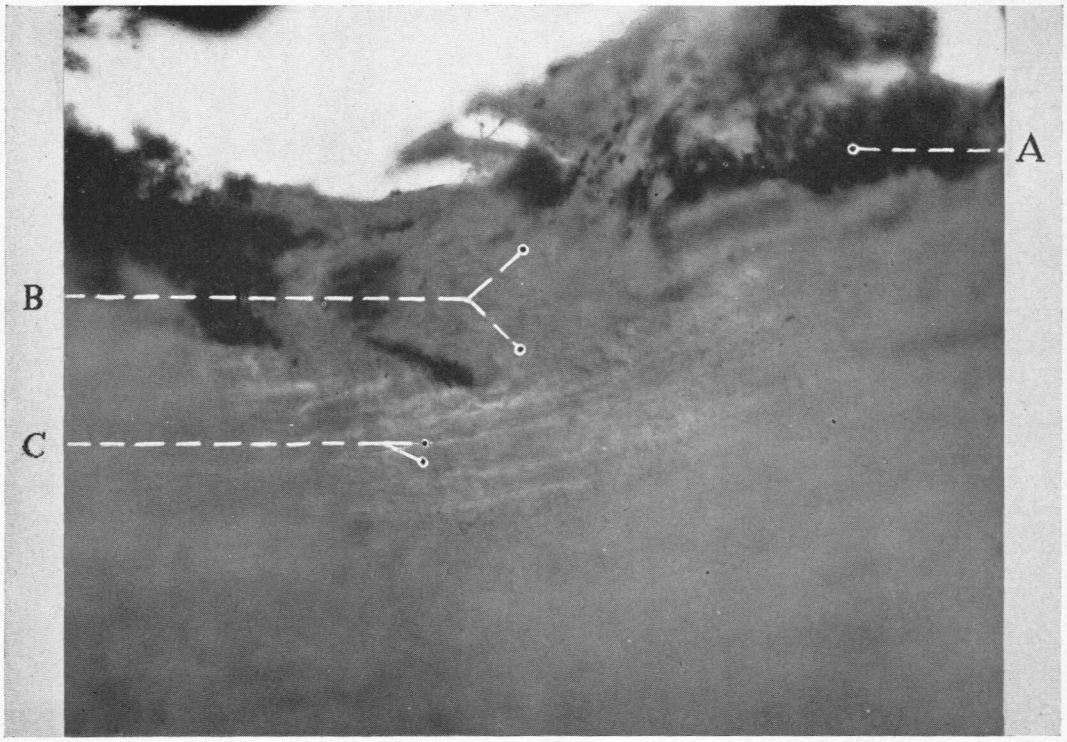

FIG. 4. ORGANIC ENAMEL MATRIX FROM THE ADULT HUMAN TOOTH 4μ NEUTRAL FORMOL FIXATION. MACCALLUM'S GRAM × 1000. ILLUSTRATING THE INITIAL CHANGES OF THE INVOLVED MATRIX IMMEDIATELY UNDERLYING THE PLAQUE.

A. BACTERIAL PLAQUE.

B. ACIDOPHILIC STAINING OF THE INVOLVED MATRIX.

C. UNINVOLVED ENAMEL MATRIX.

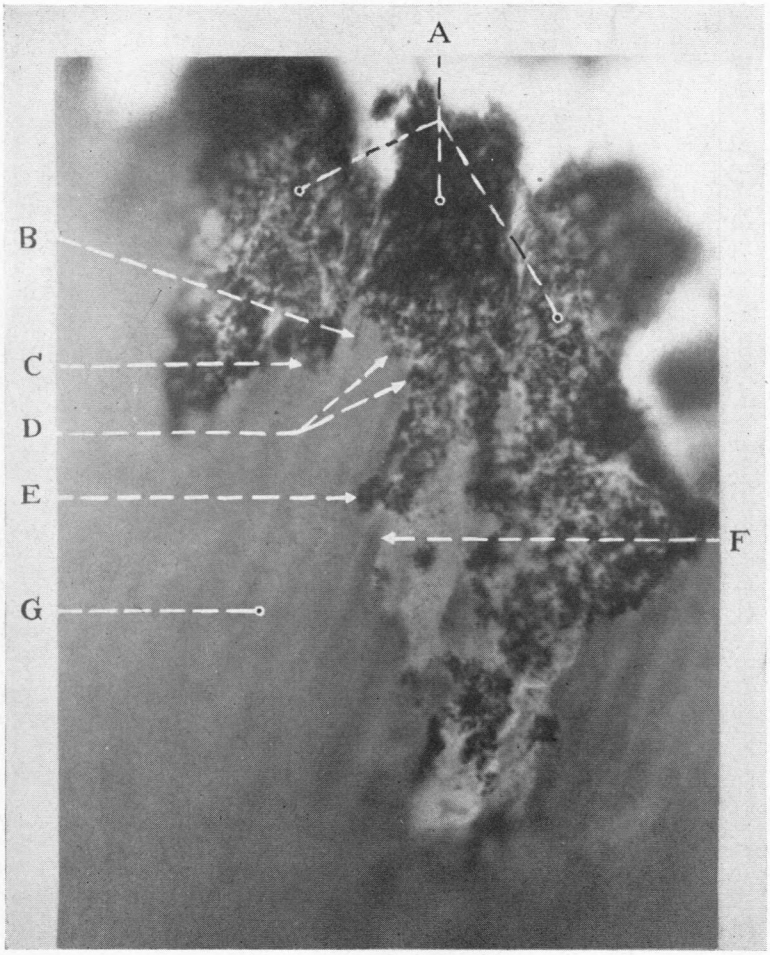

FIG. 5. LONGITUDINAL SECTION OF ORGANIC ENAMEL MATRIX FROM THE ADULT HUMAN TOOTH 4μ NEUTRAL FORMOL FIXATION. MACCALLUM'S GRAM × 1000. ILLUSTRATING SUPERFICIAL CARIES AND A CONTINUED PROTEOLYSIS RESULTING IN THE EXPOSURE OF INDIVIDUAL RODS WHICH HAVE NOT AS YET UNDERGONE SO COMPLETE A CHANGE.

A. BACTERIAL PLAQUE.
B. BACTERIA PENETRATING THE ORGANIC MATRIX VIA THE INTER-ROD MATRIX.
C. BACTERIA PENETRATING THE ORGANIC MATRIX VIA THE CORE OF THE ROD.
D. ERODED SURFACE SHOWING IRREGULARITIES OF SURFACE AND PROJECTING RODS.
E. BACTERIA PENETRATING THE ORGANIC MATRIX BY LATERAL EXTENSION INTO A ROD.
F. INTENSELY ACIDOPHILIC STAINING OF MATRIX AT THE BORDER OF THE LESION.
G. NORMAL ENAMEL MATRIX.

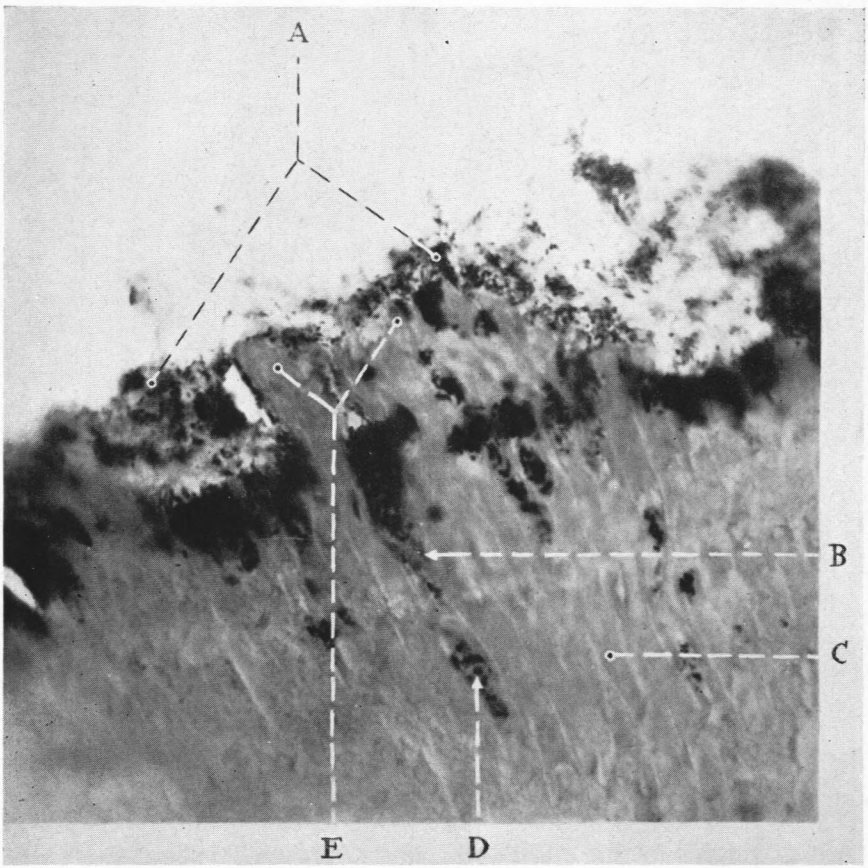

FIG. 6. ORGANIC ENAMEL MATRIX FROM THE ADULT HUMAN TOOTH 4μ NEUTRAL FORMOL FIXATION. MACCALLUM'S GRAM × 1000. ILLUSTRATING THE PENETRATION OF THE SURFACE MATRIX BY GRAM POSITIVE SPHEROIDAL ORGANISMS. THE BACTERIA ARE FOUND WITHIN THE CORE OF THE ROD AND HAVE PENETRATED FOR SOME DISTANCE BEYOND THE GENERAL AREA OF LIQUEFACTION.

A. BACTERIAL PLAQUE.

B. INTENSE ACIDOPHILIC REACTION OF INVADED MATRIX.

C. NORMAL ENAMEL MATRIX.

D. DEEPLY PENETRATING GRAM POSITIVE SPHEROIDAL ORGANISMS WITHIN THE CORE OF THE ROD.

E. LYSED SURFACE SHOWING IRREGULAR RODS PROJECTING BEYOND THE GENERAL AREA OF DECOMPOSITION.

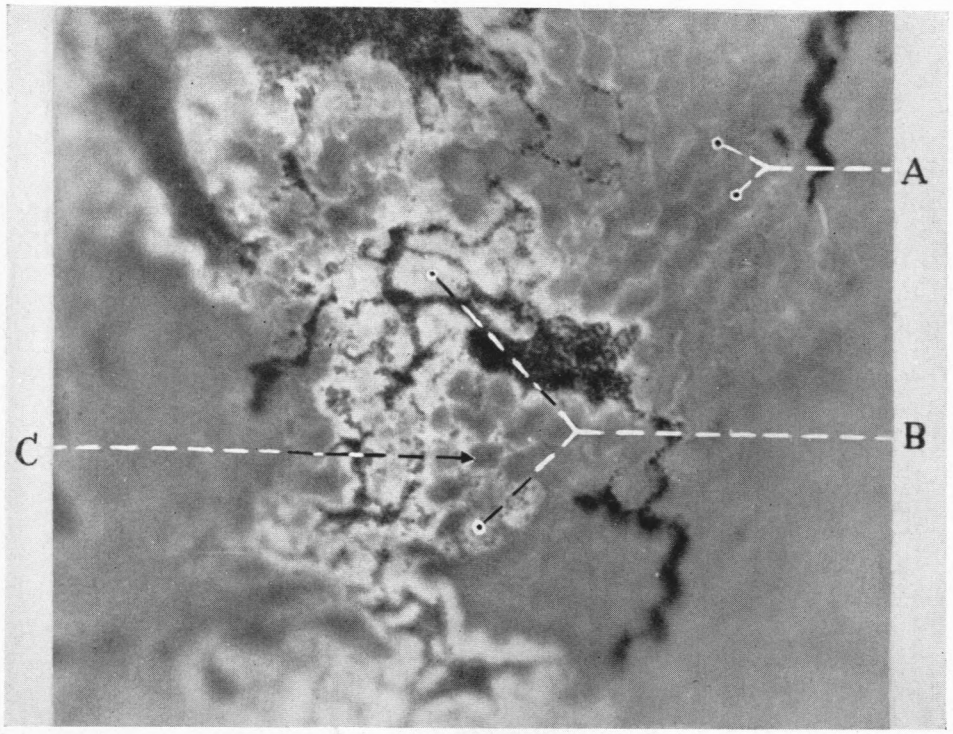

FIG. 7. ORGANIC ENAMEL MATRIX FROM THE ADULT HUMAN TOOTH 4μ NEUTRAL FORMOL FIXATION. GRAM WEIGERT $\times$ 1000. ILLUSTRATING PROTEOLYSIS OF THE MATRIX WITH THE RODS CUT IN CROSS SECTIONS.

A. NORMAL ENAMEL MATRIX.

B. AREA OF BACTERIAL INVASION SHOWING ALL DEGREES OF DECOMPOSITION.

C. ROD SHOWING AN INCREASED ACIDOPHILIC REACTION AT THE BORDER WHILE THE CORE AS YET APPEARS NORMAL.

NOTE THAT THE PENETRATION OF ORGANISMS ARE PREDOMINANTLY CONFINED TO THE INTER-ROD MATRIX. THIS TYPE OF PENETRATION IS MORE OFTEN OBSERVED IN EXTENSIVE CARIES.

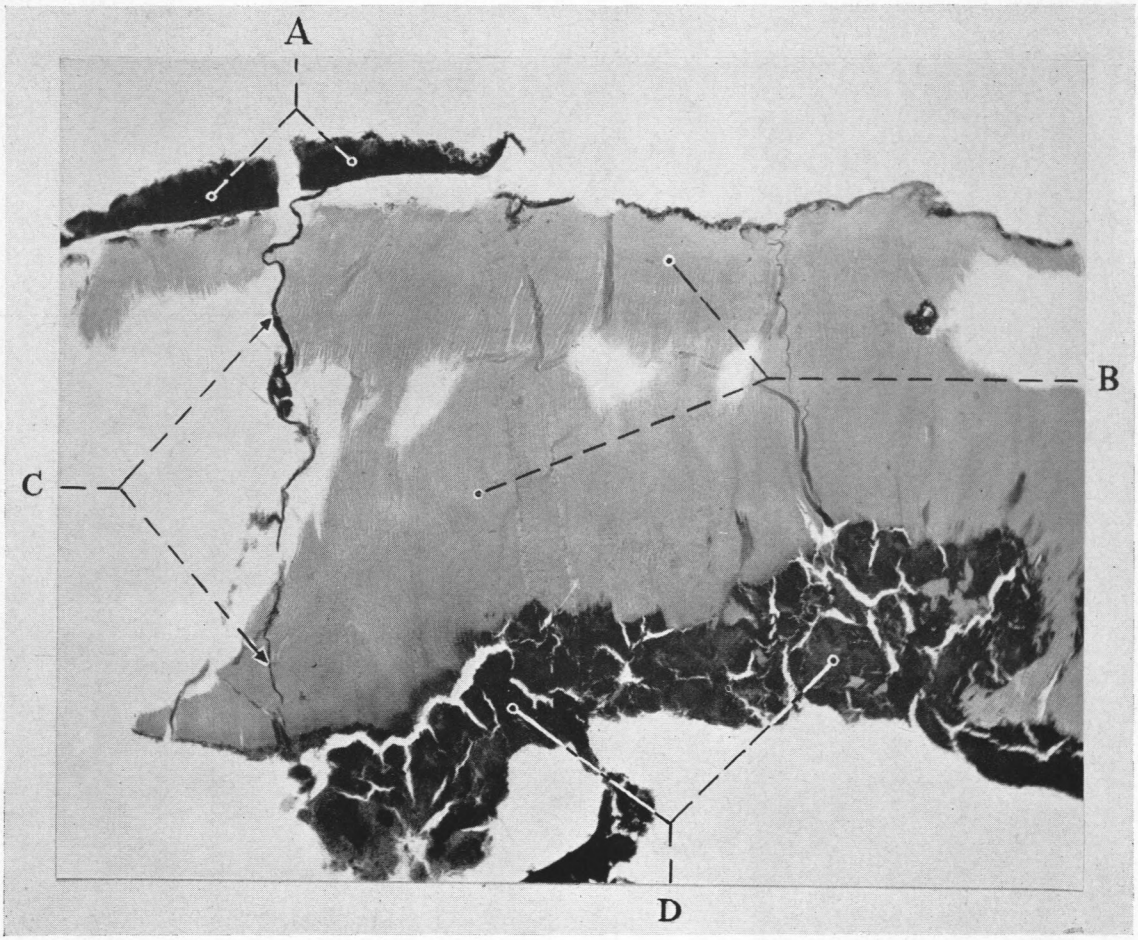

FIG. 8. ORGANIC ENAMEL MATRIX FROM THE ADULT HUMAN TOOTH 4μ NEUTRAL FORMOL FIXATION. GRAM WEIGERT × 140. ILLUSTRATING PATHWAYS OF DEEP PENETRATION FROM THE SURFACE PLAQUE VIA ORGANIC TRACTS OR "PSEUDO-LAMELLAE" TO THE DENTINO-ENAMEL JUNCTION.

A. SURFACE PLAQUE.

B. NORMAL ENAMEL MATRIX.

C. ORGANIC TRACT OF BACTERIAL INVASION.

D. LATERAL EXTENSION AND RETROGRADE SPREAD OF THE CARIOUS PROCESS AT THE DENTINO-ENAMEL JUNCTION OPENING UP THE DENTINAL EXTREMITIES OF THE RODS AND INTER-ROD MATRIX.

NOTE PARALLEL RELATIONS OF THE RODS.

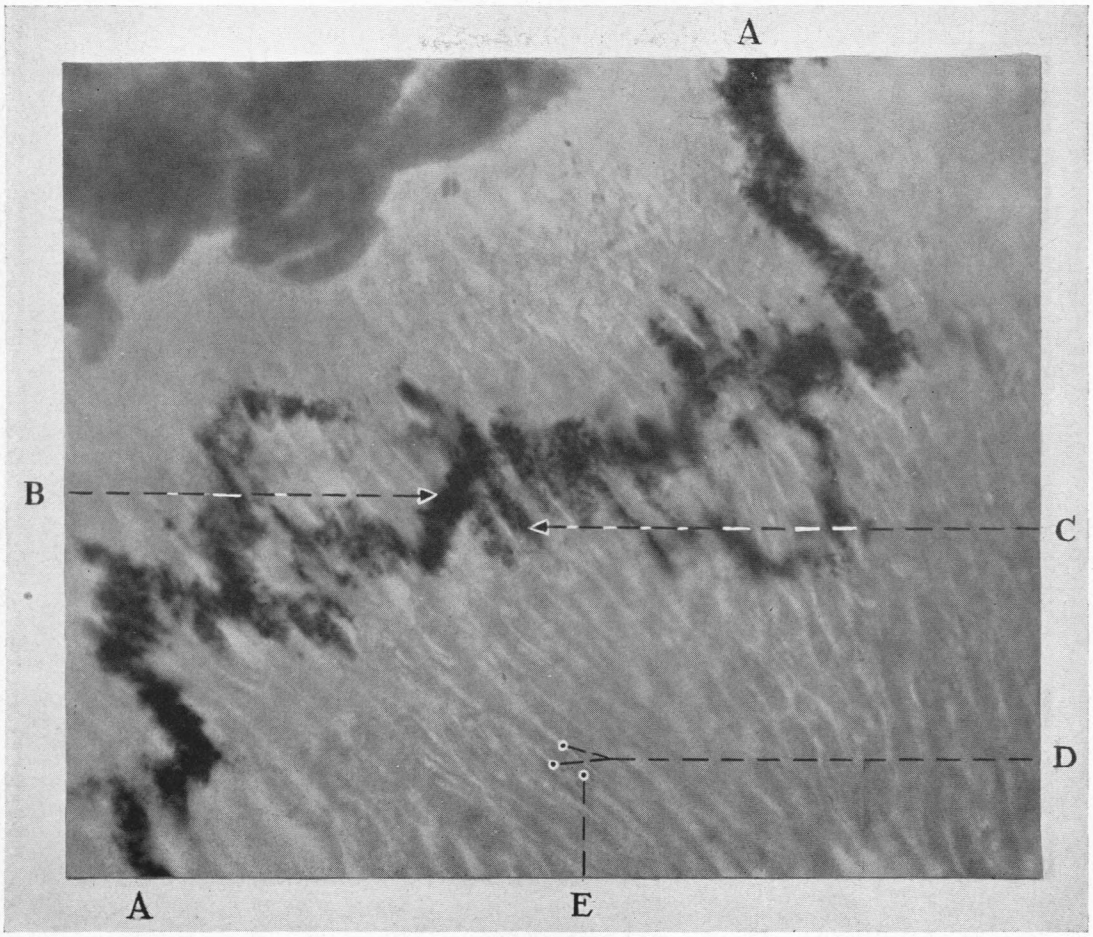

FIG. 9. ORGANIC ENAMEL MATRIX FROM THE ADULT HUMAN TOOTH 4μ NEUTRAL FORMOL FIXATION. GRAM WEIGERT × 1000. ILLUSTRATING AN ORGANIC TRACT TAKEN FROM AN AREA OF DEEPLY PENETRATING CARIES AND DEMONSTRATING LATERAL SPREADING, IMPLICATING NEIGHBORING RODS. NOTE CONTINUITY BETWEEN THE INFECTED AND NORMAL PORTIONS OF THE ROD MATRIX.

A. TRACT FILLED WITH SPHEROIDAL GRAM POSITIVE ORGANISMS.

B. LATERAL PENETRATION OF BACTERIA TO THE INTER-ROD MATRIX IMPLICATING NEIGHBORING RODS.

C. INVASION OF ORGANISMS PROCEEDING DOWN THE CORE OF A SINGLE ROD.

D. NORMAL INTER-ROD MATRIX.

E. NORMAL ROD MATRIX.

NOTE THE ACIDOPHILIC STAINING REACTION OF THE MATRIX IN PROXIMITY TO THE MICRO-ORGANISMS.

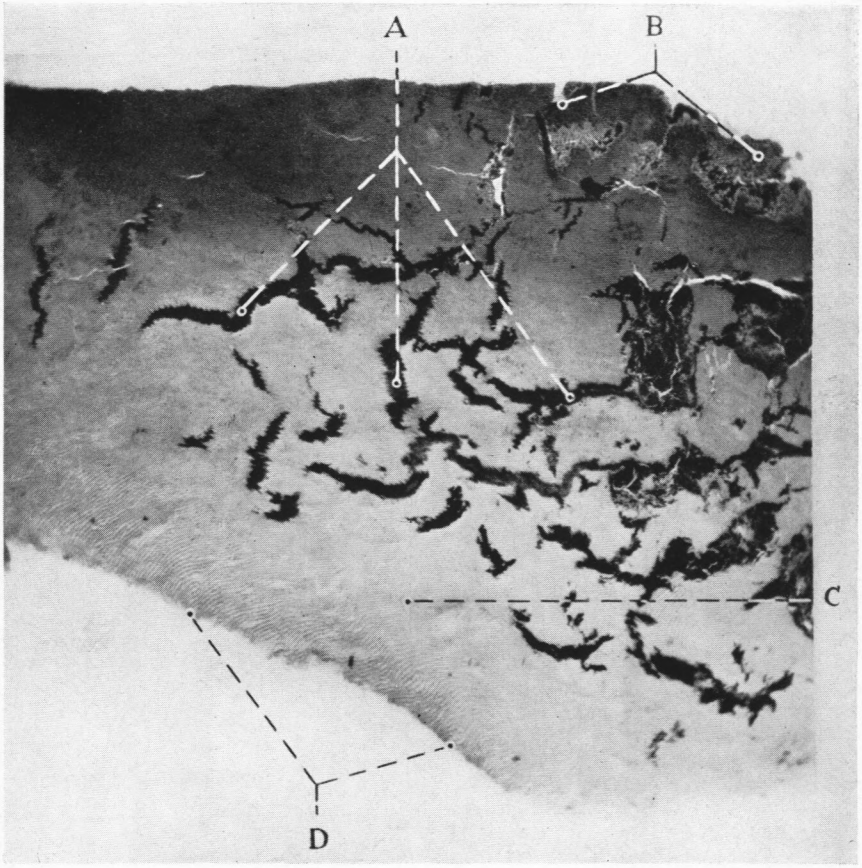

FIG. 10. ORGANIC ENAMEL MATRIX FROM THE ADULT HUMAN TOOTH 4μ NEUTRAL FORMOL FIXATION. GRAM WEIGERT × 140. ILLUSTRATING PATHOLOGIC PROCESS OF EXTENSIVE CARIES OF THE MATRIX THROUGH THE DEVELOPMENT OF MULTIPLE IRREGULAR CHANNELS OF DISSEMINATION.

A. MULTIPLE IRREGULAR CHANNELS OF DISSEMINATION OF ORGANISMS.

B. EROSION OF THE SURFACE OF THE MATRIX.

C. NORMAL ENAMEL MATRIX.

D. DENTINO-ENAMEL JUNCTION.

NOTE CHANGES IN TINCTORIAL REACTION OF INFECTED MATRIX IN CONTRAST TO THAT OF THE NORMAL MATRIX.

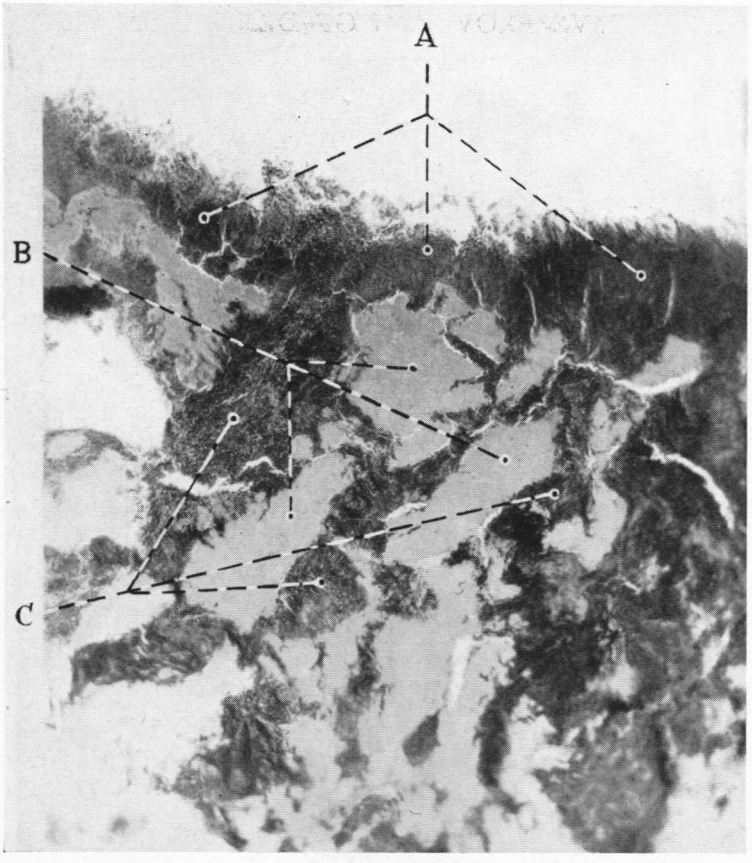

FIG. 11. ORGANIC ENAMEL MATRIX FROM THE ADULT HUMAN TOOTH 4μ NEUTRAL FORMOL FIXATION. GRAM WEIGERT $\times$ 500. ILLUSTRATING ADVANCED EXTENSIVE CARIES SHOWING ALL TYPES OF DISSOLUTION AND A BREAKING UP OF THE SUPERFICIAL MATRIX.

A. THE SURFACE PLAQUE.

B. ISLANDS OF ENAMEL MATRIX WHOSE CENTERS SHOW A NORMAL TINCTORIAL REACTION.

C. SPREADING CHANNELS OF PROTEOLYSIS ISOLATING ISLANDS OF ENAMEL MATRIX.

EFFECTIVE PROVISION OF DENTAL SERVICE TO POPULATION GROUPS*

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Change in the form of dental practice is characteristic of the evolutionary progress of our profession. The present war has accelerated the tempo of change in dentistry just as it has speeded up activity in other scientific and technical fields. And what is true of the scientific and technical phases of dentistry applies with equal force also to its social and economic aspects.

Dental patients can be divided into (1) the active demand group, those who are actually receiving service; (2) the potential demand group, those who appreciate the value of dental care but who do not receive regular treatment because they cannot afford to pay for it, and (3) the latent need group, those who need dental care but are not seeking it and are not receiving it regardless of their ability to pay.

Our professional responsibility dictates that persons in the active demand category should receive the best, the most complete and the most economical dental service possible.

For those who make up the potential demand group, i.e., those who would avail themselves of regular dental care if they could afford it, the dental profession, for the sake of its self-preservation, has the responsibility of planning the most efficient methods whereby these patients may be provided with dental service. Selection of a method of practice, however, should not be confused with the establishment of the plan whereby the cost of the dental service to be provided for this group is to be paid.

How the public should arrange to pay for dental service is not primarily the concern of the dental profession although dentists are entitled to express their views as individual citizens. On the other hand, the standard of service to be rendered is a professional question in the solution of which dentistry should not brook the interference of lay persons. Likewise, although efficient methods must be insti-

*Delivered before the New York Section of the American College of Dentists on May 18, 1944.

tuted, dentists should at no time compromise the quality of the service itself, nor should they absorb the cost entailed by accepting low fee schemes. To deviate from these principles is to jeopardize dental progress and dental health.

The latent need group can be translated into the effective demand category by organizing dental health educational and service programs for school children and by sustained programs of dental health education for adults. Persons who make up the latent need category include the well-to-do and the average wage earners as well as those who cannot afford to pay any fee. It is important to keep in mind that many plans for providing dental care have failed because a great proportion of the people for whom the plans were intended did not avail themselves of them. They simply did not feel that they needed it. Persons in families with an income over $5,000 receive approximately one-half of the service they actually need.[1] If dental care were to be provided free for the entire population as of today, the potential need group, which feels no need for sustained dental care, would still stay away from the dentist.

Unlike businessmen who are not in business primarily for their health, or the health of anyone else, dentists have been given a vested interest in the health of the public by the public itself. The public therefore expects our profession to be interested in its health. With the general growth in public health consciousness the public now expects us to furnish the service which we are prepared to render on a population basis. This calls for cooperation and experimentation. It does not mean holding out for the *status quo*.

In answer to the growing demand for health planning, the American Dental Association today is standing by the principles evolved in 1938, over seven years ago.[2] These principles, largely modeled on the oppositional attitude of organized medicine, have so far had the effect of making health planning bodies ignore dentistry altogether. The health planners feel that dentistry will be more easily dealt with after the medical profession is brought into line. It is not sufficient for dentistry to measure all health plans on the Procrustean couch of the "Eight A.D.A. Principles," as it were, and if a plan does not

fit the "Principles," reject the plan and sit back to wait for another plan to come along.

So far, the three main avenues of approach suggested for the solution of the problem of provision of dental care on a population basis are the following:

1. Change in the method and scope of dental education, along with the use of "auxiliary personnel."[3]
2. Change in the method of payment for service, including prepaid, post-paid, tax-paid and employee-employer paid systems.[4]
3. Change in the method of dental practice.[5]

The following is devoted largely to the various methods of dental practice available for the effective provision of dental service to population groups:

Public Health Dentistry[6]*:* This form of dental practice may be defined as the provision of dental care on a community basis. It includes health education and control of dental disease of patients *en masse* as well as service for the individual patient. Interest of the community, or of the government which represents it, in health is not a "New Deal Innovation." Public Health Service in the United States was inaugurated in 1798 and has gradually grown so that at present health care of the individual citizen is actually an activity of federal, state and local government agencies. About one-third to one-half of the dental service received by children is now provided by community funds.

Public health dentistry, more or less quiescent throughout the years, has been greatly accelerated under Titles V and VI of the Social Security Act of 1935.

The United States Public Health Service at present maintains regional consultants at convenient locations and also a staff of special consultants in Washington, to assist the States in developing dental health programs. At the April 1938 conference of State and Territorial Health Officers with the Surgeon General of the United States Public Health Service, minimum qualifications for public health dentists were adopted.

Before the Social Security Act was passed in 1935, there were fifteen states conducting dental hygiene programs. In 1940, there

were thirty-eight states with dental programs, and in 1941 there were 154 full-time dental health administrators employed by the various states, as against eight in 1935.[7]

Public health dentistry is receiving added impetus as a result of war conditions. The United States Public Health Service is now attempting to relocate dentists in communities expanded by war industries. Increased interest in industrial dentistry at present is due directly to population changes and dislocations caused by war conditions. Military requirements have absorbed about one-third of available dental manpower while the demand for dental service, because of better economic conditions of workers, has been increased by at least a third.[8]

With manpower at a premium, industry is especially interested in keeping available workers healthy. In industries where the worker is exposed to hazards which affect the jaws, teeth, periodontium and oral mucosa, industrial dentists are employed in increasing numbers to provide close cooperation with industrial physicians in the prevention, diagnosis and treatment of occupational diseases and accidents affecting the dental and oral organs. These dentists also conduct preemployment dental examinations, dental health education and perform emergency service.[9]

In a survey made by the United States Public Health Service, it was shown that in January 1943, industrial hygiene services were established in thirty-eight states as part of the state health departments. Since 1942 three state industrial hygiene divisions have added to their staffs the services of full-time dentists and in twenty-six other states the divisions of dental dealth and industrial hygiene are beginning to cooperate in the establishment of industrial dental programs.[10]

The American Dental Association approved "Seven Essentials of Industrial Dental Programs" in 1941. The A.D.A. Council on Dental Health now has a Committee on Industrial Dentistry and several state and local dental societies, including the First District Dental Society of the State of New York, have appointed similar committees.[11]

Unfortunately, many of the programs conducted as industrial

dentistry are merely schemes for farming out patients to dentists at predetermined arbitrarily established "low fees."

Dental Care by State Agencies[12]*:* All states with the exception of Arizona and Idaho are giving some attention to dental health, with clinic service being conducted mostly for demonstration or educational purposes and occupying a subsidiary role to dental health education. Dental service is provided mostly for children, although eighteen states sponsor some dental care for adults. Children receive treatment in schoolrooms, in dental trailers and in child-health centers. Treatment includes examination, prophylaxis, emergency care and general dentistry. Service is usually restricted either to the indigent, to certain age groups, to specified school grades, or to "those who need the service most."

The healthmobiles or dental trailers, are complete dental offices on wheels and contain a dental chair and instruments, laboratory facilities, sterilizer, and cabinets for instruments, linens, and supplies. In states where mobile units are not available, traveling dentists set up portable equipment in a room of the school being served. Maintenance of stationary dental clinics by state health departments is the exception rather than the rule.

Concentration in urban areas is greater among dentists than among physicians. There are hundreds of communities and even whole counties in some states without a single dentist. Even special inducement in the form of low or free rent offered by small towns to attract a dentist to locate there have been without avail. Some counties in the United States are too sparsely settled or too poor to support a dentist. The solution for the dental problem in these areas is to take the dental service to them—mobile dental units.

Less than one-half million dollars can be segregated as applying specifically to the cost of dental services, out of a total expenditure by states for health of $285,715,800. Three-fourths of the dental expenditures are supported in almost equal proportions by the state and federal governments. The remaining quarter is composed primarily of contributions by private foundations and secondarily of allotments from local tax funds.

Hospital dental service is usually of two types:

(1) Service in connection with the general care given to hospitalized patients; and

(2) Out-patient departments.

As far as extraction and minor oral surgery are concerned, it is easy to see the usefulness of out-patient dental clinics. For filling teeth and for prosthesis free dental clinics manned by voluntary dentists are largely inadequate.

School Dental Programs: Dental health programs in the schools are now recognized as of minor value unless associated with service programs. If the service side is neglected, children will fail to establish the dental health habits which they would develop and maintain throughout life.

In New York City, where dental service along with health education is provided in the schools by the Dental Division of the Department of Health, the child treated at the school clinic is found in many instances to be the first member of his family to have contact with a dentist or to receive dental treatment. The interest in dental care thus aroused, has a distinct educational value for the entire family.

There are ten bureaus in the Department of Health of the City of New York, covering every phase of public health. Sadly missing in New York City is a bureau on dental health.

It will be interesting to note, in this connection, that school dental service in England is being maintained and even expanded in spite of war conditions. Children who were evacuated to safe areas have been provided with dental care as far as possible. Many British dentists have been deferred from military duty on the ground that their services are essential to the continued health of their respective communities.

In the New York City Vocational Schools, the examination of over 10,000 pupils at the Central Commercial High School, showed the most frequent type of dental treatment received by pupils to consist of extractions and filling of individual teeth; pupils rarely received complete and sustained dental care.[13] The harmful effects of dental neglect show themselves in these adolescent boys and girls in the

great number of missing teeth, high incidence of dental caries and malocclusion. Of the pupils examined, 60 per cent were found able to pay average fees for dental care. Many of these children had not been receiving dental treatment because of the lack of motivation. Of the remaining 35 per cent, 26 per cent could afford clinic fees and approximately 15 per cent could not afford to pay any fee at all.

Although upward of 98 per cent of children in the country need dental care, only 25 to 30 per cent are actually receiving it. There are 35 million children in the United States between age 6 and 20 years. These children require an average of four hours of dental service to take care of their accumulated need. If this were to be done in one year, it would require the entire chair time of approximately 70,000 dentists. However, Klein[14] has shown that care of the annual increment of dental caries in children would require one hour of the dentist's time per child. This means that servicing of the yearly need in the new generation of children would require the annual services of 16,500 dentists.

The report of the subcommittee of the Committee on Dental Research of the American College of Dentists to Study Plans for National Dental Health Care, found that there would be an adequate number of dentists available to take care of the annual increment if there were an equal distribution of dentists throughout the country.[15]

Treloar[16] estimates that $40,297,894 would be necessary to take care of the 10,074,000 needy children at $4 per hour per child. Strusser proposed a universal dental care program in 1935, including dental health education for New York City children. This program has since been streamlined on the basis of later data and should receive professional endorsement and then be brought to public attention.

Private Practice[17]*:* The private practitioner can be useful in providing dental care on a community basis by participating in dental programs for population groups and by providing dental care in his own office.

The amount and kind of dental care received by the population in private offices is shown in a report of the United States Public Health

Service[18] which gives an account of the dental care actually received by 70,554 persons. According to this survey, 33 per cent of the families interviewed visited dentists during the previous year. However, 78 per cent of the visits were for extractions only, while 15 to 19 per cent of all persons in the families interviewed had never visited a dentist at any time.

The percentage of persons visiting a dentist for service other than extractions only, varied greatly with the economic and cultural level of the family, from 42 per cent for professional families to 16 per cent for those of skilled workers. For colored persons the number of those visiting the dentist was 8 per cent. Children age 6 to 15 years in families of semi-skilled workers had 31 per cent more teeth extracted than those belonging to professional groups.

The fact that our population is not receiving adequate dental care was revealed in mordant colors in the Selective Service examinations. The Army found in the 18- and 19-year boys that one out of every three teeth was affected by decay, with an average of six teeth per boy in need of filling. The Victory Corps-Physical Fitness Dental Program of the American Dental Association, in cooperation with the United States Office of Education, is now endeavoring to raise the dental standard of pre-induction age groups throughout the United States.[19]

In a survey by Ciocco, Klein and Palmer of the U.S.P.H.S.,[20] it is shown that a relatively large number of selectees gave evidence of dental defects fifteen years ago, when they were in elementary school. These investigators state:

"It is particularly disturbing to say that in spite of knowing which children in a community would grow into physically handicapped adulthood, the health professions, the lay professions, and especially society as a whole, have to date apparently failed to take advantage of the knowledge."

A survey by the Committee on Economics of the American Dental Association[21] corroborated the well known fact that a marked inverse relationship exists between different income levels and dental needs. The lower the income level, the greater the need for dental care.

The annual cost of dental care among those actually receiving it,

an American Dental Association Survey shows, is $48.96 for males 15 years of age and over and $45.43 for females in the same age range, when computed on the fee schedule of the United States Veterans Administration.[22]

Voluntary Plans: The Dental Health Service Clinic survey[23] made by the American College of Dentists shows that initial dental care required by the adult patient would cost at clinic fees $55.23, while in a low fee private practice the cost would have been $71.34. Maintenance care needed would cost $12.62 at the clinic and $16.05 in the average low fee office.

In order to meet these costs, prepayment plans are advocated on the order of those already organized for the provision of medical care. The principle on which prepayment plans of medical care are based depend upon pooling of risks and resources.[24] Prepayment medical plans exclude certain persons who have chronic, incurable or recurrent diseases. In dentistry the risk principle does not exist. There is instead a backlog of accumulated dental neglect which makes it necessary to provide a definite amount of service for 75 to 80 per cent of the population before maintenance care can be undertaken.

It is pointed out by Clark and Clark[25] that nineteen dentists, five dental X-ray technicians, nine dental hygienists and three dental laboratory technicians would be required to each 20,000 medical plan subscribers or, on a population basis, twice as many dentists as are now available. This estimate of personnel will not allow for preventive care in order to reduce future needs.

Available dental personnel, as shown by Fahs[23], could take care of only the initial dental care of 20 million people annually. If the children were not given complete and sustained dental care, there would always remain a backlog with which the dental personnel available would not be able to cope. Obviously, prepayment dental plans for adults, while of benefit in centers of concentrated population, are not the answer to the problem of providing dental care on a population basis.

Contract Practice: In the attempt to make more dental treatment possible for workers of limited income, certain schemes are being

devised by certain groups and by individual dentists outside of the organized dental profession. Many of these plans seek to establish dental practice altogether on a price basis. Some of these schemes are masquerading under the name of industrial dentistry and under other labels such as "Employees' Dental Service," "Group Dental Service," etc. The fact remains that these schemes are essentially contract dental practice which feature prearranged low fees by specified dentists, some of whom actually establish offices in the industrial plants. These methods are generally not in consonance with the Code of Ethics of the A.D.A. They are not infrequently also contrary to the legal provisions regulating dental practice in the respective states and place those engaged in this practice in unfair competition with their fellow practitioners in the community.

In a report on "Current Industrial Dental Programs in New York City,"[26] submitted to the Committee on Community Dental Health of the New York Tuberculosis and Health Association, we find that these "low fee" services are unsupervised, are not under organized professional responsibility and may actually prove inimical to public health.

In the report of the New York Tuberculosis and Health Association, we also read the following:

". . . it seems curious that organized dentistry has as yet evinced no interest in the firms where the work is done by a private dentist on the premises, intent on augmenting his own private practice; a method patently in violation of the American Dental Association standards. Our local dental society, having just adopted a plan for putting these standards into action, is at the moment in the position of dictating to employers the limits of new dental services and yet of manifesting no interest whatsoever in the work of those large firms where arrangements with private practitioners have existed for years. It is easy to see the difficulties likely to be faced by the Society in attempting to discredit old established plans, yet we sincerely hope the Society will succeed in accomplishing a uniform adoption of the American Dental Association standards before the termination of the war."

Fees charged by the majority of dentists are the result of economic stress and are so low that they have in the past afforded them a bare living at best. The number of dentists who serve patients to whom the cost factor is only of secondary importance is extremely

small. At least nine out of every ten dental patients must give heed not only to costs but are actually forced through economic stringency to seek dental service—and this is true of other more tangible commodities they purchase—at the lowest fees obtainable when they do not actually forego treatment altogether because of costs.

As a result of this condition, fees generally charged by the dentists who serve patients of limited income are so low as to allow these dentists a financial return which in some instances is lower than the wages earned by the better skilled mechanics. Dentists were found to earn an average of $63 weekly in New York City. It should be recognized also that the first $40 earned weekly by the dentist merely represents the interest on his investment for his education and equipment.[27]

As we have pointed out elsewhere[6], there are certain basic costs which must be met in the production of dental service. Certain elements are trying to circumvent these irreducible costs by having the dentist shoulder the cost involved in rendering dental treatment. It would be just as logical for the dentist to accuse the labor unions and industry of being responsible for high living costs because the latter insist on a living wage for the worker or on a fair return for their efforts and investment by management as to seek to reduce the already low income of dentists because the lower earning groups find private dental fees a burden.

The cost of dental care, we know, is burdensome to certain income groups. Experiments are needed to determine the "best buy" for the consumer of dental service. But lip-service will not produce the facts. Many plans have been proposed. None has as yet been put into action.

Dental Care on a Community Basis[28]*:* Whether dentistry is to be provided in clinics, group practices, or in the private offices as they exist at present must in the end depend on the size of the community from the standpoint of concentration of population, the economic status, the cultural level and the health consciousness of the people to be served. No "rule of thumb" decisions can be made either by the dentists or laymen in this regard.

In "An Organization Outline for a Community Dental Program,"[18] by Strusser and Salzmann, published in 1938, the appointment of community dental advisory committees was advocated, which are to include, among others, representatives from local dental societies, whose duty it would be to outline the scope, professional standards and procedures to be pursued in local dental programs where the service is to be performed at least partially in private dental offices. It should be stated here, however, that prepaid, tax-paid and other methods of payment for dental care which are not made directly to the dentist by the patient are not productive of the best dental service in the private offices, unless the work is subject to supervision and review.[28]

A community plan for dental care in relation to national health was published in the *Journal of the American Public Health Association,* in June, 1940, as a report of the Committee on Community Dental Service of the New York Tuberculosis and Health Association.[28] Under this plan, dental patients were classified according to ability to pay.

The plan proposes to provide adequate care of a complete nature for children. Complete care was to be limited at first to children age four years and under and continued to age 16. After age 16 years dental care was to be provided on an adult basis. Included in the plan were the type of dental care to be provided, the method or organization and administration of the community dental program, the provision of dental service in private offices on a community basis, the compensation to be paid to dentists, and other items of equal importance. To date, this plan has received no consideration from organized dentistry.

In conclusion, the need today is for the institution of sustained dental programs for children on a caries increment basis, along with dental health education. This will eliminate the backlog of dental decay which at present would tax dental manpower and budgetary allowances for health made by communities. Elimination of the backlog would make it possible later to provide maintenance dental care for adults on a prepaid basis. As far as the immediate dental

needs of adults are concerned, experimentation is urgently needed to provide factual data beyond that presently available.

Establishment and administration of dental plans require public and professional cooperation. It is up to the dentists to provide information on the specific professional conditions which must be adhered to in the administration of health service for population groups whether it be in private offices or in clinics.[29] On the other hand, it may well be that expert business administrators can contribute of their knowledge and experience in streamlining certain traditional methods followed by dentistry to the advantage of all concerned.

It will be difficult for some dentists to agree that compensation of the dentist in practice designed to take care of population groups is an item which must be solved by the cooperative effort of public and professional representatives. However, to refuse to sit down with the representatives of the public in order to determine equitable remuneration for the dentist and the method of administering the service is to run the risk of losing our present vested legal standing as a profession.

Professional administration and maintenance of the highest professional standard of service must remain in the hands of the dentists themselves. Dentists must not allow lay people to dictate to them how the art of dentistry is to be practiced. But, dentists must divest themselves of the strait-jacket imposed on them by tradition. Experimentation is urgently needed in the practice of dentistry on a population basis. Dentistry is not answering public demands by saying that it already has a plan which includes research, health education and service for children and indigents. Labor, industry and government are not going to be content with general statements in answer to specific demands to provide dental care for the public.

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RESEARCHES AIDED BY THE AMERICAN COLLEGE OF DENTISTS

REPORT BY THE COMMITTEE ON RESEARCH
ALBERT L. MIDGLEY, D.M.D., *Chairman*[1]
Providence, R. I.

I. INTRODUCTION

For many years, the American College of Dentists was drawn steadily and inevitably to the conviction that the dental profession could have no object more worthy than the promotion of dental research—the sure promise of professional progress and opportunity, and the dynamic support for a virile future. A progressive profession strives earnestly under its ideal of ceaseless growth to maintain the highest standard of excellence in its service, not only by meeting all present demands but also by anticipating those of the future, and by accepting with enthusiasm the challenge of new truth and increased responsibility.

Well supported scientific initiative in the prosecution of research is the watchword of the day. More and more convincing demonstrations of dentistry's zeal for research is essential, if we are to assert effectively our claim upon public confidence and support. We should direct attention to the fact that we need and deserve endowment, to ensure the continuance of progress in the solution of our problems and also of all problems of joint concern and interest to dentistry and medicine.

The Committee on Dental Research of the American College of Dentists was created in 1937. Recognizing promptly the need for advice, support and assistance from competent sources as to practicable ways and means vital to the promotion of its activities, the Committee in 1938 invited the International Association for Dental Research to appoint from its membership a standing committee to work with the College in an advisory capacity. The International Association for Dental Research promptly accepted the invitation, and since 1938 has cooperated with the Research Committee of the

[1]The other members of this Committee are: L. E. Blauch, P. J. Brekhus, J. E. Gurley, P. J. Hanzlik, A. B. Luckhardt, L. R. Main, L. M. S. Miner, L. W. Morrey, Irvine McQuarrie, Fr. A. M. Schwitalla.

College. Reports and other articles on the Committee's activities—its plans and procedures—have been published in the College *Journal* as follows: 1937 Vol. 4: pp. 61, 74, 82, 98, 256. 1938 Vol. 5: pp. 115-119. 1939 Vol. 6: pp. 276-281. 1940 Vol. 7: pp. 20, 21. 1941 Vol. 8: pp. 128, 129.

Among the Committee's chief procedures has been the promotion of dental research by awarding grants-in-aid to applicants for assistance. Thus far this has been done by supporting projects that had been successfully initiated, and could be carried to completion with the aid of relatively small grants. All of the funds for this purpose, to the present time, have been appropriated by the Regents from the treasury of the College.

The first grants-in-aid were awarded in 1940: some have been awarded annually since 1940. The grantees for the fiscal year beginning July 1, 1944, are:

Dr. Bernhard Gottlieb, Baylor University (Dental School), Dallas, Texas.

Dr. Wendell L. Wylie, University of California (Dental School), San Francisco, Calif.

Drs. Harrison R. Hunt and Carl A. Hoppert, Michigan State College, East Lansing, Mich.

Section II presents a complete list of the publications (33), *before July 1, 1944,* in which grantees described researches aided by the American College of Dentists. The following grantees, to whose work support has been given during one or more years since 1940, have not yet published reports or papers descriptive of the respective researches:

Dr. William H. Bauer, School of Dentistry, St. Louis University, St. Louis, Mo. (Researches completed: two papers in course of preparation.)

Dr. June R. Schamp, College of Physicians and Surgeons, San Francisco, Calif. (Research not yet completed.)

Dr. Morris Steggerda, Carnegie Institution of Washington, Cold Spring Harbor, Long Island, N. Y. (Research completed; paper in press.)

This bibliographic compilation was prepared by Dr. William J. Gies. It required a considerable amount of time, and a practical knowledge of the subject and its related essentials. He gave studious preliminary attention to a multitude of details and to extended correspondence with each grantee, which included submission of copies

of the list of publications for correction—later of copies of abstracts—resulting in confirmation of everything now on the manuscript. He endeavored to make the compilation not only accurate in detail and complete, but also formulated it to establish a precedent that could be followed satisfactorily in succeeding annual summaries for the convenient information of all concerned.

It is believed that the precedents established by this report for 1940-44 will be followed, in continuance of this permanent record of the interest of the American College of Dentists in the practical promotion of research in dentistry.

II. LIST OF PUBLICATIONS OF RESEARCHES, BY RECIPIENTS OF GRANTS-IN-AID DURING ONE OR MORE OF THE FISCAL YEARS 1940-44; INCLUDING ABSTRACTS OF PAPERS.

The appended list of publications, by the grantees named below (and their associates), is complete for the fiscal years 1940-44 (ending June 30):

Dr. W. D. Armstrong, University of Minnesota, Minneapolis, Minn. (7)[2]

Dr. Michael S. Bales, Tufts College (Dental School), Boston, Mass. (8)

Mr. William J. Furuta, University of Illinois (College of Dentistry), Chicago, Ill. (6)

Drs. Harrison R. Hunt and Carl A. Hoppert, Michigan State College, East Lansing, Mich. (1)

Dr. Albert H. Kniesner, Western Reserve University, Cleveland, Ohio (2)

Dr. James Nuckolls, University of California, San Francisco, Calif. (5)

Dr. Samuel Seltzer, University of Pennsylvania, Philadelphia, Pa. (3)

Dr. M. L. Tainter, College of Physicians and Surgeons, San Francisco, Calif. (4)

The abstracts of the "papers" (which include the findings stated in preliminary reports) have been reviewed and approved by the respective authors. The bibliographic groups are in the chronological order of the initial awards to the grantees. The year numerals following the name of the grantee, at the head of each group of publications, indicate the fiscal year or years in which grants-in-

[2]Each terminal numeral in this list of names and addresses of grantees indicates the sequence of the corresponding group of publications in the succeeding bibliography.

aid were available. The sequence numeral preceding each bibliographic item facilitates cross references.

(1) *Grantees:* Harrison R. Hunt and Carl A. Hoppert (1940-44)
Michigan State College, East Lansing, Mich.

Preliminary reports (1–2): 1) Harrison R. Hunt, A.M., Ph.D., and Carl A. Hoppert, M.S., Ph.D.—Statement included in a summary of findings in research on dental caries, by Hoppert, Hunt, Webber and Canniff: *Dental Caries*, second edition, p. 129; 1941. [Published by the American Dental Association.]

2) H. R. Hunt, Ph.D., C. A. Hoppert, Ph.D., and W. G. Erwin, Ph.D.—Inheritance of susceptibility and resistance to caries in rats: *J. Den. Res.*, *23*, 205; 1944, June. [Proc. Int. Assoc. Den. Res., Mar.][3]

Paper: 3) Harrison R. Hunt, A.M., Ph.D., and Carl A. Hoppert, M.S., Ph.D.—Inheritance of susceptibility and resistance to caries in albino rats *(Mus norvegicus): J. Am. Col. Den.*, *11*, 33-7; 1944, Mar.[4]

This study is the first extensive attempt to evaluate the relation of heredity (gene effects) to variations in resistance to dental caries. The experiments have been in continuous progress since 1937, under controlled laboratory conditions for genetic studies. The experimental animals have been albino rats given Hoppert's balanced diet (which induces caries in lower molars in rats)—66 percent *coarsely* ground hulled rice, 30 percent whole-milk powder, 3 percent alfalfa-leaf meal, and 1 percent common salt. *These experiments have demonstrated that inheritance in rats is an important factor in susceptibility to caries.*

In this study, breeding females are isolated when pregnant. The young are weaned when 25-30 days old; when 35 days old they are placed on the standard diet, with water from the college water-system, and thereafter are given all they will eat and drink. The

[3]The program of the meeting at which this report was presented included one by "Philip Jay, D.D.S., D.Sc. (Den. Sch., U. of Michigan): [The] incidence of [oral] *L. acidophilus* in Hunt-Hoppert caries resistant and caries susceptible rats." In the abstract of Dr. Jay's report, as published in the Proceedings (*J. Den. Res.*, *23*, 205-6; 1944, June), the names of Drs. Hunt and Hoppert were added as co-authors, in recognition of their cooperation with him in supplying the stocks that he used and in aiding him to obtain cultures from the mouths of the rats.

[4]At the head of this paper, as published, the degree of D.D.S. was mistakenly inserted after the name of each author instead of the degrees correctly given here.

lower molars are examined every 14 days, and the locations and sizes of carious lesions recorded as of the dates of observation. In this examination, the rat is held firmly, belly up, with the right hand grasping the loose skin on the back of the neck, the mouth is opened with a nasal speculum, and the lower molars are inspected under a strong light. Thus far 3164 rats have been examined—a few at least fifty times. When the experiments were begun (1937, spring), 119 rats from three local sources were placed on the standard diet. In the 116 surviving animals, initial carious cavities began to develop in from 28 to 209 days (average 70) after the beginning of the test. Susceptible rats from this first generation were crossed to start a susceptible line; resistants were paired to begin a resistant strain. Selection of individuals, progeny testing, and close inbreeding have been used throughout to extend these two lines.

The results thus far obtained may be stated in terms of the *means* of sibship (family) averages (five or more rats each) for initial incidence of caries. The means for the *susceptible* generations have steadily decreased from 57 days in the second generation to 22 days in the eleventh; variability is relatively low. The means for the *resistant* generations increased from 116 days (second generation) to 248 days (sixth), with indications that this is a maximum. Variability within this resistant line is very high and not decreasing. A very striking feature is the occasional occurrence, in the resistant line, of individuals which—although subjected to the coarse-rice diet for from 600 to 700 days—die without developing caries. *This suggests that a caries-immune strain is a possibility.*

Additional experiments are in progress. "A number of years may elapse before data are secured for a *final* genetic analysis of the problem."

(2) *Grantee:* Albert H. Kniesner (1940-41)
Western Reserve University, Cleveland, Ohio

Preliminary reports (4–6): Thomas J. Hill, D.D.S., and Albert H. Kniesner, B.S., D.D.S.—

4) Growth of oral lactobacilli in saliva: *J. Den. Res.*, *20*, 266; 1941, June. [Proc. Int. Assoc. Den. Res., Mar.]

5) A nutritional requirement of oral lactobacilli and its influence on dental caries: *N. Y. J. Den.*, *11*, 319; 1941, Sep.

6) Nutritional requirements of oral lactobacilli. I. Pantothenic acid and dextrose (*d*-glucose): *J. Den. Res.*, *21*, 310; 1942, June. [Proc. Int. Assoc. Den. Res., Mar.]

Papers (7–8): 7) Albert H. Kniesner, B.S., D.D.S., Arvin W. Mann, B.S., D.D.S., and Tom D. Spies, M.D.—Relationship of dental caries to deficiencies of the vitamin B group: *J. Den. Res.*, *21*, 259-62; 1942, June.

Forty-one selected patients—"with evidences of pellagra, beriberi, and riboflavin deficiency," or a mixture of deficiency diseases as found in repeated examinations—had low incidence of caries and correspondingly small numbers of oral lactobacilli. Using the lactobacillus count as an index of caries activity, no appreciable difference was noted in pantothenic-acid content of salivas from patients resistant or susceptible to caries. In all cases, pantothenic acid was present in concentrations sufficiently high for growth of oral lactobacilli. These patients had low incidence of caries irrespective of poor oral hygiene, of accumulations upon the teeth, and of high-carbohydrate diets containing abundance of simple sugars. There was no evidence that fluorine occurred in concentrations sufficient to lower significantly the incidence of caries.

8) Thomas J. Hill, D.D.S., and Albert H. Kniesner, B.S., D.D.S.—Nutritional requirements of oral lactobacilli: I. Pantothenic acid and dextrose (*d*-glucose): *J. Den. Res.*, *21*, 467-71; 1942, Oct.

By accredited experimental procedures it was found that pantothenic acid is essential for growth of oral lactobacilli, and is present in human saliva. The optimal proportion of pantothenic acid for growth of oral lactobacilli is not greater than that usually present in human saliva. Dextrose is essential for growth of oral lactobacilli, 1.5 mg. per cc. of medium being adequate for maximum growth. Pantothenic and dextrose requirements for maximum growth of oral lactobacilli vary for different types and strains.

(3) *Grantee:* Samuel Seltzer (1940-41)
University of Pennsylvania, Philadelphia, Pa.

Papers (9–10): 9) Samuel Seltzer, D.D.S.—Effective duration of some agents used for dentin sterilization: *J. Den. Res.*, *21*, 115-23; 1942, Apr.

The relative efficiencies of various agents used for cavity sterilization were determined in *in-vitro* tests.

Method. (a) After a cavity had been prepared under aseptic conditions for filling, and before application of the agent, shavings of the dentin (made with a sterile bur) were immediately transferred to a tube of Rosenow's brain broth. (b) The agent was then applied for three minutes and the cavity closed with a temporary filling (guttapercha and quick-setting zinc oxide and eugenol cement). One to two weeks later the filling was removed under aseptic conditions; dentin shavings were transferred to culture medium; and the cavity was filled—with guttapercha placed between the underlying dentin and amalgam. (c) A year later the second filling was removed aseptically and dentin shavings transferred to culture medium. By comparing the percentages of cavities successfully sterilized with each agent, relative efficiencies were ascertained. Results for the three stages of bacterial examination were noted in separate tests for nine agents: Morson's Kreosote; 10 percent aqueous solution of iodin; pure phenol; physiologic sodium chloride solution; isotonic solution of iodin; 50 percent solution of *thymol* in 95 percent ethyl alcohol; 50 percent solution of *phenol* in 95 percent ethyl alcohol; Howe's ammoniacal silver nitrate; 95 percent ethyl alcohol.

For cavities in 93 teeth (4 to 15 cavities for each agent; also 12 controls to which agent was not applied), previously treated in the manner stated above, the results indicated that bacteria remained in the dentin "after proper cavity preparation" and filling for one year—cultures of most cavities including controls (untreated) were positive (living bacteria). All of the teeth were vital throughout. "A hermetic seal" of a cavity "will not sterilize the dentin." The results "point to the possibility that recurrence of decay under fillings is due to inadequate sterilization of the dentin before insertion of the fillings. There is no other way, except leakage, to explain the presence in the dentin of [these cavities of] organisms commonly associated with dental caries." The results indicated that Morson's Kreosote, though inadequate, was more effective than any of the tested agents for dentin sterilization. "The search for the ideal antiseptic still continues." [See paper (10).]

10) Samuel Seltzer, D.D.S.—Effectiveness of antibacterial agents used in cavity sterilization: *J. Den. Res.*, *21*, 269-77; 1942, June.

An extension, by the same general method, of the study described

in the preceding paper (9). One occlusal cavity in each of 20 lower molars was treated with one of seven additional agents; 20 such cavities were controls (untreated). The work was restricted to medium and deep cavities—10 of each for each agent.

The agents tested, and the comparative results in terms of "percent of sterilizing value," were these: "Phenolate" (mixture of isomeric chlorophenolates), 85; metaphen (0.2 percent aqueous solution), 65; hexylresorcinol (solution S.T. 37), 60; zephiran, 45; beechwood creosote (U.S.P.), 35; 70 percent ethyl alcohol, 15; 10 percent sodium carbonate, 10. "Phenolate," which gave the best results, is "a new drug, heretofore not used for therapeutic purposes."

(4) *Grantee:* Maurice L. Tainter (1940-42)

College of Physicians and Surgeons, San Francisco, Calif.

Preliminary reports (11–12): 11) Sidney Epstein, D.D.S., and M. L. Tainter, M.D.—Study of the abrasiveness of dentifrice ingredients: *J. Den. Res.*, *20*, 273-4; 1941, June. [Proc. Int. Assoc. Den. Res., Mar.]

12) M. L. Tainter, M.D., and Sidney Epstein, D.D.S.—The abrasiveness of dentifrices: *J. Den. Res.*, *22*, 202; 1943, June. [Proc. Int. Assoc. Den. Res., Mar.]

Papers (13–18): 13) Maurice L. Tainter, M.D., and Sidney Epstein, D.D.S.—A standard procedure for determining abrasion by dentifrices: *J. Am. Col. Den.*, *9*, 353-79; 1942, Dec.

A brush-procedure and special apparatus were fully described, and their accuracy and reliability under carefully standardized conditions shown by extensive experimental data. This study indicates ways and means to test the abrasiveness of dentifrice ingredients, and to determine various factors affecting degree of abrasion, under reproducible conditions not heretofore available.

Tested in the presence of a constant lubricant consisting of 50 percent aqueous solution of glycerine, about half the abrasion (of teeth) obtained with precipitated (U.S.P.) calcium carbonate (adopted as a standard of reference) was due to the carbonate itself and the rest to the bristles of the brush. The abrasion with a toothbrush having bristles of synthetic plastic material was less than with a toothbrush having natural bristles, although the synthetic bristles were much more resistant to wear.

Dentin was abraded approximately 25 times faster than the enamel tips of the cusps of the mandibular molars tested; cementum, about 35 times faster. [See paper (14).]

14) Maurice L. Tainter, M.D., Sidney Epstein, D.D.S., and Alexander Klein, M.S.—The determination of particle sizes in dentifrice powders: *J. Am. Col. Den.*, *10*, 23-47; 1943, Mar.

In extension of the study described in the preceding paper (13), the authors determined the particle-size distributions of 27 powders used in dentifrices. From these distributions were calculated the median and mean diameters, the diameters of the particles of mean volume, and the surface areas per cubic centimeter and per gram of each powder.

In this work the microscopic method and Klein's "surface-scale hydrometer" technic were compared. The microscope gave lower proportions for the finer particles, and resultant higher mean diameters and lesser surface-area figures; the hydrometer gave higher proportions for the finer particles, and resultant data for lower mean volumes and greater surface areas. These differences were due in part to inherent limitations of each method: the microscope permitted measurement of only two of the three diameters and was unable effectively to resolve the finest particles; the hydrometer measured not the true size of particles but, instead, the diameters of spheres which would sediment at the same rate as the particles in the powder.

A survey of other available methods to estimate particle-size distribution showed that, while possibly advantageous for special purposes, they are not more generally useful or applicable than the two methods used in this research. [See papers (15–18).]

15) Sidney Epstein, D.D.S., and M. L. Tainter, M.D.—Abrasion of teeth by commercial dentifrices: *J. Am. Den. Assoc.*, *30*, 1036-45; 1943, July.

By standardized procedures based on the findings in the earlier studies (13, 14), 60 commercial dentifrices (powders, pastes, liquids) were tested as to abrasive action on human teeth (enamel). The least abrasion by pastes and powders was about half that by the control calcium carbonate; the most abrasion, about twice that by

the control. When the toothbrush moistened with equal parts of water and glycerine was used alone, the abrasion was reduced to about two-thirds of that caused by brushing with calcium carbonate powder moistened with the same liquid. Five liquid dentifrices caused very little abrasion, ranging down to as low as one-sixth that of the control calcium carbonate. Since this was less than that induced by the brush alone, when wet with glycerine, the diminished abrasion resulted from lubrication by the sulfonated detergent and gums used as substitutes for soap in these liquid preparations. Household brands of table salt and baking soda have abrasion efficiency indistinguishable from that of the specially formulated dentifrices, this fact giving support to previous claims that these simple materials may serve adequately as economical dentifrices.

The ideal degree of abrasion required of dentifrices has never been determined. The liquids cause less abrasion than the powders or pastes, but, in many persons, fail to abrade sufficiently for proper cleaning. Although powders and pastes have in general the same range of abrasiveness, the most abrasive are approximately four times as active as the least effective, affording a wide range for selection. The least abrasive solid preparations have substantially the same abrasive power as most liquid dentifrices; therefore, if minimal abrasive effect is desired, a properly selected paste or powder can be used as safely as most liquid dentifrices.

16) Sidney Epstein, D.D.S., and M. L. Tainter, M.D.—The relationship of particle size and other properties of dentifrice ingredients to toothbrush-abrasion of enamel: *J. Den. Res.*, *22*, 335-44; 1943, Aug.

The paper presents results on the correlation of the sizes of the particles in, and abrasiveness of, dentifrice powders; also the influence of H-ion concentration and of hardness on abrasion. The carefully controlled standardized method described in an earlier paper (13) was used—72 measurements in each test of the wear on the enamel cusps of 18 human mandibular molars after 200,000 strokes (calcium carbonate control).

The best measure of abrasive power was comparison of the slopes of the curves relating their *median* particle-size to ascertained abra-

sion. Comparison of the relative abrasiveness of any two dentifrice powders, even at the same particle-size, gives ratios that do not hold for any other particle-size unless the curves are parallel. According to this criterion, dicalcium phosphate (7 samples) was about one-sixth as abrasive as precipitated calcium carbonate (10 samples). Limited data are presented relating the abrasiveness of fourteen other powders to their particle-sizes, H-ion concentrations, and Moh hardness numbers. The data indicate that the Moh hardness number gives little information as to prospective abrasion of enamel for substances softer than enamel. Pumice and silica—Moh numbers higher than for enamel—were very abrasive.

Among the unsolved problems of abrasion deserving extensive study are: influence of pressure, type of bristle in the brush, differences produced by lubrication, critical level of H-ion concentration, optimum degree of abrasion for satisfactory dental hygiene. Also, should the desirability of a dentifrice be judged by its effects on enamel, or on dentin and cementum?

17) Sidney Epstein, D.D.S., and M. L. Tainter, M.D.—Glass slide procedure for detecting undue abrasion of dentifrices: *J. Am. Den. Assoc.*, *30*, 1590-4; 1943, Oct.

A critical analysis with many dentifrices has shown that if glass microscope slides are abraded with a 5-cent coin for 200 strokes, under a steady pressure of 1,500 grams, there is a significant correlation of +0.40 between the amount of abrasion—by the dentifrice materials—of enamel of human molar teeth and the degree of scratching of the glass. For this test, the flat surface of the coin is used and the powders are moistened into thick slurries by adding small amounts of 50 percent glycerine solution in water, while the pastes and liquids are used undiluted. But the curve relating scratching of the slides to abrasion on teeth is so flat, and the scatter of the individual results is so great, that it is impractical to use the glass-slide procedure to determine the absolute or relative abrasive power on human teeth of an individual material.

The glass-slide procedure is useful mainly for the detection of the presence of traces of highly abrasive materials. Thus the pres-

ence of 0.0156 percent of flour of pumice, or 0.0037 percent of a "240-mesh" silica, in precipitated calcium carbonate, may be detected through increased scratches on the slide. This test is useful to help to identify dentifrices that might scratch the enamel. [See paper (18).]

18) M. L. Tainter and Sidney Epstein—Use of metal plates for testing the abrasiveness of dentifrices: *J. Den. Res.*, *22*, 381-9; 1943, Oct.

The abrasion, by a series of dentifrice ingredients and commercial dentifrices, of plates of copper, silver and antimony (loss of weight after 20,000 strokes), was compared with the effects on the enamel of human teeth in the special abrasion-testing machine previously described (13). There was no correlation between the amount of abrasion of any of these metal plates and that of the enamel. The results showed that these metal plates cannot be used as test objects to establish the degree of abrasiveness of dentifrices for human enamel.

The metal plates can detect the presence of small amounts of a highly abrasive material, such as emery dust, in precipitated calcium carbonate, and hence might be used in qualitative tests for the presence of undesirable constituents, as in the case of the glass-slide procedure indicated in the preceding paper (17).

(5) *Grantee:* James Nuckolls (1940-41 and 1942-43)
University of California, San Francisco, Calif.

Preliminary reports (19–24): 19) James Nuckolls—I. Lobular development and calcification in the tooth: *J. Calif. State Den. Assoc.*, *17*, 73-5; 1941, May-June. II. Photomicrographs: *Ibid.*, *17*, 105-6; 1941, July-Aug.

20) James Nuckolls, D.D.S.—Primary centers of lobular development and calcification in the tooth: *J. Den. Res.*, *20*, 270; 1941, June. [Proc. Int. Assoc. Den. Res., Mar.]

21) James Nuckolls, D.D.S., J. B. deC. M. Saunders, M.B., Ch.B., F.R.C.S. (Ed.), and Harry E. Frisbie, D.D.S.—Further observations concerned with amelogenesis and calcification in the first molar of the rat: *J. Den. Res.*, *22*, 210; 1943, June. [Proc. Int. Assoc. Den. Res., Mar.]

22) H. E. Frisbie, D.D.S., and James Nuckolls, D.D.S.—Dentinogenesis: histologic study of the development of the dentin in the cat: *J. Den. Res.*, *22*, 221-2; 1943, June. [Proc. Int. Assoc. Den. Res., Mar.]

23) H. E. Frisbie, D.D.S., and James Nuckolls, D.D.S.—Distribution of the organic matrix in adult human teeth and its role in caries: *J. Den. Res.*, *23*, 215; 1944, June. [Proc. Int. Assoc. Den. Res., Mar.]

24) James Nuckolls, D.D.S., J. B. deC. M. Saunders, M.B., Ch.B., F.R.C.S. (Ed.), and Harry E. Frisbie, D.D.S.—Histologic study of the development of epithelial attachment and the formation of secondary cuticle in rat first molars. [This paper was listed on the printed program of the meeting of the International Association for Dental Research (March, 1944), where it was read by title; but, owing to a mishap, the abstract was not included in the published Proceedings.]

Papers (25–26): 25) J. B. deC. M. Saunders, M.B., Ch.B., F.R.C.S. (Ed.), James Nuckolls, D.D.S., and Harry E. Frisbie, D.D.S.—Amelogenesis: A histologic study of the development, formation and calcification of the enamel in the molar tooth of the rat: *J. Am. Col. Den.*, *9*, 107-36; 1942, June.

With the aid of "more delicate, precise and direct techniques" than any heretofore employed—and by avoidance of acid decalcifications—the authors studied some aspects of the "development, formation and calcification of the enamel," one of the most "confused and controversial field[s] in the whole realm of dental histology." Their technique favored not only the positive staining *in situ* of the calcium complex, but also provided histological material that revealed details of the mode and development of enamel in the first molar—from birth of the rat to eruption of the tooth.

The internal enamel epithelium, just before establishment of the enamel, differentiates into two types of cells: ameloblast or ganoblast, and kionoblast.[5] The latter initiates enamel formation by establishment of the dentino-enamel membrane, and may give rise to enamel tufts, lamellae and spindles. The earliest evidence of cellular activity in the ameloblast is increasing granulation of the cytoplasm, followed by development of a large terminal vacuole—the future Tomes process. As the ameloblast approaches full functional activity, basal granules appear in the cytoplasm.

Immediately after establishment of the dentino-enamel mem-

[5]This new term, applied by the authors to a cell they believe has not hitherto been clearly recognized, is explained by them as follows in a footnote: "Kionoblast from the Greek Κίων (σνος) a pillar and βλαστός a germ."

brane and the terminal vacuole, the formal elements of the enamel matrix arise as delicate parallel processes extending peripherally on either side of the cell, to merge by their apices with the cell membrane of the ameloblast. The development of these structures, which the authors term *"pre-enamel processes,"* converts the distal end of the ameloblast into a hollow cylinder having walls that taper somewhat proximo-distally. The central core of the more proximal part of the cylinder is occupied by the terminal vacuole and constitutes the Tomes process. The terminal vacuole is studded with minute, highly refractile, spherical granules, which stain specifically for keratohyaline and should not be confused with the so-called "calcospherites." The authors regard these granules as "specific secretion granules of the ameloblast from which the enamel processes and the enamel matrix are derived." Soon after formation of the pre-enamel processes, the body of the ameloblast, beyond the terminal vacuole, undergoes hyalinization to form a solid cylinder. Progressive hyalinization of the distal part of the cell establishes the organic structure of the enamel rod. The so-called terminal-bar apparatus—never observed where fixation was good and shrinkage absent—is believed to be "entirely an artifact."

Extensions of the cell protoplasm in the region of the terminal vacuole into the core of the pre-enamel rod constitute the well known Tomes processes. If acid decalcification is severe enough to cause partial dissolution of the enamel matrix, the processes rupture and produce conical projections, which frequently have been illustrated as Tomes processes, although they are artificial, caused by shrinkage and rupture. In preparations in which decalcification was conducted under microscopic control to expose the matrix, the cell membrane of the ameloblast was traced throughout the entire thickness of enamel. The enamel matrix undergoes progressive alterations in tinctorial reactions with age, the strongly basophilic enamel becoming more and more eosinophile and, when fully mature, almost chromophobe. Reduction of the ameloblast is associated with changes indicative of more generalized hyalinization of the cell. The pre-enamel processes are shortened and may not be present as such; the matrix is deposited more uniformly. Unusual numbers of secretion

granules within secondary vacuoles fuse to form so-called "calcospherites," which constantly exhibit tinctorial reactions identical with those of the pre-enamel processes and early matrix; composed of eleidin, they are termed "*hyaline or eleidin globules.*" In the final stage of matrix formation, hyalinization extends throughout the remainder of the reduced cell. Pre-enamel and Tomes processes are absent; the nuclei become pyknotic and are incorporated into the final layer. Hyalinization may extend to the stratum intermedium.

Calcification of enamel matrix begins at the dentino-enamel membrane as soon as this structure is established. The calcium density of this layer increases until eruption, and exceeds that of the rest of the enamel and of the dentin. Calcification of the enamel rod and interprismatic substance commences as soon as the central core begins to undergo hyalinization. Minute calcific crystals are deposited first on the lateral walls of the hyaline rod, the deposition extending peripherally as far as the base of the pre-enamel processes. The core of the rod calcifies a little later than the walls. The calcium density of the core is never so great as that of the margin of the rod. Calcification of interprismatic substance begins slightly behind the limit of the newly calcified rod. The deposition in the rod and interprismatic intervals is finely granular, the granules fusing to form large particles. Calcification progresses from the dentino-enamel membrane toward the surface, gradually increasing in density as the enamel matures. After staining with silver salts, the tinctorial reactions of the ameloblast indicate that the calcium of enamel is derived from the tissue juices of the surface cells.

Enamel is derived from, and is a modified form of, ectoderm. Modification of skin into enamel organ is a far less radical change than is suspected. The ameloblast has preserved its essential and primitive function as a keratin-producing cell; its product undergoes a slight chemical or physico-chemical modification (nature unknown), from which it derives its affinity for the calcium complex. The progressive changes from the strong basophilia of the pre-enamel processes which do not calcify, to the progressive acidophilia of the more fully formed enamel matrix which rapidly calcifies, are similar to the staining reactions of bone in the development of its organic

matrix. The changes suggest alterations in the pH related to the deposit of the inorganic salts, which is more in conformity with a humoral theory than of one of enamel secretion. [See paper (26).]

26) James Nuckolls, D.D.S., J. B. deC. M. Saunders, M.B., Ch.B., F.R.C.S. (Ed.), and Harry E. Frisbie, D.D.S.—Amelogenesis: A further study of the development of Tomes' process and the enamel rod matrix in the molar and incisor teeth of the rat: *J. Am. Col. Den.*, *10*, 241-68; 1943, Dec.

A continuation and extension of the preceding study (25). The development of the Tomes process, and of the enamel matrix in undecalcified first molars and incisors (rat), were followed from birth to three days thereafter. The closest attention was given to problems of fixation and staining; comparisons were made of effects of various agents in both decalcified and undecalcified materials.

Initial development of the Tomes process is signalized by the appearance of a vacuole—called "terminal vacuole"—filled with clear homogeneous material containing minute refractile bodies. With increasing maturity of the ameloblast, the Tomes process develops proximal and distal portions. The *proximal* portion, in direct succession to the cell, is virtually a single vacuole, its content being similar to that of early development of the terminal vacuole, which seems to arise by confluence of small vacuoles at the distal extremity of the cell. The *distal* portion of the Tomes process is conical and hyalinized, and its apex projects to a variable extent into the core of the enamel rod. The proximal portion of the Tomes process is bounded laterally by pre-enamel processes which, more refractile than the body of the Tomes process, are slender structures, their apices terminating just short of the distal end of the cell; their bases blend and are continuous with the lateral walls of the rod; as they mature, they thicken and give rise to rod cortex.

The developing rod consists of the rod cortex, rod core, and rod membrane. The *cortex* results from continuation of the progressive change first observed in the pre-enamel processes, and is the first portion of the rod to undergo complete maturation and also calcification. The *core* is the less completely hyalinized inner part of the rod, and seems to be formed by continued hyalinization of the Tomes

process. In some rods interrupted areas of incompletely hyalinized material, having staining reactions identical with those of the distal portion of the Tomes process, occur deeply within the core. In extreme examples, the entire rod is apparently immature, and the distal part of the Tomes process may extend almost to the end of the rod. Surrounding the hyaline rod—therefore external to the cortex—a *membrane* can be selectively stained. This membrane is assumed to be a thickened continuation of the cell membrane of the ameloblast. The hyalinized rods seem to be connected by intercellular substance having staining reactions, suggesting that it consists of material similar to that of the enamel-rod matrix. The origin of the interprismatic substance could not be determined, but appearances suggest that it may have arisen in the region of the proximal part of the Tomes and pre-enamel processes.

"Terminal bars" are found in the intercellular intervals at the level of the junction of the distal end of the ameloblast and the base of the Tomes process. A second system of terminal bars, less in degree, may occur in the direction of the developing rod. These appear as differentially staining globules at the periphery of the rod. The authors, in harmony with the deductions presented in the preceding paper (25), have again found the terminal-bar apparatus to be an artifact. Further study has shown that terminal bars are revealed, but less numerously, when undecalcified material is treated with acid stains; also that these structures appear, in most exaggerated form, as heavy discrete knots, when the material has been subjected to the severe action of acids and precipitating fixatives. The appearance of segmentation may be produced not only in the rod and distal hyalinizing portion of the Tomes process, but also in the process itself and in the body of the cell well above the level of the so-called terminal bars. In the opinion of the authors, these appearances are related to the effects of precipitating fixatives that grossly distort and exaggerate the normal process, and therefore are false.

(6) *Grantee:* William J. Furuta (1940-41)
University of Illinois (College of Dentistry), Chicago, Ill.

Preliminary report: 27) Murray M. Hoffman, B.S., D.D.S., M.S., Cecilia

Schuck, Ph.D., and William J. Furuta, A.B., M.S.—Histologic effects on rat teeth by fluorine administered in dry and moist diets: *J. Den. Res.*, *20*, 276-7; 1941, June. [Proc. Int. Assoc. Den. Res., Mar.]

Paper: 28) Murray M. Hoffman, Cecilia Schuck and William J. Furuta—Histological study on the effects of fluorine administered in dry and moist diets on teeth of young albino rats: *J. Den. Res.*, *21*, 157-70; 1942, Apr.

Gross and histologic changes in teeth of young rats on diets containing added sodium fluoride were noted. Pigment changes in incisors, and ground sections of incisors and molars, indicated that (a) animals that had a daily fluoride intake of 0.05 percent of the diet, given in the drinking water, were most severely affected; (b) those receiving this amount of fluoride in milk were less affected; (c) those to which this amount of fluoride was given in the dry portion of the diet were least affected. Since the basic diets were adequately controlled, and the daily intake of fluoride was nearly uniform, the observed variation in toxicity was ascribed to "difference of absorption of the mineral [fluoride] in the gastro-intestinal tract."

The histological changes, in each of the three groups, showed degeneration of ameloblasts with enamel hypoplasia and cyst formations, and accentuation of the incremental lines in both dentin and enamel, the rate of incrementation remaining within the normal (approximately 16 microns). There were rachitic-like changes in the dentin of incisors and molars (interglobular dentin). Excessive osteoid occurred in the alveolar bones. Eruption of third molars was retarded. There was persistence of enamel organic matrix in incisors and molars, possibly associated with poor mineralization—calcification disturbed by the absorbed fluoride. Referring to fluoride as a currently recommended preventive of caries, the authors say:

"The apparent toxicity of fluorine and the ensuing pathological changes when administered, strongly suggest that very careful investigation and rationalization of findings be borne in mind to offset the misfortune that often follows the publications on 'cure alls'."

(7) *Grantee:* Wallace D. Armstrong (1941-44)
University of Minnesota, Minneapolis, Minn.

Preliminary reports (29–30): 29) W. D. Armstrong, Ph.D., M.D., and

M. Knowlton, B.S.—Fluorine derived from food: *J. Den. Res.*, *21*, 326; 1942, June. [Proc. Int. Assoc. Den. Res., Mar.]

30) W. D. Armstrong, Ph.D., M.D., and J. W. Knutson, D.D.S., Dr.P.H.—Bacteriostatic effects of quinones: *J. Den. Res.*, *22*, 214; 1943, June. [Proc. Int. Assoc. Den. Res., Mar.]

Papers (31–32): 31) W. D. Armstrong and J. W. Knutson—Effect of quinones on acid formation in saliva: *Proc. Soc. Exp. Biol. Med.*, *52*, 307-10; 1943, April.

Previous observers noted that small amounts of *synthetic vitamin K* (2-methyl-1,4-naphthoquinone) prevented production of acid in incubated (4 hours) saliva-glucose mixtures (saliva plus 10 percent of its weight of glucose), and suggested that this vitamin might be used to prevent dental caries. The authors found, by procedures described in detail, that the observed antibacterial effect was due to the quinone structure of synthetic vitamin K; that the same result was obtained in variable degrees with other quinones (thirteen studied); that the antibacterial property of the compound is unrelated to the "vitamin-K activity"—that its inhibitory effect on acid formation by oral microörganisms is *incidental* to its properties as a vitamin. Illustrative quotations follow:

"The data . . . indicate that while 1 mg. of 2-methyl-1,4-naphthoquinone per 100 cc. [of] saliva effectively decreases acid formation to a degree below that required for decalcification of tooth structures, a concentration of upwards of 3 mg. per 100 cc. is required for complete inhibition of acid formation.

"Under the conditions of these experiments . . . 1,4-naphthoquinone has an antibacterial effect somewhat superior to that of 2-methyl-1,4-naphthoquinone; yet the first-named substance is reported to exhibit not more than 10 percent [of] the vitamin-K potency of the second. Furthermore, 1,2-naphthoquinone and benzoquinone, whose vitamin-K activities are negligible, are shown to possess in substantial degree an ability to interfere with acid formation from glucose by salivary organisms.

"It might be supposed that there could exist a relationship between the oxidation-reduction potentials of quinones and their relative effectiveness as bacteriostatic agents . . . [but the data suggest the probability] that some character of quinones other than their oxidation-reduction potentials is also determinant of their antibacterial properties.

"We found [in disagreement with previous observers] that 3 and 5 mg.

of . . . [2-methyl-1,4-naphthoquinone] per 100 [cc. of] saliva-glucose mixture, and corresponding amounts of benzoquinone, caused a progressive decrease in the *L. acidophilus* counts in samples withdrawn at hourly intervals over a 4-hour incubation period. The final count was reduced to about one-third the original number of organisms while that of untreated controls increased by about 30 percent." [See paper (32).]

32) W. D. Armstrong, Wesley W. Spink and Jeanne Kahnke—Antibacterial effects of quinones: *Proc. Soc. Exp. Biol. Med.*, *53*, 230-4; 1943, June.

There has been increasing evidence that the activity of some antibiotic substances is due to their quinone structure (31). The present paper contains quantitative data on the "required bacteriostatic and lethal concentrations" of some quinones against two species of Gram-positive pathogenic cocci: staphylococcus (one strain) and streptococcus (one strain). The bacteriological procedures were indicated in detail; the comparative results were presented in three tables. Illustrative quotations follow:

"In Table I is shown the minimum amounts of several [thirteen] quinones and [three] other substances required to inhibit the growth of staphylococcus for 24 hours and for 48 hours, and the quantities of the same substances required to kill the organisms present in the original inoculum . . . 2-methyl-1,4-naphthoquinone equals, and the other [six] naphthoquinones approach, the ability of 2,6-dimethoxy-benzoquinone to inhibit the growth or to kill the strain of staphylococcus used in these experiments.

"The data [Table II] . . . permit the calculation that the penicillin was approximately 13 to 18 times as effective, on a weight basis, as 2-methyl-1,4-naphthoquinone or 2,6-dimethoxy-benzoquinone . . . the two most potent quinones." [Impurities in the penicillin led the authors to conclude that the antibacterial activity of *pure* penicillin is much greater.]

"The results in Table III [for six compounds, including five quinones, indicate] . . . that several of these substances appear to exert a considerably greater antibacterial action against the . . . streptococcus than . . . [against] the staphylococcus. None of [these substances] . . . in a concentration as high as 29×10^{-6} mols per 100 cc. exerted any bacteriostatic or bactericidal effect against a strain of *Escherichia coli*."

(8) *Grantee:* Michael S. Bales (1942-43)
Tufts College Dental School, Boston, Mass.

Preliminary report: 33) M. P. Sheldon, B.S., D.M.D., and M. S. Bales, B.S., D.M.D.—Correlation between formative defects in enamel and sys-

temic debilities: *J. Den. Res.*, *23*, 220; 1944, June. [Proc. Int. Assoc. Den. Res., Mar.]

Paper: Research completed; paper to be published during the ensuing fiscal year (1944-45).

III. GENERAL SUMMARY OF MAIN PUBLISHED FINDINGS

Caries: fluorine; dental effects of toxic doses (27–28).[6] Sodium fluoride, administered in large doses equal to 0.05 percent (500 parts per million) of the daily diet, was most toxic when added to the drinking water; less so when taken in milk; least poisonous when swallowed in dry portions of the food. The inequalities were attributed to differences in absorption from the gastro-intestinal tract. Histological changes in teeth in each of the three groups of rats, under the conditions of this excessive dosage, included degeneration of ameloblasts with enamel hypoplasia and cyst formations; rachitic-like changes in dentin; excessive osteoid in alveolar bones; and delayed eruption of third molars. The authors suggest "very careful investigation and rationalization of findings" before acceptance of claims that fluorine is a desirable preventive of caries.

Caries: inheritance; important factor (1–3). Inheritance is an important factor in susceptibility to caries in rats. Results of experiments in continuous progress since 1937—in which susceptible and resistant lines have been obtained—indicate that, by selection of individuals, progeny testing, and close inbreeding, a caries-immune strain may be evolved.

Caries: pantothenic acid; relationship (4–8). In selected patients having a variety of deficiency diseases (vitamin-B group) there were low incidence of caries and correspondingly small numbers of oral lactobacilli. These conditions were not due to salivary deficiency of pantothenic acid, which—essential for growth of lactobacilli—was present in adequate concentrations in salivas of each of these patients.

Caries: recurrence; under fillings (9–10). Continuance of bacteria for at least one year in the dentin—"after proper cavity preparation and filling," including use of accredited agents for cavity sterilization—indicates that recurrence of decay under fillings is due

[6]The numerals in parenthesis, after marginal headings, refer the reader to the corresponding units in the preceding bibliography.

(when not a result of leakage) to inadequate sterilization of the dentin before insertion of the fillings. The most effective antibacterial agent of those tested (all inadequate) was phenolate, a "new drug." Hermetic sealing of a cavity does not assure sterilization of the dentin.

Caries: vitamin K; supposed preventive (30–32). The action of synthetic vitamin K (2-methyl-1,4-naphthoquinone), in preventing production of acid in incubated saliva-glucose mixtures, led previous observers to suggest that this vitamin might be a preventive of caries. The authors found that this antibiotic effect on oral lactobacilli was due to the quinone structure of the vitamin; was obtained by other quinones having little or no vitamin-K activity; and was only incidental to this activity. Tests of the relative bacteriostatic effects of a group of quinone compounds, including synthetic vitamin K, indicated that in this respect all were inferior to penicillin.

Dentifrices: abrasiveness; methods and results (11–18). Methods to determine abrasiveness were subjected to thorough comparative studies, the results described, and the most effective procedures used to obtain findings for dentifrices. The relative degrees of abrasiveness of many commercial dentifrices having been determined and recorded, the authors concluded that "household brands of table salt and baking soda have abrasion efficiency indistinguishable from that of the specially formulated dentifrices"—support for "previous claims that these simple materials may serve adequately as economical dentifrices." When *minimal* abrasive effect is desired, "a properly selected paste or powder can be used as safely as most liquid dentifrices."

Enamel: amelogenesis; and other processes in development of teeth (19–26). With the aid of more delicate, precise and direct techniques than any previously employed—and by avoidance of acid decalcification—some aspects of the development, formation, and calcification of enamel were studied, new views were presented, and revisions of old opinions were indicated, in one of the most confused and controversial fields in dental histology. The abstracts (25–26) are, in effect, general summaries of the many findings described in the detailed papers.

EDITORIAL

DENTAL RESEARCH: POST-WAR PLANNING[1]

The dental profession possesses an intimate knowledge of the substance, extent and solidarity of its research program, its attainments, the recognition it has been accorded, and its future potentialities. We must now beware lest our leadership becomes self-satisfied and falls in line with the "easy way," resting on past achievements as a guaranty of continued interest and support. Our leaders in research are indeed too wise and too energetic to ignore the fact that research, like everything that lives and seeks to grow, must face the problem of adjusting itself to an everchanging environment. Especially should this philosophy be our stimulus and our guide in the midst of situations that arise and conditions that are revealed in a nation at war.

To meet the requirements of the immediate future, it is necessary to set up a well-planned effort toward clearly-defined objectives under capable direction, so that we shall be prepared to move forward even more forcefully, continuously and effectively. Otherwise, our attitude, our ideals, and the commonsense of our aims and purposes are likely to be obscured or quenched in dismal failure. The main objective at present, as at all times, is to awaken and keep awake the spirit of scientific research, and so to vitalize our efforts that we shall slight no professional obligation and neglect none of dentistry's opportunities, whenever and wherever they may arise.

There are at least three items which appear essential to growth in extent, content and quality, which should be ever in our minds as we develop our post-war program of research: (1) an appraisal of the present status of dental research, so that we may find ways and means to publicize the true values of dental and oral health-service, (2) the attainment of an effective medico-dental relationship, and (3) funds for the promotion of research.

[1]Submitted as a report at the Regents' meeting in Cincinnati, Ohio, Oct., 1943, but altered slightly for this editorial.

At the present moment, extended discussion of the status of dental research seems neither appropriate nor necessary. We should, however, appraise what we have done and what we possess if we are to use their influences to good purpose in the attainment of our objectives. However, brief reference is made to this item in the discussion of funds for the promotion of research.

While dentistry is admittedly a specially-equipped health-service profession, it cannot attain its widest degree of usefulness because some of the problems of common interest and concern to dentistry and medicine still remain unsolved. If the healthy functioning of the human organism depends upon the effective care of all its parts, and if an alliance between dentistry and medicine will promote the highest degree of well-being for both medical and dental patients, then it logically follows that a completely satisfactory program of dental research is not attainable while the interrelated problems of dentistry and medicine remain unsolved. Consequently, the medico-dental relationship is not only a professional relationship but an essentially human relationship. High purpose, unified effort and a dauntless will to succeed have made possible a splendid growth in the field of dental research. In cooperative research with medicine, dentistry certainly has something to give as well as to receive.

The widespread attention which has been bestowed upon dentistry in recognition of its important functions, possibilities and attainments, especially during the past ten or fifteen years, indicates that there are an increasing number of philanthropic agencies and individuals who may be willing to lend a sympathetic ear to those who seek funds for the solution of dentistry's many problems. A striking instance of this philanthropic trend was the gift of $1,300,000 to Harvard University for an experiment which soon proved as hopeless as it was conspicuous and untimely. The publicity given to the initiation and the abandonment of the Harvard plan should be of very definite help to those representatives of organized dentistry who have opportunities for enlightening potential benefactors upon the real needs, aims and values of dental education and dental research, and for emphasizing dentistry's proved efficiency in oral

health-service. Progressive advancement in education, in programs of scientific investigation, and in consciousness of public responsibility, is well documented in our professional literature; so that an appraisal of what we have done can readily be made for the benefit of interested philanthropic laymen. They can be shown convincingly that funds intended to finance prevention of dental diseases will be most effectively employed if they are given directly to dental education.

To well-informed observers, the most recent growth of dentistry has placed the profession in a position where it is admired and trusted; and the subtle scheme of combining complimentary allusions to the supremacy of American dentistry in manual and digital activities with derogatory insinuations that the biological laws are ignored in its ministrations, can no longer succeed, save only, in the dark confines of ignorance and prejudice. It is our task to develop intensive effort and sustained action to the end that detractors may be silenced and benefactors may be deeply and permanently impressed with dentistry's needs and just deserts. When this has been well begun, continuous publicity for dentistry's aims and projects should bring us the contributions and endowments which we require. So I say, appraise what we have done, set forth what we hope to do, and do not forget that cooperation with medicine is a necessary part of our program if we are to impress benefactors and proceed vigorously with our research.

A. L. M.

JOURNAL OF DENTAL RESEARCH

WILLIAM J. GIES ENDOWMENT FUND

Financial Statement as of May 15, 1944

Compiled by LEUMAN M. WAUGH, D.D.S., *Treasurer*[1]

This Fund was created, on the initiative of the voluntary Committee on Endowment—organized in New York City in 1937—to insure the continuance of the *Journal of Dental Research* "in full accord with the highest ideals of strictly professional journalism devoted to the advancement of research," to which the *Journal* was dedicated at its establishment in 1919.

I. AMOUNTS RECEIVED FROM ORGANIZATIONS

(March 12, 1943, to May 15, 1944)

*Alpha Omega Fraternity	$ 25.00	
*American College of Dentists—completes pledge of $5,000	1,000.00	
American College of Dentists—		
Kentucky Section	170.00	
Kentucky Section—given by J. H. Springsted	5.00	
*Atlanta (Georgia) Dental Society	25.00	
Baylor University, College of Dentistry—given by P. Knutzen, Acting Dean	15.00	
Eastman Dental Dispensary, Inc.	300.00	
International Association for Dental Research—		
American Division	192.45	
Minnesota Section	25.00	
*Nevada State Dental Association	15.00	
*Ohio State Dental Society—annual payments for 1942-43	120.00	
Omicron Kappa Upsilon—		
Rho Chapter	200.00	
*Xi Chapter	25.00	
Peoria (Illinois) District Dental Society	25.00	
Psi Omega Fraternity, Phi-Rho Chapter	10.00	
*San Francisco (California) District Dental Society	25.00	
*Wisconsin State Dental Society	165.00	
		$2,342.45

*Additional contribution.

[1]A financial statement as of March 17, 1944, was published in *J. Den. Res.*, *23*, 226; 1944, June.

II. AMOUNTS RECEIVED FROM INDIVIDUALS

(March 12, 1943, to May 15, 1944)

$200.00:	*In memoriam: John and Ophelia E. Gies, Reisterstown, Maryland, 1872; (John Gies III, William J. Gies, II)—completes pledge of $1,000	$ 200.00
180.00:	*Arthur H. Merritt	180.00
100.00:	Captain James T. Gies, Captain Robert H. Gies, I. Hirschfeld, *Leuman M. Waugh, and one contribution from the following group: Drs. Louie T. Austin, Boyd S. Gardner, Stanley A. Lovestedt, Jerry A. Millhon, Edward C. Stafne—all of the Section of Dental Surgery, Mayo Clinic, Rochester, Minnesota (5 x $100)	500.00
75.00:	R. J. Rinehart, *Simon Shapiro (2 x $75)	150.00
25.00:	Ernest N. Bach, M. K. Baklor, John J. Fitz-Gibbon, Charles W. Freeman, *H. D. Grubb, M. M. House, Malvern D. Huff, *Arthur S. Litten, L. D. Pankey, Marcus L. Ward, Maurice William (11 x $25)	275.00
20.00:	J. F. Cart, Clarke E. Chamberlain, Charles D. Hermon, W. F. Lasby, *Albert L. Midgley, W. S. Peters, *C. M. Smith, *Charles I. Taggart, R. C. Willett (9 x $20)	180.00
18.50:	*Joseph E. Psayala (War Bond—Sec. VI)	18.50
15.00:	*George M. Anderson, John C. Black, *O. W. Brandhorst, L. C. Hemsworth, Otto B. Litweiller, P. V. McParland, Arthur Miller, E. Milloversum, C. M. Taylor (9 x $15)	135.00
10.00:	Edward L. Ball, Charles M. Barnwell, A. H. Bassman, *Frank J. Bell, *I. B. Bender, Paul N. Bennett, Frank E. Beube, *Charles F. Bodecker, *David	

*Additional contribution.

W. Brock, *E. H. Bruening, George C. Brown, *A. W. Bryan, *H. L. Bunker, E. A. Charbonnel, *Oscar J. Chase, Jr., *George Wood Clapp, *Frank C. Cole, C. B. Coleman, J. V. Conzett, Ivan R. Cottrell, Wilbur McL. Davis, William R. Davis, Captain H. R. Delaney (D.C.), *Robert L. Dement, *M. M. Devan, Gerard A. Devlin, Meyer Eggnatz, *Samuel S. Eisner, E. O. Ellington, R. H. Fladeland, C. O. Flagstad, Willard C. Fleming, *C. G. Fletcher, Willard H. Foss, W. Talbot Foster, *Geneva E. Groth, *John E. Gurley, *G. Fred Hale, T. A. Hardgrove, L. A. Harker, James F. Henegan, J. D. Hertz, *Thomas J. Hill, W. N. Hodgkin, W. J. Hollingshead, *Conrad L. Inman, James T. Ivory, *Andrew F. Jackson, George B. Jersin, Ernest L. Johnson, Raymond E. Johnson, *Alfred W. Kany, W. N. Kelly, Paul C. Kitchin, Albert H. Kniesner, Alfred L. Kohn, Leonard Kohn, William Kress, *H. O. Lineberger, W. E. Lundy, W. J. B. Mason, *C. J. Masters, Fred E. Maxfield, *John Oppie McCall, John F. McParland, *Harry A. Mesjian, Sydney R. Miller, E. L. Mitchell, Waldo H. Mork, Emory W. Morris, L. R. Musser, *Ivor P. Muzzey, *Charles Nelson, *Walter T. Newton, *James Nuckolls, Drs. Bertha P. and Joseph T. O'Leary, J. W. Outlaw, E. B. Penn, Henry C. Petray, William R. Pond, *Lowrie J. Porter, Kyrle W. Preis, K. C. Pruden, Charles E. Rudolph, Robert Wm. Rule, Jr., *Major J. B. Schneer, *Clyde H. Schuyler, Benjamin Shapiro, Harry B. Shuman, W. K.

*Additional contribution.

	Slater, *Ernest G. Sloman, D. M. Small, Franklin A. Squires, *H. Hayward Streett, Charles A. Sweet, *R. Hamill D. Swing, W. C. Tannebring, *Isidore Teich, G. D. Timmons, Jerome H. Trier, *John E. Tyler, E. G. Van Valey, Ralph Waldron, Leonard T. Walsh, *C. R. Wells, Frederick T. West, Harry T. Wood, W. A. Wood (108 x $10)	$1,080.00	
9.45:	George P. J. Pritchard Contribution was $10.00, less commission and service charge.	9.45	
9.28:	Arthur L. Walsh Contribution was $10.60, less discount and service charge.	9.28	
5.00:	Harry Bear, *Reuben L. Blake, *Frank Carothers, *O. M. Davis, Joseph D. Eby, *Henry C. Fixott, Hannah Frankowitz, Guy R. Harrison, A. W. Haskell, *Abraham Lees, Harry Lyons, *Maurice Markowitz, Leon W. Marshall, A. L. McDonough, Leroy M. S. Miner, *Gerald Mitchell, P. S. Neuwirth, *Edith B. Oblatt, M. Webster Prince, Jacob Shapiro, William C. Stillson, *Gordon L. Teall (completes pledge of $10), J. W. Vogan, Leonard P. Wahl (24 x $5)	120.00	
4.50:	*F. A. Richmond (interest on bond)	4.50	
3.00:	*J. C. Curry	3.00	
2.00:	*Earl W. Pellien	2.00	
1.00:	*Zachary Blum	1.00	
	Contributions from organizations carried forward		$2,342.45
			$2,867.73
	Total contributions March 12, 1943 to May 15, 1944		$5,210.18
	Total cash receipts were	$5,191.68	
	One contribution was in form of a War Bond—see Section VI	18.50	

*Additional contribution.

III. INTEREST, INCOME AND EXPENSE

(Current year)

No expenses were recorded.

Interest to date, May 15, 1944, on War Bonds:

Number of Bonds	Denomination of Bonds	Date Purchased	Interest Accrued to Date		
10	$1,000.00	March 1942	$ 90.00		
22	1,000.00	May 1942	198.00		
8	1,000.00	July 1942	40.00		
2	1,000.00	Dec. 1942	4.00		
1	1,000.00	March 1943	2.00		
5	1,000.00	March 1944	.....		
2	1,000.00	May 1944	.....		
1	25.00	Feb. 1944	.....	$ 334.00	334.00

Total addition to the Fund as of May 15, 1944 ..... $5,544.18

IV. SUMMARY OF TOTAL RECEIPTS (ASSETS): 1937-44

Total receipts for the Fund, as of March 12, 1943		$32,732.99
Paid for 42 War Bonds	$31,080.00	
Balance on deposit	1,230.99	
Bonds and shares of stock—contributions from members previously reported	422.00	
Receipts in 1943-44:		
From organizations (Sec. I)	$ 2,342.45	
From individuals (Sec. II)	2,867.73	
		5,210.18
		$37,943.17
Payments for 50 War Bonds (Sec. VI)	$37,000.00	
Balance on deposit	502.67	
Bonds and shares of stock	440.50	

V. CUMULATIVE SUMMARY OF ANNUALLY RECORDED ITEMS OF RECEIPTS AND EXPENDITURES

Year	Contributions (Payments)	Earned Interest	Expense	Net Receipts	Cumulative Annual Net Receipts
1937-38	$10,652.50	None	$134.06	$10,518.44	$10,518.44
1938-39	4,748.17	None	None	4,748.17	15,266.61
1939-40	5,693.78	None	None	5,693.78	20,960.39
1940-41	5,190.55	$206.78	3.53	5,393.80	26,354.19
1941-42	4,166.35	333.95	None	4,500.30	30,854.49
1942-43	1,766.31	112.19	None	1,878.50	32,732.99
1943-44	5,210.18	334.00[2]	None	5,544.18	38,277.17
	$37,427.84	$986.92	$137.59	$38,277.17	

VI. STATEMENT REGARDING INVESTMENT IN WAR BONDS

By authorization of the Trustees of the Association, fifty War Bonds of Series F ("12-year appreciation bonds"), costing $740 each, have been purchased from the funds in the Endowment Committee's treasury, as of the following dates and serial numbers:

Purchased	(10) March 1942: M140400F to M140409F, incl.	$ 7,400
Purchased	(22) May 1942: M197811F to M197832F, incl.	16,280
Purchased	(8) July 1942: M237558F to M237565F, incl.	5,920
Purchased	(2) December 1942: M423990F and M423991F	1,480
Purchased	(1) March 1943: M204356F	740
Purchased	(5) March 1944: M750832F to M750836F, incl.	3,700
Purchased	(2) May 1944: M752375F and M752376F	1,480
		$37,000

The payee named on each bond is "Committee on the William J. Gies Endowment Fund for the Journal of Dental Research (an unincorporated body)." All of these War Bonds (and the Bethany Methodist Hospital Bond and Farnsworth Television and Radio Corporation Stock, indicated in Section IV) are in the custody of the Treasurer of the Committee, in a safety deposit box, Empire Trust Company, 580 Fifth Avenue, New York City, in trust for the Committee.

The contribution of $18.50 listed in Section II from Dr. Joseph E. Psayala was in the form of a $25 United States War Bond Series F ("12-year appre-

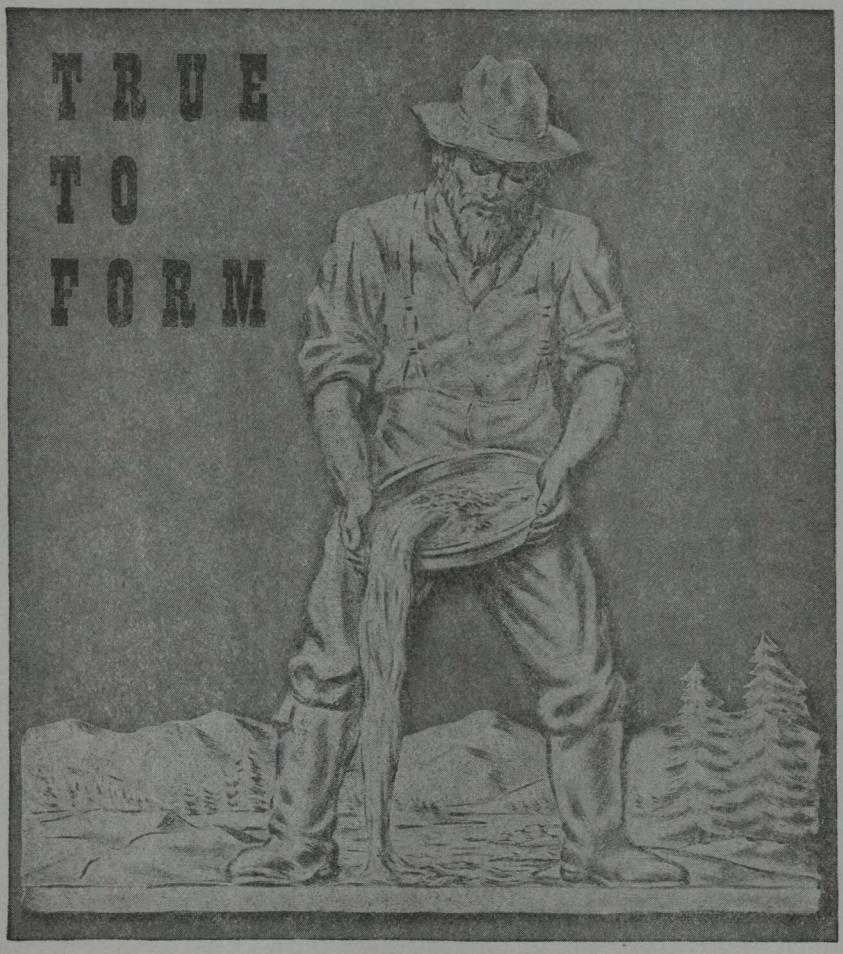
[2]Accrued interest on War Bonds; not available before maturity, or sale, of the bonds.

ciation bond") Q631670F, the payee being "William John Gies Endowment Fund for Journal of Dental Research." It is in the custody of the Treasurer in a safety deposit box, Empire Trust Company, 580 Fifth Avenue, New York City, in trust for the Committee. The bond was purchased in February, 1944.

VII. SUMMARY OF OPEN ACCOUNTS RELATING TO PLEDGES

A. Organizations

	Pledge	Paid	Balance	
International College of Dentists	$ 800	$200	$600	
Kansas: First District Dental Society	100	25	75	
J. B. Mann Study Club, Washington, D. C.	1,000	100	900	$1,575
B. Individuals				
Harry Kaplan	250	100	150	
George C. Paffenbarger	250	235	15	
R. J. Rinehart	200	75	125	290
				$1,865



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