SOME DETAILS IN THE HISTOPATHOLOGY OF THE ENAMEL.

(A lecture delivered before the Odontological Section of The Royal Society of Medicine, 24th April 1950.)

By

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The lecture touched upon some of the features of the histogenesis histology and pathology of the enamel, domains selected because the problem of the enamel seems to have retained international interest, and because the material has been acquired by means of a special uniform technique: namely, the study of the organic structure of the decalcified enamel. A brief account of the problems considered was given, illustrated by a series of colour photomicrographs.

I. Histogenesis, with particular reference to the kionoblasts.

The first series of pictures illustrates some points of topical interest in the histogenesis of the enamel, studied with rats, monkeys and humans. It is well-known that the ameloblastema is the first of the hard structure-forming blastemas in the dental organ to be differentiated; following this, the differentiation of the odontoblastema, the appearance of the predentine and the primary mineralization of its peripheral part take place. Not until the dentine formation is complete does the enamel production begin.

Fig. 1 illustrates the pulp with the odontoblastema, the predentine with its Tomes' canals at an angle of 45 degrees, the early stage of mineralization in its globular form, and the ameloblastema's beautiful palisade of cells, with nuclei on practically the same level. Outside this lies the merged inner and outer epithelia. The ameloblastema's cell-row is delimited by the structure which, in American research, is known as the "terminal bars". There

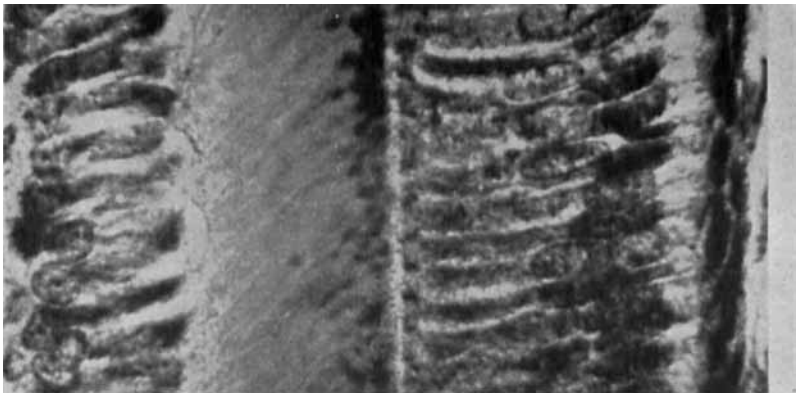


Fig. 1.

has been much lively discussion on the existence of this structure. In many quarters it has been held that the markings are but an artefact, resulting from the handling of the material. The author disputes this theory, maintaining that the boundary structure does in fact exist, and in the way that the name "terminal bars" conveys; this assertion is based on the study of sections from both decalcified and non-decalcified specimens. In the figure the membranes of the ameloblasts can be followed over the border line and between the Tomes' processes which are not yet homogenized in the upper part of the picture where the enamel is only just beginning to form.

In a later stage of development it is possible to follow more clearly the cell-membrane's bridging and to see the initial mineralization of the now homogenized primary substance. Among the ameloblasts other types of cells appear, with bodies so narrow that they are barely half the width of the ameloblasts themselves. They have peculiar, long nuclei whose structure is indistinct. These are the kionoblasts, whose function, according to the literature, is to be found in the fact that they are spread out fork-shaped into the membrana limitans, imparting firmness to it, and thereafter leading to the formation of the Nasmyth membrane. More attention will be devoted to these cells later on in an attempt to show that they have probably another function, Fig. 2 (colour) Pl. I.

In Fig. 3a the course of the histogenesis of the enamel can be followed more closely. Nearest the mineralized dentine surface we find the primarily mineralized enamel whose formation of

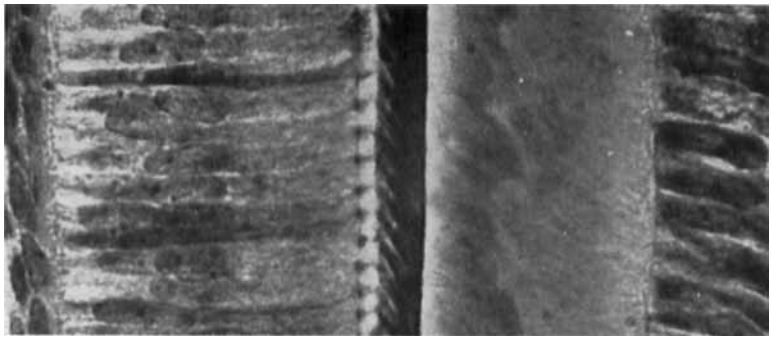


Fig. 3 a.

prisms at an angle of forty-five degrees to the ameloblastema occurs in such a way that the homogenized series of Tomes' processes is mineralized from the membrane walls: they assume a funnel shape, each opening on to the ameloblast. One gets the impression that the kionoblasts as such take no part in the formation of the prisms but may be followed singly or in numbers between the ameloblasts — a point which will be returned to. Between the terminal bars and the enamel surface are the connecting membrane walls.

Fig. 3b is the negative of 3a and is included because it illustrates so well the channel form of the kionoblasts; and, in addition, the course of the enamel mineralization from the funnel walls of the prisms.

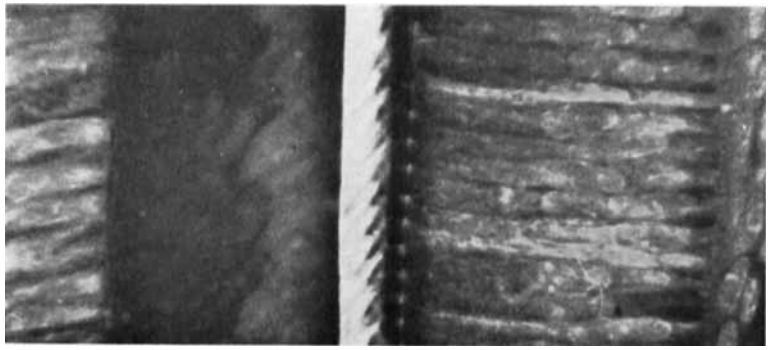


Fig. 3 b.

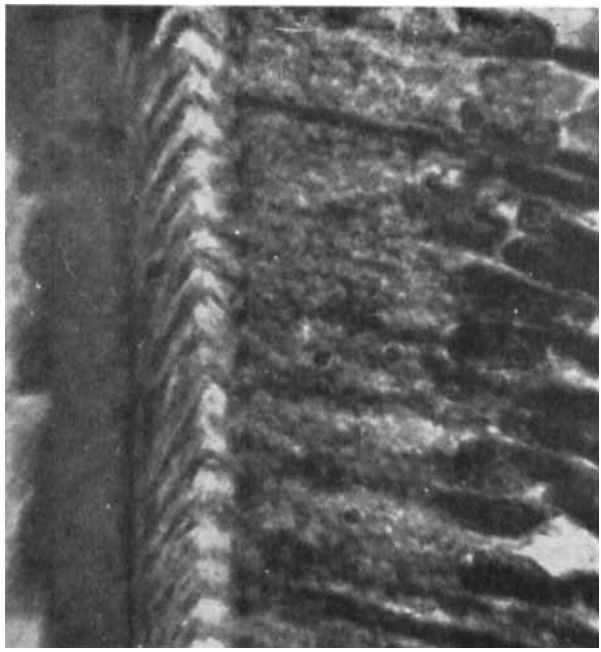


Fig. 4 a.

Fig. 4a demonstrates a further histogenetic detail of importance. The membrane band of the blasts may be followed as a double structure of two lines pressed one into the other. In this somewhat oblique section it can be observed in various places, moreover, that the terminal bars not only appear as a regular row of points, but form oblique lines in the membrane's longitudinal direction or in the angle of the hexagonal pattern. Fig. 4b, the negative of 4a, brings out the channel form of one of the kionoblasts, the funnel shape of the prisms and the angular structure of its membrane extensions to the terminal bars.

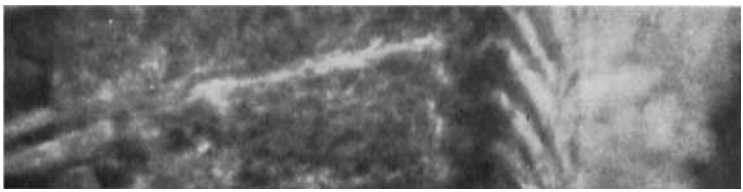


Fig. 4 b.

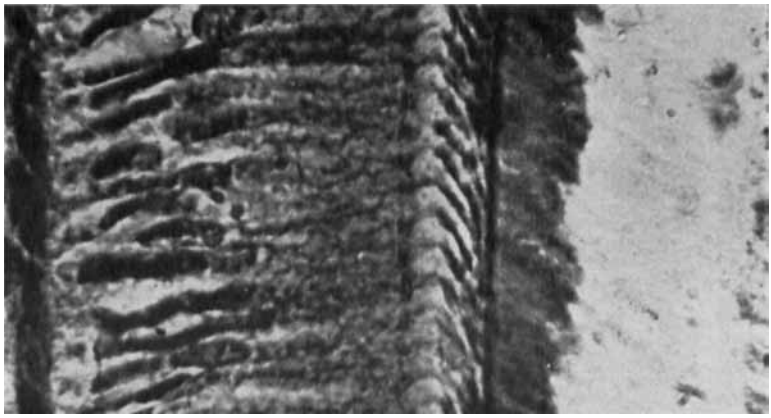


Fig. 5.

If rickets appears during the period of formation of the enamel the mineralization is checked, with the result that the Tomes' processes are not filled with mineral salts in the normal way, Fig. 5. The pattern will therefore be dominated by the thin prism wall with the double lines of the membranes from the blasts to give the newly-formed enamel its peculiar design. The various shapes of the terminal bars are an interesting feature.

In an attempt to learn more about the kionoblasts the author has reconstructed in a number of serial sections the arrangement and distribution of the cells in the ameloblastema by using oblique sections. Fig. 6 (colour) Pl. I demonstrates how the kionoblasts are sandwiched between the ameloblasts in a very characteristic fashion, and how the considerably smaller and thinner cells build themselves into an irregular system which seems to be most comparable with the serpentine course of a system of blood vessels.

If one follows, in different section layers, the fate of these cells during various phases of production of enamel, one cannot detect their participation in the formation of the prisms and in their mineralization. On the other hand, ample support is found for the interpretation of their function according to the previously stated hypothesis — that the cells in question comprise the apparatus for transport of blood through the ameloblastema.

In Fig. 7 one can follow a succession of three kionoblasts, demonstrating the transport of blood through the cell bodies.

Fig. 7.

Their difference in shape from the ameloblasts wich have round nuclei should be noted.

In Fig. 8 the difference in shape and structure between the narrow elongated kionoblasts and the broader ameloblasts with their rounded and well-defined nuclei is still better defined.

Fig. 9 (colour) Pl. I portrays red blood corpuscles which have been forced in through the cells in a characteristic manner, and can be followed through them in consecutive rows. This occurrence is most readily apparent in the last stage of the enamel production in the case of humans and monkeys, with their voluminous enamel pulp. But with rats, where the enamel pulp is not of the same volume, one can find this process of corpuscular flow through the blasts at a much earlier stage, as Fig. 10 (colour) Pl. II shows.

The pictures previously described are all of them chosen from material obtained from monkeys and rats. The following three

Fig. 8.

Fig. 11.

photomicrographs from the human embryo have been selected to illustrate the characteristics and reactions of the kionoblasts. The decalcification has but slightly affected the outermost region of the enamel, severing the ameloblasts' connection with the prism system. But as can be confirmed in Fig. 11, the kionoblast acts in quite a different way; despite the decalcification, it maintains its organic connection with the enamel and its cell-body seems to continue as an integral part of the enamel substance.

In Fig. 12, in which a negative is used for greater clarity, the

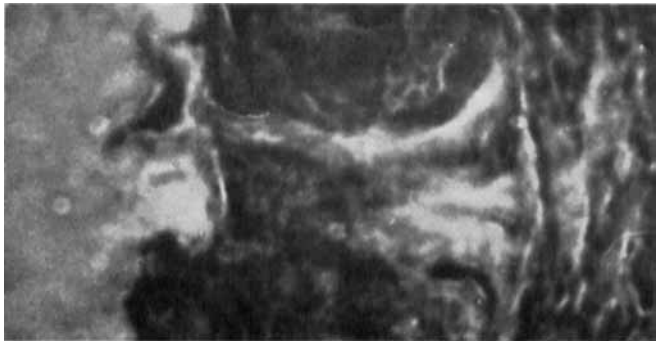


Fig. 12.

differences in appearance and function between the two types of cells in the ameloblastema are indicated by the narrow winding body of the kionoblast which can be followed deeply into the enamel structure.

In greater magnification, that intimate organic connection between the kionoblasts and the enamel really does seem to confirm the hypothetical rôle of the kionoblasts in the histogenesis of the enamel's lamellar system, Fig. 13.

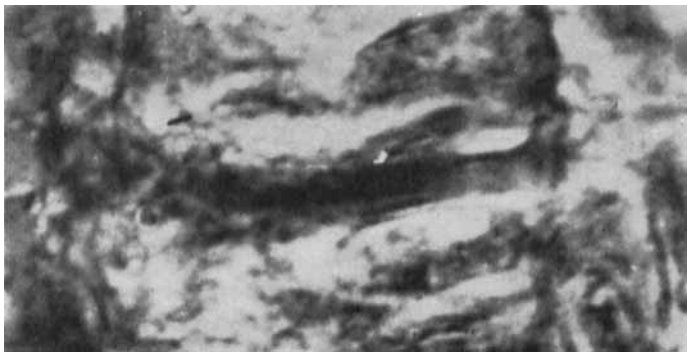


Fig. 13.

As far as can be judged, the kionoblasts take no part in the formation of the prism structure which seems to be solely the result of the functioning of the ameloblasts. On the other hand, they seem to be interspersed, separately and in groups, between the ameloblasts and to occur just as if they represent a blastema blood system: that is, they admit of a flow of blood-serum and corpuscles. They themselves are not blood vessels, but act as a medium for the passage of blood. If, in the research that is continuing, this working hypothesis proves itself to be justified, one is not far from the conclusion that the histogenesis of the lamellar system in the enamel may be a reproduction in the prism structure of the arrangement of the kionoblasts in the ameloblastema.

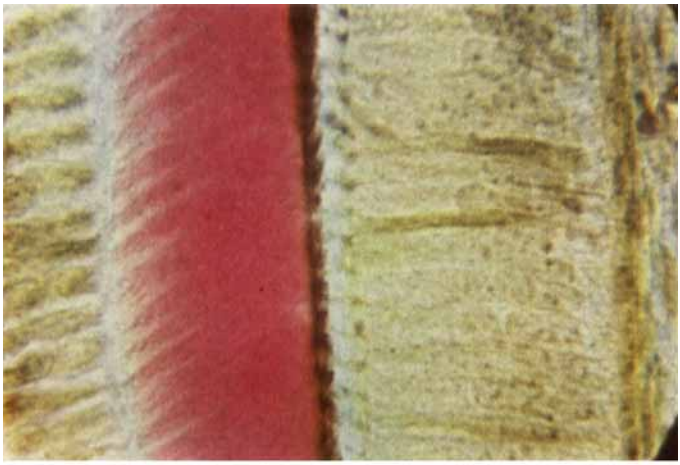


Fig. 2.

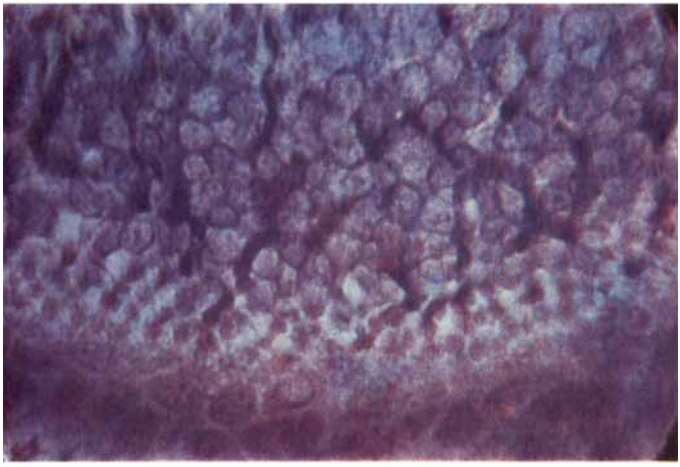


Fig. 6.

Fig. 9.

PLATE II.

Fig. 10.

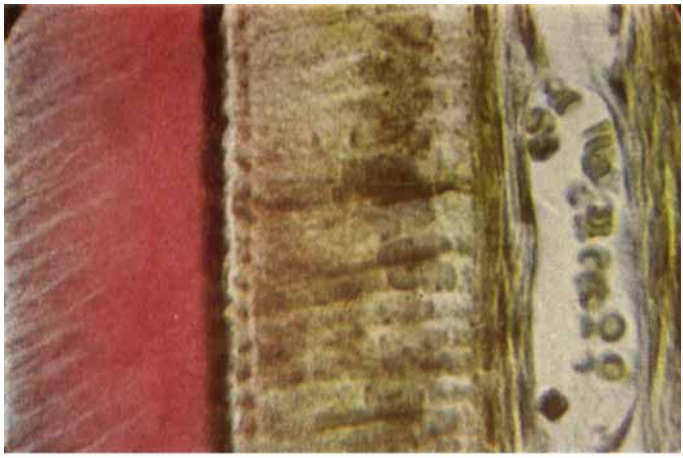


Fig. 14.

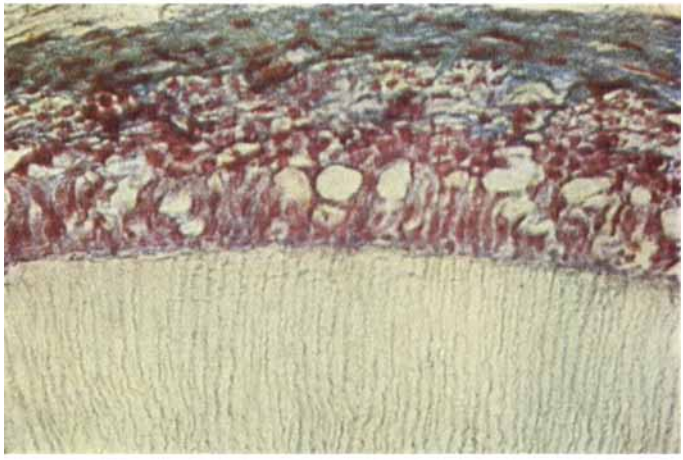


Fig. 22.

II. Histological details of the organic structure.

The design of the structure of the enamel can in its outer part be closely followed from Fig. 14 (colour) Pl. II from monkey. Outermost is to be found the dental sac's collagenous cap, and then the combined inner and outer epithelia with the ameloblastema in vacuolate degeneration. The decalcified enamel structure is characterized by its prisms, free of mineral substance, and by the residual prism sheaths which are recognizable from their origin — that is, the membranes around the ameloblasts.

Fig. 15 depicts clearly the decalcified enamel's organic structure, the double membrane plaiting of the prism sheaths and

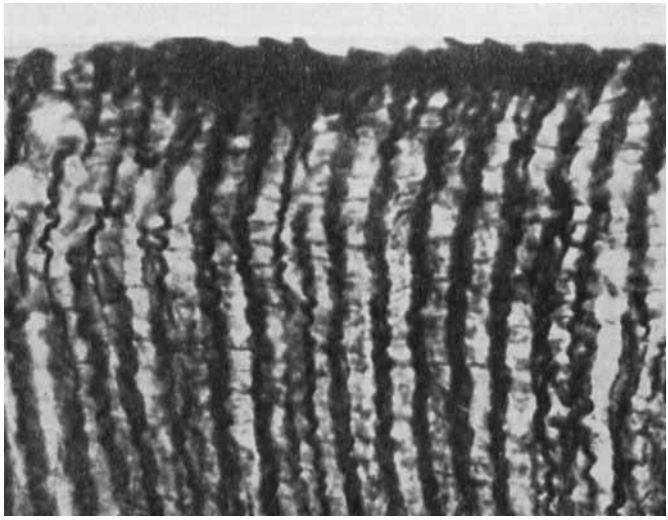


Fig. 15.

the repeated curves, formed, as previously shown, in the primary stage of development. Between the sheaths runs the inter-columnar bridge system of thin transverse lines. These seem, as it were, to split up the prisms into a series of thin parts: when their organic structure is exposed by the action of acid they disclose the enamel's cross-striation. The plaiting of the thin bands of the sheaths, the structure of the decalcified prisms, and the inter-columnar system are more clearly seen in detail in Fig. 16.

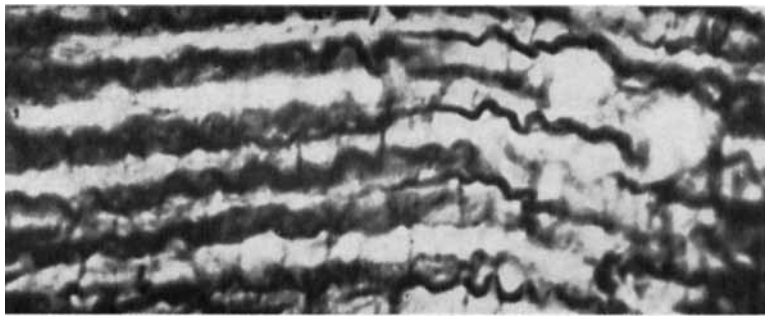


Fig. 16.

For those who may perchance interpret the serpentine form in the picture as an artefact of demineralization and sectioning, it may be of interest to follow the next two photomicrographs, Figs. 17 and 18. The first of them shows the fang of a dog, around the crown of which methylene blue has been allowed to penetrate from the surface, in accordance with Berggren's experimental method. From ground sections of the parts where the colour penetrated, Fig. 18, there are, it seems, quite the same peculiar markings of the organic structure (the repeated curves and the plaiting) as appeared in the decalcified preparation. There can, therefore, be no question of an artefact.

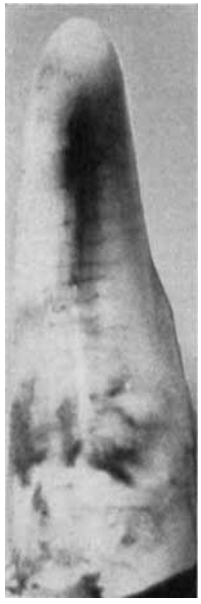


Fig. 17.

The deeper the decalcification the more clearly do Hunter's bands stand out, Fig. 19. Thus they are not, as so often alleged, the result of variation in the mineralization, but illustrate a typical and regular arrangement of the prism system which is more clearly unmasked the more the organic structure is exposed through prolonged decalcification.

With the same method of decalcification of the enamel of extracted human teeth, the lamellar system most often appears as indicated by Fig. 20. The formation of the lamellae seems at least not to contradict the working hypothesis assumed above: that is, that it should originate from cell elements of another type in the ameloblastema, which, in the

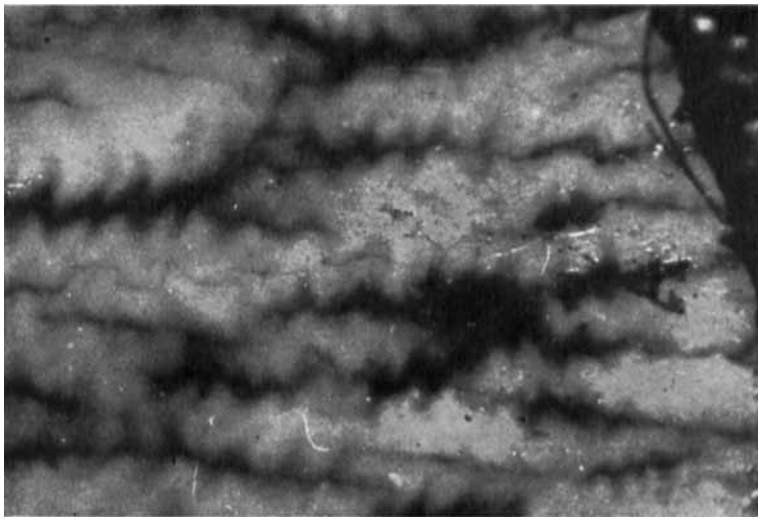


Fig. 18.

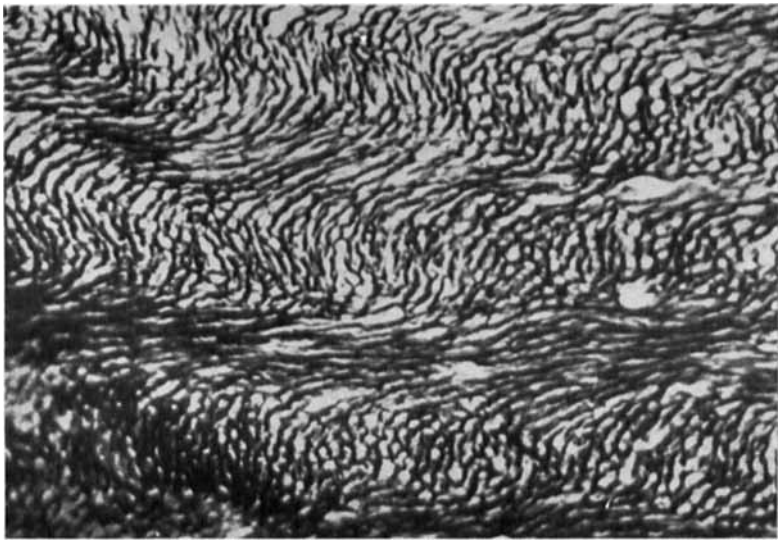


Fig. 19.

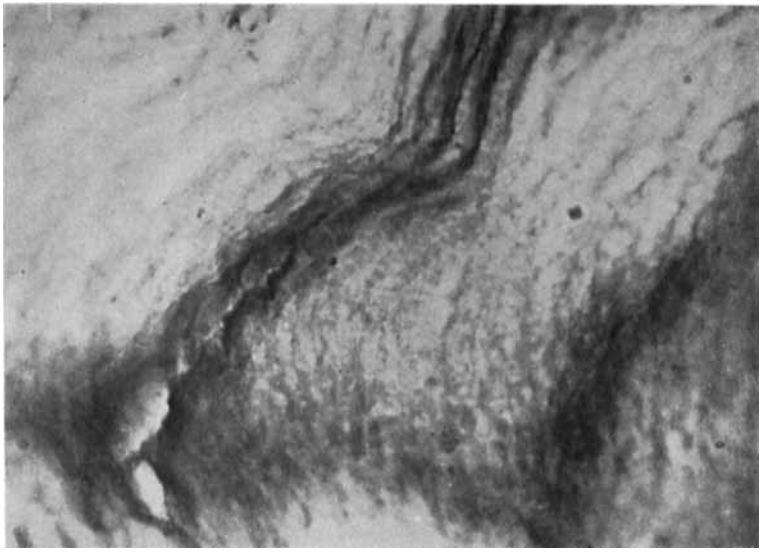


Fig. 20.

process of development, are imprisoned in the prism system.

In these lamellae of the human enamel, Fig. 21, it is not unusual to observe lumina of varying size and length. Whether these are artefacts it cannot always be decided.

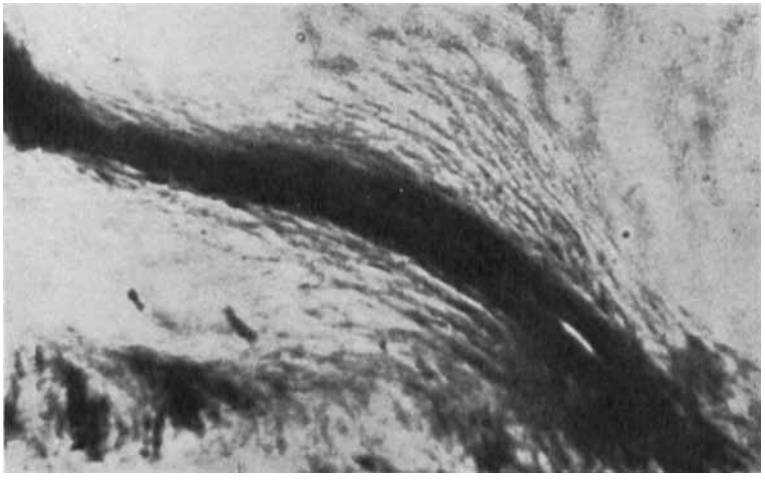


Fig. 21.

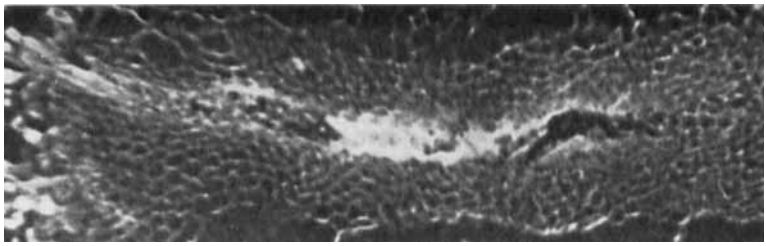


Fig. 23.

If such luminae really exist, one would expect to find them present if, in its final stages, the process of mineralization of the enamel were interrupted. Photomicrographs from horizontal sections in the deep gingival layers of the enamel in cases of D-hypo-vitaminosis in monkey, *Macacus rhesus*, show a remarkable configuration of the lamellar system. One cannot avoid the impression of a bloodvessel-like feature in the enamel structure, running between the dentino-enamel junction and the surface, Fig. 22 (colour) Pl. II.

In many other pictures, e. g. Fig. 23 (negative), clear lumina can be traced whose content further emphasizes the validity of the assumption. It is not unusual to find ramifications, a fact which tempts one to believe in an enclosed vessel-like system of canals. It is of importance, however, not to confuse such a phenomenon with the fissure system in obliquely sectioned enamel such as is illustrated in Fig. 24.

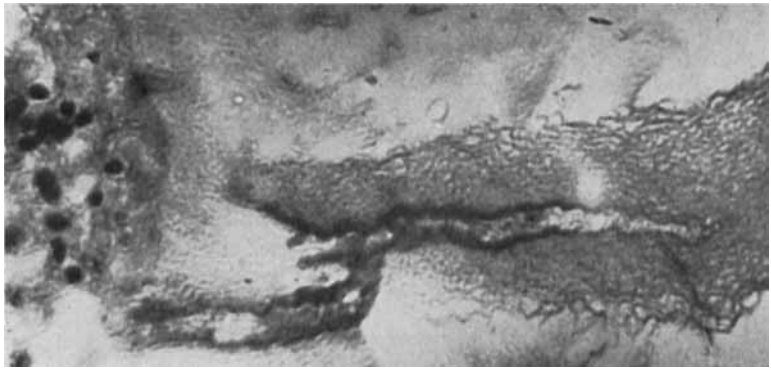


Fig. 24.

III. Carious decay of the organic structure.

It is clear that it would be of value to caries research if it could be shown, as assumed here, that the lamellar system forms a canal system, deriving from cell elements in the blastema, which elements facilitate the blood transport. The accepted fact that primary caries most often develops around lamellae, extending from the seat of the surface injury towards the boundary of the dentine, should, in accordance with this tentative explanation, offer many interesting links for continued research.

A general outline of the early development of caries in the occlusal fissures of the six-year molar is depicted in Fig. 25 (colour) Pl. III. The so-called decalcified zone of the enamel caries is represented by the densened eosinophile zone. The section cuts the plaque in the occlusal fissure. In this zone, proteolysis subsequently occurs, leading to the formation of caverns in the presence of powerful bacteria cultures.

It is of particular interest to observe how the carious zone is walled off behind a dense barrier; Fig. 26 shows at its centre the passage of the convolute lamella path towards the dentine. The

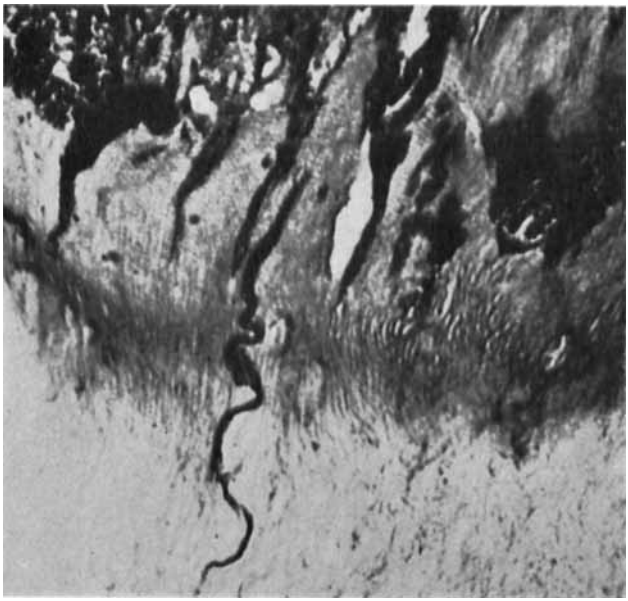


Fig. 26.

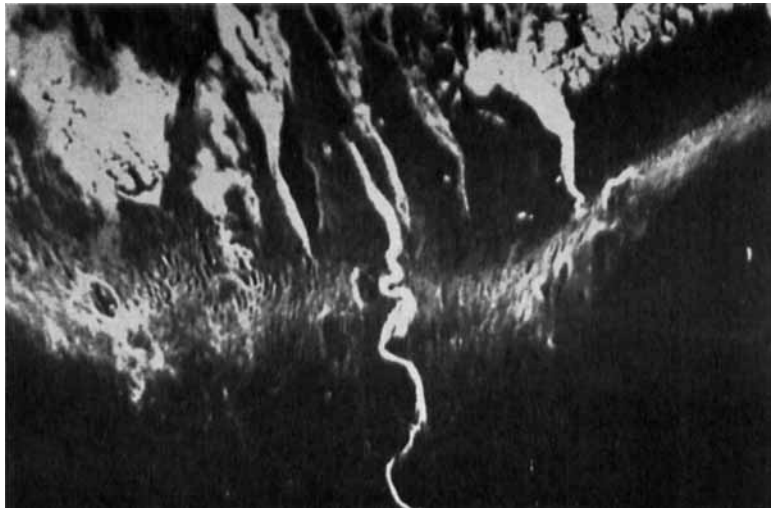


Fig. 27.

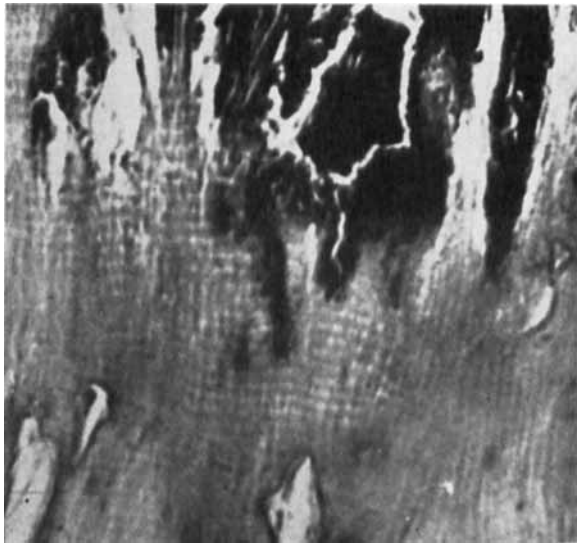


Fig. 28.

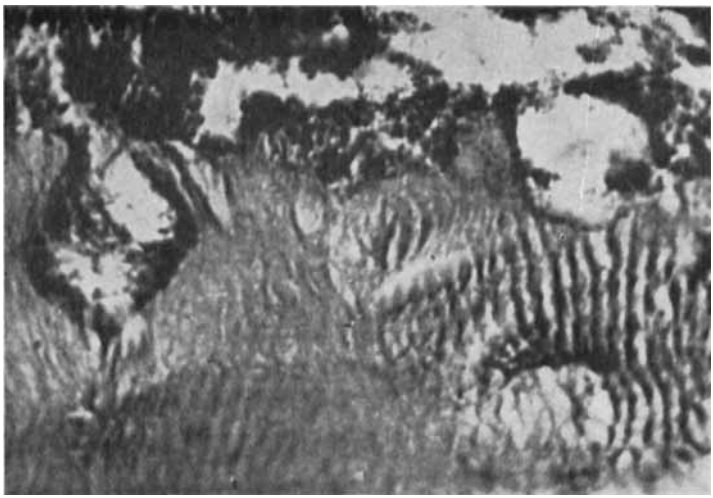


Fig. 29.

negative, Fig. 27, brings out very clearly the wall and the penetrating lamellar system.

Fig. 28 demonstrates the irregular advance of the cavern formation in the enamel; but of greater interest is the illustration of cross-striation which appears as a regularly progressing division of the organic enamel structure: the long bands of the enamel sheaths are eventually divided up into distinctive, distended, bulbous fragments which are finally dissolved, thus extending the proteolytic caverns. How these caverns develop is seen from Fig. 29. In one area the result of the carious acid effect with decomposition along the intercolumnar bridges and the spherical development of proteolytic tissue destruction may be observed. The corresponding caverns which are filled with bacteria are

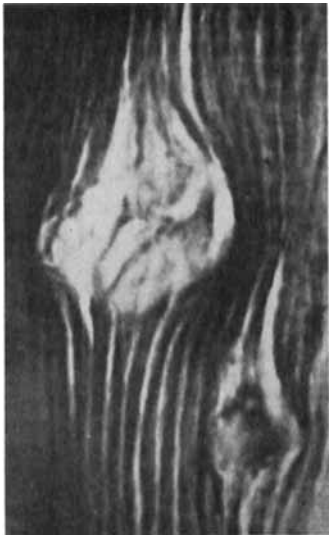


Fig. 30.

visible, both in open connection with the completely destroyed zone lying above.

That the prisms of this zone have lost their mineral content is clear from Fig. 30 which shows the cavern walls distended by the internal pressure.

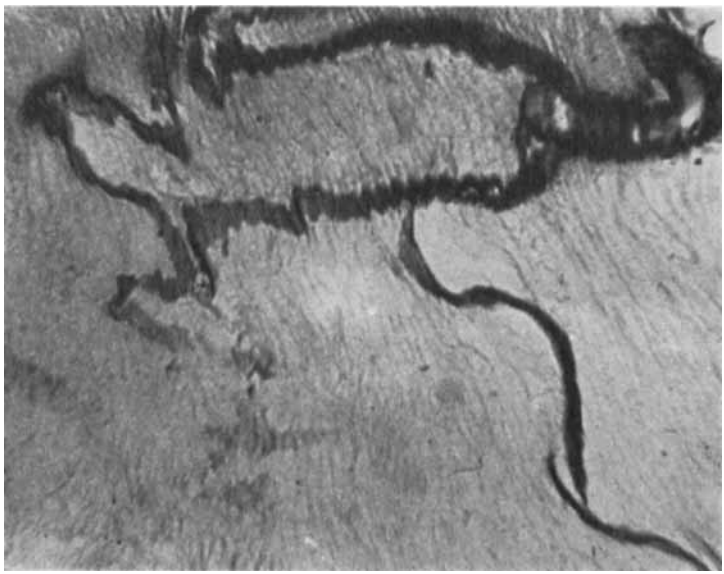


Fig. 31.

If one cuts obliquely from the bottom of the decomposed and softened zone, lamellae are found, winding inwards, and this lends support more clearly than ever to the view of the lamellar system as the enamel's concealed path of infection to the inner tooth, Fig. 31.

The conception of a convoluted "vessel" system in the enamel seems further confirmed by Figs. 32 (negative) and 33, which, in a series of sections, trace the lamellar path down to the boundary dentine.

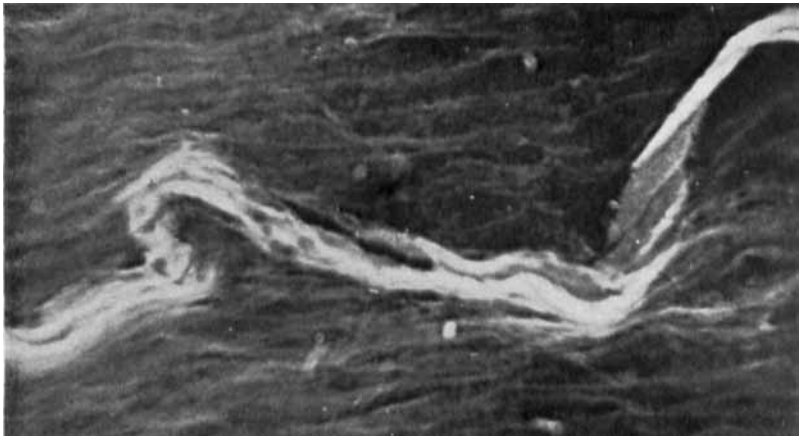


Fig. 32.

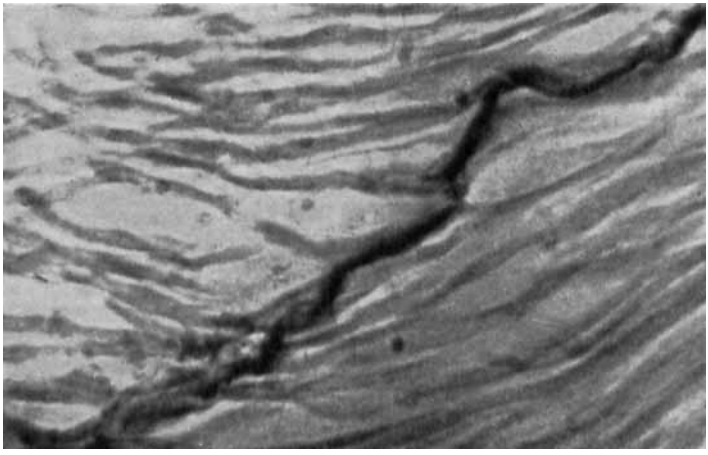


Fig. 33.

IV. The histogenesis of "textbook hypoplasia".

The ameloblastema is, as pointed out above, the first of the blastemas building up the dental hard substances to be differentiated. One would expect that, as in the question of blastemas derived from the mesenchyme, the production of hard substance would occur on the first differentiation of the blasts. After the differentiation of the ameloblastema, however, the whole dentine formation proceeds as far as the mineralization of the first dentine layer, immediately after which the enamel calcification starts. If any conclusion is to be drawn from this, it should probably be that the mineralization of the preceding mesenchymal dentine cap is a necessary condition of the enamel production. If the dentine mineralization is disturbed during the period of dentine production in such a way that the dentine cap is defectively — or not at all — mineralized, that particular dentine cap will reach the dentino-enamel junction but with a non-mineralized dentine zone. The Retzius' stripe which, from the upper margin of this dentine zone, progresses into the enamel, marks an interruption in the production of enamel. Such a break appears as an organic membrane — just as the enamel cuticle. Where this reaches the enamel surface there accordingly appears a hypoplasia, in the literal sense of the word, signifying a lack of substance to be built up. If the mineralization of the dentine is arrested for long, the enamel will not develop normally and a thin poorly mineralized enamel covering will result, on a cusp or proximal part, for example. If, however, the interruption of the mineralization of the dentine is only brief and is followed by mineralization of the next dentine cap, there will remain the incompletely mineralized or entirely unmineralized preceding dentine cap, thereafter unalterable. This is recognizable in the complete dental structure by a dentine cap, either wholly mineral-free or with interglobular spaces of varying sizes. It is possible to detect on the longitudinal section of a 6-year molar, for example, all the variations in the mineralization of the dentine caps, from shortly before birth to the age of 12 or 14 years. At each site where caps, characterized by irregularities in mineralization, reach the dentino-enamel junction, the corresponding production in the enamel exhibits a simultaneous disturbance. If the disturbance is insignificant, the change appears in the enamel as a thinner enamel cap with defective crystal structure of

the hydroxy apatite. If the mineralization of the dentine cap ceases completely, the above-mentioned line of interruption appears with associated enamel hypoplasia. When the succeeding dentine cap is mineralized normally a fresh enamel cap is immediately

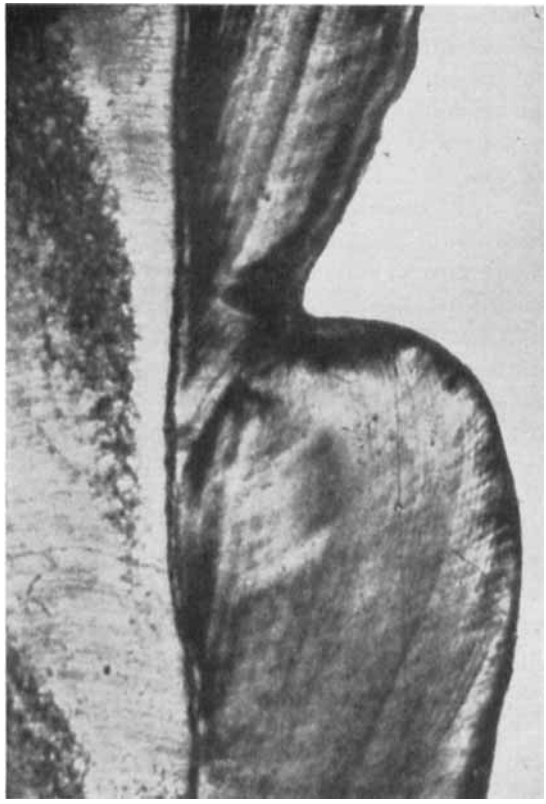


Fig. 34.

built up. This is structurally unlike the previous one since it is oriented differently. The ameloblastema follows the margin of the hypoplastic enamel; when the fresh enamel cap is built up it assumes the original thickness of the enamel, the bowed line so formed, to which the ameloblastema conforms, overlapping the hypoplasia. The hypoplasia is thus followed by an apparent hyperplasia. The two layers of enamel are separated by the interrupting membrane and manifest in their mineralization of the

prisms the two distinct periods of construction. If there should subsequently recur an injury which causes disturbance in mineralization, a dentine cap would exhibit once again imperfect mineralization, and enamel hypoplasia would reappear. The cause of this irregularity in mineralization is here called the R-reaction (see note, p. 52). It affects the whole bone structure of the body, but is clearly evidenced in the formation of the teeth. The pathogenesis lies most frequently in D-hypovitaminosis or D-avitaminosis (rickets), spasmophilia, fluorosis, enteritis-colitis, etc.

When studying enamel hypoplasia in ground sections, the section should be made vertically through the centre of the enamel hypoplasia, parallel to the longitudinal axis of the tooth. Fig. 34 gives the familiar picture of enamel hypoplasia as it then appears under the microscope. It is of particular interest to confirm how, with its tip, the non-mineralized dentine cap reaches the dentino-enamel junction just below that point from which the corresponding line of interruption in the enamel is directed towards its surface. At the site where this line emerges in the surface, enamel hypoplasia is again in evidence, followed by the apparent hyperplasia whose curved contours indicate a radial course of the prism structure. This picture recurs in every enamel hypoplasia of this type.

To give a clear experimental picture of the pathogenesis of such hypoplasia, recurrent D-avitaminosis in young apes, *Macacus rhesus*, has been studied. The following photomicrographs of dental organs during growth and formation of the hard substances have been chosen with the intention of illustrating the parallel with the final state, Fig. 34. The first of them, Fig. 35 (colour) Pl. III, gives a general survey. It shows the occurrence of the interruption in the mineralization of the dentine which results in the pathologically widened predentine zone and the long predentine extremity of the margin of the crown. The corresponding enamel formation has ceased altogether, indicated by a hyperchromic line. Higher in the dentine the previous period of D-avitaminosis is registered by the deficient mineralization of its dentine cap. The corresponding interruption in the enamel production is visible as a hyperchromic line in the enamel structure, directed towards the enamel surface following the Retzius' system. Where this line reaches the enamel surface, hypoplasia can be confirmed; in the cavity so formed the ameloblastema follows the curvature. Between the associated dentine cap and that cap which now marks

the repeated interruption in the mineralization lies a row of dentine caps with normal mineralization, indicating the interval of healing between the two periods of D-avitaminosis: this was when vitamin D was supplied to the animal. The enamel which

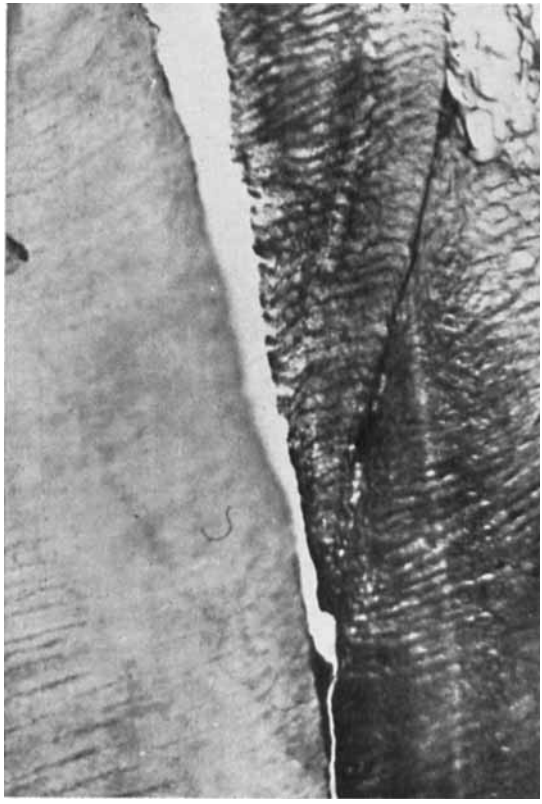


Fig. 36.

is deposited during this healing period corresponds to the enamel between the above-mentioned hypoplasia and the current interruption in the enamel production. Since this enamel formation has resumed its normal width it appears as a hyperplasia and lies, bow-shaped, pressed up against the earlier cavity. It is apparent from this picture that the enamel hypoplasia marks a change in structure in the enamel construction. The healing enamel has quite another orientation of the prisms from the enamel which

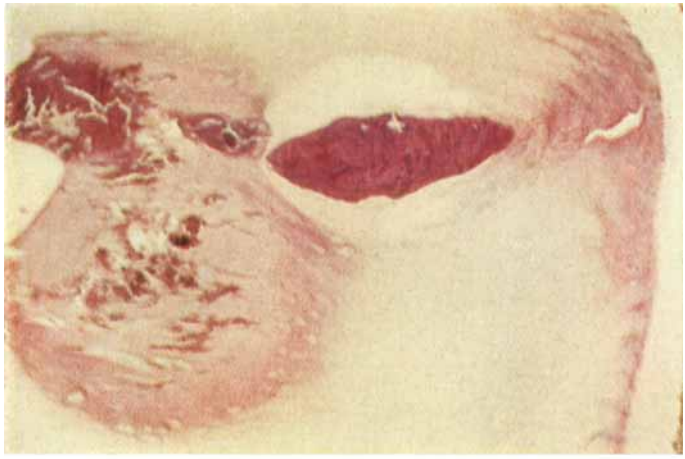


Fig. 25.

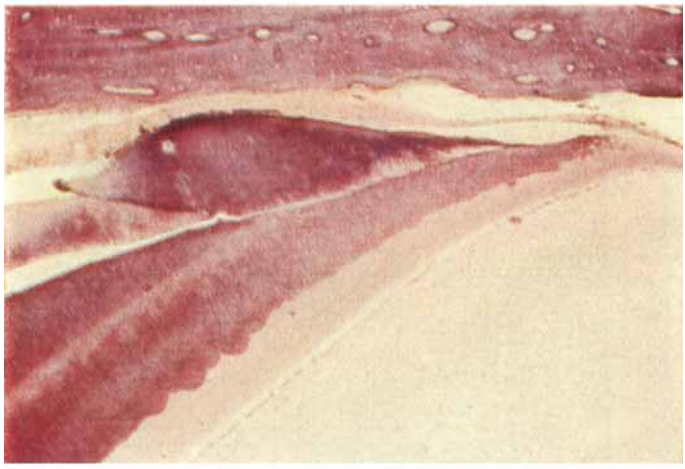


Fig. 35.

Fig. 37.

Fig. 47.

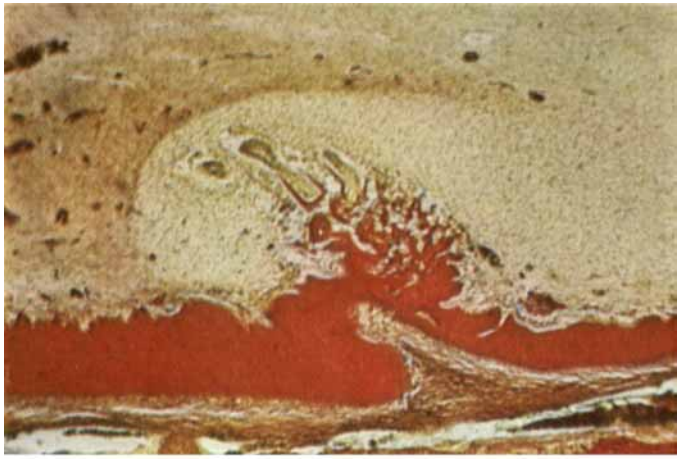


Fig. 51.

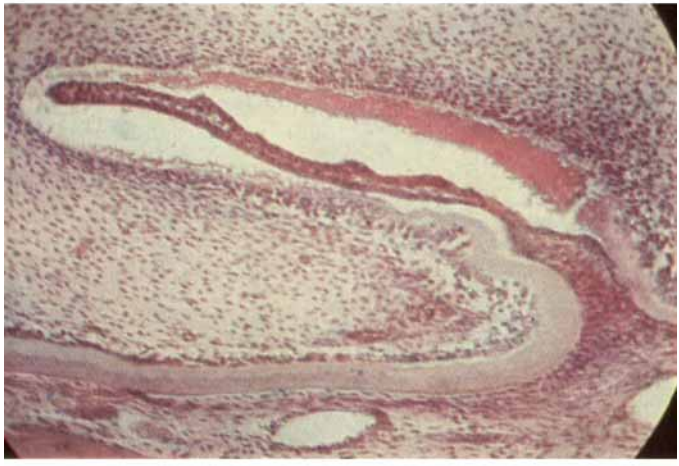
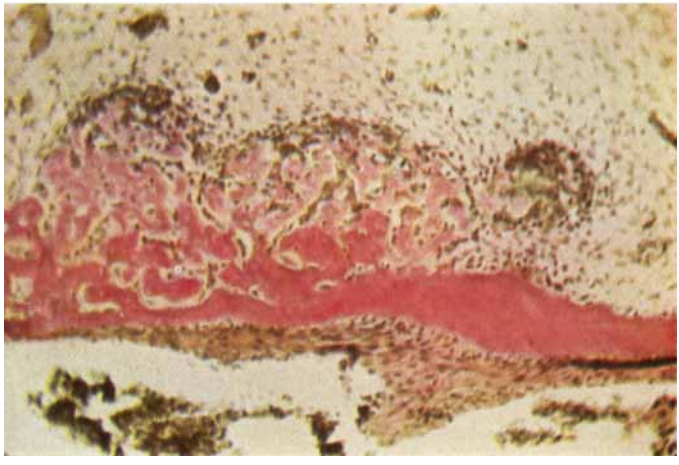


Fig. 52.



formed before the break in production. This statement will be more clearly illustrated in the following detailed photomicrographs.

Fig. 36 shows the non-mineralized dentine zone remaining from the first period of D-avitaminosis; from the point where this zone's upper margin meets the dentino-enamel junction, the membranous organic marginal line indicates the break in the enamel formation; this line divides the enamel substance into two parts of entirely different character in the direction and the arrangement of the prisms.

The hypoplasia of the enamel in this form, the so-called "textbook hypoplasia" which has its origin in the inhibition of the mineralization, conceals in the enamel block a structural prismatic variation in two layers, the one which precedes the period of interruption and the other which follows in the recovery periods, Fig. 37 (colour) Pl. III; between them runs the cuticular membrane of the period of interruption displaying amorphous calcification.

V. The histo-pathogenesis of the pulpal enamel pearls.

A peculiar phenomenon in the pathology of the enamel is the formation of enamel pearls in the pulp tissue; they appear during the development of changes in the hard structure in the incisors of rats in cases of experimental A-avitaminosis.

In Fig. 38 a typical enamel pearl with central ameloblastema is photographed in the pulp tissue near the cementum-covered wall of the rat incisor. An attempt will be made here to explain in detail the pathogenesis of this formation so much discussed in the literature. For this purpose it is necessary to give a brief account of the pathological changes in the formation of the dental hard substances during the development and healing of experimental A-avitaminosis in growing rats. A fact of particular interest is that experimental animals show a gradual decrease to zero in the ascorbic acid value of the blood during the period of vitamin A deficiency; the most profound changes in the dentine and cementum formation take place in just these last stages of the A-avitaminosis. On recovery promoted solely by renewing the vitamin A supply, with no alteration in the C-content of the diet, the ascorbic value of the blood rises again to normal. This phenomenon of a bio- and cyto-chemical relationship between the A-avitaminosis

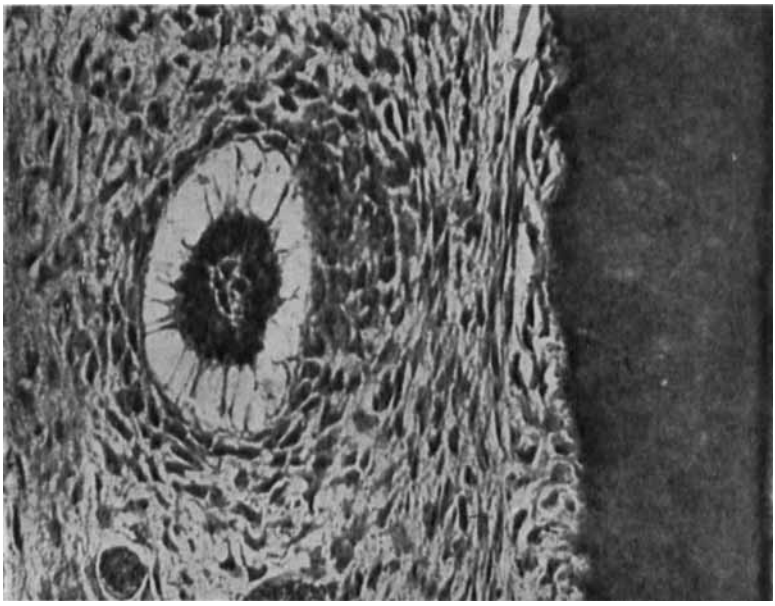


Fig. 38.

and C-pathology has given rise to the theory that the skeletal and dental pathological changes in A-avitaminosis are to be interpreted as secondary scurvy. This must be called in question; the dental pathology of A-avitaminosis as a whole presents the interesting picture of an exceptional and very specific disease which may be created experimentally in but one manner — the complete suspension of the supply of vitamin A. It is true, however, that there occur, among the dentinal and pulpal changes in A-avitaminosis, changes of a type that may be principally classified as an S-reaction.[1]

[1] The terms *R-reaction* and *S-reaction* are derived from "rachitis" and "scorbutus", respectively. The terms — expressable as an either/or principle — refer to two diametrically opposite series of pathological skeletal and dental changes concerning the building up of new hard substances, their quality and quantity, their structure, mineralization, collagenous content, differentiation of the blastema, etc., as well as destruction in the form of osteoclastic and osteolytic resorption, osteoporosis, atrophy, etc. This either/or principle in the bone pathology is to be found in all details most pronounced in D-avitaminosis, rachitis (R) and C-avitaminosis, scurvy (S) (This notation is parallel with WILTON's N- and W-factor).

These terms are in general use in the author's previously published works and postgraduate courses in histopathology of the hard substances, and are particularly conveniently applicable in schematic surveys of all the different reactions in the skeletal system and teeth resulting from specific infections and inflammations, from endocrinal and nutritional disturbances, from vitamin deficiencies, from ageing and from traumatic lesions.

The significant physio-pathologic characteristic of dental A-avitaminosis is the folding-in against the pulp tissue of the apical blastema in the growing incisors of the rat. In vitamin A deficiency, as in C- and D-avitaminosis, there will be observed a very interesting difference in reaction capacity between the two parts of the odontoblastema of the enamel- and cementum-covered root-halves. In these two factors — the singular folding-in, and the difference in reaction between the blastema halves — is to be found the histogenesis of the pathologic morphology of the teeth in A-avitaminosis.

In the first phase of development of the disease, the simultaneous characteristics of the R-reaction are predominant in the form of enamel hypoplasia and defective mineralization of the dentine. This latter change takes place chiefly in the enamel-covered half.

Fig. 39 shows the apical part of the enamel-covered root-half with the remarkable folds typical of A-avitaminosis. The photograph of the amelo-odontoblastema demonstrates the comprehensive R-reaction in the form of numerous hypoplasia and of defective and checked mineralization of the dentine.

The great difference in reaction between the enamel- and cementum-covered halves of the tooth will be clear from Fig. 40 which gives a general picture. To the left is the enamel-covered half with its typical R-reaction; imperfect mineralization of the dentine with large interglobular spaces but with odontoblastema comparatively intact. To the right is the corresponding cementum-covered wall; this is noticeably narrower, with a blastema which no longer has the characteristics of the normal odontoblastema but resembles rather an osteoblastema in the type of the cells; there is also a widened predentine zone but at the same time structural alterations are beginning to appear. In the continued development of the pathological changes of the dentine structure along the cementum-covered wall the odontoblasts are further deteriorating and the blastema disappearing: the R-reaction ceases and is followed by a tendency towards development of the S-reaction with the formation of hard substance with enclosed cells and the appearance of an osteo-cementum-like layer, Fig. 41.

In this phase of development, the innermost layer of the dentine wall of the enamel-covered half also begins to show structural changes of a special type, Fig. 42, peculiar lacunae of varying depth appearing in the dentine; irregular dentine forms in these, and

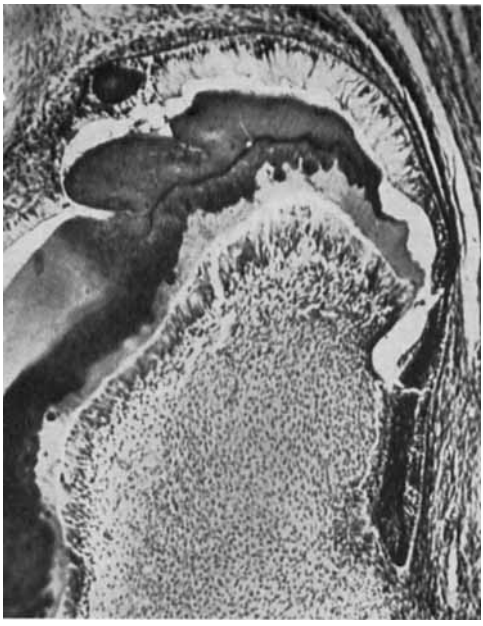


Fig. 39.

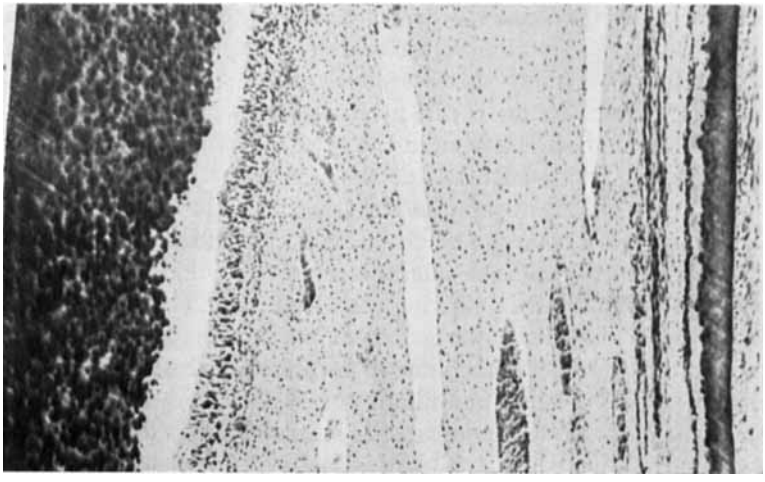


Fig. 40.

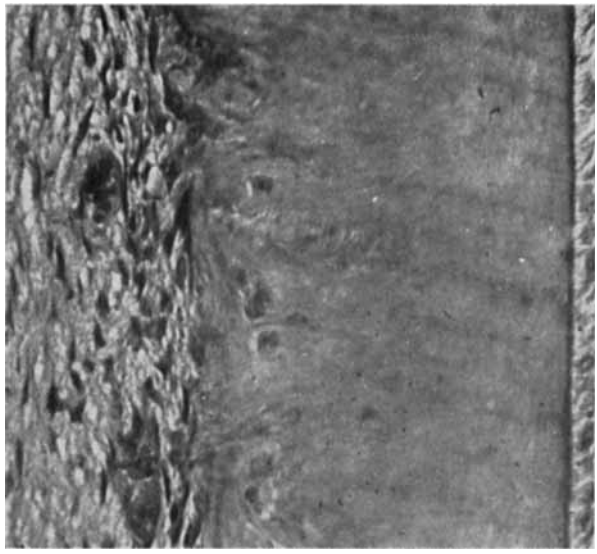


Fig. 41.

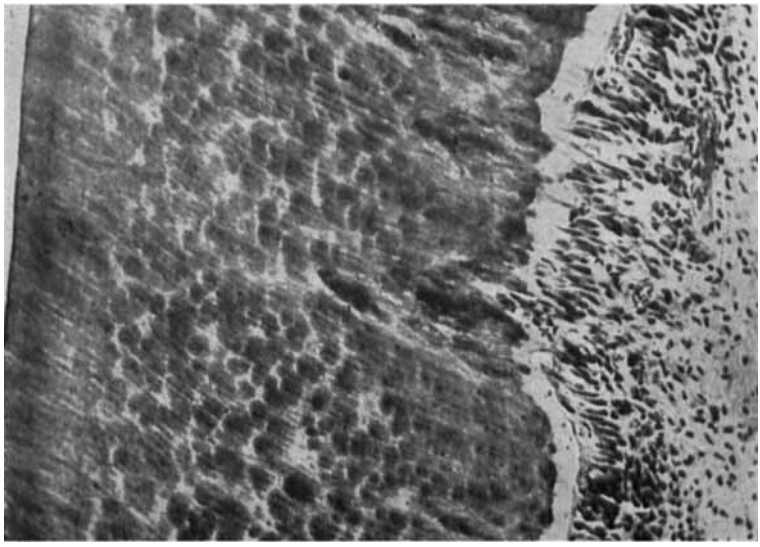


Fig. 42.

hyperaemic vessels can generally be followed into the dentine whilst changes are also occurring in the odontoblastema; the dentine with the R-reaction is then followed by an innermost dentine layer with more normal mineralization but with structural S-reaction. In the final stage of experimental A-vitaminosis there appear, with the repeated folding of the apical amelo-odontoblastema, the prominent changes in the formation of dentine depicted in Fig. 43; within each new fold the peculiar structure recurs.

The rhythmically repeated folding of the apical cementum-covered dentine wall results in the formation of adherent denticles, typical of the final stages of A-avitaminosis. The denticles are attached along a very thin and pathologically altered dentine wall, without odontoblastema or predentine. The structure of the denticles can be closely followed in Fig. 44. Their pathogenesis is fully explained by the two following figures: at the site of folding, that part of the blastema which is folded in towards the pulp continues its formation of the pathologically altered dentine, Fig. 45, and when the root renews its growth in the apical direction during the period preceding the next fold, the dentine formation increases in volume and takes the shape of a denticle attached to the wall and projecting into the pulp, Fig. 46. The collagenous content of these denticles is seen in Fig. 47 (colour) Pl. IV.

All these changes will be repaired by the subsequent normal dentine formation in the course of the healing period brought about by the resumed supply of vitamin A. In the cross-section, Fig. 48, the recovery will soon be complete. The history of the disease — its development to severe A-avitaminosis and its recovery — may be followed here in detail; in particular, observe the marked difference in reaction between the right enamel-covered and the left cementum-covered halves; the pathological hard substances, formed during the period of A-avitaminosis, are now completely imbedded in normal dentine. Fig. 49 gives a very interesting impression of the repair of the root during the healing period: the right part shows the formation of the new apical portion of normal dentine; the connection with the left part comprises small bridges of hard substance developed during the first stage of restoration; the apical extremities of the left part show the pathological deformations from the period of A-avitaminosis, now partially enclosed in repairing dentine.

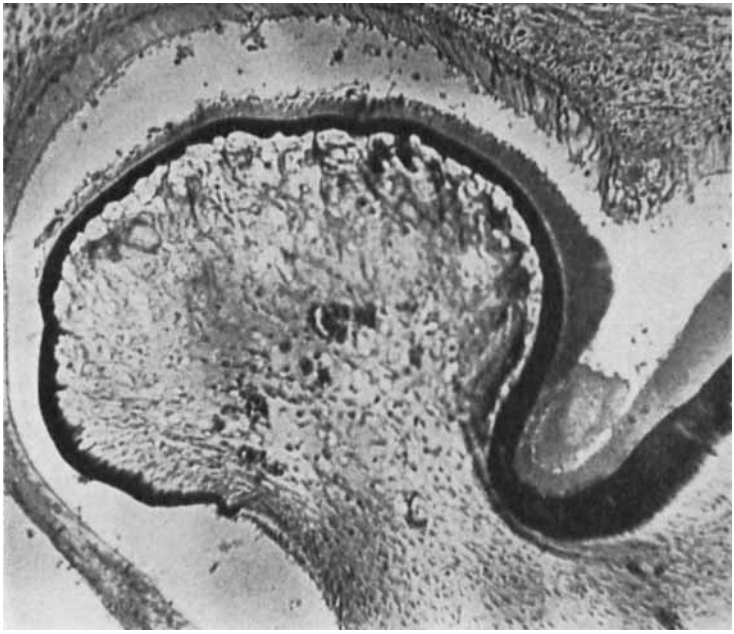


Fig. 43.

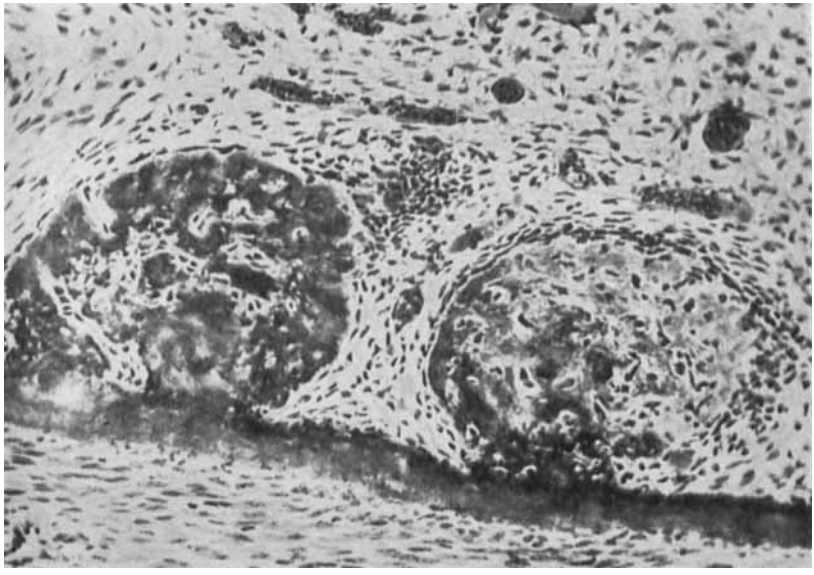


Fig. 44.

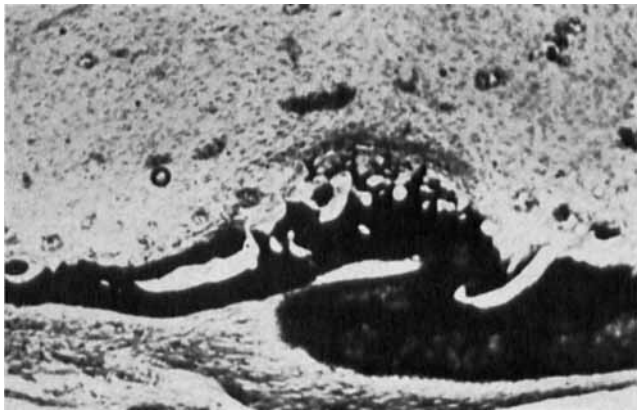


Fig. 45.

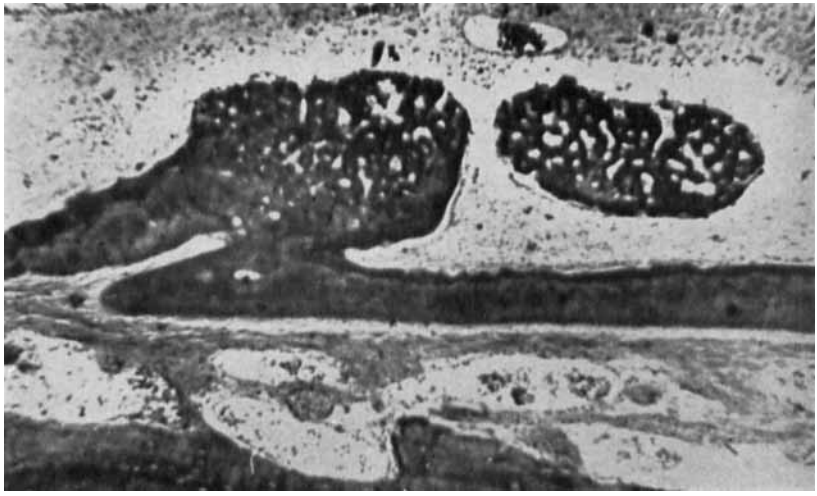


Fig. 46.

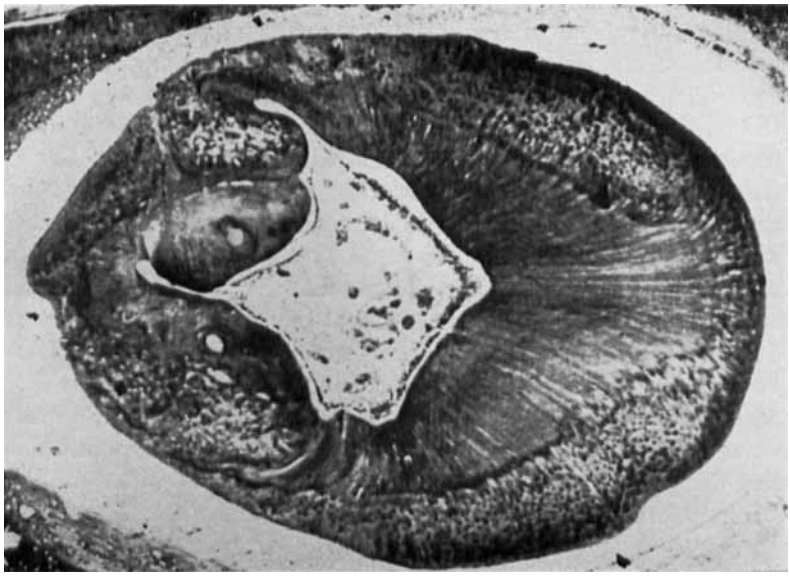


Fig. 48.

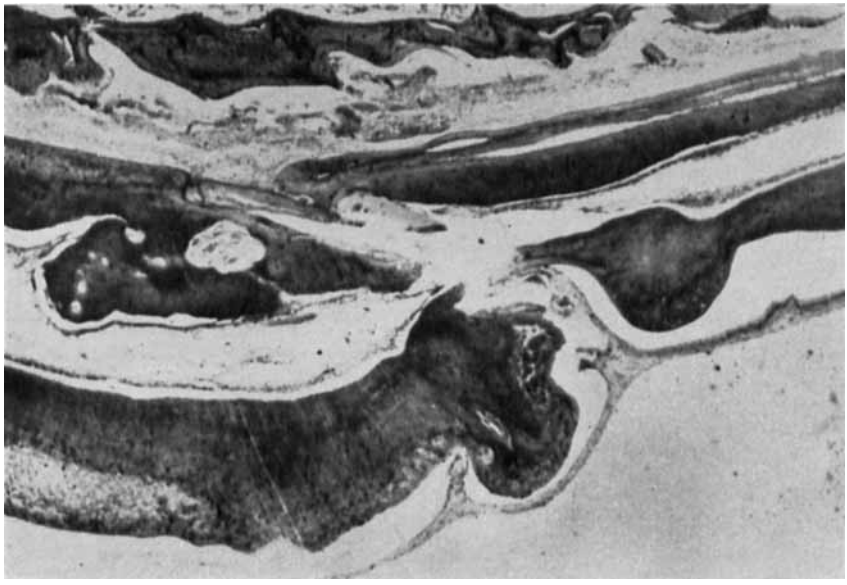


Fig. 49.

As has been shown in this description of the pathological changes in the development of A-avitaminosis, and their repair in its healing, the serial longitudinal sections of the enamel- and cementum-covered halves at the root nowhere expose the pathogenesis of the pulpal enamel pearls which must be counted among the most peculiar characteristics of vitamin A deficiency. From a series of thin sections it can be confirmed that a pearl is the result of the production of enamel from a fold of ameloblastema at the top of an adherent denticle of the cementum-covered wall, Fig. 50. This observation suggests the following conclusions: only along one zone of the long incisor roots can the repeated construction of adherent denticles be completed with a simultaneous folding-in of an ameloblastema towards the pulp. This zone will follow the border-line where the cementum-covered half passes directly into the enamel-covered half. When the serial sections were laid exactly along this line the pathogenesis was easily confirmed. Fig. 51 (colour) Pl. IV. demonstrates the development: just at the transition to the enamel,

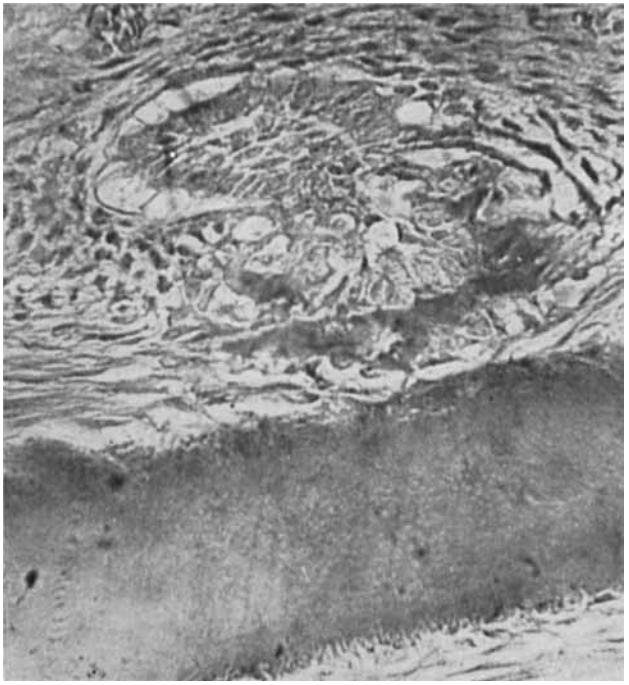


Fig. 50.

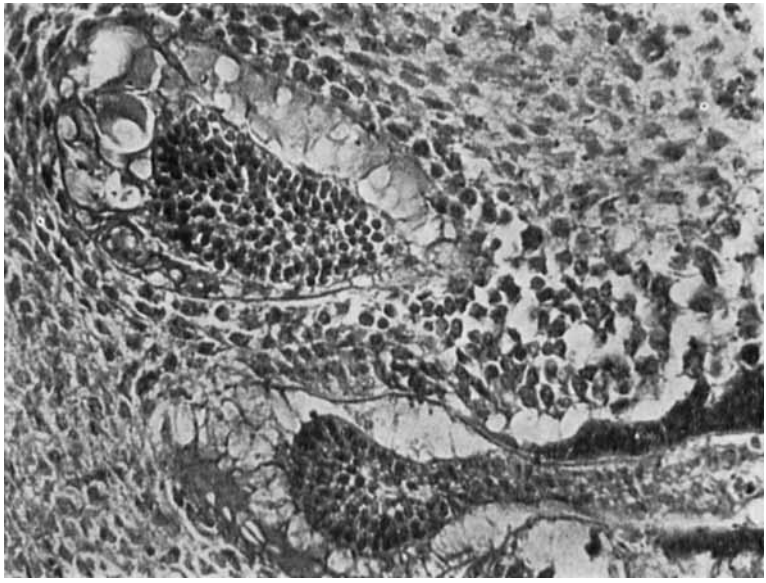


Fig. 53.

the folding of the cemento-odontoblastemas occurs, and there follows a fold of the ameloblastema in towards the pulp. The continued enamel production is now directed towards the pulp.

In Fig. 52 (colour) Pl. IV, the construction of the adherent denticle has started at the point of the apical fold. On its cusp three different enamel caps are recognizable. All of them are the result of the continued production of enamel from the folded ameloblastema. One gets the impression that the uppermost ameloblastema layer with produced enamel cap lies free in the pulp tissue. The enamel production round the point of folding of the ameloblastema, and the apparently free enamel pearl of definite shape in the pulp are clearly depicted on the last photo, Fig. 53. On resumption of growth of the root such pearls will be found situated adjacent to the dentino-cemental wall, along which they were formed, as the first photomicrograph in this series illustrates (see Fig. 38).

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