

PATHOLOGY OF THE DENTAL PULP

By THOMAS J. HILL, D.D.S., Cleveland, Ohio

In this paper, an attempt will be made to review the more important considerations associated with pathologic changes of the dental pulp which may be misunderstood or are controversial and to contribute some new information to an understanding of these conditions.

In a general way, pathologic changes are the same in a dental pulp as in other parts of the body, but, in addition, there are local conditions which contribute to the incidence and severity of injuries to the pulp. These conditions are: inability of the protecting tissue to repair itself, lack of collateral circulation and *definite* encasement within unyielding walls.

The functions of the pulp are: the formation of dentin, the maintenance of vitality of the dentin and the conveyance of sensation. In adult life, when the first of these functions, formation of the dentin, stops, or is reduced to a minimum, histologic changes take place, so that the structure of a normal pulp varies with advancing age. There is a progressive decrease in the number of pulp cells and a gradual increase in the amount of connective tissue. These changes are retrogressive and

the rapidity of their occurrence is increased by external irritation such as from caries, abrasion, pyorrhea and trauma. Figure 1 is a photomicrograph of a pulp from an unerupted third molar of a 21-year-old patient. It is composed of loosely arranged well-vascularized embryonal tissue and contains the usual number of pulp cells. The odontoblasts are regular in outline and of average height and the predentin is of normal width.

Figure 2 is a photomicrograph of an upper lateral incisor which was carious, with slight recession of the gum, also from a patient 21 years old. The pulp cells are decreased in number and much of the embryonal tissue is replaced by fibrous connective tissue. The arterioles have greatly thickened walls and there are areas of perivascular round-cell infiltration and a small amount of fat infiltration. The odontoblasts are lost and no predentin is present. The regressive change is severe and indicates that the pulp has been subjected to injuries for a prolonged period.

Figure 3 is a photomicrograph of a caries-free, pyorrheal upper lateral incisor from a patient 41 years old. In this pulp, the embryonal tissue has been replaced by hyalinized connective tissue and contains but few stellate cells. This pulp also has been subjected to irritation over a long period and represents the histologic changes commonly found in advanced pyorrhea. Comparable changes are found in teeth from people of ad-

*From the Institute of Pathology, Western Reserve University.

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vanced age in whom there is no evidence of external irritation other than normal function.

The pulp can, within some limitations, react to these irritants with progressive changes, such as tubular calcification, the formation of secondary dentin and possibly calcification of the pulp tissue itself.

has previously been believed that this transparent zone is the result of the direct deposition of calcium by Tomes' fibers or by calcification of the Tomes' fibers themselves, but Weber ² and also Euler³ have recently demonstrated that the presence of fat precedes calcification. This indicates that the deposition of calcium is the

Fig. 1.—Normal pulp of unerupted third molar from a 21-year-old patient. (X30.)

Under the stimulation of active caries, calcium is deposited within the dentinal tubules. The location of this deposition is known as the transitional zone, the reactionary zone or the zone of sclerosis.¹ It

result of a degenerative process rather than a physiologic deposition by Tomes'

1. Beust, T. B.: Reactions of Dentinal Fibril to External Irritation, J.A.D.A., 18:1060 (June) 1931.

2. Weber, R.: Neue Untersuchungen über das Auftreten von Fett im Zahn, Deutsche Monatschr. f. Zahnheilk., 48:14-89, 1930.

3. Euler, H., quoted by Kronfeld, R.: Histopathology of Teeth and Their Surrounding Structures, Philadelphia: Lea & Febiger, 1933; p. 89.

fibers. It must be recognized that this deposition does not occur in pulpless teeth. The impermeability of the reactionary zone to dyes (Fish⁴) suggests that the tubules have been blocked, and therefore would retard the progress of caries and obstruct the irritating influence of caries on the pulp.

The extent of this transparent zone is greater in young individuals and in teeth

but when the carious process is rapid, or the penetration is deep, alteration of the pulp is found. In the first case, secondary dentin is formed in response to a mild stimulation, and in the latter case, the irritation of the pulp causes reactions which are destructive in nature.

Such irritations as those caused by caries and abrasion, if not too severe, may stimulate the pulp to increased activity.

Fig. 2.—Pulp of carious incisor having recession of gum, from 21-year-old patient; illustrating retrogressive changes. (X100.)

wherein the penetration of dental caries is slow. In early caries of the dentin, beneath the reactionary zone, pulps may be found which are histologically normal,

The functional ability of the pulp as far as the formation of secondary dentin is concerned may be estimated by the width of the area of predentin, which, when calcification is normal, is in proportion to the activity of the odontoblasts. Fish⁴ has stated that, in the histologic examination

4. Fish, E. W. : Pathology of Dentin and the Dental Pulp, *Brit. D.J.*, 53:351 (March) 1932.

of pulps beneath caries and secondary dentin, reduction in the number or complete loss of odontoblasts is frequently seen. He states that tubular dentin is formed when the odontoblasts have survived the injury and hyaline dentin is

Figure 4 is a photomicrograph of a cross-section of the pulp of a noncarious lower incisor taken from a 26-year-old patient. In the central portion, the odontoblasts are lost and there is no predentin. At either extreme, where predentin is

Fig. 3.—Pulp of caries-free pyorrheal lateral incisor from a 41-year-old patient; illustrating histologic changes commonly found in advanced pyorrhea. (X120.)

formed when the odontoblasts are lost. Examination of our material shows a close relationship between the proportional amount of predentin and the number and structure of the odontoblasts.

present, the amount is in proportion to the height of the odontoblasts. We have observed, particularly in adult teeth and in the teeth from individuals of advancing age, that the height of the odontoblasts

Fig. 4.—Pulp and dentin of lateral incisor from patient 61 years of age; illustrating formation of dentinal substance by accretion of calcific bodies which have been formed within pulp. (X220.)

decreases toward the apical end of the tooth, and with it is seen a decrease in the thickness of the predentin. In some teeth, and especially in those which show fibrosis and some degree of calcification, the odontoblasts are completely lost in the radicular portion and a dentinal substance has been formed by an accumula-

incisor from a patient 61 years of age. The tooth was without caries, prostheses or pyorrhea. The pulp shows a marked decrease in the number of cells and a moderate degree of hyalinization, but no increase in the thickness of the vessel walls. In the depicted portion of the canal, there are no odontoblasts present,

tion of calcific deposits within the pulp which have become adherent to the dentinal wall. This dentinal structure is neither tubular nor hyaline, but globular in character. Figure 5 is a photomicrograph of the pulp and dentin of a lateral

but calcareous bodies have been formed within the pulp' and become adherent to the dentin wall. This dentinal material has been formed at the same rate of speed as the tubular dentin toward the coronal portion of the tooth, where odontoblasts

and predentin are seen. We interpret this condition as the formation of calcific deposits within the pulp which, by the increase in their size, have become part of

is frequently observed in adult teeth. This vacuolization is believed by Willman⁵ and others to be a regressive process which is often found with advancing age.

Fig. 6.—Reticular atrophy of pulp. (X 120.)

the dentinwall. Such Calcific deposits are to be more fully discussed later in this paper.

Vacuolization of ih wt Hastic layer

Similar vacuolization of odontoblasts has

5. Willman, Warren: Changes in Dentin and Pulp Produced by Caries and Dental Restorations, abstr, J. D. Res., 12:460 (June) 1932.

been described by Wolbach and Howe⁶ in experimental scurvy in rats. That such changes do occur there can be no doubt, but careful study of our material reveals the fact that histologically similar vacuoles can be produced by improper fixation of pulp tissue. Good fixation of pulp

pulps by immediate grinding of the side of the tooth under water until the coronal pulp was freely exposed and then placing it in 10 per cent formalin or Bouin's fixative. Realizing that immediate penetration of the pulp by the fixative is necessary, early in our study of pulps we

Fig. 7.—Penetration of caries through dentin and resultant inflammatory reaction in pulp. (XH0«)

tissue can seldom be attained by placing extracted teeth *in toto* in a fixative. We obtained satisfactory fixation of dental

cut off part of the radicular portion of the tooth. We found that, in the coronal horns, in many cases, vacuolization of the odontoblasts occurred over a normal thickness of predentin. This we believe to be an artefact due to poor fixation, a diffi-

6. Wolbach, S. B., and Howe, P. R.: Inter-cellular Substances in Experimental Scorbutus, Arch. Path. & Lab. Med., 1:1 (Jan.) 1926.

culty overcome in large part by free opening of the pulp. When with proper fixation vacuolization of the odontoblasts is found accompanied by a diminution in the amount of predentin, which indicates a decrease in the functional ability of the odontoblasts, the condition is clearly degenerative. Vacuolization of the odontoblasts is usually accompanied by reticular

accompanied by a diminution in the number of pulp cells.

Euler⁷ and Schneider⁸ have described cystic areas within the pulps of clinically intact teeth where the odontoblastic layer had separated from the dentin to form the cavity. Hammer,⁹ who has made a study of this condition, believes that the cysts which he has seen and which are

atrophy of the parenchyma of the pulp. Figure 6 illustrates early reticular atrophy, which consists of an increase in collagenous material and the formation of small vacuoles. As the severity of this condition increases, the vacuoles become larger and the collagenous material is aggregated into denser strands, always

formed by the dentin wall on one side

7. Euler, H. : *Über Zystenbildung der Pulpa*, Ztschr. f. Stomatol., 28:1019, 1930.

8. Schneider: *Zur Frage der Pulpacysten*, Deutsche Monatschr. f. Zahnheilk., 50:1089, 1932.

9. Hammer, H. : *Zur Frage Pulpacysten*. Deutsche Monatschr. f. Zahnheilk., 50:1089, 1932.

and the odontoblasts on the other, were without signs of degeneration of the surrounding cells and are the result of shrinkage due to technical methods. We have seen examples of such cysts in our preparations which, in addition to the

tioned normally and tend to confirm the conclusions of Hammer.

Hyperemia of the pulp easily and frequently results from thermal and chemical irritation following the loss of the protecting tissue. Clinically, hyperemia

lack of degenerative manifestations in the pulp tissue, had areas of predentin normal in width. These observations indicate that the odontoblasts had func-

of the pulp is characterized by paroxysms of pain on subjection of the tooth to irritating influences. Sudden rushes of blood into a tissue encased within rigid walls

cause a pressure which is relieved with the removal of the irritation and the subsequent equalization of blood pressure. These conditions are readily amenable to

There is little or no laboratory evidence that pulps die because of hyperemia produced by the thermal conductivity of large metallic restorations.

Fig. 10.—Calcification of hyalinized connective tissue. (X370.)

treatment by proper protection of the pulp. It is probable that long-continued hyperemia of thermal or chemical origin might result in degenerative changes.

Caries sufficiently deep to require restorations extensive enough to cause thermal shock is likely to result in bacterial invasion of the pulp. This is the most

important factor in the death of such pulps. Most forms of pulp inflammation and degeneration are the direct result of bacterial invasion of that tissue. Bacterial

times demonstrable. Henrici and Hartzell,¹⁰ in a study of pulps of carious teeth, found in all some histologic change, either inflammation or fibrosis. They concluded

Fig. 11.—Perivascular calcified bodies in pulp. (X14-0.)

occupation of dental tubules in advance of the necrotic areas of caries has repeatedly been shown and bacteria within the pulp under areas of caries are some-

times demonstrable. Henrici and Hartzell,

10. Henrici, A. T., and Hartzell, T. B.: Microscopic Study of Pulps from Infected Teeth, J.A.D.A., 8:213 (March) 1921.

ies over a long period before they undergo degeneration. It cannot be inferred that all histologic change found in pulps of teeth with caries is the direct result of bacterial invasion of the pulp, because some change may result from products of bacterial activity or the products of caries.

are comparable to inflammations in other parts of the body. Before actual exposure of the pulp occurs, sometimes even in superficial caries, infection of the pulp through dental tubules may take place and areas of inflammatory reaction are seen just beneath the odontoblastic layer.

Fig. 12.—Calcified mass showing calcification of marginal collagenous fibrils. (X385.)

It is evident that the proximity of caries to the pulp bears a direct relationship to the pathologic change found within the pulps.

The most common pathologic change found within dental pulps is inflammation. Inflammations of the pulp *per se*

Figure 7 is a photomicrograph of the carious portion of a tooth illustrating the penetration of caries through firm dentin and the resultant inflammatory reaction in the pulp. These reactions are at first confined to a small area of the pulp and are characterized by the presence of serous

Fig. 13.—Corpora amylacea in pulp of carious tooth. (XH60.)

exudate and of polymorphonuclear leuko- inflammation usually increases in severity cytes. Seldom does resolution occur. The and extent of area involved. It may be

manifested as a localized abscess immediately beneath the area of infected dentin of pulp infection is clinically accompanied or the inflammatory process may involve by prolonged pain which is exaggerated

Fig. 14/—Calcification around and in neighborhood of corpora amylacea. (XH60.)

the entire pulp. In areas of localized on the application of thermal or chemical pulp abscesses, bacteria are easily demonstrated, the severity of which gradually

decreases over a lengthy period of time.

This partial or localized pulpitis may result in slow progressive necrosis of the pulp provided the caries has caused an exposure and permitted an opportunity for accommodation of inflammatory swelling. Owing to the encasement of the pulp within unyielding walls and its consequent inability to accommodate vascular change, total pulpitis will result in stasis of the blood stream and thrombosis. Under these conditions, complete death of the pulp will occur in as short a period of time as forty-eight hours.¹¹ The clinical symptoms of total pulpitis are steady pain, increasing difficulty in localizing the tooth and a decreasing response to cold with an *increasing response to heat*. Figure 8 is a photomicrograph of a pulp of a deeply carious molar tooth. It has a localized abscess in the central portion and inflammatory cellular exudate throughout the coronal portion of the pulp.

The influence of silicate cements on the pulps of teeth was recently summarized as follows^{11 12}:

The death of pulps beneath silicate cement fillings has long been a clinical observation. The cause of the death of these pulps has been the subject of some controversy and, as yet, is only partially understood. The use of synthetic enamels is largely confined to anterior teeth. As the death of pulps beneath fillings is well known to be more frequent in anterior than in posterior teeth, it is quite possible that many pulps that have died beneath silicate fillings would have died beneath other fillings. There is little available evidence which helps to distinguish between pulps that die because of approaching caries and those that die because of

the irritation caused by the silicate cements. The theories that the pulps beneath silicate cements die because of the heat generated by the chemical action of phosphoric acid and the oxides of silicon, aluminum or calcium, or by the hygroscopic action of these cements during the setting period, have not been substantiated by scientific evidence.

Palazzi¹³ conducted a series of experiments by using silicate cements in the teeth of dogs. He found definite changes in the pulps after from twenty to forty days. These changes were manifested as hemorrhage, reticular atrophy and fibrosis.

Rohrer,¹⁴ in an article on the effects of silicate cements on protoplasm, agrees with Palazzi that silicate cements have an injurious effect on the dental pulp. He suggested that the results obtained by the experiments of Palazzi might be exaggerated because in the teeth of dogs cavities were mechanically made and consequently lacked the reactionary zone of dental caries.

G. Fasoli¹⁵ conducted a series of experiments on dogs by preparing cavities of various depths and filling these cavities with cement. After a period of time had elapsed, the pulps were examined microscopically. Some of his conclusions were:

1. Pulps exhibit no harmful reaction to the use of zinc phosphate cements.
2. The use of silicate cements in deep cavities produces hyaline and reticular degeneration and definite areas of necrosis in from thirty to forty days.
3. When silicate cements are used in cavities less deep, the pulp exhibits dis-

11. Romer, O.: Erkrankungen der Zahn-pulpa taken from "Die Pathologie der Zahne, p. 231, Handbuch der Speziellen Pathologische Anatomie und Histologie (edited by F. Henke and O. Lubarsch), Vol. 4, Berlin: Julius Springer, 1928.

12. Hill, T. J., and Van Natta, H. C.: Review of Researches on Protection of Vital Pulps, Proc. Am. A. D. Schools, March, 1932, p. 273.

13. Palazzi, S.: Über die Anatomischen Veränderungen Zahnpulpa in gefolge von Silikat-zement füllungen, Ztschr. f. Stomatol., 21:279, 1923.

14. Rohrer, A.: Action of Silicate Cements on Protoplasm, Brit. D. J., 45:196 (Feb. 1) 1924.

15. Fasoli, G.: Silikat-zemente und Pulpav-eränderungen, Ztschr. f. Stomatol., 22:225, 1924.

turbance of the capillaries with the production of hemorrhagic infarcts and the formation of areas of serous exudate in the central portion of the pulp. Abnormal production of secondary dentin is found in these teeth. In this series of experiments, the changes followed a slower

phoric acid could not be demonstrated to be the main factor. It was either absent or negligible after the cement had been allowed to set for fifteen minutes, but there was apparently a soluble acid salt of phosphoric acid present.

6. Some protecting substance must be

Fig. 15.—Calcification, showing nidi in pulp of carious bicuspid. $n > nidi$. (X?0.)

course and the remote portions of the pulp were unaffected.

4. The lesions in the pulp are not limited to the regions directly beneath the cavity.

5. Silicate cement contains some substance harmful to the pulp. Free phos-

used between the pulp and the silicate cement and oxyphosphate cements are entirely satisfactory for this purpose.

Internal resorption of the walls of the pulp chamber is occasionally found. It is not so common as external resorption of the root surface and seldom is it so exten-

sive. Its etiology is obscure. Most cases of internal resorption are repaired by the filling in of the lost area with a substance similar to cementum or bone. Figure 9 is a photomicrograph of a pulp of a lower lateral incisor having distal caries and advanced pyorrhea, from a patient 48 years old. The photograph is made of the central third of the root canal. The pulp exhibits no marked pathologic change. The

areas of resorption have taken place. The resorption has been largely repaired by replacement with bone. While the origin of such bone in the repair of the dentin may be controversial,⁶ it is worthy of note that, in this tooth, the bone can be traced along the sides of a lateral canal to a point near the cementum. This might support the belief that it has its origin in the peridental membrane, although

Fig. 16.—Calcification within pulp; showing atypical odontoblasts and small amount of predentin. (XI00.)

number of pulp cells and the amount of connective tissue are normal. A few small areas of calcification are present. The odontoblasts are lost, but this condition is not uncommon in this portion of the root canal at this age. The marked change is in the dentin, where extensive

there is nothing inconsistent in the theory that such conditions are the result of metaplasia.

Calcification within the pulp has been the subject of much discussion in relation

16. Euler, H.: Metaplasie der Pulpa, Vierteljahrsschr. f. Zahnheilk., 37:303, 1933.

to its importance as an etiologic factor in obscure facial neuralgia. In the microscopic examination of 132 teeth, we have found calcification in 80 per cent of the pulps. This percentage is in keeping with the observations made by Kronfeld. The age distribution in our examination of teeth is shown in the accompanying table.

It will be observed that calcification is common in young individuals and that there is a gradually increasing tendency to the condition with advancing age. We have observed these areas of calcification in unerupted and newly erupted teeth.

The great frequency of calcification within the pulp and the inability to inter-

relationship between pulp calcification and other systemic disease has not been definitely established. If it were possible to establish such a relationship, roentgenograms would be of aid in the recognition of only a few of the larger areas of calcification.

There are three types of calcification of the dental pulp,¹⁹ * each formed through different pathologic processes, the recognition of which is possible only by use of the microscope. In this paper, the use of the common terms for the products of these types of calcification such as denticles, pulp stones, pulp nodules and calculi, will be avoided, because no one is generally applicable to the various classes of calcification, which are:

1. Pulp calcification caused by an infolding of the odontoblasts during the development period. This infolded region becomes separated from the main body of the odontoblasts and islands of dentin are formed. This type of calcification results entirely in the formation of dentin. Such calcification within the pulp has been described by Fridrichowsky and by Wolbach and Howe ²¹ in vitamin A deficiency in rats. Similar infolding was previously described in vitamin C deficiency in guinea-pigs. It is probable that this is not an important type of calcification in human teeth because we seldom see calcification of the pulp resulting entirely in dentin formation.

2. Calcification areas within the pulp which are the result of calcification of hyalinized connective tissue. Figure 10

19. Ottolengui, Rodrigues, and Cahn, L. R.: Pathologic Conditions Discovered Within Pulps of Unerupted, Impacted and Imbedded Teeth, D. Items Int., 48:897 (Dec.) 1926.

20. Fridrichowsky, H.: Zur Histologie der Dentikel, Ztschr. f. Stomatol., 25:124, 1927.

21. Wolbach, S. B., and Howe, P. R.: Incisor Teeth of Albino Rats and Guinea Pigs in Vitamin A Deficiency and Repair, Am. J. Path., 9:275 (May) 1933.

AGE DISTRIBUTION OF CALCIFICATION IN PULPS OF ONE HUNDRED AND THIRTY-TWO TEETH

No. Teeth Examined	Age Years	No. Teeth Showing Calcification	No. Teeth Showing No Calcification	Percentage
9	10-20	6	3	66
30	20-30	20	10	66
16	30-40	13	3	80
46	40-50	38	8	82.6
20	50-60	18	2	90
11	60-70	10	1	90.9

pret roentgenograms with sufficient accuracy to recognize but a small percentage of them would lead one to question the results arrived at in an investigation of pulp calcification based wholly upon roentgenographic evidence.^{17 18} It is recognized that there is some clinical evidence to support the common belief that the removal of teeth showing pulp calcification has, in certain instances, relieved facial neuralgia. This relationship or the

17. Kronfeld, Rudolf: Histopathology of the Teeth and Their Surrounding Structures, Philadelphia: Lea & Febiger, 1933; p. 57.

18. Stafne, E. C., and Szabo, S. E.: The Significance of Pulp Nodules, D. Cosmos, 75:160 (Feb.) 1933.

is a photomicrograph of the pulp of a pyorrheal tooth from a patient 50 years old. It illustrates the calcification of hyalinized connective tissue. This type of calcification, which is usually perivascular or perineural, is frequently found associated with fibrosis of the pulp. An example

Similar calcification areas in a more granular form can be observed within the walls of the arterioles of the pulp in Figure 19.

3. Calcification which begins as a deposit of calcium salts in corpora amylacea. Figure 13 illustrates corpora amylacea²²

Fig. 17.—'Unstained ground section; showing irregularity of dentinal tubules. (X100.)

of this condition is found in Figure 11, a photomicrograph of a pulp of a pyorrheal tooth from a 50-year-old patient. Figure 12, a photomicrograph of a tooth from the same patient, shows a large calcification area with marginal calcification about the collagenous fibrils.

within the pulp of a carious tooth from a patient 29 years old. Corpora amylacea

22. Romer, O.: *Erkrankungen der Zahnpulpa, Die Pathologie der Zähne*, p. 266, *Handbuch der Speziellen Pathologische Anatomie und Histologie* (edited by F. Henke and O. Lubarsch), Vol. 4., Berlin: Julius Springer, 1928.

occur as concentrically lamellated bodies of irregular size and stain metachromatically, with methyl violet. Their origin is probably in degenerated nuclear fragments. Figure 14, a photomicrograph taken from the same tooth, illustrates a granular deposition of calcium around

content is distributed in uneven lamellation. When these calcification areas are cut through the center, a nidus is always demonstrable. Figure 15 is a photomicrograph of a pulp canal of a carious bicuspid tooth from a patient 47 years old. It illustrates extensive calcification of this type,

Fig. 18.—Calcification in pulp; showing well-organized odontoblasts, wide border of pre-dentin and regular dental tubules. (X130.)

and in the immediate neighborhood of the corpora amylacea. By concretion and by conglomeration, these deposits increase in size. The uneven staining in this type of calcification indicates that the inorganic

and also the nidi in those calcification areas that have been cut through the center. This is the type of calcification which was responsible for the building of a dentinal substance onto the dentin wall,

previously described in this paper and illustrated in Figure 5.

Most of the larger calcification areas contain dental tubules and at their margins odontoblasts are found. Euler,²³ Kronfeld²⁴ and Neuwirt²⁵ believe that these odontoblasts do not arise in the odontoblastic layer but are formed from the stellate cells of the pulp. These odontoblastic cells are usually of a lesser height and are either cuboidal or round. They form irregular dentin and, from the small amount of predentin usually present, one would believe the formation of dentin to be slow. That there is a tendency for odontoblasts to form around calcific areas in the pulp is further exemplified by the works of Neuwirt²⁵ and of Feldmann.²⁶ They demonstrated, in mechanical exposure of the pulp, where fragments of dentin had been forced into the pulp, that odontoblasts arising from the stellate cells of the pulp form around these fragments and further deposition of dentin is made. Figure 16 is a photomicrograph of the pulp of a carious molar tooth from a patient 29 years old. Small odontoblasts are seen over portions of the calcification area, which has a thin area of predentin. At other margins of this area, the increase in size appears to be by accretion and by calcification about the collagenous fibrils. Figure 17, an unstained ground section of an area of pulp calcification, illustrates the irregularity of the formation of dental tubules.

Calcification areas occasionally show well organized odontoblasts, the activity

of which is evidenced by the wide area of predentin and the regularity of the dental tubules. Such a condition is illustrated in Figure 18, taken from a 44-year-old patient who had pyorrhea. Figure 19 is a photomicrograph of a pulp of a molar tooth which had mesial caries and an occlusal filling, in a patient 19 years old. It illustrates the centers of calcification which have become fused and further united by the formation of a dentinal substance having tubules. This is the most common composition and arrangement of large calcification areas within the pulp. It also illustrates calcific deposits within the walls of arterioles, which have been previously described in this paper. Figure 20 is a photomicrograph of the pulp of an abraded molar tooth from a 42-year-old patient who had pyorrhea. It illustrates the extent to which calcification of the pulp occurs and the attachment of the calcification area to the dentin wall. This attachment is usually by the formation of dentin around the calcification area. Except for the calcification and some fibrosis, which is comparable to the amount usually found in the fifth decade of life, no marked pathologic change is observed in this pulp.

SUMMARY

1. Throughout life, there is a gradual histologic change in the pulps of teeth. The amount of embryonal tissue and the number of stellate cells become reduced. There is increased fibrosis, which is hastened by pyorrhea, caries and abrasion.

2. Hyperemia may be produced by thermal shock, but there is evidence that bacterial invasion is the most important etiologic factor in the death of pulps.

3. Formation of secondary dentin and tubular calcification act to retard the progress of caries.

4. The rapidity with which dentin is formed in adult teeth may be estimated

23. Euler, H.: Sekundäre Odontoblastenbildung, *Deutsche Monatschr. f. Zahnheilk.*, 45: 488, 1927.

24. Kronfeld, Rudolf: Footnote 17, p. 55.

25. Neuwirt, F.: Die Reparativen Fähigkeiten der Pulpa, *Ztschr. f. Stomatol.*, 31:291, 1933.

26. Feldman, G.: Neue Wege in den Therapie von Zähnen mit Entzündeter Pulpa, *Vierteljahrsschr. f. Zahnheilk.*, 48:211, 307, 1932.

Fig. 19.—Fusion of centers of calcification and union by tubular dentinal substance; calcific deposits within walls of arterioles. (X100-)

by the width of the area of predentin, which, when calcification is normal, is in proportion to the activity of the odontoblasts.

5. The pulps of teeth sustain injuries

from the indiscriminate use of silicate cements, which may be prevented by proper protection of the pulp.

6. Calcification within the pulp arises from three different sources, the resultant

Fig. 20.—Large area of calcification within pulp and its attachment to dentin wall. (X O.)

types being morphologically recognizable :

(a) Metabolic disturbances such as vitamin deficiency and the resultant infolding of the odontoblastic layer to form

islands of dentin.

(b) Calcification subsequent to and associated with hyalinization of connective tissue.

(c) Calcification of amyloid bodies.

7. Calcification of the pulp is usually associated with connective tissue which, in the pulp, is largely perivascular and perineural. Aside from this anatomic relationship, there is no evidence that calcification *per se* interferes with the blood supply to the pulp or with the nerves, to cause neuralgia.

The more common calcification of the pulp follows in the wake of degenerative processes. It might be possible that the etiologic agent which was responsible for

the degenerative processes preceding calcification is independently the cause of the neuralgia which is sometimes relieved by the removal of teeth. Since calcification of the pulp cannot be established as the cause for facial neuralgia and the degenerative processes which precede this condition are not roentgenographically demonstrable, it is evident that roentgenograms of the pulps are of little value in establishing the cause of obscure facial neuralgia.