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The Effects of Filling Materials and Caries on the Dental Pulp and Adjacent Hard Tissues

Warren Willman
Loyola University Chicago

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THE EFFECTS OF
FILLING MATERIALS AND CARIES
ON THE
DENTAL PULP AND ADJACENT HARD TISSUES

BY

WARREN PAGE WILLMAN, B.S.M., D.D.S.

A Thesis Submitted in Partial Fulfilment
of the Requirements for the Degree of
Master of Science
in
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VITA

Warren Page Willman

Born in Kankakee, Illinois, August 15, 1903.

Attended Kankakee High School, 1917-1921. James Millikin University, Decatur, Illinois, 1921-1923. Entered Chicago College of Dental Surgery, Dental Department of Loyola University, 1923. Degree of Bachelor of Science in Medicine, Loyola University, 1926. Degree of Doctor of Dental Surgery, Chicago College of Dental Surgery, 1927.

Instructor in Crown and Bridge Prosthesis, Chicago College of Dental Surgery, Dental Department of Loyola University, 1927-1928. Instructor in Periodontia, 1928-1930. Instructor in Operative Dentistry, 1930-1932. Associate and Junior Lecturer in Operative Dentistry, 1932-1933. Assistant Professor of Operative Dentistry, 1933-1934. Member of Chicago Dental Society, Illinois State Dental Society, American Dental Association. Member of the International Association for Dental Research. Member of the Odontographic Society of Chicago. Member of the Federation Dentaire Internationale. Member of the Omicron Kappa Upsilon Honorary Fraternity.
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INTRODUCTION
INTRODUCTION

The original objective of this investigation was to discover, if possible, the nature and extent of the changes which occurred in the tissues of the human dental pulp when the various types of filling material commonly in use were placed in the calcified structures of the teeth. Shortly after the work was started it seemed wise to include the effects of dental caries and other forms of external irritation, such as abrasion, erosion, or traumatic injury, in the study of pulp changes. At a still later date it became obvious that a study of the related changes in the adjacent calcified tissues would also have to be included while observing the pulp, in order to have the histopathologic picture reasonably complete.

The human dental pulp has been the subject of much curiosity and interest, both in its normal and pathologic states. Folklore teems with ludicrous and fantastic remedies for the excruciating pain produced by an inflamed or decomposing pulp, while the writings of the early physicians or dentists run it a close second describing their earnest but bungling efforts to provide relief. As scientific knowledge has gradually grown, it is not the least remarkable that this tiny organ, normally so well protected and entirely unfelt, should have become the object of much study and investigation.
In the past several decades the histology of the normal pulp has been elaborately worked out. Its mode of origin from the original mesodermic tissues is thoroughly understood, and its normal adult form has been minutely, even if somewhat too idealistically, described. Embryonal stellate or spindle-shaped connective tissue cells have been shown to form the most obvious bulk of the organ, lying in a matrix of gelatinous ground substance and shot through with arteries, capillaries, and veins that are endowed with exceptionally frail endothelial walls. The nerves, which uniquely identify the organ to the laity, have been followed most carefully from their entrance at the apical foramina to their end arborizations between the odontoblasts and even into the dentin. The odontoblasts themselves, though no longer burdened with dentin formation, have received much attention since they send out their protoplasmic attenuations, the Tomes' fibers, into the tubules of the dentin, thus linking the pulp with its surrounding structures. And in recent years other, newly observed structures have been dealt with, such as the histiocytes and other defense cells, found in the pulp as well as in various regions of the body, and the argyrophile fibers, now thought to be primarily concerned with dentin development.

In addition, pathologists, and especially dental pathologists, have devoted much energy to the various aberrations of the pulp. The effects of acute and chronic inflammation
have been noted, along with the results of injuries and infections. And in very recent years, much work has been started in the direction of correlating these effects with whatever causes may have anteceded them.

It has long been known, especially clinically, that certain of the changes that occur in the pulp tissues take place after dental restorations, such as fillings, have been placed in the teeth. Just what changes occur, and when and to what extent had not been fully determined, so the present objective was planned with that intention. The writer will confess that at the outset, particularly of the experimental work, he was naive enough to hope that a definite and different deformation of the pulp cells would be observed under each type of filling material, and that he would so be enabled to make a brilliant report of startling findings to the profession. Alas, as Gottlieb has said, "He who would eat of experimental bread has a hard crust to chew!"
CHAPTER I

REVIEW OF THE LITERATURE
CHAPTER I

One of the earliest and most colorful of genuinely scientific writings on the subject of the human dental pulp is to be found in the Treatise on the Natural History and Diseases of the Human Teeth by John Hunter, first published in 1771. He describes the pulp chamber under the heading, "Of the Cavity of the Teeth", in this way:

"Every tooth has an internal cavity, which extends nearly the whole length of its bony part. It opens or begins at the point of the fang, where it is small; but in its passage it becomes larger, and ends in the body of the tooth; the cavity at this end is exactly of the shape of the body of the tooth to which it belongs, and in general it may be said that the whole of the cavity is nearly of the shape of the tooth itself, larger, that is, in the body of the tooth, and thence gradually smaller to the extremity of the fang; Simple where the tooth has but one root, and in the same manner compounded when the tooth has two or more fangs.

"This cavity is not cellular, but smooth in its surface: it contains no marrow, but appears to be filled with blood-vessels, and, I suppose, nerves, united by a pulpy or cellular substance. The vessels are branches of the superior and inferior maxillaries, and the nerves must come from the second and third branches of the fifth pair.

"By injections we can trace the blood-vessels distinctly through the whole cavity of the tooth; but I never could trace the nerves distinctly even to the beginning of the cavity (12:25-26)."

It is indeed remarkable that Hunter was able, with the meager means at his disposal, to keep so accurately on the narrow path of scientific thinking. Amidst all the error and
superstition of his day, he found it possible to be simple enough to stick to facts and describe what he had observed: "This cavity appears to be filled with blood-vessels" (he had seen them) "and, I suppose, nerves, (deduced presumably from the known sensitiveness of the organ) "united by a pulpy substance (loc. cit.)." This is probably the earliest approach to the present term of pulp for this organ.

A little farther on in the same treatise Hunter gives the first description of the formation of secondary dentin, laid down in response to extreme abrasion, in a paragraph entitled, "Of the Cavity filling up as the Teeth wear down":

"A tooth very often wears down so low that its cavity would be exposed if no other alteration were produced in it. To prevent this, Nature has taken care that the bottom part of the cavity should be filled up by new matter in proportion as the surface of the teeth is worn down. This new matter may be easily known from the old, for when a tooth has been worn down almost to the neck, a spot may always be seen in the middle, which is more transparent, and at the same time of a darker colour (occasioned in some measure by the dark cavity under it), and generally softer than the other. (Plate 1) Any person may be convinced of the truth of these observations by taking two teeth of the same class, but of very different ages, one just completely formed, the other worn down almost to its neck. In the last he will observe the dark spot in the centre, and if as much is cut off from the complete tooth as has been worn off from the old one, the cavity of the young tooth will be found cut through; and on examining the other its cavity will be found filled up below that surface. Now this observation contradicts the idea of the hole leading into the cavity of the tooth being closed up; and what is still a further proof of it, I have been able to inject vessels in the cavities of the teeth in very old people when the alveolar process has been gone, and the teeth very loose in the gum (11:50-51)."

Figures 25 and 26 are drawings of abraded teeth showing the darker and more translucent secondary dentin filling the space formerly occupied by the pulp chamber.
During the end of the eighteenth century and the beginning of the nineteenth, the dental writers followed very largely the fundamental ideas laid down by Hunter. In the middle of the nineteenth century, however, a new era of research was ushered in by the introduction of the compound microscope. This period has well been called the birth of modern histology.

Among the dental investigators of this time who wrote extensively on the early findings by the use of the microscope was John Tomes who published *A System of Dental Surgery* in 1859. In it, in a long chapter entitled "The Dental Tissues", he describes the character of the dentin, as then understood, stating that up to that time the dentinal tubules were considered by others, such as Kölliker, to contain clear fluid during life, which, he says, "appeared to indicate so satisfactorily the offices of these canals, that the subject was regarded as one which had been fully investigated (20:324)." However, he further observed that freshly denuded dentin was exquisitely sensitive while the dentin of a pulpless tooth was never so. "These considerations indicate sufficiently clearly", he argues, "that the sensitiveness of the dentine is dependent upon its connection with the pulp of the tooth, and that it has no inherent sensibility of its own hard tissue (20:326)."

Tomes anticipated some of our best modern research when he added, "After a portion of dentine has been for some time exposed, or if the exposure be brought about gradually by the
slow wearing away of the enamel, that acute sensitiveness which has been described is not then found to exist. In parts which have been subject to the foregoing conditions, it will on examination be found that the dentinal tubules the peripheral extremities of which have been exposed, are more or less obliterated in some part of their course between the surface and the pulp cavity (20:326).

The obvious discrepancy of these facts with the notion that the tubules contained only color fluid ("inasmuch as we have no instance of sensation being manifested in a fluid") led Tomes to investigate this subject further. "I had", he announces, "little expectation of finding that one of the most important points in dental structure had been overlooked, namely, that each dentinal tube is permanently tenanted by a soft fibril (20:327)." (Plate 2.) This was the first description of the dentinal fibrils which now so frequently are termed Tomes' fibers.

In another work Tomes observed that in persons dying of suffocation the dentin frequently was stained red, showing this color through the enamel. He deduced that the blood was decomposed so that the pigment of the erythrocytes was free in the serum. "The tubuli of the teeth", he says, "though too small to admit the red globules of the blood, freely admit the liquor sanguinis, and, if this be colored red, the tooth itself will necessarily take the same hue (21:120)." This shows
Plate 2. - Facsimile of figure 125, Tomes, (20:330).

It shows the dentin at d, the odontoblasts at b, and the then newly discovered dentinal fibrils (Tomes' fibrils) at c.
how well Tomes had divined the permeability of the dentin, which has only recently been demonstrated experimentally.

He also brought out that caries of the dentin produced a vital reaction, just central to the lesion, consisting of a translucent zone. "The dead is not separated from the living part of the tooth", he says, "but it is circumscribed by the consolidation of the living that is next to the dead (21:203)."

And a few paragraphs later, "Nature erects a barrier, more or less perfect, to save the pulp from irritation (21:205)."

Farther on he mentions the role of secondary dentin in this respect also. "An attempt is made", he states, "to keep out the disease, by placing a barrier of secondary dentin between the pulp and the spot towards which the disease is advancing (21:206)."

In 1869 C. Wedl published his excellent atlas of the Pathologie der Zähne. In it are sixteen plates, each with ten or twelve engravings of exceedingly good quality. Two of these are of particular interest, dealing with the formation of secondary dentin (23:22). The first shows a longitudinal section of an upper cuspid which was highly abraded on its lingual surface and decayed on its labial surface. In the incisal portion of the pulp chamber secondary dentin is to be seen, and on the labial wall of the chamber a "corresponding .... new-formation of dentine is attached (23:23)." The second shows a section of an upper central incisor with proximal caries. Wedl describes
"A new formation of dentine bending into the pulp-cavity and united with the original dentine on the part corresponding to the caries (23:24)."

In 1894 C. F. W. Bödecker brought out his large Anatomy and Pathology of the Teeth. A short chapter on the normal pulp is summarized by the results:

"I. The dental pulp is a variety of connective tissue termed myxomatous, representing an embryonal form of it. Some authors have called this form of tissue "adenoid", but this term is admitted to be erroneous.

"II. The myxomatous tissue of the pulp is intermixed with a delicate fibrous connective tissue in varying amounts.

"III. The pulp-tissue is traversed by a closed system of blood-vessels, viz., arteries, veins, and capillaries. At least one artery is invariably found in the pulp, and it is by no means of exceptional occurrence that the pulp contains several arteries. Lymphatics in small numbers are also present.

"IV. The pulp-tissue is richly supplied with nerves, which, in the shape of bundles of medullated nerve-fibers, traverse the myxomatous tissue. Toward the periphery of the pulp they lose their myelin sheaths, become non-medullated, and, in the shape of minute beaded fibrillae, branch between the odontoblasts.

"V. The odontoblasts at the periphery of the pulp are elongated protoplasmic formations with rows of nuclei. They are medullary corpuscles such as we see wherever a new tissue arises from a former one. They build up the basis-substance of the dentine by solidification (transformation into glue, and infiltration with lime-salts). The reticulum of living matter traversing the odontoblasts remains unchanged in the basis-substance of the dentine.

"VI. The dentinal fibers originate between the
odontoblasts. Being formation of living matter, they are in direct connection with the reticulum of living matter, first of the odontoblasts and afterward of the basis-substance of the dentine. The connection between the ultimate nerve-fibrillae and dentinal fibers is very probably an indirect one by means of the intervening reticulum of living matter (6:253-54).

In a later chapter Bödecker takes up the morbid anatomy of pulpitis, describing acute non-purulent pulpitis, purulent pulpitis, chronic pulpitis, chronic purulent pulpitis, and gangrene of the pulp at considerable length. (6:397-411). This is followed by two chapters on degenerations and atrophies of the pulp, which describe loosely the findings on calcifications of the pulp (plate 3); a process termed "eburnification and ossification of the pulp"; "sclerosis" of the pulp; reticular atrophy, first ascribed to C. Wedl; and "atrophy of the nerves". The text indicates that much observation and study had preceded its writing, but it also contains much material that is speculative and fanciful. (6:412-31).

In 1903 W. D. Miller brought out much evidence in support of the theory that the transparent zones in the dentin, first described by Tomes (op. cit.) represented a truly vital reaction on the part of the dentin and not a decalcification, as was commonly asserted at that time by some writers. He stressed, first of all, the necessity of correct interpretation of specimens and pointed out certain treacherous artefacts that closely resemble transparent dentin. (13:254). He then described in detail experiments he had made with several
Plate 3. - Facsimile of figure 163,  

This shows a ground section of a tooth with the enamel, E, abraded so as to expose the primary dentin, PD. A deposit of secondary dentin, SD, is shown in the incisal portion of the pulp chamber.
acids in various dilutions in an attempt to produce transparent zones artificially. These failed, as he anticipated. (13: 256-58). He also tells of attempts to make analyses of the material taken from these zones, to show that they were not decalcified. The results from two series, one run by himself and one by a Dr. Jeserich, a Berlin chemist, show a slight excess of calcium present instead of a decalcification, in the average of results. (13:264). Their methods of working were minutely accurate for that time but would be crude by present day standards of microanalysis. That may be why Miller failed to interpret the slight excess of calcium found (0.7%) as meaning that these zones were actually hypercalcified, as is believed nowadays.

G. V. Black, in his text-book, Special Dental Pathology, devotes fifty pages to the histology, functions, and diseases of the dental pulp. (4:235-85). He states that "the most common diseases to which the dental pulp is liable are hyperemia and inflammation (4:248)." To these he adds hypertrophy, traumatisms, and calcifications. Black raises objections to extended classifications of inflammations of the pulp on the grounds that they are distinguishable only microscopically and not clinically. (4:249). He describes his long-fixed habit of noting down the symptoms of diseased pulps along with the condition of the tissue he anticipated finding, and his results when he correlated these with the actual microscopic findings.
"My conclusion", he says, .... "is that inflammations of the pulp in earlier stages can not be differentiated from hyperemia; furthermore that it is impossible to determine from the symptomatology, whether or not inflammation exists in the pulp (4:250)."

In the following chapter Black describes hyperemia, its symptoms, the changes that occur in the blood-vessels when it becomes more insistent over a prolonged period, and the sequela, usually death of the organ by stagnation, called infarction. (4:254-57).

In the next chapter he deals with inflammation of the pulp due in most cases, as he says, to dental caries. He describes the pathologic changes, though not in any detail, microscopically, and the symptoms and signs upon which to base diagnosis. (4:258-64).

The remaining twenty pages are devoted to the calcifications in the pulp chamber and their effects upon the pulp tissue. He classifies calcifications into two main groups: (1) those attached to the walls of the pulp chamber and (2) those free in the tissues of the pulp. In the group of attached calcifications he makes three distinctions: (a) growths in which the tubules are continuous with the primary dentin, i.e., secondary dentin, (b) growths beginning as secondary dentin but in which the tubules progressively disappear, and (c) growths which are non-tubular from the beginning. He remarks that
"in any of these, calcospherites, or small nodules, which have previously formed free in the tissues of the pulp may occasion­ally be included (4:265)." It will be seen that this classifi­cation is followed closely by the more recent one of Euler and Meyer which will be considered presently.

In the group of free calcifications Black makes four dis­tinctions: (a) nodular formations, usually confined to the bulbous (coronal) portion of the pulp, (b) fusiform calcifica­tions, in the root portion of the pulp, (c) jointed calcifica­tions in the root portion, and (d) more extensive calcific growths which fill up the pulp chamber and, more or less, the canals, especially in molars. (4:266).

In the discussion following this classification Black says of secondary dentin, "These calcifications occur under many conditions, most of which are abnormal. They occur oftenest ... in cases of abrasion and erosion (4:269)." He obviously the considers abrasion entirely pathological, contrary to/present tendency to discriminate between physiological and patholog­ical, or excessive, abrasion, for he states it as his conclu­sion from observations that "extensive abrasion of the teeth is in a degree hereditary (4:268)."

Black does reason that "the physiological import would seem to be that the growth of secondary dentin is a response to irritation of the dentinal fibrils, and has a definite in­tention of placing the soft tissues of the pulp farther from
the source of injury, and thus protecting it (4:268)." But curiously enough he comes to the conclusion that secondary dentin formation is not "a local formation, confined mostly to the protection of the pulp over the area which is threatened by the injury to the dentinal fibrils", but is a generalized formation in all of the teeth of the individual due to a reflex impression "made upon the nerve centers by the irritation of the dentinal fibrils (4:269)." This error seems incredible, coming from so good a scientist as Black, until one realizes that all of his microscopic specimens were either dry ground sections or sections of embedded pulps without the adjacent dentin. Had he been able to study decalcified sections, such as those described in the present investigation, he could not have slipped into such a fallacy.

In discussing the effect of this secondary dentin formation on the primary dentin and enamel, Black says that while the "secondary dentin may grow and narrow the pulp chamber without affecting the dentinal fibrils", the dentin will die if these fibrils drop out and a clear calcification begins. (4:271). Biologically this is true, of course. Modern investigators often refer to this "dead tract". But in sharp contradiction of present concepts Black says, "Those tubules which are exposed to the saliva become filled and soddened with the materials of decomposition which occur in the mouth, and in time the whole of this area of dentin becomes softer than
normal (4:271)." He even adds that such softening of the dentin "renders the enamel much more liable to break away from it than from healthy dentin."

Oddly enough he goes on to observe that in central ground sections through an area of exposed tubules, the tract they occupy is darker by transmitted light. (4:271). He apparently never noticed that these same areas appear lighter by reflected light, as we shall see later, and he never dreamed that they were harder, not softer, than the adjacent dentin.

In his discussion of the effect on the pulp, Black observes that as calcification increases in amount, the pulp degenerates and becomes "more distinctly fibrous until its structure is greatly changed. As this goes on, the pulp usually becomes insensitive and fails entirely to respond to temperature changes. The condition is a more or less complete loss of function by the pulp." He then immediately adds what seems a gratuitous assumption, "Finally death of the remaining portions of the pulp occurs (4:273)."

In writing of the free calcifications, Black discusses his findings of them, as to the age frequency and their distribution in the pulp, and concludes that they can also strangle a pulp by their numbers. He astutely observes, however, that they occur oftener without painful symptoms than with, and says that his studies "indicate that the growth of these calcifications produces no particular symptoms (4:276)."
Among modern investigators, the work of E. Wilfred Fish of London is especially related to our present experiment. Besides research into the vitality of the enamel, with which we have not concerned ourselves, he has studied experimentally the circulation of the tissue fluid in the dentin, between the Tomes' fibers and the walls of the tubules (10:21-32), the calcium metabolism of the dentin itself (10:33-50), the reactions of the dentin to peripheral injuries (10:51-61) (which has come to be included as part of our present investigation), and the reaction of the dental pulp to peripheral injuries of the dentin (10:62-77) (our original intended subject).

The larger part of Fish's work was done by means of experimentation on monkeys and dogs at Hale Laboratory, Royal Dental Hospital, London. Human material was used when possible for correlation, but not, of course, experimentally in vivo. The findings and conclusions of Fish will be noted in a later chapter along with the findings of our specimens.

An American research worker who has been able to discover a great deal of information by a thorough study of ground sections of human teeth is Theodore B. Beust of Louisville, Kentucky. He has found that, not only in response to external irritation but even as age advances, a consistent change takes place in the dentin which he terms sclerosis. (1:1060). This hardening consists of the filling up of the tubules with calcium salts so that they show darker than the adjacent tubules.
by transmitted light, due to the more unbroken surface they present. (1:1060). Sclerosis may occur in isolated tracts, Beust has found, especially when the peripheral ends of the tubules of such a tract are exposed by any means, but with increasing age it tends to occur rather generally throughout the dentin, beginning at the coronal surface and extending toward the pulp. Since this occurs regularly and, apparently, physiologically in teeth, Beust has given the term maturation to the process. (2:2186). This study of sclerosis and density changes in the dentin has recently become the center of much interest because of the relationship between it and the onset and progress of caries. The findings of Beust will be discussed again, in another chapter.

In the *Pathohistologie der Zähne* by Hermann Euler and Wilhelm Meyer of Breslau, Germany, there is a classification made of the different forms of secondary dentin. Their Class I is that of secondary dentin which differs from the primary dentin only by a slight angle of deviation that all the tubules make in passing a given line, which demarcates the end of the primary. (7:38). (Plate 4). This is in sharp contradistinction to the definitions of Fish, who would call this irregular dentin still primary. Fish recognizes as secondary dentin only that which is laid down centripetally to an observed calcium barrier, and which is not in fluid communication with the primary dentin, as that of Class I of Euler and Meyer undoubtedly

Irregular dentin, called Class I by the authors. The section is taken from an upper first molar. D is primary dentin, ID irregular dentin.
Class II is given as the more wavy or irregular type of secondary dentin with unusually abundant lateral anastomosing branches. (7:39). (Plates 5 and 6).

Class III is shown as a type of secondary dentin wherein the tubules are greatly reduced in number, giving it a more homogeneous or hyaline appearance. (Plate 7). This is teleologically described as being a hurried and consequently rather poorly formed structure. (7:39-40). This view is further substantiated by the occasional inclusion of an entire odontoblast with its dentinal fibril still in the primary dentin. (7:43-44). The few other odontoblasts that survive the original stimulus or injury recover and extend with the new deposit, forming the scattered tubules that it does contain, and ultimately lie adjacent to its inner surface. (Plate 8). According to the photomicrographs in their book, only this latter class would meet Fish's definition of true secondary dentin. New dentin laid down without the prior formation of a calcium barrier is called "additional primary dentin" by Fish (loc. cit.). (Plate 9).

Some interesting experimental research in the production of secondary dentin is being done at the present time by B. Orban and Joseph Weinmann, under the direction of Bernhard Gottlieb at the University of Vienna. Weinmann has shown that by feeding rats high or even toxic doses of vitamin D he can
Plate 5. - Facsimile of figure 40, Euler and Meyer, (8:42).

This section shows Class II secondary dentin. It is taken from an upper first bicuspid. D is the primary, ID the secondary dentin. The greater amount of branching of the tubules can be plainly seen.
Plate 6. - Facsimile of figure 41, Euler and Meyer, (8:42).

This also shows Class II irregular dentin. A more wavy type of tubules with much lateral anastomosing is illustrated.
Plate 7. - Facsimile of figure 42, Euler and Meyer, (8:43).

This illustrates these authors’ Class III irregular dentin. D is the primary dentin, ID the secondary. The diminution in number of the tubules is to be noted. At E.O. an odontoblast was covered and enclosed by the rapid formation of new dentin.
Plate 8. - Facsimile of figure 44, Euler and Meyer, (8:44).

Upper first molar. The irregular dentin encloses odontoblasts. D, normal dentin; PD, predentin; O, groups of odontoblasts enclosed in the predentin; P, pulp.
Plate 9. - Facsimile of figure 45, Euler and Meyer, (8:45).

Mesiodistal section of an upper central incisor. Extensive production of secondary dentin has followed abrasion of the incisal edge.
produce a hypercalcification of the dentin. (24:1216). In order to determine whether any such changes could be produced by local application of vitamin D he also cut deep cavities in the teeth of dogs and sealed crystalline vitamin D (Calciferol) in the cavities with cement. At the end of three months a new layer of secondary dentin was produced in each tooth. This new layer plainly showed over-calcification, indicated by the excessively heavy staining it took. (24:1218). Vitamin D, given per mouth to rats, has been shown to produce over-calcification of all hard tissues, and in this case its local application had the same effect on the secondary dentin of the dog. (24:1219).

These zones of over-calcification correspond very closely to the hypercalcific zone or barrier described by Fish as occurring between primary and secondary dentin. (10:56-61). The application of Calciferol caused this zone to be much more pronounced. Weinmann concludes that these experiments, up to the present time, show that vitamin D can cause not only typical changes by way of general calcium metabolism, but also local changes in the primary dentin, and further, that by its use a wide, intensely calcified barrier may be produced. (24:1220).

Orban has been experimenting on dogs, producing secondary dentin by the application of varying percentages of paraformaldehyde. When strong (50%) paraformaldehyde was placed on the
surface of the exposed dentin, the pulp tissue became inflamed. (15:443-46). When, however, the paraformaldehyde was mixed with zinc oxide in concentrations of 10 per cent or less and the resulting powder made into a water-base cement, it could be placed in a cavity having no pulp exposure and the pulp tissues would remain vital, though showing microscopically some signs of degeneration. (15:316). What was the more remarkable was that gigantic deposits of secondary dentin developed in four to nine months. Cavities filled with plain cement or amalgam were used for controls. The comparative results can easily be seen in plate 10. The deposit of secondary dentin at B was laid down in response to a 2 percent paraformaldehyde filling which was in place for five months. The control tooth, taken from the same dog, contained an identical cavity which had been filled for the same period with amalgam. Although the pulps are identical in appearance, no secondary dentin is to be seen. (15:390).

Plate 11 shows two teeth from another dog. In A plain cement filling was placed and in B the cement powder was mixed with 4 percent paraformaldehyde. Both were left in place for nine months. The astonishing difference in results is instantly apparent. (15:395).

Orban has found that as high as 20 percent of paraformaldehyde may be used if removed within a day or less and plain cement or amalgam substituted. This will cause secondary den-

Figure 6 (left) shows at B a heavy deposit of secondary dentin in a dog's tooth, resulting from a filling of cement containing 2% paraformaldehyde, placed in the cavity, a, for five months.

Figure 7 (right) is a control tooth from the same dog. The cavity, a, was filled with amalgam during the same period.

A shows a section of a dog's tooth which had had a cement filling in the cavity for nine months.

B shows another tooth from the same dog which had had a filling containing 4% paraformaldehyde for the same period. a, cavity; d, line of demarcation between the primary dentin and the heavy deposit of secondary dentin; c, pulp.
tin production but not marked pulp degeneration. (15:434). It seems feasible to allow a percentage of 10 or less to remain in a cavity indefinitely. The experiment has been performed on nine dogs, using 210 teeth in all. The effect on human pulps and dentin is still open to investigation.

In Milan, Italy, two men have been carrying on extensive research work on the pathology of experimental pulp lesions of all kinds. These are Professor G. Fasoli, director of the Dental Institute of the University, and Silvio Palazzi, the present director of the Dental Institute of the University of Pavia, who has worked under his direction there for the past thirteen years.

Fasoli has experimented chiefly with silicate cements. It has been claimed that the damage to pulp tissues which so often results from the use of these materials is due to a release of free phosphoric acid from the filling after it has set. Fasoli has found that this is not the case, because if a sensitive reagent, such as methyl orange, is used to detect phosphoric acid, it is found that there is no trace of it free after the silicate has set. It is his opinion that the damage is caused by a formation of soluble acid salts, because the presence of such acid salts can easily be demonstrated chemically. (9:226).

Fasoli reports a series of experiments made on dogs, extending over a period of from one to six months. Experimental
cavities were cut in the canine teeth (analogues to cuspids in humans) and filled, some with silicate cement, others with oxy-
phosphate cement. Untouched teeth were taken for controls. He 
states that under oxyphosphate cement fillings, regardless of 
the depth of the cavity, there was a formation of secondary 
dentin, with the number and size of the underlying odontoblasts 
reduced somewhat. Otherwise the pulps were intact and appar-
ently unaltered. (9:234). (Plate 12).

Under silicate cement fillings, however, Fasoli found a 
much more marked reaction of the pulp. (9:232) (Plate 13): (a) 
In the case of shallow cavities there was extensive formation 
of secondary dentin, and there was hyperemia of the pulp with 
minute hemorrhages. Cystic spaces were sometimes found in 
the center of the pulp. (b) In the case of deep cavities 
there were extensive diffuse hemorrhages. In some cases the 
changes ranged in severity from hyaline degeneration of the 
pulp to complete destruction. (9:231-33). These experiments 
were made with many types or brands of silicate cements in-
cluding some of the most modern. Fasoli summarizes his ob-
servations and conclusions as follows:

1. Oxyphosphate cements do not damage the pulps in dogs' 
teeth, even when placed in deep cavities.

2. Silicate cements do cause severe pulp changes. In 
deep cavities reticular and hyaline degeneration and beginning 
pulp necrosis may be observed as soon as 30 or 40 days after
Plate 12. - Facsimile of figures 7 and 8,
Fasoli, (9:234).

Two dog's teeth containing cavities filled with oxyphosphate of zinc cement for six months. The cavity on the right was cut somewhat deeper than that on the left. Both show secondary dentin, SD, formed in response to the injury of the cavity preparation. Otherwise the pulp remained normal.
Plate 13. - Facsimile of figures 3 and 4.

Fasoli, (9:232).

Two dog's teeth containing cavities filled with silicate cement fillings for five months. Besides the deposit of secondary dentin, SD, the pulp on the left shows marked hyperemia, hemorrhages in the odontoblastic layer, and a serous space in the center of the pulp. The pulp on the right shows diffused miliary hemorrhages with large infarcts toward the left and a large serous space in the center with centrifugal compression of the surrounding pulp tissue.
as poisons to the dog's pulp, causing hyperemia and hemorrhages in the pulp with various forms of degeneration and finally necrosis. He terms these conditions, "hemorrhagic pulpiteis"; no bacteria are present. (18:257).

Recently Stafne and Szabo of the Mayo Clinic, at Rochester, Minnesota, have published results of research on pulp calcifications done by consulting the files of dental roentgenographs. They brought out that pulp nodules, which have been the subject of so much discussion, are found in teeth of nearly half of essentially normal patients. (19:163). They reduced their findings from 500 patients to two tables. The first listed many diseases, including cholecystic and renal disease with lithiisis, arthritis, and trifacial neuralgia, and figures stating how many of the patients suffering from each disease had pulp nodules and how many did not. "Any deductions made from this table", is their conclusion, "must be that pulp nodules are probably not important as etiologic factors in the production of systemic disease (19:163)."

The second table was made to show the possibility, if any, of these nodules being associated significantly (that is to say broadly, as a result rather than as a cause) with disease elsewhere than in the dental pulp. This table gives the number of cases studied, starting with 200 "essentially normal" and continuing, in most cases, with 100 cases each of the various diseases, and gives the percentage of cases with pulp nodules in
each instance. This is 46% for the normal and ranges from 42% (renal lithiasis) to 57.1% (acromegaly) for the different diseases. The authors state that the significance of these rather small variations "is largely problematic (19:163)."

C. F. Bödecker (son of C. F. W. Bödecker) and Applebaum have written recently on the variations in permeability of the dentin. Much of Bödecker's work, like that of his father, lies in the realm of speculation, and the paucity of his data sometimes makes his deductions very questionable. In this article he sums up three views extant concerning the reaction of the dentin due to peripheral injury: (1) the vital reaction, or the sclerosis of the dentin due to an activity on the part of the fibrils, (2) the death of the fibrils in the affected tubules, leading to their encapsulation by secondary dentin centrally and sclerosis of sound tubules laterally (Bödecker ascribes this theory to Fish, but, as will be shown later, Fish also includes the first in his presentation), and (3) a metamorphosis, or change, in the nature and permeability of the affected dentin. (5:1178-79). He commits himself to the latter theory, saying that "We believe that many fibrils in such areas have undergone a fatty metamorphosis, thus filling the tubules with fatty-gaseous casts (5:1180)."

In discussing the zones to be found in the dentin under a carious cavity, i.e., the transparent zone, the "dead tract" of tubules (according to Fish), and the secondary dentin, he makes
one important observation, well supported by the evidence. Namely that the oft-described transparent zone actually consists of two zones, an outer decalcified one and an inner hypercalcified one. (5:24). He submits the results of different tests to prove this, one of the best being a comparison of a photomicrograph of a ground section of a tooth made by transmitted light and a roentgenograph of the same section enlarged to identical over-all dimensions. By transferring the sharp line in the roentgenograph (plate 14, A) where the decalcification ends to the photomicrograph (plate 14, B), it may be seen that this precisely divides the translucent zone in the latter in two parts. This shows that only the inner part of this zone is hypercalcified, while the outer part is actually completely decalcified.

The left shows a roentgenogram of a section of a cuspid. The decalcified area is sharply bounded where the arc is drawn in ink. The right is a photomicrograph of the same magnification of the same ground section. By a transfer of the ink line, it may be seen that the decalcification ends in the transparent zone, TZ, instead of at its edge.
CHAPTER II

EXPERIMENTAL INVESTIGATION
CHAPTER II

As a first step toward determining the reactions of the pulp, a record sheet was composed and printed (plate 15) in such a way that the person engaged in obtaining specimens could gather accurate and exact information about a number of points in the history of both the patient and the tooth. All the possibilities or alternatives in each item were printed out with little ballot squares opposite, so that only a check mark was needed to complete the notations.

Junior students were assigned each day to watch in the extraction room of the college clinic for suitable teeth. They were instructed to retain only teeth that had vital pulps, dental restorations, and that could be obtained with a satisfactorily complete history from the patient. The kind and age of the restoration, the designation of the tooth itself, whether or not it had been sensitive to chemical or thermal changes and if so when, were noted on the chart. A roentgenograph was taken before the extraction, and after the tooth had been removed the apical one-third of the root was cut sharply off to give the fixing fluid better access to the pulp tissue. The tooth was then dropped into either 10% formalin solution or Zenker-formol, which was also recorded.

In this manner one hundred and twenty-five teeth were
Research in Tissue Changes of Vital Pulps

CASE HISTORY

1. PATIENT

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2. GENERAL CONDITION OF MOUTH

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<th>Extensive</th>
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<tr>
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<td>Slight</td>
<td>Extensive</td>
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<td>Slight</td>
<td>Extensive</td>
</tr>
<tr>
<td>Erosion</td>
<td>Absent</td>
<td>Slight</td>
<td>Extensive</td>
</tr>
<tr>
<td>Hypoplasia</td>
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3. INDIVIDUAL TOOTH

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<tr>
<td></td>
<td></td>
<td>Mercurial chiorid</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Silver nitrate</td>
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<td>Phenol</td>
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<table>
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4. EXPERIMENTAL FILLINGS

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<tr>
<td>Medium</td>
<td>Hypersensitive</td>
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<tr>
<td>Deep</td>
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</tbody>
</table>

Plate 15. - Reproduction of the history sheet used in collecting human specimens.

Original blank measured 11" x 8½".
collected, by students, my various associates, and myself. On three occasions we were fortunate enough to be able to place fillings in vital teeth of patients some weeks before those same teeth were extracted. The delay involved made this procedure insuperably difficult, or we should have experimented further in this way.

Since we were denied human material, however, we made use of canine. Experimental work on animals is always open to criticism on the basis that we have no positive assurance that a given experimental result in the laboratory animal is necessarily paralleled by a like result in the human tissue. This is undeniably a sound objection, but we had the hope that should there be certain definite changes observable after our experiments, they might then be corroborated by similar changes in our collection of human pulps.

Accordingly we operated on four dogs, placing them in a profound narcosis by means of injections of morphin and scopolamin, and working on their teeth by methods as nearly like clinical procedure on humans as was possible. Class V cavities were prepared on the labial or buccal surfaces of many of the teeth on one side of the mouth. (The opposite side was left untouched for control.) These cavities were started with moist, sharp, carborundum stones and finished with steel burs, exactly as would be done in the mouth. Great care was used not to overheat the tooth structure. Amalgam fillings, both well
and poorly condensed, silicate cement fillings, mixed both thick and thin, oxyphosphate of zinc cement, also mixed vari-
ously, gold foil fillings, and gold inlays were placed in the cavities. An occasional cavity was left entirely open, and several drugs were sealed in deep cavities on moist dressings.

The dogs were kept alive for several weeks and were then again narcotized and a few more fillings were placed, on the same side of the mouth as before. Thus, when the animals were dispatched a few weeks later, we had two groups of teeth in each dog which had contained fillings for two different periods of time. About twenty teeth were filled in each dog. As a rule it is very difficult to extract dogs' teeth without frac-
turing them. The resistance of the periodontal structures is greater than that of the tooth structure itself, so that prac-
tically all anterior teeth break if one attempts to extract them. For this reason it is necessary to saw the teeth apart, allowing part of the surrounding bone and soft tissue to re-
main with each tooth. It was feared that the time lost from the time a dog was killed till the teeth were sawn apart and especially till the fixing fluid had reached the pulp tissue would be so great that a precise fixation of the minute cell-
ular arrangement of the pulps might not be possible. And more, there was the great possibility that the fixing fluid, having to diffuse through solid tissue and then into the tiny apical foramina of the teeth, might never reach the pulp in sufficient
quantity to fix it adequately.

So a method was devised for reaching every minute part of the tissues of the entire head with the fixing agent at the instant of death. Two glass funnels were erected securely on ringstands, the one containing warm physiologic saline solution, the other Zenker-formol. These were fitted with rubber tubes and pinch-valves, and united by a glass Y-tube to flow into a common rubber tube. (See diagram, plate 16). By manipulation of the valves, either of the two solutions could be made to flow down out of the common duct. Below this another Y-tube divided the stream again and two long rubber tubes, with valves, were attached with glass canulae at their ends.

When the dog was deeply anesthetized, an incision was made on each side of the neck and both the common carotid arteries and the internal jugular veins exposed. Then the valve under the funnel of saline solution was opened and the liquid allowed to fill all the tubing, excluding every bubble of air. Next a hemostat was clamped on one of the carotid arteries and a longitudinal incision made in the artery wall distal the hemostat. One of the canulae was placed in the incision, pointing distally and securely tied in place. The same was done with the other canula and the opposite carotid. During the time consumed with this part of the experiment, the vertebral arteries, of course, provided a sufficient blood supply to the brain to maintain fe.
Plate 16. - Diagram of apparatus used for fixation of pulp tissues of animals.
Then, as nearly simultaneously as was possible, the jugulars were cut, the valves of the canulae opened, the carotids cut between the hemostats and the insertions of the canulae, and the hemostats removed. In this way the saline solution passed by force of gravity (the funnels were about 150 centimeters above the dog's head) into the common carotid arteries and through their entire capillary bed, which of course includes all of the anterior part of the head, while the blood ahead of it was being washed out through the jugular veins. The freeing of the proximal ends of the carotids allowed the blood pressure to fall so rapidly that no blood could come forward from the vertebral arteries, by anastomosis, to the region of the jaws.

The effect was most spectacular. One would scarcely be prepared to believe that all of the various colors and shades of the tongue, gingivae, and cheeks were caused solely by the color of the blood beneath the surface showing through, but such proved to be the case. As the warm saline solution pursed through the vessels, washing the blood out of arteries, arterioles, capillaries, venules, and veins, in sequence, the tissues assumed the utter whiteness of fine bleached linen. This was maintained only for a matter of seconds, until it was certain that every trace of blood had been removed. Then the valve under the nearly empty funnel of saline solution was closed and simultaneously the one under the fixing fluid opened.
Zenker-formol is a brilliant yellow from the potassium dichromate it contains, and consequently, as soon as it passed into the tissues, there was a quick change from their extreme pallor to a vivid color closely resembling a severe jaundice.

By the time the fixing agent reached the brain, if not before, the dog was dead, and it only remained to dissect out the two jaws and cut the teeth apart. The completeness of the fixation could be seen macroscopically in the deep structures of the glands and bone marrow, where everything was uniformly yellow. Such an effect could scarcely have been achieved by weeks of soaking the whole jaws in a fixing solution. The one objection to the method is that all vessels are utterly devoid of blood cells in the finished specimens.

All of the teeth, both human and canine, were decalcified in 5% nitric acid, embedded in celloidin, and sectioned, stained, and mounted in the usual manner.
CHAPTER III

OBSERVATIONS AND DEDUCTIONS
CHAPTER III

Included in the human specimens collected for the investigation were several which were without dental restoration and could so be used for controls. In the collection of specimens in the Research Department of the Chicago College of Dental Surgery, Dental Department of Loyola University, a great many more normal human teeth were available for study and comparison also. In the introduction of this thesis it was forementioned quite deliberately that while the normal histology of the pulp had been carefully studied, its description was somewhat too idealistic. This, in fact, constituted our first major observation.

In laying out a study of divers deviations from a normal, the primary consideration, obviously, is to establish that normal. In this case, however, the more pulps were studied the more the supposed normal became vague and ill-defined. The orthodox text-book description of a human dental pulp was answered by so few specimens that they stood out as rare exceptions and, by very definition, the antithesis of normal. Plates 17, 18, and 19 show such a normal pulp; it was taken from a very young individual (8 years). But even in this specimen, in other slides of the series, slight traces of degeneration, so called, could be found.
Plate 17. - Photomicrograph of a normal human pulp.

D, dentin; O, odontoblasts; N, nerves; V, blood-vessels; P, pulp cells (stellate embryonal connective tissue cells).
Plate 18. - Higher magnification of the pulp shown in plate 17.

The dark area to the left is dentin. O, odontoblasts; N, nerves; V, blood-vessels; P, pulp cells.
Plate 19. - Higher magnification of the pulp shown in plate 18.

N, nerve; V, blood-vessels (capillaries); E, erythrocytes (red blood corpuscles); P, pulp cells.
Ordinarily the teeth of adults and older persons (although there are occasionally curious anachronisms) show many involutionary changes in the pulp tissues and the calcified structures as well. To call all of these abnormal or pathologic would now seem reduced to an absurdity. The thymus gland atrophies at puberty, by twenty many people find a few gray hairs, by thirty a few wrinkles put in their appearance, at forty the female sexual function begins to wane, by fifty the accommodation of the lens of the eye is reduced, by sixty the bones are brittle, the blood pressure high, all functions slowed, and at 64.4, by life insurance averages, we die, from one cause or another.

All of these changes are in the direction of declination. They are all involutions or degenerations, if one cares for that term. But they are all undeniably physiologic or, at least, normal. In like manner it seems necessary to consider most of the changes and so-called degenerations of the pulp as perfectly normal. In the following pages we shall discuss these changes, both of the pulp tissues and then of the hard tissues, one at a time, taking up the relationship, or lack of it, with dental restorations as each separate item is considered.

1. Reticular atrophy of the pulp

Reticular atrophy of the pulp is a change in structure of the entire organ whereby the compact formation of the connec-
tive tissue cells is thinned out, so to speak, giving a section of pulp tissue a net-like (reticular) appearance.

Reticular atrophy was observed in more than half of the human pulps studied, more often in the pulps of older individuals but also occasionally in young ones. In older pulps it tends to appear in a more advanced stage, as coarse reticular atrophy, whereas in younger pulps it more often will not even involve the entire organ but be limited to a small area, such as a pulp horn. A typical instance of this is shown in plate 20. Reticular atrophy is arbitrarily divided into a fine and a coarse form. Plate 21 shows the fine type and plate 22 the coarse. Both are magnified the same amount, which makes the difference in the size of the "mesh" instantly apparent. They represent an earlier and a later stage of this type of degeneration.

This sort of atrophy is unique with the dental pulp. It represents the characteristic diminution in size of an organ with advancing age, but with one striking peculiarity. Where other organs simply shrink in volume as they atrophy, the pulp, being attached on all its peripheral surface to the surrounding unyielding dentin, is obliged to do its shrinking at the expense of its density instead of its volume. Consequently, the more it atrophies the scantier its parenchyma becomes, until eventually it may consist of only a few scattered strands of live connective tissue with a few blood vessels and nerves, with
Plate 20. - Reticular atrophy of the pulp.

Pulp taken from a lower molar of a boy 18 years old. The tooth had been filled with amalgam approximately eight years and was also affected by superficial caries.
Plate 21. - Fine reticular atrophy of the pulp.

Any shrinkage of the pulp must be at the expense of its compactness, since its volume remains fixed by the walls of the pulp chamber.
Plate 22. - Coarse reticular atrophy of the pulp.

The magnification is the same as for plate 21, so that the difference in size of the vacuoles is easily seen. These spaces are filled with fluid *intra vitam*. D is the dentin. No odontoblasts remain.
tissue fluid in the spaces included between them. Clinically such a pulp is unresponsive, but it is emphatically an adequately serviceable pulp since, as we shall see later, there is so little true vitality left in such a tooth for the pulp to preside over, and is normal in a tooth of an aged person. If it be found in a tooth of a younger individual, it must still be considered as harmless; it is in a like category with prematurely gray hair.

Reticular atrophy of the pulp, in any stage, is not observed to occur more frequently in teeth with fillings or other dental restorations than in intact teeth. On the contrary, the evidence points to a non-relationship.

2. Vacuolar degeneration of the odontoblasts

Another tissue change of a similar character that frequently occurs along with reticular atrophy is vacuolar degeneration of the odontoblasts. It was found to be present to at least a slight degree in the majority of the human specimens. Like reticular atrophy it represents a degeneration characterized by accumulations of tissue fluid between cells, but in this instance the peripheral cells of the pulp, the odontoblasts, are the ones involved. The term vacuolar degeneration of the odontoblastic layer is given to this involutionary change because fluid collects in the site of the odontoblasts, forming tiny vacuoles. (Plate 23). These vacuoles appear to
Plate 23. - Vacuolization of the odontoblastic layer.

This shows a pulp horn of an upper first molar which contained an amalgam filling for ten years. D, dentin; P, pulp; O, odontoblasts; V, vacuoles.
Plate 24. - Vacuolar degeneration of the odontoblasts.

This shows how the odontoblasts may be grouped together and pulled slightly away from the dentin. D, dentin; V, vacuoles.
Cysts of the pulp are very rare; in fact some investigators even question their reality. Hill, among others, thinks they are merely artefacts arising during and after fixation in the technical procedure of preparing the specimens. (11:828-29). A careful examination of plates 25 and 26, however, leads to the counter criticism that it is difficult to account for one very large shrinkage space in a specimen where all the rest of the tissue is so well fixed.

The most frequent site of these cysts is in one of the pulp horns (plate 25), although they occur elsewhere along the periphery of the pulp or even in the substance of the organ itself (plate 26). Euler has pointed out that they must be differentiated from old abscesses of the pulp and that they are without clinical symptoms. (7:1030).

They are not numerous in our specimens, but the evidence weighs against the thought that they might grow in size to the point of causing death of the pulp. There is apparently no relationship between the occurrence of these cysts and the presence or absence of dental restorations.

4. Calcifications in the pulp

Every one who has studied dental pulps under the microscope is familiar with the various masses of calcification so frequently found in them. They are called pulp stones or pulp
Plate 25. - Cyst of the pulp.

Photomicrograph of a pulp horn of a lower second molar of a young woman 22 years old. It had carried a gold inlay for three years. There was also recurrent caries. D, dentin; P, pulp; C, cyst.
Plate 26. - Pulp cyst.

D, dentin; P, pulp; C, cyst. At V there may also been extensive vacuolar degeneration of the odontoblasts.
nodules or denticles. Most of these calcifications are either of a generally rounded shape, laid down in successive lamina-
tions like a pearl (plate 27), or they are of a jagged, irregu-
lar shape, often roughly spindle-shaped. The latter may be termed diffuse fibrillar calcifications. (Plate 28). Occasion-
ally denticles are observed which show a tubular structure sim-
ilar to that of irregular dentin. These are frequently called true denticles and are often fairly large and nearly always attached to the walls of the pulp chamber. Calcifications with-
out tubular structure are then called false denticles.

In studying these formations, particular attention was given to the frequency of their occurrence. It is the usual belief that they are far less common than is actually the case, and they are frequently blamed as the etiologic factor of tic douloureux, obscure neuralgias, or elusive cases of pulpitis. Therefore, in order to secure as many data as possible, an actual count was made, using not only the pulps with clinical histories collected especially for this investigation, but utilizing many more that were available in the laboratory col-
lection.

In compiling this material histologic sections of 164 teeth were examined and in each case the designation of the tooth itself -- that is, which particular tooth of the denture it was --, the history available, the number of sections, the condition of the tooth, whether abraded, decayed, artificially
Plate 27. - Dentine or pulp nodule.

This photomicrograph shows the concentric lamellations of this type of calcification.
Plate 28. - Diffuse fibrillar calcification of the pulp.

Extensive calcification of an otherwise intact pulp. D, dentin; O, normal odontoblasts; C, calcifications.
cut, or intact, the condition of the pulp, whether intact or 
exposed and inflamed, and the type of pulp, whether juvenile, 
adult, or senile, were recorded on a chart. In addition it 
was noted for each one whether or not secondary dentin was 
present and then whether diffuse fibrillar calcifications, 
microscopic denticles or macroscopic denticles (plate 29) were 
visible. It seemed important for this study that the latter 
division be made, because most studies of pulp nodules hereto­
fore have been made on the basis of roentgenographic evidence 
alone, so that a comparison was needed to show the relationship 
between the actual number of pulps containing calcifications 
and the number of such calcifications that might have been de­
termined by roentgenographs alone. In making this distinction, 
it should be remarked, all benefit of doubts was given the lat­
ter. Some of the nodules recorded as macroscopic, or roentgen­
ographically visible, were so small that an exceptionally sharp 
image would be needed to reveal them.

Of the total number of teeth examined, 33 were incisors, 
25 cuspsids, 28 bicuspids, and 78 molars. Of the 33 incisors, 
28 showed calcifications of some sort microscopically, of the 
25 cuspsids, 23, of the 28 bicuspids, 22, and of the 78 molars, 
70, making a total of 143 showing calcifications out of the 
164 specimens. This is approximately 87.2%. It must not be 
concluded from this, however, that nearly 13% of teeth are free 
of these bodies. The number of sections available from each
Plate 29. - Large denticle in a lower molar.

Such a calcification would be distinctly visible roentgenographically. It has formed by the coalescence of several smaller nodules of the type shown in plate 27.
specimen varied from a single section in two cases to as high as 39 in one instance, but the arithmetic average was only 13 sections per tooth. In no case was the entire pulp retained in an unbroken series. On the contrary, the average total thickness of the 13 sections was only 0.325 millimeters, which is probably less than one-tenth the entire width of an average pulp. Moreover, the given sections from a particular tooth were almost never in perfect series; sometimes they represented widely separated planes of the same pulp. How important this point is will be realized when it is pointed out that more than once, in a set of sixteen or twenty sections, a conspicuous denticle would be visible in only one section, while all the others contained none whatever. And since calcifications show no predilection whatever for any one region of the pulp chamber, it can be readily understood that there was a hazard of more than 10 to 1 of missing calcifications when they were present. Arguing from such a basis it seems probable that only very few teeth could be reasonably expected to be entirely free of them.

The high incidence of pulp calcifications is not the only remarkable thing about them, however. Their age distribution is equally surprising. Calcification is essentially a characteristic of mature or even decadent tissues. It is usually associated -- except, of course, in normal bone and tooth development where its physiological purpose is obvious -- with a de-
creased blood supply and a lessening of function. Denticles do occur in increasing numbers as the age of the patients advances, but what is unexpected is that they should occur in the very young at all. Concretions elsewhere in the body do not, but here is the exception. Not only have denticles been observed in impacted teeth (plate 30) and young teeth (plate 31), but they have been found in teeth not yet fully formed (plate 32). But to go farther, they have even been found in deciduous teeth (plate 33) and, most remarkable of all, in the tooth-like structures of dermoid cysts (plates 34 and 35).

The etiology of these nodules, so far from being explained by these findings, is rendered more obscure than ever. Their excessively high incidence almost precludes calling them abnormal, and yet their presence serves no apparent purpose but has, in the past, actually been considered a detriment. This latter view becomes increasingly hard to accept. And since denticles are found in virtually all teeth, obviously there can be no evident connection between them and dental restorations of any kind. In observing our slides, no more denticles were apparent in teeth with ridings than in those without.

Stafne and Szabo, as mentioned in the second chapter, have done considerable work in studying the correlation, if any, between the presence of these bodies and the presence of disease elsewhere in the body, and conversely, the possibility of systemic disease being responsible for the presence of the nodules.
Plate 30. - Denticle in the pulp of an impacted tooth.

A pulp taken from an impacted lower third molar; patient 3 years old. The pulp is of a juvenile type with virtually no degeneration. D, denticle.
Plate 31. - Calcifications in juvenile pulp.

Normal young pulp. O, intact layer of odontoblasts; C, calcifications.
Plate 32. - Dentine in tooth not fully formed.

This shows a buccolingual section of the mandible of a child of 11 years. A is the second deciduous molar and B the erupted second bicuspid, the root of which is still forming.

e: enamel; D, dentin; P, pulp; and at C a denticle may be distinctly seen.
Plate 33. - Dentine in a deciduous tooth.

Buccolingual section of part of the maxilla of a small year old child. A is the first deciduous molar; B is the crown of the first bicuspid. At C there is a typical denticle in the pulp of the deciduous tooth.
Plate 34. - Calcifications in the pulps of teeth found in a dermoid cyst of the ovary.

"A" and "B" are fairly true tooth forms taken from a dermoid cyst. E, enamel (lost by decalcification); D, dentin; B, bone resembling the alveolar bone; C, capsule of the cyst. The teeth are erupting from the inner surface of the capsule into its lumen. x marks calcifications of the pulp tissue.
Plate 35. - Calcifications in the pulp of a tooth from a dermoid cyst of the ovary.

This tooth is more irregularly formed than the teeth in plate 34, but the elemental tissues are just as well formed. Dentin; E, enamel, showing resorption and replacement by ne at R; P, pulp, showing a large number of calcifications; cyst capsule.
Their conclusions were that the evidence in favor of either idea was negative or, at best, extremely meager. (19:163). Their observations were all made on the basis of roentgenographs, so that only macroscopic denticles could be counted. Even so, they found them surprisingly common, stating that they found them in 46% of "essentially normal" patients. What the percentage was of teeth with nodules was not given, but it is a frequent observation that if they are roentgenographically visible in one tooth of a given individual they are rather more likely than not to be seen in a number or even nearly all of the teeth in that mouth.

In a recent conversation with Dr. Stafne the writer learned that at the time of his writing the article he had been fearful lest his 46% seem a fantastic overstatement. He was reassured by the figures of this present investigation. Of the 143 teeth that showed calcifications of some type, 122 or 85% showed only the fibrillar calcifications or microscopically small denticles, while but 21, or a scant 15%, showed denticles that could possibly have been visible in a roentgenograph. So if Stafne and Szabo found 46% of patients to have macroscopic denticles in one or more teeth, and these represent but 15% of the total number of denticles to be found in a few sections taken of each of many teeth, then one can only conclude that very few if any people are entirely without them.
5. Sclerosis of the dentin

The term sclerosis was given by Beust to describe the filling up of the tubules of the dentin with calcific material, so that the dentin becomes relatively homogeneous instead of being honeycombed with the tubules. Such sclerosis frequently occurs in isolated tracts when the external surface of the involved tubules is opened, as by abrasion or caries. (3:633). So far from being "soddened" or "softer than normal" (4:271), as we previously quoted Black as believing, these areas are much more dense than the adjacent dentin. They do appear darker in ground sections when observed by transmitted light, as Black saw them. This is due to the relatively greater amount of calcium salts present. But by reflected light they appear as lighter than the adjacent dentin. This is due to the fact that the more nearly homogeneous substance of the sclerosed area reflects light much better than the tubular dentin, because the tubules opened in grinding the section are present as thousands of tiny grooves, all of which tend to break up the light and so fail to reflect it.

Additional evidence that these areas are more nearly solid is given by the work of Fish. (10:51-61). His staining experiments show that dyes placed in the pulp penetrate readily into the tubules of the ordinary dentin but fail entirely to penetrate the sclerosed dentin. These experiments were carried
on with monkey's teeth and dog's teeth, both in vivo and in vitro, with very similar results. Similar experiments with human teeth in vitro were done with the same results.

Bödecker believes the tubules in these areas are filled with "fat-gaseous casts" (5:1180), but by far the most conclusive evidence we have of the actually greater calcification of these areas is found in the work of Van Huysen, Hodge, Warren, and Bishop. They found by means of the Roentgen ray, and a precision densitometer to measure the relative translucency, that these areas in ground sections were definitely more radiopaque. (22:729-38). The only remaining evidence that has not been obtained would be that of a microanalysis of a section of one of these areas to show that a greater amount of calcium is present.

In plate 36 is shown an incisor, the tip of which has been abraded to the dentin. The characteristic dark area (the specimen was photographed by transmitted light) is seen, where sclerosis has taken place. However, sclerosis does not occur only when the peripheral ends of the tubules are opened, as by abrasion, caries, cavity preparation, and the like. Beust has shown that it also occurs rather regularly in the peripheral part of the coronal dentin as the tooth becomes older, progressing toward the pulp. (1:1060). He has given the term maturation to this process, because it truly seems to represent a physiologic change to a more mature type of dentin.
Plate 36. - Sclerosis of the dentin.

The tip of an incisor abraded to the extent of opening the dentinal tubules involved in the sclerosed tract, S. It appears darker than the surrounding dentin, because it was photographed by transmitted light.
Since this occurs, not only in teeth without fillings or other dental restorations but also in teeth with intact enamel, it obviously is not related to external irritations. By far the greater majority of our specimens showed sclerosis of some part of the dentin.

6. Secondary dentin

In the preceding discussion of the changes in the pulp tissues proper and the calcifications of pulp nodules and sclerosed dentin, it has been noted that all of these occur independently of dental restorations. With secondary dentin formation such is not the case.

Secondary dentin is somewhat difficult to define. When the tooth is first developed, both enamel and dentin develop rather simultaneously, having the dentinoenamel junction as their common starting point. When the crown is erupted into the mouth, the outward growth of the enamel is unquestionably at an end, whatever may or may not be thought to happen to its density. At some similar period the inward growth of the dentin presumably comes to an end also, or at least its rate of growth approximates zero. Whether this is so or not is extremely difficult to verify. It is well known that the thickness of dentin relative to the size of any given tooth in the denture is quite constant; that when the dentin grows beyond this relative thickness, causing the pulp chamber to be rela-
tively small for that tooth, there can usually be discerned a line of demarcation indicating, apparently, a pause in growth in the location of the expected normal. For these reasons most histologists believe that the primary dentin is laid down to a certain fairly normal thickness and that subsequent growth, if it occurs, follows a resting period, and is indicated by at least a slight change in the direction of the dentinal tubules. Dentin that is thus laid down after the "completion" of the primary or original dentin is usually called secondary dentin. Fish takes exception to that definition, admitting only dentin the tubules of which are not confluent with those of the primary dentin to be secondary dentin. (10:56). Euler and Meyer, however, as noted in Chapter II, do include what Fish calls "accelerated growth of primary dentin (loc. cit.)" in their classification of secondary dentin. (8:41).

Early in the observation of the material collected for this investigation it was observed that localized formations of secondary dentin, confined precisely to the inner ends of the cut dentinal tubules, occurred with great regularity under the various types of fillings. Plate 37 shows a buccolingual section of an upper second bicuspid, taken from a woman 34 years of age. It had carried a large gold inlay for four years. At A the only part of the cavity preparation that is visible in the plane of this section may be seen; and at B, exactly covering the inner ends of the dentinal tubules included in
Plate 37. - Secondary dentin formed in response to cavity preparation and filling.

Buccolingual section of an upper second bicuspid. A, occlusal portion of the cavity preparation; B, boss of secondary dentin; C, pulp cyst; D, denticle; E, beginning reticular atrophy; F, vacuolization of the odontoblasts.
he cavity preparation, is a boss of secondary dentin. In this specimen, incidentally, may be observed:  
  C, a cyst of the pulp;  
  D, a denticile;  
  E, an early stage of reticular atrophy;  
  and  
  F, vacuolar degeneration of the odontoblasts. Of all of these, only the presence of the secondary dentin can be laid definitely to the cavity preparation and the placing of the inlay. The possibility of a connection between the other changes and the inlay would be entirely speculative.

But cavity preparations and the placing of dental restorations were not the only observed causes of secondary dentin formation. In plate 38 is shown a mesiodistal section of a lower second molar, taken from a woman of 45. It had contained a mesioocclusal amalgam filling for approximately seven years. At  
  A is a tract of secondary dentin formed apparently in response to the cutting of the occlusal extension of the cavity. But at  
  B is seen secondary dentin that was obviously present when the cavity was prepared, since otherwise the pulp would have been exposed. This dentin, then, was laid down in response to the opening of the peripheral ends of the tubules by caries, instead of by instrument. And at  
  C is seen another example of this phenomenon. The distal cavity,  
  D, near the gingival line, had just penetrated the enamel and reached the dentin when the tooth was extracted. The tubules affected by the caries may readily be traced to the pulp chamber and there it will be seen that the secondary dentin very precisely covers
Plate 38. - Secondary dentin formed in response to different stimuli in the same tooth.

Lower second molar which carried a mesioocclusal amalgam filling for seven years. A, secondary dentin resulting from the cavity preparation; B, secondary dentin stimulated by the original caries; C, secondary dentin from the new cervical caries at D.
In addition to the opening of dentinal tubules by caries or a surgical cut, abrasion that reaches through the enamel and into the dentin, or erosion, will also cause a formation of secondary dentin. Plate 39 shows an upper incisor, without caries or filling, that was subjected to much abrasion. The enamel has been lost by decalcification; the dentin on the incisal edge has been abraded to the level of the original pulp chamber. At A is a formation of secondary dentin that undoubtedly started as soon as the abrasion reached the dentin and now occupies a space that was formerly pulp chamber and comprises part of the incisal edge of the tooth. At B is the secondary dentin that has been formed in response to this abrasion on the lingual surface, C.

The result of a cavity preparation may be seen in plate 40. This is a labiolingual section of a lower cuspid, taken from a woman, 32 years of age, which contained a gingival amalgam filling for seven years. At A a deposit of secondary dentin may be observed, corresponding exactly in location to the tubules involved in the cavity preparation. At B may be seen a small deposit due to incisal abrasion. Some vacuolar degeneration of the odontoblastic layer may also be noticed in this region.

Plate 41 shows this same point. At A is seen an island of secondary dentin stopping the tubules opened by the preparation of the cavity, B.
Plate 39. - Secondary dentin formed in response to abrasion. An upper incisor abraded on the incisal and the lingual surface. At A is a deposit of secondary dentin caused by the incisal wear, and at B a deposit caused by the lingual abrasion.
Plate 40. - Secondary dentin formed in response to cavity preparation.

Section of a lower cuspid taken from a woman 32 years old. It had contained a gingival amalgam filling for seven years. A is a deposit of secondary dentin precisely covering the central ends of the tubules involved by the cavity preparation. B is secondary dentin due to incisal wear.
Plate 41. - Secondary dentin caused by cavity preparation.

A is a deposit of secondary dentin corresponding exactly in location to the central ends of the tubules cut in preparing the cavity at B.
Plate 42 illustrates a combination of stimuli in an upper incisor. At A and A are two proximal cavities; at B and B are the two resulting deposits of secondary dentin. While at C is seen secondary dentin that was laid down in response to abrasion of the incisal edge of the tooth.

Some of these observations are so conclusive that it can be readily understood why surprise was expressed, when reviewing G. V. Black's work in Chapter II, that he should have fallen into the error of supposing that a local stimulation provoked, through the intervention of the nervous system, a generalized production of secondary dentin in all the teeth. He based this idea on teeth he had examined, especially those of one mouth in which abrasion was extensive on all but two unoccluding teeth, in which calcification was exceptionally extensive. (4:269). Had he had the decalcified sections made nowadays, with pulp and adjacent dentin intact, he would have been able to see what was not to be learned from ground sections of dry teeth and embedded sections of pulps studied separately.

In studying the material from the dog experiments, the same result was always noted. Plate 43 shows a lower incisor taken from one of the dogs. It contained an oxyphosphate cement filling for nine weeks, the cavity having been treated with a 1:500 solution of mercuric chlorid before the filling was placed. During this short time a noticeable deposit of secondary dentin, A, was laid down, opposite the position of the cavity, B.
Plate 42. - Secondary dentin resulting from a combination of stimuli.

Section of a lower incisor with two proximal cavities at and A. At B and B are two resulting deposits of secondary dentin, and at C is a deposit produced by the irritation of incisal abrasion.
Plate 43. - Secondary dentin formation in a dog.
Lower incisor of dog. The experimental cavity contained oxyphosphate of zinc cement filling for nine weeks. Secondary dentin, A, was laid down opposite the position of the cavity, B.
Plate 44 shows an upper incisor from the same dog. The cavity in this case was allowed to remain open during the entire nine weeks without any treatment whatsoever. Here again, at A, is a deposit of secondary dentin. It is somewhat larger than that in the preceding tooth, but whether this difference is due to the difference in depth or location of the cavities or to the differences in their treatment can only be conjectured.

Plate 45 is a section taken from another upper incisor of this dog. The cavity, B, contained a silicate filling during the nine weeks. At A is a heavy deposit of secondary dentin.

Plate 46 shows a lower incisor of another one of the dogs. The cavity was cut very deep, quite by accident. In fact, at the time of operation the pulp seemed virtually exposed, so that an anodyne was sealed in the cavity with cement. An examination of the entire series of sections under the microscope failed to show an actual exposure, but it may be seen in this section that the cavity was exceedingly deep and that there was a very heavy deposit of secondary dentin laid down in defense.

Plate 47 is a higher magnification of the incisal portion of the pulp chamber. Here again secondary dentin from two sources of stimulation may be observed. At A is the portion produced since the beginning of the experiment in response to the cutting of the cavity, B. But at C is seen secondary dentin that was preëxistent at the time of operation, as evidenced by the
Plate 44. - Secondary dentin formation in a dog.

Upper incisor of a dog with experimental cavity left undilled for nine weeks. Deposit of secondary dentin at A corresponding to the central ends of the exposed dentinal tubules.
Plate 45. - Secondary dentin formation in a dog.

Another upper incisor from same dog shown in plate 43. The cavity, B, contained a silicate cement filling for nine weeks. A, secondary dentin.
Plate 46. - Secondary dentin formation in a dog.
Lower incisor of another animal. B, deep cavity; A, secondary dentin. The cavity was sealed with an anodyne dressing for fifteen weeks.
Plate 47. - Higher magnification of plate 46.

At A is secondary dentin produced in response to the cutting of the cavity, B. It may be seen to overlap the secondary dentin at C, which was already present at the time of the operation, probably in response to incisal wear.
overlapping of the newer portion, A. Abrasion of the incisal tip of the tooth was probably responsible for the deposit at C.

The production of secondary dentin, as Fish has observed, "is a specific reaction to peripheral injury of the dentine (10:76)", but the amount of it that will be produced would seem to depend (1) upon the severity of the injury, (2) the length of time that elapses, and (3) the individual quality of vitality and reaction of the pulp. The type of secondary dentin, whether as tubular as the primary dentin, or less so, or entirely hyaline in character, must depend on how many, if any, of the odontoblasts survive the injury. Orban has shown quite conclusively that the odontoblasts do not form dentin, as their teleological name would infer, but merely contribute the contents of the tubules. (14:1572). Dentin can be formed readily without them, but it will then be entirely nontubular in structure. (14:1581).

Fish, in his animal experiments, not only cut cavities in the teeth of the dogs and monkeys (plates 48, 49, 50, 51, and 52), but in addition, after the animals were killed and the teeth removed, introduced a dye (methyl blue) into the pulp chamber and allowed it to diffuse through the dentin before sectioning the teeth. (10:51-61). Since he had previously shown that normal dentin in these animals is freely permeable to this dye (10:21-31), he showed by his results that the secondary dentin formed over the inner ends of the cut tubules.
Plate 48. - Facsimile of figures 27 and 28,

Fish, (10:112).

The left is a photomicrograph of a dog's tooth in which the cavity, C, had been filled with cement for eleven weeks. There is no change visible in the pulp tissue. The right shows an identical experiment except that the cavity was allowed to remain open. A large boss of secondary dentin, S, was formed over the cut tubules which were sealed by the calcium barrier, B.
Plate 49. - Facsimile of figure 29, Fish, (10:112).

Photomicrograph of a tooth of a monkey. The experimental cavity, C, was made twelve weeks before death. The calcium barrier, B, and secondary dentin, S, correspond precisely to the tubules cut by the cavity. Minute hemorrhages in the pulp may be seen at H.
Plate 50. - Facsimile of figure 30, Fish, (10:112).

Photomicrograph of tooth of dog two weeks after cutting the experimental cavity. The deposit of calcium salts, B, is observed to extend a short distance up the injured tubules, A, and forms a barrier between the highly tubular secondary dentin, S, which is beginning to form, and the injured primary dentin.
Plate 51. - Facsimile of figure 51, Fish, (10:113).

Photomicrograph of monkey's tooth twelve weeks after cutting an experimental cavity. The injured tubules of the primary dentin are sealed with calcium salts at A. A calcified barrier, B, may be seen between the primary and secondary dentin. The origin of the tubules of the secondary dentin is at T.
Plate 52. - Facsimile of figure 32, Fish, (10:113).

Another experimental cavity of twelve weeks' duration in a monkey's tooth. Irritation to the early secondary dentin resulted in a second barrier of calcification being formed. A, first barrier; B, second barrier.
hermetically sealed these tubules from the pulp, because the dye entered the normal dentin in all other regions except the one tract so sealed. The dye also penetrated the tubules present in the island of secondary dentin, showing the fibrils to be alive there. A hyaline barrier of calcium salts laid down in an organic matrix was also demonstrated between the primary and secondary dentin. It is by the presence or absence of this barrier that Fish discriminates between secondary dentin and accelerated growth of primary dentin, as before mentioned. (loc. cit.).

But he also noticed another type of reaction to the cut cavities which apparently made secondary dentin production unnecessary, at least for a time. He terms this a "translucent zone type of reaction (10:56)", whereby the peripheral ends of the tubules, instead of the central, become occluded by lime salts. This is in direct correspondence with Beust's (op. cit.) observations on sclerosis of dentin following peripheral irritation, although it is confined to the peripheral part of the tubules. This translucent zone was first noted by Tomes who observed it under caries. (21:203). Fish is the first to have seen it under experimental cavities. Sometimes both types of reactions occurred simultaneously, as shown in plate 53. This shows an experimental cavity three months old. Some of the groups of tubules, as at D, were sealed off by the calcium barrier, H, and a deposit of secondary dentin. The dye
Plate 53. - Facsimile of figure 26, Fish, (10:112).

Experimental cavity three months old. At D groups of tubules sealed off by calcium barrier, H, and deposits of secondary dentin. The dye could not penetrate these groups of tubules. The intervening groups were not sealed off, so that the dye was able to penetrate them almost to the cavity, C, except for the translucent zone, TZ.
could not penetrate the tubules from the pulp chamber. Intervening groups of tubules were not cut off at their central ends, so that the dye penetrated them almost to the cavity, C. The dye did not quite reach the cavity, however, because the translucent zones, TZ, sealed the tubules at their peripheral ends. Additional primary dentin, A, (according to Fish's definition) was added, in those areas where the fibrils had remained alive and the tubules permeable, so that the whole deposit had a regular outline.

Since neither our human or dog material was treated with a dye like methyl blue, because of the methods of fixation, it is not impossible that some of these bosses of secondary dentin observed in our specimens are composed in part of what Fish calls true secondary dentin, occluding tracts of dead fibrils, and in part of the Class I type of secondary dentin of Euler and Meyer (op. cit.) which is in fluid communication with the original primary dentin.

Fish also states that in 3% of his experimental cases, a more severe type of reaction occurred in the pulp, so that lesions in the peripheral areas resembling granulomata sometimes appeared instead of secondary dentin. (10:72). Plate 54 shows such a case. At C is a granulomatous mass, while at S is a boss of secondary dentin. These indicate "a difference in the severity of the reaction at different points along the pulp margin under the injured tract of tubules (10:73)." The
Plate 54. - Facsimile of figure 39, Fish, (10:114).

remainder of the pulp is normal, these granulomatous areas being quite local. Fish also states that on rare occasions the pulp dies from a peripheral injury to the dentin, and in some other cases, no reaction whatever is observable, the dentin being unsealed at either the peripheral or central end. (10:74).

It is Fish's conclusion (10:83) that the translucent zone just below the lesion is due to sclerosis of the primary dentin and "occurs when the contents of the tubules", i.e., the fibrils, "are irritated, but not destroyed (10:84)." While the secondary dentin at the pulpal ends of the tubules is produced "in response to a necrosis of the primary dentine (10:83)."
CHAPTER IV

CONCLUSIONS
CHAPTER IV

Approximately two hundred human teeth and eighty canine teeth were available for this study. From the study of this material and the literature bearing on the subject, it is concluded:

First, that our conception of a normal pulp must be materially altered. Instead of the ideal being thought uniquely normal, an average pulp, with the common involutionary changes of tissue, such as vacuolar degeneration of the odontoblastic layer, reticular atrophy, occasional cysts, and the like, should be substituted in our concept. The presence of calcifications in the pulp, also, seems well within the pale of the normal. The possibility of these bodies causing disturbance either locally or systemically seems very remote.

Second, that of all the changes that can occur in the pulp and surrounding dentin, the production of a translucent zone of sclerosed dentin at the periphery of the dentin or of secondary dentin seems to be the only two that may follow directly as effect from cause, the irritation of cavity preparation and a subsequent filling operation. Evidence has been adduced that fillings of silicate cement cause tissue damage to the pulp if placed in freshly cut, non-caries cavities. The same result might be expected if a carious cavity were extended widely into
sound dentin for retention or immunity during cavity preparation. Other types of filling materials seem to have no effect whatever on the tissues of the pulp. It must be added that while barrier formation and secondary dentin formation appear to be the only tissue changes resulting from the procedures of operative dentistry, the converse is by no means true. These changes also occur if the dentinal tubules are opened at their periphery by any other means, as by caries, abrasion, erosion, or trauma, and they may even be induced experimentally by drugs, such as paraformaldehyde.
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