

normal in depth. T+. Glaucomatous excavation of optic disc. Fields markedly contracted, only a small temporal field remaining (Fig. 24).

Feb. 16, 1903. Removal of left superior cervical sympathetic ganglion under morphia-chloroform narcosis by Dr. A. E. Halstead. The operation was somewhat difficult on account of the patient's thick neck. The ganglion was very small, but was removed with about 3 cm. of the nerve. Spinal accessory nerve was cut and was afterward united with catgut suture.

Feb. 17, 1903. Patient recovered well from the operation. Pupil is contracted, ptosis marked. Partial anesthesia of left side of face and neck. T+ as before.

Feb. 26, 1903. L. upper lid droops about 2 mm. more than right. Right pupil 4 mm. L. pupil $2\frac{1}{2}$ mm. L. V. with Sph.+1.50=20/40. T+. Pain in left side of face and jaw, slight hoarseness and some loss of power in left shoulder. The hyperesthesia of the left side of the face about the jaw and ear was intense, so that he could not bear the slightest touch. Eserin was used regularly.

March 13, 1903. L. V. with Sph.+1.50=20/40. T.+1. Ptosis and miosis persist. Hyperesthesia of the face gradually getting less. Fields much contracted, being reduced to a narrow slit (Fig. 25).

March 17, 1903. Anterior sclerotomy. Slight reduction of tension.

March 21, 1903. Tension again as high as before. Under cocain and holocain anesthesia an iridectomy was done upward. Immediately after completing the corneal incision the wound gaped widely and after a portion of the iris had been excised vitreous began to present in the wound and the patient complained of intolerable pain. After cutting off the portion of extruding vitreous the lens was forced into the wound and had to be removed. Then followed more vitreous and a copious hemorrhage, the patient all the time experiencing great suffering. The bleeding was finally checked and the eye bandaged. The patient was given an opiate and put to bed, and immediately had a severe chill.

March 22, 1903. Patient was fairly well and not suffering much. In the gaping corneal wound was seen a clot of blood and a tissue resembling iris or choroid. There was comparatively little reaction after the operation. The wound became smooth by the separation of the protruding mass and the blood in the anterior chamber was absorbed. At the present time, April 20, 1903, the wound has healed. Eye sensitive to touch. T.—2. V.=0. Sensitiveness of the side of the face has disappeared and the patient has completely regained the power of the arm and shoulder.

The excised ganglia in the last four cases were prepared by Dr. E. V. L. Brown, the pathologist of the Illinois Eye and Ear Infirmary, and were examined by him and also by Dr. Sydney Kuh, professor of diseases of the nervous system at the Post-Graduate Medical School. To these gentlemen I am indebted for the notes on the histologic appearances of the specimens. All the specimens were hardened in 5 per cent. formalin and corrosive acetic solution and stained with toluidin blue and with hematoxylin and eosin.

In all of the specimens there was increased pigmentation of the ganglion cells. In Cases 4 and 5, mast cells were seen. In Cases 4, 5 and 7 it was noted that the cells were not round, but of irregular shapes.

In one case, No. 6, cells slightly vacuolated were observed. In two cases, Nos. 4 and 5, it was noted that the cells showed excentric nuclei, and in the same cases marked vascularity was present. The histologic examination lacks completeness, as, unfortunately, there was not available for control study normal ganglia from individuals of the same age as the patients. As the pigment in the ganglion cells is normally increased in advancing age, it can not be said that these present an abnormality.

(To be continued.)

SOME HISTOLOGIC FACTS THAT CONTRADICT THE GENERALLY ACCEPTED ODONTOBLAST THEORY.*

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While I feel very much honored by your kind invitation, I nevertheless approach my task with some trepidation, because my histologic examinations lead me to conclusions that are considerably at variance with those

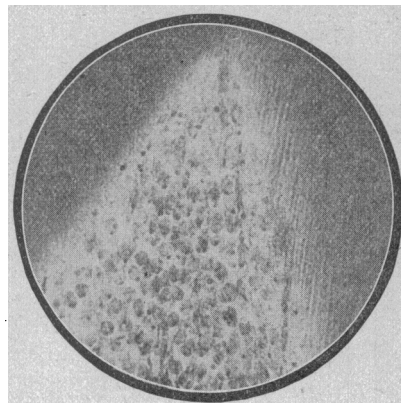


Figure 1.

of others who have worked along similar lines in past years. In Germany, I have for the present only a small number of followers, and I fear that the majority of you who are assembled here to-day are also fanatic adherents of the old odontoblast theory; consequently, I assure you that I expect only a moderate amount of applause and approbation at your hands.

But it is in your country precisely that the first voices were raised years ago against the prevailing views in regard to the rôle of the odontoblasts, and while I do not fully share the views of Heizmann, Boedecker,

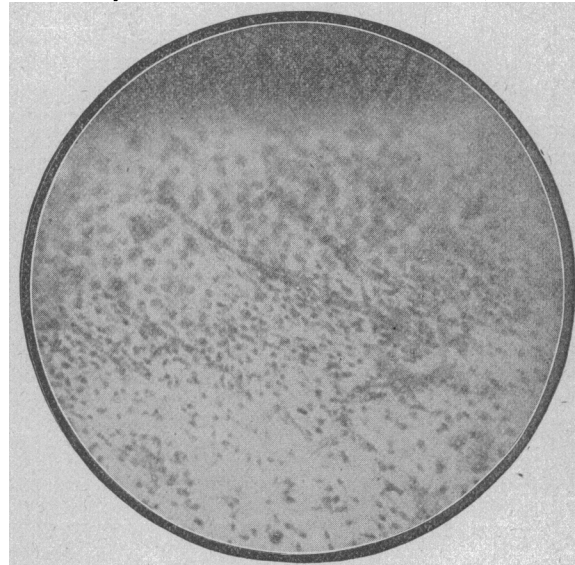


Figure 2.

Abbot and Andrews, I nevertheless recognize in these men brave pioneers, and their example inspires me with the courage to appear with my results before an audience that is composed of the greatest investigators in our particular field of research.

* Read at the Fifty-fourth Annual Session of the American Medical Association, in the Section on Stomatology, and approved for publication by the Executive Committee: Drs. M. L. Rheln, M. H. Fletcher and Wm. E. Walker.

According to the views that are prevalent in regard to the rôle of the odontoblasts, the surface of the pulp is covered with a single layer of cells that are in intimate contact with each other as are epithelial cells. These are the dentin cells or odontoblasts. Each one of these cells sends a protoplasmic process (dentin fiber) into the dentin. It is claimed that these odontoblasts are perennial, i. e., that they persist during the formation of the dentin, and are neither reduced in number during this process nor replaced by other cells. It is further claimed that the nutrition and the sensation of the dentin is exclusively bound to these dentin cells and their protoplasmic processes. All subsequent formative changes in the dentin, as, for instance, the genesis of the transparent zone in caries and the broadening of Neumann's sheath in senile teeth are also believed to be exclusively due to metaplastic activity going on in the protoplasmic processes of the odontoblasts.

The great advantage of this theory is its simplicity, for the most complicated physiologic processes can be explained on a very simple, one might almost say unitarian histologic basis. The very fact, however, that everything seems so simple should lead us to be skeptical. Recent investigations into the structure and the composition of the albumins teach us that things that appear exceedingly simple are nevertheless most complicated, and it seems hardly probable that different physiologic processes can be explained on the basis of one histologic unit. True, the adherents of the odontoblast theory point out that in lower orders of animal creation a single cell or group of cells may occasionally assume all the functions of the organism and that similar conditions might obtain in the case of the odontoblasts; against this argument we can formulate the objection that the odontoblasts are simply parts of a more complicated organism, and that in higher orders of animal creation the anatomic differentiation proceeds *pari passu* with the physiologic division of labor that becomes operative in complicated organisms. One might further object that in no vertebrate is any other cell variety known to exist whose protoplasm can at the same time perform the functions of nutrition, of sensation and of metaplastic transformation.

These theoretic objections alone do not, however, lead me to doubt the correctness and the validity of the odontoblast theory as it is accepted to-day; I have, moreover, been able to discover a number of histologic facts that can not be reconciled with this theory. It is the main object of this address to demonstrate these histologic facts to you with the aid of illustrations that I have made from my microscopic preparations.

I have frequently seen in the so-called pulp horns of the dentin germ in the fetus and the new born that the dentin cells were arranged in a multiple layer. Figure 1 is one of a series of sagittal sections through the anterior dental germs of a new-born child. You will notice that each dentin cell is pear shaped; you will also notice the direction of each dentin fiber and of the capillaries, for all these features show that we are actually dealing with a longitudinal section and not with a diagonal section in which the multiple arrangement of the cells might be simulated. The layer I am discussing consists of ten to twenty rows of cells. The individual cells show no trace of lateral flattening from mutual pressure; on the contrary, capillaries and fibrillæ will be seen running between the different cells in a direction that is parallel to the longitudinal axis of the dental germ. It is clear, therefore, that the cells are not

arranged in several layers on account of crowding or lack of room and mutual pressure.

The structural changes in the dentin cells also call for discussion. It will be seen that the cells become paler from the bottom toward the surface of the layer; that the nuclei disappear and that the cell boundaries become indistinct, until finally near the margin of the dentin hardly any cell remnants can be seen; for in this zone the changes in the substance and the structure of the cells are so far advanced that the dentin cells have become fully disintegrated and have disappeared from view.

If with Figure 1 we compare Figure 2 that is made from a frontal section through the pulp ridge of a young calf tooth, we will find the same conditions. Here, again, we see a broad layer of oval and round cells that are already beginning to become pale, extending up to the edge of the dentin. The layer here consists of some twelve rows of cells. These cells are odontoblasts in a state of dentinogenous metamorphosis, and show evidence of beginning disintegration. Between the cells we see capillaries running in the same direction as the dentin canaliculi. Below this zone we see the pulp cells in close aggregation, arranged in rows and extending their processes into the layer of disintegrating odontoblasts. These cells are in a state of conjugation and lead by that the formation of odontoblasts.

In preparing a tooth germ according to the method of Koch-Weil and in making a section without decalcification, spherical solid structures are seen in place of the pale disintegrated odontoblasts. These spheres were formerly considered to be dentin spheres or globular masses, and were described by these names. In another stage of the formation of dentin a transparent, homogeneous layer composed of single trabeculæ is occasionally seen in place of the disintegrating odontoblasts.

If a tooth that is still in a developmental stage is only incompletely decalcified, spherical structures are also seen in the place of disintegrating odontoblasts. Many of these little globules, however, contain remnants of cells and nuclei; in injected specimens I was even able to detect capillaries between the spheres. These capillaries could be sharply differentiated from the colorless spheres of dentinogen by their intense color.

After this discursion you will be able to understand Figure 3, which is prepared from a frontal longitudinal section through the germ of a molar tooth in a new-born infant. The preparation was decalcified until all the calcium salts were removed. In the natural state dentinogenous masses of spheres were found throughout the whole visible portion of the pulp. After careful and thorough decalcification, a wide layer of disintegrating cells was seen instead, in which odontoblasts could no longer be found. Toward the center, pulp cells are seen that are odontoblasts, and that send processes or fibrillæ into certain portions of the area of disintegrating cells and through this zone into the dentin.

In a later stage of formation dentin appears in the place of the mass of globules, and the pulp cells are seen to have undergone metamorphosis into trabeculæ of odontoblasts. Some of them become distended and appear as cartilage cells that are surrounded by a light areola. This is the stage that preceded the disintegration or the dentinogenous formation of globules and that is illustrated in Figure 4. This specimen is prepared from a longitudinal section through the dentin germ of a sheep's fetus.

As long as the formation of dentin continues, the arrangement of dentin cells in multiple layers can be demonstrated. In a preparation from the longitudinal section through a normal bicuspid in a girl of 12 years, the layer of odontoblasts still consists, at some places, of seven to eight rows of cells. I have been able to show in innumerable sections that the odontoblasts that are arranged in several rows underneath each other are not merely different layers of a single row that have become displaced so as to lie below one another, but really consist of several rows of cells.

I have been able to demonstrate this conjugation of pulp cells for the purpose of odontoblast formation in the earliest developmental stages. In Figure 5, made from the tooth germ of a human fetus, the genesis of the small number of odontoblasts that is formed in this specimen from confluence or conjugation of pulp cell (mesoblasts) can be clearly seen. In those places in which no odontoblasts have been formed, single fibrillæ, i. e., preformed dentin fibers, appear underneath the layer of enamel cells at the margin of the dentin germ.

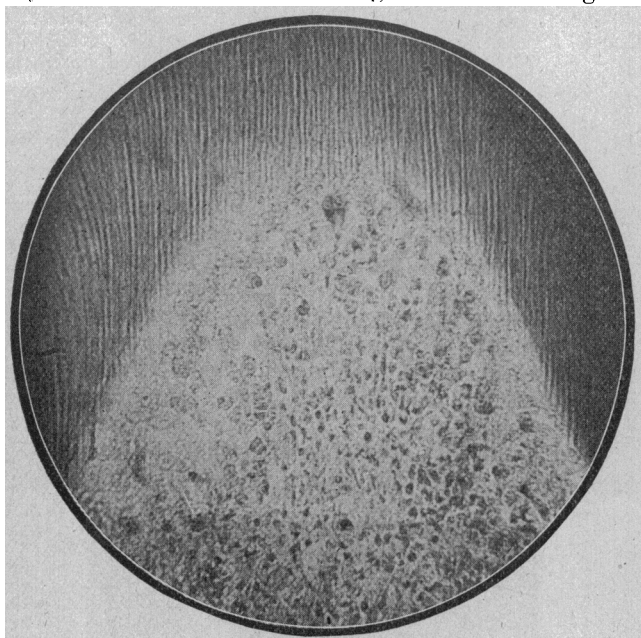


Figure 3.

In a somewhat later stage and after dentin has already been formed, odontoblasts arranged in trabeculae appear. Many of these later odontoblasts, however (as shown in Fig. 6), no longer possess a nucleus, whereas the odontoblasts in Figure 5 may even possess two or three nuclei.

I believe that I have adduced valid arguments against the existing odontoblast theory by the demonstration of these illustrations (Figs. 1 to 6), for I have been able to show (1) that, at least in the earliest developmental stages of the teeth an odontoblast originates from several cell units; (2) that during the different periods of tooth formation a multiple layer of odontoblast cells can be demonstrated; (3) that, finally, the external rows of this layer undergo metaplastic transformation so that they disintegrate. Consequently I argue that it is impossible for each odontoblast to persist throughout the whole period of tooth formation without being destroyed and subsequently replaced by other cells from which the dentin is ultimately formed.

The innumerable smaller cells that are seen underneath the layer of odontoblasts have been completely

ignored by the adherents of odontoblast theory. The fact, moreover, that the nuclei of the odontoblasts show no karyokinetic figures has been misinterpreted by them. The replacement of the odontoblasts that are destroyed in process of dentin formation does not occur by cell regeneration and karyokinesis of nuclei, but by proliferation of the small underlying pulp cells that are connected by innumerable protoplasmic anastomoses with the dentin cells.

The salient feature of this whole investigation is to show that dentin cells are actually used up in the process of dentin formation. If it should be possible to demonstrate the presence of transition forms between the different forms of cells that are in process of metaplastic metamorphosis this postulate would be fulfilled. When the formation of dentin proceeds quietly and uniformly, such transition forms are not easy to find when the formation of dentin, however, proceeds in an irregular manner, i. e., when "functional irritants influence the formation of dentin," then it is an easy matter to find such forms of cells. I take the liberty of demonstrating by the following illustrations what I wish to show. The specimens are taken from the

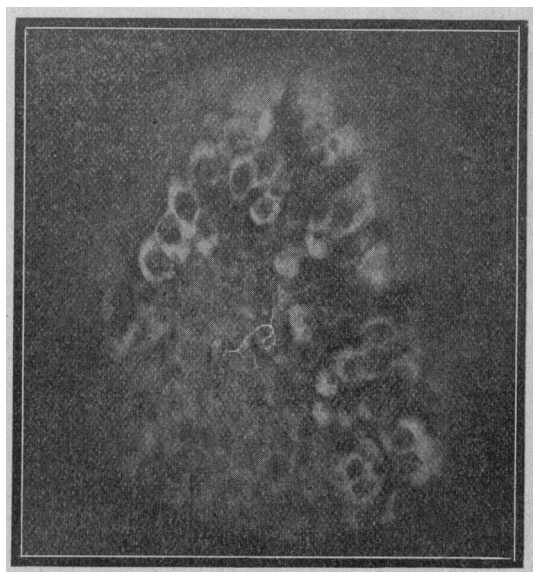


Figure 4.

roots of teeth from ruminants, rodents and fish. I have already in a separate monograph described the results of these particular investigations on the formation of dentin in these species.

Figures 7 and 8 are made from cross sections through the root of a cow's tooth in its first stage of development. We see a narrow strip of dentin with fenestrations; within the openings of the dentin are seen odontoblasts, some of them still connected with one another by protoplasmic processes. The substance of the majority of the cells is changed, and owing to "dentinogenous transformation" that is taking place, they appear black. In the narrow odontoblast layer we see pale odontoblasts, and in addition some cells that are colored black. In this layer, therefore, we see the same change in the substance of the cells throughout.

In Figure 8 the layer of odontoblasts is irregular, somewhat broader than above and interrupted in many places by protruding plugs of dentin. The odontoblasts of this layer correspond in regard to arrangement, form and substance to those that are found in the interior of the dentin.

The gradual transition of pulp cells into the layer of odontoblasts, as well as the transition of the odontoblasts into particles of dentin, can be seen clearly in Figure 9, made from the first dentin appearing in the

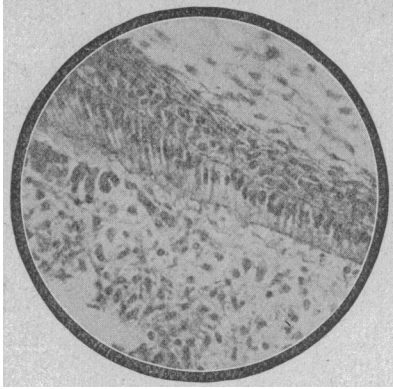


Figure 5.

roots of a cow's tooth. The changes in form that the odontoblasts undergo in the process of dentin formation can be seen particularly well. In Figure 10, in which we see that the dentin is broader, the arrange-

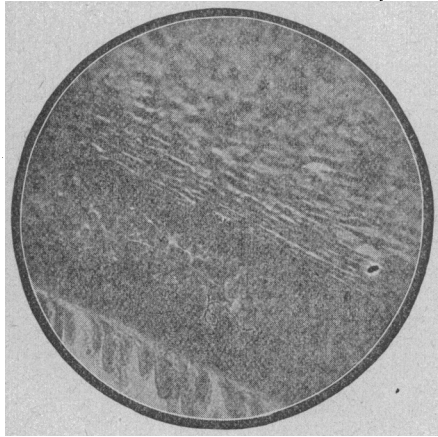


Figure 6.

ment in rows, the conjugation and the changes in the substance of the odontoblasts can be seen on a somewhat larger scale. In Figure 11 the dentin contains two plugs, consisting of different kinds of cells. The other

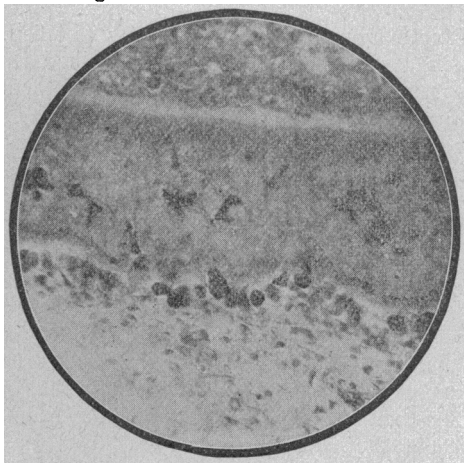


Figure 7.

margin of these plugs is very pale and is seen to be undergoing metamorphosis to dentin.

These plugs that frequently pass through the layer of odontoblasts and almost penetrate to the cement layer in the dentin, may easily create the impression that

dentin had been previously formed, had again been absorbed and had been subsequently replaced by pulp tissue that had penetrated into the spaces created in this way. This view, however, is opposed by very valid arguments. I have studied the peculiar formation under discussion through all developmental stages, i. e., from the very beginning of tooth formation to the completion of root growth, and I could determine on the basis of thousands of observations that these plugs never

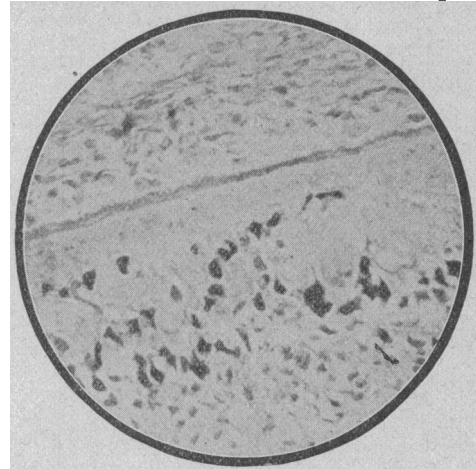


Figure 8.

penetrate into finished dentin, but are only found before the formation of dentin is complete. The plugs represent masses of pulp that remain behind in certain areas, owing to an irregular development of the dentin. Later these plugs are destined to undergo the same metamorphosis as other portions of the pulp that are further advanced in their development. Finally these plugs disappear and the area they occupy is replaced by normal dentin.

Gero Rudas has recently stated that the presence of cells in the dentin is always due to the invasion of

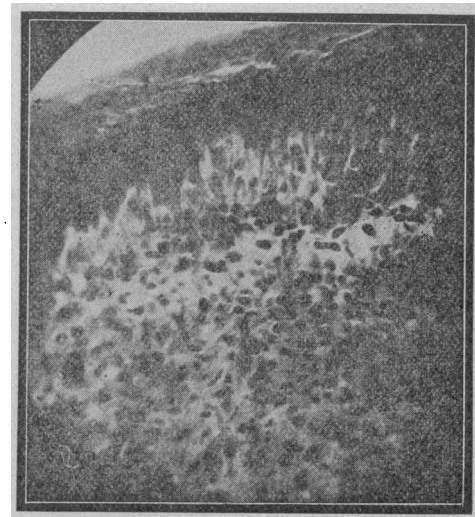


Figure 9.

leucocytes, and that the development of the pulp is deficient in those places in which incapsulated pulp cells are found. I am forced, however, to deny the correctness of these statements from my own experience. I believe I have shown that the cells within the dentin do not consist of invading pathologic leucocytes, but of mesodermic cells and dentin cells that have been passively inclosed. Under normal conditions these cells

disappear without leaving a trace. Only in those cases in which the developmental processes are arrested—as, for instance, by rapid and great destruction of the cutting edges of the teeth from overuse with resulting

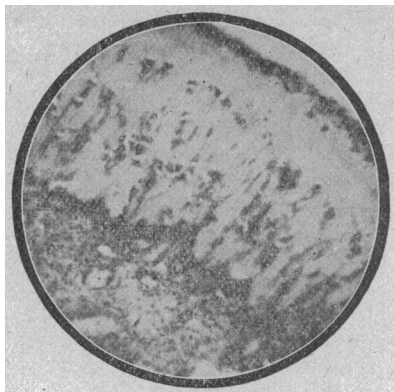


Figure 10.

atrophy of the pulp—do we see deficient development of dentin in such places.

In the dentin of certain animals, however, we sometimes see single cells, cell groups and cell territories

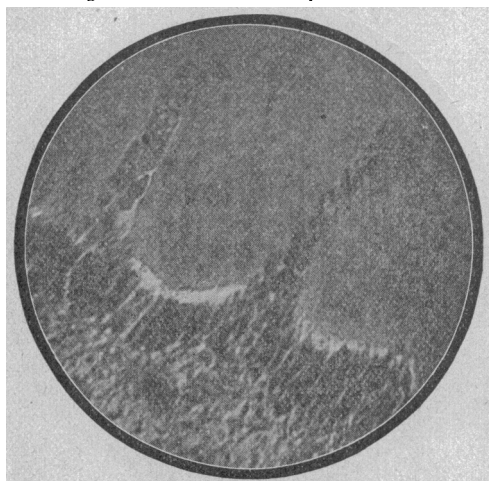


Figure 11.

that probably persist during the life of the animal; they must either persist as living cells that are normal or as decayed cell rudiments. I have been able to find such cells, particularly in the teeth of rodents and most fre-

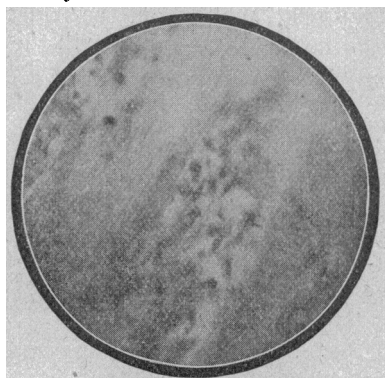


Figure 12.

quently in the molar teeth of rabbits. In Figure 12 such cell territories are shown, and in Figure 13 single cells inclosed in the dentin of rabbits. We know nothing positive in regard to the significance of those cells.

What we learn from the formation of dentin under the influence of functional irritants is that dentin may

be formed without a typic layer of odontoblasts and that the pulp cells that were heretofore considered to be completely indifferent are directly concerned in this process; and finally, that the latter are arranged in rows during this developmental process without ever being transformed into typic odontoblasts. The transformation of these cells into dentin may, however, be

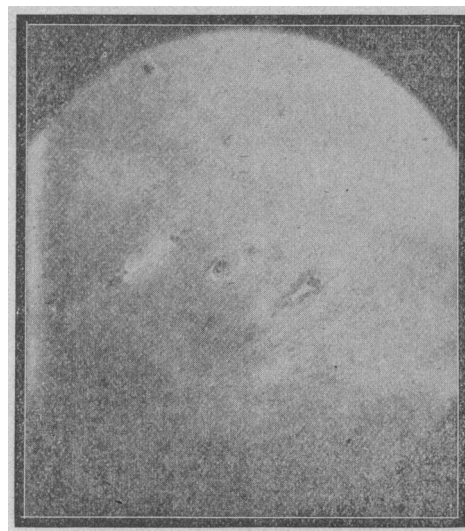


Figure 13.

followed step by step, for it can readily be shown that they, as well as the odontoblasts that are inclosed within the dentin, are gradually converted into dentin; and this, I argue, proves that in the formation of dentin, dentin cells are used up.

At all events we may assume as positive that no conclusions can be drawn from the fact that the odonto-

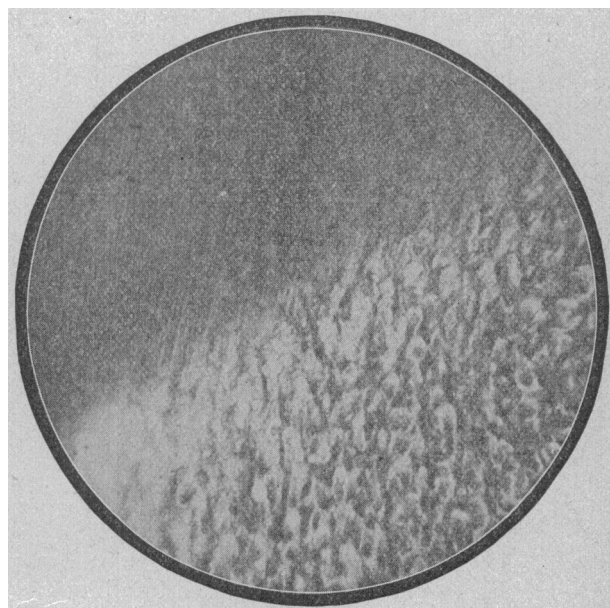


Figure 14.

blasts form one layer in regard to the permanence of the odontoblasts, nor in regard to the continuous disintegration of these cells and the replacement of the destroyed cells by new ones. I am inclined to the belief that the formation of a single layer of odontoblasts constitutes merely a transitory stage in the development of dentin, and a stage, moreover, that need not necessarily be observed in all cases.

In connection with the old odontoblast theory, the idea is often expressed that the perennial dentin cells (whose number always remains the same) advance toward the pulp as well as toward the dentin, and that in process of advancing toward the pulp they displace this tissue. The question is not decided, however, whether or not all the constituents of this displaced pulp undergo atrophy or whether they are merely compressed. In the light of my discovery of the metamorphosis of pulp cells into dentin cells and of the continuous degeneration of the latter, this view is altogether untenable.

Only one explanation seems satisfactory, namely, that more and more of the pulp becomes included in the

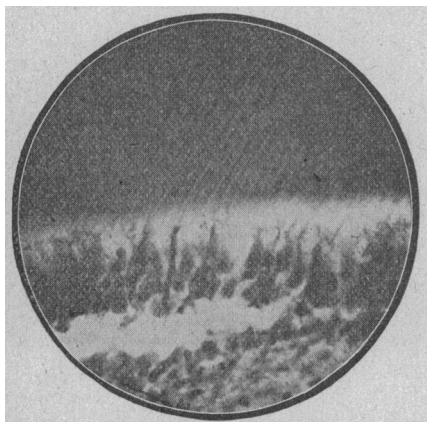


Figure 15.

process of dentin formation, must assume consequently that the pulp becomes smaller and smaller, and we do not need to postulate compression nor atrophy of the pulp to explain its gradual shrinkage.

What becomes of the blood vessels and the nerves in those portions of the pulp that are converted into territories of dentin cells?

I attempted to solve this query many years ago, and have made it the subject of a comprehensive investigation. The main results of my studies have been de-

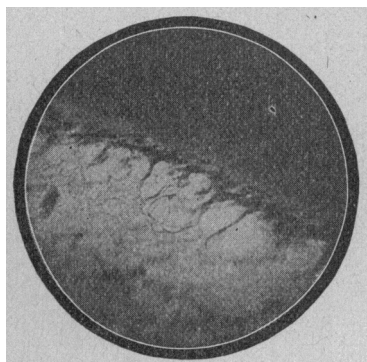


Figure 16.

scribed in a lecture. I found that the blood vessels either undergo atrophy or are converted into intraglobular spaces. I also showed that the nerves, with exception of the axis cylinders, are converted into a hyaline substance that later undergoes calcification and that the axis cylinders partially disappear and partially persist. At the same time I showed that these cylinders are very difficult to demonstrate histologically.

One of my arguments in favor of the view that new pulp areas are constantly included in dentin formation, is that the histologic structures within and below the odontoblast layer undergo continuous changes. At one

time we may see numerous capillaries between the odontoblasts, occasionally even between them and the layer of dentin; in other cases, a layer of odontoblasts will be found containing no trace of blood vessels. Sometimes the pulp immediately below the odontoblasts contains very many cells and sometimes it contains very few. Frequently a large number of very fine fibers are seen in this zone, constituting what is called Weil's layer; in other instances this layer will be found to consist of a finely granular material containing neither cells nor fibers. The vaso-dentin of fish teeth is a classical example of the continuous participation of pulp territories in dentin formation without the agency of odontoblasts. If we look at the question in this light, the dentin fibers must need assume an altogether different histogenetic relation to the odontoblasts than has hitherto been postulated. It seems impossible that the fibers should arise exclusively from the odontoblasts that are situated at the surface of the pulp, and they must also be imagined to arise in part from pulp cells situated deeper down in the pulp. In other words, they must be more or less preformed in the pulp, and can not be said to originate by metaplastic transformation as a

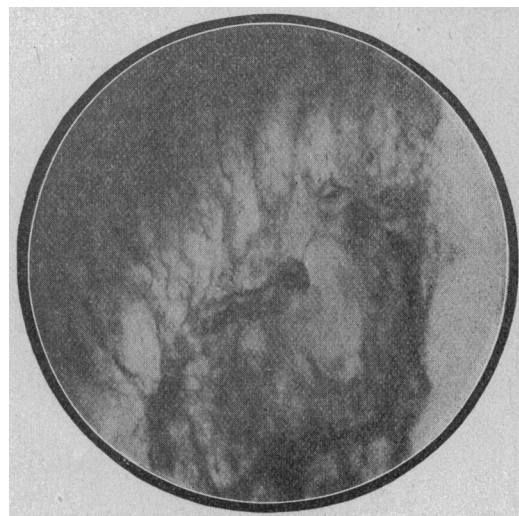


Figure 17.

residuum of the dentin cells in the process of dentin formation.

The next slides demonstrate these postulated conditions. In Figure 14, made from the molar tooth of a young rabbit, a layer is seen underneath the young dentin that contains very few cells and that is traversed by fibers. Here dentinogenous disintegration of odontoblasts has taken place and the dentin fibers originate from the deeper cells.

Figure 15 is made from the tooth of a sheep fetus and under a high power shows fibers that run between the multiple layer of odontoblasts. In other words, these fibers pass through the whole layer of the odontoblast cells into the dentin.

Figure 16 is made from the pulp of a young human premolar. Fibers will be seen passing from the layer of odontoblasts deep down into the pulp; these fibers constitute the so-called layer of Weil.

Figure 17, made from the same series of cuts as Figure 16, shows a spot in Weil's layer under a higher power. Numerous fibrillæ are seen to originate from the circumference of a single capillary. These fibers enter the odontoblast layer. They are very different in appearance; most of them stain like fine connective tis-

sue fibers; some of them are fine tubules that are occasionally found to contain a few red blood corpuscles. In most cases, however, they are so fine that blood corpuscles can not pass through. Formerly I described these tubes as lymphatic channels, but am now inclined to the belief that they are blood vessels and that they constitute very fine nutritional or tissue fluid fibers of a variety that has hitherto never been described in dental tissues. By a special method of preparation I have been enabled to demonstrate large numbers of them between the dentin cells, and could also repeatedly demonstrate their presence in dentin. These channels, that vary in thickness from one-fourth to one-third the

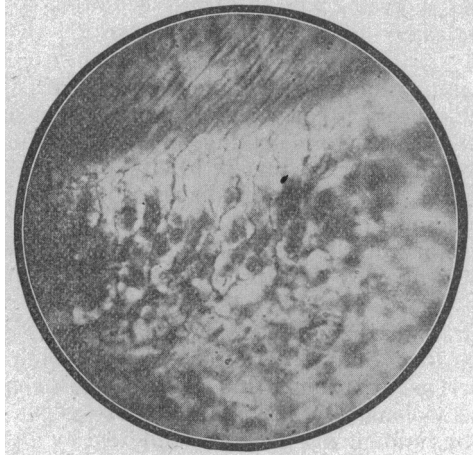


Figure 18.

width of a cell nucleus, usually terminate in a knob-like loop.

I call your attention particularly to these channels and capillaries that pass between the dentin cells, for there is resemblance in regard to distribution and location between these structures and the dentin fibers. In Figures 15, 16 and 17 both structures are seen to pass side by side through the odontoblast layer and below it.

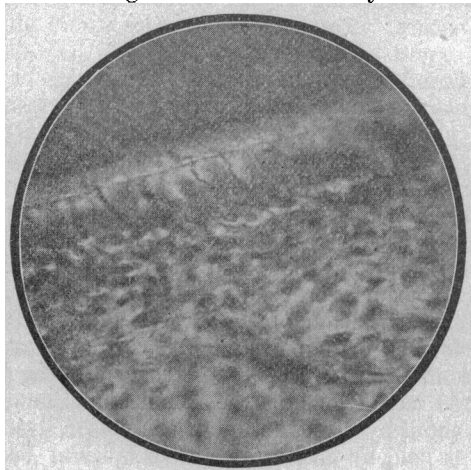


Figure 19.

i. e., in Weil's layer; but I have also been able to show repeatedly that in addition to Tomes' fibers fine channels frequently enter the dentin, the method I employed for demonstrating their presence being either injection of the specimens or special preparation of a series of cuts made from the fetus of cows, fish and sheep.

The question of the nutrition of dentin can only be solved after the relation existing between Tomes' fibers and the fine channels that I have demonstrated between the odontoblasts is explained.

I believe, however, that I have definitely solved the question of the sensibility of dentin by my demonstration of the entrance of nerves into the dentin. That the sensibility of dentin is not dependent on the presence of odontoblasts in the dentin, as taught by Black, Walk-off and other authors, could be demonstrated clinically by the fact that teeth whose pulp was completely calcified and that contained no more odontoblasts were still highly sensitive. In these cases of general calcification the axis cylinders were not involved in the process and remained intact.

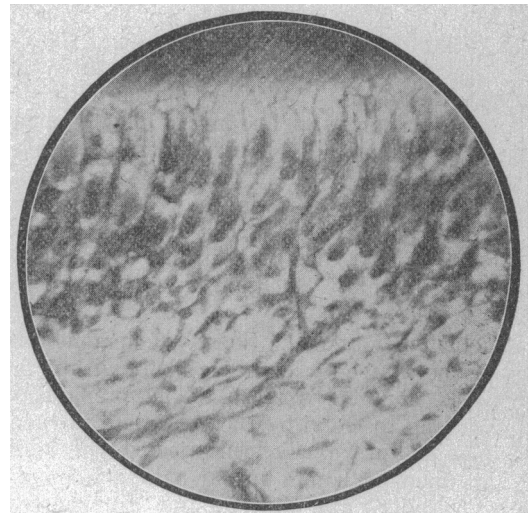


Figure 20.

Boll in 1868, Boedecker in 1883, the writer in 1892 and Römer in 1899 demonstrated the fact that pulp nerves pass into the layer of odontoblasts. The deviation of nerve fibers between the odontoblasts and the dentin was observed in 1892 by Retzius and by Huber in 1899. The entrance of nerves into the dentin I demonstrated in 1892 and Römer in 1899.

In older specimens nothing but pale fibers are seen in those places where nerves entered into the dentin.

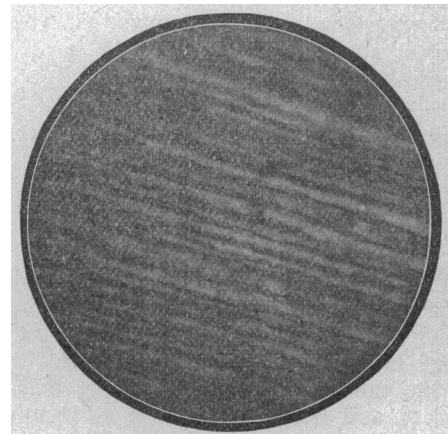


Figure 21.

These fibers, however, are clearly differentiated from the other tissues. This may be seen in slide 25, made from an old specimen prepared from a young rabbit's tooth.

In preparations made according to my modification of Mallory's stain, the axis cylinder is clearly differentiated from all other stained tissues. Preparations of this character, as will be seen from the following slides, can be preserved for a passably long time.

In Figure 18, made from the dentin germ of a sheep's fetus, a few of the axis cylinders that run toward the

center of the odontoblast layer between the dentin cells can be clearly recognized. They pass from the parietal sheath or the nerves of the dentin germ and can be followed through the whole odontoblast layer beyond the boundaries of the dentin.

In Figure 19 the entrance of nerves into the dentin can be seen still more clearly. One of the axis cylinders is seen to terminate in a knob-like ending in the dentin, and another one terminate in a similar way before reaching the dentin, i. e., in the odontoblast layer.

The course of the nerves in the odontoblast layer varies greatly. They often traverse the layer in the same direction as the dentin channels and the odontoblasts. In some places they deviate from this direction. Sometimes they form a veritable plexus with all their small lateral branches. The nerve fibrils apparently originate from cells and cell nuclei. If one looks carefully it will be seen, however, that they are merely close to the cells and really pass alongside of them.

At the point of division of the nerves of the odontoblast layer I have frequently seen one or more spindle-shaped enlargements. Such places are shown in Figure 20.

In conclusion, I take the liberty of demonstrating in Figure 21 the course of the nerve fibers within the dentin channels in a cow's tooth. The preparation was made according to Golgi by the sublimate method after the specimen had previously been injected with methylene blue. The fibers of Tomes appear pale; the fibers that I call axis cylinders are darker and frequently form spirals around the dentin fibers. They are situated between the walls of the dentin channels and the fibers of Tomes.

Basing on a series of histologic facts that I have attempted to illustrate to you with the aid of a few photographic slides of some of my specimens, I believe I have demonstrated the generally accepted odontoblast theory, the theory of dentin formation and of the sensibility of the dentin are untenable. While I do not dare to substitute a new and complete theory for the old one, I hope, nevertheless, to have contributed a few new facts by my investigations that may form the basis of an odontoblast theory that will satisfy us both in an anatomic and physiologic sense.

DIABETES AND OBSCURE PHYSIOLOGY.

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Of the many unsolved problems in pathology none perhaps possesses a greater interest to both theorist and practitioner than does the origin of diabetes mellitus. Its etiology is even yet shrouded in mystery, notwithstanding the enormous amount of study that has been devoted to it, and the situation may be summarized in the statement that of no other pathologic condition is there so much known, and yet so little. Many and various have been the theories advanced from time to time, each having a few facts apparently supporting it, but as they conflicted with each other in important particulars, one after another they were abandoned, for the advancement in physiologic chemistry made it possible more and more to explain the different and complex processes by means of which the several classes of foods passed through stage after stage of preparation and elaboration until they were fit to become incorporated into the tissues of highly organized beings. One after

another the more conspicuous organs were taxed with the responsibility, and were believed to be the seat of the diabetic phenomena. But with the progress of science the theories on which these opinions had been based were discredited, and they were reluctantly given up.

The theory that will be set forth in this paper is believed to be new as concerns its application to diabetes mellitus; but it has, however, appeared previously in medical literature in relation to purely physiologic researches concerning the ultimate state of the carbohydrate foodstuffs immediately preceding their absorption into the tissues. The various known facts on which this hypothesis is erected will be cited as required, not always chronologically, but in the order in which they apply here.

The inquiry concerns physiology as well as pathology, and, if the theory is correct, is as important to one as to the other. But before making any definite statements it will be in order to review briefly the experimental and clinical knowledge of diabetes mellitus, adding thereto our own observations.

The history of the scientific study of the subject up to the present, may with propriety be divided into four periods.

The first was inaugurated by the brilliant "piquere" experiments of Claude Bernard. The important results finally arrived at through this class of experiments were, first, that violent stimulation applied to various parts of the nervous system was generally followed by glycosuria; second, that this was the result of a sudden liberation of glycogen in excess sufficient to pass through the renal parenchyma. Here we have strong evidence to show that under some conditions the nervous elements alone may be concerned in the production of some of the diabetic phenomena. This possibility has been further strengthened, in some instances, by necropsies showing definite pathologic conditions in important nervous locations. To this class may perhaps belong the occasional instances of diabetes following severe shocks of emotional and psychic nature; also those grave cases where no morbid anatomy could be found.

The second period began with the series of experiments that demonstrated the power of certain drugs, notably phloridzin, to produce temporary glycosuria. But the glycosuria obtained from phloridzin was found to be very different from that derived from the preceding experiments on the nervous systems. According to Von Mering and others it does not appear to depend upon glycogen liberation, the blood being poor in dextrose, and the glycosuria occurring in starving animals, and even after extirpation of the liver. The dextrose here must be formed principally from the albuminous tissues, and it seems probable that the glycosuria is the result of an altered condition of the renal functional tissue, brought about by direct action of the poison itself. This opinion is held by no less an authority than Von Noorden, who suggests the possibility that in some clinical cases the glycosuria may be the result of unknown toxins analogous to phloridzin circulating in the blood and acting on the kidneys. It seems evident from these researches that there are poisons existing in Nature capable of causing diabetic symptoms through their influence on the renal functions, and therefore clinical examples of diabetes mellitus from such sources are not at all beyond the range of possibility. There is no question but that the kidneys frequently exhibit pathologic conditions in diabetes, many showing catarrhal nephritis and at times fatty infiltration. One authority