panies the eruptions of the wisdom tooth. It is true that in this particular type of cases, we have a greater degree of pain, more swelling, and inflammatory action extending to the surrounding tissues, than is the case in true hypertrophy of the gums, but the difference in the exciting cause, will explain the difference in symptomatic results.

In true hypertrophy, usually the result of some chronic inflammation of a root membrane, or the irritation of the gums produced by the products of the degeneration of the hard tissues of the teeth, we have the train of symptoms or conditions as above given; but in this particular type of cases caused by undue pressure on the surrounding tissues, and still further augmented by the close relations of the parts, and the mechanical action of mastication we account for the difference in subjective symptoms.

In chronic catarrhal inflammation, included under division second, or passive congestions we find the mucous membrane thickened and of a rose color with white spots which appear and disappear with the exfoliation of the epithelium. The patient complains of unpleasant sensations such as burning, itching and tickling, and as a result of this congestion there is a grey mucilaginous, and at times a puriform secretion poured out upon the surface of the gums. Of course the degree of this inflammation is modified to a greater or less extent, according to the time that the lesion is allowed to run, and as to whether the patient is suffering from other systemic troubles, or from catarrhal inflammations of surrounding membranes.

Phlegmonous inflammation, is somewhat sudden in its onset, but is attended with but little or no fever. The congestion is confined to both the corium and submucous connective tissue, with ulcerations occurring in small, shallow, yellowish spots or in the form of one large phagedenic ulcer. If this ulceration is allowed to continue, we will have necrosis of the osseous substance, beneath. Recovery is very slow and the surrounding membrane is spongy, from infiltration of the liquor sanguinis, very sensitive and bleeds easily. This type of cases is common, and is found in the well nourished, as well as in those who have systemic complications.

Suppurative inflammation, is where the lesion has been such, as to cause the passive congestion to result in the formation of pus, this pus may find its way to an external opening, or it may undergo, resorption. If the exciting cause has been mechanical such as compression, contusion, or laceration, the recovery is speedy with suppuration, but if on the other hand the exciting cause is a deceased root membrane of a chronic character, and the irritant becomes extended to the gum a fistula is formed, which will continue to discharge its secretion, until the parts are made healthy within.

Again referring to division one, under which we consider inflammatory œdema, acute catarrhal, herpetic and croupous inflammations, there is in common the rapidity of onset, severe pain, tumefaction and sponginess of gums, attended with the unpleasant sensation of itching and burning, in addition to this, the herpetic inflammation is characterized by the formation of vesicles, which rupture and remain a superficial erosion healing without cicatrization; closely allied to the herpetic condition is the inflammatory affection of the gums, developed during the course of an acute exanthemata, such as scarlet fever, measles etc.

The croupous inflammation, not only has the subjective symptoms that attend all active congestions, but it is characterized by the greyish structureless membraniform exudation, which may be very easily detached. This exudation takes place very rapidly and as rapidly undergoes degeneration with the production of an offensive, sanious mass, which dips down between the gum and the tooth thus exciting the root membrane into an active congestion with the corresponding loosening of the tooth. If the action is not checked the alveolar process is destroyed, and the teeth are lost.

In considering the treatment of the above pathological conditions, it must be governed according to the nature and type of the case in hand. But to generalize, I will say that in treating division first, or active congestions, with the exception of those cases due to acute exanthemata, I have found in galvanic and faradic electricity my most valuable agent, and it is so valuable, from the fact that by the proper manipulation of this subtle agent, we can correct circulatory disturbances, promote absorption of the transudated fluids, and cause the secretions and excretions to be changed into their normal condition.

Take for instance a case of noma, destructive in its results, and obstinate to cure, here by an application of electro-chemical cauterization from the positive pole, we can arrest the superficial destruction of tissue, and produce a healthy restoration of the parts beneath.

In treating the passive congestions I can apply electricity with benefit, only to the chronic catarrhal inflammations, the other types of cases of the second division are treated according to the principles of good surgery.

In dealing with hypertrophies, fibrous tumors and epitheliomata the electro cautery has been my best and most successful remedy. In order to cope with the papillary and vascular tumors, the galvanic current has been my most potent agent.

THE CONDITION OF THE DENTINE IN PULPLESS TEETH.

Read in the Section of Oral and Dental Surgery, at the Forty-third Annual Meeting of the American Medical Association, held at Detroit, Mich., June, 1892.

BY W. C. BARRETT, M.D., D.D.S.,
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I had promised the Secretary of this Section that I would prepare and read a paper at this meeting upon the "Condition of the Dentine in Pulpless Teeth." It was a subject upon which I had bestowed some thought, but with the literature of which I had not made myself thoroughly acquainted. I had read Prof. Miller's work on "The Microorganisms of the Human Mouth," and his communications to the various journals, notably *The Dental Cosmos*, but not with this special subject in mind. I had not collated his writings on this theme, and hence had not collated them. When this had been done, I was astonished at the thorough manner in which the ground had been covered. Had I examined my subject earlier, I should not have attempted an excursion in the field, but having engaged to present this paper, I will endeavor to redeem my promise, premising that I can do little save to call attention to the great work done by Prof. Miller in this direction, a work

which does not seem to be fully appreciated, and which perhaps will not be until each attempts, like me, specially to study it for himself.

In view of his exhaustive experiments, one can but wonder at the seemingly crude theories now held and taught by some men to whom we naturally turn for illumination. Perhaps I shall do more good by placing Dr. Miller's comprehensive studies before you for discussion than I would by effort in any other direction, for it must be remembered that his are not mere generalizations, such as mine must necessarily be; they are the result of long series of conclusive, scientifically conducted, original experiments and observations, and they are not to be overthrown by any inductive reasoning whatever. In view of the overwhelming original testimony which he introduces, no man has the right to dispute his deductions until he is fortified by a like mass of opposing facts, deduced from like carefully conducted experiments; or until he can indisputably demonstrate that the conclusions reached by Dr. Miller are not warranted by the results obtained.

The dentine, deriving its nutriment from the pulp of the tooth through the dentinal fibrillæ, is separated from all the other tissues of the body, through investment by the cementum and the enamel. The latter tissue is not only practically devoid of living matter, but it is not in direct relation with any tissues save the dentine itself. For our present purposes then, the enamel may be looked upon as a simple protecting cap, whose office it is to preserve the dentine and pulp from injury.

The cementum is of a different character. It is but slightly differentiated from true bone. It is in relation with a periosteal membrane, and it is nourished like bone through a system of lacunal corpuscles, connected by canaliculi.

In young teeth it is not infrequently the case that there is even a kind of system of Haversian canals, traversing the cementum and dentine between the pericementum and pulp, whose office it is to assist in maintaining the vascular supply, and incidentally, perhaps, to afford nutriment to the deeper cementum corpuscles.

The dentine is yet more differentiated, and in its structure has lost the lacunal corpuscles, while the canaliculi are modified into the dental fibrillæ. Their office is not materially changed, nor are we to suppose that the contents of the dental tubuli differ very widely from the canaliculi of true bone. They form the living portion of the dentine, as the lacunal corpuscles and the canaliculi form that of true bone. The dentinal fibrillæ are in connection with the vascular pulp, and from it derive the nutriment necessary to their continued life. The corpuscles found in true bone and in cementum are absent, and hence there is little that bears a resemblance to that concentric texture of true bone which forms its lamellated structure. At the extremities of the dentinal fibrillæ, we have the irregular inter-tissual spaces which mark the boundary line between dentine and cementum, and which may perhaps be considered as modifications of the cementum corpuscles, but they are not intimately connected with the dentinal structures.

The dentinal fibrillæ, depending as they do on the vascular pulp for nutrition, certainly lose their protoplasmic vitality when separated from it. Let us consider for a moment the condition of the dentine in a tooth the pulp of which has, without medica-

tion, just been removed. The fibrillæ have been freshly torn from their relations with the pulp, and remain filling the dental tubuli as before their violent separation. Like all protoplasmic material, they are composed of the great quaternary of elements which enter into the composition of all organic elementary structures.

But the dentinal fibrillæ probably also contain other elements, such as sulphur and phosphorus, which make it more near the nature of albumen. Indeed, it is probable that if we consider it as clearly albuminous, we shall not do much violence to nature. Carpenter says that this substance is the proper pabulum of the animal tissues generally, and so the dental fibrillæ, possibly modified somewhat, may be looked upon as albuminous in their nature.

The dentinal tubules which contain the fibrillæ are exceedingly minute. For the putrefaction of the contents of the tubuli it would be essential that putrefactive organisms should enter them. While it is quite possible that these may penetrate the tubuli, it must be to but a limited extent. In an article in the *Dental Cosmos* for May, 1890, upon "The Decomposition of the Contents of the Dental Tubuli," Dr. Miller shows that from their minute size and the very small amount of nutriment for the microorganisms which they contain, any bacteria which enter them must soon perish from want of pabulum, to say nothing of the fact that they are mostly ærobic, and so could not possibly live in the tubuli for any length of time.

But not satisfied with *a priori* reasoning, he commences a rather remarkable series of experiments and observations, from which he incontestably deduces the conclusion that bacteria do not penetrate the tubuli of undecayed dentine to any marked extent; certainly not enough to cause any necessity for the disinfection of the dentinal substance in teeth in which there has been no decay within the pulp canals.

I need not enlarge upon this. The record is open to the inspection of all, and no one has yet dared to take issue with Dr. Miller upon this point. It must be accepted that in the dentine of the roots of the teeth in which decay has made no progress, there is no infection of dentine that demands any antiseptic treatment aside from that usual in pulp canals.

What then becomes of the contents of the dentinal tubuli? It is but necessary to consider their character to form a clear estimate of this. The dentinal fibers cannot be considered as pure albumen, for that is only found in connection with other substances in the body. But whatever its exact composition it presents certain characteristics, one of which is its tendency, when at rest and separated from living tissue, to dissociate the watery portion, and to form a dried residuum which is insoluble. This condition may also be produced by most acids, by the metallic salts and by creosote.

The natural tendency then, for an albuminoid fibril, would be to assume a mummified condition, most conducive to its indefinite preservation, when kept in the state in which it will be found in the dentine of a tooth root.

Fibrine is but comparatively slightly differentiated from albumen. It contains the same elements, but it offers certain physical characteristics that distinguish it. Albumen is coagulated by heat, and by certain agents. Fibrine coagulates spontaneously

when exposed to the air. If then the fibrillæ of the dentine approach fibrine in their characteristics, they will undergo the change called coagulation without the intervention of any medicinal agent whatever. It is in this condition that, if sealed up from external influences, they present the very best state for the preservation of the tissue in which they are found. Hence the condition of the dentine is best in which the contents of the tubuli have been coagulated, or separated from the watery elements, and permitted to become a kind of minute mummy within their investing sheaths.

But the investigations of Miller, to which I have already referred, determine another matter. He did not find that the mouths of the dental tubuli, in teeth from which the pulp had been removed, were opened toward the pulp chamber.

He had under examination nineteen teeth. In ten of the roots of them he found the canals to be completely or partially lined with a homogeneous structure, containing few or no tubuli, but occasionally a formation resembling a bone lacuna, and bearing a much more near resemblance to cementum than to dentine. This layer is impenetrable to microorganisms, and therefore forms a protection to that tissue when the pulp has been removed. We find then, that the dentine is not only protected by the cementum on its external aspect, but very often by a modification of this tissue on its inner surface.

There is, therefore, little danger of infection of the contents of the dental tubuli. But even if this were possible, I cannot conceive that it would matter much. The amount of infectious matter would be so small, and the chance for the penetration of the gaseous products of decomposition through the dental tubuli, past the zone of demarcation between dentine and cementum, and finally through the cementum corpuscles and canaliculi which are themselves filled with living matter, would be so remote, and its influence upon the pericementum so infinitesimally small, that it seems folly to take it into consideration in the presence of so much more potent factors.

Clinical experience too, as is so plainly stated by Dr. Miller in the article to which reference has been made, does not sustain the theories of those who insist upon the possibility of infection from dentine. If the contents of the pulp canal be completely removed, the territory made thoroughly aseptic and the space finally filled, there is, so far as I am aware, no record of any trouble from the dentine. Even admitting that there exists within the dental tubuli matter which is susceptible to putrefactive changes, it is too small in amount to induce any disturbing conditions in the presence of a pulp canal carefully filled with a material which must seal the exceedingly minute mouths of the tubules wherever they are not already stopped by the modified dental layer of which Miller speaks. In the presence of much more important things, it seems like bad judgment to elevate such a mole-hill into a mountain, for it has a tendency to carelessness in things that need more attention. I doubt whether there has ever been a case of re-infection of the tissues about a tooth that has had its root filled after antiseptic treatment, that cannot be directly traced to some imperfection in the manipulation, or to some infected pockets outside the tooth itself. "It is well known that the usual place for the formation of an infected

pocket is at or near the apex, and that there may be a penetration of the microorganisms and the formation of infected pockets for some distance back in the bone. I have myself seen at least three such, extending back from the point of an incisor tooth until the last one was over the second bicuspid, and each of these separate pockets connected with the original one at the apex of the incisor.

They were only reached by a long incision through the gum and external alveolus, extending from one to the other until the last one had been reached. If the infection had been from the dentine, it would have manifested itself quite differently.

Undoubtedly there are cases in which the infected spot is at some point in the pericementum between the gingival margin and the apex of the root. But this does not by any means prove that it came from infected dentine. Perhaps after the apical foramen, the most usual place for the formation of an abscess is at the bifurcation of the roots of a bicuspid or molar. I believe this to be due to the fact mentioned earlier in this paper, that something analogous to an Haversian canal there may penetrate the cementum and dentine, from the pericementum to the pulp. I have demonstrated such many times.

Not infrequently an abscess refuses to yield to the usual treatment through the pulp canal, and for the reason that the medicaments fail to reach the infected point. But such instances cannot be said to be produced by infected dentine.

There is, in truth, another foramen analogous to that at the apex, and it becomes septic in precisely the same manner as does the latter.

In what I have previously urged, we have presupposed that caries has not commenced within the pulp canal. And yet we are all well aware that in cases in which the pulp had been long dead, and there has been extensive decay of the coronal portion of the tooth, there was caries of the dentine commencing within the pulp chamber and extending down the canal.

In instances like this, of course there will be thorough infection of the dentine. It will not, however, be due to decomposition of the contents of the dental tubuli, but to infectious matter which may find its way into the hollow from external sources. It would be as manifestly improper to fill such a canal without removing the carious portion, as it would be to fill a cavity from the outside without careful excavation.

Here would be an instance in which the reaming out of the canal to the depth to which caries had penetrated would be eminently proper practice. It is very seldom that this decay will extend far down the root canal, or be very deep, without such destruction of the crown and cervical portions of the root as to make preservation practically impossible.

As regards the use of coagulating agents in the treatment of root canals, I cannot conceive of any objection to them. If by any it be thought necessary that dentine should be disinfected and made more aseptic, this can only be accomplished by agents which have the power of penetration in a marked degree. These are usually coagulants, like chloride of zinc, bichloride of mercury, etc. Upon this subject I can only refer to the experiments and observations reported by Dr. Miller, in an article on "The Relative Rapidity with which various Antiseptics Penetrate Pulp Tissue," published in the *Dental Cosmos* for May, 1891.

Discussion.

Dr. E. L. Clifford said the discoloration found in dead teeth was probably caused by the formation of sulphides of some of the different metallic agents used in medicaments. The less we treat the teeth in the vast majority of cases, the better will be the results. No law can be laid down to which exceptions could not be found, but in the majority of cases the quicker we fill a root-canal and the cavity over it, the more satisfactory the operation will be. Two things, however, work against immediate root-filling: first, the difficulty of drying the root; and second, soreness of the tissues about the root. If there is no way to dry it thoroughly, or if it is too sore to work on, then it is better to wait. In all favorable cases he advocated immediate filling.

Dr. W. W. Allport said the Section was under obligation to Dr. Barrett for a most excellent paper.

In our practice we have two classes of pulpless teeth to treat. Those we devitalize leaving the dentinal fibers in the condition when devitalization took place, are in the most favorable condition for successful treatment. The other class is where the nerve has been dead for a long time. Here the condition is different, and the treatment must be adapted to the altered condition.

Our aim should be to prevent discoloration of the dentine. Whether this can best be done by the use of oils or of chlorine, or by thoroughly drying the cavity and canal, so as to leave the fibers in a mummified condition that will prevent decomposition, is the question to decide. In old cases we should use every means in our power to restore the normal color, but in freshly devitalized teeth little or no treatment is necessary.

Dr. Gish said that whatever treatment was necessary could as well be done in one sitting as in twenty, and that the duty of the dentist to his patient calls for the course of treatment necessitating the least trouble and pain, therefore he advocated immediate filling.

Dr. M. H. Fletcher said that to him it seemed that the pulp-canal before filling must be surgically clean; that is, it should have all substances removed which might contain or support any spores or bacteria. In a case reported by Dr. H. A. Smith in a paper read before the Mississippi Valley Dental Society, trouble arose at the apex of a tooth that had been filled for fourteen or fifteen years. In the discussion it was said that there had been a continual effort at the apex of the root to dissolve the root as a foreign material. This will always happen if there should be an excess of filling-material forced through the end of the root, and if the tubuli had not been thoroughly disinfected the septic matter contained in them would set up suppuration.

In regard to the substance covering the tubules from the pulp-chamber, he had learned that after the tooth is fully formed there is a deposit, as it has been called, a translucent zone, lining the chamber and pulp-canal, but the deposit as he had seen it is penetrated by the tubuli. This must be so or the deposit could not take place, and he thought the tubuli were sufficiently numerous to need disinfection.

Dr. G. L. Curtis said the paper was an argument in favor of removing the pulp while it was healthy, and not destroying it by any substances that would enter the tubules. He could understand how in teeth so treated the deposit spoken of would occur, but in teeth dead for a long time it would not be there.

Dr. A. E. Baldwin did not agree with Dr. Barrett's idea that anything was so well proven that a dentist should finally accept it as a truth without satisfying himself by independent investigation. Because another had formed a theory and by experiment satisfied himself of the truth of it, was no reason anyone else should accept it. In regard to bacteria and microbes, and the necessity of making our root-canals aseptic, he disagreed with the paper. He did not think we could get them in better condition than by desiccation. He seldom uses anything in the way of antiseptics except carbolic acid in five per cent. solution, and has very good success. If he can get the root cleaned out, in an ordinary sense, whether it is, "surgically clean" or not, and get it thoroughly dry, he did not worry about the success of the filling. He thought a great deal of the irritation about the end of the root could be treated from the outside rather than through the root-canal.

Dr. Barrett said we would have to modify our view in regard to the pathology of alveolar abscess. He believed it to be thoroughly demonstrated that the pericementum furnishes all blood-supply to the growing tooth. He did

not believe anybody ever saw arteries and veins passing through the apical foramen. He believed there is something like Haversian canals that pass through the dentine and the cementum to the pericementum, and that pus will often be found about diseased teeth at the bifurcation of the roots, and this shows why the treatment through the roots will not succeed. He believes that thorough drying will make a tooth aseptic, but sometimes the point of infection will be found away off from the tooth that seems to be the cause of suffering to the patient. In these cases, of course, the treatment must be on the outside, and followed up till the point of infection is discovered and broken up and cleaned out.

THE ETIOLOGY OF LARYNGISMUS STRIDULUS.

Read in the Section of Laryngology and Otology, at the Forty-third Annual Meeting of the American Medical Association, held at Detroit, Mich., June, 1892.

BY JOHN O. ROE, M.D.,

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Scarcely any affection has excited so much speculation in regard to its cause as laryngismus stridulus. The first definite theory regarding its etiology was put forth by Kopp in 1830, who asserted that it was caused by the enlargement of the thymus gland.¹ In 1836 Ley maintained that it was always due to "paralysis of the abductor muscles arising from the pressure of strumous, bronchial or cervical glands on the recurrent or pneumogastric nerves."² Marshall Hall ascribed laryngismus to a reflex cause, believing that it resulted from irritation in some other part of the body;³ and this theory was supported in 1849 by Reed, who observed that the spasm was frequently caused by retained meconium, fecal masses and worms.⁴

These opinions, however, were not universally accepted, for it was generally believed, as Mackenzie observes, that the weight of evidence pointed to the practical acceptance of molecular changes in the nerve centres as the essential cause of the phenomenon.⁵

In view of the fact that it occurs most often in rachitic children, Elsässer⁶ in 1843, and Lederer⁷ in 1852, maintained that it was caused by craniotabes and softening of the occipital bones, and that the attacks were excited by the pressure to which the brain was subjected when the child lay on its back.

From recent observations made from a physiological and neurological standpoint, Hughlings Jackson (than whom there is none more competent to speak on this subject) asserts that the relation of rickets to attacks of laryngismus stridulus in children is due to the abnormally soft ribs, that render the action of the diaphragm insufficient (the infant's breathing being almost wholly abdominal), and thus, especially during sleep, when the attacks commonly occur, there is an overstimulation of the respiratory centres by supervenous blood, and an excess of the natural stimulant, which result in convulsions.⁸ In a previous paper on this subject, Hughlings Jackson⁹ was at a loss to explain how this spasmodic effect was brought about by overstimulation of the

1 "Denkwürdigkeiten in der ärztlichen Praxis," Frankfurt, 1830.

2 "Essay on Laryngismus Stridulus," London, 1836.

3 "The Nervous System," 1841.

4 "On Infantile Laryngismus Stridulus," London, 1849, p. 71.

5 "Diseases of the Throat and Nose," London, 1880, vol. 1, p. 479.

6 Der Weiche Hinterkopf, Stuttgart, 1843, p. 161.

7 Beobachtungen über Spasmus Glottidis, Jour. für Kinderkrankheiten, xviii, 1852.

8 "Neurological Fragments," British Med. Jour., 1892, vol. 1, p. 489.

9 "A Contribution to the Comparative Study of Convulsions," Brain, London, April, 1886.