

lesion of the eye, and I wondered how much good those averages would do me when I came to consider his case. Averages are all right from the anatomic standpoint, but from the clinical standpoint—when one works with the brain immediately above, and dangerous blood vessels immediately at the side, and nerves which one must let alone in close contact with the cavity—averages are of no value. It is the individual case which is to be considered. In doing some cadaver work it occurred to me that the posterior pharyngeal wall and the back wall of the sphenoid cavity form the arc of a circle, of which the anterior nasal spine forms the center. In making measurements it was observed that the distance from the nasal spine to the posterior pharyngeal wall was practically the same as to the posterior wall of the sphenoid cavity. If one can pass a probe in the direction of the sphenoid sinus as far as he can pass it directly backward, he must of necessity be in the sphenoid cavity and no place else. It seems to me this is a better rule than any other measurements can be.

While the curved forceps shown by Dr. Skillern has the advantage of allowing one to cut downward a little more, it interferes with the turning of the forceps to one side, as one must do to enlarge the opening laterally, as one can not well turn a curved forceps sideways in the nose. An anatomic arrangement which must be borne in mind in cutting away the anterior wall is this: The anterior wall close to the septum is very thick and strong, while on one side a little out, one-third or one-fourth of an inch, it becomes very thin, as suggested, sometimes only membranous. These cavities should be explored very much more frequently than they are. By using a probe with a bend it can be done in the majority of cases without the necessity of removing the middle turbinate or resorting to any other surgical procedure, and it has happened to some of us to find a great many more cases of sphenoid disease since we have made the exploration of the sphenoid a part of our routine examination.

DR. ROSS H. SKILLERN, Philadelphia: In answer to Dr. Andrews, the forceps can easily be turned when in position, and I think can be entirely rotated while in the nose. One is thus enabled to bite in any direction. The curve is not sufficient to interfere in any way with mobility.

ALVEOLITIS OR SOME PATHOLOGIC CONDITIONS ARISING FROM INEFFICIENT CARE OF THE MOUTH.*

M. H. FLETCHER, D.D.S., M.D., M.S.
CINCINNATI.

Civilization brings with it the necessity of conscious intelligent care of the body, in order that good health may be enjoyed for the allotted three score years and ten. This is particularly true in case of the teeth.

I believe it possible, by the proper selection of prepared foods, to live a moderately healthy life without teeth, yet to live under such conditions could hardly be called enjoying good health. To have good teeth and healthy gums is one of the first requisites of perfect health. The belief that everybody can secure this condition, even by proper care, may be utopian, but this ideal can be nearly attained by most persons. Not only this, but a large percentage of diseased teeth and gums can be restored to comfort and usefulness by proper treatment and continual cleanliness. If every surface of every tooth could be kept perfectly clean one would have neither decayed teeth nor diseased gums. It is rare to see a mouth as clean as it is practicable for one to be kept, yet much time, money and energy are spent

for luxuries and fads, and much is also paid to doctors and dentists for the repair of ills that could be prevented by conscious, intelligent care.

To take no care of the teeth, or inefficient care, seems to be almost a universal habit. One patient put it very nicely when she said: "I find I have been cleaning my conscience all these years instead of my teeth."

Among the most prevalent diseases, as well as the most surely preventable, is that which attacks the alveolar process, resulting in its necrosis and the loss of the teeth. This disease is known by several names, but commonly by that of pyorrhea alveolaris. In my opinion it is almost wholly brought about by calcareous deposits which can be kept off by the patient.

NOMENCLATURE.

The names by which the disease is and has been known are serving their purpose, but for simplicity and clearness the usage in other branches of surgery and medicine will be adopted. That is, the name of the organ or part involved will form the foundation or generic name,

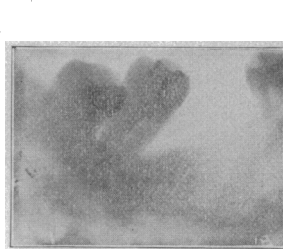


Fig. 2.—First lower molar lost from alveolitis. Second is beyond recovery. Third has recovered.

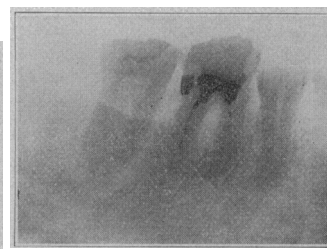


Fig. 4.—Lower first and second molars. The first molar and bicuspid have been curetted, the second molar has not. Picture taken three months after treatment. Typical of many cases.

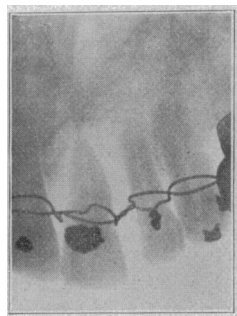
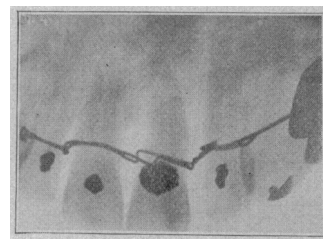


Fig. 3.—Superior incisors and canine after curetting. With wire splints to sustain them until recovery.



the nature of the disease being indicated by a suffix, and the stage or condition by a descriptive adjective. Under the name "gingivitis," inflammation of the gums follows the same rule.

The alveolar process being the part primarily involved in the disease under special discussion, the term "alveolitis" will be used in this paper in the sense above indicated, and the different stages of the disease distinguished as acute, chronic, suppurative and necrotic alveolitis. The adjectives "tuberculous," "syphilitic," "pyemic," etc., are also usable under this plan, and each ascribes a definite pathologic characteristic to the disease whose name it modifies.

Riggs' disease is a better term than pyorrhea alveolaris, since the latter name describes only the suppurative stage, and does not permit of use to describe other stages without producing a paradox or a contradiction. Such terms as "Bright's disease" and "Graves' disease" are, however, being eliminated from general medicine,

* Read in the Section on Stomatology of the American Medical Association, at the Fifty-ninth Annual Session, held at Chicago, June, 1908.

* Owing to lack of space, the article is here abbreviated by the omission of parts of text and illustrations. The complete article appears in the author's reprints.

and purely descriptive terms, formed on the principles indicated above, are being substituted.

The term "interstitial gingivitis" is subject to the criticism that it implies that the soft tissues are the ones primarily involved by the inflammation, whereas in this disease the bone seems to control the health or disease of the soft tissues overlying it. Subsequent to the initial lesion in alveolitis, the gums seem to have little more to do with the disease than the skin does in cases of underlying bone disease.

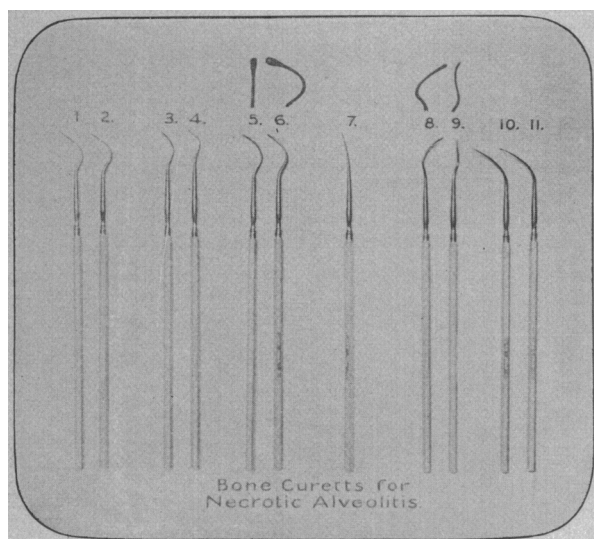


Fig. 6.—Bone curettes for removing dead and diseased tissue about the teeth and in deep pockets. The longest ones will reach beyond the apices of the roots.

PATHOLOGY.

Alveolitis in its progress includes all the processes known to other surgical diseases of bone, viz., acute, chronic, suppurative and necrotic stages. In my opinion, the initial, exciting cause, in at least nine-tenths of the cases, is the formation of calcareous deposits about the necks of the teeth. The tartar being adherent, Nature

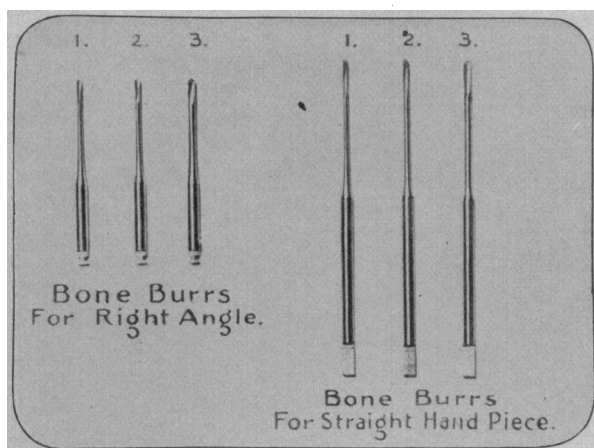


Fig. 7.—Alveolitis burs for both straight and right-angle hand pieces, for the same purpose as the curettes. The burs work more rapidly and thoroughly and are usable in a majority of cases.

can not remove it, and the inflammation becomes more extended and violent, resulting in raw tissue and open blood vessels at the point of contact with the tartar; this naturally results in the formation of pus, in small quantities at first. The surrounding of a foreign body by pus or the floating of a foreign body in pus, until the surface is reached and an opening made for

its discharge, is a familiar process in the case of a thorn or splinter in the flesh.

Tartar against the gum margin is a foreign body, and Nature endeavors to dispose of it just as she would the splinter. The tartar, being fixed, only continues the irritation, with pus as a continual result. The thin edges of the alveolar process, being continually attacked by pus or osteoclasts, soon begin to melt away, and the membranes covering the bone and roots of the teeth disappear with it. So long as the irritation remains, this process continues, its rapidity varying with environment and individual. Thus it is seen that Nature in this particular malady, if unassisted, in her attack on her enemy, the calcareous deposits, destroys the tissue about teeth until the tooth with its attached tartar is extruded, thus finally accomplishing what she started to do (Fig. 3).

The acute or incipient stage of alveolitis may continue for a lifetime, with no appreciable inconvenience or pain to the patient, the progress often being so slow that there is nothing to reveal the presence of the disease aside from the receding of the tissue about the necks and roots of the teeth. But infection may take place about one or more teeth at any time, producing periostitis.

either inside or outside of the socket, proceeding from one stage to another, ending in necrosis either of limited or extended degree.

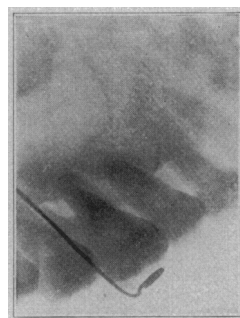


Fig. 5.—Superior molars and bicusps when antrum is involved. The wire passes through the opening into antrum.

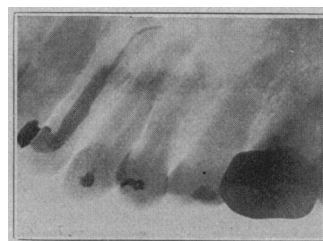


Fig. 8.—The case reported in which disease from superior lateral had perforated to floor of the nose.

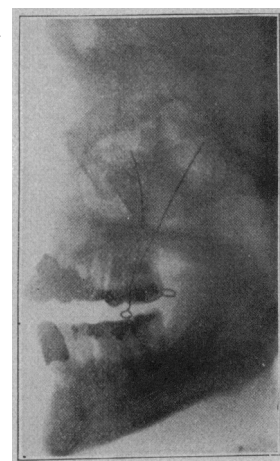


Fig. 9.—Showing a typical case in which necrotic alveolitis has perforated the antrum above first molar. The wires show the opening into the antrum to be about the diameter of the crown of the tooth. Typical of thirteen cases.

ETIOLOGY.

Abundant evidence can be presented to maintain the position that, without local irritation, destructive diseases of the soft and hard tissues about the teeth and alveolar process would be confined to those due to traumatism, thrombosis, nutritional changes and bacterial stomatitis, with now and then an infection from the circulation, as in other tissues; but these affections would then be attributed to their special causes and treated accordingly.

The most frequent point of attack for alveolitis is in the septums between the teeth, where tartar is not disturbed by lips, tongue, food or brush; but any place

about the necks of the teeth, where tartar is undisturbed, is apt to be the point of attack (Fig. 3).

The opportunity for the entrance of infection to the alveolar process is almost unlimited, for calcareous deposits about the necks of the teeth are found in every adult human mouth and in the mouths of most children.

The quantity of tartar and the irritability of the soft tissues in its presence varies in different individuals, and with each individual according to his power at different times to resist disease after its onset.

Autointoxication or lack of resisting power, however caused, makes one more susceptible to other diseases as well as to this; but a low state of health could hardly be the sole cause of an infectious disease. The microbes of infection are ever present in great abundance, Miller having isolated more than one hundred different kinds of bacteria from the juices and deposits in the mouth. So, when the gums and periosteal membrane are sore and bleeding from the impingement of tartar, bands, the wedging of food, or any other irritant, infection may readily start and the disease follow. Lesions of the gums about the teeth are so universal that the wonder is, not that there are so many diseased mouths, but that there are not more.

A lesion of the gums, because of immediate proximity to the bone, seems pre-eminently a suitable place for the beginning of any of the surgical infections of bone. The infections to be considered in alveolitis are those that produce osteomyelitis, caries or tuberculosis of bone, gummata or syphilitic lesions, and actinomycosis. The ultimate result in each one of these infections is dead bone.

Necrosis from lead, mercury, phosphorus and all other chemical causes, are not to be considered here, since their etiology is apparent.

The diagnosis, then, in any single case, lies between an infection by pus-producing germs, such as accompany osteomyelitis; tuberculous germs, which produce caries; syphilitic germs, which produce gummata; and actinomycosis. The last named produces "lumpy jaw" in cattle, and is not frequent in man, especially in this country. It is not uncommon, however, in countries where camels are used as domestic animals, and is confined largely to these countries. It is sometimes seen in cattle in the United States, and now and then in man in cattle districts; but in this paper it hardly need be considered in the light of a factor in alveolitis.

Then, in consideration of the above, we have three kinds of bacteria to consider as the most probable sources of infection, namely, pyogenic, tuberculous and syphilitic.

SYPHILIS.

The consensus of opinion seems to be that syphilitic infections are far less numerous than either pyogenic or tubercular infections. In my opinion this is especially true in regard to the alveolar process, although the mouth seems particularly favorable to other lesions of syphilis. In patients who seem to be syphilitic, recovery is much slower, the infected tissue being more sensitive, and persisting in showing inflammation long after other patients with other infections have recovered. The characteristic thickening of the periosteum in the gummata of syphilis can be felt over the diseased bone in the neighborhood of the apices of the teeth, either on the buccal surface or inner surface, or both. Gummata in the periosteum must be distinguished from hardened lumps at the apex of dead teeth or exostosis of bone. Gummata usually appear, however, on the septum of

bone between the roots rather than over them. This is because syphilitic bone is more apt to be in the cancellous portion of the body or between the roots. These conditions are rare, however, in comparison with pyogenic and tuberculous infection. Local treatment is practically the same in all infections, excepting that it must be continued longer in syphilis. Syphilitic infection may be either congenital or acquired. The congenital form appears more frequently in the bones of children, and particularly in the long bones, but the acquired form frequently shows in the bones of the head and face, the alveolar process and maxillæ being no exception. With our present incomplete knowledge, however, syphilitic alveolitis is not easy to distinguish from tuberculous alveolitis.

OSTEOMYELITIS.

In considering osteomyelitis, which is pyogenic, it is to be remembered that in a typical case the seat of trouble is in the marrow of one of the long bones, oftenest the thigh bone of a child or youth, although it may occur in the periosteum, in the form of a felon, for instance. It is considered that the infection comes from the blood and oftenest finds a lesion or point of least resistance in the marrow of some bone and begins its work there. The bony walls being unyielding, the patient's suffering is intense, and the spread of the infection to the surrounding marrow and bone is rapid. The destruction and pain continue to increase as long as there is no exit for the pus and other products of inflammation. Early interference by the surgeon is demanded here, for if time must elapse for the pus to burrow its way to the surface through bone and flesh, many days at least, and probably many weeks must pass, during which time much tissue is destroyed and untold suffering endured.

The above description, if somewhat modified, applies to an abscess at the apex of some dead tooth, or an infection under the gum tissues, either from the blood or by infection from a lesion at the alveolar border. Very acute and painful infections about the alveolar process imply that the pus is confined at some depth below the margin of the gums. If the infection were nearer the gum margin, either in the socket or on the alveolar process, the discharge would either not be confined or would make its exit at an early stage of the disease, giving comparatively little pain in either case. The infection remaining, however, continues its destructive work, and a chronic stage of suppurative alveolitis or true pyorrhea alveolaris is the result.

If not interfered with, this condition may continue indefinitely, now and then assuming an acute stage when new foci become infected, quieting down again when the sinus is opened to the surface. But each time this occurs there is loss of more bone in the alveolar process or in the deeper cancellous portion, often extending into the body of the jaw, and many times into the antrum. This destruction continues slowly or rapidly, according to the acuteness or chronic activity of the disease, until the tooth or teeth are lost. When the tooth is out, if the infection is not mixed with other germs, such as tuberculous or syphilitic, healing becomes prompt and complete in the normal time for the building of bone structure, that is, in two or three months. Under proper surgical treatment, however, healing can be made just as prompt and complete, and the teeth retained and made useful, provided the dead and infected bone be removed before the foundations of the tooth are too greatly destroyed (Fig. 4).

TUBERCULOSIS.

In considering tuberculous infection as a cause, my experience makes me believe that alveolitis in all its stages, after its initial lesion, is more largely due to this infection than to any other.

Descriptions, by two of our best authors and surgeons, of this disease in other bones than the alveolar process, will better serve than my own, as a description of tuberculous alveolitis and its ravages. The action of tubercle bacilli on bone, as here described, is particularly applicable to carious alveolitis.

Park¹ goes into minutiae in regard to the "infectious granuloma" of Virchow, which plays so large a part in the slow destructive process in caries of bone. This granulation tissue is the effort on the part of Nature to repair the damage done by tuberculous or other infection, but the granules also become the seat of infection and serve as a further means of increasing the progress of the disease, especially that of tuberculous type. Carious necrosis into the alveolar process and jaw bones seems exactly the same in character and extent as this infection in other bones, and in my hands has yielded to the same character of treatment that has proved successful on other bones (Fig. 4).

Although many microscopic tests have been made, no satisfactory results have been obtained. Probably none can be obtained until the work is taken up under proper laboratory environment for both culture and microscopic tests; and even then the work might be uncertain, as Miller has shown² in his endeavors to find a special bacterium of this disease. He sums up his work as follows:

From these experiments, we might conclude that, if there is a specific bacterium of pyorrhea alveolaris, it does not readily grow on gelatin, a result which is of value in so far as it indicates that in further experiments on this subject, media should be employed which admit of being kept at the temperature of the mouth. At the same time the thought suggests itself that possibly the bacterium of pyorrhea alveolaris, like so many mouth bacteria, is cultivable on none of the artificial nutrient media, which would of course render all experimenting useless.

TREATMENT.

In the first place, in extensive cases, the tissues will need to be cocaineized, a 5 to 10 per cent. solution being injected into the pockets. If the tissues are very sensitive, a weaker solution can be injected into the gums, the same as when teeth are extracted. The periosteum is very sensitive, but bone is not so, and dead bone not at all so. The use of cocaine must be determined by the conditions. I use it as sparingly as possible, for the reason that if enough is used to produce anesthesia it usually produces a sleepless, disagreeable effect, if it does not prostrate the patient for a day or two. Usually the cocaine can be postponed until the point of curetting and burring is reached.

Next, all calcareous deposits must be removed. There are hundreds of instruments made for this purpose, each operator having his own preference in instruments and his own methods of use. But the deposits must be most thoroughly removed and the teeth and roots polished with an orange stick and pumice, floss, discs or brush, or whatever the operator can best use to restore the teeth to a smoothness equal to that they originally had.

Experience shows that scratches and grooves on the roots from instruments are nearly always present, al-

though they should not be. These scratches, when under the margin of the gums, do not cause the roots to decay nor prevent the tissues from becoming healthy; but calcareous deposits remaining, however slight in amount, will continue to irritate and increase, resulting in a repetition of the various stages of the disease, namely, initial lesion, increased inflammation, infection and destruction of bone. One of the most important parts of the treatment consists in teaching the patient how to keep the teeth free from calcareous deposits. If tartar is not allowed to form about the gum, it will not form below, and the disease is prevented; otherwise it is likely to return (Fig. 1).

After removal of deposits comes the removal of necrosed bone. No repair is ever complete unless this is done. It is oftenest done, however, by Nature herself without intentional aid from the doctor.

Now, it would seem the rational thing for the physician to seek out the points of attack, and by conscious effort to assist Nature by tearing down and removing her enemy's citadel, the infected and dead bone, be it ever so small an amount, thereby reducing the enemy's forces and stimulating Nature to a renewed and stronger effort, and thus not only shortening the time of repair, but making it more sure. In this way recovery can be induced in many cases which would otherwise end unfavorably. The progress of tuberculous infection in bone is so slow that many patients, even if not entirely cured, may be much relieved, in that the disease becomes quiescent, and the affected teeth, which otherwise would be lost, remain useful for years.

The conscious effort referred to consists in the use of delicate bone curettes and suitably shaped burs for removing dead bone from about the necks and roots of affected teeth. Examination should first be made about the neck of every tooth with a delicate probe; one of the bone curettes is usually good for this purpose, but a more delicate instrument, like a smooth broach, is often necessary. The sense of touch readily reveals the presence of bone not covered by soft tissue. When such is present there is very little if any sensitiveness, whereas if gums or periosteum still cover the bone, the patient quickly responds to the pain and the touch reveals the difference between live and dead bone. No matter what one may believe as to the causes of the disease, the fact remains that, in the mildest cases of alveolitis in its second or chronic stage, there is always more or less death of the bone about the teeth, and the dead and diseased tissue should be removed in order to give Nature the most assistance.

The third or necrotic stage is that in which the alveolar process is more deeply involved, the necrosis usually progressing along the root in one tract, until near the apex, then gradually encircling the root, also continuing its ravages into the maxilla. In the upper jaw the infection often goes into the antrum, now and then into the floor of the nose, and many times destroys large sections of the cancellous portion of the superior maxilla (Figs. 5 and 9).

In the treatment of these cases, packing is not called for except in instances of extensive removal of tissue, such as curetting the antrum or the removal of considerable portions of the body of the bone.

If systemic treatment is demanded it should be directed toward the restoration of normal health by such means as the elimination from the diet of foods which do not become wholly digested and assimilated, and the copious drinking of pure water. If the local trouble

1. *Treatise on Surgery: Tuberculosis*, I, 160.
2. "Microorganisms of Human Mouth," p. 329.

is complicated with other diseases of moment, the general practitioner or specialist should take charge of these.

TIC DOULOUREUX.

Besides these deep-seated extensive cases, there is another class which calls for knowledge of local pathology and great keenness of diagnosis. They are non-suppurative and attended by more or less neuralgic pain, sometimes a matter of small discomfort, again reaching the magnitude of tic douloureux. They usually occur about a perfectly good tooth, and there is no evidence of inflammation, either on inspection or on percussion. The one symptom which calls attention to them is entirely subjective; that is, the patient complains of discomfort or pain in the neighborhood. The tooth affected is usually next to one more deeply affected or next to the space where one has been lost. With a smooth broachlike probe inserted close to the tooth some place will be found which is not sensitive, usually approximating the space where the adjoining tooth was, or the tooth more deeply affected with the disease. The bone is found nude of periosteum and eburnated, that is, abnormally hard. This hardened condition is easily detected by the touch; the bone is very hard to cut even with a bur, and is not sensitive. The treatment consists in the removal of the eburnated bone with a curette or bur; the bur is the better.

Disease of the periosteum, wherever found, is usually accompanied by neuralgic pains. In view of this fact, I have been led to believe³ that tic douloureux might be due to a disease of the periosteum, starting as above described, and continuing even after all the teeth have been lost.

Subsequent experience has very much strengthened my belief in this theory, and I believe it lies with the stomatologist to work out this hypothesis and find the proper treatment for this most distressing malady. At the present time I would call this variety periosteal alveolitis from tubercular infection, or periosteal caries.

DIAGNOSIS.

The diagnosis of chronic cases of alveolitis needs suitable instruments, sensitive touch and great care; and the treatment, in addition to this, requires a perfect knowledge of the anatomy of the structures and their pathology.

To operate in any of these cases is surgery and not dentistry, so that the stomatologist also needs to be skilled in operative surgery to a degree which gives him suitable knowledge and confidence in himself to handle a patient undergoing the removal of part of the alveolar process either above or below. He must also be capable of opening and properly treating the antrum of Highmore, and of knowing if this cavity is diseased beyond its connection with the teeth, or whether it is diseased at all or not, and of handling a patient under collapse, either from shock or local anesthetic. Further, the operator should be so in touch with his patient and the extent of the operation as to know whether the operation should be performed under local or general anesthesia, and whether it should all be done at once or at intervals of a few days or weeks; for general and systemic complications from secondary and acute infection may occur at any time.

These features will present themselves more strongly when a history of some of the cases is given and the radiographs are shown.

INSTRUMENTS.

Under the impetus, first, of an effort to be thorough, then of surprise at the prevalent ravages of the disease, then of a desire to learn its full extent and how to treat it, I found myself bewildered at times. Consequently, I began to invent instruments which would reach the extended tracts of necrosis. These instruments are here presented for criticism (Figs. 6 and 7).

The curettes, or hand instruments (Fig. 6), are all of the hoe and hatchet type, varying only in size of blade and length and shape of shank. The attempt is made by these variations to reach any extended tract of necrosis. The necrosed portions are usually friable—that is, in the state of osteoporosis—and can easily be cut away with the curettes; but certain phases of the disease and certain kinds of infection often result in osteosclerosis; that is, hardened or eburnated bone, on which the curettes make little headway. For cutting these hardened bones I have made some extra long bone-cutting burs (Fig. 7), both for the straight and right-angle hand-pieces. The contra-angle seems to be more suited to the work, however, than the right angle. The burs for the contra-angle will reach all cases in the lower jaw and most of the upper, but a bur two and one-half to three inches long—that is, one long enough to reach to and into the antrum—is often necessary for the upper jaw.

Experience has taught me that the burs must be of the fissure-bur type, with rounded ends, and small enough to cut away the septums of bone without injury to the roots. Often this can not be done with any bur, since it would necessarily be so delicate that it would break very easily, an accident which occurs quite frequently. The same thing also occurs with the curettes. The alveolitis burs are made without the usual shoulder near the head in order to prevent them from breaking so easily. In many instances the roots of teeth are so close to those adjoining that no instrument sufficiently strong can be inserted into the space.

OPERATIONS.

On account of proximity to the floor of the nose, the antrum and the roof of the mouth, necrotic and suppurative alveolitis may be more serious in the upper than in the lower jaw, since all these cavities may be involved singly or at once. It has fallen to my lot to be obliged to remove much of the alveolar process and to excavate more or less extensively into the cancellous bone of the superior maxillary.

My records show that in the past twenty-two months I have treated 112 patients with necrotic and suppurative alveolitis. This does not include patients in whom moderate amounts of tartar have been removed and the teeth polished. Out of these 112 patients, in 13 the antrum was perforated; 17 had acute osteomyelitis; 8 a considerable portion of the alveolus and bone from the maxilla was removed; 5 had symptoms approaching tic douloureux; and all required more or less curetting of the alveolar process and maxilla. Twenty-four had one or more teeth that could not be saved. In one of the antrum cases the disease was caused by intranasal disorder and has been troublesome for five years. Two patients went to a nose specialist, and I do not know their later condition. In the other ten the conditions have been cleared up, as shown by transillumination, and the patients have been discharged, so far as antrum trouble is concerned.

3. Fletcher, M. H.: Periosteal Caries, *THE JOURNAL A. M. A.*, Sept. 2, 1899, xxxiii, 585.

Affections of the antrum caused by the teeth are much more amenable to treatment than when the disease is from intranasal disorder.

In one instance, in the lower jaw, it was necessary to remove all the teeth and alveolar process, back of the canine on one side, curetting extensively into the body of the bone. In another the patient was operated on by a general surgeon and much of the body of the lower jaw removed on one side, in addition to all the alveolar process from the canine back. The patient was afterward sent to me to have the upper teeth and jaw treated. I found extensive necrosis running back into the cancellous bone from above the superior lateral and central. The harder plates of bone under the floor of the nose and the antrum had not been perforated, but their shapes could easily be felt with the instrument. This patient has been under treatment for six months and is nearly well.

By proper handling, these patients can be conservatively treated and the structures often induced to heal without the radical operation of extended removal of maxillary substance, as is now so frequently done in the hands of the general surgeon.

The laws of regeneration do not permit of complete healing of bone tissue inside of several weeks at the shortest, and often require several months, so that patience and careful watching are necessary on the part of both patient and doctor. One patient now on my list for nearly a year, who would not submit to a radical removal of the cancellous bone in the superior maxillary, has submitted to a small amount of removal from time to time and is gradually recovering under two dressing treatments a week. This case, however, was the result of a dental abscess arising at the apex of the superior lateral which had discharged into the floor of the nose (Fig. 8).

The extensive ravages of alveolitis must be seen to be fully understood, and it is my desire that stomatologists may take up this task and unitedly work out the complete history of the disease, together with the most approved methods and instruments for its successful treatment.

REGENERATION.

At the present time, the greatest uncertainty about alveolitis is in regard to the origin of the cause or the kinds of infection producing it. The symptoms are nearly the same in all instances, and the treatment is practically the same in all cases, namely, the removal of deposits and of any necrosed or dying tissue, which constitute the hindrances in Nature's path to recovery.

After curetting and burring have been done, the cavities should be washed out with a warm antiseptic solution to remove the cuttings. The blood should be allowed to clot in the cavity. My plan is to be careful not to disturb the blood clot so long as it remains aseptic. If there is a tendency for pus to form, the wound should be washed out every one, two or three days, according to conditions, and, if pus continues after ten days, a second, third or even more attempts must be made to remove the offending material.

Aseptic blood clot is Nature's "false work" or scaffolding on and into which she builds all new tissues, no matter of what kind. The less the healthy clot is disturbed, the more prompt is the repair. In the blood clot is formed the granulation tissue of repair, which is the second stage of the building of new tissues. Any disturbance to these granules is also a hindrance to repair; hence packing is seldom called for.

Following the formation of granulation tissue is its gradual change into tissue of the kind from which the granules sprang. This process continues until the wound is healed. The hindrances to this normal process are foreign bodies, dead or dying tissue, and the presence and increase of bacteria. Nevertheless there is a continuous and never-ceasing effort to extrude or to tear down and carry away any foreign body.

This new or granular tissue thus endeavoring to envelop the diseased or dead tissue is called the involucrum. Dead tissue thus completely involved is attacked from every point, and often an enemy thus attacked can be destroyed, whereas, if only partly surrounded, it would hold its ground.

This process is one of the most common things to be seen in the human mouth, especially in patients who have been or are being treated for alveolitis. When the crown of a tooth has disappeared, the involucrum begins to grow over the edges of the root, and at times a portion of the root may be completely enveloped and finally become absorbed. Inflamed gums, from irritation of tartar or other causes, constitute an attempt to involve and destroy the enemy and replace lost tissue.

And now to the point of the above explanation. What really happens in a large number of cases treated for suppurating or necrotic alveolitis is that an effort is made by the doctor to remove the deposits. Some doctors may try to remove dead bone. In any event, the tissues are wounded in the effort, and all or most of the deposits and dead bone removed and the limiting zone or membrane at least partly broken up. Thus Nature is stimulated to a new and vigorous effort to repair the damage done. The involucrum grows up and around the root, enclosing more securely any deposit that may have been left. Of as much importance, however, is any necrosed or infected bone which the involucrum may enclose, for it is likely to increase in extent and, as stated, many times involves the deeper portions of the maxilla, often reaching the antrum (Fig. 9).

If I have suggested anything new or valuable, I believe it is the necessity of either curetting or burring about all teeth where the disease is found, and of more thorough removal, if the disease is deep seated—that is, of consciously assisting Nature to do what she is always trying to do. These deeper-seated sequestra, however, do not always prevent the involucrum from closing tightly about the neck and roots of the teeth, especially if the involucrum is composed of bone tissue. In such a case, at some point about the tooth, dead bone may be found or a place which can be penetrated to dead bone or to a cavity above. This, however, can be done only with a delicate but sufficiently stiff probe such as I will show. A small portion of necrosed bone may be absorbed and complete regeneration finally take place after Nature has been stimulated to a new effort, but the approved surgical treatment is to remove all dead or dying bone if it be practicable.

It must be borne in mind that the process of regeneration of bone in these cases is accompanied by acute inflammation of the surrounding tissue. The new-forming bone is soft and yielding; the callus does not show much tendency to harden for three or four weeks, and does not become ossified for six or more weeks. Often, therefore, after the removal of bone about the roots, the teeth are looser than ever, and at times need splinting with wires to other and firmer teeth, the splint remaining until ossification is completed, when the teeth

will become firm and useful, provided there remained in the beginning enough live bone, periosteum, and endosteum for the purposes of regeneration.

CONCLUSION.

In conclusion, I apologize for the length of this paper. To me, however, every portion of the subject is fraught with so much interest and clinical experience that it has been difficult to select that which seemed most necessary in presenting the subject.

The radiographs will somewhat support my practical experience, but only your own experience, past and future, can reveal to you the great prevalence and ravages of this disease.

11 East Seventh Street.

ABSTRACT OF DISCUSSION.

DR. EUGENE S. TALBOT, Chicago: Dr. Fletcher says that a large percentage of diseases of the alveolar process are of local origin. Here I disagree with him. He says, however, that "to have good teeth and healthy gums is one of the first requisites of perfect health." No one will dispute this statement. Perfect health, however, means health of the body, generally, as well as the mouth. Dr. Fletcher says that "the term interstitial gingivitis is subject to criticism since it implies inflammation of the soft tissues as being the primary tissues involved." The constitutional changes which cause this disease nearly always take place in the arteries, as demonstrated in my work on "Interstitial Gingivitis or So-called Pyorrhea Alveolaris," and, therefore, the bone is liable to be the first involved. The term "interstitial gingivitis" was given for the very reason that the disease was deep-seated; the word "interstitial" means between tissues including both hard and soft. From Dr. Fletcher's reasoning the word "alveolitis" is objectionable since it does not refer to the gums and soft parts which are always first involved when the disease is of local origin, as he asserts it is, in a large percentage of cases. The term "pyorrhea alveolaris" should be used only when pus is present. Dr. Fletcher ignores the autointoxications of pregnancy and those caused by drugs and diseases. Certainly arteriosclerosis and endarteritis obliterans, which are present in almost every case, are not local irritations. He says that a low state of health could hardly be the cause of an infectious disease. Researches have shown that the only infection which takes place in this disease is pus infection. The inflammatory process precedes this stage. Dr. Fletcher describes the effects of syphilis, tuberculosis and actinomycosis as infections and classes all pathologic processes as infection. These diseases may or may not cause infection of and destruction of bone, etc. Very few patients come to me for treatment suffering from these infections. Dr. Fletcher speaks about necrosis and necrotic tissue; asserts that tartar is the great cause; that "infection of the alveolar process is almost unlimited;" that "calcareous deposit is found in the mouth of every adult human and of most children." If it be due to tartar, how is the disease accounted for in animals which do not have tartar but which do have deposits, the result of the disease? My studies on animals and human beings have demonstrated that this disease (in the constitutional and large majority of cases) is the result of irritation in the blood, which sets up absorption of bone (not pathologic in the sense of necrotic), but precisely such absorption as results in the alveolar process when a tooth is extracted or in tooth movement. Dr. Fletcher forgets that the alveolar process is wholly unlike any other in the body, being a doubly transitory structure. Its end is annihilation. It is natural for this bone to be absorbed; not so with other bones. The alveolar process is only waiting for some slight irritation (not sufficiently active to cause necrosis) to set up absorption. This irritation is caused by changes in the blood stream (auto-intoxication and acidosis). Syphilis, tuberculosis, actinomycosis and all diseases will cause such changes in the blood, and always act on the alveolar process. Dr. Fletcher brings forth no researches in proof of the correctness of his premises. I still

believe that there are local causes, but a large proportion are constitutional. It is my experience that regeneration of the alveolar process, if it does take place, is a process similar to that of bone absorption. If acute inflammation sets in the formation of osteoclasts and osteoblasts, halisteresis, and Volkmann's perforating canal absorption can not take place.

DR. G. V. I. BROWN, Milwaukee: When Dr. Fletcher uses the termination "itis," without modification, then he lays himself open to the necessity of accepting the well-known etiologic factors that come into play with reference to all inflammations. He would lead us to infer that he believes the questions of constitutional characters are very slight; but again he tells us that he does consider them important when he says that the cause is entirely local. His paper speaks of "autointoxication, or greater susceptibility to disease." The latter is not autointoxication, but may be a result of it. Do you believe for a moment that by any kind of scraping you could prevent the dark line appearing at the gingival border, or the effect on the structures of excessive mercury administration? Would scurvy do the same thing? I have shown several times a series of slides of an individual who had what under Dr. Fletcher's term would be alveolitis; of course he had alveolitis, but every organ in the man's body—heart, kidneys, liver, spleen—showed the same round-celled infiltration, the same destructive process going on as in the alveolar process, but worse in the alveolar process than elsewhere, because he had alveolitis the result of leukemia, which is a constitutional disease. I thought that everybody was willing to admit that where inflammation, especially of such a character as this exists, there are both predisposing and exciting etiologic factors. Dr. Fletcher gets good results from his treatment, and I am perfectly willing to accept his surgical principles and methods of treatment, because I know he knows what he is talking about. There is no difference between us except in the matter of trying to make it appear that this one factor, calcic deposits, so greatly overshadows the constitutional and all other generally recognized factors.

DR. STEWART L. McCURDY, Pittsburg: The terms "caries" and "necrosis" denote incidents in the history of disease, and not diseases themselves, if we take into consideration the etiologic factors that produce disease. Caries of any tissue, hard or soft, is the result of some local or constitutional disease. Necrosis, likewise, implies a history of constitutional disease. We must go back to the etiologic factors that produce the disease rather than consider these as distinct diseases. From Keen's new work on surgery I find that primarily considered tuberculosis of the alveolar process is rather rare compared to syphilis and other diseases of this structure. I would like to apply the word "hereditary" rather than "congenital" to these diseases. Syphilis is rarely congenital. The syphilitic condition exists at the time of birth, but not the disease itself. "Congenital" means existing at birth, and "hereditary" means that the disease develops at any time in after-life, but that it was latent at the time of birth. The majority of these conditions develop later. I made the estimate in 1897 that 50 per cent. of all bone and joint diseases of children had syphilitic ancestry and this view was so radical at that time that the paper was not published; yet since that time many more radical papers than mine have been published. Most practitioners of medicine and surgery had not seen as many of those general syphilitic diseases as those of us who practice among rail-roads, mill-workers and the laboring classes. Men who are in sky-scrapers do not see the cases that men see who are treating this class of cases.

DR. VIDA A. LATHAM, Chicago: The whole of this discussion goes back to the first principles. What is pyorrhea alveolaris, Riggs' disease, or interstitial gingivitis? Let us have a pathologic classification; in the lack of it lies our trouble! Dr. Fletcher is misunderstood by some people, Dr. Talbot by others, and I think that Riggs himself was misconstrued. The condition termed alveolitis exists as a later period. The disease starts with a gingivitis, and I believe that Dr. Fletcher's statement is true that thrombosis plays a part; that it is a primary factor; in other words, the vasomotor system losing control and being soon affected by thrombosis and the degeneration of

the nerve fibers of the gingivæ, prevents contraction in some cases and gives advanced retraction in others, and thence result two forms of pyorrhea, if I may use that term: 1, the acute, with the spongy gum, bleeding, pus formation, with chronic suppurative alveolitis as the termination or sequela; 2, the chronic, in which there is typical fibrosis, recessional gum, with that peculiar black stain running around the teeth—few know what it is—which produces a mechanical abrasion that gradually causes the tooth structure to become less and less and the alveolar process may be denuded of the periosteum, and on top of that we get recession of the gum which finally exposes the cementum of the tooth. This form of gingivitis is seldom or never mentioned as a pyorrhæal condition. I believe it is pyorrhæal myself. Another reason why I think that this classification is going to be borne out is this: In both cases, whether of the pus-forming type or the fibrosis variety, there is eversion of the teeth. In some cases of fibrosis the teeth are turned right out, so that they have to be extracted instead of being replaced. I think that the fibrous form of alveolitis goes more particularly with Dr. Talbot's theory; when there is faulty elimination or metabolism accompanied by liver disease, very often cholangitis is present, and anemic areas in the stomach, and the pancreas is at fault. In these cases there is a change in the tooth structure showing that there is arteriosclerosis behind it.

DR. DANIEL HOPKINSON, Milwaukee: I have frequently tried to isolate some organism as an etiologic factor in this condition, but have met with negative results. But I think that possibly another way of isolating organisms, as the tubercle bacilli, is by the inoculation of the guinea-pig. If we question whether a certain lesion is tuberculous or not, by inoculating a guinea-pig, say in the groin, we can positively state whether the tubercle bacilli are present; even if in few numbers they will positively develop in the guinea-pig. So far as the histologic changes in tuberculosis or syphilis of these structures are concerned, they are frequently identical. If, however, the *Spirochæta pallida* be found, and we are positive that it is the pallida and not any other spirochete, then we can state positively that we are dealing with a syphilitic lesion. But unless an individual has frequently found these *Spirochæta pallida* and has been able to differentiate them from the other spirochetes present, such a diagnosis would not be reliable. Pus-producing organisms might possibly be isolated by another inoculation.

I think that the tubercle bacilli are very rarely, if ever, obtained in sputum in a pure type. Especially in the mouth we find almost every form of suppurative organism; therefore, if we are dealing with a tuberculous lesion, there is undoubtedly present a secondary infection with pus-producing organisms.

DR. M. L. RHEIN, New York: I believe that the apparent variance of opinions on this subject is due entirely to the fact that we have never had a proper classification or nomenclature of pathogenic conditions involving what may be termed the periodontal aspects. The exciting cause ought to be recognized in the nomenclature and associated with it the predisposing cause. A good example for such a nomenclature is that adopted by the dermatologists. We have been discussing here a large variety of diseases and attempting to include them under one particular head. I thoroughly believe that a large number of diseases can be properly termed "alveolitis," with an adjective expressing the predisposing cause, just as well as I am assured that a large number could be as appropriately termed "gingivitis," with its proper predisposing cause expressed by an adjective. I am sure that the class of cases in which faulty metabolism is a predisposing cause is an entirely different class. Those are the cases in which the tooth has a tendency to become everted or protrude. The result of my investigations on that particular type of disease is that it is due to the abnormal condition of the blood which is circulating in the pulp of the tooth and acting as an exciting cause. Faulty metabolism of some kind produces an irritation that makes itself felt in the end circulation that we have in the pulp of the tooth. I have given considerable attention to the microscopic examination of pulps of this nature, and I have found in such cases the

most remarkable pathologic conditions in the pulps of teeth that have never even been abraded on their exterior surfaces. The removal of such pulps has been sufficient to produce a cure of such conditions. Dr. Fletcher and Dr. Talbot are discussing different diseases. Nothing can better demonstrate the absolute necessity for this Section to produce a proper classification of the different diseases of the alveolar or, what may be a better term, periodontal, regions of the mouth.

DR. EDWARD C. BRIGGS, Boston: That interstitial gingivitis is an entirely separate thing from infectious alveolitis seems to me perfectly clear. There is no question that infection often supervenes in interstitial gingivitis and brings about the true infectious alveolitis which Dr. Fletcher describes. But that it exists as a first condition in all cases I could not concede. Interstitial gingivitis, as I understand it, exists where there are no deposits, and that is one thing that is often overlooked in the treatment—that many cases of true interstitial gingivitis show very little evidence either in the way of pus or of deposits. These cases of infection we see and may confuse them at first with cases of interstitial gingivitis. I have seen two cases of actinomycosis which at first I thought were severe cases of interstitial gingivitis. I proved them to be cases of actinomycosis, and subsequent surgical treatment cured the condition.

Destruction of the alveolar process is not necessarily always caused by inflammation, but is the physiologic condition due to any irritation that is set up about the alveolar process, as after extraction. So we get it in the irritation that comes in gingivitis. The first treatment of these cases of interstitial gingivitis is the removal of the pulp, which is the beginning of the treatment in a great many cases, to offset the pathologic changes.

DR. M. H. FLETCHER, Cincinnati: I agree with Dr. Rhein that the nomenclature is still unsatisfactory. I have plainly stated in the paper that the soft tissues are first involved and that the bone becomes involved subsequently. The word "gingivitis" indicates the earlier stages of the disease, in which the part would recover with the simplest treatment did not the bone become the foundation or primary factor in the chronic stages. At the present I believe that the use of "alveolitis" as a generic term allows of the best description of the diseases of the soft tissues by the use of qualifying terms, as described in the paper. The name, however, is insignificant compared with the pathology and etiology. Both Drs. Talbot and Brown ignore this and other parts of the paper in their discussion. Arteriosclerosis is given great prominence in Dr. Talbot's book as well as in his discussion. The terms "arteriosclerosis" and "endarteritis obliterans" are practically synonymous, the latter term being a description of the closure of the arteries in arteriosclerosis. This disease of the arteries and the high pressure that accompanies constitute practically a disease of the large vessels, not usually found in the capillaries, and yet the capillaries, smaller arterioles, and Haversian canals are the only blood vessels found in the tissues about the teeth. Faulty metabolism, from any cause, fosters and augments any existing weakness or lesion, and this was set forth in Dr. Briggs' paper as in my own. Dr. Talbot says that I "ignore the autointoxications of pregnancy, drugs and disease" as a special cause of the disease. I do not believe, as he does, that this is a special factor here any more than it would be in any portion of the body where a point of least resistance is found. If Dr. Talbot will read the paper more carefully he could hardly make the remark that I class "all pathologic processes as infection," and I refer him and Dr. Brown to the paper itself for answers to most of their objections and to a more full discussion found in reprints of the paper and discussion. Dr. Talbot further says: "Dr. Fletcher forgets that here is a condition wholly unlike any other part of the body, the alveolar process being a doubly transitory structure. Its end is annihilation. It is natural for this bone to be absorbed; not so with other bones." I can not see any foundation for this statement. If by "doubly transitory structure" he means that the alveolar process is gradually becoming obsolescent and thereby is deficient in quality of structure or lacking in resisting power, I differ with him most emphatically. According to the laws of evolution, if an organ is becoming obsolescent

from disuse it gradually grows smaller, and may grow weaker as far as muscles are concerned, but it always receives the requisite amount of blood and is perfectly nourished, but each generation finds it smaller. Under natural selection many hundreds of generations are required to make an appreciable change in a genus or a species. I believe that the written history of man is not long enough to make evident much change in him. According to the laws of evolution the teeth would disappear before the alveolar process. The teeth stimulate its growth; when they are extracted the process disappears. If, however, new teeth were present in the jaw and were to erupt, the alveolar process would be rebuilt as in the second or third dentitions. In rodents, elephants and all animals that have teeth of continuous growth, the alveolar process is also continuously renewed, showing that in vertebrate types the alveolar process is anything but a weak or transitory structure. The tail of the tadpole might be called transitory, but up to the time when it begins to disappear it is well supplied with circulation and nourishment and is perfectly strong for its function.

[Limitations of space in THE JOURNAL necessitate abbreviation of the discussion here, but copies of the paper and full discussion can be obtained by sending to Dr. Fletcher.]

CIRCULATORY DISTURBANCES IN DIPHTHERIA.*

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Matthew Arnold, in his "Essays on Criticism," says: "It is true that all time given to writing critiques on the works of others would be much better employed if it were given to original composition."

From the truth of this statement we must occasionally except the subject of medicine, for it is not the work of one man that is analyzed, but the opinions and hypotheses of many. These often give us information of great value in regard to matters which are insusceptible of direct experimental investigation, and from such an analysis we may often see the gaps in our knowledge that require to be filled.

It is not possible within reasonable limits to give a systematic discussion of the whole subject of the circulatory disturbances in diphtheria, but I wish rather to consider the underlying causes of these disturbances, some aspects of the symptoms, and the rational therapy indicated to oppose or counteract these underlying causes.

A thorough knowledge of the cardiac lesions which result from diphtheria is requisite for a consideration of the production of symptoms, and this has been afforded by the careful and extensive studies of Hayem, Birch-Hirshfeld, Leyden, Unruh, Steffen, Ribbert, Romberg, Councilman, Mallory and Pearce and many others. Though they differ in regard to certain unessential details, their agreement is general and striking. Moreover, Aschoff and Tawara, who on the basis of a critical study opposed many of the views in regard to the heart muscle in other diseases, state that the changes in diphtheria are extensive and severe. Thrombosis, endocarditis and pericarditis play only an occasional rôle. The two chief lesions are the parenchymatous and interstitial.

Fatty degeneration is extremely frequent. It varies in degree from an extremely mild to a marked change and it constantly accompanies the severer lesions. This degeneration may occur in the first few days, but it is

also found late in the disease. A much more severe degeneration which affects all parts of the muscle fiber, the contractile elements, the protoplasm and the nucleus, and which leads to the formation of granular detritus and large irregular hyaline masses, is also found. This is both focal and general and is at times so extensive that it was estimated by Councilman that in one of his cases one-third of the heart was destroyed. This extreme degeneration is only found late in the course of the disease, rarely earlier than the seventh day.

The interstitial changes are of two types. In the one type there are focal collections of lymphoid and plasma cells. In the other there is an invasion of the degenerated and necrotic muscle cells with endothelial cells and polymorphonuclear leucocytes. The first of these types is usually accompanied by some degeneration of the myocardium, but it is not dependent on it, while in the second type, during the process of repair, an extensive formation of connective tissue may take place. It should be emphasized that the interstitial changes are essentially late changes. Up to the sixth or seventh day, then, fatty degeneration is practically the only abnormal condition present in the heart.

There has been no satisfactory investigation in regard to the intrinsic nerves and ganglia of the heart. The findings in the pneumogastric nerves will be considered later.

It is usually customary to divide the circulatory symptoms according to the time of their appearance into the early, or those occurring during the period of local lesions, which is usually febrile, and those occurring late or during the period of convalescence, which is almost always afebrile. This is not strictly correct, but is more satisfactory than dividing them according to their mode of production, which would take for granted things that are still sub judice.

EARLY CIRCULATORY DISTURBANCES.

The early circulatory disturbance occurring at the height of the disease is extraordinarily fatal. When fully developed it is practically never recovered from and but a few hours at most separate the beginning of the symptoms and the termination. Before the introduction of antitoxin it was greatly feared, and with reason; now, thanks to antitoxin, it is rarely seen. We meet it most frequently in older children or young adults in whom for several days the throat process has been neglected. The unfortunate patient presents all the evidences of malignant diphtheria. The process in the throat is extensive with membrane covering tonsils, uvula, soft palate, pharynx, sometimes extending into the larynx. The nasopharynx is filled with membrane, making nasal respiration impossible, and the upper lip is excoriated with the bloody purulent discharge which escapes from the nostrils. The cervical glands are greatly swollen so as to fill all the depressions in the neck and make a veritable collar around the throat.

The temperature is sometimes greatly elevated, at others little above normal, but the prostration is extreme and there is marked pallor. The heart's action is usually rapid, but the sounds are for a time of fair quality and the blood pressure is not much below normal. Before long, however, though sometimes after the local process in the throat has begun materially to improve, the pulse becomes weaker, the blood pressure falls rapidly and progressively, the heart's action becomes more and more feeble, and the patient quickly passes into a

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