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## PREFACE

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THE rôle of *Endamœba buccalis* as the specific cause of pyorrhœa alveolaris, or Riggs's disease, has very recently been recognized. It happens, as it seldom has happened before in the history of medicine, that whenever the specific cause of the disease was found, a specific, efficient remedy against the parasite was already known.

This infection and disease is so wide-spread that it is practically universal, and is the cause of the loss of more than 50 per cent. of all the permanent teeth that are lost from any cause. The slow, incipient nature of the disease results in its presence usually not being suspected until considerable irreparable damage has been done, and often not until many teeth are practically lost.

The most successful treatment of this disease will naturally depend, as in other diseases, largely upon a knowledge of the specific cause of the disease, the manner in which this agent produces the disease process, and finally upon a knowledge of the manner in which the specific treatment acts on the causative agent.

The object of this book is to present the subject in a simple, concise way, in the light of recent information. Former theories and ideas as to cause and treatment are left out altogether. No attempt is made to include a review of the literature on the subject except in so far as it may seem essential in presenting the present view.

Though diseases involving the teeth are always of special interest to the dentist, pyorrhœa alveolaris, with its long-drawn-out suppurating process in the mouth, the possible effect of absorption of infectious and toxic substances, and the harm that may come from being unable to properly masticate the food, is of special interest to the physician also. The book is intended to be of practical use to both the physician and the dentist. It is also believed that many of the more intelligent laymen will find it a convenient source of information as to the nature, and especially the prevention, of this disease, from which all sooner or later lose their teeth if they live long enough.

In order to make the work more convenient when required for quick reference, the essential points in each chapter are summarized at the end of the chapter.

THE AUTHORS.

NEW ORLEANS, LA.,  
May, 1915.



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# ALVEOLODENTAL PYORRHEA

## CHAPTER I

### DEFINITION

MANY terms have been employed to designate the disease which is discussed in this book. Among them are pericementitis, alveolitis dentalis, alveolar periostitis, Riggs's disease, Fouchard's disease, and pyorrhœa alveolaris. In selecting a name we have taken into consideration the pathologic process and the tissues involved.

The term pyorrhea can hardly be objected to, for there is pus formation and flow in every case from the very incipiency to the loss of the last tooth. In the smallest lesions the amount of pus produced may be so small as to require microscopic examination to demonstrate it, but it is always present. Later the quantity becomes much greater, coincidentally with the increase in the extent of the lesion. The term pyorrhea, however, could be applied to processes in which there is pus production and flow involving other structures and tissues than those of the mouth. It is necessary, therefore, to be more specific and to indicate what structures are in-

volved. *Pyorrhœa alveolaris* is perfectly correct in speaking of the advanced stage of the disease, but, as will be seen in the chapter upon Morbid Process, the pyorrhea disease exists for months or years before it involves the alveolar bone. During this early stage the peridental soft tissue above the level of the alveolus is alone involved. The term *pyorrhœa alveolaris* hardly covers this early stage of the disease. At the time when the cause of the disease was unknown and when this early stage was not recognized and appreciated, "*pyorrhœa alveolaris*" met the requirements, and it remains a question whether usage may not remove any objection to its continued use.

The word alveolodental means pertaining to a tooth or teeth and to a socket or sockets. Alveolodental pyorrhea would more correctly describe the disease under consideration, and we shall employ it in this book. A proper definition for alveolodental pyorrhea is, "A destructive disease of the supporting structures of the teeth," and we may now add, "caused by *Endamœba buccalis*."

**Summary.**—Alveolodental pyorrhea: A destructive disease of the supporting structures of the teeth, the specific cause of which is *Endamœba buccalis*.

## CHAPTER II

### HISTORIC

THE occurrence of minute, unicellular, ameboid organisms has been a matter of record for many years. There are now known to be many different classes and species. Many varieties are entirely free living—feeding on smaller animal and vegetable organisms in many warm, moist places, such as in ponds, among accumulations of decaying vegetation, etc. Other varieties live and reproduce as parasites in, or upon, the higher animals. Some of these parasitic amebæ are most probably only harmless commensals; others, by their constant occurrence and reactions in morbid processes, are now thought to be pathogenic, either directly or indirectly. At present we are chiefly concerned with the species known and described as *Endamæbæ buccalis*, which we will show to be constantly present in the lesions of alveolodental pyorrhea. The full evidence of its pathogenicity will be given more attention in the succeeding chapters.

As far back as 1849, when this form of life first began to attract attention, Gros described an

ameba found in the mouths of adults; and gave it the name of *Amæba gingivalis*.

In 1862 Sternberg gave a detailed description of an ameba found mixed with the deposits of tartar removed from the teeth. This ameba differed somewhat from the ordinary free-living ameba, or the *Amæba coli* found in other parts of the alimentary canal, and received the name of *Amæba buccalis*.

In 1879 Grassi recorded the finding of an ameba in the necrotic material from carious teeth, to which he gave the name of *Amæba dentalis*.

The above findings were restudied by Prowazek, when, in 1904, he not only confirmed the described ameba of Sternberg, but redescribed it in detail in the light of the then present knowledge; and in one of the first attempts at the classification of the various forms of ameba placed it in the class *Entamæba*. He also found the same organisms present in scrapings from carious teeth, and further concludes that all amebæ that had been described as occurring in the mouth, except *Entamæba coli*, were in reality the same species, or *Entamæba buccalis*.

Kartulis, in 1900, working in Egypt, described an ameba found in suppurating tumors of the jaw which he thought to be the causative agent. This was also the opinion of Doflein, who confirmed

the work of Kartulis, and named the organism *Entamæba kartulisi*. Craig, in his notable work on "The Pathogenic Amebæ of Man," reviews the descriptions given of this ameba, and, finding that they are so similar to the "tetragera" type of *E. histolytica*, concludes that they are probably identical. The abscesses referred to are secondary infections from a preëxisting amebic infection of the intestines. Secondary abscesses as a sequel to the intestinal infection with *histolytica* often occur in the liver, brain, bone, etc.

Recently, in July, 1914, Smith and Barrett announced at a meeting of the Pennsylvania State Dental Society the finding of *Endamæba\* buccalis* in nearly all cases of Riggs's disease (pyorrhœa alveolaris), and their belief that the organisms were pathogenic on account of the favorable effect on the lesions of the disease produced by the local use of emetin hydrochlorid, a well-known amebicide.

In August of the same year Dr. Angelo Chiavaro presented a paper to the American Dental Society of Europe in which *Endamæba buccalis* was reported to have been found in 22 cases of pyorrhœa,

\* Throughout the remainder of this book the term *endameba* is used in place of *entameba*, proposed by Barbagallo and Casagrandi in 1897, because of clear historic priority, first pointed out to us by Allen J. Smith (personal communication). *Endameba* was proposed for a parasitic ameba of the cockroach in 1897 by Joseph Leidy (Proc. Phila. Acad. Nat. Sciences, vol. xxxi, p. 204), eighteen years before *entameba* was first proposed for parasitic amebæ (Annali d'Igiene Sperimentale, 1897, vol. vii, p. 103).



and 14 other cases not affected, out of a total of 68 persons examined. This author concludes that the amebæ are not pathogenic, but were in reality an adjuvant to the auto-disinfection of the mouth.

Independently of the work of these last two authors, we noted the occurrence of *Endamæba buccalis* in 86 cases of unmistakable alveolodental pyorrhea, while in numerous normal controls they could not be found. These findings were reported to the Orleans Parish Medical Society in September, 1914, at which time we expressed the opinion that the endamebæ were pathogenic for many reasons, chief among which was the fact that they disappeared and the lesion promptly began to heal upon the hypodermatic administration of emetin hydrochlorid. Again in February, 1915, in the Journal of the American Medical Association, our former observations were confirmed by further observations in which we had found endamebæ present in more than 300 cases of pyorrhea.

Middleton and Barrett, in the Journal of the American Medical Association (vol. lxiii, No. 20, p. 1746), record the finding of *Endamæbæ buccalis* in chronically inflamed tonsillar tissue, and that the condition was greatly benefited by the hypodermatic use of emetin.

Up to the present time there is no conclusive



evidence that any pathogenic ameba has been cultivated artificially either in pure or contaminated culture. Thus the differentiation of the various species of ameba remains a comparative study of the organism along structural and reproductive lines in general.

In the large series of cases of pyorrhœa that we have studied all the organisms appear to agree with the descriptions given for *Endamœba buccalis* except that occasionally we find in addition an ameba of the *Endamœba coli* type; the latter, particularly in preparations from the surface of the lesion, or from deposits upon the buccal mucous membrane.

The literature bearing upon ameba gives bare mention of the occurrence of this very common ameba, probably on account of the very generally accepted view of its non-pathogenic rôle. In his book, "The Pathogenic Amebæ of Man," Craig gives a very excellent review of much of the important literature upon this organism. It is from this source and recent observation that we have compiled the following brief description of *Endamœbæ buccalis*.

*Geographic Distribution.*—World-wide.

*Occurrence.*—In pyorrhœa lesions around the teeth of almost all human adults and many

younger people and also in the follicles of diseased tonsils.

*Morphology.*—Relatively small. Usually from 6 to 30 microns in diameter, averaging about 25 microns. The ectoplasm is distinct and clear. The endoplasm is granular and vacuolated, having a reticular structure. The vacuoles contain various particles of phagocytized material, and none of them are contractile. The nucleus is well defined, spherical or oval in shape, and has a thick nuclear membrane containing refractile material or chromatin. A small centriole is usually situated near the center of the nucleus.

*Motility.*—This is somewhat sluggish in character, and is produced by the extrusion of ectoplasmic pseudopodia into which the endoplasm is drawn. The motility is not nearly as marked as *E. histolytica*, but more so than *E. coli*.

*Reproduction.*—This is usually accomplished by simple binary fission—the nucleus dividing by mitosis, the nuclear spindle often being observed. Schizogony has been described.

*Cultivation.*—There are no records of successful cultivation. A personal communication from A. J. Smith states that he has kept the organism alive and motile for twenty-four hours in a mixture of equal parts of egg-white and blood-serum.

*Pathogenicity.*—Up to the work of Smith and

Barrett, and later our own, these endamebæ have been looked upon as harmless commensals. The following pages will deal more in detail with this question.

The reader is referred to works on protozoölogy for more complete and scientific description of this and other species of amebæ which this book makes no pretense to cover.

## CHAPTER III

### ETIOLOGY

**Specific Cause.**—Endamebæ, of the species *buccalis*, in most instances, if not in all, are present in the lesions in all cases of alveolodental pyorrhœa. We have found them in all of more than 300 cases in which the disease had developed to the extent that it could be diagnosed. In two cases of very early disease, in both of which the diagnosis remains in doubt, we failed to find endamebæ. In each negative case only one examination was made, and that before we had acquired the best technic.

They cannot be demonstrated in material taken from normal gums. They are often found in material taken from gums which appear on casual observation to be little or doubtfully affected. Upon more careful examination with proper instruments, however, pyorrhœa pockets can usually be demonstrated. These may be so shallow as to be doubtful, but more often they are much more extensive than the appearance would indicate. There may not be sufficient pus for it to be recognized by the unaided eye, but in every instance

in which we found endamebæ many pus-cells were present and recognizable with the aid of the microscope. It should be understood that a considerable amount of pus could be present in material, and still not be recognized on macroscopic examination.

We have failed to find endamebæ in microscopic preparations that contained no pus, and have learned to discard such preparations and not to waste time in fruitless search for endamebæ in them.

Bárrett and Smith found endamebæ in all of 46 cases of pyorrhœa, and did not find them in 7 normal mouths. Chiavaro found them in all of 22 cases, and also in 14 cases not diagnosed pyorrhœa alveolaris. This is to be explained by the reasonable probability that he did not consider the diagnosis indicated until the disease process had reached the alveolar level. Strictly speaking, pyorrhœa alveolaris must involve the alveolar structure, but, as is shown in the chapter on Pathology, the early stage of this disease is pyorrhœa dentalis, which later extends to become pyorrhœa alveolaris.

The constant presence of endamebæ is one argument that they are the cause of the disease, but it is far from certain proof to that effect. Another important bit of valuable evidence is the

location of the endamebæ in the lesions. They are much more numerous in the depth of the lesion, and just at the juncture of the dead and dying tissue with the living. In fact, their habitat (see chapter on Pathology) is the dying tissue in the bottom of the lesion, where bacteria and other agencies are few or entirely absent. This fact is

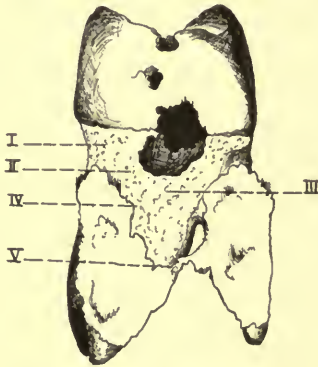


Fig. 1.—Drawing of an extracted tooth from which material the photomicrographs, Figs. 2, 3, 4, 5, and 6, were made. A part of the peridental membrane came out attached to the roots, and is indicated by the large white areas with ragged edges.

illustrated by the pictures on pp. 28, 29, 30, 31. Other evidence that the endameba is the specific cause of alveolodental pyorrhea is the very certain and rapid results that follow treatment with a specific amebicide. It might be argued that application or injection of emetin or other drugs into the lesion might benefit by some local healing effect or by killing harmful bacteria, and not certainly by its ame-

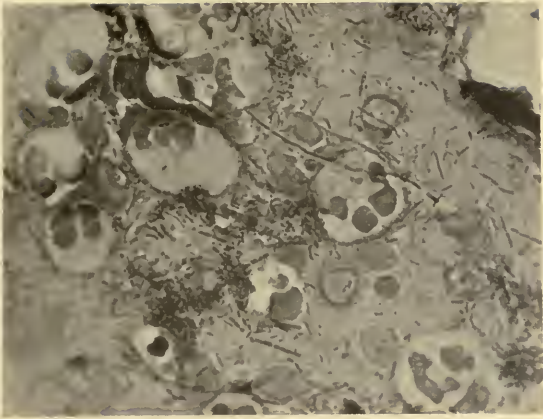


Fig. 2.—Photomicrograph of representative field of specimen made of material removed from area marked I in Fig. 1. Shows pus and bacteria, but no endamebæ.

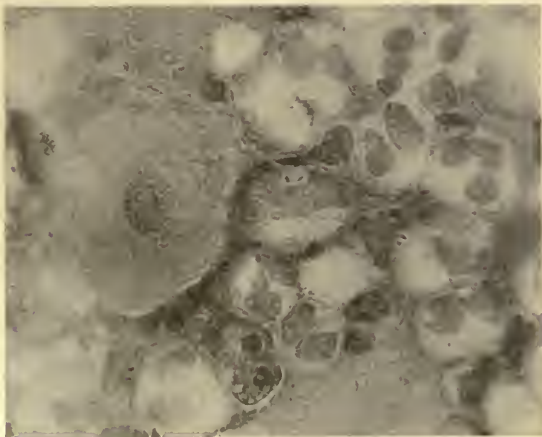


Fig. 3.—Photomicrograph of a representative field of specimen made of material removed from area marked II, Fig. 1. Shows pus, bacteria, and one endameba.



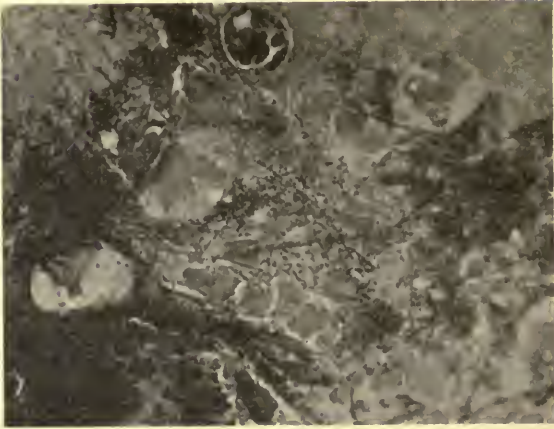


Fig. 4.—Photomicrograph of representative field of specimen made from material removed from area marked III, Fig. 1. Shows pus, bacteria, and one endameba.

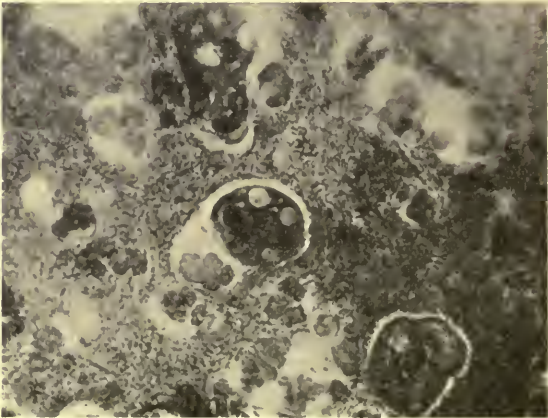


Fig. 5.—Photomicrograph of representative field of specimen made of material removed from area marked IV, Fig. 1. Shows pus, many bacteria, and three endamebæ.



bicidal action. The results are just as definite and striking if the drug is injected hypodermatically into some part of the body remote from the disease. When emetin is given hypodermatically, it is diluted so much by the blood before it reaches the seat of the disease until it has certainly lost

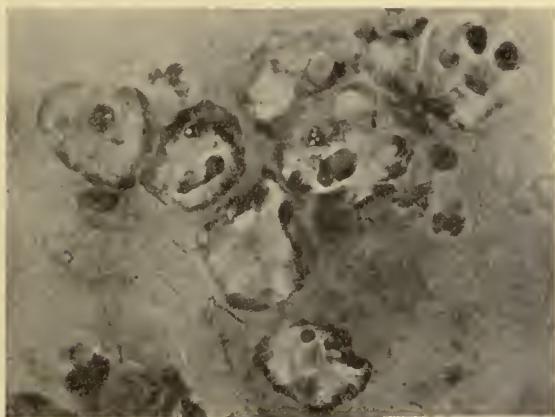


Fig. 6.—Photomicrograph of representative field of specimen made of material removed from area marked V, Fig. 1. Shows bacteria, very few pus-cells, and many endamebæ. This shows how much more numerous the endamebæ are in the very bottom of the lesion at the edge of the peridental membrane. Could there be greater evidence of their etiologic rôle?

all its possible antibacterial action, though not its specific amebicidal power. These favorable results are coincident with the disappearance of the endamebæ. The bacteria and other factors, such, for instance, as malapposition, thought by some to be the cause of the disease, are not affected.

The belief that endamebæ are the specific cause

of the disease is somewhat strengthened by the similarity of the lesion and the relation of the parasites to it as compared to another disease of man, amebic dysentery, caused by another species of endamebæ. The parasites are most numerous in the depth of the undermined ulcer of amebic dysentery (see Fig. 7), just as they are most numerous in the very depth of the pyorrhea lesion.



Fig. 7.—Drawing to illustrate amebic ulcer of the intestine. Note undermined edges. At the very extremity of the excavation the endamebæ are most numerous. Note similarity of the lesion to that of alveolodental pyorrhea in which also the endamebæ are most numerous and active in the very bottom of the lesion.

The hemorrhagic tendency during the early stage of the disease is also like amebic dysentery.

We fully appreciate the fact that Koch's postulates have not been satisfied yet. The endamebæ have not been isolated in pure culture, injected into another individual, thus producing the disease, and finally reisolated in pure culture. We would point out that this has not been done with other

diseases due to protozoa. In fact, it was impossible with malaria until recently. The malarial plasmodium was found in the blood of all cases of malaria—it was not present in the blood of normal individuals; it disappeared coincidentally with the disease, and therefore was well known to be the specific cause of malaria. Endamebæ are present in all lesions of all cases of pyorrhea: they are not present in normal gums. They disappear when the lesions get well, and the disease gets well when the endamebæ are killed by a specific amebicide. We have, therefore, fully as much evidence of specific etiology in this case as we had in malaria ever since the parasite was discovered, until very recently, when the parasites were first cultivated outside of the body, and inoculation experiments with pure culture made possible.

**Contributing Factors.**—Though endamebæ are the specific cause of pyorrhea, there are contributing factors, one or more of which are thought to be almost as essential as the endameba itself. However, these would be quite incapable of producing the disease in the absence of the endamebæ. Perhaps the most important of these is trauma or damage to the gum margin involving the peridental membrane. It is not at all probable that endamebæ would produce disease or could affect the normal or unbroken tissue. Damaged tissue

—favorable soil—must first be furnished before the infection can be established. Let it be understood that it would be quite possible for this favorable soil of damaged tissue to be too little for its existence to be recognized by the patient or by others. It might be microscopic in extent, and still be amply large to receive many endamebæ. The disease affects first the dental periosteum at the attachment of the gum to the tooth. It begins in the tissue between the teeth most frequently, and therefore those things most likely to damage the interdental peridental membrane would be the greatest source of harm. No doubt the use of the common toothpick is a very common source of producing the traumatic lesions favorable for inoculation of endamebæ. We are not at all certain but that the use of dental floss for the same purpose may be a fruitful source of harm in this same way. Unless one acquires unusual skill, it very often damages the interdental soft tissue, and bleeding is not uncommon. The bleeding always means damaged tissue. The gum attachment may also be damaged while chewing particles of food, etc.

Food-particles, especially hard substances forced between the teeth and resting, making pressure on the soft edge of the gum if allowed to remain for sufficient time, cause inflammation and ulceration

of the tissue. Such diseased tissue, usually in the form of a pocket, would furnish ideal soil for the inoculation of endamebæ.

We are inclined to think that tartar allowed to accumulate on the teeth by insufficient oral hygiene may also create favorable tissue for the establishment of endamebic infection. If not removed, it continues to increase until, at the gum margin, it may more or less close the little groove that normally exists between the tooth and the very edge of the gum. After such unnatural condition is established small particles of food that may get into this more or less closed groove, by their pressure and decomposition, cause inflammation of the tissue. Such inflamed tissue would furnish ideal soil for endamebic infection.

We are inclined to think that the improper vigorous brushing with a stiff brush may also damage the gums sufficiently to make favorable damaged tissue for infection by endamebæ. Brushing across the teeth instead of from gums toward the end would be most likely to do harm.

Crowns forced down into the gum are a source of chronic inflammation and finally formation of pockets favorable for endamebic infection. Almost all the crowns we have examined produce inflammation of the gum, usually evidenced by



redness, and, on microscopic examination, pus formation. We do not know whether crowns could successfully be used without their being made to exert pressure on the gum. We think that if they cannot, the advisability of their use is quite doubtful.

The bacterial flora, including spirochetæ, is also very probably a secondary factor in bringing about the local conditions necessary for infection with endamebæ and also in the progress and course of the disease. There is a great variety of bacteria in the mouth of most individuals, but they vary very much in variety and numbers with different individuals, being influenced largely by the care taken of the mouth and teeth. Naturally, one with the foulest mouth would be more likely to develop a favorable pocket for endamebic infection following trauma to the gums, than one who had a much smaller number and variety of bacteria with which the wound would promptly be infected.

Finally, different individuals show different degrees of resistance to infections of various sorts, and there is no reason to suppose that such would not be the case also in this infectious disease.

## SUMMARY

The specific cause of alveolodental pyorrhea is *endamebæ*, chiefly, if not altogether, of the species *buccalis*. They are present in all lesions, and they are not present in the absence of pyorrhea.

Secondary factors, one or more of which no doubt are essential, are trauma or wounding of the gums at the point of attachment to the teeth, lack of oral hygiene, and possibly individual resistance.

Damage to the gums producing favorable soil for infection may be done in a number of ways, among which are picking the teeth, cleaning with hard brushes, floss, rubbers, and the effect of hard particles of food, particles of food between the teeth making pressure on the gums, tartar on the teeth, ill-fitting crowns, etc.

## CHAPTER IV

### MORBID PROCESS

IN our opinion, it is of vital importance for all who would fully understand any particular phase of this disease—diagnosis, prophylaxis, or treatment—first to familiarize themselves with the morbid process. They should know something of the structures involved, their histology, functions, etc., and especially how such tissues break down or give way in the presence of the disease, as well as how they repair whenever the cause of the disease is removed. One who undertakes to diagnose the presence, or especially the absence, of the disease without knowing where and what the disease is, is likely to make many errors. One who looks for endamebæ without knowing something of the kind of lesion the disease produces and where the endamebæ are located is likely to come to the erroneous conclusion that endamebæ are not present in all cases of pyorrhæa, as we say they are. One who undertakes to treat the disease without having some idea of the disease process—how it is produced and how it is repaired, as well as the limits of nature's ability to repair the tissues



involved—is almost certain to be disappointed. It will be necessary for us to present a brief description of the structures involved, but only in so far as such description sheds light upon the disease itself. It should be understood that this is a disease of the peridental membrane, but, as a result of damage and final destruction of this tissue, other structures, the tooth and the alveolar bone, are involved also.

#### DESCRIPTION OF A TOOTH

A tooth consists chiefly of dentin, a very hard substance. The crown is covered with a harder substance, the enamel. The root has a layer, or several layers, of cementum covering it. The cementum serves to attach to the tooth the connective-tissue fibers of the peridental membrane which hold the tooth in place. Its development depends upon certain cells, cementoblasts, contained within the dental portion of the peridental membrane.

**The Alveolar Bone.**—The teeth are set deeply in sockets, or alveoli, in the maxillæ and mandible. The outer surface of the alveolar process is comparatively smooth and covered by normal periosteum. The alveoli, or sockets, into which the roots of the teeth fit, are bounded by a thin, definite bony wall which is pierced by a great many open-

ings. The surface is irregular. These cribriform plates unite the cortical plates of the bone at the

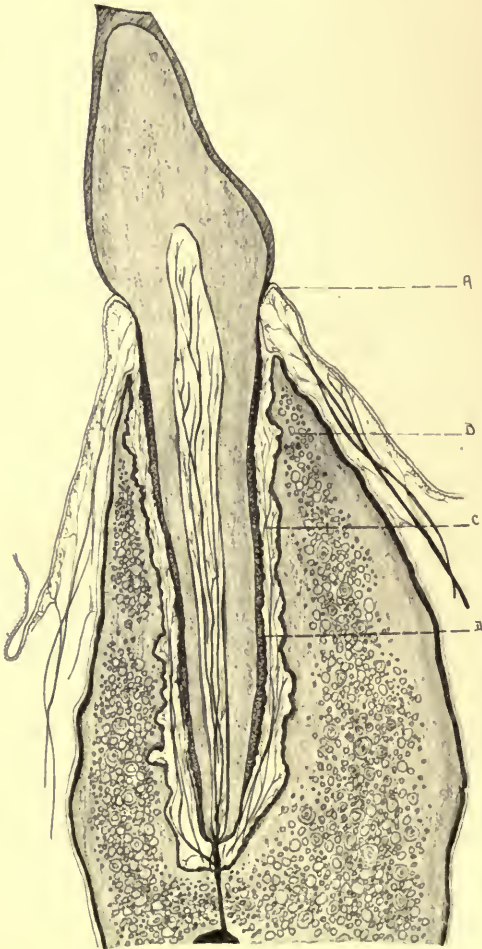


Fig. 8.—Drawing to illustrate the different structures involved in alveolodental pyorrhea: A, Gum margin; B, alveolar process; C, peridental membrane; D, cementum. The peridental membrane is well supplied with blood-vessels, and the alveolar wall is very irregular in outline.

border of the alveolar process, and are fused with it on their labial and lingual sides. They are really made up of a thin layer of subperidental bone, which has been built on the plates of cancellous bone to attach the fibers of the peridental membrane.

**Peridental Membrane.**—The peridental membrane is the soft tissue which fills the space between

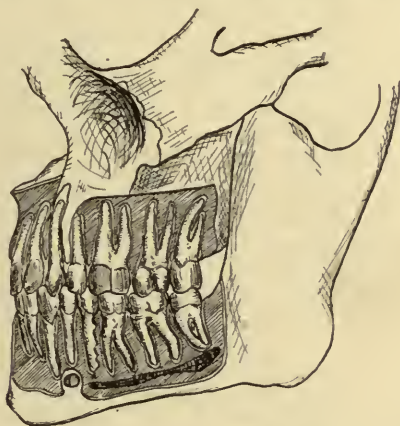


Fig. 9.—Drawing of a dissection to show the great depth to which the teeth are normally set in the bone. It necessarily requires a long time for a suppurating process to remove the structures shown surrounding the teeth.

the surface of the root of the tooth and the bony wall of its alveolus, surrounds the root occlusally from the border of the alveolus, and supports the gum. Its chief function is to support or maintain the tooth in proper relation to adjacent hard and soft tissues. It consists largely of strong connective-tissue fibers running in various directions

and at various angles from the root of the tooth to the wall of the alveolus, and from the root of the tooth into the gum. By fibers running from their attachments to the alveolar wall toward the apex of the tooth the tooth is suspended or swung

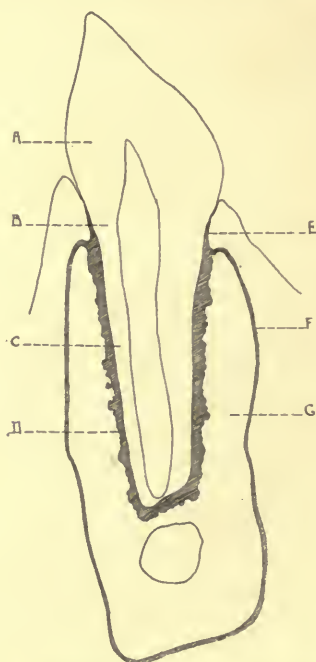


Fig. 10.—Drawing to illustrate the periodontal membrane and its continuity with the alveolar periosteum.

in its alveolus; by other fibers running in the opposite direction it is held down in its socket; by still others it is held from turning in its socket in one direction or another. Later we shall refer to this tough connective-tissue network as favorable

tissue for long-continued suppurating processes,

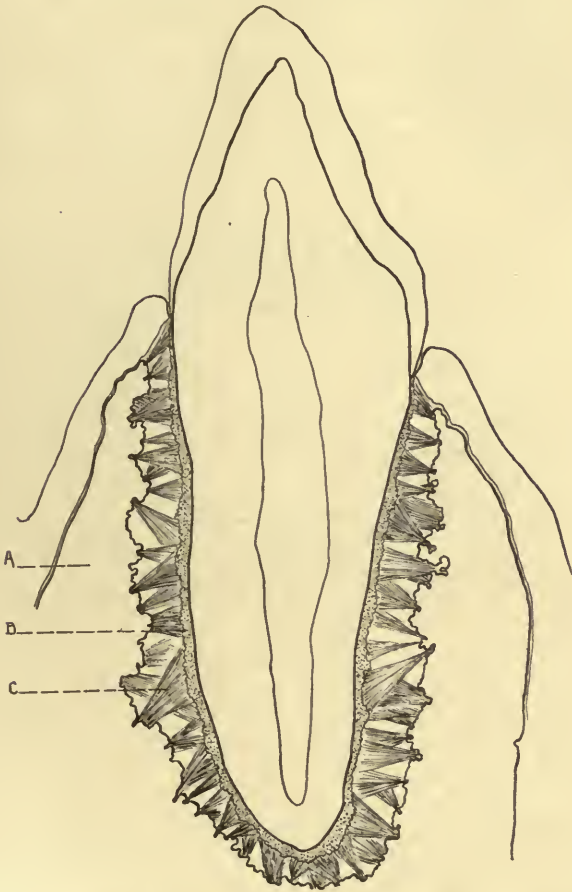


Fig. 11.—Drawing suggesting the manner in which the tooth is suspended and held in its socket by connective-tissue fibers running in many directions. A, Alveolar bone; B, cementum; C, fibers of periodontal membrane.

and especially unfavorable for or impossible of regeneration.



The peridental membrane also contains blood-vessels, nerves, and certain tissue-forming cells, osteoblasts on the alveolar side and cementoblasts on the side next to the root of the tooth. These are capable, under favorable conditions, of forming bone or cementum. In addition to these,

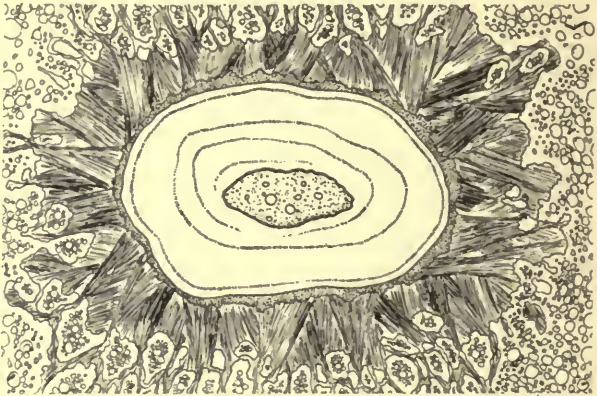


Fig. 12.—Cross-section of a young tooth showing how the fibers run in different directions, preventing the tooth from turning in its socket. Contraction of these fibers pulls the tooth to one side or twists it in its socket whenever the fibers producing the opposite action have been destroyed by the pyorrhea process.

osteoclasts are also present and capable, under favorable conditions, of removing bone or cementum. Under the age of maturity there are also some fibroblasts present, and probably capable, under favorable conditions, of making new connective-tissue fibers.

## ENDAMEBÆ THE SPECIFIC CAUSE

As is discussed more fully in the chapter on Etiology, endamebæ are the specific cause of alveolodental pyorrhea. The disease would not occur but for them. On the other hand, we do not believe it possible for endamebæ to attack successfully the normal tissue any more than tetanus bacilli, for instance, could attack the undamaged tissue. The wide distribution of these endamebæ (*buccalis*) will be fully discussed in the chapter on Prophylaxis. Suffice it to say here that the infection is practically universal. Practically all grown people have them in pyorrhea lesions in their mouths, and many younger people have them also. In fact, we think that almost all are infected before they are twenty years old. Any person who has pyorrhea is throwing off some endamebæ in the saliva all the time. All our associates, *including the immediate family*, are a source of infection, as well as many others with whom we do not come in so direct association.

These endamebæ have lived for countless generations in pyorrhea pockets around the teeth and some other locations. Their habitat has been, not normal tissue, but diseased tissue. Their food and other requirements have been supplied

in this particular kind of diseased tissue for countless generations. We know of no evidence that these parasites live in nature in any other kind of material. Normal tissue, even though it be the mouth and gums, would be very far from being the kind of material this parasite is accustomed to live in. There is every reason, therefore, to think that before *Endamœbæ buccalis* can establish themselves and produce their disease, the proper peridental tissue must be damaged so as to produce favorable soil and environment for them.

**Favorable Soil.**—Since the disease is practically one of peridental membrane only we may expect the first lesion or infection in any given person to be in this tissue. There are many ways in which damage to the peridental membrane may be produced. It is to be understood we are dealing with a very small microscopic parasite, no larger than many of the tissue-cells are. Therefore it is not necessary that the lesion produced should be large enough to be seen with the unaided eye, or to give rise to pain or in any way attract attention. In fact, the gum and peridental membrane are not very sensitive to pain. Considerable trauma to the gum may be produced without much pain being experienced.

No doubt the most common source of damage to the edge of the peridental membrane is hard



particles forced against it and efforts to remove particles of food that have lodged.

Normal teeth are "self cleaning," to the extent that food that passes between them gets loose as it gets nearer to the gum. Normally, the teeth come closer together (in fact, touch) near the grinding or cutting surface, but they are further apart and the space between them becomes wider toward the attachment of the gum and peridental membrane. As a result of this, food forced between them in biting and chewing comes out usually without any effort to remove it being necessary. In spite of this kind provision of nature, hard particles of food may occasionally be forced between the teeth in such a way as to damage the peridental membrane at once or later, as a result of the pressure continually made against the soft tissue. It does not require much pressure to produce inflammation and breaking down of soft tissue anywhere, especially if aided by such a bacterial flora as is always to be found in the mouth.

Efforts to remove food that may have lodged between the teeth, by means of hard instruments, such as toothpicks, usually damage the interdental soft tissue. How often does one pick the teeth without breaking the continuity of tissue at some place or other? Often bleeding is produced and

the blood is seen or tasted. Many other times the damage occurs, but is not sufficiently great to attract attention at all. In fact, even whenever enough trauma is produced to cause noticeable quantities of blood to flow, there is little or no pain and the individual does not realize at all that any harm has been done.

The brunt of mastication of food is borne by the first molar and the teeth just in front and just behind it. Damage is most likely to occur here, and as a result pyorrhea generally begins around one of these teeth. These teeth are also generally lost first. The examination of a number of mouths will bear out this statement. It is not proposed for a single moment that the disease may not begin around any other tooth. It does do it in many instances.

If the peridental membrane is torn loose from the tooth, no doubt it would heal back in a very short time if left alone and given an opportunity. Healing, however, is likely to be interfered with or prevented by food that is driven into the space at each meal. Small particles remain and act like a foreign body, not only irritating and preventing healing directly, but they favor the growth and harmful action of bacteria which are always present in the mouth. Such a lesion containing food-particles and bacteria will have some pus-

cells in it within twenty-four hours, and if healing is prevented by repeatedly forcing food into it, it soon becomes ideal soil for an *Endamebæa buccalis*, if one should chance to be planted there. Of course, as long as an individual has no focus of infection in his own mouth as a source of endamebæ and must depend, therefore, upon outside sources, the chances of a particular traumatic lesion getting infected are not great. If such a wound does not get infected with endamebæ, it heals in a few days, as soon as nature's reacting and healing powers have time to accomplish this result. The bacteria and irritation by food are not sufficient to prevent healing in a few days, any more than they would prevent healing of other lesions involving other tissues in the mouth.

#### **The First Lesion a Source of Infection to Others.**

—Let us suppose that, on account of the character, size, or location of the lesion or some other influence a lesion or little pocket by the side of a tooth did not heal promptly, and did get infected with endamebæ. These endamebæ multiply rapidly and crawl about from place to place, by their ameboid movement burrowing about among the granulations and inflamed tissue of the lesion, and actually penetrate the diseased tissue. It is quite probable that they are in search of some particular

kind of cell or product of the inflammatory process for their food.

Pus from various sources, including that from pyorrhea lesions, contains certain cells or portions of cells which usually contain a very dark-staining nucleus. In fact, such bodies may be found in most any pus from chronic inflammatory lesions. These same bodies are found quite regularly in *Endamœba buccalis*. Frequently one endameba may contain as many as eight or ten of them. They appear to be the food of the endamebæ. These bodies are comparatively few in number, and are mixed or distributed among the pus-cells. In order for an endameba to secure them it would be necessary for the parasite to move about in the material containing them—to forage, as it were, for them. Endamebæ thus move about back and forth over the ulcerated surface and among the granulations of the lesion. It is not known whether they damage directly or attack the living tissue, but it is not at all necessary for them to do so for them to be pathogenic. They may produce great harm and actually be the essential cause of the pyorrhea by continually carrying and planting and replanting harmful bacteria in the tissue.

Though endamebæ are small, microscopic objects, the largest of them are many hundred times larger than the small bacteria of different kinds

present also in the lesion. These bacteria are not capable of preventing healing themselves, because the resisting powers of the tissues and body fluids keep them walled off or pushed back out of the living tissue. The endamebæ, however, carry them in and out of the granulation tissue as they move about in it. Bacteria can be seen on and

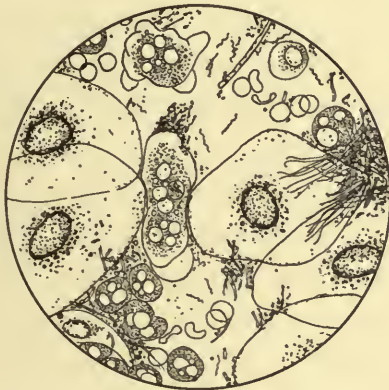


Fig. 13.—Drawing of actual observation of *Endamebæ buccalis* passing through a narrow place between cells. Note mass of bacteria being drawn with it. In this manner they carry and plant bacteria in the granulation tissue of the lesion.

in moving endamebæ by watching them under the microscope. One can see endamebæ transport bacteria from one place to another in the preparation, and it would seem reasonable to suppose that they do the same thing in the diseased tissue.

Bacteria that are being continually carried and planted into the diseased tissue could thus do



much more damage than if they were not aided. One effect they surely have is to cause the production of pus if they are pyogenic bacteria. It would seldom occur that such a lesion would not have some pyogenic species. No doubt the kind of secondary bacteria present influences the amount of pus produced in a given case, and to a great extent the course of the disease.

In addition to bacteria, there are also different species of spirochetes present in the mouth, and these are often very numerous in pyorrhea lesions. There is good reason to believe that they may also be important secondary factors in most cases.

We do not know of any conclusive evidence that the endamebæ do or do not directly damage or break down tissue. It is quite possible that they could not produce pyorrhea unaided by their symbiotic bacteria and spirochetes, any more than the bacteria and spirochetes can produce the disease unaided by the endamebæ. There are many instances of such obligatory symbiosis in nature. This is a subject for future investigation.

After one such focus of infection is established, from which endamebæ are being thrown off into the mouth all the time, a favorable lesion produced by subsequent damage to the peridental membrane around other teeth is much more likely to become infected. The larger the number of infected

lesions, the more probable it becomes that whenever other lesions are produced they will get infected. It is here seen how important it is to prevent the first infection, which is almost certain to occur during childhood. Unfortunately, our former knowledge of the disease only led to interest and concern in the disease in older people after they began to lose their teeth or were about to do so. Now that we know the specific cause of the disease, that it is contagious, and, fortunately, how to prevent the establishment of the infection, there is no doubt that dissemination of this knowledge will lead to the prevention of this unnecessary veritable scourge of man, to a great extent.

The importance of preventing the primary infection cannot be emphasized too forcibly. One who has no endamebæ in his mouth would not be in much danger of getting wounds, that chance to be produced, infected with endamebæ. It must be quite an accident for the first endameba to get planted or lodged in a wound in the edge of the peridental membrane. The number introduced into the mouth from without must be comparatively small. Sooner or later, however, the accident happens. After the endamebæ are established in one pyorrhea lesion there is a constant overflow of them into the mouth. They can be identified in the saliva of advanced cases with



ease. By first diluting the material and then centrifuging, we recovered 11 endamebæ from a single mouthful of "spit" in one instance. Not only is such a person likely to be a source of infection to others, but he is a fruitful source of infection to himself, or rather of spreading infection in his own mouth.

**The Course of the Disease in a Given Lesion.**—Whenever the disease is established, its course and progress vary considerably, dependent upon many variable factors. The lesion at first consists of a small pocket by the side of the tooth. The tooth from which the peridental membrane was torn makes one side of the pocket, while the rest of its wall consists of inflamed granulation or ulcerated tissue—an ulcer held against the tooth. One side, however, is open and permits the outflow of pus. Depending upon the location of the lesion, it will be more or less subject to food being forced into it. Food-particles are a source of increased inflammation and bacterial growth, and no doubt they contribute to the progress of the disease in many instances. Usually, however, many months or even years pass before the disease has destroyed the peridental membrane as far as the edge of the alveolar bone. The pocket gets deeper and wider, and more and more pus is produced because of the increased ulcerated surface.

The gum is red and inflamed whenever the lesion is on the labial or lingual side of the tooth in most



Fig. 14.—Drawing to illustrate an early pyorrhea lesion by the side of a tooth. The disease has just reached the level of the alveolar process, which is almost exposed.

instances, but in others casual observation hardly detects that it is abnormal. The disease begins much more frequently between the teeth, but the

interdental soft tissue is not so likely to appear red and inflamed. In fact, it is only by careful exam-



Fig. 15 —Drawing to illustrate a more advanced stage of pyorrhea by the side of a tooth. The periodontal membrane has been destroyed for a considerable distance down the alveolus. The bone has begun to be removed, and the gum to retract as the bone gives way. Whenever the progress of the disease is rapid, the alveolar bone is laid bare.

ination with a suitable instrument that some of the small lesions between the teeth are found.

One is in doubt often until he examines the material removed from a suspected lesion microscopically and finds pus-cells and endamebæ.

During this early stage the gum bleeds easily from slight trauma, such as picking the teeth, brushing them, sucking air between them, etc. The cause of this tendency to bleed readily is the granulation surface or ulcer. Granulations in other tissues of the body bleed from slight trauma in the same way. The lesion probably seldom ever entirely encircles the tooth-root at this stage, though this sooner or later occurs in most instances. We have seen instances, however, where the disease extended to the very end of the root on the side next to another diseased tooth, while the opposite was well and the interdental soft tissue intact and normal.

Up to this time there is little or no pain produced. The tissue involved is not very sensitive to pain. There may be considerable discomfort and uneasy feeling, especially following a meal, produced by particles of food forced into the pocket (or, as is usually the case, pockets) or held against the diseased tissue between the teeth. This leads to picking the teeth or effort to remove food in other ways. Many have been educated up to passing a cord or dental floss between the teeth for this purpose. This is probably fully as

harmful as picking the teeth, because it is seldom done without traumatizing the interdental soft tissue.

After the disease has existed long enough, there begins to be more or less retraction of the gum, leaving exposed dentin which is often sensitive to changes of temperature and physical force. Holding hot or cold water about the teeth or forcing it between them gives rise to pain. This may be noticed for months or years before the patient realizes that pyorrhea exists.

Though the disease begins around a single tooth, it is unusual to find people over twenty years old who haven't more than one tooth involved. We have seen patients in whom we could demonstrate lesions, pus, and endamebæ around every tooth in their mouth. It is common to find the disease around at least four, six, eight, or ten teeth in the same individual. Of course, the lesions are of different shape and extent around different teeth.

Peridental membrane, being composed chiefly of tough fibers, is destroyed slowly by suppurative processes. Finally, however, the pocket reaches the level of the alveolar bone. The destructive process continues in the peridental membrane now between the tooth and the wall of the alveolus. As it progresses the peridental membrane is gradually sloughed or eaten away.

In case of a rapidly progressing disease the pocket



Fig. 16.—Drawing to illustrate the last stage of a pyorrhea lesion by the side of a tooth. The periodontal membrane has been entirely destroyed, except a small portion in the very bottom of the socket (not shown here) which is still holding the tooth in place. There has been great destruction of alveolar bone and consequent retraction of the gum. The rapid destruction results in considerable exposure of the bone.

now has bare tooth-root on one side and bare alveolar bone on the other, with ulcerated gum



at its edge and the granulating, ulcerated edge of peridental membrane in the bottom. Figs. 14,

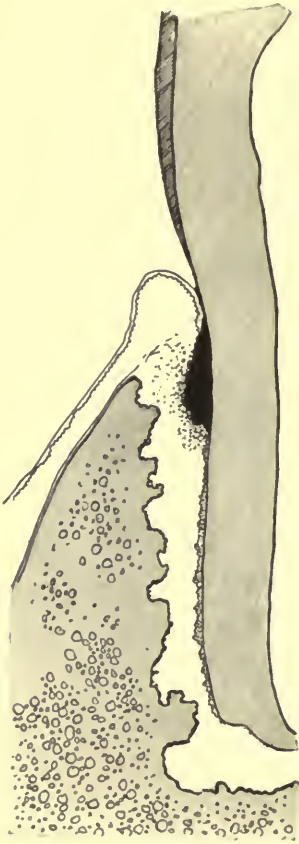


Fig. 17.—Early stage of slowly progressive disease. The bone is not laid bare at any time.



Fig. 18.—Drawing to illustrate more advanced stage of slowly progressing disease. The bone has been absorbed as the disease progressed.

15, and 16 illustrate such lesions. In the usual slowly progressing lesion, however, the bone is



absorbed as the lesion extends and is not exposed or denuded of all soft tissue. Figs. 17, 18, and 19 represent such lesions. Large numbers of endamebæ are demonstrable on and in this latter granulating tissue. The pocket contains food particles that have been forced into it, myriads of bacteria and spirochetes, much pus, and many endamebæ. The latter, however, are much more numerous in the very bottom of the lesion, and this point should be kept in mind when examining for them.

The decomposing contents of the pocket necessarily have a bad odor. This varies with the bacterial flora present, some giving rise to different and stronger odors than others. Certain spirochetes contribute largely to the odor of the contents of pyorrhea pockets. During the hours when one is awake and the mouth is being worked, the material does not accumulate so much, and therefore the decomposition is not so great. During sleep and quiet pus accumulates, and the decomposition with resultant odor is greater. It is sufficient to give rise to a foul breath and often a bad taste in the mouth. The bad taste in the morning is largely due to this material. The thought would distress many people if they knew how much pus, and what, they were swallowing each day—and up to now there has been no escape

from it. Some people lose their teeth earlier than others, but *all* lose them from this suppurating process sooner or later. Some happen to have

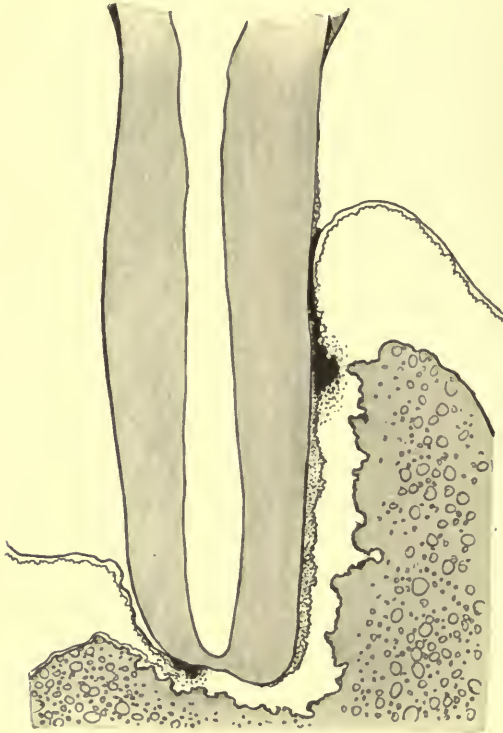


Fig. 19.—Drawing to illustrate still more advanced or final stage of slowly progressing disease. Periodontal membrane destroyed to the very apex of the root and alveolar bone entirely gone on one side. Such a tooth is loose and usually falls out of normal position.

different bacterial flora in their mouths or they have different spirochetes, and therefore their breath is worse than that of others, or they have

more pus than others, but it is a question of degree only. It cannot be said that foul breath is due to pyorrhea altogether, but it is practically so. If everybody's breath smelled to himself like it does to the other fellow, and if it was known by all that the odor results chiefly from pyorrhea, it is doubtful whether there would be enough ipecac produced in South America and India combined to supply the demand!

The destructive process continues, and finally the peridental membrane that remains is insufficient to hold the tooth rigid in the socket. It gets loose and can be moved from side to side with little force. It continues to get looser and looser during several months or years until it finally falls out, or, as is usually the case, it is removed artificially on account of pain or worry that it may cause. Sometimes a tooth will hang on for several years after it has gotten loose.

The socket enlarges as a result of slow absorption of the alveolar bone, which is denuded of peridental membrane. It is constantly bathed in pus, but it is doubtful whether it is removed as rapidly as by nature when a tooth that has not been previously diseased with pyorrhea is extracted. In the former instance it usually requires many months for complete absorption of the alveolar process. In pyorrhea the removal of the alveolar

process goes on during the time the tooth remains after the bone begins to be stripped of its peridental membrane. Frequently the removal of bone has been very great before the tooth is finally lost (Fig. 19). As the bone is removed the gum retracts, and frequently half the length of the root,



Fig. 20.—Photograph of postmortem specimen showing great retraction of gum, with consequent exposure of roots of teeth, in a case of pyorrhea of long duration. Some people's teeth hold fast much longer than others do in the presence of the disease.

or even more, stands above the level of the gum before the tooth finally comes out (Figs. 20 and 21).

Sometimes the disease results in more or less destruction of the periosteum covering the edge of the alveolar bone, in which instance there may be considerable caries. This leads to thickening of

the gum over the edge of the bone. Where there is much carious bone, the healing process follow-



Fig. 21.—The lingual side of the same specimen shown in Fig. 20.



Fig. 22.—Skiagraph of normal teeth showing the interdental alveolar bone (Courtesy of Dr. C. Edmond Kells).

ing the falling of a tooth is very slow. The gum over it remains thick for many months. Where

there is little caries, the disease stops and the socket soon heals up. We do not know what becomes of the endamebæ, but it is probable they



Fig. 23.—Skiagraph showing disappearance of alveolar process. Note how little support the teeth have. This lost bone cannot be regrown. The teeth now lean to one side for lack of support.



Fig. 24.—Skiagraph showing loss of alveolar bone from pyorrhea.

disappear from the particular socket because there is no periodontal membrane left for them to live in. This is their natural habitat.



**Amount of Blood and Pus Lost During the Course of the Disease.**—It is interesting to speculate on the amount of blood and pus lost during the course of the disease. If one sucks or picks or brushes the tooth during the early stage of the disease, a drop or two of blood oozes out in many instances. This is noticed by the patient, but perhaps it is done only once or twice a day. Some people get much larger quantities from their teeth. In fact, some bleed without any trauma at all. True, others have little bleeding. There is a very much larger amount of blood caused to flow by chewing food. It is mixed with the food and not noticed. One who has bleeding gums makes them bleed whenever he masticates food until they “bleed out.” This is repeated at every meal. Many people waste one to several drams of blood in this way every day for weeks, months, and years. Though the loss of such a small quantity of blood for a short time only could hardly be considered likely to be very harmful, it is quite probable that, kept up for a sufficient length of time, it may be a source of much harm. Only three drams a day for a year would be about a gallon, and it is not at all improbable that the loss of a gallon of blood each year would be harmful.

As for the pus, one can obtain one or two drops



from a pyorrhea pocket by massaging it, and after five or six hours a similar amount can be obtained. It is not a high estimate to say that at least four drops of pus can be secured from the average pyorrhea pocket during twenty-four hours. When mixed with the saliva in driblets and swallowed, it is not noticed, however. This would be an ounce in one hundred and twenty days, or three ounces in a year. We believe it would be a very conservative estimate to say that the average tooth suppurates more than ten years before it is finally removed by pyorrhea. Thus about one quart of pus would be produced for each tooth, and for all 32 teeth, about eight gallons. The disgusting thought about it is that all this eight gallons of pus is produced in the mouth and that most of it is swallowed. It would be no great surprise if the production and loss of this large amount of pus should have marked harmful influence on the health and perhaps the longevity of the individual.

That this disease may influence the longevity of individuals by the loss of their teeth and the resulting failure to masticate and digest their food well can hardly be doubted. In fact, it will be conceded. This, coupled with the probable harmful influence of the loss of blood and pus, serves to indicate that the disease now under discussion is of vast importance to all humanity. Think what

it would mean in comfort, health, pleasure, and longevity to man if this veritable scourge could be blotted out! What a different people we would have if all retained and enjoyed their teeth as long as they lived! At present the average person begins to get along without a part of his teeth or substitutes artificial ones by the time half of his life is spent. During the remaining half of life this disease, consisting of a filthy, sloughing process in the mouth, slowly but surely removes the remaining teeth, which the better-to-do may substitute with artificial teeth,—a poor makeshift,—but the majority have to “gum it” as long as they live. Think of the pleasure lost by being unable to masticate and enjoy the food for a large portion of life, all due to this now preventable and curable disease!

#### SALIVARY CALCULUS

What is called salivary calculus and thought to be due to the precipitation of calcareous material from the saliva is in fact due to pyorrhea. It is now generally thought to be the cause of pyorrhea, in some cases at least, but we feel certain that it will be found to be the result, rather than the cause, of pyorrhea. We have not had the opportunity up to this time to investigate the subject extensively, and the literature on the subject that

has been available to us is worse than useless. Many explanations of the cause of the condition are absurd.

Pus discharging from a pyorrhea lesion (even a small one) flows along by the side of the tooth, and especially in the ditch between two teeth. It is held and made to flow against the tooth more completely in certain locations by the lips, tongue, or cheek than in others, but it is sufficient in some cases to insure calculus formation around any tooth involved. As the pus mixes with the saliva in certain individuals calcium salts are thrown out of solution and are deposited on the tooth. This continues to accumulate until, finally, a pretty heavy deposit of yellowish calculus is formed on the tooth or teeth. It is heaviest just above the edge of the gum, because more pus passes over that location than further from the lesion. As the discharge passes further from the lesion it has lost a part of the precipitable matter and it is diluted more. Therefore the calculus formed is thinner the further from its source we go. On account of the fact that the pus is caused to flow along the channel where two teeth stand side by side, in greatest quantity, the calculus is thicker on the edges of the teeth, as a rule.

## SERUMAL CALCULUS

Not only may certain salts in pus of certain individuals be thrown out of solution and thus form calculi on the enamel of the teeth, but certain substances down in the pyorrhea pocket (bacteria and their products) in certain individuals cause precipitation and formation of calculi on the root of the tooth and deep in the lesion. This is "serumal" calculus, and is probably a source of considerable irritation in pyorrhea lesions wherever it exists.

## CHANGE OF POSITION OF THE TEETH FROM PYORRHEA

One of the things formerly thought to be the cause of pyorrhea is malocclusion. Many an unfortunate victim has had his or her teeth ground off by the dentist as a remedy for pyorrhea, exposing sensitive dentin for the balance of the life of the tooth. Instead of malocclusion being the cause of pyorrhea, pyorrhea is the cause of much of the malocclusion seen.

The teeth are held in place by tough fibers which run from the wall of the alveolus to the cementum of the tooth. Whenever these fibers, the periodontal membrane, are destroyed on one side of a tooth and remain intact on the other side, the tooth is drawn to the side on which the peri-

dental membrane is still living. Sometimes the peridental membrane is destroyed in such a way that the tooth is twisted in its socket by the opposite fibers. Teeth are sometimes turned almost one-fourth around in this way. It often occurs that the incisors pitch forward because the peridental membrane is destroyed on the lingual side. Strain on the teeth, such as biting them together or biting food, gradually increases the tilting. Tilting is also encouraged or favored by the swelling of the inflamed tissue on the diseased side.

The fibers of the peridental membrane are directed both apically and occlusally from their attachment to the alveolar wall. The tooth is held down in its socket by those directed occlusally, most of which are located at the border of the alveolar process. Whenever the peridental membrane all around a tooth is destroyed to below the original border of the alveolar process, the chief fibers that hold it down are gone. The swelling of the soft tissue produced by the inflammation as the disease progresses now tends to crowd or force the tooth from the socket. If the tooth strikes its opposite fellow, it is held down in the socket, but if it does not rest against another tooth, it gradually rises out of the socket and stands higher than the other teeth. It may be remarked here that such a tooth can be forced back in place and

retained by proper mechanical means after the disease is stopped.

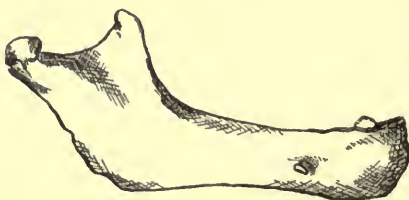
#### LOSS OF THE ALVEOLAR PROCESS

Whenever teeth are lost from any cause the alveolar process is absorbed or removed in the course of several months or years, leaving the maxillæ narrow and quite different in appearance from their appearance before the teeth were removed (Fig. 25).

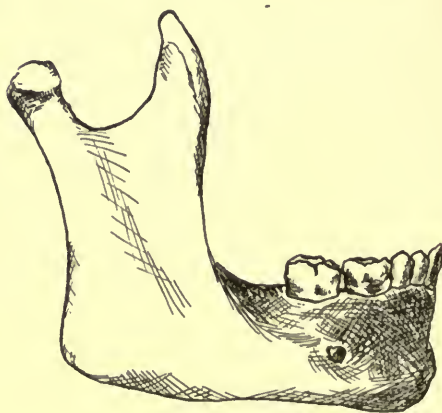
**Pyorrhæa as a Source of Other Systemic Diseases—Septicæmia, Endocarditis, Rheumatism.**—The opinion is held by quite a good portion of the medical profession that pyorrhæa is the source of various systemic diseases. We are not prepared to express an opinion one way or the other. It must be confessed that it is not proved to be so, however much we may believe it. The fact that a patient who is suffering with one of these systemic diseases also has pyorrhæa is not sufficient evidence that there exists any etiologic relation whatever between them, because practically all other grown people who have not such systemic disease have pyorrhæa also. On the other hand, it would seem that the chances of pathogenic bacteria reaching the blood through pyorrhæa lesions are great. Most pyorrhæa pockets contain one or more species of pathogenic bacteria, staphylo-



cocci, streptococci, pneumococci, diplococci, etc. The endamebæ are moving about among the bacteria and carrying them into the granulation tissue among the granulations. They plant and replant the bacteria where they are likely to gain entrance into the blood-stream. It will be remembered that granulations consist of loops of blood-vessels, as it were, with very thin, permeable walls. This phase of the subject is one for future investigation.



Infancy

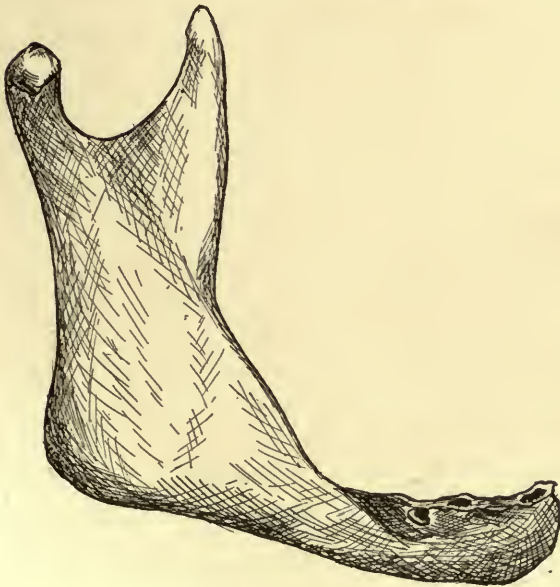


Childhood





Maturity



Senility

Fig. 25.—Similar pictures to these are common in text-books on anatomy and physiology, which show the usual changes in the mandible produced by age. The loss of teeth and disappearance of the alveolar process are looked upon as physiologic processes because they occur in practically all people if they live long enough. Instead of being a physiologic change, it is due to alveolodental pyorrhea, and in the light of our present knowledge is preventable.

## SUMMARY

A tooth is held in its bony socket by a tough peridental membrane which consists largely of fibers attached to the alveolar wall and to the cementum of the tooth.

Alveolodental pyorrhœa begins in a lesion produced by damage to the edge (tearing loose) of the peridental membrane. Such lesions may be prevented from healing until they chance to become infected by endamebæ, by food particles forced into them from time to time.

Once endamebæ are inoculated into such a lesion, they prevent healing and cause the lesion to extend slowly but surely. The peridental membrane is slowly destroyed, leaving the alveolar wall and tooth bare. Large quantities of pus are formed.

As the peridental membrane attaching the alveolar wall to the tooth is destroyed, the bone, which no longer can perform the function for which it was intended,—supporting the tooth,—is slowly absorbed or removed.

It is not known whether the endamebæ damage or attack the tissue directly. They probably do their damage by crawling about among the granulation tissue in search of certain nuclear bodies or cells for food, and incidentally planting and re-planting bacteria among the granulations. The bac-

teria could not invade the tissue unaided by the endamebæ. Different bacterial flora in the mouth of different individuals is one of the influences which leads to great variation in the character and progress of the disease in different individuals.

Whenever the destructive process has advanced far enough and the supporting structures have been destroyed sufficiently, the tooth gets loose in its socket. Finally, whenever there no longer remains sufficient tissue to support it, it either falls out or is removed.

One lesion infected is a constant source of infection to any other favorable soil that may be prepared by damage or trauma that may occur to the periodontal membrane. Sooner or later the supporting structures of all the teeth are involved. After months and years of suppuration one tooth after another is lost, until finally all are gone.

The amount of blood lost during the active early stage of the disease is estimated to be at least a gallon a year. This may be harmful. The amount of pus produced during the time required for all the teeth to be removed by this disease is estimated to be at least eight gallons. This may also have an influence on the health and possibly on the longevity of the individual.

It is also quite probable that pyorrhea lesions may be the portal of entry of pathogenic bacteria into the blood-stream as well as bacterial poisons.

## CHAPTER V

### CONTAGIOUSNESS

BEFORE we knew the specific cause of alveolo-dental pyorrhea and how to diagnose the disease certainly in all stages, it was impossible to know whether or not it was contagious. The length of time necessary for the disease to develop to a stage where it is recognized by the patient and likely to be diagnosed by the dentist or physician is so great that, naturally, the source of infection is never thought of. No doubt in most all cases several years intervene—in certain cases even twenty or more years—between the date of primary infection and recognition of the disease.

Another factor which would lead to difficulty in determining the contagiousness of the disease is its many sources. Practically all adults have the infection, and may, therefore, be sources of infection to others. In the presence of such a condition it would be impossible to trace the infection in a given case to its source. It is quite true that many have thought the disease contagious or infectious in some way. The common observation of its much greater prevalence in certain

families than in others is an indication, but not proof, of contagiousness or infectiousness. It resembles other contagious diseases—measles, smallpox, tuberculosis, etc.—in this respect. In regard to any disease due to specific infection there is greater tendency for it to occur in households and communities in which other cases exist or have existed.

Now that we know the specific causative agent in pyorrhœa, *Endamœba buccalis*, we can understand the greater prevalence in certain families and groups of people who are closely associated. Let us suppose a husband has the disease. Close association with his wife, eating and drinking after each other, kissing, etc., expose her especially to the infection, which she sooner or later surely gets. Finally, whenever their children grow up and get old enough to become infected, they could hardly escape on account of the constancy and favorability of the source of infection to which they are exposed. A child might be taught early not to eat or drink after others than their immediate families, but in what home is the individual drinking-cup to be found? Where the father and mother are in the most advanced and infectious stage, nothing short of individual drinking-cups and other precautions could promise to protect the children who are growing up. The common

drinking-cup in public places, stores and shops, parks, factories, etc., must be a great source of infection. Such a cup must have endamebæ on it almost all the time.

We are often asked whether we think the disease is "catching." To this we reply there is not the slightest known evidence that *Endamœba buccalis* lives and reproduces anywhere in the world except in the mouth and adjacent structures of man and perhaps certain animals, and since this parasite is the essential cause of pyorrhea, it is caught from some other person in every case. Everybody who has pyorrhea got it from somebody else, usually, if not always, by putting more or less saliva and pus from the other person's mouth into his own mouth. It is not necessary that the quantity be large enough to be seen on ordinary observation to be sufficient to infect. Microscopic quantities may contain many endamebæ. One should be sufficient to start the disease if planted in favorable soil.

The spread of pyorrhea would soon be checked materially if the facts that we now know about the disease could be brought clearly to the attention of the public. If the real facts were known, the pernicious habits of drinking and eating after each other, putting things of unknown cleanliness



in the mouth, kissing in the mouth, etc., would be rapidly reduced, at least.

Another factor that may some day contribute to reduction in the incidence of the disease is the cure of cases or reduction in the number of cases. Every case cured is just one less source of infection. Not only that, such an individual will usually be a kind of educator for others, for no one is likely to be cured and remain cured without learning, during his treatment, some of the fundamental lessons about the disease. Now that the disease is curable and its return preventable, there should be an ever-increasing number of cured persons. In addition, other younger people will avoid infection, and in time the proportion of the population of the community who have the disease should get less than it is at present.

#### SUMMARY

*Endamœba buccalis*, the specific cause of pyorrhœa, is not known to live and reproduce anywhere in the world except in the mouth and adjacent structures of man. It is "caught," either directly or indirectly, from another person who has pyorrhœa by putting more or less saliva and pus-containing *endamebæ* from the infected person's mouth into the mouth of the one who becomes infected.

## CHAPTER VI

### SYMPTOMATOLOGY

THE symptoms of alveolodental pyorrhea vary with the stage of the disease, the bacterial flora in the mouth, including probably the spirochetes, the oral hygiene practised by the patient, the teeth involved, and individual influences, as well perhaps as other factors that we do not understand at this time. It should be clearly understood that the disease is one of many years' duration, and that different teeth are usually found to be in different stages at the same time. In the same patient one or more teeth may be found very loose and in the very last stage; one or more others may be in an advanced stage, but still firmly held in their sockets; one or more others may be in a less advanced stage, but have the disease well established; one or more others may be in the earliest stage, in which careful microscopic examination may be required to demonstrate the pus and endamebæ which can be obtained by carefully removing material from beneath the collar of gum, especially that between the teeth; and still one or more other teeth and their

peridental membrane may be perfectly normal all in the same mouth.

#### BLEEDING FROM GUMS THE EARLIEST SYMPTOM

Bleeding from the gums is probably the earliest symptom recognized by the patient. This comes on so insidiously and is supposed to be such a harmless thing that little attention is paid to it. It occurs in almost all cases, but is very variable in amount with different individuals. Picking the teeth may be followed by the flow of a little blood. The patient thinks he wounded the gum and pays it little attention. Sucking the teeth may cause a little bleeding of the gum around one or more teeth. Brushing the teeth may be followed by the flow of a little blood. Perhaps only enough blood to color the first spit or two of saliva is all that is noticed. In other instances when early lesions exist several drops of blood may be expectorated. We have seen a few patients whose gums would occasionally break loose and bleed apparently without any provocation. The blood comes from the granulation tissue which makes one side of the lesion or pocket. Granulation tissue generally bleeds freely upon the slightest manipulation. The tissue that bleeds is chiefly between the teeth. Chewing food causes bleeding when one has pyorrhea, especially in the early

stage, but the blood is promptly diluted with the food and not tasted. The quantity of blood that oozes from the gums and is swallowed during a meal in some cases must be considerable—several drops, at least, in many cases. There is less bleeding in far-advanced cases, as a rule.

#### SORENESS OF THE GUMS AND OF THE TEETH ON PRESSURE

There is often a slight, sometimes considerable, soreness or unnatural feeling of the diseased gum from time to time, but the patient usually mistakes it for a little something that has lodged between the teeth and is pressing against the gum, or a little injury that has occurred in some way. In fact, the slight unnatural feeling is not seriously thought of by the patient, and usually passes away in a day or two, to recur in a few days. After the disease advances far enough, pressure on the tooth, as by biting the teeth together or eating, elicits soreness. Sometimes this is considerable and gives rise to more or less continued discomfort for days or weeks at a time. Probably this is brought about by food-particles being carried into the lesions. Other patients suffer very little pain or discomfort during the many years the disease lasts.

## BAD TASTE IN THE MOUTH

During the early stage of the disease, if only one or two teeth are involved, perhaps there is not enough pus formed or decomposition in the pockets to be very noticeable to the patient. As the disease advances more pus is produced, there is more dead or dying tissue in the lesion, and there is more decomposition of food and pus in the larger pockets. This gives rise to a bad taste in the mouth, especially after the patient has been asleep and quiet for a few hours. During the waking hours the pus and decomposing material are swallowed or spit out every few minutes and therefore not allowed to accumulate. On the other hand, during sleep they accumulate and upon waking they are in sufficient quantity to give rise to a bad taste in the mouth.

## FOUL BREATH

This pus and sloughing, decomposing material usually have a very bad odor. At first it is so little as to be unnoticed, except perhaps after several hours of sleep and quiet. As the quantity gets larger an offensive breath is present, noticeable at all times to others, and after a little accumulation, to the patient himself. The odor, no doubt, is produced in most instances by spirochetes in the

lesion, but in some the characteristic fecal odor strongly suggests that produced by the colon bacillus. In other cases the odor is the sickening, raw flesh odor produced no doubt by profuse flow of pus.

#### PYORRHEA

Pyorrhœa is present during all stages of the disease, but the quantity of pus is not large enough to be recognized macroscopically until it has existed for a considerable length of time. The amount of pus produced by lesions of equal size in different people varies considerably. Perhaps the secondary pyogenic infection and individual idiosyncrasy are largely the cause. When only the posterior teeth are involved, the patient does not notice the pyorrhea. Whenever the front teeth are sufficiently diseased, pus may be noticed oozing from around the teeth. Properly applied pressure squeezes out sometimes a drop or more of thick, creamy pus. We have seen many patients who had learned to massage the pus out of their pyorrhea pockets. Pressing against the end of the tooth often will cause pus to appear at the edge of the pocket. So also will pushing the tooth from side to side. The manipulation in either case simply forces the pus out of the pocket.



## SENSITIVENESS OF THE NECK OF THE TOOTH

After the disease has existed for a time the periodontal membrane which normally covers the neck and the root of the tooth is slowly but surely destroyed, leaving that part of the tooth exposed. In certain individuals the dentin is especially sensitive to heat and cold, sweet and sour, etc. This symptom is present to some extent in all advanced cases, but varies greatly in different cases. It is quite noticeable in some early cases, even before retraction has occurred.

## RETRACTION

Retraction of the gums is not an early symptom, except in those cases where it has been produced by overuse of the tooth-brush and powders and pastes containing powders. As the alveolar process is absorbed the gum retracts, the tooth appearing larger and longer. In some cases the tooth gets loose and is lost before the alveolar bone is absorbed, and in such cases there is not much retraction. In others, perhaps the less rapidly progressing cases, the bone is absorbed and retraction of the gum occurs, until sometimes the length of the part of the tooth that is uncovered by gum is more than twice as great as normal.

## LOOSENESS OF THE TEETH

The teeth get loose sooner or later in all cases if pyorrhea is allowed to run its course, because it always causes the loss of the teeth. For a time the tooth will get very loose and then tighten up



Fig. 26.—A case of pyorrhea involving all the teeth. The gums were massaged to bring out the pus, which can be seen at the gum margins.

again. Sometimes months or years before it is finally lost it may be quite loose. The least force moves it from side to side. The patient can move it aside with his tongue. In addition to this loosening, due to destruction of the tissues which

normally hold the teeth firm, there also sometimes occurs sufficient thickening of the peridental membrane to lift the tooth from its bed in the socket, in which instance the tooth gives considerably when force is applied to it.

#### CHANGES IN THE POSITION OF TEETH—MALOCCLUSION

Inflammation of the peridental membrane causes more or less swelling or hyperplasia of the tissue. This tends to lift the tooth from the socket, especially when no opposing pressure is exerted to prevent it. When one of two teeth that normally articulate with each other is lost for any cause, the other is not kept pressed in place, and if diseased with pyorrhea, is forced outward from the bottom of the socket by the thickened peridental membrane. The incisors normally do not articulate in the majority of people, and therefore are especially likely to present this symptom of pyorrhea. The tooth may be noticed to be elongating. This is often the first symptom that attracts attention. It often occurs that the pyorrhea lesion is much more extensive on one side of a tooth than on another. In such an instance the thickening of the peridental membrane exerts constant pressure in the opposite direction, causing the structures to give way gradually and thus the tooth is moved sideways. The space between the teeth widens.

More frequently there is combined action, lifting the tooth in the socket and tilting in one direction or another. Teeth may be lifted and tilted to one



Fig. 27.—The teeth of some people seem to be more firmly set than others. This is one of them. There is no moving of the teeth in spite of a bad pyorrhea. The interdental soft tissue has been destroyed. There is no soft tissue connection between the labial and lingual gums.

side or the other. Often they are tilted forward, giving rise to increased prominence of the front teeth. Sometimes considerable twisting of a tooth from its normal position may be caused in this

way. It is remarkable that so much change in the position of the teeth can be produced with so little



Fig. 28.—Bad pyorrhea in a young woman. There are deep pyorrhea pockets around each of the teeth, and some of them have been moved recently from their normal positions. The endamebæ disappeared from the oral administration of ipecac. No emetin was given hypodermatically.

and often no pain whatsoever. No doubt it is due largely to the slowness of the process. The action



is so slow that the tooth often remains fairly firmly held for several years.



Fig. 29.—Rapidly progressing case of pyorrhea in a young woman. Note retraction of gum from canine tooth, and how other teeth have moved out of their normal position—the extent to which two of the teeth have risen out of their socket. The endamebæ disappeared from this case after three daily hypodermics of one-half grain of emetin each.

The molar teeth are often tilted in one direction or another in the line of least resistance whenever



they are lifted in their socket by thickened peridental membrane. When one has been lost, and the absorption of the alveolar bone makes quite a gap, the tooth behind it tilts forward and sometimes almost lies down in the gap before it is finally lost from the pyorrhea. Changes in the position of the teeth of an adult may, therefore, be considered a most significant symptom of pyorrhea.

#### GUM-BOILS

Gum-boils may sometimes occur without pyorrhea being present. Infection extending down a root-canal and escaping into the peridental soft tissue may produce abscesses. Most of them are produced by poorly drained pyorrhea pockets getting stopped up in some way or another. Foreign substances, like food particles, forced into them may interfere with their drainage and cause an abscess.

#### CHANGES IN THE GUMS

Retraction of the gums has already been mentioned as a symptom of pyorrhea of long standing. In the early stage there occur more or less redness and slight swelling of the edge of the gum. This may be so slight as not to be noticeable, especially in the interdental gum. Usually, however, the interdental papilla extend a little higher than

normal and more or less edema is present. Redness is not so marked as in the gum in front of or behind the tooth. Often, in spite of very deep pockets beside a tooth, there is little or no redness.



Fig. 30.—The last stage of pyorrhea. Note the pus on teeth. This was brought out by massaging the gum just before the picture was taken. Note that lower teeth have fallen over because of the loss of the supporting structures. Such teeth are held by only a very little bit of living tissue at the bottom of the socket.

In fact, the gum may appear to be attached to the tooth in the normal manner until it is examined carefully, when it is found that a suitable narrow instrument can be passed down by the side of the

tooth for a considerable distance without meeting with resistance. If the instrument is then manipulated, a little blood wells out of the pocket.

After the disease is advanced often there is a red or purple appearance of the gum over the



Fig. 31.—The last stage of the disease. Imagine what a fruitful source of infection such an individual may be to others! Such teeth pain or worry him. He picks at them unconsciously with his fingers, and is likely then to handle things handled by others.

tooth, and extending perhaps as far as the level of the apex of the root of the tooth. We think in most cases of this kind there has also been considerable destruction of the periosteum covering the alveolar bone over the tooth, as well as that lining

the socket. Though the absence of redness and inflammation of the gum cannot be depended upon to exclude pyorrhea, its presence is a symptom of great diagnostic value. It is true that inflamma-



Fig. 32.—The last stage of pyorrhea.

tion may be produced by trauma or certain other things, but it is usually temporary.

#### ABSORPTION OF ALVEOLAR BONE

Absorption of alveolar bone begins as soon as any part of the periodontal membrane between it and the tooth is destroyed, but it is usually many

months or years before there has been sufficient absorption for this to become a prominent symptom. The absorption continues after the tooth is lost until the alveolar process has been entirely removed. This may, therefore, be more correctly considered as a symptom or evidence of the previous existence of pyorrhea, instead of its presence.

#### SUMMARY.

The symptoms of alveolodental pyorrhea are variable in different individuals.

The earliest symptom usually is bleeding from one or more gums. It may be produced by the least manipulation or force, like brushing the teeth, picking the teeth, sucking air between them, etc.

Soreness and unpleasant feeling of the gums and of the teeth on pressure may be present for a long time. Often in the last stage there is considerable soreness at times.

Bad taste in the mouth, especially in the morning, is present in most cases, but usually it comes on so insidiously and has existed so long that the patient simply accepts it as a natural condition. He is more impressed with it whenever he gets rid of it from appropriate treatment. The relief attracts his attention.

Foul breath is a symptom in all advanced cases.

It varies with the bacterial and spirochetal flora in the individual's mouth.

Pyorrhea is a constant symptom. Pus can always be demonstrated by competent microscopic examination, but in the advanced stage macroscopic pus can be demonstrated.

Sensitive dentin is often exposed by retraction of the gums.

In the late stage of the disease, involving a given tooth, the tooth gets loose in its socket and may be tilted about by little force.

Malocclusion occurs especially with teeth that do not articulate with or press against others. Sometimes they rise out of their socket one-eighth inch or more.

Absorption of alveolar bone and the resulting retraction of the gum are constant symptoms of the late stage of the disease.



## CHAPTER VII

### DIAGNOSIS

HERE, as in most other diseases, the first thing necessary for the diagnosis is to suspect the presence of the disease. On account of the great prevalence of alveolodental pyorrhea one may feel practically sure of its existence, to some extent, at least, in almost all adults and in a large percentage of children. Its presence should be suspected in all people until proved otherwise.

It should be understood that pus formation results from any lesion accompanied by a break in the soft tissues of the mouth. The amount of pus may be great, or it may be so small as to require microscopic examination to demonstrate it. Unless endamebæ are present, lesions in the gums heal rapidly provided the cause is removed. During the period the lesion exists there is more or less pus formation. Such, however, does not constitute the specific disease discussed in this book.

### HISTORY

The history given by the patient of bleeding gums for a considerable time, changing in the posi-

tion of certain teeth, soreness of the teeth, gum-boils, looseness of the teeth, and finally pus discharging from around the teeth is reasonably certain to mean that the patient has endamebic pyorrhea. Any one or all of these symptoms could be caused by other things, but if so, the cause would usually be apparent or known.

When a patient is examined for pyorrhea all of the teeth and gums should be inspected systematically, both on the labial and on the lingual side. By inspection inflamed, swollen gums, retracted gums, teeth out of position without being pushed out by other teeth, and rather wide lesions may be observed.

Suitable instruments to feel for lesions or pockets in different suspected places are very valuable. An ordinary wooden toothpick will do in the absence of something better, but the most convenient instrument for this purpose is a Younger dental scaler No. 22 (Fig.



Fig. 33. — Younger dental scaler No. 22, the best instrument with which to obtain material containing endamebæ from most pyorrhea lesions. Its mate, No. 23, is better suited to a few of the lesions.

33). The mate to this, No. 23, is often useful to reach places to which the No. 22 is not adapted. With this little instrument the presence or absence of lesions can be determined by gently pressing the point of this instrument down by the side of a tooth where the disease appears to exist. Frequently it goes very deep without meeting with resistance—much deeper than one would have expected from the appearance. This is especially true of lesions between the teeth. More or less pus is brought out by scooping with the flat side of the scaler. Frequently there may be sufficient pus present to be squeezed out either by massaging the gum from the level of the end of the root of the tooth toward the crown or by moving the tooth from side to side. The pus appears between the margin of the gum and the tooth. Such a condition justifies a practical diagnosis of pyorrhea without further evidence. Loose permanent teeth hardly result from any other condition than pyorrhea.

#### MICROSCOPIC EXAMINATION

Endameba, being a microscopic parasite, it would not be possible to say positively that any case of pyorrhea had endamebæ present without making a microscopic examination. We wish to say here, however, that though the ideal way to make the diagnosis is by microscopic examination,

diagnoses sufficiently reliable for practical purposes can be made without it. We advise all whom circumstances permit to make or confirm their diagnoses by microscopic examination.

**Method of Obtaining Material for Examination.**

—In obtaining material for microscopic examination for endamebæ the fact that most of the parasites are in the edge of the peridental membrane in the very bottom of the lesion should be kept in mind, and an effort should be made to scrape the material for examination from the bottom of the lesion. Some endamebæ can usually be found in the pus that can be massaged out or obtained by working the tooth back and forth, but not as many as in material scraped from the bottom. With the dental scaler or other instrument one should try to scrape some material from the very bottom of the lesion. In advanced disease much pus and other material is obtained also, but in early lesions often a very small amount is obtained. Avoid causing bleeding as much as possible. Blood dilutes the material too much.

**Preparation of Stained Specimens for Examination.**—Endamebæ can be demonstrated either in stained preparations or in unstained wet preparations, in which case the living endamebæ are seen showing their characteristic ameboid motion.

To prepare a stained specimen the material

obtained from the lesion is spread thinly on a glass slide and stained. There are many methods of staining, each having certain advantages, perhaps, but the following is good enough for all ordinary purposes. The stains used are among those commonly in use in all laboratories.

1. The dried film is fixed with heat by passing it rapidly, film side upward, through the flame of an alcohol lamp or Bunsen burner two or three times. The slide should not get so hot but that it can be touched to the back of the hand without burning.

2. Cover the film with carbolfuchsin—one or two drops are all that is necessary.

3. Wash off at once with water. (Do not dry.)

4. Cover the film with Löffler's solution of methylene-blue. One or two drops are sufficient. Allow to stain for about one-quarter to one-half minute.

5. Wash off with water. Dry by blotting or by simply fanning in the air for a few minutes.

The carbolfuchsin stains all objects on the slide—pus-cells, bacteria, etc. It is a quick, strong stain. Löffler's methylene-blue is also a strong stain, and if the preparation which has first been stained red with the carbolfuchsin should be stained with the methylene-blue long enough, the red would be entirely displaced by the blue and

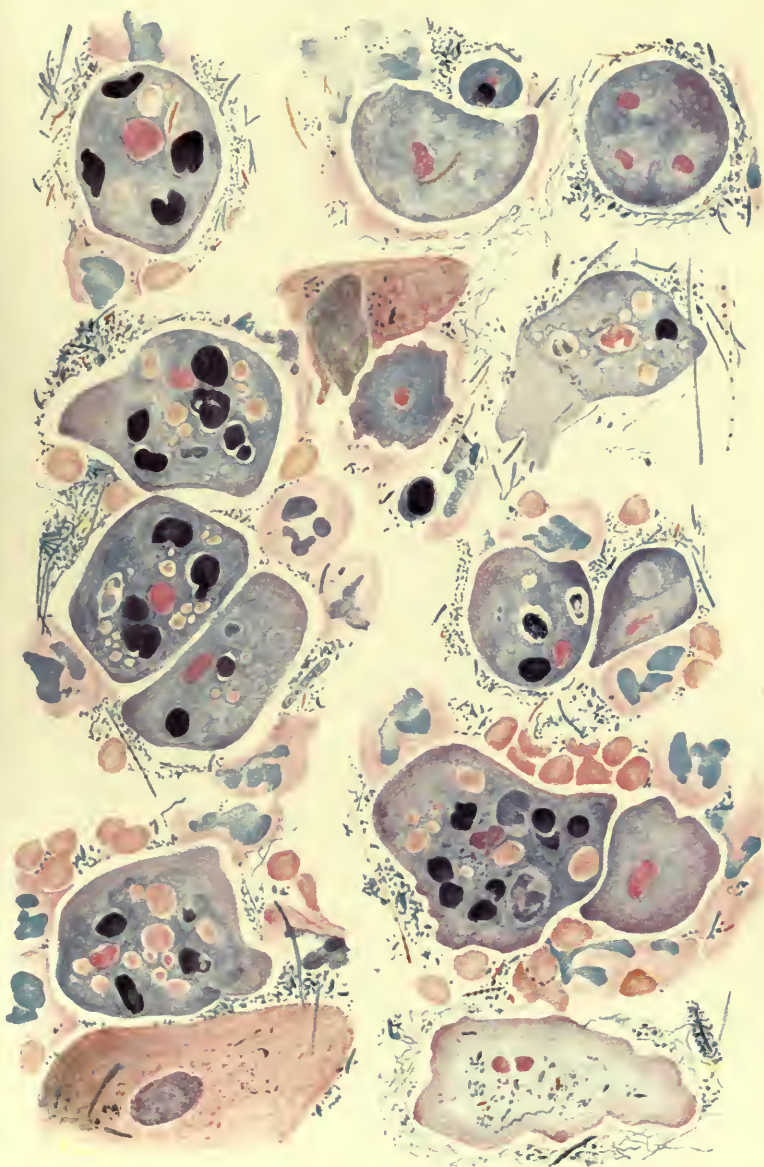


everything on the slide would be stained blue. The red is displaced by the blue more rapidly in some objects on the slide than others, and in certain portions of a given cell or endameba than others. If we carry the staining with the blue just far enough, we have a beautiful combination of blue and red staining in different objects and different portions of objects. This serves to show up their morphology and different parts to great advantage, especially the endamebæ. There is considerable difference in the length of time necessary to stain different specimens with the methylene-blue to get the most perfect results, but from one-fourth to one-half minute is sufficient in most cases. The film appears purple to the eye when properly stained.

**Examination of Stained Specimens for Endamebæ.**—After the specimen has been prepared and stained it is ready for microscopic examination. Place a drop of immersion oil on the film and examine with the oil-immersion lens. Select a part of the film for examination that is fairly thin. Generally speaking, it is better to search for the endamebæ in parts of the film where it is only about one cell thick. They can be seen and recognized better in such surroundings than where the film is much thicker. If the film is made of material from exactly the proper part of the lesion,



PLATE I



Selected *Endamoeba buccalis* and other material from pyorrhea lesions illustrating different sizes and features found in stained specimens. These are all camera lucida drawings from specimens stained with the carbolfuchsin and methylene-blue stain.



often one or more endamebæ may be found in every field. On the other hand, if the preparation is made of the pus, as it is likely to be, and not material scraped from the bottom of the lesion, it may be necessary to search many fields during several minutes before finding an endameba.

Those familiar with microscopic examination of material of this kind, especially if familiar with the appearance of stained specimens of other endamebæ, will usually have no difficulty in recognizing the *Endamœba buccalis* and in differentiating them from the other much more numerous cells, bacteria, débris, etc., present. (See Plate I.)

The bacteria present are usually of many different kinds. Different species stain by this method blue, purple, or pink. The spirochetes, which are often present in very large numbers, usually stain purple. Red blood-cells stain pink. The pus-cells present are chiefly polymorphonuclear leukocytes. In well-stained specimens the cytoplasm stains pink and the nucleus stains purple. The contrast between nucleus and cytoplasm is good. Some epithelial cells may be present. Their cytoplasm stains pink and their nuclei purple. The nucleus is round or oval, and is comparatively small, while there is a large amount of cytoplasm. There may also be endothelial or other cells ob-

tained from the granulating surface of the gum side of the lesion. They are usually quite large, being as large as, or larger than, epithelial cells. The nucleus is larger and the cytoplasm stains a pale purple.

**Appearance of the Endamebæ.**—Among this mixture of various kinds of cells, bacteria, etc., endamebæ stand out in striking contrast in most instances.

Endamæbæ buccalis vary in size from about 32 microns down to about 6 microns in diameter. Some are round or oval, others are more or less irregular in shape, depending largely upon the rapidity of drying when the material was spread on the slide. Two parts of the parasite are clearly shown by this stain—the endosarc, or inner part, and the ectosarc, or outer portion. The ectosarc, which stains pink or light purple, constitutes a covering of the endosarc, varying in thickness at different places and in different individuals. In some places it consists of an almost indistinguishable band, while in other places it may be as thick as one-tenth to one-fifth the diameter of the parasite. In places it may project a considerable distance without a corresponding projection of the endosarc.

The endosarc stains blue or slightly purple and there is apparently variation in its density. The

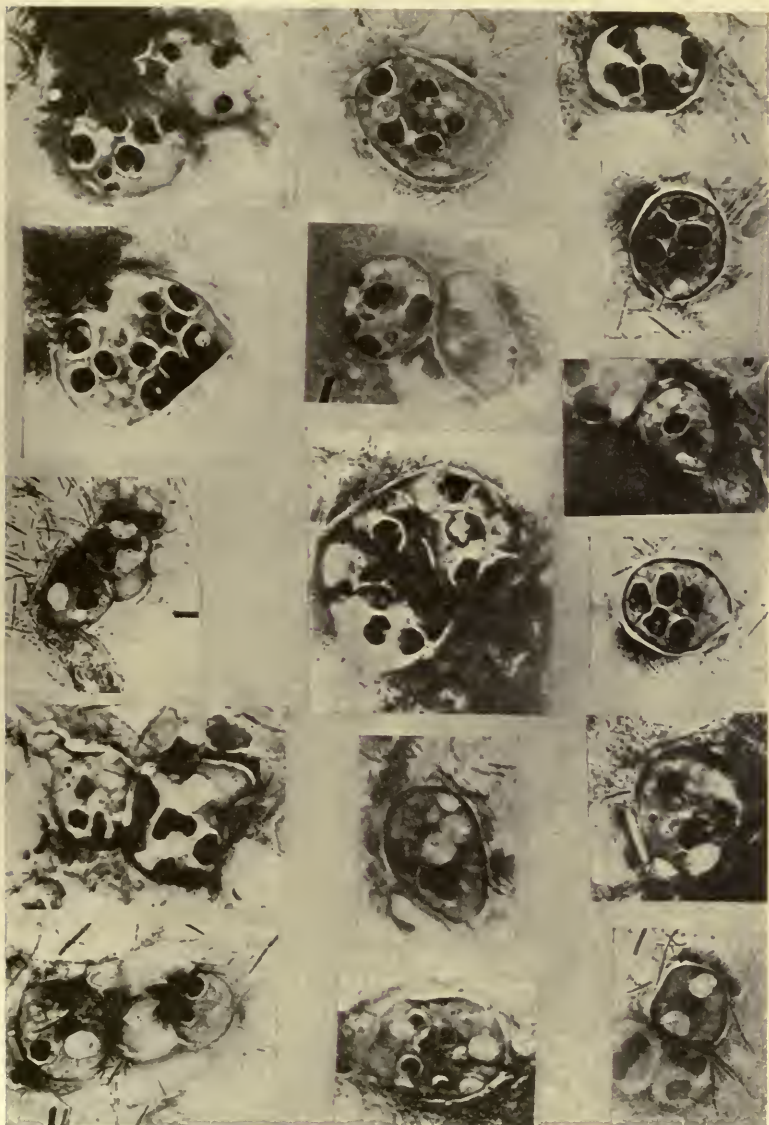


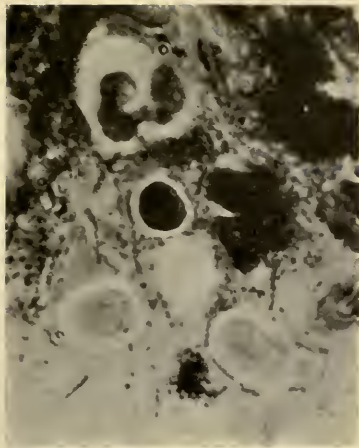
Fig. 34.—Photomicrographs of selected *Endamœba buccalis* representing different sizes, shapes, etc., in stained specimens. The large picture in the center is probably two or three endamebæ. The large parasite to the left and above contains 11 of the dark-staining inclusion bodies, about the largest number ever found in a single parasite.



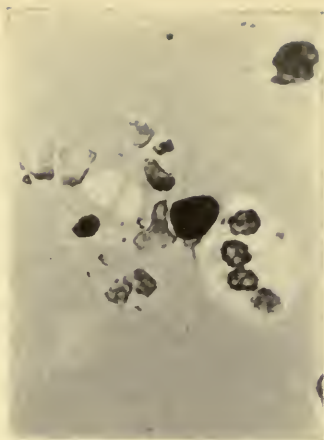
shape is round or oval or irregular, but is never as irregular as the ectosarc around it. The endosarc contains a variable number of very dark, almost black stained bodies. Some of the smallest parasites may not contain any of the bodies, while the largest may contain as many as ten. These inclusion bodies vary in size from about one to three microns in diameter. Most of them are round or oval. Quite a few are polymorphous, resembling the nuclei of polymorphonuclear leukocytes more or less shrunken. Some of these have more or less pinkish staining protoplasm around them, which still further indicates that they are leukocytes or other cells. In fact, it seems very certain that these bodies are the remains of cells, perhaps of different kinds, the cytoplasm of which is consumed or digested by the parasite. It is probable that the food of the parasite consists largely of these cells obtained from the diseased tissue. We have often recognized them scattered about free in the preparations. These bodies attract attention to the parasites under the microscope perhaps as much as any other thing. Usually they appear even more prominently because of a clear ring (due to retraction?) around them.

The endosarc also contains red blood-cells or remnants of red blood-cells in various stages of destruction. These stain pink. They may be

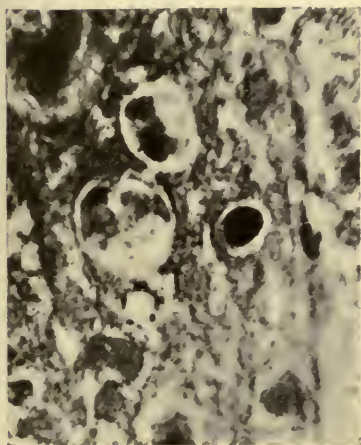




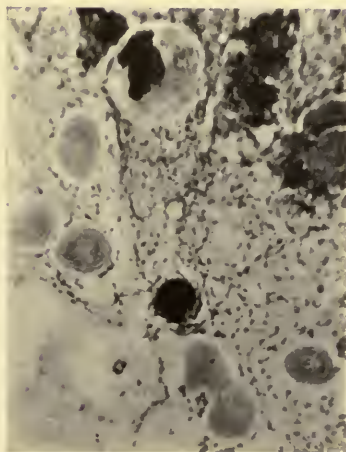
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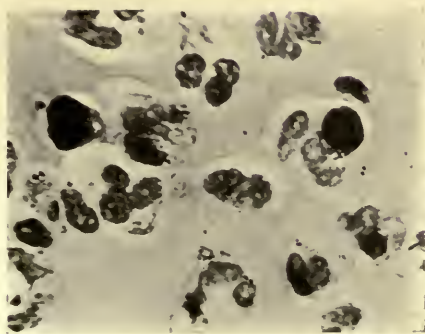
B



C



D



E

Fig. 35.—Photomicrographs of pus from different sources showing the dark staining bodies apparently similar to those seen in *Endamoeba buccalis*. A, From pyorrhea; B, chancroidal gland; C, granulating eczema surface; D, staphylococcal infection of abraded surface; E, acne vulgaris. Note the phagocytosis of one of these bodies by a pus-cell at the right.

absent from the smallest and some other endamebæ.

Bacteria of various sorts may be seen apparently in both the endosarc and the ectosarc of some of the parasites. Sometimes they are arranged in groups or masses. They do not appear to be present in a large portion of the endamebæ in most preparations.

A collection of chromatin can frequently be seen in specimens stained with this stain, but it is better shown in specimens stained with the modifications of the Romanowsky stain (Wright's, Giemsa's, etc.). It is round or oval, not more than one micron in diameter, and may be situated in most any part of the endosarc.

**Examination of Unstained Specimens.**—Endamebæ can be diagnosed with satisfaction in unstained specimens provided the examination is properly made. The advantage of this examination over stained specimens lies chiefly in the fact that the endamebæ are seen alive and showing their characteristic ameboid movement. On the other hand, perhaps a little more expert knowledge of the use of the microscope and of the appearance of other objects in such preparations is required than is required in examination of stained specimens.

Material is obtained from the bottom of the

pyorrhea lesion or pocket in the same way that it is obtained for stained specimens. It is diluted five or ten times by mixing it on a slide with an appropriate quantity of patient's saliva and at once covered with a cover-glass. A good plan is first to place about one-fourth drop of the saliva on the slide at about the middle of the slide. Then obtain the material to be examined. It usually is a little slow to come off of the instrument and to mix with the salt solution. A small, common, hard-wood toothpick is a very convenient instrument with which the material may be removed from the point of the dental scaler or other instrument used to obtain it and to facilitate mixing with the saliva. Water may be used instead of the saliva, but the endamebæ do not live as long in it.

The examination should be made with the high dry or 4 mm. objective. Owing to the fact that the objects to be studied are hyaline, or have no color now, it is necessary to reduce the light considerably. This may be done by closing the iris diaphragm under the Abbé condenser or by lowering the condenser to some extent.

Bacteria are usually very numerous and of various kinds in such preparations. Some are motile, others are non-motile. Spirochetes are also usually very numerous, and their activity

attracts attention. It is quite possible that they are of considerably more importance as secondary factors in producing pyorrhea than the bacteria are.

There are more or less red blood-cells in most preparations. Pus-cells, chiefly polymorphonuclear neutrophilic leukocytes, are the most numerous cells present. A few may still be living, but they seldom show any considerable ameboid movement. Their smaller size, small characteristic nuclei, and their granular cytoplasm serve to differentiate them from endamebæ.

Epithelial cells, which may also be present, show a small round or oval, clear-cut nucleus, and relatively large amount of clear cytoplasm. They are non-motile.

**Appearance of Living Endamebæ.**—Endamebæ first attract attention by being larger than the much more numerous pus-cells present. It is true that the smallest endamebæ are smaller than pus-cells, but whenever they are present there are also many more of the large parasites, many of which are two or three times the diameter of pus-cells. They are more or less irregular in outline (see Fig. 36), and with proper adjustment of the light and close focusing the parasite is seen to consist of two separate portions. The ectosarc is clear greenish and quite homogeneous in appear-



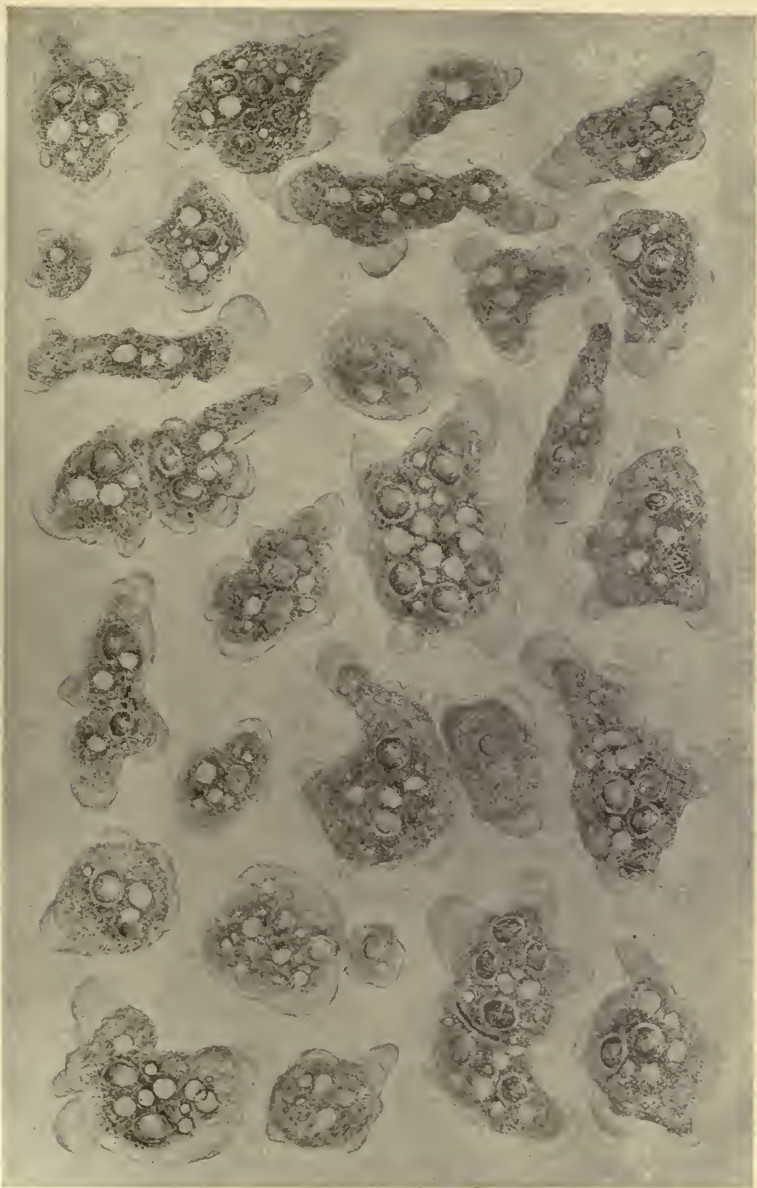


Fig. 36.—Large number of selected living *Endamoeba buccalis* showing different sizes, shapes, etc., that may be seen.

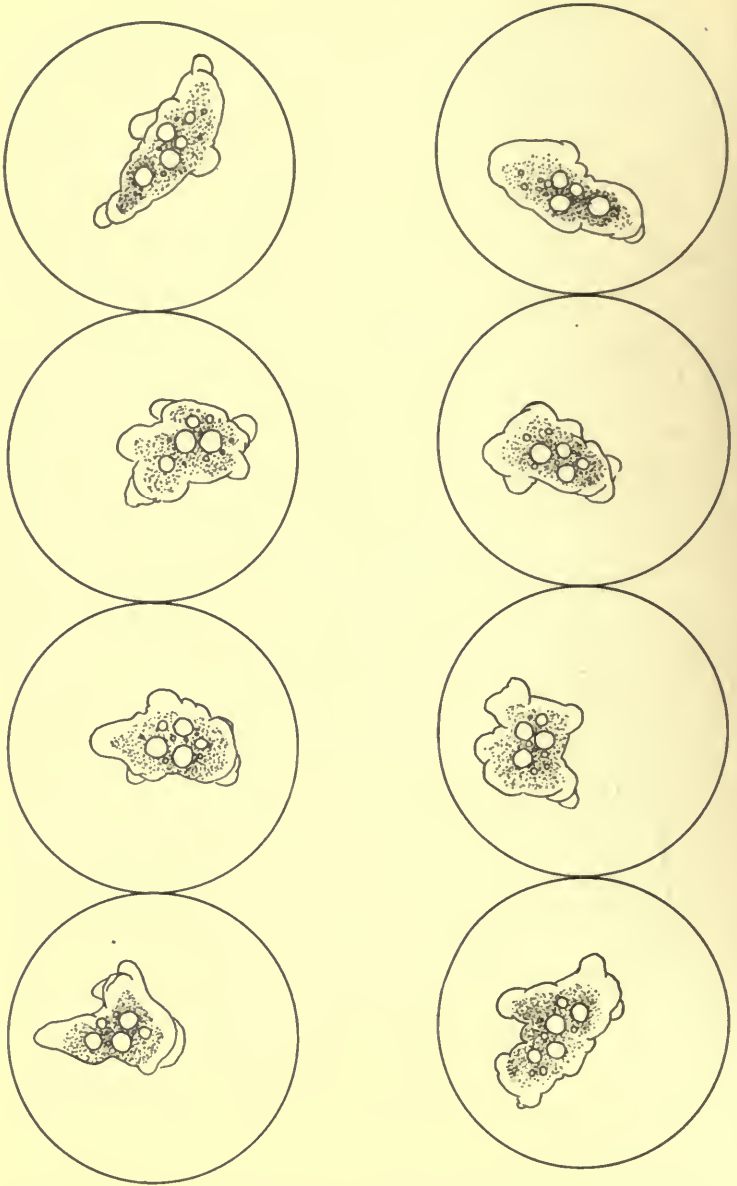


Fig. 37.—Drawing of an active *Endamoeba buccalis*, illustrating changes in shape and position at intervals of about one minute.



ance, while the endosarc is darker and contains many coarse granules, cell detritus, and vacuoles.

If one watches the parasite for a moment the characteristic ameboid movements may be seen. A pseudopod of ectosarc projects in one direction and perhaps is retracted in a few seconds. Another one may project in another direction, and it may either be withdrawn or it may continue to enlarge, carrying with it a part of the endosarc. Finally the entire cell protoplasm has flowed into the pseudopod and actually moved from its original position. In the space of a minute or two of time the most active endamebæ may change their shape many times, and may move the distance of their diameter or more. Often two or more pseudopodia may be projected in different directions at the same time. Sometimes a pseudopod will be projected from a pseudopod. With a little practice one acquires the capacity to recognize the endamebæ when not actively moving, but the diagnosis should be made on these alone with caution.

Endamebæ in such preparations live and move for several minutes or an hour or two if kept warm. If the room is cold, they quickly cease motion. As they die they become round or oval in shape and clear, so that they are difficult to recognize.

After a few hours most of them disintegrate and no other trace of them can be found.

**Differentiation of *Endamœbæ buccalis* from Other *Endamebæ*.**—We do not know of any method by which this endameba can be differentiated from the many species of amebæ that exist that would not involve a knowledge of protozoölogy greater than this book is intended to convey, and far greater than the authors possess. It will require much more study of the protozoa of the mouth before we will be able to say but that what we are now calling *Endamœba buccalis* may represent several different species, which our present knowledge does not lead us to differentiate. It may be possible that harmless species may be found in the mouth associated with the pathogenic species. In one instance of bad pyorrhea in which endamebæ having all the appearance of *Endamœba buccalis* were present deep in the lesions, we found large numbers of what we took to be *Amœba coli* on the surface of the gums, well up under the lips and cheeks. They seemed in this instance to be living and reproducing in the mouth without doing any harm.

**Apparatus and Material Required for Microscopic Examination.**—For the benefit of those who may intend to prepare to make microscopic examinations for *Endamœba buccalis*, we give here a

complete list of just what we would get for the purpose under the circumstances:

1. Microscope, Bausch and Lomb. B. B. 8. Ocular X, Objectives 16 mm., 4 mm. (N. A. O. 65), and  $\frac{1}{12}$  oil-immersion lens. This is a standard microscope for general clinical labor-

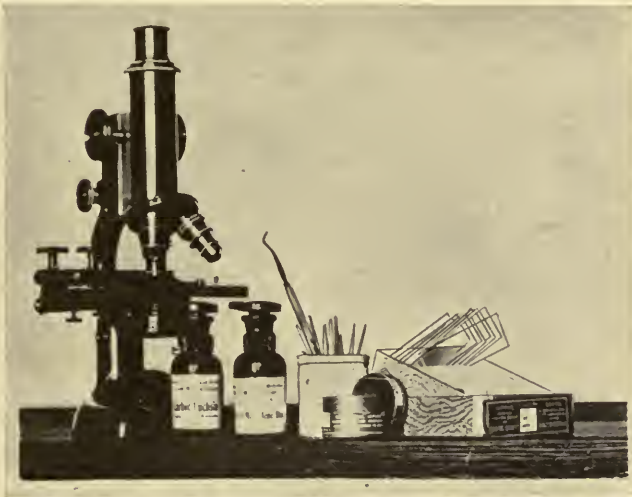


Fig. 38.—The apparatus and material required in making microscopic examination for endameba. See list, pages 117 and 118.

- atory work. (Should have case made to hold microscope with mechanical stage attached.)
2. Mechanical stage. Though this is not essential, it is very valuable to all except those who have had a great deal of practice.
  3. One box regular size, medium thickness microscope slides.

4. One box cover-glasses  $\frac{7}{8}$  inch square, No. 2.
5. One or more pairs of Younger's dental scalers Nos. 22 and 23.
6. A few hard-wood toothpicks.
7. One alcohol lamp, or Bunsen burner if gas is available and preferred.
8. Two T. K. drop bottles, flat top, 30 c.c. each. The best have the dropper come out from the side of the top and not from the neck of the stopper below the flat top.
9. Two fluidounces carbolfuchsin (Czaplewsky's formula).
10. Two fluidounces Löffler's methylene-blue solution.

Description of the apparatus and material required is given in such detail for the benefit of those who are not familiar with clinical laboratory work, and it should be clearly understood that other equipment will serve the same purpose.

#### SUMMARY

The history of bleeding gums, gum-boils, and loose teeth and teeth changing position all indicate pyorrhea.

On account of the great prevalence of the disease in all adults, it should be suspected in all grown people. To suspect its presence is the first thing necessary in diagnosis.

The endamebæ are most numerous in the bottom of lesions, and proper material for examination can best be obtained with a Younger scaler No. 22 or No. 23.

The endamebæ are more easily demonstrated in stained specimens than in unstained specimens. The technic for preparing and staining preparations is given on pp. 102, 103, 104. A list of the apparatus and material required for microscopic examination for endamebæ is given on pp. 117 and 118.

Endamœbæ buccalis are from 32 to 6 microns in diameter. Most of them contain very dark-staining inclusion bodies, which are in striking contrast to the color of the endamebæ and to all other material on the slide. These serve to attract attention to the endamebæ and assist very much in finding them. We could not give a description that would tell as much about the appearance of the parasites as the several drawings, photomicrographs, and colored plate in this book, to which the reader is referred.

## CHAPTER VIII

### TREATMENT

**General Discussion.**—The treatment of alveolo-dental pyorrhœa has for its object, first, the destruction of the *Endamœba buccalis*, the specific cause of the disease; and, second, the healing of existing lesions. Since the damage done by the disease consists largely of destruction of structures and tissues, it would seem that little repair of the damage done could be expected. Nature can no more grow new alveolar bone after it has been removed nor grow gum back onto the part of a tooth from which it has retracted than she could grow a new tooth in place of one that has been lost or a new limb in place of one that had been amputated. For the purpose of obtaining the first object mentioned, we are fortunate to have a drug—*ipecac*—which is highly toxic to *endamebæ* and not harmful in proper doses to man.

It is needless to explain that healing of the lesions cannot reasonably be expected to take place until the cause has been removed. Though we can destroy the *endamebæ* with the specific, *ipecac* treatment, we have to depend largely upon



nature's processes for the healing of the diseased or damaged tissue after the cause has been removed. Certain things can be done to facilitate healing, however, and they will be considered after the specific treatment has first been explained.

**Description of Ipecac.**—Ipecac is the root of *Cephaelis ipecacuanha* (Richard), belonging to the *Rubiaceæ*. The plant is a small shrub, indigenous to Brazil, and cultivated in certain other parts of the world, especially in India. It contains three alkaloids: emetin ( $C_{15}H_{22}NO_2$ ), cephalin ( $C_{14}H_{22}NO_2$ ), and psycotin, and certain gums or resins. The action of the drug seems to be due entirely to the alkaloids, chiefly emetin and cephalin.

If ipecac is applied to the skin in sufficient concentration and for a sufficient length of time, it finally causes ulceration. Applied to the mucous membrane in sufficient concentration, it causes inflammation and ulceration. When taken into the stomach, nausea and vomiting are produced, apparently as the result of local irritation to the gastric mucosa and not of central irritation. Two to five grains of powdered ipecac are sufficient to cause vomiting in most individuals. The effect of a much larger amount taken into the stomach does not cause greatly increased symptoms, because the excess is vomited.

Emetin has about double the nauseating effect

of cephalin. Psycotin is present in small quantity, and is not irritating to the skin nor the mucous membrane, and is only slightly nauseating, even in large doses.

Both emetin and cephalin lower arterial tension when given in sufficiently large doses, emetin being the more powerful of the two. Neither of them produces nausea when given hypodermatically to man, but in animals, as shown by d'Ornellas, emesis is produced. Sufficiently large doses given hypodermatically or intravenously produce death in animals, and no doubt would do so in man. Ten milligrams given intravenously kills 2000-gram rabbits within a few minutes. Weakness and collapse follow in two or three minutes. This corresponds to about 0.38 gm. (6 grains) for a normal adult man.

Emetin is a non-crystalline, monacid base, but forms crystalline salts. The hydrochlorid is the one used therapeutically. Cephalin is a crystalline, monacid base, and forms crystalline salts. Both are colorless, but are decomposed by light. Their salts are stable. Emetin was found by Rogers to be much the more powerful amebicide, and it is therefore employed in preference to cephalin. Ipecac is amebicidal according to the amount of emetin it contains. Different samples vary much in their emetin content. The U. S.

P. standard requires that powdered ipecac should contain 1.5 per cent. total ether-soluble alkaloids.

**Manner in which Emetin Affects Endamœba buccalis in Alveolodental Pyorrhœa.**—Emetin is thought to be a powerful poison to endamebæ, just as quinin is a poison to the malarial plasmodia. Quinin is also toxic to man, and a sufficiently large dose of quinin would kill a man as certainly as a sufficiently large dose of strychnin would. Quinin is so much more toxic to the malarial plasmodia than it is to man, however, that practically harmless doses produce sufficient concentration of the drug in the blood to kill the plasmodia present. The virtue of most of our other specific drugs used against parasitic diseases depends upon their capacity to destroy the parasite with the minimum amount of harm to the patient.

Emetin given hypodermatically in doses so small as to produce no demonstrable constitutional effect in man furnishes sufficient concentration in the blood to eradicate *Endamœba buccalis* with which it comes in contact. This is thought to be because of the specific toxic action of the drug on the endamebæ. It is not equally toxic for other protozoa on which we have tried it—malarial plasmodia, trypanosomes, and trichomonas.

Rogers found that emetin hydrochlorid was amebicidal in dilutions of 1 : 100,000 or less. Less

than one-half grain given hypodermatically to a normal sized man should theoretically produce more than this concentration of the drug in the patients' blood, and thus change it to an amebicidal fluid.

Emetin is slowly eliminated, requiring more than forty-eight hours for the elimination of a single dose. The maximum concentration, however, is reached in about fifteen hours. Therefore it should not be necessary to repeat the dose frequently to maintain a considerable concentration in the blood. In this respect emetin is quite different from many other alkaloids which are quickly absorbed from the tissues, quickly reach the blood-stream, and produce their effect, and are quickly eliminated.

**Dosage.**—Emetin hydrochlorid has been employed for the treatment of amebic dysentery in doses of from one-third to two grains. In the case of amebic dysentery, the dose had to be determined more or less empirically. The lesions containing endamebæ are not accessible for frequent examination to determine just when they disappear nor what the effect of different doses is. With *Endamæbæ buccalis* in pyorrhæa lesions which are accessible for examination as frequently as desired, it is possible to observe the effect of different sized doses, given with different intervals

between doses, and the length of time necessary to eradicate demonstrable endamebæ. We have done considerable experimentation with the object of shedding light upon these questions, controlling all experiments by microscopic examination for the endamebæ. The doses of emetin experimented with have been from one-half to three grains. We have not been able to determine that any larger doses than one-half grain destroy the endamebæ any more quickly than this amount does. The local reaction is greater the larger the dose, and the cost is also proportionately increased; therefore it is desirable to employ the minimum dose that is certainly efficient. At this present writing we think one-half grain of emetin hydrochlorid given hypodermatically is the proper dose for average sized adults. The dose may be reduced for children in proportion to age.

**Interval Between Doses.**—We have also experimented considerably to determine what interval between doses leads to the best results. One dose during a day is about all that would be practical in most instances. We have no doubt but that slightly better results might sometimes be obtained by more frequent administration than once a day, but we do not think the probable advantage worth the inconvenience. On the other hand, we are convinced that emetin given one



dose every day without intermission will destroy endamebæ more certainly and quickly than when an interval of more than one day is allowed to intervene between doses.

**Technic of Giving Emetin Hypodermatically.**—Emetin hydrochlorid is the salt used for this purpose. The most convenient form for ordinary use is the sealed ampules containing one-half grain in solution ready for use. It is supplied in this form by several pharmaceutic houses. An ordinary hypodermic syringe (all glass syringes are the best) is sterilized by boiling. The neck of the ampule is broken, and the contents are drawn into the syringe. The injection is usually made in the arm, about the insertion of the deltoid muscle. A good way to sterilize the skin where the needle is to be inserted is to touch the skin with a little cotton applicator moistened (not wet) with pure lysol. A place one-fourth inch in diameter is sufficiently large. Stick the needle through this and pretty deeply. Make sure of going through all the skin. Less local reaction is produced the deeper the drug is placed intramuscularly. Inject the emetin, withdraw the needle, and wipe off the lysol.

**Local Effect of Emetin.**—There is always more or less local reaction following an injection of emetin. It varies very much with different indi-



viduals, but is generally worse in women than in men. There are redness and soreness in an area of from about three-fourths inch to two inches in diameter. It reaches its maximum in about two days and subsides in about three days. Sometimes there is considerable itching about the fifth day, accompanied by formation of small vesicles and shedding of epithelium. We do not know of an abscess occurring in any case, but we think such a thing may sometimes occur.

**Urticaria Produced by Emetin.**—About 1 per cent. of the cases treated with emetin hypodermatically have developed general urticaria. It was mild in all except one case, in which it was quite severe. The urticaria resembled that often produced by quinin or other drugs, and passed off in from one to three days. This is an objection to the use of emetin, but the same objection could be raised to several of our most valuable drugs. So far as we know and believe there is no way to prevent this, nor to know in advance those who possess this idiosyncrasy to emetin. We have not known of its occurrence when the emetin was taken internally, but should expect it.

**Length of Time Necessary for Endamebæ to Disappear.**—The length of treatment required before the endamebæ disappear from the lesions may depend upon several factors that we know of,

and probably others that we do not now recognize. There is considerable difference in the absorption, distribution, and elimination of drugs by different individuals, all of which may influence to some extent at least the effect of the drug. The most important source of variation, however, is the difference in the lesions in different individuals as well as in the several lesions in the same individual.

Emetin in the blood eradicates endamebæ which are in tissues bathed by the blood, but many of the endamebæ in a pyorrhœa lesion are in dying and dead tissue not well supplied with blood. It is true there is some exudation of body fluids through this material, but there is no such rapid change of fluids as in the living tissue. Large pyorrhœa pockets contain a considerable amount of pus, food particles, bacteria, etc., in which there are also many endamebæ. These substances are not likely to get much emetin from the blood. Endamebæ often disappear within twenty-four hours after a single dose of emetin from an early lesion in which all the endamebæ are either in living tissue or directly in contact with tissues well supplied with blood. Emetin gets to them promptly. In other more advanced lesions it requires longer—sometimes several days—for the contents of the pyorrhœa pocket to become sufficiently impregnated with the emetin to destroy all the endamebæ.

This perhaps is especially true whenever it includes spongy bone containing pus and endamebæ.

In our experience of more than 300 cases the endamebæ disappear in from three to six days from the time daily hypodermatic treatment is begun in all but a small percentage of cases. In these they disappeared in this time from all except perhaps one or more lesions of extreme type in each case.

**Treatment with Emetin Injected into the Pyorrhea Pockets.**—If the reader is familiar with the chapter upon Morbid Process, it will be clearly understood that it would not be practical or in most instances possible to find all pyorrhea lesions and to inject medicine into them so as to reach all the endamebæ. A pyorrhea pocket is practically a deep ulcer whose walls consist of bone or tooth and granulating soft tissue.\* The shape is such as to make it less practical to reach all parts with a local application than it would be to reach all parts of an open, superficial ulcer of the skin, for instance. There are many antiseptics which we can apply to such an open ulcer of the skin in sufficient concentration to kill bacteria, but such application fails to disinfect the ulcer, though applied constantly. The infection extends too deep, speaking in microscopic terms, to be reached

\* Cavernous sinuses extend into the soft alveolar process, and strands of undissolved connective tissue traverse the bottom of the lesion.

by the antiseptic solution applied to the surface. It is no more likely that we could disinfect a pyorrhea pocket by injecting the endamebicide into it than it is that we could disinfect it of bacteria by washing it out with an antiseptic solution, a thing that could not be expected. Washing out pyorrhea pockets with some harmless, non-irritating fluid, preferably physiologic salt solution, however, would undoubtedly be of value and would facilitate healing. Washing out the pockets would remove food-particles that would otherwise decompose, accumulations of bacteria that sometimes form considerable masses, pus, and débris, as well as some endamebæ. If emetin is added to the salt solution in proper quantity, it will do no harm and no doubt it will kill some endamebæ. The endamebæ washed out or killed are chiefly those in the dead material in the lesion, and not those deeper which are actually damaging the tissue. These are the endamebæ, however, that are least likely to be destroyed by emetin in the blood.

Emetin diluted 100,000 times will kill endamebæ, and therefore it is not necessary to employ a strong solution for this purpose. Strong solutions of emetin are irritating and tend to prevent healing. On the other hand, it is desirable to use a solution sufficiently strong that it will still kill endamebæ

after being diluted somewhat by the fluid secretion with which it may be mixed in the pocket or by being absorbed into the dead material. We think a solution of 1 : 10,000 or 1 : 15,000 would be harmless to the tissues and still be strong enough under all circumstances. This can be made up extempo-



Fig. 39.—Berlin abscess syringe suitable for washing out pyorrhea pockets with solutions of emetin.

raneously and economically by dissolving a one-half grain tablet of emetin hydrochlorid in a pint (bottle) of salt solution. The salt solution may be made sufficiently accurate for practical purposes by dissolving 60 grains (one teaspoonful) of sodium chlorid in a pint (bottle) of water.

Any suitable syringe may be used for this purpose. The best we have seen is the Berlin abscess syringe (Fig. 39), which may be obtained from dental supply houses and some drug stores. We have had two patients to volunteer the information that after being treated by their dentist by injecting iodine into the pyorrhea pockets they had supplied themselves with syringes and had made the injections themselves. This suggested the practicability of patients' washing out their own pyorrhea lesions, or having it done by some one at the home, with the emetin solution. We recognize that this can be practised only by certain patients and not with all lesions. Wherever practical, however, we think it might be found to be of considerable value.

Barrett injects a 0.5 per cent. solution of emetin into the pyorrhea pockets and also infiltrates the surrounding tissue with this solution, using a hypodermic syringe with proper needles. Good results have been obtained, including rapid healing of the lesions and disappearance of endamebæ. For the treatment of individual lesions, no doubt this method would give as good results in the hands of skilled operators familiar with the disease as any method. It is doubtful, however, whether it can be extensively applied successfully. It would require rather persistent use to disinfect the mouth



of endamebæ in the average case of pyorrhœa. Remember that in addition to the advanced recognizable lesion there are usually many others in the earlier stages of the disease, and some of these at least would be difficult to locate and treat. The greatest good will come to the patient from disinfecting his mouth and curing the earlier lesions as well as those more advanced, which at best leave a tooth very much impaired in usefulness because of the loss of supporting structures.

**Treatment with the Alkaloids of Ipecac Taken Internally by Mouth.**—The treatment by hypodermatic administration, though efficient, is necessarily limited because of the discomfort produced, the expense attached, etc. Almost all grown people have the disease and are doomed to lose their teeth after years of sloughing and pus production around each one of them, if they live long enough. Only a small fraction of all the victims of the disease could or would avail themselves of this treatment. Recognizing these facts, we set out to find some more practical and economic method of treatment than by administration of the drug hypodermatically or injection into the gums, requiring expert service each time. This we now seem to have.

**Alcresta Ipecac.**—John Uri Lloyd discovered that a colloidal form of hydrated aluminum silicate

possesses the power of adsorbing alkaloids from an aqueous, neutral, or acid solution. The alkaloid is then not given up except in the presence of an alkaline fluid, when it is promptly set free. The only source of the alkaloids of ipecac prepared in this way, so far as we know, is supplied by Eli Lilly and Company under the name of "Alcresta Ipecac," in the form of tablets each containing the alkaloids of 10 grains of ipecac. When taken into the stomach in this form, where the reaction is acid, the emetin and cephalin remain inactive until the alkaline intestinal juices are reached, when they are set free and absorbed. Quantities corresponding to 50 or even 100 grains of ipecac do not produce any nausea. The rate and the completeness of absorption necessarily vary somewhat with different individuals. Emetin is irritating more or less to all tissues. Certain individuals are more susceptible to its action than are others. The irritation to the intestinal mucous membrane is sometimes sufficient to give rise to griping and a little diarrhea. We have not seen these symptoms severe enough to necessitate the patient discontinuing the medicine, but we have no doubt this will occasionally occur. In most instances no bad effect is noticed. If sufficient diarrhea should be produced, there might be so little emetin absorbed and thus reach the blood

that it would not destroy endamebæ in the pyorrhea lesions. Such, however, seldom occurs.

The amount of ipecac necessary to contain one-half grain of emetin, our regular hypodermatic dose, is about 45 grains. It usually requires about twice as much of a drug given by mouth to have the same effect as when it is given hypodermatically. Therefore we should give an amount of the Alcresta Ipecac representing about 90 grains of ipecac per day. A convenient way is for the patient to take three tablets three times a day after meals—preferably one hour after meals.

We have now tried it out sufficiently to state that it causes the disappearance of endamebæ about as quickly as emetin given hypodermatically in a large percentage of cases. The Alcresta ipecac tablets should be continued for from four to six days or longer, the same as the hypodermics of emetin. A convenient way to write the prescription for them is—

*Rx.* Alcresta ipecac tablets (Lilly), 40.

*Label.*—Three tablets three times a day an hour after meals.

Forty is about the number required for a course for the average case, and the manufacturers supply them in original bottles containing 40 tablets each.

**Alcresta Emetin.**—Since the cephalin and psychotin are also contained in the Alcresta ipecac

tablets, we have recently had the manufacturers make for us Alcresta emetin instead of Alcresta ipecac (combined alkaloids). It was hoped that the slight discomfort of griping and looseness of the bowels might not be produced by this preparation. This matter is still under investigation.

**Relapse or Reinfection.**—As a result of a proper course of from four to six days or more of emetin treatment, we are unable to find endamebæ in the lesions except perhaps in a small percentage of cases, and usually only in certain bad lesions. It is not possible to say that no endamebæ remain. We can only say that they have been reduced to such small numbers that they are not demonstrable, or else they have entirely disappeared. It may perhaps be desirable for us to explain why we advise to discontinue the internal administration of emetin as soon as the demonstrable endamebæ disappear, instead of continuing it longer. The pain and expense accompanying the hypodermatic treatment make it desirable not to continue the treatment any longer than necessary. The intestinal disturbance sometimes produced by the Alcresta ipecac (alkaloids) is one reason for desiring to discontinue it as soon as it is no longer needed. The Alcresta emetin may not have this objection to the same extent, but there is another reason applicable to all forms. Many alkaloids, whenever taken for

more than a few days at a time, rapidly lose their effect. They are either neutralized or eliminated without getting into the blood in the same concentration that they did when first taken. The maximum effect is obtained when the alkaloid is taken for only a few days, and repeated if necessary after an interval of at least a few days. We do not want to set the course of treatment arbitrarily at four to six days. We simply say it is sufficient for most cases, but we have no doubt but that a longer course will be found, sometimes, at least, more successful. In fact, we feel that if we were deprived of the microscopic examination with which to control the progress of treatment, we would be inclined to make the course of treatment longer, perhaps ten days or two weeks in the worst cases.

Though the specific emetin treatment causes the disappearance of demonstrable endamebæ, the lesions or pockets remain and will require days, weeks, or months to heal, depending largely upon the size, extent, and nature of the individual lesions as well as upon the natural healing powers of the individual patient. In many lesions there is considerable alveolar bone denuded of its periosteum, which, therefore, must be removed by the long, slow process of nature, if not aided by artificial mechanical means. It requires nature several



months to absorb and remove the alveolar bone after a tooth has been extracted, and it requires several months also to remove the alveolar bone above the level of the living peridental membrane in the case of pyorrhea. After the endamebæ have been destroyed and the blood no longer contains emetin, these lesions or pockets offer the most favorable soil for reinfection.

**This disease is almost universal.** Nearly everybody has the infection and is throwing off endamebæ with the secretions of the mouth at frequent intervals. With our present habits of drinking and eating after each other and putting things into our mouths that have been exposed to infection by others, we must be putting endamebæ into our mouths almost daily. With several healing pyorrhea pockets in the mouth offering most favorable places for lodgment of endamebæ, it could hardly be reasonably expected that one would go many days without getting reinfected. Reinfection established in one place would be a source of reinfection of other lesions as soon as the endamebæ have time to multiply sufficiently. This would seem to take away the promise of complete cure by use of emetin unless it is repeated at frequent intervals. We have, however, been able to eliminate this discouraging feature, to some extent, at least, by prophylactic measures, especially the



local use of ipecac or emetin by the patient. The reader is invited to turn to page 160, where, under the heading "Local use of emetin as a prophylactic," this subject is fully discussed. Emetin used in this way would tend to prevent reinfection. It could not, however, be expected to furnish absolute protection, because endamebæ may be introduced at any time of the day or may be introduced deeply, while the emetin is used only one or two or three times a day and cannot reach deeply situated endamebæ. In addition to the prophylactic use of emetin to prevent reinfection, patients should endeavor to minimize the introduction of endamebæ according to the suggestions made in the chapter upon Prophylaxis, page 144.

In spite of our prophylaxis, we may expect a certain percentage of cases to get reinfected long before nature could heal all the lesions present. In our experience, endamebæ are demonstrable in some of the lesions in at least 12 or 15 per cent. of cases in from three to six weeks after all had apparently been destroyed. It is not possible to determine whether these are cases of reinfection or cases in which all endamebæ had never been destroyed, and therefore more correctly

called cases of relapse.\* On account of the considerable number of reinfections or relapses, as the case may be, that occur, we believe it advisable that the course of emetin should be repeated after an interval of from two to four weeks in all cases, unless thorough microscopic examination is made and no endamebæ found present. The courses of emetin should be repeated again and again until all the lesions heal.

#### **Scaling and Scraping the Roots of Teeth.—**

If a tooth surrounded by a pyorrhœa pocket is extracted and examined, considerable accumulation of deposit of various kinds is found on the part of the root that was between the level of the edge of the gum and the edge of the remaining periodontal membrane. This may be either soft or hard. There is a deep-set opinion held by the dental profession that it is important to scale or scrape this off to facilitate healing. This might be true if there was any hope that the destroyed periodontal membrane would be regrown and again cover the root of the tooth where it formerly existed. We have not the slightest evidence, however, that such a thing can occur. Apparently, therefore, the only benefit that would

\* Though we have not been able to demonstrate it, we think it quite possible that encysted endamebæ in the tissues escape the effect of emetin, and after it gives way, reproduce and give rise to another crop of the parasites.

result would be what results from cleanliness and removal of foreign material, which may in some instances be a source of irritation. However, if done properly, no harm should result.

**Removal of Redundant Tissue.**—In many pyorrhea lesions we have alveolar bone denuded of periosteum projecting upward by the side of or around the tooth. This bone can never be recovered with periosteum and will certainly be removed by nature by a long, slow process of liquefaction. This requires months in many cases, when it could be done at once by surgical procedure. The results to be expected from such operation done now are not to be compared with what may have been obtained when it was tried before we knew the specific cause of the disease and had a specific remedy against it. Cutting away redundant gum that helps to make the pocket ought also very much to facilitate the healing process. If left to nature, it finally retracts to the level of living peridental membrane, to which the edge of it is finally united. As long as the pocket is present it collects food and retains material from deeper down, which decomposes and keeps up irritation. The real disease is in the very bottom of the lesion, and converting the pocket into a wide-open, freely draining lesion very much facilitates healing.

Though we have not had sufficient observation to be able to recommend the operation of removing gum and bone that nature finally removes, we believe that it is the proper thing to do. One obstacle will be met in most cases, and that is objection on the part of the patient. It will not be easy to get patients and others to appreciate the real extent of the disease, and the fact that the overhanging bone and gum will finally be removed by nature anyhow. We are now able to explain the certain amount of success formerly obtained by different "pyorrhea specialists" by cutting down the gum and often cutting it away and by the application of acids into the pyorrhea pockets.

**Supporting Teeth that are Loose or Have not Sufficient Support to Give Them Strength.**—It frequently happens that a tooth or several teeth are more or less loose as a result of the great depth to which the peridental membrane has been destroyed. Healing and repair are prolonged by the strains to which the little remaining supporting structures are subjected. Steadying such teeth so they cannot move about, by binding them to other teeth in such a way as the individual case requires, may often be done to great advantage. In fact, some teeth may be saved whose insertion into the supporting structures may be so shallow

after retraction is complete that they will not stand up in their natural position, in which case permanent attachment to other teeth may be indicated. We shall not enter into any discussion of the many methods of accomplishing this object, and which are familiar to all dentists.

NOTE.—Since our early publications we have seen a considerable number of cases in which endamebas have reappeared after they had been absent for various periods of time following the use of emetin in one way or another. We have also seen a considerable number of cases in which endamebas have not been eradicated even for a short time by emetin. We know of instances in which endamebas disappeared from individual lesions as a result of local use of emetin after emetin given hypodermatically had failed to get rid of them. This all goes to show that the methods of specific treatment now followed fall far short of the ideal and that further experience should lead to great improvement. No doubt it will be found that there is good in both local and systemic use of emetin at the same time in most cases.

We do not want to be understood as underestimating the value of proper dental treatment of any abnormal condition, whether it be the result of alveolodental pyorrhea or other diseases or abnormalities of the teeth and supporting structures. We have not taken up the surgical treatment of pyorrhea because the methods that have been employed are familiar to the dental profession and are described in the dental literature, and because no new methods have yet been worked out in the light of our recently acquired knowledge of the specific cause of the disease. It remains for future investigation to indicate the proper surgical treatment to be combined with the specific emetin treatment.

## CHAPTER IX

### PROPHYLAXIS

THE reader should read at least the chapters on Etiology and on Morbid Process before reading this one, unless he is already familiar with the specific cause of the disease, the manner in which endamebæ produce the disease process, and the nature of the process.

**Care of the Teeth to Prevent Development of Favorable Soil for Infection.**—As has been pointed out, the destructive disease, alveolodental pyorrhea, is caused by a specific living microscopic animal parasite, *Endamœba buccalis* (and possibly other species), which lives as a parasite in the affected tissues. These endamebæ, however, are harmless when applied to normal gums and tissues. They cannot take hold and establish themselves on normal mucous membrane any more than tetanus bacilli applied to the normal skin could produce tetanus. Not only must tetanus bacilli be introduced into the tissue, but that tissue must be damaged, crushed, dying, or dead to constitute favorable soil for the organism to start growing in. Many other disease-producing organisms, some of



them of the most virulent type, are not capable of producing their disease when applied to normal unbroken skin or mucous membrane, for instance, plague bacillus, rabies virus, vaccine virus, *Treponema pallida*, *Leishmania*, etc.

*Endamœba buccalis* as a parasite of man has been accustomed to growing in a certain kind of tissue and environment for countless generations. This has been so for so long a time that it is more or less a fixed requirement of the species. It is not healthy tissue that they have been accustomed to live in, but it is dead, dying, and diseased tissue constantly irritated by food-particles and bacterial action and the action of the endamebæ themselves. Their food is largely broken-down tissue and especially certain pus-cells. If we could in some way remove all the endamebæ from a pyorrhœa lesion in which they are living and reproducing in their natural way, and not alter the lesion or its contents, we would have left present the most favorable soil for endamebæ. No better soil could be found than that in which the parasite naturally grows. Such soil, consisting of damaged peridental membrane with a little pus formation, microscopic in quantity, perhaps, plus bacteria and food-particles, can be produced in a number of ways.

**Use of the Toothpick Dangerous.**—Picking the

teeth often damages the gum. The soft tissue may be torn away from the tooth sufficiently to cause the flow of a little blood, and if we would examine carefully at once, we might be able to recognize a little wound or pocket by the side of the tooth. It is not necessary, however, that the wound should be large enough to be seen by the unaided eye. A very small wound would be sufficiently large to hold microscopic particles of food, many bacteria, and many endamebæ. Picking the teeth should, therefore, be avoided, and whenever thought absolutely necessary, it should be done with great care not to wound the gums. The instances in which it is necessary to use tooth-picks are extremely few.

**Dental Floss and Rubbers.**—Cleaning the teeth, as is often practised, by passing dental floss, thread, or a rubber strip between them, often damages the tissue at the attachment of the gum to the tooth. Considerable force is sometimes required to pass the cord between the teeth where the crowns are normally in close apposition, and consequently when it does slip through, it strikes the interdental gum with considerable force, often sufficient to wound the soft tissue. Who ever cleaned all his teeth in this way without drawing a little blood? Bleeding always means a break or wound in the tissue. True, such a wound may

be very small, but it is of ample size to furnish lodgment for many endamebæ. We do not believe the use of such means to clean the teeth is advisable, except perhaps in extremely rare instances, when they should be employed with great care. We are fully aware of the fact that this is contrary to the general opinion and advice, but we believe that in the light of present information the latter opinion and advice appear to be incorrect.

Brushing the teeth is often a source of damage

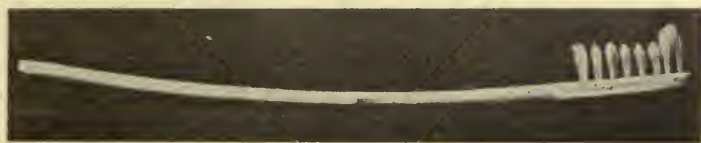


Fig. 40.—An ordinary tooth-brush from which the bristles shown in Fig. 41 were taken.

that should be avoided. The ordinary coarse bristle tooth-brush, when vigorously used, may be quite a source of trauma to the gums. Though the bristles are simply coarse hairs, and appear harmless if we look at them with the unaided eye, whenever examined under considerable magnification with the microscope the end of each one resembles a coarse nail, and many of them very sharp pointed at that. If one presses such a brush against the closed teeth he feels a pricking or sticking sensation at the edge of the gum. True,

it is not very painful. The normal gum is not very sensitive to pain. The sticking sensation is due, however, to the sharp ends of some of the



Fig. 41.—Photomicrograph of bristles taken from the tooth-brush shown in Fig. 40. Could it be possible to brush the teeth (and gums) with such an instrument without damage to the gums?

bristles sticking in the gum, and especially at the line of attachment to the tooth by the peridental membrane. The little wounds made by the bristles are very small,—microscopic, in fact,—

but they are still large enough to furnish a favorable field for the inoculation of several endamebæ if they chanced to be introduced at the proper time. It is true that many such wounds heal very quickly. If one brushes the teeth (and gums) vigorously with such a brush, there are often so very many of such little wounds produced that more or less bleeding occurs.

The finer and softer the bristles of the tooth-brush, the less damage is done, but perhaps it is almost impossible to avoid doing at least a little damage, especially if the brush is drawn across the teeth. Much less damage would be done, it would seem, if the brush is drawn from above downward in brushing the upper teeth and from below upward in brushing the lower teeth.

The frequency with which the teeth are brushed is of some importance. If a certain amount of damage is done each time, then it would be important not to brush the teeth more frequently than is necessary. This leads us to inquire for what purpose the brushing is done. We think of two—to remove or prevent accumulation of tartar consisting of mucus, bacteria, and other substances on the teeth; and to remove food-particles from around and between the teeth.

**Removal of Tartar.**—For the purpose of removing tartar it could hardly be argued that brushing



more frequently than once each day is necessary. The heroic brushing to which many people subject their teeth (and gums) several times each day is hardly warranted for this purpose. It is quite probable that tartar, if soft, can be removed as effectually by rubbing the teeth with a suitable piece of gauze or cloth and without damaging the gums as the brush usually does. Or perhaps some genius will invent some other instrument which will serve this purpose.

**Brushing to remove food** is only partially successful and not necessary. Food in the form of grease and very small particles may adhere to the surface of teeth, and after a meal the teeth feel just a little less smooth on the surface due thereto. A few strokes with the brush remove this and put a cleaner feeling in the mouth than before. Most of the roughness is washed away by the saliva in an hour or two if let alone, or it could be removed with a suitable piece of gauze without injuring the gums, as the ordinary brush does. For the purpose of removing food-particles from between the teeth the most vigorous brushing is a failure. The brush does not reach much of the material it is desired to remove. This can be accomplished much more effectually by thoroughly rinsing the mouth by forcing water between the teeth several times. We therefore question the



advisability of brushing the teeth with a bristle brush for the purpose of removing food-particles. In the event it is employed it should be done with great care.

**Polishing Powders and Pastes.**—Various powders, either as dry powder or incorporated in pastes, are used for the purpose of polishing and cleaning the teeth. Chalk and pumice are the chief ones. Any of them to be effectual must be gritty, but it may be that the powder is so fine that its gritty nature is not appreciated on ordinary examination. Under the microscope, however, such material can be seen to consist of irregularly shaped, sharp-cornered, sharp-edged, and sharp-pointed particles. Power to polish depends upon this. Such material, placed on the ordinary tooth-brush and used moderately, may not injure the enamel of a normal tooth—the enamel is so hard. It is quite different with the soft edge of the gum, which yields much more readily to such grinding or rubbing. A little damage is done, some cells are rubbed away, and perhaps if we could examine microscopically we would find the superficial layer of epithelial cells removed and blood-cells passing out through the damaged tissue. Repair of such damage is so rapid that the harm done ordinarily is not great unless the performance is repeated too often. Some people, in their

zeal to keep their teeth scrupulously clean, grind or damage the gums in this way several times a day. Before the damaged tissue has had time to be repaired by nature's processes it is again subjected



Fig. 42.—Photomicrograph of prepared chalk from one of the common tooth-powders. Imagine brushing the teeth (and gums) with such a brush as that pictured in Figs. 40 and 41, and with this sharp, gritty material on it. Not only would it damage the gum and cause retraction, but it would wear away grooves in the root of the tooth after it is exposed. Most other powders and pastes that are effective "cleansers" contain similar gritty material. This is the chief cause of "erosion."

to the same trauma. By proper, delicate technic we can remove with a suitable small instrument a very small amount of secretion from the edge

of the gum where it is attached to the tooth of an individual who uses these substances vigorously, provided the teeth have not been brushed during the preceding hour or two. Though this material is insignificant in quantity, a microscopic examination shows it to contain many pus-cells. Pus is not thrown off by a normal or unbroken tissue. In addition, we can also often recognize some of these sharp, gritty particles which have been rubbed, as it were, into the soft tissue. Actually the edge of the gum is kept in more or less of an ulcerated, inflamed condition from which pus is being thrown off. Though the extent of the damage may not be sufficiently great to attract attention, or, in fact, to be recognized without microscopic examination, it exists, nevertheless.

This continued grinding and damaging of the gums results in wearing them back slowly, but surely, if sufficiently vigorously practised. Such damaged gums offer favorable soil for *Endamœbæ buccalis*. Tooth-powders and pastes containing powders are therefore harmful, and may be a source of alveolodental pyorrhea. It may be advisable to polish the teeth once in a great while with such preparations, in which instance it should be done with care.

**Erosion of Teeth Caused by Brushing with Powders.**—Though not directly bearing upon

pyorrhœa, it perhaps is permissible for us to point out in connection with powders and pastes containing gritty substances the fact that a very important condition of the teeth, erosion, is produced chiefly by their use. Though the enamel of the tooth is hard and not ordinarily damaged by rubbing with gritty substances, the neck and root exposed from brushing or pyorrhœa are much softer, and vigorous brushing finally grinds away trenches or depressions in the part most exposed. Erosion is caused in this way.

We have on several occasions advised persons to quit the use of pastes, powders, and dentifrices, and in most instances the question was asked, "What must I use to clean my teeth and remove the bad taste?" Most people who try to take care of their teeth use some one or more of the many patent, proprietary, or other preparations as a habit, and imagine that they are very essential. Little do they realize that the bad taste they relieve by their use is the result of damage done by previous use of the same preparation. The amount of money that is worse than wasted in the United States annually for the many harmful preparations of this kind would be astounding if we knew what it was.

**Ill-fitting Dental Work.**—Whenever considerable pressure is made on soft tissue, like the gum,

for a sufficient length of time, inflammation and ulceration result. A crown forced against the gum, as they usually are, soon produces such a condition, especially if the gum is torn away from the tooth to some extent at the time it is put on. Such ulcerated lesion furnishes favorable soil for *Endamœbæ buccalis*. Sometimes the rough edge of a filling extends into the gum, causing inflammation and ulceration. Other ill-fitting dental work may also be a source of damage to the soft tissues, producing tissue subject to infection by *endamebæ*. During our studies of pyorrhea we do not recall having seen a single cap crown that was not irritating the gum more or less. There has been pus at the edge of the gum in contact with the metal in every instance we have examined. We are not informed whether crowns can be put on without doing this damage, but if they cannot, they must be recognized as a fruitful source of harm.

**Salivary Calculi.**—We are in considerable doubt at this time as to the cause of salivary calculi. They either cause irritation, inflammation, and ulceration of the gums, or the disease of the gums produces the deposit. We have not been able to find a single instance where there was not microscopic or macroscopic pus present at the edge of the gum, and in a large percentage of cases end-



amebæ have been found also. The condition of the gum around a calculus is favorable for infection with *Endamebæ buccalis*. Removal or preventing these calculi would, therefore, be a prophylactic measure. Removal by dental instrumentation need not be described here. Oral hygiene—cleanliness—would probably tend to prevent their formation.

**Some Probable Sources of Infection That May be Avoided.**—As has just been pointed out, there are many ways in which lesions favorable for infection with endamebæ may be produced. Some are likely to be produced in spite of care to avoid them. It, therefore, becomes important to avoid as much as possible introducing endamebæ which may find their way into any lesion present, into the mouth. There is no evidence that this species of endamebæ live and reproduce anywhere except in the tissues of man, so far as we know, nor do we believe it at all probable that such is the case. It, therefore, is necessary that infection come either directly or indirectly from another infected individual. During the many years required for the sloughing out of all the teeth, there must be countless millions of endamebæ thrown off with the pus. The pus and endamebæ are mixed with the saliva in the mouth. This infected saliva may be a source of



infection if in any way it is introduced into favorable soil for the endamebæ. So far as we are aware, it is not known whether *Endamœba buccalis* can become encysted and resist drying for long periods of time, but it is known that certain non-pathogenic species of amebæ retain their vitality in a dried state for considerable periods of time. The amebæ cultivatable from hay or road dust are examples. It is believed that cysts of the endamebæ that cause amebic dysentery in man retain their vitality in the dried state for considerable periods of time also.

**Drinking After Others.**—Whenever a person drinks from a cup or other container he touches the sides of the container more or less with the lips. Saliva is always left where the mouth touched. It is quite true that the quantity may be very small in many instances, but it may contain many microscopic endamebæ. If another person drinks and places his lips at the same place, his chances of getting endamebæ on his lips and in his mouth are good. If the endamebæ retain their vitality under such conditions, as we believe highly probable, one might be infected by drinking from a cup or glass that had been infected many hours previously, if not washed between times. Washing removes part or all of the infection, depending upon its thoroughness. Washing with

hot water kills as well as washes away the infection. The public drinking-cup should, of course, be avoided. The common drinking-cup or glass in the home is also dangerous. Endamebæ obtained from the dearest relative can produce the disease just as certainly as those obtained from a stranger.

**Eating After Each Other.**—Eating food that has been bitten by others or using knives, forks, and spoons that have been used by others without proper cleaning may likewise be a source of infection and therefore should be avoided.

**Kissing.**—Often, in kissing, saliva from one individual's lips is transferred to the lips of another. This saliva is quite likely to contain endamebæ. Parents, most of whom have the infection, could possibly infect their children in this way.

**Putting Infected Things in the Mouth.**—Putting in the mouth articles of any sort that have been in the mouths of others is likely to transfer endamebæ and should be avoided. Pencils, money, papers, etc., may be mentioned. It is not unusual to see a parent, nurse, or other older person show a child how to blow a toy or other instrument and immediately return it, infected, to the child to place in his mouth. Nearly all such older people have pyorrhea in some stage, and therefore endamebæ in their saliva. Such things should be

avoided, and children should be taught as early as possible not to put things in their mouths that had been in the mouths of others. Nearest relatives and friends are not to be excepted.

A possible way of introducing endamebæ into the mouth is putting things that have been contaminated indirectly into the mouth. For instance, some person having a bad form of pyorrhœa may in some way contaminate his own hands. With them he may handle articles of various sorts, which in turn are soon handled by some other individual. The latter may place his hands on the same place that had been contaminated and thus contaminate his own hands. It is true that the amount of saliva containing endamebæ thus transferred may be very small, but during the course of a few hours, especially in public places, a person would stand a chance to touch several contaminated articles. He may now introduce the contamination into his mouth in a number of ways. Many people put their hands or fingers to their lips or tongue for various purposes, or often for no purpose, many times during each day. Much of this is bad habit and could be avoided.

**Eating with Unclean Hands.**—The chances of contaminating the hands in one way or another are so great that they may be presumed to be contaminated at most any time unless they have

been recently well washed. It is not uncommon for people to go to the table and eat their regular meals without washing their hands, to say nothing of handling and eating various things between meals. Such habits should be discontinued.

**Spitting.**—Sputum consists of saliva and secretion from the mouth and the respiratory tract. When pyorrhea exists, there are more or less endamebæ contained in it. In some cases there must be very large numbers. In fact, Smith, Middleton, and Barrett have recently found *Endamœba buccalis* present in the tonsils in a considerable percentage of cases of chronic tonsillitis. The possibility of such material becoming dried, made into dust, and this transferred through various agencies to the mouth of others, thus being a source of infection, is probably not great, but may be thought of.

**Local Use of Emetin as a Prophylactic.**—In our early experimental work on pyorrhea we recognized the necessity of preventing reinfection in cases whose endamebæ had been destroyed by the specific treatment. Otherwise, reinfection would surely occur long before the lesions in most cases could heal. We naturally selected emetin as the drug to be applied locally for this purpose. On account of the cost of the alkaloid at that time, we thought it would be more economic and prac-

tical to employ the fluidextract of ipecac containing the emetin instead of the pure emetin. The method of applying it was to have the patient apply one drop of the fluidextract of ipecac to the wet tooth-brush and brush the teeth with it after first having brushed them to clean them. The patient was instructed to force the solution of ipecac formed in the mouth between the teeth, and to spit out the excess, but not to rinse it out. Our idea was that the ipecac probably would kill endamebæ that may have been introduced through the day, but that were located only superficially in the diseased tissue. To determine the efficiency of this to destroy superficially located endamebæ we tried it on a number of patients who had shallow lesions with endamebæ. In a number of them, to our great satisfaction, the endamebæ disappeared from shallow lesions in a few days and the lesions rapidly healed. This was without any other treatment. If the local use of ipecac in this way destroys endamebæ in superficial lesions of pyorrhea, it would more certainly destroy those recently introduced but not established.

Fluidextract of ipecac in sufficient concentration causes some irritation and soreness of the mucous membrane. Many patients, when instructed to use one drop, think that if that small amount will do good more will do more good, and



therefore use enough to make their mouth sore. A more practical method of application that we have recently employed is to instruct the patient to rinse the mouth with water to half a glassful of which one or two drops of fluidextract of ipecac has been added. The amount of ipecac present is even much more than necessary to kill all endamebæ with which it comes in contact.

More recently we have employed a solution of emetin hydrochlorid in water instead of the ipecac. The prescription usually given the patient is:

℞. Emetin hydrochlorid..... 5 grains  
Distilled water ..... 1 ounce

Mix. Dispense in sprinkler-top bottle.

*Label.*—Put one or two drops in one-fourth glass water. Use as a mouth-wash.

The five grains of emetin used in this prescription cost the druggist at present prices about 50 cents, and the quantity should last for at least one year, if used daily. The best way to use it is to take some of the dilute solution in the mouth and force it back and forth between the teeth. Spit it out and repeat. The object is to wash out anything that may be lodged between the teeth and to leave a trace of the solution of emetin where it is likely to reach any endamebæ that may have recently been introduced.

The question may be asked, why prescribe the drug in concentrated form and not much more



diluted? The reply is that there is no necessity for the patient to buy the diluent when he can use water, which is probably the best and costs nothing. It has also been suggested to combine the emetin or ipecac with other more pleasant dentifrices, pastes, powders, etc. It is our opinion that they are valueless, and often harmful, and that the after-effect of the emetin is fully as pleasant as any of them. The taste is bitter at first, but it is not an unpleasant bitter, and soon passes away. We have had several persons to tell us that it produces the most pleasant effect of any preparation they ever used, and that they never had such a pleasant, clean taste in the mouth as is produced by the use of emetin or ipecac. In the light of our present information, ipecac or, preferably, emetin, should be used as a mouth-wash once every day or two for prophylaxis. It may be that future work may show that some other method of using it is better than that here suggested, but this is practical and efficient.

#### SUMMARY

Endamœbæ buccalis cannot attack normal tissue. It must be damaged. Damaged tissue, constituting favorable soil for endamebæ, may be produced by hard particles of food forced against the gum, picking the teeth, vigorous brushing,

pressure of ill-fitting crowns, and collections of tartar and bacteria, etc. These should be avoided.

The source of infection is pus and saliva from the mouth of some other person, most all of whom are infected. Drinking and eating after others should be avoided; also putting anything not known to be free from contamination into the mouth.

Rinse the mouth once a day (at night) with a solution of fluidextract of ipecac or of emetin by forcing the solution back and forth between the teeth. The solution should be made up just before use by adding one or two drops of fluidextract of ipecac or one or two drops of a solution of 5 grains of emetin in one ounce of water, to half a glass of water.

If ipecac or emetin was used in this manner from childhood through life, it is doubtful whether the individual would ever get the disease.

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