DENTAL SEPSIS, ITS NATURE AND SYSTEMIC EFFECTS.

By Captain S. H. Woods.

The Army Dental Corps.

INTRODUCTION.

One of the greatest advances in medicine of the present century is the recognition of the mouth as a focus of infection.

To William Hunter, of London, belongs the credit of having aroused the medical profession to the importance of dental sepsis. In 1900, he said, "It is not the absence of teeth but the presence of sepsis; it is not dental defects per se but chronic septic poisoning; it is not defective mastication but effective sepsis; it is not accumulation of fermenting food debris between teeth, but the presence of virulent streptococcal sepsis in open wounds in the gums and sockets, teeth and bone, that underlie the ill-health so frequently associated with bad mouths."

Mitchell Bruce, in his presidential address to the British Medical Association in 1910, said of oral sepsis: "Its effects are so widespread, so multiple and frequently so grave as to make us ashamed of our previous blindness to a common source of blood infection staring us in the face all these years. And how many years, be it added! The last discovered fossil skull found in Rhodesia, at the Broken Hill Mine, showed widespread dental caries with evidence of extensive alveolar abscesses, and associated was a condition of the upper end of the tibia suggesting chronic arthritis. This skull is considered as belonging to the Pleistocene Period."

In the early days of this movement somewhat extravagant claims were made, and there was a general tendency to condemn teeth as the cause of all sorts of systemic conditions, on insufficient evidence and, it must be confessed, on insufficient knowledge.

On the one hand, the extremist physician advocated wholesale extraction of teeth in order to eradicate at the outset any possibility of dental sepsis, while on the other hand the extremist dentist endeavoured to save every possible tooth for his patient, however diseased it was. But soon the moderates of each side tended to co-operate in the interests of the patient, and gradually evidence was gathered tending to show the undoubted relationship of dental to systemic disease.

The recent great increase in the knowledge of dental pathology brought about by the development of dental radiography, coupled with intensive research into the histology of the tissues, broadened enormously the point of view of the dental profession. At the same time, the tendency of medicine along the line of preventive treatment stimulated increased interest in the elimination of those hitherto unsuspected areas of periapical bone infection which the X-rays revealed.
This brought about a closer co-operation and a more rational attack on the problem, with encouraging results. But the whole problem is still far from solution and presents peculiar difficulties, not the least of which is an imperfect knowledge by the general medical practitioner of the nature of dental sepsis.

When it is considered that every adult mouth not subject to frequent examination and treatment presents some degree of obvious sepsis—a point Medical Inspectors of Recruits will appreciate—and also that a considerable degree of quite unsuspected latent sepsis may exist in a mouth periodically treated, the importance of the problem cannot be exaggerated. The mouth is not to be regarded as a sort of watertight compartment outside the body from which no leakage can take place into the system, and of importance only to the dental surgeon. It does most certainly concern the modern physician who endeavours to investigate and remove every possible focus of infection which may be associated with the condition under treatment.

It is essential for him to have some idea of the nature and possible extent of those septic and infective conditions of the dental tissues embraced in the comprehensive term "dental sepsis."

In the words of Sir William Willcox, "There is no problem in medicine for which careful scientific investigation and well-balanced judgment are more required. To solve this problem, it is essential that there should be the closest co-operation between dental surgeon, physician, radiologist, bacteriologist and all associated with the investigation."

Such co-operation in the Army has only become possible since the formation of The Army Dental Corps in 1921 and the posting of dental officers to the large military hospitals. It has been my good fortune during the last three years to be stationed at Queen Alexandra Military Hospital, Millbank, London, where an ideal co-operation has existed from the outset, resulting in an increasing number of hospital patients being sent by the various departments and wards for the elimination of dental sepsis.

The experience gained in this connexion has prompted me to write this paper, and though the cases recorded are few and the investigation very limited, it is hoped this article, however incomplete, may be of some interest.

It is written primarily for medical officers and purely from the standpoint of Army practice, and attempts only to serve as a general introduction to the problem, concluding with a consideration of the question with particular reference to the Army.

It attempts to answer three questions:

1. What is the nature of "dental sepsis"?
2. What are the factors governing its systemic effects?
3. What are the systemic lesions produced?

Each question embraces a vast field, and it is only possible to touch on essential points in each.

Dental Board of the United Kingdom Lectures, "Systemic Effects," 1923.
The paper is arranged as follows:—

Section I.
Subdivision (a) Anatomy
    ,, (b) Pathology
    ,, (c) Bacteriology

To answer Question (1).

Section II.
Subdivision (a) Factors governing effects Questions (2) and (3) treated
    ,, (b) The systemic effects

SECTION I.—SUBDIVISION (a)

ANATOMY AND HISTOLOGY.

While the normal appearances of the teeth and gums are well known, it is essential to draw attention to some important points in dental anatomy and histology.

Dental tissues are divided into:—

(A) Hard Structures.—Tooth, alveolar bone, bone of jaw.
(B) Soft Structures.—Gum, periodontal membrane, pulp.

(A) Hard Structures.

A tooth is divided into:—

Crown, that part normally above gum and composed chiefly of dentine covered by enamel.

Root or roots, normally hidden in bony socket and composed of dentine covered by cementum.

Neck, the junction of crown and root, normally covered by the gum margin.

The only part of the tooth which we need consider in some detail is cementum, which is the smooth, yellowish-white and very hard substance covering the root.

Cementum is similar to bone in structure, is arranged in concentric laminae around the dentine and contains cementoblasts, contained in lacunæ, with long processes, lodged in canaliculæ, running towards the exterior. These cells intercommunicate and cementum is therefore permeated by a living protoplasmic network. It contains no blood-vessels.

Alveolar bone is the socket or alveolus in which the tooth is embedded and consists of cancellous bone lined by a thin layer of dense compact bone. It is a special structure dependent upon the presence of teeth, and when a tooth is extracted it is this bone which shrinks and disappears by a process of absorption.

The spongy nature of alveolus is beautifully indicated by the odontogram, in which it appears as a fine interlacing network, and the compact bone of the lining as a continuous thin, white line round the root.

The force exerted during extraction is inclined to give a totally false idea of the density of alveolus and its open texture must be borne in mind. The strength of alveolus depends purely on the arrangement of its lamellæ.
which are placed at angles to one another. Each lamella has a covering of endosteum, the cells of which have the power to form new bone or destroy that already present, depending on the stimulus reaching them.

Maxillary alveolus is much more spongy than mandibular, particularly in the molar region, hence it follows that infection spreads more rapidly and extensively in the former.

Bone of jaw is similar to alveolus, with which it is directly continuous. Here, also, the main body of the maxilla is more spongy than that of the mandible.

(B) Soft Structures.

Gum covers the alveolus and is continuous with the mucous membrane of the mouth, from which it differs chiefly by its greater density. It consists of a dense submucous layer, closely attached to the underlying bone by tendinous fasciculi and a mucous membrane of many layers of squamous epithelium. It is pale pink, firm, free of tenderness and is designed to withstand very considerable pressure.
The triangular spaces between teeth, just under their points of contact, are entirely filled in by gum known as the "interdental papillae."

The part of the gum which embraces the neck is known as the "cervical gum" or "gum margin," and plays a most important part in the pathology of dental sepsis. It is not attached flush to the tooth but in such a manner as to leave a slight V-shaped depression round it, about one-eighth inch in depth, known as the "gingival trough." The floor and outer wall of this depression are covered by epithelium which forms a smooth lining and is reflected upon the tooth, thus effectively sealing the trough and shutting off the periodontal membrane from the fluids of the mouth.

![Diagram](Fig. 2.—High-power photomicrograph, showing—\(a\), cement cell; \(b\), epithelial cell-nests; \(c\), cementum; \(d\), dentine; \(c\) \& \(f\), cemental fibres, starting near \(b\), and running in between \(a\) to enter \(c\); \(p\) \& \(m\), periodontal membrane.)

This epithelial lining decreases in thickness from gum margin towards the floor and fades off into a thin squamous layer at the apex of the V, thus presenting an area of lesser resistance at this site (fig. 1).

Normally the gum margin is very closely applied to tooth and the gingival trough must be regarded as a potential space only. This will be readily appreciated if one tries to pass the point of a fine probe between normal tooth and gum. Allowing 1 inch as the average circumference of the thirty-two teeth in the mouth and \(\frac{1}{2}\) inch as the average depth of the troughs, it will be seen that their area in health is 4 square inches.

The periodontal membrane is a thin single membrane which invariably intervenes between tooth and alveolus and is directly continuous with the
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gum margin immediately under the gingival trough. It must be clearly emphasized here that the main source of infection from the mouth is the periodontal membrane, and it is essential to study it in some detail, as the whole problem of dental sepsis is centred in this tissue.

Particular attention must be focused on (a) the nature and arrangement of its fibres; (b) its lymphatic system; (c) its cells.

(a) Fibres. The periodontal membrane is chiefly composed of bundles of white fibrous connective tissue, entirely non-elastic, which run transversely through it and pass right into the substance of bone and cementum, the portions so contained in these tissues being known as "bone" and "cement" fibres respectively (fig. 2, c f).

![Diagram showing the structure of the periodontal membrane.](http://militaryhealth.bmj.com)

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The fibres run in an oblique upward direction from tooth to bone except round the neck, where they assume a very definite and regular arrangement. Here they are densely packed together and run horizontally across from tooth to bone, and some arch over the alveolar crest to unite with its periosteum. This dense horizontal band of fibres which embraces the neck of each tooth is known as the "circular ligament," and is immediately under the gingival trough (fig. 3).

The oblique fibres suspend the tooth in its socket, allowing of a very limited movement and preventing undue pressure on the apical vessels.
The circular ligament is protective to the underlying part of the periodontal membrane and also acts as a strong ligament.

The bone and cement fibres penetrate the hard tissues in more or less parallel lines and are lodged in distinct channels in them.

The periodontal membrane is more strongly attached to tooth than alveolus and is always removed with it on extraction.

It is important to gain some idea of the area of the periodontal membrane when stripped off the tooth. Fig. 4 indicates the exact surface area of the different teeth, a point which will be considered in the section on pathology.

The periodontal membrane is thicker just around the apex than elsewhere,

![Diagram of teeth showing surface area](image)

**Fig. 4.** Exact surface area of average tooth from the gum margins to the apices.

this thickening or pad acting as a shock-absorber. In the radiogram the membrane is indicated as a very thin continuous black line between cementum and bone, and shows a slight increase in width at the apex, where the tissue is thickest.

(b) The Lymphatic System.—Up to 1907 no lymphatics in the periodontal membrane had been demonstrated. It was presumed there must be a lymphatic system similar to that found elsewhere in the body. In 1907 it was shown that a wreath of lymphatics surrounds the neck of each tooth, just above the circular ligament.

In 1918, by means of the Prussian blue injection method, the complete lymph system in gum and membrane was demonstrated for the first time, thus filling a large gap in our knowledge of the functions of the

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1 From "Pyorrhoea Alveolaris." D. A. Crow.
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periodontal membrane. It was shown that the wreath-like system just described is continued through the circular ligament and passes down the membrane to the region of the apex, where it joins the lymphatics from the pulp and then anastomoses with the lymphatic network in the medullary spaces of the bone.

(c) The Cells.—There are three kinds, each playing a different part in the pathology, and may be classified as follows:—

1. Cement cells (fig. 2, a), in contact with cementum.
2. Bone cells, similar to osteoblasts, in contact with alveolus.
3. Epithelial cell-nests, scattered here and there between 1 and 2, chiefly round the apical region.

These are regarded as remains of an embryonic membrane present in the early stages of calcification (fig. 2, b). The periodontal membrane is directly continuous with the pulp at the apical foramen and has a rich blood supply, the vessels passing down its centre.

The pulp is the soft vascular tissue which is enclosed by dentine, and corresponds more or less to the shape of the tooth. It is directly continuous with the periodontal membrane at the apical foramen, through which enter its vessels and nerves.

Its periphery is lined by special cells, each of which sends off long processes which penetrate the entire thickness of dentine and anastomose with cells at the junction of dentine and cementum. We have already noticed the anastomotic network in the latter. This is continued through the periodontal membrane, via the cement cells, then by ordinary connective-tissue cells to the bone cells lining the alveolus. Thus there is direct protoplasmic continuity from pulp through dentine, cementum, periodontal membrane and alveolus, a point to be borne in mind.

SECTION I.—SUBDIVISION (b).

Pathology.

Those acute conditions which, by their marked clinical signs and symptoms, demand and receive early attention play no part here. I refer to acute alveolar abscess, acute ulcerative gingivitis, and any of the usual conditions for which the soldier reports "sick" and receives attention.

We are concerned only with those periodontal infections which give rise—at any rate in their earlier stages—to no marked local sign or symptom, and which may be present for long periods without causing pain.

It is only possible, within the limits of this paper, to treat this section in a general manner, and to indicate the extent of the tissue reaction following infection of the periodontal membrane.

This infection takes place in two ways:—

1. Externally, via the gingival trough.
2. Internally, via the apical foramen.
(1) The External Infection of the Periodontal Membrane.

We have noticed that the epithelium lining the gingival trough effectively seals off the periodontal membrane from the oral fluids. In addition, it is believed on strong evidence that the trough is normally sterile, leucocytes being found in it in abundance, which act as phagocytes, taking up and ingesting any organisms finding their way into this space.

Any injury to the gum margin not healing by resolution leads to a breach in its epithelium, followed by infection by the ever-present bacteria which swarm on its surface, resulting in a destructive inflammation at the site of injury.

The soft wall of the gingival trough is directly involved, becomes hyperemic, swells away from the tooth and thus converts the potential space into an actual one. Its smooth epithelial lining is broken, and at once the underlying periodontal membrane is open to infection by the organisms which are swept into the breach by the saliva (fig. 5).

Whatever the exciting cause may be, these early changes are constant, and of the numerous causes of the initial injury it is only necessary to consider three:

(a) Impaction of food debris.
(b) Deposits of salivary calculus (tartar).
(c) Overhanging edges of fillings and crowns.
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(a) Food Debris.—This occurs most frequently in the premolar and molar region, where the large contact area is more likely to hold strands of meat, fish and fruit than the much smaller and self-cleansing points of contact of the incisors.

A stagnation area is set up immediately around the site of injury, followed by a low-grade inflammation and slow destruction of the periodontal membrane in a cup-shaped manner, forming pockets. The crest of alveolus is involved and absorbed by the osteoclastic action of its bone cells, and the condition results in a destruction of the investing tissues of the tooth around the site of injury.

When both sides of a premolar or molar are involved, the tooth becomes progressively loose after a time, the infection encroaches on the apical portion of the periodontal membrane, the pulp is involved and changes are started in it which lead to its slow death.

Pus is generally absent or only present in small quantity, and, apart from the discomfort of food impaction, there may be no pain and the crowns may be free of caries.

From the onset of the condition, the toxins are absorbed directly via the lymphatics, and as several teeth are often involved, the toxin-forming area may be extensive (fig. 4). On extraction the periodontal membrane will have an offensive odour, and if it be scraped off the root and the latter sterilized externally by passing it through a flame, the tooth substance and pulp will be found equally offensive when cracked open.

(b) Deposits of Calculus.—Tartar is of two varieties, superficial and subgingival. Superficial tartar is deposited at the gum margin and is confined chiefly to the lingual surface of the lower incisors and the buccal surface of the upper molars. If removed at frequent intervals it causes no harm, but if allowed to increase over a long period it causes extensive damage to the gingival troughs and leads to stagnation areas with subsequent infection of the periodontal membrane, and sequelae similar to those just described.

Subgingival tartar is always pathological, is not obvious and is found round any tooth subject to a marginal gingivitis. It is hard and dark, sometimes forming a ring round the tooth, and always causes a local injury to the gingival trough in contact with it.

It is very frequently found on the lingual surfaces of lower molars and the upper incisors, areas which are often untouched by the toothbrush and in which considerable destruction of investing tissues may result. If not removed, destruction of periodontal membrane progresses and the condition resembles that of impaction of food debris with similar results.

It has been noted that the potential area of the gingival troughs is four square inches, and while a deposit of tartar involving all this area is necessarily rare, that involving one square inch or more is quite common, and as it persists for long periods the amount of toxic absorption via the lymphatics may be considerable.
(c) Overhanging Edges of Fillings and Crowns.—We have just seen the results of local injury to the gingival troughs and can readily appreciate the sequelae of any filling leaving an edge below the gum margin.

The neck is the most constricted part of the crown and great care is needed to restore the exact contour in this region. While the edge of a filling or inlay is limited to a portion of the neck, that of a crown at once involves the whole circumference of the gingival trough. The crown plays such an important part in dental sepsis that it must be considered in detail.

Crowns are of two kinds: (1) Collar; (2) Flush.

The collar crown is essentially a metal cap, which embraces the tooth at the neck and is designed to render serviceable a tooth which is too broken down by caries or injury for conservation by the usual means. Theoretically, it should be so shaped and the root so prepared in the form of a truncated cone that the collar should pass just under the gum margin without causing direct pressure on the floor of the gingival trough. However carefully this fitting is done, it is obvious the edge of metal is a foreign body in the trough and that a stagnation area will result under its edge followed by the usual pathological changes in the investing tissues.

But it must be confessed that the great majority of such crowns I have seen in the mouths of officers during the last four years show little, if anything, of this careful adjustment of collar or preparation of root and, quite apart from the apical condition of the teeth, the cervical tissues in every case showed marked sepsis.

The overhanging edges of collar crowns are clearly shown in radiograms, and I have a large number exhibiting every degree of approximation of
metal and tooth, some indeed showing the extreme toleration of the tissues to ill-treatment (fig. 6).

The flush crown is a porcelain tooth which is ground to fit flush with the previously prepared root, and here it is possible to obtain a very close adjustment with hardly a perceptible edge. It is kept in place by a metal post which is cemented into the root canal.

Fig. 7.—Section showing initial stage in pyorrhea. A, Breach of epithelium of gum margin; T, deposit of tartar in gingival trough; L, infiltration of gum with leucocytes; G, normal gum epithelium; D, dentine; C, cementum; P. memb., periodontal membrane; B, normal crest of alveolus.

A variation is a porcelain facing to which a metal backing is cast. These gold-backed crowns are frequently mistaken by medical officers for the collar gold crown.

They are usually found on the anterior teeth and occasionally on the bicuspsids. However close the fit to the root, the crown must be adjusted below the gum margin and, theoretically, without causing damage to the gingival trough. If left above the gum the enamel would decay, so it is entirely removed.
FIG. 8.—This section shows that the inflammatory process proceeds from the periphery, namely, from gum towards the bone. i, Gum infiltrated with leucocytes; c, normal gum; y, normal bone.

FIG. 9.—In this section the implication of the bone in the disease is shown. i, Infiltration approaching from the gum towards the bone; s, bone transformed into fibrous intervening tissue; o, osteoid tissue; d, bone partially decalcified; n, bone corpuscles; l, line dividing the healthy from the unhealthy bone; v, periodontal membrane; D, dentine.
Chronic marginal gingivitis is very common round such crowns, which are most frequently fitted to teeth previously septic, and will be considered again in relation to root treatment.

Collar crowns are not made in Army dental practice, except in the special circumstance of absolute necessity for the retention of splints and fragments in gun-shot wounds of jaw with loss of substance.

We have so far examined purely local conditions confined to individual teeth and have indicated the possible extent of the toxin-forming surface of the periodontal membrane in each case.

We must now consider that general suppurrative periodontal infection known as "pyorrhea" which, by virtue of its extent and chronicity, is responsible for so much widespread ill-health.

*Pyorrhea alveolaris* embraces a series of connected pathological processes, and for the purpose of this paper may be regarded as a local periodontal infection of a suppurative character, affecting several or all the teeth in the mouth, the initial cause being injury to the cervical gum by food debris and due to the character of the diet of the present day.

The earliest changes in the gingival trough are similar to those already
described, but the subsequent pathology\(^\text{1}\) is different and may be summarized thus:

1. The periodontal membrane is slowly destroyed in spite of its attempt to form a barricade of granulation tissue. The circular ligament disappears and the crest of the alveolus is involved (figs. 7, 8).

2. This crest of bone is first decalcified, then transformed into an osteoid tissue and subsequently into a fibrous intervening tissue (figs. 9, 10).

3. The deeper portions of alveolus containing bone-marrow are rapidly absorbed by a process of rarefying osteitis, in which "giant cells" are very active (fig. 11).

4. Bone infection is not limited to that in contact with periodontal membrane, but spreads laterally and in depth, involving the external and internal alveolar plates and even the body of the maxilla and mandible, producing not an area but a cubical content of infected bone (fig. 12).

These pathological changes are very clearly indicated in figs. 7 to 12, which present the earliest, intermediate and last conditions as a connected series.

Concurrently with these changes outside the tooth, the cementum is infected from the periphery inwards, and this infection spreads into the dentine via the dentinal processes. The pulps of teeth involved are always affected, and the apices of such teeth are translucent when held up to a bright light. Bone recession precedes gum recession, the latter being

\[^{1}\text{J. F. Colyer, "Dental Surgery and Pathology," condensed from pp. 531, 532.}\]
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no indication of the extent of the former, which is often surprisingly extensive, even in young adults. Pockets are thus formed, from which a free flow of pus is obtained, but it must be noted that the disease may have progressed very extensively before this classical symptom is evident.

Pyorrhoea thus involves four structures, viz., periodontal membrane, gum, bone and tooth, from each of which bacteria and toxins are absorbed into the system.

![Diagram of jaw and investing tissues](http://militaryhealth.bmj.com/)

(By courtesy of the "Lancet.")

(From a drawing by Mr. A. Hopewell-Smith.) Showing extensive changes in bone of jaw and investing tissues.

Fig. 12.—A, Pulp cavity; B, dentine of tooth; C, hyperplastic cementum around apex of root; D, periodontal membrane, greatly thickened—hyperplastic; E, indifferent tissue at apical region greatly increased in amount; F, free edge of bone of socket becoming converted into fibrous intervening tissue; G, bone of socket presenting earliest signs of osteoporosis; H, large osteoporotic space in bone of jaw filled with bone-marrow; I, bone of socket partially decalcified and converted into osteoid tissue; J, junction of living with decalcified bone; K, osteoclasts producing lacunar absorption; L, bone of jaw only slightly altered by disease; M, sequestrum undergoing peripheral absorption; N, soft, cancellous tissue slightly changed from normal; O, inflammation of gum at neck of tooth.

It is important to obtain some idea of the pus-forming area. In an advanced case involving thirty-two teeth, it has been estimated that the periodontal membrane area, allowing for loss in early stages, is twenty square inches. While such a condition is rare, frequently ten or fifteen
teeth are involved, presenting an area from seven to ten square inches. The surface area of infected alveolus is approximately the same, but here we must remember that the infection produces a cubical content of infected bone many times greater than the roots of the teeth involved.

The free margin of infected gum also presents an extensive pus-forming area, and the histamins from the infected pulps of teeth are absorbed via their lymphatics.

While there is a free flow of pus into the mouth, it must be emphasized that a considerable volume of toxins and bacterial products is absorbed via the lymphatics of the affected tissues and particularly from the deeper medullary spaces of the bone.

When it is considered that the disease may be present for long periods, it is not astonishing that so much general ill-health has been attributed to it.

(2) The Internal Path of Infection of the Periodontal Membrane.

This implies direct infection of the periodontal membrane via the apical foramen, following sepsis or injury of the pulp.

The pathological changes in the membrane and contact tissues following such internal infection are either (a) productive, or (b) destructive.

(a) Productive Changes.—There are three, viz., cementosis, abscess sac, granuloma.

Cementosis is an increased production of cement due to the cementoblastic reaction of the cement cells (fig. 2, a) produced by the histamins or toxins from the periapical infection.

This thickening of cementum is indicated in the radiogram when it is often quite unsuspected, and is an important symptom of chronic periodontal irritation.

Abscess Sac.—When suppuration occurs on the inner surface of the periodontal membrane, it may gradually expand the latter and absorb the bone, thus forming a sac. The inner surface is lined by granulation tissue and its wall gradually thickens and becomes more fibrous. It most frequently remains attached to the tooth on extraction and is found chiefly at the apical region. It is not uncommon, and I have over twenty specimens, most of which were the size of a small pea, in the recent state.

Granuloma.—This differs from the sac in structure, consisting of a solid mass of granulation tissue, and is regarded as a proliferation of the epithelial cell nests (fig. 2, b). Controversy rages as to the pathogenicity of the sac and granuloma. The odontogram, while indicating a periapical bone absorption, gives no hint as to the nature of the soft structure which is contained in the dark area between tooth and bone. The present opinion regards the sac, at any rate, as a highly pathogenic lesion from which toxins and bacteria may be absorbed by the lymphatics.

Destructive Changes.—Chiefly absorption of the root by a process of rarefying periodontitis, produced by the cementoclastic reaction of the
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cement cells. A considerable part of the root may thus be destroyed, the absorption being either smooth and continuous, or rough and irregular.

While caries is not dealt with in this paper, we must briefly consider the question of the treatment of teeth with putrid pulps resulting from pulp infection due to caries. Such a tooth is commonly known as "septic," and presents a cavity filled with food debris, under which is a putrid pulp chamber and root canal filled with necrotic matter, this condition usually being present for some time before treatment is begun, and necessarily involving the periodontal membrane. It offers a challenge to the dental surgeon, and he is loth to extract it, and until very recently the usual routine treatment carried out was to remove all caries and infected material from the body of the tooth, enlarge the root canals by suitable instruments to remove infected dentine, and seal into the cavity powerful formalin dressings which were changed at frequent intervals. When the last dressing was considered to be free from any abnormal odour, the tooth was judged ready for conservation.

It must be clearly emphasized here that such infected teeth which have received this treatment are the most potent source of systemic infection from the mouth. Such an unscientific guide as the olfactory sense is now recognized as hopelessly inadequate, and every possible avenue is being explored in the attempt to render such treatment more satisfactory.

Odontograms are taken before and during treatment; root dressings are bacteriologically examined until the last one gives no growth on culture media; the apices are sealed up as far as improved technique allows and checked by radiograms.

The non-irritating dichloramine T and acriflavine compounds are being tried in root treatment, and a few enthusiasts are experimenting with the lactic acid Bacillus bulgaricus, on account of its markedly acid reaction which destroys the streptococci in the septic canals while itself remaining non-pathogenic. This last treatment is an attempt to use some innocuous organism in place of those antiseptics which are powerful destructive irritants.

It cannot be said that dental surgeons are not alive to the seriousness of the attack by the medical profession on root treatment, but they are loth to sacrifice every septic tooth to the forceps. But the very nature of the tissues and their inaccessibility render direct sterilization impossible. Both hard and soft tissues are deeply impregnated by bacteria and their toxins, as the result of their protoplasmic continuity, and after every possible bacteriological and radiological test has been carried out, it cannot be said definitely that there is not some residual infected hard and soft tissue.

The position is summarized thus by Price and Mouldenhauer, after an exhaustive research into the possible sterilization of septic teeth:

1 (1) "Infected dentine and cementum are not readily sterilized by

Dental Cosmos, October, 1919.
medication even when the amount of medication is largely in excess of the mass of tooth structure.

(2) "The sterilizing agents contained in root dressings very readily and rapidly lose their disinfecting power.

(3) "The most efficient agents, silver nitrate and formalin, are very objectionable, the former by its discoloration and the latter by its destructive and irritating properties.

(4) "The results show that an exceedingly small percentage of teeth that have been treated by the dental profession in the past have actually been sterilized. Nature is, and has always been, very tolerant, and much of the credit that has been given to and taken by the dental public for sterilization of infected roots has been due to Nature for her kindness in tolerating, at least without local irritation, what is probably an almost universal condition of infection."

It may be argued that there are tens of thousands of such treated teeth in the mouths of people who show no constitutional symptoms. This will be granted, but they are potential foci of infection from which toxins are directly absorbed by the lymphatics, and preventive medicine rightly demands their eradication.

When it is remembered that practically every crown—collar or flush—is fitted to such a potential or actual focus of infection, it will be realized that such means of conservation is but an insult to injury and cannot be too strongly condemned.

It may be of interest here to record that, during the last three years at Millbank, I have extracted 180 collar crowns and thirty-nine flush crowns from officers who were either hospital patients or otherwise receiving medical attention for some disability, and also twenty-eight collar crowns from R.A.M.C. officers at their own request, a most encouraging sign of their appreciation of the dangers of such treatment.

In over 90 per cent of these teeth the local symptoms of an infected periodontal membrane were obvious, and in every case both the exterior and interior of tooth were foul-smelling. There is no better way of convincing the patient that such teeth are definitely harmful than by the test of his olfactory sense.

Mention must briefly be made also of the vicious circle set up by the gingivitis so frequently resulting from systemic diseases, such as:

(1) Acute infections, as in the enteric group, cholera, dysentery, acute rheumatism, malaria.

(2) Nutritional disorders, as gout, diabetes, chronic rheumatism, scurvy, anæmias.

(3) Those resulting from the use of toxic drugs, such as mercury, lead, iodides.

In all these conditions there is some degree of gingivitis which leads to loss of gum margin and its sequelæ, opening up the periodontal membrane to infection. This is particularly the case in the acute conditions men-
tioned above. The marked decrease in quantity of saliva which invariably occurs, aided by the stagnation of food particles round the teeth and the much lowered tissue resistance, render the gum margins very susceptible to infection, and an extensive and sometimes acute gingivitis is set up during the illness.

On recovery, it is discovered by the patient that his gums have retracted, are soft and tender, and that food impacts between the teeth, setting up a vicious circle, the effects of which we have noticed. I have seen an extensive loss of gum, periodontal membrane and crest of alveolus resulting from an attack of typhoid fever of six weeks' duration, and simulating a chronic generalized pyorrhoea of many years' standing.

The importance of this gingivitis is that the tissues destroyed are never replaced in their entirety, and that it is only a question of time before a true pyorrhoea is established.

Of the acute oral conditions for which the soldier often seeks the medical officer's advice, two must be mentioned in particular. Firstly, the acute alveolar abscess, which, if untreated, "goes down" in due course and leaves a chronic sinus which may or may not give rise to local symptoms, but, nevertheless, is constantly discharging pus into the mouth, while at the same time some of its toxins are absorbed by the lymphatics of the invisible infected tissues. Secondly, the acute specific ulcerative gingivitis known as "Vincent's angina" or "trench mouth," which, if left untreated, may eventually be overcome by the barrier of granulation tissue set up by the gum and periodontal membrane, but which most commonly causes extensive destruction of the investing tissues before this occurs, and here dental sepsis is fully and rapidly established.

(To be continued.)