Section of Odontology

President—W. WARWICK JAMES, O.B.E., F.R.C.S., L.D.S.E.

[*March* 19, 1937]

Local and Remote Sequelæ of Infection in the Parodontal Sulcus

By E. WILFRED FISH, M.D., D.D.Sc., D.Sc.

(*From the John Hampton Hale Research Laboratory, Royal Dental Hospital and School of Dental Surgery, London*)

ABSTRACT.—(1) It is shown in this paper that the infecting organisms in the parodontal sulcus are confined to the surface, but may be forced into the blood and lymph stream by traumatic interference.

2. Local and general disturbances may therefore arise both as a result of the absorption of soluble toxic matter from the pocket into the tissues and also as a result of this traumatic introduction of organisms into the blood-stream.

3. The effect of toxic absorption on the local tissues is destruction of the attachment of the tooth—pyorrhœa.

4. The remote effects of toxic absorption may be a similar destruction of the connective tissue generally—arthritis and fibrositis. The liver and kidney may suffer since they excrete the toxic matter, and other susceptible tissues may also be involved.

5. Traumatic bacteræmia may produce local bone necrosis or "dry" socket. Acute suppurative lymphangitis produces the "pyorrhœtic abscess".

6. The remote effect of bacteræmia may be to produce osteomyelitis (e.g. of the tibia) or to convert a simple endocarditis into the bacterial type.

7. The mechanism of bone absorption and deposition in response to irritation is discussed.

8. Methods of eliminating parodontal infection are referred to and the importance of complete elimination is stressed. It is shown to be not incompatible with the conservation of the teeth.

RÉSUMÉ.—1. Il est démontré que l'agent infectieux dans le sulcus paradentaire est limité à sa surface, mais peut être introduit dans la circulation sanguine ou lymphatique par des interventions traumatiques.

2. Des troubles locaux ou généralisés peuvent donc survenir comme suite de l'absorption de toxines solubles dans la poche paradentaire ainsi que par l'introduction traumatique des germes dans la circulation sanguine.

3. L'effet local de l'absorption des toxines est la destruction de l'attache de la dent—la pyorrhée.

4. L'effet éloigné de l'absorption toxique peut être une destruction analogue du tissu conjonctif en général—arthrite et fibrosite. Le foie et le rein peuvent en souffir, car c'est par eux que les toxines sont éliminées, et d'autres tissus susceptibles peuvent aussi être attaqués.

5. La bactériémie traumatique peut produire une nécrose osseuse locale ou alvéole "sèche". La lymphangite suppurée produit l' "abcès pyorrhéal".

6. L'effet éloigné de la bactériémie peut se manifester comme ostéomyélite, par exemple du tibia, ou transformer une endocardite simple en endocardite bactérielle.

7. Le mécanisme de l'absorption et de la déposition de tissu osseux comme réaction à l'irritation est discuté.

8. L'auteur parle des moyens d'éliminer l'infection paradentaire, faisant spécialement ressortir l'importance de l'élimination complète, qui n'est pas incompatible avec la conservation des dents.

ZUSAMMENFASSUNG: 1. In der vorliegenden Arbeit wird gezeigt, dass die die paradentale Tasche infizierenden Keime auf die Oberfläche begrenzt sind, jedoch durch traumatische Einflüsse in den Blut- und Lymphstrom hineingepresst werden können.

2. Oertliche sowie Allgemeinstörungen können daher sowohl durch Resorption von löslichem toxischem Material aus der Tasche in die Gewebe als auch infolge eben dieser traumatischen Einpressung von Organismen in den Blutstrom entstehen.

3. Der Einfluss der Giftresorption auf die lokalen Gewebe äussert sich in Zerstörung der Zahnbefestigung—Pyorrhoe.

4. Die Fernwirkung der Giftresorption kann in einer analogen Zerstörung des Bindegewebes im allgemeinen bestehen—Arthritis und Fibrositis. Da Leber und Nieren die toxischen Stoffe ausscheiden, können sowohl diese als auch andere empfindliche Organe ebenfalls in Mitleidenschaft gezogen werden.

5. Die traumatische Bakteriämie kann zu lokalen Knochennekrosen oder zu "trockener" Alveole führen. Eine akute eitrige Lymphangitis führt zum "pyorrhoischen Abszess."

6. Durch Fernwirkung der Bakteriämie kann eine Osteomyelitis (z.b. der Tibia) enstehen oder eine einfache Endokarditis in die bakterielle Form übergeführt werden.

7. Der Mechanismus der Knochenresorption und -ablagerung, wie sie als Folge der Reizung auftreten, wird besprochen.

8. Methoden zur Beseitigung der paradentalen Infektion werden erwähnt und es wird die Bedeutung der restlosen Beseitigung betont. Es wird gezeigt, dass diese mit der Erhaltung der Zähne durchaus vereinbar ist.

INTRODUCTION

THE purpose of this paper is to review the immediate effect of the organisms which inhabit the periodontal sulcus on the surrounding tissues, and also to consider their remote effect on the general health.

The subject is approached mainly from the clinical aspect and is discussed in the light of recent findings which are firmly supported by experimental evidence. These have shown on the one hand that, when undisturbed, the bacteria remain localized to the surface in the chronic pyorrhœa pocket and only the toxic products of their activity are absorbed (Fish and Maclean [2]), whereas on the other hand any traumatic disturbance of the inflamed gum margin may drive the organisms into the blood-stream and produce a transient bacteræmia (Okell and Elliott [7]) and Round, Kirkpatrick, and Hails [8]). These observations only refer to chronic infections.

The discussion, therefore, deals with the effect of this absorption of toxic material both on the parodontal tissues and on the system generally, and is also extended to include the effect on both the local tissues and the more remote parts of the body when the infection itself is disturbed by trauma and actually gains access to the blood-stream or lymphatics.

The toxic material absorbed causes locally a breakdown of the parodontal tissues which is known as pyorrhœa, and upon general absorption produces various chronic disturbances of bodily function. The traumatic entry of organisms into the tissues produces acute septic inflammation either locally or at some distant point, but this only happens if they gain a foothold in a nidus of non-vital tissue. If they remain in the blood-stream they are soon eliminated quite harmlessly. The difference between acute and chronic inflammation when produced by the mouth streptococci is that in the former the organisms are violently introduced into the tissues and are being vigorously attacked by leucocytes, while in the latter they have been successfully confined to a necrotic nidus and the reaction in the surrounding tissues is due to the absorption of their toxic products [2].

LOCAL SEQUELÆ OF PARODONTAL INFECTION

Pyorrhœa, as its name to some extent suggests, is a chronic suppurative inflammation of the marginal tissues of the gum. In an established case it is characterized by a deepened periodontal sulcus, ulcerated, and lined with granulation tissue, on which a

degenerated epithelium grows either in sickly luxuriance, like a plant kept in a cellar, or wilts as a scanty remnant.

This condition, though almost universal in civilized communities, is neither healthy nor normal. Fig. 1, taken from the erupting molar of a rat, shows how the horny cuticle of the gum and that of the enamel (Nasmyth's membrane) are originally continuous and protect the living parodontal tissues beneath from injury by shooting the food over the streamline contour of the gum-enamel junction.

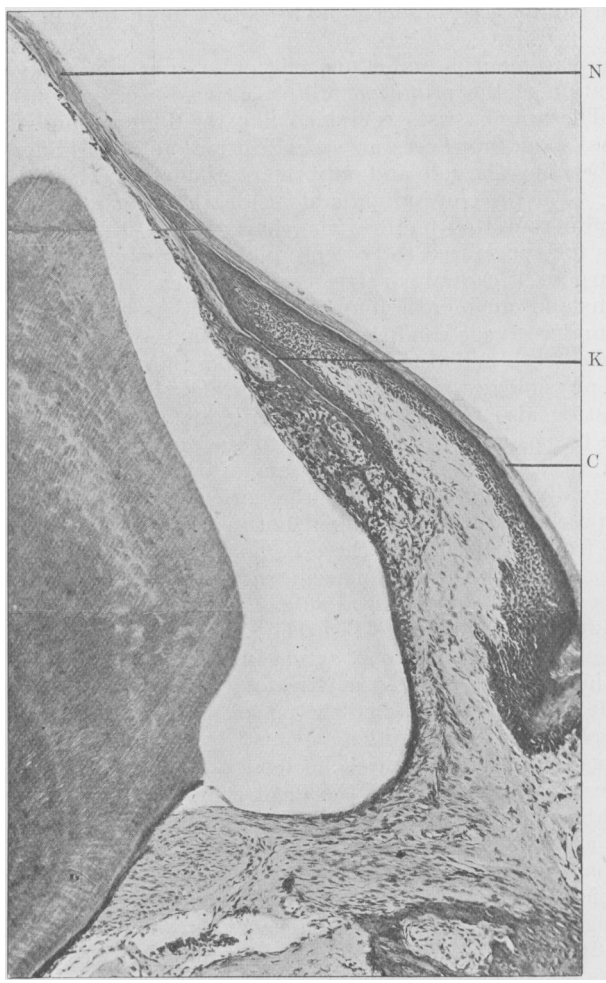

FIG. 1.—Photomicrograph (× 100) of partly erupted molar tooth of rat, showing continuity of Nasmyth's membrane (N) and the horny cuticle of the gum (C). There is a keratinous invagination at K but no periodontal sulcus.

There is, however, as shown by Stewart-Ross, a dipping-in of the keratinized cuticle together with its supporting epithelium at the place where the remnant of the enamel organ (the epithelial attachment) joins the mouth epithelium. This arrangement gives a triple horny layer at the actual edge of the gum, and so provides for a rapid proliferation and keratinization of the epithelium at this all-important point where most of the masticatory stress falls. Unfortunately the device contains

within itself the germ of its own undoing, and so, like all Nature's biological experiments which inevitably end in death, it is, from the human point of view, a failure. This horny invagination tends to split and only friction by the roughest primitive food can prevent the periodontal sulcus from developing (fig. 1A).

There is a similar dipping-in of keratinized material at the base of the nail-bed, and even in the unmanicured monkey this keratinous invagination also shows some tendency to split (fig. 2). In over-manicured fingers, softened by disuse, chronic infection of the nail-bed, onychia, is not uncommon. The infection enters this crevice, and the nail may be loosened and lost like a tooth, only in such an event a new nail will grow.

Such easy replacement is, unfortunately, only an attribute of very simple structures. Observation of the primitive protozoa suggests a conception of life akin to immortality. The amœba rises revivified like the Phœnix and duplicated at each translation. As soon, however, as specialization and cell-differentiation appear, somatic death becomes the rule and only the reproductive cells preserve this heritage of immortality. The price of individuality is death, and this principle applies to the teeth. The undifferentiated teeth of the shark persist in an endless series, but the human tooth is too specialized to be reproduced so freely, and though well adapted to its native purpose, it cannot survive the changed environment imposed upon it by civilized customs and our increased span of life—unless we compensate it for its lost stimuli. Even under savage conditions, as age advances the gums recede and become infected, and the teeth are lost, but for a number of years an almost ideal state of structural and physiological perfection persists like that in fig. 1.

It is interesting also to observe the interdental area of the young rat's tooth. Here again the tissues are firmly adherent to the enamel and there is no interdental space. Indeed, in fig. 3, the rat's molars, one of which is perhaps still only partially erupted, are actually stuck together by the cuticle which is common to both at the contact point, precluding for the moment both caries and pyorrhœa.

In material from civilized man there is, however, always some indication of the constant trauma to which the gum margin, softened by relative disuse, is submitted. There is always a definite periodontal sulcus and, apparently, always some chronic inflammatory infiltration round it (Fish [1]). If the pocket is shallow and its wall firm, abrasion, often amounting to an actual ulceration, is produced at the very edge of the gum at the spot on which the masticatory stress falls when the food slides over the crown of the tooth. Later, when the pocket is deeper, ulcers may be seen at the bottom of the pocket, caused by the continual tearing-back of the pendulous interdental papillæ as the teeth sink into solid food.

As time goes on, the surface of the dead cementum inside the pocket becomes covered with tartar and bathed in serous exudate in which organisms live and multiply; so that apart altogether from a continuance of the trauma which originally caused the lesion, there is a continuous toxic irritation of the ulcerated surface inside the pocket, which prevents healing. The organisms are, however, precluded from actually growing into the tissues by the leucocytes which form the floor of the ulcer in the pocket [2]. Some of these leucocytes are killed from time to time and form pus, which leaks out in a constant ooze.

In just the same way, pus oozes out from a sinus which leads down to a sequestrum of infected necrotic bone. Such a sequestrum also lies in a bed of exactly similar granulation tissue. The processes are strictly comparable and have many essential points in common. In each case there is a necrotic refractory mass of which the tissues cannot rid themselves and which yet harbours germs. In the one case it is necrotic cementum and tartar, in the other necrotic bone. In either case the mass forms a refuge for germs, enabling them to live in full enjoyment of the food and oxygen carried by exuded lymph, yet for the most part out of reach of the leucocytes. The polymorphonuclear leucocytes do indeed venture to the very brink of the lesion

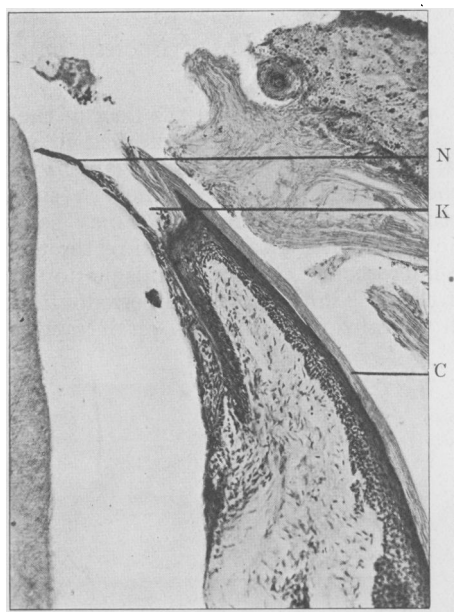

FIG. 1A.—Photomicrograph (× 100) of molar tooth of rat showing early split of keratinous invagination (K), forming a sulcus. N, Nasmyth's membrane. C, Horny cuticle of the gum.

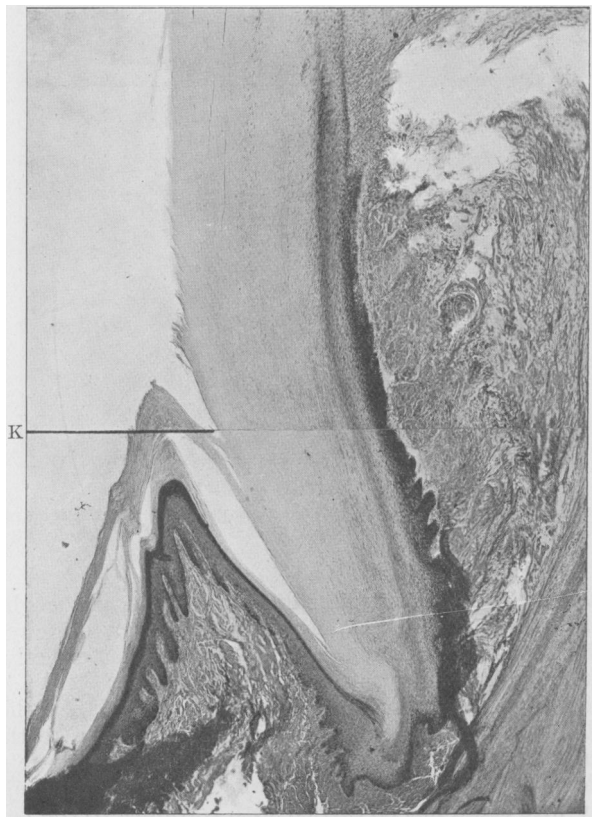

FIG. 2.—Photomicrograph (× 46) of base of finger nail of monkey (Mr. H. B. Harding's specimen), showing a keratinous invagination (K), similar to that in fig. 1, which also displays a tendency to split.

in search of food and, packing solidly, form the floor of the pyorrhœtic ulcer, just as they form the surface of the granulation tissue bed in which the sequestrum lies. Here they wait to catch and devour any stray organism which drifts their way, but if they venture into the pus they will probably never return.

The position is actually one of stalemate and may be demonstrated quite easily without relying on the histological interpretation of the tissue reactions as observed in sections under the microscope. If the organisms do actually penetrate and live in the tissues amongst the cells in the bone and periodontal membrane, then the apex of a pyorrhœtic tooth will always be infected on extraction, whatever we do to the

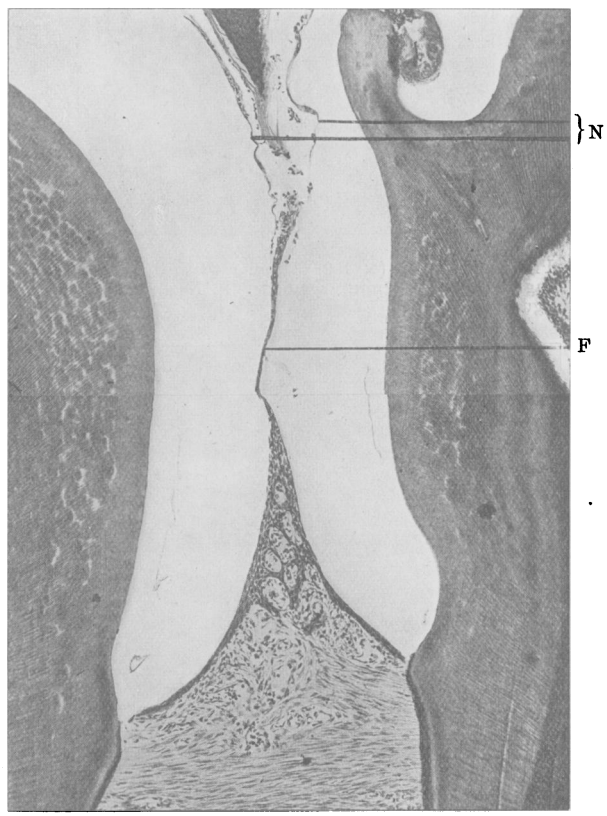

FIG. 3.—Photomicrograph (× 100) of interdental papilla of rat's molars, showing the Nasmyth's membranes (N) fused at F and healthy tissue filling the whole interdental space.

pocket. Alternatively, if thorough sterilization of the pocket before extraction enables one to deliver the tooth with a perfectly sterile apex, quite regularly, however severe the pyorrhœa, then it is clear that the infection was entirely confined to the pocket and was killed by the sterilization.

It has been found possible to do this as described in an earlier paper [2] and Dr. Maclean and I have now a series of 30 investigations of teeth extracted from pyorrhœa cases after cauterization of the pocket, all having sterile roots and apices, but such an unblemished record was only obtained by rigorously rejecting any tooth where the cautery wire could not be carried with certainty to the very bottom of the

pocket all round ; or if there was any bleeding which could carry infection from the surrounding teeth or mucous membrane on to the root as it came out, and of course, if the root accidentally hit the lips or other teeth as it came away, it was rejected. We have not yet had a single positive culture where we were satisfied that all these stringent conditions were fulfilled and had predicted a sterile apex.

It seems necessary therefore to accept the histological interpretation, thus supported, that the organisms are confined by a wall of leucocytes to the necrotic surface cells of the ulcers and to similar debris in the pockets. It must then follow that the reaction of the tissues behind the barrier of leucocytes is due to the toxic products of these organisms diffusing into them.

The same reaction is observed in the neighbouring bone whether the irritant be an infected sequestrum of bone or an infected sequestrum of cementum, and also whether the infected cementum be at the neck of a pyorrhœtic tooth or at the apex of a dead one. In each case, toxic products diffuse into the surrounding tissues although the

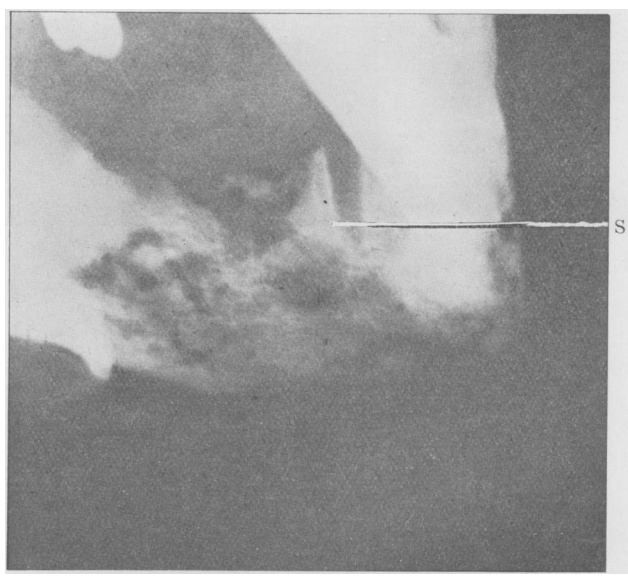

FIG. 4.—Skiagram of bone necrosis in human lower jaw (by Dr. Blackman from case referred to Mr. Ainsworth). S is socket of extracted tooth, forming sequestrum and surrounded by extensive absorption.

organisms themselves cannot penetrate. The result is generally a breakdown of the adjacent connective tissue, whether it be the fibres of the periodontal membrane or the matrix of the bone, though more rarely fibrosis and sclerosis occur. Perhaps the best way of appreciating the effect of this toxic absorption on the bone and the soft connective tissue round pyorrhœtic teeth is to take first a more massive type of reaction where, as the result of trauma, bone is actually invaded by organisms, an acute reaction follows, and a sequestrum is formed ; we may then consider the sequence of events.

Fig. 4 shows the sequestrum infected with organisms forming the centre of an area of widespread bone absorption, yet the sequestrum itself is quite unabsorbed. It consists of the lamina dura or shell of bone which once surrounded the extracted tooth. When the tooth was extracted, organisms were propelled into the vessels in this bone from the periodontal sulcus, as they were into the vessels of the pulp [2],

or of the arm [7]. At that most unfortunate moment, the trauma of extraction bruised the bone forming the socket wall and occluded the vessels imprisoning the germs. These latter made the best of their unique good fortune and, no longer in danger of attack from leucocytes, as they would have been if they had escaped into the general blood-stream, they increased enormously, ate every shred of soft tissue and converted the damaged bone into a bare, infected, necrotic sequestrum—the " dry socket ". From this focus the poisons and the germs started to spread in all

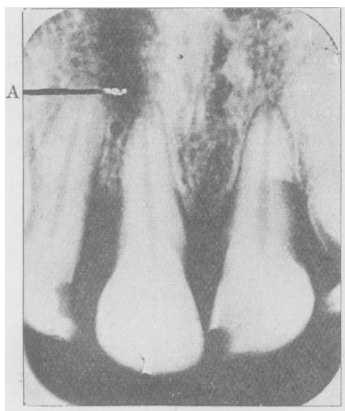

FIG. 4A.

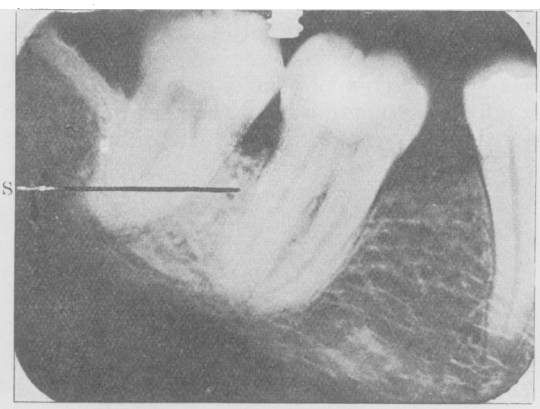

FIG. 4B.

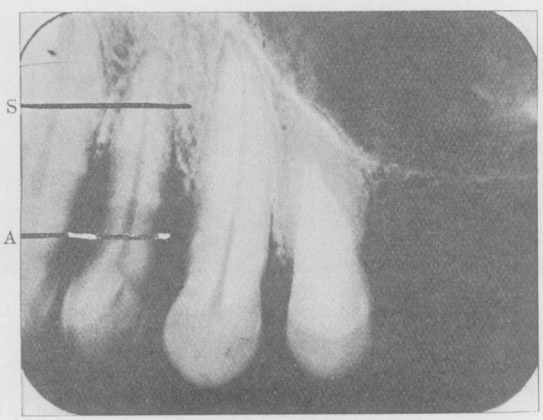

FIG. 4C.

FIGS. 4A, B and C.—Skiagrams of human teeth (by Dr. Blackman) from cases of parodontal infection, illustrating: Bone absorption, A in fig. 4A; bone sclerosis, S, in fig. 4B; Bone absorption, A, and sclerosis, S, in fig. 4C.

directions, although the poisons spread by diffusion more quickly than the non-motile germs could spread by sheer multiplication. In response to this toxic diffusion, leucocytes hurried to the spot. The polymorphonuclear leucocytes got there first and formed their solid barrier as effectively as possible in a tissue which, by virtue of its lacunæ and canaliculi, offers such strategic advantage to the invaders. The leucocytes, nevertheless, at least delayed the spread of the actual organisms, though the poisons continued to diffuse out and irritate the cells of the surrounding bone

behind the wall of leucocytes. The result of this toxic irritation is the absorption of bone which is so very well marked in the skiagram.

Similarly fig. 4A shows an infected sequestrum of cementum and tartar harbouring germs which are prevented from entering the surrounding tissues by the remnant of epithelium and the wall of leucocytes which form the lining of the pyorrhœtic pocket [1]. At the same time the toxic products of this struggle diffuse into the surrounding bone, and again the result is bone absorption.

There are two ways in which the textbooks say that bone can be absorbed; one is by the action of osteoclasts, and the other by halisteresis or vascular absorption. There appears, however, to be no evidence of the existence of the latter phenomenon, and indeed it is by no means clear how it would work. If we accept the rule that inflammatory hyperæmia produces bone absorption, and ischæmia promotes bone deposition (Leriche and Policard [5]), are we willing to overlook or deny that the most active hyperæmia accompanies the opposite phenomenon, to wit repair, when bones are fractured?

Even if we accept the view that, despite the perfect buffering of the blood and tissue fluid, a local inflammatory change in the pH sufficient to use up the alkali reserve and attack the bone salts could take place despite the hyperæmia which is flushing out the tissues, we must still explain how the organic part of the absorbed bone matrix is disposed of. This collagenous material must be digested by a proteolytic ferment which, in turn, can only be produced by a cell, whether we call it an osteoclast or anything else.

The theory of bone absorption by halisteresis may perhaps have been devised because it is not often that we see osteoclasts actually at work, and realizing this difficulty, Hopewell-Smith [4] suggested that even the round cells are capable of absorbing the bone; yet in fact we would not expect to see osteoclasts at work very often. It may not be generally recognized that they work very quickly and then break up and disappear. Gottlieb and Orban [3], however, showed that if a tooth is moved, in a dog, by means of an appliance, a considerable part of the interdental septum is absorbed away by newly formed osteoclasts in as little as thirty-six hours. At this rate the osteoclasts could eat the whole skeleton in a few months, as indeed they very nearly do in cases of parathyroid tumour.

In a chronic disease like pyorrhœa, therefore, we find a very large number of Howship's lacunæ in which osteoclasts, having previously removed a contaminated fragment of bone matrix, have now disintegrated and disappeared, so that only a very occasional one is actually caught in the act (figs. 5 and 5A).

If the irritation becomes too severe, the osteoclasts break up and are replaced by round cells, as was shown by Stewart-Ross [9] when he infected the dentine experimentally from the root canal. At first the permeable apical cementum was absorbed in his experiments, then as the tubules of the dentine were opened up, the released toxic material killed the osteoclasts, whereupon leucocytes entered and filled the lacunæ. This often happens if an acute infection spreads towards an osteoclast; as the toxicity of its environment increases, the osteoclast is killed and replaced by cellular infiltration (fig. 5, R). In this way, Hopewell-Smith's observation is explained. Conversely, if the irritant is diminished, the cellular infiltration and the osteoclasts disappear and osteoblasts are stimulated by the fading irritant to build up the bone again and fill in the lacunæ, as is shown hundreds of times throughout the bone in fig. 5, or in any long-standing case of pyorrhœa.

Observations on chronic inflammatory reactions in connective tissue suggest, therefore, the following summary:—

During the invasion, streptococci having a tendency to grow into the tissues are met by a wall of polymorphonuclear leucocytes, while their toxic products diffusing into the surrounding tissues are encountered by round-celled infiltration. When repair sets in, wandering cells (histiocytes or macrophages) eat up the soft

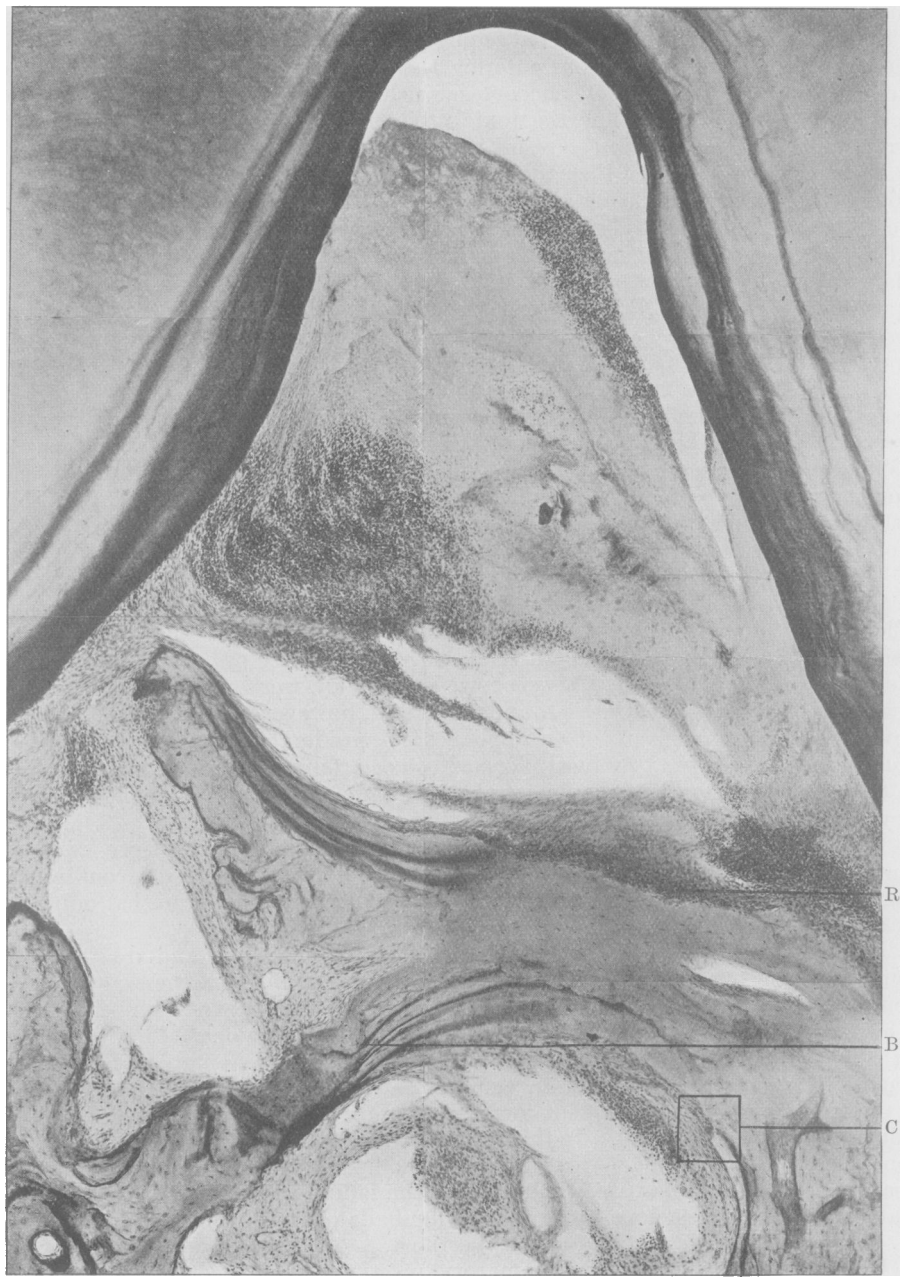

FIG. 5.—Photomicrograph (× 60) of parodontal pocket (human) extending between the roots of a lower molar, showing numerous Howship's lacunæ from which the osteoclasts have disappeared and been replaced by round cells (R) where the toxic irritation has increased. At B, where the irritatiou has subsided, the Howship's lacunæ are built up again with bone matrix and at C (*see* fig. 5A) an active osteoclast is to be seen.

tissue debris, while fibroblasts are stimulated to produce scar tissue or " fibrosis " as the irritant is diluted and removed.

Similarly osteoclasts, formed in response to toxic irritation, eat the contaminated bone, while if the irritant becomes diluted, or if it is less severe, osteoblasts are stimulated to lay down new bone and produce " sclerosis." Osteoclasts never phagocytose either living or dead organisms, and are killed by too severe a poison or by the actual presence of streptococci.

The sequence of events, therefore, in such a case of suppurative osteitis as that illustrated in fig. 4, becomes clear.

The organisms being trapped in the damaged bone of the socket, the surrounding bone matrix becomes contaminated by their soluble toxic products which diffuse along the lymphatics and into the canaliculi of the matrix. This stimulates the production of osteoclasts at an appropriate distance from the infection, and unless these osteoclasts can clear away a zone of bone around the infected area before the organisms can reach them, they will be killed and the necrosis will continue to spread

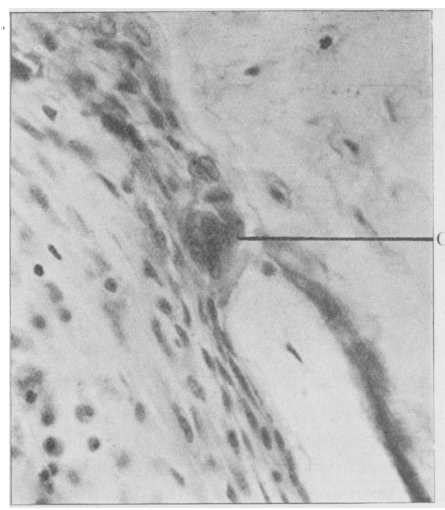

FIG. 5A.—Higher magnification of fig. 5 (× 412). C is one of the few osteoclasts to be found.

If, however, the osteoclasts accomplish their task in time, the sequestrum becomes separated from the rest of the bone and the gap is filled by a safety barrier of leucocytes. The organisms spread to the edge of the sequestrum but cannot cross the gap because of the leucocytes. Absorption of the surrounding bone on the other side of the gap, however, continues as long as the poisons diffuse into it, that is, until the sequestrum with its colony of organisms is taken away. The dead infected bone can never be absorbed, unless at a much later date the infection dies out of it and it becomes a sterile foreign body.

The Howship's lacunæ seen on the surface of all sequestra were cut by osteoclasts before the actual infection reached the periphery. Similarly, the living bone of the alveolar crest near a pyorrhœa pocket, being uninfected, is absorbed, yet the cementum in the pocket, being infected, is not absorbed. The apex of a dead tooth may be absorbed while toxic material only is escaping from the infected root-canals into the bone, but if the infection itself reaches the osteoclasts they die, pus fills the lacunæ, and absorption ceases. A peri-apical rarefaction area is evidence of past toxic irritation, not necessarily of present infection : though the once irritated area may have

become infected or may be still the site of toxic irritation. If all irritation had entirely gone, new bone would have appeared and filled the gap.

In fig. 4A the alveolar crest, and even the deeper bone, is absorbed because the toxic matter diffusing into it was sufficiently irritating to stimulate the embryonic mesoderm cells to produce osteoclasts, which have now disappeared and been replaced by round cells. In fig. 4B, which is taken from a patient with a clinically clean mouth, the toxic diffusion is much smaller in amount and not so highly irritating, hence only osteoblasts were stimulated and sclerosis of bone was produced round the pocket. In fig. 4C, both grades of irritation are seen. Absorption of the crest of the alveolar bone and sclerosis at a lower level where the toxic matter had become diluted in the tissues.

In each case the toxic diffusion follows the path of lymphatic drainage from the gum margin, as described by Macgregor [6]; and before leaving the local sequelæ of infection in the sulcus the possibility of actual organisms being thrust into the lymphatic vessels of the gum margin and periodontal membrane must be considered. No harm would be likely to follow if the organisms were carried direct to the lymph-nodes, but not infrequently they become arrested halfway down the periodontal membrane—perhaps by the pressure of the lingual bar of a denture or at other times just owing to temporary stasis in the lymphatic channel. This commonly occurs between the upper lateral and canine, and the result is, as occurs elsewhere in the body under similar circumstances, acute suppurative lymphangitis.

The condition is recognized clinically as a parodontal abscess. The tooth concerned becomes very tender to pressure, a continuous gnawing pain is experienced, the gum margin is red and swollen, and ultimately the pus points either through the alveolus or into the pocket, thereby enormously deepening it overnight. The condition is significant as showing how impossible is the conception that these mouth organisms really have their normal habitat in the tissues.

Remote Sequelæ of Parodontal Infection

In view of this evidence it is possible to assess very accurately the dangers of pyorrhœa to the patient's general health, and to place the true value upon any method of treatment which may be devised. The danger to health is first from the bacterial shower into the blood-stream which occurs on eating [8], or on extraction of the teeth [7]. Normally this transient bacteræmia disappears very rapidly and does no harm, but there are exceptions. The organism may, as Okell and Elliott suggest, become implanted on a fibrinous vegetation of a heart valve in cases of active endocarditis, since these fragments of non-vital fibrin lie actually in the blood-stream and offer a safe breeding ground for the germs. The result is a fatal bacterial endocarditis which in 95% of cases is due to *Streptococcus viridans*, the true mouth streptococcus; but much may be done to avoid such a catastrophe.

If possible no tooth should be extracted while fibrinous vegetations are still present. Any living tooth necessarily extracted on account of pain should have the periodontal sulcus cauterized first; it is then quite safe to extract it, but it would be wiser to treat the pulpitis with arsenic. The extraction of any dead tooth is, however, still a grave danger even if the pocket has been cauterized. It should only be extracted if an acute abscess forms and causes severe pain, and of course the sulcus must still be cauterized. Even so, we have found that with a dead tooth a bacteræmia may occur from the infected apex alone, even after the sulcus has been cauterized, so this risk should not be lightly taken.

If, however, a patient with active non-infective endocarditis has severe pyorrhœa with heavy granulations round the gum margins, he is in very serious danger even if we do not interfere surgically. The risk of a bacteræmia from chewing is so grave that he should be put on a soft diet. He must not brush or rub the gums while the

heart vegetations are present, but may only hold very hot mouth washes, such as permanganate of potash and peroxide of hydrogen, in his mouth frequently.

The vegetations may persist, however, for a long time, and the danger of a shower from eating is so grave that it is a matter of great urgency to devise a method of treating these cases. It is not safe to leave the mouth condition as it is, much less, of course, to extract the teeth or even manipulate the gums. We have even had a positive blood culture after simple scaling. It is really a choice of evils ; gingivectomy will cure the pyorrhœa, but may cause a bacterial shower. In one case out of four Dr. Maclean and I have found this to occur, though it may depend on the dexterity with which the operation is performed. Perhaps the safest course is to pack the pockets with zinc oxide and oil of cloves, mixed rather thin and incorporated with wisps of cotton-wool. This dressing is tucked, rather than packed, into the pockets with the utmost gentleness. Mr. Arthur Bulleid tells me he has observed histologically that, if properly carried out, this treatment eliminates all pus from the pockets for the time being. This agrees with clinical experience and it would therefore appear to be our best safeguard in all such cases. The dressing can, of course be repeated every few days throughout the acute phase of the illness, and as the inflammation subsides, scaling, and even gingivectomy, may be carried out quite safely.

A second danger from a bacterial shower, which may occur in anyone however healthy otherwise, is that during the few minutes, or possibly hours, that the organisms are circulating in the blood, the individual may bruise a bone and imprison some of the bacteria in the clot. If this happens there is nothing whatever to prevent them growing and setting up an osteomyelitis in, for example, the tibia, in just the same way as necrosis was produced in the mandible by damaging the alveolus during extraction at a moment when the vessels in that bone were carrying the infection which had just been thrust into them by the pumping action of the forceps.

On the other hand, the bruising of a bone at a time when the blood is sterile, is not liable to produce infective osteomyelitis, since a subsequent bacteræmia cannot introduce organisms into a vessel which is already thrombosed in the bruised bone. Nor, even, will mouth organisms penetrate into the bone of a socket after tooth extraction if they were not trapped in it at the time. The streptococci and staphylococci are not motile, whereas leucocytes are ; and the injured vessels do not remain open for germs to be swept in but are closed by blood-clot at once. Even burnishing infected saliva into newly opened bone, as is often done with an elevator in the socket of a partially extracted tooth, generally fails to infect the bone.

Apparently the only other possible danger from a bacterial shower, since septicæmia from it is uncommon, is that these organisms, temporarily in the blood-stream, might find a home in such places as the joints and ends of the bones, thereby producing arthritis, or perhaps in fibrous tissue, producing fibrositis. We have, however, shown [2], that the streptococci can only continue to exist in the tissues if they establish a necrotic nidus, which in this case would have to be a pyæmic abscess. No such lesions have ever been demonstrated in the tissues in fibrositis or rheumatoid arthritis although they would be very easy to demonstrate if they were there. It may, therefore, be assumed that the effect of oral sepsis on these diseases is due to the absorption of soluble toxic material and not to the direct spread of the germs themselves to the site of the lesion.

To summarize, therefore, the theoretical position is that the bacteræmia of pyorrhœa is only of danger to a patient suffering from active, non-infective endocarditis, or, in the case of a healthy patient, if trauma to a bone should occur at a moment when the organisms are present in the vessels at the site of such injury. Three clinical cases of the former catastrophe have come within the author's experience recently, while the clinical incidence of " dry " socket is strong evidence of the latter view since it occurs only when the bone is badly bruised at the moment of extraction, that is, at the moment of invasion by organisms. It has, in recent months, been entirely

eliminated from the author's practice by cauterizing the gum margin in doubtful cases before extraction.

The chronic toxic absorption into the tissues is, however, a more constant menace though perhaps not so dramatic. Not only do the soluble toxic products of the organisms' activity in the sulcus diffuse into the periodontal membrane and alveolar bone and therefore destroy the attachment of the teeth but, being taken into the general circulation, they are liable to irritate other remote tissues, as shown by Dr. Graham.[1] The defence against this poison is twofold. The round cells, chiefly lymphocytes, which infiltrate the parodontal tissues are a response to the presence of the poison locally, and presumably destroy it to some extent, otherwise they would hardly be likely to be so constantly attracted by it. Secondly, much of the toxic matter is carried away by the lymph-stream [6], as is shown by the chronic lymphangitis which surrounds the lymphatics [1]. This poison will, therefore, meet a further large number of lymphocytes in the lymph-nodes.

There is, however, a mass of clinical evidence that a considerable amount passes into the general circulation, and before it is ultimately destroyed in the liver and kidneys, which form the second line of defence, it may irritate any tissues which are inherently susceptible and temporarily sensitized, such as the joints and fibrous tissue generally. It is significant that these structures are composed of the same type of connective tissue as the parodontal tissues themselves. Moreover the morbid changes in infective arthritis, which include bone absorption and "sclerosis" or lipping, round-celled infiltration, and fibrous tissue breakdown or hypertrophy (Willcox [10]), are very similar to the changes which occur in the parodontal tissues in response to irritation from the toxic material in pyorrhœtic pockets.

Fig. 6 shows a skiagram of the hand of a patient suffering from rheumatoid arthritis. The bone absorption round the joints is well marked. In fig. 7, another case of infective arthritis, there is sclerosis or bone deposition, particularly on the iliac side of the joint, though this is a less common phenomenon in the infective type than in simple osteo-arthritis.

When, therefore, a patient exhibits, on the one hand, inflammatory and degenerative changes described as fibrositis or arthritis in the connective tissue generally, and at the same time exhibits an ulcerated gum margin, and is absorbing poisons from it which are causing hypertrophy, or destroying the same sort of tissue in the jaw in exactly the same way, it seems reasonable to associate the two conditions. How far the extent of the local tissue destruction may be taken as a measure of the damage being done to the more remote structures is a matter of conjecture, but it seems unlikely that much harm can be caused by the diluted toxic material to a distant joint if the concentrated poison at the point of entry is not causing sufficient irritation to destroy or affect the local connective tissue. The radiographic demonstration of local bone destruction or sclerosis is at least a very useful indication that toxic absorption is taking place, and the absence of any local radiographic change in the bone is not without its significance.

Patients vary very widely in their sensitivity to this toxic matter ; one will show widespread bone destruction with only very slight pocketing or ulceration, while another may suffer very little local bone damage from quite extensive and old-standing "dirt pyorrhœa". Such inherent frailty, or resistance, in the patient's protoplasm—or perhaps it is acquired susceptibility or immunity to toxic irritation—may not be present in all the tissues of the body. Structures under constant strain may succumb more readily, and it must be admitted that toxic matter which fails to stimulate the production of osteoclasts at the point of entry may nevertheless irritate such tissues as the retina, or a joint which has been sprained, even when in much greater dilution. It does not seem reasonable, however, to extract a dead tooth with no radiographic or clinical sign of bone change round it if the patient is in good health, since in such a case there are for the moment neither local nor general signs of toxic absorption.

[1] *Proc. Roy. Soc. Med.*, 1937, **30**, 1165 (Sect. Odont., 51).

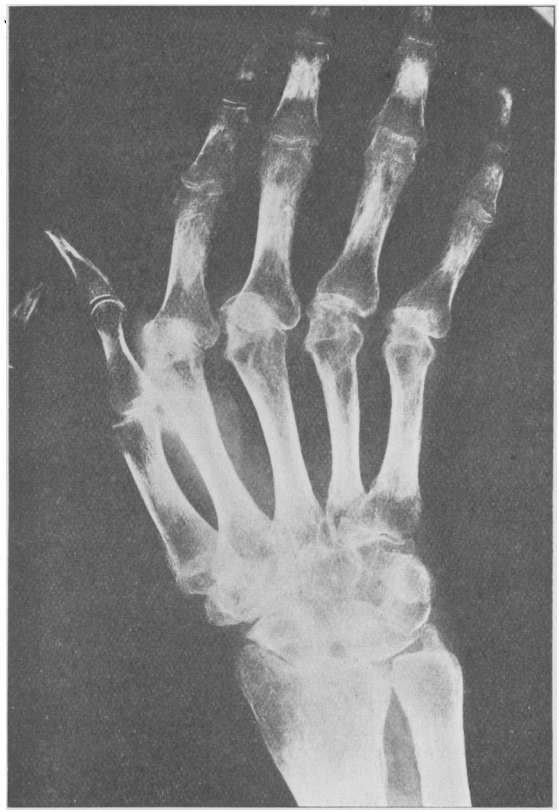

FIG. 6.—Skiagram (by Dr. Rohan Williams) of infective arthritis of hand (human) showing bone absorption.

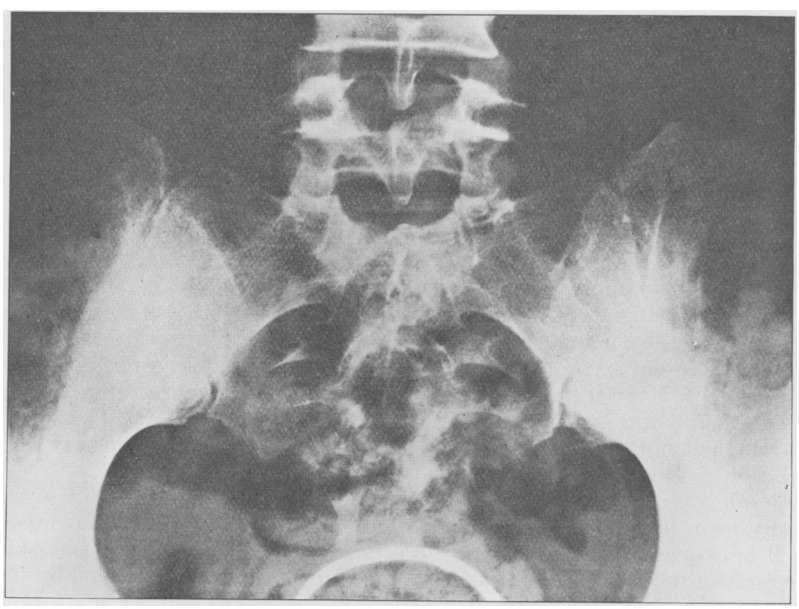

FIG. 7.—Skiagram (by Dr. Rohan Williams) of infective arthritis of sacro-iliac joint (human), showing sclerosis especially on the iliac side of the joint.

On the other hand, certain obvious indications for thorough eradication of all doubtful or even possible sources of toxic absorption do constantly occur. Derangement of the liver or kidney function is perhaps the most certain, since these organs are ultimately responsible for the elimination of the poisons. Similarly, in pharyngitis and gastro-intestinal disease, the danger of swallowing irritating discharges must not be overlooked. Even in respiratory disease, such as asthma, two recent cases of a dramatic recovery following gingivectomy illustrate an association of these conditions with oral sepsis which has been recognized in some quarters for a very long time.

Elimination of Parodontal Infection

The list might be considerably extended, but only by encroaching on the province of the general physician. It is, however, very important that we, as physicians of the mouth, when called upon to eliminate the possibility of toxic absorption or a bacterial shower from the parodontal tissues, should do so thoroughly and completely and not merely carry out an isolated dramatic operation. It is not of much benefit to the patient to extract a useful, but dead, front tooth because there is a trace of periapical bone absorption present and yet to leave the gum margins round the back teeth ulcerated, inflamed, and liable to bleed at a touch. Incidentally such an operation is also seriously misleading to the general physician. There is more toxic absorption, as shown by the soft tissue ulceration and bone loss round these periodontal sulci, than could possibly have been taking place from the infected apex, and the organisms are the same in each case. The patient would be much healthier and happier if the chronic inflammation round the back teeth had been cured and the front tooth left; though, of course, both areas should be treated. Perhaps apicectomy of the front tooth would have been effective.

Similarly, it is almost impossible to find a case in which a few teeth are so badly affected by pyorrhœa that they have to be extracted and yet the rest of the teeth are not in need of any treatment at all. It is almost invariably necessary to carry out a toilet of the gum margins round the remaining teeth if the patient is not to lose all possible advantage from the extractions. It may be that our failure to complete the eradication of sepsis in such cases has often misled physicians, and made them, as well as the general public, wonder whether oral infection is indeed a factor at all in general disease. Certainly we cannot expect any clear-cut results from such mismanaged experiments.

Even if all the teeth are extracted, the operation may be associated with so much physical and mental trauma and such a flood of toxic matter and actual bacteria into the blood-stream that the patient is often irreparably damaged, whatever he may ultimately gain.

It is, however, seldom necessary to extract all the teeth. Even teeth which are slightly loose are far more useful than most dentures. Once it is realized that, if a tooth is reasonably firm and its pockets can be cut away without leaving it absurdly denuded, it is thereby made quite healthy, a very large number of teeth may be saved which are at present extracted, and the deformity and discomfort of full dentures will be avoided. Even if the patient's general health does not improve, he has gained by the treatment and is the better for having a clean mouth and most of his teeth intact. He has not lost all his teeth and gained nothing; moreover, he will be grateful, not disgruntled.

Wholesale extractions may lead to very serious disability, and it is no wonder that a physician hesitates to expose a patient to the risk of edentulous misery which may well be worse than the original disease; but he nevertheless wishes his patient to have a clean mouth if it can remain intact, and no patient, if he realized it, would willingly have a chain of suppurating sores in his mouth—more loathsome than a festering boil on the surface of his body.

To turn for a moment to the actual treatment of parodontal infection. The demonstration that the organisms are all lodged superficially in the pocket has allayed any misgiving about the necessity of treating, or maltreating, the underlying bone which was previously thought to be infected. It is only necessary to cut away the pockets to ensure that the organisms shall lose the crevice in which they found sanctuary. The pernicious association of the infected sequestrum of cementum and the granulation tissue bed of the ulcer is broken up. Moreover, if we can render the epithelial covering of the gums accessible to the patient's efforts at mouth hygiene, the horny cuticle of the gum can be restored by friction right up to its attachment to the tooth. The toxic absorption will cease completely, for the poison cannot penetrate the horny cuticle, and we can give an assurance that the teeth are as void of offence as any teeth can be.

If, however, the disease is too far advanced, or through lack of co-operation, the patient or the dental surgeon fails to secure firm keratinization right up to the cement attachment, so that granulations remain, the only cure is to extract the tooth concerned. Mr. Arthur Bulleid has pointed out that a perfect control of the success, or otherwise, of the treatment is the continued presence or absence of pus cells at the neck of the tooth. With this dictum the author emphatically agrees, but would crave indulgence, for it is difficult to obtain complete success in any human endeavour, and a few pus cells can generally be found at the gum margins between the back teeth of even people with very clean mouths ; so much so that somebody once described the leucocyte as a normal inhabitant of the sulcus. Such a view was, of course, misguided, but to show that we have cured pyorrhœa, we must undoubtedly eliminate the possibility of there being any marked collection of pus cells at the gum margins.

Fortunately it is seldom necessary even now to extract the tooth in order to do this, and we may surely hope that, by constant effort to save every living tooth and render it healthy, our methods will become simplified and improved and the reproach of wholesale extraction be lifted from us.

The author wishes to express his indebtedness to Sir William Willcox for his advice and to Dr. Maclean for his continued co-operation ; to Mr. Ainsworth, Dr. Blackman, and Dr. Rohan Williams for the loan of skiagrams ; to Mr. Harding for the loan of a section, and to the Medical Research Council for a grant towards the cost of the experimental work involved.

REFERENCES

1 FISH, E. W., *Brit. Dent. J.*, 1935, **58**, 531.
2 FISH, E. W., and MACLEAN, I. H., *Brit. Dent. J.*, 1936, **61**, 336.
3 GOTTLIEB, B., and ORBAN, B., " Die Veranderungen der Gewebe bei ubermaziger Beanspruchung der Zahne ", Leipzig, p. 26.
4 HOPEWELL-SMITH, A., " The Normal and Pathological Histology of the Mouth ", 1919.
5 LERICHE, R., and POLICARD, A., " The Normal and Pathological Physiology of Bone ", (Trans.), London, 1928.
6 MACGREGOR, A., *Proc. Roy. Soc. Med.*, 1936, **29**, 1237.
7 OKELL, C. C., and ELLIOTT, S. D., *Lancet*, 1935 (ii), 869.
8 ROUND, H., KIRKPATRICK, H. J. R., and HAILS, C. G., *Proc. Roy. Soc. Med.*, 1936, **29**, 1552.
9 STEWART-ROSS, W., *Brit. Dent. J.*, 1935, **58**, 473.
10 WILLCOX, W. H., *Brit. M. J.*, 1921 (i), 804.

The Toxicity of Sterile Filtrate from Parodontal Pockets

By J. WALLACE GRAHAM, M.D., D.D.S., M.R.C.P.

ABSTRACT.—The local effect of the absorption of toxic material from pyorrhœa pockets on the hard and soft tissues around the teeth is well known. In this experiment an attempt was made to study the toxic effect on remote structures by injecting the sterile filtrate fresh from pyorrhœa pockets into various animals.

The filtrate was obtained from patients with chronic pyorrhœa by removing the contents from parodontal pockets and passing them through a Seitz filter. The sterile filtrate obtained was then injected into cats, guinea-pigs, rabbits, and rats, in varying amounts.