DENTAL FOCI OF INFECTION*

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My experience as a pathologist and a teacher of both general and dental pathology influenced the selection of, and limitation of this subject to, "Dental Foci of Infection." What can be said that will be helpful in your evaluation of suspect dental lesions?

The problem of physician, dentist, or both is essentially one of diagnosis. Proper appraisal of the history, physical findings and accessories or aids is the only satisfactory method. Many failures, which should not be failures, result from too much attention to accessories. The history is more or less neglected and the clinical examination slighted. Appreciation of the time factor should be the greatest help and in those cases suspected of dental foci, knowledge of this time element will often be a most valuable link in the diagnostic chain.

The concept of infection seems to have strayed from the narrow path of its original meaning. It depends on the struggle between the invading agent and the defending host.[1] First, the pathogenic organism must exhibit the ability to gain access, survive and multiply; second, it is opposed by the defensive mechanism of the body which includes immunological processes and tissue changes. The latter is the primary concern of a pathologist.

The importance of the oral cavity as a portal of entrance and a breeding place for microörganisms is apparent. When the anatomy and physiology of the teeth, their supporting structures, the gingivae and alveolus are considered, they offer a constant nidus for the entrance and propagation of infection. This was recognized by several investigators. William Hunter,[2,3,4] in his "Oral Sepsis as a Cause of Systemic Disease," did much to disseminate this idea.

The overemphasis of laboratory aids or accessories, to the neglect of history and clinical findings, calls for comparison and evaluation, especially for bacteriology and x-ray.

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* Presented October 21, 1940 at the Graduate Fortnight of The New York Academy of Medicine.

Bacteriology

The literature on bacteriological investigation of dental foci of infection concerns three groups of bacteria. The first includes the fusiform bacilli and spirochetes or Vincent's organisms. Although there is increasing evidence that fusospirochetosis may cause systemic disease,[5, 6] the other two groups are of more concern now. The second contains mainly the B. acidophilus group. Though the etiology of caries is not fully understood, there are two schools of thought. One emphasizes the internal mechanism and nutrition of the tooth, and the other advocates the external or bacteriological and chemical processes. By the fermentation of carbohydrates, these organisms produce organic acids and enzymes active in cavity formation. The dissolution of enamel and dentine or cement leads to involvement of the pulp, and a direct circulatory route for the entrance of bacteria through the apical foramen. This is the portal of entry for the third group, in which the chief offender is the Streptococcus viridans.

Demonstration of Bacteria

Bacteria have been demonstrated in many ways and in various sites. Smears, cultures, and animal inoculation have been used to isolate and identify organisms. Tissue stains have aided in both biopsy and postmortem material. Periapical tissues, pyorrheic pockets, saliva, carious enamel and dentine, pulp, pulp stones and periapical bone have yielded various positive findings. Microörganisms have been found in blood, joints, heart, kidney, gastrointestinal tract, muscle, prostate, eye, nose, throat, bone, feces and urine which were considered to have their origin from dental foci. However, bacteria can be active long before they produce histopathological tissue changes or x-ray evidence of their presence.

Blood Cultures

Recent investigations can be presented succinctly in two groups:

I. Occasionally positive blood cultures have been demonstrated in healthy individuals;[7] temporary or transient bacteriemias have been reported following operative procedures, dressing manipulations,[8] urethral operations[9] and tonsillectomies.[10, 11] In 5,310 blood cultures on hospital cases Fox and Forrester[12] obtained their greatest percentage of

positives in subacute bacterial endocarditis (238 positive cultures).

II. Immediately after extraction, positive blood cultures occur when previously the blood has been negative, and when in a relatively short time, the blood again becomes negative.[13] With periodontal disease the incidence of positive blood cultures after extraction may be even higher.[14, 15] Rocking of teeth or massaging of the gingiva has been followed by positive bacteriemia.[15, 16] Mastication of hard candy has been followed by temporary bacteriemia.[17] Serratia marcesens have been recovered from the blood following extraction, having been thus introduced into the gums.[18]

The phenomenon, anachoresis, whereby certain foreign blood-borne substances, including bacteria, are attracted to and fixed in inflammatory areas, has a direct application to infected teeth. Csernyei's experiments[19] indicate that chronic periapical inflammations have an anachoric effect on microörganisms, and that organisms do not remain in the blood, but take refuge in the area of inflammation. H. B. G. Robinson and L. R. Boling[20] present the results of their experiments in which they were able to produce injury to the pulps of animals, following which they injected certain bacteria into the vein of a hind leg and later definitely recovered organisms from the site of injury.

It has been demonstrated that bacteriemias (determined by positive blood cultures) may be transient, and in many instances be produced by artificial means or in extremely unusual circumstances. Isolation of a bacterium from the blood stream gives neither its origin, nor tells what relation it has to systemic disease already present. In practically all cases of subacute bacterial endocarditis, we incriminate the Streptococcus viridans. In hospitals where repeated positive blood cultures have yielded a Streptococcus viridans, the patient ultimately proves to have either clinically, or at autopsy, endocarditis lenta. Bacteriological evidences as to the type of organism about the tooth and supporting structures favor the green producing streptococcus. However, is it not presumptuous to assume in most of these cases that the organism in the tooth, then in the blood and finally on the heart valve is necessarily the cause?

Finally, it is pertinent to evaluate bacteriological technique and reports. Good technique is extremely difficult, time consuming and expensive. Most of the work, to date, has been with aerobes. Negative results do not always mean that organisms are not present, nor do positive findings always mean that such organisms are causal. Special tests for viru-

lence and specificity, except in the hands of a few experienced workers, are rarely performed and need careful evaluation.

X-RAY

X-ray examinations, when devoid of clinical data, should be regarded only as diagnostic aids or accessories. Compared with the knowledge of the presence of bacteria from a suspicious area, positive x-ray findings are more valuable. Assuming good standard technique, comparisons are based on radiolucent and radiopaque changes. Pathological changes may be present, and even good x-rays show nothing. On the other hand, the size of an x-ray area is not necessarily an index of the activity of a process. When changes are present in hard tissues, i.e., enamel, dentine and cement (the tooth proper) or the bone (the alveolus), the x-ray will be of greater aid than when changes are found in the soft tissue, where, however, sometimes by inference, conclusions may be justified.

As an index of dental infection, a radiolucent area is more indicative of activity than a radiopaque area. Changes have occurred to bring about a decrease in the harder tissue, a removal of the calcium. The most important cause of a radiolucent area is usually destruction, as: (1) In caries due to actual loss of tooth substance; (2) when a root of a tooth has been removed, destroyed or resorbed; and (3) in bone with an abscess; granuloma or cyst; with sequestration in osteomyelitis; with a destructive tumor. Whether the infection is active or latent, and how little or how much it is contributing to a systemic infection is the main question. A single x-ray of a radiolucent area may be difficult or impossible to interpret. When however, successive x-rays reveal further progress and extension, associated with a history and clinical signs and symptoms suggesting a focus of infection, such evidence is strongly convincing.

In the light of infection radiopaque areas deserve less consideration. Increased density or condensation can be illustrated by: (1) enamel nodules; (2) metallic fragments; (3) hypercementosis; (4) productive lesions, as osteogenesis, osteosis (exostosis), osteoma. As a result of infection, radiopaque areas do occur but in general they are best interpreted as bone scars and are testimony to the fact that healing has been adequate.

When successive x-rays originally reveal a radiolucent area, and

subsequently a change to more and more radiopacity, with a history and clinical symptoms and signs suggesting a dental focus, such evidence is quite convincing that the probability of such a lesion causing systemic manifestations is less and less.

Other Tests

The sedimentation test in dentistry, particularly with the present problem has received more attention of late.[21,22] It is a laboratory aid or accessory which only exceptionally may indicate the activity of a dental focal infection. When a series of successive tests are made for comparison, the information is much more valuable than with a single test, especially in interpretation of the progress of a condition.

In dental literature an increasing number of claims for short wave diathermy have been made. They are no doubt, a direct reflection of the popularity of this procedure in medicine.

Benefits have been claimed not only from a therapeutic standpoint, but even as an aid in the diagnosis of dental infections—"Provocative irradiation."[23]

Several have claimed that the application of "Short Wave" to a suspect dental focus, if active, will be followed by an increase in the sedimentation rate and that this test has considerable diagnostic value.[24,25]

To elicit information on the vitality of a tooth, pulp testing is valuable. While instruments may be employed for this, there are various clinical methods which are adequate in most cases.

Dental Focal Infection

The crux of this situation is intimately related to a number of conditions frequent in dental practice. It is to be regretted that in many of our medical schools they receive little or no attention. Space will not permit as much consideration as they deserve. Some of the more important conditions are briefly discussed.

Caries, i.e., dental decay is the major problem of dentistry. From such cavities, leading directly to pulp involvement, it may readily be appreciated how bacteria and infection can gain entrance (the most frequent pathway of entrance) to the pulp and the systemic circulation. When evidences of infection are open, i.e., in direct communication with the outside world, drainage is more easily established and the dangers of systemic or distant involvement are less. The lamina dura

when intact, indicates to many the absence of periapical infection.

Periodontoclasia (pyorrhea) represents another major problem in dentistry. Many papers have been written on "pyorrhea" as a probable dental focus and this thought cannot be dismissed lightly. Three possibilities as to the modus operandi should be mentioned: Absorption of bacteria or their products directly into the systemic circulation from pockets; an exudation of purulent or infected material into the mouth and subsequent swallowing of such infected material; lastly, interference with mastication as the result of loose teeth. While the positive proof in support of any of these contentions is difficult, appreciation of the fact that most of the lesions in this condition are open lesions, at least diminishes the probability of absorption of bacteria or their products directly into the systemic circulation. This was one of the main contentions of William Hunter.[2]

In pyorrhea, the chief pathological features are: alveolar resorption, pocket formation, loosening of the teeth with or without suppuration. Usually, calculus and evidences of infection in pockets are present.

Retained Roots. Many statements concerning the danger of such roots are found. The cause of the root retention necessarily influences its danger. Information from the history, as to the probable cause and especially a knowledge of the length of time such retention has been present will be very helpful in evaluating such a focus. Where roots have been retained for years and the x-ray is negative or possibly shows condensation in contrast to a radiolucent area, less consideration as to the probability of an active focus of infection seems justified.

Dead Tooth. When a tooth is traumatized severely so as to completely sever its periodontal attachment with obvious resultant death of the pulp (some reserve the term dead tooth for such a condition) this may or may not act as a dental focus. Usually, such injury will result in loss of the tooth within a short time after the accident. In such cases on account of open socket and free drainage if infection does occur the local defense will usually be adequate. In some cases, however, certain changes may usually be expected, resulting in resorption of the root and eventual loss of the tooth.

It cannot be denied that inadequate or unsatisfactory root canal therapy, sometimes the fault of the dentist, often the neglect of the patient, may be responsible for dental foci. As a result of recent encouraging advances especially in root canal therapy in prevention, diag-

nosis and treatment,[26, 27, 28, 29, 30, 31] we are of the opinion that all pulpless teeth should not be needlessly sacrificed on the altar of focal infection.

Radicular—Granuloma—Cyst. The next group is also frequent and important. As a rule they appear as more or less well localized radiolucent areas as a result of destruction. Their infectious nature cannot be denied. They are practically all associated with, or due to, devitalized teeth, sometimes a tooth which has had root canal therapy, but more often has not.

They are, in contrast to some of the others we have shown, closed lesions; they represent infection, possibly of a latent type, and are thereby a menace.

The more localized, the less the danger; but neither can it be denied that they are very frequently present without active local clinical evidences and in many individuals whose apparent health is excellent, and who remain so indefinitely.

Pulp stones are not uncommonly found in one or more vital teeth. While it is claimed they are of infectious origin,[32] dental foci, or frequently the cause of neuralgia, the evidence appears more against than in favor. Dr. S. Sorrin, one of my associates on the Dental Service at Montefiore Hospital, advises me that in the dental examination of 200 cases of arthritis of various types (composed for the most part of cases of infectious arthritis) he found: 100 were edentulous; 53 had less than 20 teeth and 47 had 20 teeth or more. In these 47, isolated pulp stones (4 teeth or less) were present in 15 patients.

While pulp stones may be composed of dentine or calcium, based upon an understanding of pathological changes in such conditions they are interpreted as degenerative rather than inflammatory.[33]

Periosteal condensation, osteosis and osteoma. It is not always easy to decide whether these changes are dystrophic, neoplastic or inflammatory. As said previously, in general, when of inflammatory or infectious origin, we interpret them as bone scarring and believe they represent evidence of a successful or adequate local defense.

Secondary Manifestations

An extensive list of conditions has been attributed to dental foci. The organs involved include: joints, muscle, nerves, kidney, heart, eye, gastrointestinal tract, nasopharynx and gall bladder. From this list there are three upon which to comment:

I. The possible or probable relation of dental infection to heart disease is so extensive that comment will be made only on the relation of extractions to endocarditis. Recently, several reports [34, 35] on subacute bacterial endocarditis developing within a short time after extraction of (infected) teeth have appeared. In a personal communication, I. Salman, Oral Surgeon and S. P. Schwartz, Cardiologist, of Montefiore Hospital reviewed 215 hospital cardiac cases of rheumatic, arteriosclerotic and syphilitic patients from whom a total of 1126 teeth had been extracted under local anesthesia. No cases of subacute bacterial endocarditis followed.

II, III. Nasopharynx and Eye: More direct mechanisms and pathways of extension have been emphasized by several authors in affections of the antrum[36] and eye.[37] In striking contrast to the other parts of the body, the pathogenesis strongly indicates a direct extension or direct pressure on dental nerves rather than the usually accepted explanation of hematogenous spread.

By better medico-dental coöperation (and lately there have been encouraging evidences of it), greater progress can be made in the prevention, diagnosis and treatment of dental foci of infection. Sometimes, however, it is well to remember that although the ravages of infection may be incapable of repair, the progress of infection may be arrested and the source eradicated; if such cases cannot be improved, at least they should not be left otherwise.

Finally, lest I be misunderstood in my attitude toward bacteriology, let me paraphrase the words of Brutus, It is not that I love Bacteriology less but Pathology more.*

REFERENCES

1. Zinsser, H., Enders, J. F. and Fothergill, L. D. *Immunity; principles and application in medicine and public health.* New York, Macmillan, 1939, p. 2.
2. Hunter, W. Oral sepsis as a cause of disease, *Brit. M. J.*, 1900, *2:*215.
3. Hunter, W. Oral sepsis as a cause of disease in relation to general medicine, *Brit. M. J.*, 1904, *2:*1358.
4. Hunter, W. Rôle of sepsis and of antisepsis in medicine, *Lancet,* 1911, *1:*79.
5. Smith, D. T. Rôle of fusospirochetal organisms in acute and chronic infections of the mouth, *J. Am. Dent. A.*, 1936, *23:*1340.
6. Barrow, W. H. Systemic reaction to oral fuso-spirochetosis without local lesions, *Ann. Int. Med.*, 1938, *12:*508.
7. Cameron, G. C, Rae, C. A. and Murphy, G. N. Blood cultures and focal infec-

* I acknowledge with pleasure valuable advice and material from Julius A. Klosterman, Associate Professor of Bacteriology, New York University College of Medicine and New York University College of Dentistry; Abraham L. Greenfield, Professor of Radiography, New York University College of Dentistry; Morris Cohen, Assistant Professor of Pulp Canal Therapy, New York University College of Dentistry.

tions, *Canad. M. A. J.*, 1931, *25:*131.
8. Seifert, E. Über Bakterienbefunde im Blut nach Operationen, *Arch. f. klin. chir.*, 1925, *138:*565.
9. Barrington, F. J. F. and Wright, H. D. Bacteriæmia following operations on the urethra, *J. Path. & Bact.*, 1930, *33:* 871.
10. Bartlett, F. H. and Pratt, J. S. Streptococci isolated from excised tonsils and post-tonsillectomy blood cultures, *Am. J. Dis. Child.*, 1931, *41:*285.
11. Fischer, J. and Gottdenker, F. Über transitorische Bakterieneinschwemmung in die Blutbahn nach Tonsillektomie, *Wien. klin. Wchnschr.*, 1936, *49:*177.
12. Fox, H. and Forrester, J. S. Clinical blood cultures, *Am. J. Clin. Path.*, 1940, *10:*493.
13. Okell, C. C. and Elliott, S. D. Bacteriæmia and oral sepsis, *Lancet*, 1935, *2:* 869.
14. Fish, E. W. and MacLean, J. Distribution of oral streptococci in the tissues, *Brit. Dent. J.*, 1936, *61:*336.
15. Elliott, S. D.: Bacteriæmia and oral sepsis, *Proc. Roy. Soc. Med.*, 1938-39, *32:*717.
16. Richards, J. H. Bacteremia following irritation of foci of infection, *J.A.M.A.*, 1932, *99:*1496.
17. Round, H., Kirkpatrick, H. J. R. and Hails, C. G. Further investigation on bacterial infections of the mouth, *Proc. Roy. Soc. Med.*, 1935-36, *29:*1552.
18. Burket, L. W. and Burn, C. G. Bacteremia following dental extraction. Demonstration of source of bacteria by means of a non-pathogen (Serratia marcesens), *J. Dent. Research*, 1937, *16:*521.
19. Csernyei, J. Anacoric effect of chronic periapical inflammations, *J. Dent. Research*, 1939, *18:*527.
20. Robinson, H. B. G. and Boling, L. R. Anachoretic effect in pulpitis; bacteriologic studies, *J. Am. Dent. A.*, 1941, *28:*268; and *Personal communication.*
21. Rault, C. V. Value of routine blood sedimentation tests in dental patients, *Mil. Surgeon*, 1938, *83:*132.
22. Morgenroth, E. [Methods and evaluation of blood sedimentation test in stomatogenic focal infection], *Deutsche Zahnärzt. Wchnschr.*, 1938, *41:*1137.
23. Gutzeit, K. Zur Frage der dentalen Infektion und ihrer Diagnosestellung durch Kurzwellenprovokation der Zähne, *München med. Wchnschr.*, 1938, *85:*164.
24. Held, H. R.: Short waves in dentistry, *Province dent.*, 1937, *23:*297.
25. Wolf, H. F. Short wave diathermy in dentistry, *Dental Outlook*, 1940, *27:*170.
26. Hatton, E. H., Jiminez, F., Lenberg, J. V. and McMurrough, W. Comparison of roentgen ray, transillumination and bacteriologic examinations of pulpless and infected teeth, *J. Am. Dent. A.*, 1936, *23:*190.
27. Blayney, J. R. Present-day evaluation of the pulpless tooth, *J. Am. Dent. A.*, 1936, *23:*533.
28. Grossman, L. I. Changing concept regarding pulpless teeth, *J. Am. Dent. A.*, 1937, *24:*1928.
29. Auerbach, M. B. Clinical approach to problem of pulp canal therapy, *J. Am. Dent. A.*, 1938, *25:*939.
30. Dixon, C. M. and Rickert, U. G. Histological verification of results of root-canal therapy in experimental animals, *J. Am. Dent. A.*, 1938, *25:*1781.
31. Grossman, L. I. Present status of the pulpless tooth, *Ann. Int. Med.*, 1939-40, *13:*1805.
32. Seybold, J. W. Pulp stones and devitalized and impacted teeth as a factor in systemic disease, *J. Am. Dent. A.*, 1939, *26:*1627.
33. Stones, H. H. Histologic study of investing tissues of teeth, dentine and dental pulp, *Brit. Dent. J.*, 1938, *65:* 737.
34. Palmer, H. D. and Kempf, M. Streptococcus viridans bacteremia following extraction of teeth, *J.A.M.A.*, 1939, *113:* 1788.
35. Hopkins, J. A. Streptococcus viridans: bacteremia following extraction of teeth, *J. Am. Dent. A.*, 1939, *26:*2002.
36. Shea, J. J. Infections of the paranasal sinuses of dental origin, *Surg., Gynec. & Obst.*, 1938, *66:*408.
37. Kirby, D. B. Dental infection and the eye, *Ann. Dent.* 1939, *6:*65.