PRESENT CONCEPTIONS OF DENTAL FOCAL INFECTION

CHARLES A. RESCH, D. D. S.

The significance which has been attached to the rôle of dental focal infection in the causation of systemic diseases has led to extremes in its application in many cases, with the result that its importance has been doubted by those practitioners who attempt to justify therapeutic measures by rational rather than by empirical methods. The hypothesis of this interrelationship is complex and a comprehensive knowledge of the physiology and pathology of the body as a whole is essential before the problem may be evaluated properly.

The purpose of this paper is to attempt to correlate the observations which have been made and the present conceptions of the relationship between dental infection and systemic disease. To these will be added the results we have obtained in attempting to apply the observations made by investigators in this field.

The theory and principles of focal infection are not new. Historians record such consideration by the Egyptians some 2600 years ago, and Hippocrates is said to have recommended the removal of decayed teeth for the relief of rheumatism. In 1818 Benjamin Rush[1] of Revolutionary fame, and one of the signers of the Declaration of Independence, reported cases of systemic disease which he ascribed to dental infection and about which he wrote, "I have been made happy by discovering that I have only added to the observations of other physicians in pointing out a connection between the extraction of decayed and diseased teeth and the cure of general diseases . . . When we consider how often the teeth when decayed are exposed to irritation from hot and cold drinks, and ailments from pressure by mastication, and from the cold air, and how intimate the connection of the mouth is with the whole system, I am disposed to believe that they are often the unsuspected causes of general, and particularly nervous diseases . . . I cannot keep from thinking that our success in the treatment of all chronic diseases would be very much promoted by directing our inquiries into the state of the teeth in such people and advising their extraction in every case in which they are decayed. It is not necessary that they should be attended with pain in order to produce diseases." Rush's ideas would have been just as timely a hundred years later, and it is well to remember that he wrote long before the advent of the roentgenogram.

Following Rush, many other eminent clinicians associated dental sepsis with systemic disease. Black[2] states that, as early as 1839, cases of dental diseases that were related to lesions of the eyes were

reported in the literature, and Garretson[3] in 1890 described various systemic derangements, such as spasms, skin diseases, diarrhea, and "irritation," which he ascribed to dental origin.

In 1891, C. W. Miller[4], an outstanding bacteriologist, wrote, "During the past few years the conviction has gradually grown continually stronger among physicians as well as dentists that the human mouth as a gathering place and incubation of diverse pyogenic germs performs the significant rôle in the production of various disorders of the body, and that if many diseases whose origins enveloped in mystery could be traced to this source, they would be found to have originated in the oral cavity."

These early clinicians were limited to clinical observations only, and even without the aid of roentgenograms, they recognized the relationship between abnormal conditions of the mouth and certain systemic conditions. The reason for such an association could be explained only by the direct extension of the infection into the surrounding tissues by way of the blood stream thus producing a septicemia, by the absorption of pus and bacteria through the alimentary system, or through reflex irritations from dental pulps. In 1896, König[5] made demonstrations of dental radiography. Upson[6] in 1909 attempted to establish a relationship between dental lesions and serious mental disturbances. Two years later, Pancoast[7] attempted to establish a relationship between dental effects and idiopathic epilepsy, and Sparrevohn[8] suggested the possibility that impacted third molars might be an etiological factor in the production of Ludwig's angina. This work introduced the era of dental radiography as an aid in diagnosis and it initiated many lines of investigation.

As interest in dental radiography increased and as improvements were made in technic, members of both the medical and dental professions began to place greater emphasis on dental sepsis as a causative focus in systemic infections. This marked the beginning of a broader concept of focal infection, and this was increased by the definite attempts of bacteriologists and other scientists to place the hypothesis on a basis of rational therapeutics rather than on the empirical observations which had prevailed prior to the use of roentgenology and bacteriology. At approximately this time, William Hunter, an eminent English internist, presented a paper at Yale University entitled, "The Rôle of Sepsis and Asepsis in Medicine."[9] In this paper he severely criticized the American dental profession for its part in producing "mausoleums of gold" in the mouths of the American people. He felt that under these "mausoleums" were the ideal culture media for the growth of the particular bacteria associated with the anemias and nephritis. To Hunter, the problem was not one of teeth and conservative dentistry, but of

sepsis and asepsis, with conservative dentistry assuming the rôle of septic dentistry, incubating infection. Needless to say, this paper aroused a storm of protest from the dentists of America, but in the end, it served to stimulate interest in the scientific study of dental sepsis.

Frank Billings[10] and his associates in Chicago published the first truly scientific papers concerning this problem. In 1916, by careful correlation of clinical, bacteriological, and experimental findings, they were able to present some apparently definite facts concerning the relationship of systemic diseases and periapical infections. The more universal use of radiograms for the diagnosis of oral infection was strongly urged. With the inauguration of this method of research, other clinicians entered the field and since that time, the medical profession has led the way, and the dentist has assisted by providing clinical observations and the results of roentgenographic studies. Rosenow and later Haden presented carefully controlled and substantiated experimental work which has made the general acceptance of the hypothesis more secure.

Before discussing the experimental studies on this problem, a definition of the condition should be stated. Billings' early definition of focal infection was simply, "a circumscribed area of infection," but this has been elaborated in the following manner: "focal infection is a chronic process beginning in some epithelial defect and later involving remote parts of the body by establishing new bacterial colonies or by toxemias, and caused chiefly by Streptococci and sometimes Staphylococci." This definition has been formulated by Hatton and his associates[11] of Northwestern University.

Rosenow[12], in a series of experiments, was able to demonstrate that organisms in chronic foci vary greatly in their affinity for different tissues of the body, and he emphasized that a focus is not only an area in which organisms multiply and enter the blood stream, but also a place where tissue affinity is acquired. By studying the migratory movements of bacteria in an electrical field he has since endeavored to determine the reasons for this elective localization.

He found that streptococci from similar sources in patients with different types of disease had a definite variation in cataphoretic time and velocity. Of particular interest were his observations of the shift of the cataphoretic time and velocity of streptococci isolated from the nasopharynges and apices of the teeth of patients suffering from arthritis, encephalitis and other diseases.

However, the question in which the dental practitioner is vitally interested is, how can we demonstrate with the diagnostic means now at hand that dental infection actually exists? The answer simply stated is—such a condition cannot actually be so demonstrated. Rosenow's

work has shown the complexity of the ramifications surrounding the problem. If it were possible to classify the histopathological phase by microscopic study of the tooth in question and to examine the tooth and tissue without sacrificing either, the solution might be obtained.

Haden[13] approached this problem from the bacteriological viewpoint. He attempted to correlate the bacteriological and roentgen findings. He found that in 500 pulpless teeth, which were radiographically negative, 46.2 per cent contained cultures of ten or more colonies. Of 500 pulpless teeth which were roentgenographically positive, 62.8 per cent on culture contained ten or more colonies. In the controls, 400 vital teeth were examined and on culture, but 4.8 per cent had ten or more colonies. Viewing his results from a negative aspect, we find that of the positive pulpless teeth, 26 per cent were negative to bacterial growth in deep agar culture medium. Of the negative pulpless teeth, 44.3 per cent were found to be negative in deep agar culture medium. Of the vital control teeth, 85.5 per cent were found to be negative

In explanation of the 4.8 per cent of the vital teeth in which ten or more colonies were cultured, it is necessary to again refer to Rosenow's work in which cultures of Staphylococcus albus were planted in the anterior vital teeth of dogs. After a period of time, extraction of these vital teeth revealed the presence of the organisms in the pulp canals and at the apices of other nonvital teeth in situ which had not been contaminated experimentally. After a longer period of time, other sound teeth that were removed were found to be sterile, but the nonvital teeth extracted later remained contaminated. From this work it was concluded that all teeth are affected metastatically by bacteria if such bacteria have access to the general circulation. However, vital teeth in general have the power of overcoming invasion, provided the general resistance of the host is normal, while nonvital teeth apparently do not have this ability or resistance and remain as a source of continued infection.

Haden and Rosenow recognize four possible groups of dental focal infection: (1) The positive and negative pulpless tooth with chronic periapical infection, (2) pyorrhea alveolaris and pockets surrounding partially erupted teeth and under poor restorative appliances, (3) chronic pulp infection in vital teeth, and (4) residual alveolar infection following dental extraction.

Rhoads and Dick[14] extracted radiographically negative pulpless teeth from all parts of the mouth and studied them bacteriologically. They concluded that it seemed justifiable to regard all pulpless teeth as probable foci of infection whether or not radiographs showed changes in the apices, and they further emphasized that this is true in the presence of systemic disease of a type which usually is associated with focal infection.

Recently, we had the opportunity of removing two teeth from a hospitalized patient. One was a lower left bicuspid which was devitalized and showed definite evidence of periapical involvement with a large area of rarefaction, and beyond this there was condensing osteitis. The other tooth, which also was devitalized, was an upper left lateral and it was absolutely roentgenographically negative to any apparent periapical pathosis. Both teeth were cultured by our bacteriologist, Mr. Reich, who used a brain agar culture medium and brain broth culture as contrast. Considerably more than two hundred colonies of bacteria grew on the agar culture. The bacteria from both teeth after incubations for 24 hours were later proven to be Streptococcus nonhemolyticus. The profuse growth of a single species of bacterium on agar ruled out contamination. The essential point here was that the removal of the definitely radiographically positive teeth would not have excluded all the possible factors of dental focal infection.

The Northwestern University group[11] recognize the following primary dental foci: (1) The periapical involvement which is primarily of metastatic origin or the result of a bacteremia in which a focus is set up at points of weakest local resistance, (2) pyorrheal infection which is primarily toxic and not a true lateral abscess, but a circumscribed one caused by poor drainage and a low grade chronicity, (3) the pulp infections or a localized abscess in an otherwise normal pulp, which is predominately toxic and is the most potent factor in dental focal infection. In this group, the symptoms of discomfort are present but often referred. Listing the vitality of such a tooth may show a reaction varying from normal to sluggish (hypesthesia) to hypersensitivity. The tooth usually shows a large restoration and in such instances, the radiogram is of little diagnostic value. Pulp stones may be recognized and be the only visual clue roentgenographically. The fourth class recognized by the group at Northwestern is the partially erupted third molar with its recurring pericoronitis and its tendency to become toxic. These workers have studied focal infections not only from the bacteriological standpoint, but also from the histopathological angle and present slides of the microscopic picture found at the apices of these teeth in situ. Areas of periapical rarefaction do not necessarily mean infection. This group has been able to demonstrate areas of rarefaction at the apices of teeth in which there was no round cell infiltration or bacteria.

One particular phase of this type has been investigated by E. C. Stafne[15] who applied the term "periapical osteofibrosis" to the condition, which is the forerunner of the cementoma and is most prevalent in the anterior region of the mandible.

Blayney[16] demonstrated, by microscopic sections of apical tissue, that devitalized teeth with root canals incompletely filled may be entirely free from all indications of inflammation or bacterial invasion. He showed

a carious lower first molar with definite roentgen evidence of periapical involvement around both roots and, histologically, an area of inflammation and destruction of the pulp in the gingival or middle portion of the mesial root. Other microscopic studies of the same tooth showed that the periapical tissues did not show the slightest sign of inflammation or degeneration. His conclusions were that bacteriological study of root ends wherein the inoculum is obtained after extraction is very misleading and that for a reliable study, the material must be collected before the root is disturbed. He also concluded that only roentgenographic evidence of root resorption in the absence of bony changes is not sufficient to justify the extraction of teeth so involved.

In the investigations which we have conducted, we have not had the opportunity to study microscopic sections and have relied on the bacteriological method. Approximately eighty teeth have been cultured, and fifty-seven or 71.3 per cent showed pure cultures of nonhemolytic streptococci; four were of the viridans type. The majority of these teeth were devitalized and there had been some therapy to the root canal. Radiographically negative and positive teeth were present in about equal numbers. Six of the cultured teeth were negative, two having been removed because of peridental involvement. Others gave cultures of streptococci but contamination was apparent because other organisms were reported to be present in the culture. The wide variation in our findings from those of Haden may be explained partially on the basis that our patients were suffering from some systemic disorder and, due to the metastatic nature of organisms and the sites of lowered local resistance, we would expect to find a greater number culturing bacteria.

In the group which we studied there was an upper bicuspid. This was a devitalized tooth, the root canal of which was well filled, and no evidence of periapical involvement was demonstrable roentgenographically. The patient, a young woman, suffered from acute nephritis; some low grade infection was suspected but could not be demonstrated. In the absence of any other etiological factor, the questionable tooth was removed and cultured. The organisms were injected into a rabbit with the result that no demonstrable lesion was found after four days when autopsy was performed. Subsequent reports from the patient, however, have revealed no further recurrence of the systemic symptoms.

In conclusion, since it has been shown bacteriologically and clinically that the teeth and oral sepsis play an important rôle in the general health in the majority of cases, we should attempt to eliminate the practice of extracting teeth that do not appear to be causative factors in the production of systemic dyscrasias. No tooth should be removed on the mere assumption that it is the definite cause of the complaint, because it is a recognized fact that the products of such teeth may act as sensitiz-

ing agents and, therefore, should be considered a secondary rather than a primary cause. Indeed, I feel safe in regarding the majority of infectious teeth in this light rather than as etiological factors. However meager may be our total means of evaluating such conditions, the application of "snap diagnosis" is to be less condoned than extolled as a sensible economy. The most thorough diagnostic studies at our command must be employed. From the dental standpoint, these include a history of dental discomfort, a complete digital examination of the mouth including a test of the vitality of the teeth, and a complete roentgen examination. Roentgen examination of two suspicious looking teeth for the purpose of determining the general condition is inexcusable from the standpoint of conscientious and thorough practice and is usually worthless from an economic viewpoint. If, after a complete survey, there is evidence of abnormality of the oral structures, the age, the position of the tooth and its proximity to important structures, the condition of the tooth, the importance the patient attaches to his teeth as to care and condition of the mouth, and finally the general complaint of the patient in addition to evidence of low grade infection, determine whether a suspicious tooth should be extracted. It is in such problems that dentistry and medicine merge, and only after the fullest coöperation between both professions does the final therapeutic result justify the measures attempted. In dealing with these problems, it is not entirely a question of how much infection is present, but how well can we ascertain that such conditions exist. Unless a complete understanding of the condition can be procured before the teeth are condemned, many normal teeth will be sacrificed and even with all due caution, teeth that may be causing no trouble will be removed, but this number will become smaller with continued observation, study, and more complete records.

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