Chapter VII
The Role of Focal Infection

The Stress-Adaptation Syndromes developed by Selye and our study of the vegetative nervous system give us the physiology and morphology of disease mechanisms as a whole. Focal infection is important to the dentist both because it is an independent pathogenic influence within the body and because it is a source of localized stress. It is not possible in all instances of pathology which depend upon dental structures to determine the relative amount of influence of the stress-adaptive process and of focal infection. Both must be considered.

I. The Nature of Focal Infection

Billings gave the first definition of a focus of infection:

A focus of infection may be described as a circumscribed area of tissue infected with pathogenic organisms. Foci may be primary or secondary. Primary foci usually are located in tissues communicating with a mucous or cutaneous surface. Secondary foci are the direct result of infections from other foci through contiguous tissues, or at a distance through the blood stream or lymph channels. Primary foci of infection may be located anywhere in the body.¹

More recently, Solis-Cohen elaborated this concept by pointing out that “a focus is bacterial and not structural.”²

Recent writers are careful to distinguish between focal infection and a focus of infection. Shuster states that

...focal infection implies metastasis from the infected foci, of bacteria or their toxins, capable of injuring contiguous or distant tissues.³

Curtis suggests that:

...a focal infection is a localized or generalized infection caused by the dissemination of organisms or toxic products from a focus of infection. It probably would be better defined as a metastatic infection.⁴

II. The Significance of Foci of Infection

Through the years, there has been considerable discussion, pro and con, on the theory of focal infection. Several men have questioned whether a focus is capable of the wide-spread somatic damage which is ascribed to
it. Eustermann, in 1924, summarized the principal ailments which were then considered to be dependent upon focal infection:

Patients with acute articular rheumatism, subacute or chronic infectious endocarditis, chronic myocarditis, chronic infectious arthritis, appendicitis, gastric or duodenal ulcer, chronic cholecystitis, iritis, glomerulonephritis, pyelonephritis, neuritis, neuralgia, herpes zoster, myositis, erythema nodosum, symptomatic purpura, vague aches and pains in the muscles and joints, certain types of headache, anaphylactic phenomena of a cutaneous visceral nature, and disturbance in function of organs supplied by the autonomic nervous system are quite generally suspected of harboring foci of infection.5

Several items in Dr. Eustermann’s report are interesting in light of our present knowledge of the adaptive processes of the body. The reader will note that some of the items mentioned in Eustermann’s list have also been shown to be diseases of adaptation according to Selye’s Stress-Adaptation Syndrome, and the latter has demonstrated the mechanism of several of these as it results from stress. This would seem to reinforce the importance of foci as sources of stress. At the time of the publication of Eustermann’s work, the pathways from focus to disease were not known entirely, and it was presumed that bacteria or toxins were carried mechanically through the body. Now it is possible to postulate that bacteria need not be transported from the focus to the site of systemic disease, since the adaptive processes of themselves could account for a breakdown in these areas. For this reason, the literature no longer lists several of the diseases mentioned by Eustermann as the result of focal infection.

The degree of importance granted to the theory of focal infection has varied through the years. In 1938, Cecil commented:

Many of us who originally accepted the theory of focal infection with enthusiasm have watched with interest and some trepidation its rapid development in the various fields of medicine but are now wondering if the time has not arrived for a reevaluation of the whole theory. Many thoughtful students today question seriously its validity, and some are quite willing to throw it overboard...Scientific men are becoming a little wearied of the universal acceptance of a theory as though it were an established fact.6

In 1940, Reiman and Havens stated:

It may be said, therefore, that (a) the theory of focal infection, in the sense of the term used here, has not been proved, (b) the infectious agents involved are unknown, (c) large groups of patients whose tonsils are present are no worse than those whose tonsils are out, (d) patients whose teeth or tonsils are removed often continue to suffer
from the original disease for which they were removed, (e) beneficial effects can seldom be ascribed to surgical procedures alone, (f) beneficial effects which occasionally occur after surgical measures are often outweighed by harmful effects or no effect at all and (g) many suggestive foci of infection heal after recovery from systemic disease or when the general health is improved with hygienic or dietary measures.⁷

Despite these differences of opinion, contemporary medical thinking does not entertain serious doubt that foci of infection have widespread, harmful effects within the body. In 1950, Kern summarized the principal arguments in favor of the focal infection concept:

1. Primary foci may be found in association with secondary systemic disease, such as tuberculosis, syphilis, gonorrhea, brucellosis, and sarcoidosis, which frequently have no other cause to account for secondary disease. Recognized clinical improvement may occur in the systemic or secondary disease when the primary focus is eradicated.

2. There is evidence of transient bacteremia in patients with primary foci and secondary systemic disease. Moreover, when a focus of infection exists, mechanical factors favor recurrence of bacteremia, e.g., pressure of the jaw muscles, compression of the tonsils in swallowing, and stresses of the prostate.

3. There is selective affinity of certain bacteria for certain tissues.

4. It is well known that exacerbations of arthritis, myositis, and iridocyclitis may follow surgical procedures on the primary focus.

5. Recognized clinical improvement may occur in the systemic or secondary disease when the primary focus is eradicated.

6. A patient may be rendered vulnerable to bacteremic episodes by lowering of his defense mechanisms.

7. Conditioning factors such as traumatized tissues, fatigue, exposure, malnutrition, anemia, avitaminosis, mental depression, and particularly disease of the leukipietic system result in secondary disease far more readily when a focus of infection is present. For example, the flora of the colon can kill the host when the body defenses are lowered by radiation.

8. Some persons are more susceptible to infection than others, and it is in these especially that secondary infection takes place.⁸
III. The Mechanisms of Focal Infection

The principal subject of research and the source of the controversy over the theory of focal infection is the determination of the pathways by which a focus of infection can affect the body, even at locales considerably removed from the focus. Appleton has summarized the mechanisms of focal infection. This summary was made in 1949 and reiterated by Dr. George Coleman in 1953. It seems subject to some modification in the light of the work of Selye, Whatmore and others but it presents a view of the classically accepted mechanisms of focal infection.

1. Bacteria may be discharged from the focus into a free surface whence, conveyed by mechanical means, they determine an extension of the disease by reinoculation.

2. Bacteria may be conveyed to distant parts of the body by way of the lymphatics or the blood stream. They may be arrested in the nearest lymph nodes, leading to lymphadenitis or even to abscess formation. If the bacteria pass this barrier, one of three things may happen: They may multiply in the blood stream. They may be conveyed alive to a suitable nidus, where they will multiply and infect the surrounding tissues. They may, by the process designated by Adami as “subinfection,” produce a slow, but progressive atrophy with replacement fibrosis in various organs of the body.

3. Products of bacterial metabolism may reach and damage remote parts of the body.

4. Bacteria at the focus may undergo dissolution. Dissolution products diffusing into the blood or lymph may sensitize various tissues of the body in an allergic sense, and later liberation of dissolution products may result in an allergic reaction. On the other hand, if absorption of bacterial toxins is prevented by chronic inflammatory changes or by encapsulation of the primary focus or if upon absorption the toxins are effectively neutralized by natural or acquired antitoxins, ill health is not to be expected.

Selye objects that it is still something of a mystery as to how focal infection works. The difficulty is that the development of focal infection is quite unpredictable. At times, a localized infection will be followed by rheumatic heart disease or arthritis, and at other times by nephritis or some other change in organs far removed from the site of infection. Sometimes surgical removal of the infected focus (for instance, the teeth or tonsils) leads to a cure and sometimes it does not. It is difficult, therefore, to prove a causal relationship between a localized infection and the disease-manifestations elsewhere in the body.

In 1955, Selye and his associates began to research the connection between focal infections and the Adaptive Syndromes. Some difficulty was encountered in the early research due to the problem of producing
constant, identical infections for experimental purposes. However, it was soon learned that rats given repeated, subcutaneous injections of various putrefied proteins experienced a syndrome characterized by endocarditis and myeloid infiltrations in various organs, particularly the spleen, the renal pelvis, and the liver. This group of changes became known as the Focal Syndrome, i.e., a syndrome of systemic disorders which seem also to be not-specific in the sense that they result from focal infection regardless of its location. In this syndrome, there were certain major, indicative symptoms: 1) endocarditis, 2) nephritis-like renal lesions, 3) adrenal changes which were not merely non-specific manifestations of the general stress reaction, and 4) a marked tendency for ectopic myelopoiesis in the spleen, liver, and adrenals.

A further and very significant development was the discovery that this same set of symptoms could be produced by overdosage with somatotropic hormone (STH) without the presence of a focus. Furthermore, the syndrome could be controlled by the proper injections of corticoids. This suggested to Selye that the events which follow upon foci of infection – the Focal Syndrome – might not be specifically the result of the foci themselves but rather a case of improper adaptation of the body to the invading organism.

Two other studies are of importance in consideration of infections. In 1955, Stoerk presented a study of the relationship of immunization to cortisone and the lymphoid tissues of the body. His summary is as follows:

From the sum of these observations, it is apparent that the cellular components of lymphoid tissue play an important part in the development of immunity. The suppression of lymphoid cells by cortisone or other agents is associated with an inability to form circulating antibody of the classic type. However, for the duration of the lifetime of antibody gammaglobulin, humoral immunity is not dependent upon lymphoid cells and persists even after nearly complete elimination of these cells. The situation is necessarily different in those instances where immune activity is absent from the body fluids and is entirely dependent for its persistence upon viable cells, capable of circulation. In the case of delayed hypersensitivity (immunity), significant reduction of these cells by cortisone should be followed by prompt diminution of immunization, i.e., sensitization, and thereby account for the anti-allergic action of the steroid.10

Thus, we can glimpse the interaction of the adaptive processes and the processes of focal infection. In 1962, G.J. Therbecke and B. Benaurraf reported research which gives greater light to the reaction of the body to pathologic foci. Their findings can be summarized as follows:

1. Reticulo-endothelial organs are involved in the defense against disease.

2. Specific immunity to organismic attack, and therefore to allergy is mostly bound to humoral components formed by reticulo-endothelial tissues.


3. Non-specific immunity is regarded as being mediated by the reticulo-endothelial cells proper.

4. In experimentation, antibodies have been synthesized \textit{in vitro} by spleen, lymph nodes, lung, and bone marrow tissue. However, attempts to localize the site of antibody formation intracellularly have been unsuccessful to date.

5. Plasma cells (mature and immature) have been shown to contain antibody.

6. Upon the introduction of antigens and thus upon the formation of antibodies certain histological and morphological changes have been observed. The lymphoid tissue of spleen and lymph nodes display phenomena leading to the appearance of enlarged secondary nodules. A complex process at the site of disturbance takes place involving interaction of both white and red lymphoid pulp.

7. We may say changes in the number and function of cells of the reticulo-endothelial system seem to account for many of the modifications of natural resistance to allergenic materials.

8. It is highly significant that researchers have found that adrenal corticoids have been able to inhibit both primary and secondary antibody production.\textsuperscript{10}

\textbf{IV. A Summary of the Theories of Focal Infection Mechanisms}

A survey of this material presents the following generalizations:

1. Foci of infection are important etiologic influences in a number of systemic disorders.

2. There are definite connections between a focus and the general adaptive processes of the body.

3. The body's reactions to topical irritants, such as a focus, are controlled by the reticulo-endothelial system understood in a broad sense.

4. Heavy dosage with glucocorticoids decreases the antibody-producing faculties of the body. This has significance when we consider the combination in one person of infection and stress. As will be elaborated later, this is the apparent case in the patients who present themselves to the dentist.

5. A considerable divergence of opinion exists as to relative function of foci and their precise mechanisms, but few will deny their importance as pathogenic influences upon the system.
V. Treatment of Focal Infection

Because of the well-established concept of the part played by bacteriology in disease, and because of excellent success obtained through the use of the sulfonamides and antibiotics, there has been a tendency to overrate the usefulness of these drugs and to regard them as the only curative measure necessary. Although their use may contribute effectively to the eradication of a focus, they usually are not striking at the cause of the infection. Therefore, in many cases, antibiotics are no more than mitigating agents. Their importance must not be minimized, but they are not the ultimate in the treatment employed by the thorough dentist. They will be used to bring the situation under control, but the true cause of the pathology must be sought.

VI. Comment

Recent literature accords less and less importance to foci of infection within the dentition itself. Most researchers feel that the eradication of any focus within the body is of benefit to the patient in general, but that this eradication cannot be expected to alleviate the systemic conditions in all cases.

Infections of the middle ear and especially of the respiratory system are a principal concern of the present work. Abraham Ribicoff, as Secretary of Health, Education, and Welfare, stressed the importance of respiratory disorders in quoting medical statistics which stated that 91% of the diseases of teenagers are respiratory illnesses and that the “shock troops” of the armies of disease are acute respiratory ailments.

To what extent these disorders are the cause of systemic difficulty through the classical mechanisms of focal infection or as a source of stress, it is impossible to say. They may, in fact, be an effect rather than a cause. The fact remains that they routinely exist in malocclusion patients and that they are greatly ameliorated or completely eliminated with the correction of the occlusion and the consequent alleviation of muscular imbalance and malarthrosis of the temporomandibular joint, as the author’s research has demonstrated repeatedly. Thus, foci of infection must be given careful consideration in the total health care extended to all patients by the dentist.

References


15. Fonder: The role of the dental physician, *op. cit.*