

THE RELATIONSHIP OF ARTHRITIS
AND PERIODONTAL DISEASE
A SURVEY OF THE LITERATURE

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ABSTRACT

The purpose of this study is to locate as much of the already published materials on periodontal disease and arthritis as possible to see if some type of relationship exists between the two. There was no laboratory investigation conducted; only a survey of the literature was done. This survey of the literature shows that many similarities exist between arthritis and periodontal disease. A gradual body of deterioration accompanying these conditions seems to affect one's resistance to infection which is a key step in the process of deterioration. It appears that neither arthritis nor periodontal disease causes the other, but evidence of the previous investigations leads to the conclusion that a common condition or link exists between the two.

GENERAL BACKGROUND

Joints are delicate structures which readily respond to injurious stimuli. In a long life there is much stress and strain, not to mention abuse such as that caused by athletics. Needless to say, diseases of the joints are extremely common. Weight bearing joints; i. e., lower limbs and vertebral column, suffer the most.

The joints are composed of six structures: the articular surfaces of the bone covered by hyaline cartilage, the epiphyses, in some cases the epiphyseal cartilage and metaphysis, and the synovial membrane. "The most striking feature of the microscopic appearance of intercellular substances over cells, the chondrocytes lying quietly in lacunae in the ground substance" (Boyd 1974:1344). Nourishment is of great importance to the articular surface, because the articular cartilage is devoid of blood vessels. "The lateral articular area is furnished with a delicate perichondrium continuous with the synovial membrane and containing numerous capillaries, but the central area possesses no perichondrium, the surface being formed of clear matrix containing no cells" (Boyd 1974:1344). Nourishment, therefore, is dependent entirely upon the synovial fluid.

If the natural function of the joint is tampered with, this low threshold of nourishment must be kept in mind. When the joint is placed in a forced position, clinical evidence shows joint degeneration due to inadequate diffusion of nutritive fluid. A lesion results, and usually cannot be repaired, for it may interfere with the growth of epiphyses or become the starting point of osteoarthritis.

The protein content of the synovial fluid is thought to be the key to the correct functioning of a normal synovial membrane, the amount of protein here being only half the amount contained in the lymph. This probably accounts for the feeble powers of resistance and the readiness of degeneration of this part of the articular cartilage and its absence or slowness of repair.

There are two kinds of joints: the first is fibrocartilaginous joints and the second is synovial joints.

FIBROCARTILAGINOUS JOINTS In this case, skeletal components are united by fibrocartilage, which is separated from the bones on either side by thin plates of hyaline cartilage. The classic example is the intervertebral disc which unites, but separates, two vertebrae. The disc of an adult is avascular and depends on diffusion for its nutrition, probably through the plates of hyaline cartilage.

SYNOVIAL JOINTS These types of joints consist of two cartilage covered bones joined in a mutual embrace. This type of joint is designed for free movement. Naturally, the major joints of the legs and arms fit into this category.

Articular cartilages are separated from each other by a thin layer of synovial fluid. When the joint is at rest, the articular surfaces are in direct contact. With increased speed in movement, pressure builds up and the articular surfaces then become separated by a lubricating fluid which absorbs or inhibits friction. The viscosity of the synovial fluid is due to hyaluronic acid, a non-sulfated mucopolysaccharide. A condition known as "use-destruction" or "wear and tear" always occurs during movement and this results in the gradual wearing away of the articular cartilage. Movement of heavy loads and decrease in viscosity which accompanies ageing

accentuate this process. "Osteoarthritis may be an exaggerated form of 'use-destruction' in which the metabolism of the synovial fluid and the articular cartilage play a part of prime importance" (Boyd 1974:1344-1345).

Diseases of the joints can be classified thus:

- A. Acute arthritis
 - 1. Suppurative
 - 2. Non-suppurative
- B. Tuberculous arthritis
- C. Chronic arthritis
 - 1. Rheumatoid arthritis
 - 2. Ankylosing spondylitis
 - 3. Psoriatic arthritis
 - 4. Osteoarthritis
- D. Gout
- E. Tumors
- F. Other arthropathies (Brothwell 1967:353).

ACUTE ARTHRITIS Suppurative arthritis is one type of acute arthritis. Many cases of this sort usually affect the knee and hip joints. Staphylococci and streptococci are common infecting organisms for suppurative arthritis. However, in the intervertebral joints, tubercle bacillus, Brucella organisms, typhoid bacillus and syphilis are the usual infections (Brothwell 1967:353). These organisms can enter the joint by any one of three means:

- " (1) by bloodstream transmissions from a distant focus of infection
- (2) by the extension of an infection (osteomyelitis) from nearby bone, or
- (3) by direct introduction from a penetrating wound" (Morse 1969:15).

When the infectious organism enters the joint initially, an edema of the synovial membrane is caused. Joint fluid is increased, and swelling and hyperemia occur. The disease can stop here and hence no damage occurs. If the disease continues, a substance is formed which is able to destroy cartilage (Morse 1969:15). Many times, the articular cartilages may be eroded and underlying bones exposed. Ligaments tend to soften causing the joint to become disorganized and dislocated. When the infection is a mild one, only

the synovial membrane is affected and no destruction of the joint occurs. If there is no tissue destruction; i.e., destruction of articular surfaces, a fibrous union will be cartilaginous in nature. However, if the articular surface is destroyed, the fibrous union will be bony.

A second type of acute arthritis is non-suppurative. This includes most acute arthritides. Inflammation is confined to the synovial membrane and there is no tissue destruction or permanent joint damage (Brothwell 1967:353).

TUBERCULOUS ARTHRITIS Tuberculous arthritis will not be dealt with here because of its irrelevance to the nature of this study.

CHRONIC ARTHRITIS Chronic arthritis is the arthritis with which most people are familiar. By the word chronic, this does not mean to imply a chronic joint inflammation. Rather it means a slowly progressive disease. Here the disease is separated into four specific conditions.

The first is rheumatoid arthritis. This condition is generally common in women from the ages of 20-40 years. This progressive disease begins with the small joints, then later afflicts the larger joints. At any time the disease can be arrested, but joint changes can never be reversed (Brothwell 1967:354). At first, inflammation of the synovial membrane occurs. This in turn produces a series of swelling, thickening and scarring which produces deformities and limits motion (Morse 1969:13).

The disease may stop at synovitis or, as usually occurs, the articular cartilage is involved.

"The synovial membrane grows over the articular cartilage from the side and forms a thick vascular covering (pannus), which adheres to the cartilage and erodes it. The cartilage is damaged by granulation tissue which is formed in the superficial layers of the epiphysis as part of the inflammatory reaction. The cartilage is destroyed

and adhesions form between the two layers of the pannus covering the articular surfaces, and the joint cavity may be obliterated. Fibrous ankylosis of the joint develops and bony ankylosis may follow. In the rheumatoid process there is no development of osteophytes at the joint margins" (Brothwell 1967:354).

A second type of chronic arthritis is ankylosing spondylitis. This disease is more commonly found in males 15-35 years of age than in women. A polyarthrititis is characteristic of this disease. Ossification occurs in the outer fibers of the annulus fibrosus (the outer portion of the intervertebral disc) and also in the apophyseal, costo-vertebral and sacro-iliac joints (Brothwell 1967:354). Sometimes there is a bony fusion of the symphysis pubis. It is not necessarily true that other joints in the body are involved, though some degenerative disease may be detected. Many degrees of ankylosing spondylitis exist. They may range from involvement in the sacro-iliac joints to fusion of the entire spine. Osteophytes sometimes occur but usually the bony connection between the joints has a smooth undulating appearance which is somewhat conformed to the spinal countour (Morse 1969:15). There is a slow loss of the exact lumbar curve and development of thoraco-lumbar kyphosis is gradual and continual. Calcification and ossification continue until the typical picture of a "bamboo spine" results (Brothwell 1967:354).

A third type of chronic arthritis, psoriatic arthritis is diagnosed from soft tissues only and will not be discussed here.

Lastly, in the category of chronic arthritis there is osteoarthritis. This degenerative joint disease, also known as degenerative arthritis is as common in men as in women, especially the form which affects the hip joint. There are no general symptoms yet the disease generally is associated with the aging process (Boyd 1974:1359).

"Although efforts have been made to use the degree of osteoarthritis as a measure of age, any such estimation would be only approximate and quite unreliable due to the great variation in individuals to the time of development and the extent of degenerative joint disease" (Morse 1969:13).

So far studies have shown no evidence that a toxic factor is involved. Mainly the large joints are affected, but in some cases, joints of the hands and feet are involved. Swollen, knobby knuckles are not uncommon.

'Heberden's nodes, which are much commoner in women, are often present. These are small bony outgrowths at the sides of the terminal phalangeal joints. There is a striking difference from the usual sex incidence of osteoarthritis. This seems to depend on a single autosomal gene, sex-influenced to be dominant in women and recessive in men, with the onset in women being closely related to menopause" (Boyd 1974:1359).

The nodes start out containing a small bead of mucoid material. This arises as the result of degeneration of the periarticular soft tissue with subsequent ossification (Boyd 1974:1359).

Osteoarthritis can be characterized by five statements:

1. Osteoarthritis is a degeneration of articular cartilage and bone.
2. Cells of cartilage degenerate and the smooth surface becomes rough.
3. The cartilage cells swell, burst and disappear.
4. Cartilage is gradually worn away and underlying bone becomes exposed. (This also degenerates, but the latter undergoes condensation and hardening).
5. Cartilaginous excrescences are formed at the articular cartilage margins and cause lipping of the joint edge. They increase the available articular surface and may be compensatory. They tend to ossify so that atrophied bone is surrounded by a ring of excrescence which restricts movement. These changes have typical radiological signs (Brothwell 1967:355).

Osteoarthritis is one of the oldest known diseases. It was present in our primitive ancestors as evidence shows in bones from Nubian caves dating back 8,000 to 10,000 B. C. Lesions have also been found in bones of pre-diluvial dinosaurs. Hence, the great occurrence of this condition in present day populations cannot be entirely blamed on modern conditions of life. The cause of the disease is not known.

"It is a slow involutonal process often associated with marked arteriosclerosis so that local ischemia may play a part. It is always difficult to form a correct judgment on the relation of trauma to any pathological process, but the common idea that trauma is an etiological factor especially in hip joint disease, appears reasonable" (Boyd 1974:1359).

Three other types of arthritis exist. They are gout, tumors

and a category of "other arthropathies". However, because of the nature of this study, a description and an analysis of these conditions will be omitted.

Investigations in all areas of arthritis confirm that patients usually suffer from a general deterioration of health. This may include sluggishness in the vital functions of the organs, incomplete digestion of food, impaired elimination of toxins and metabolic waste from the body, a weakened nervous system and circulatory system only to mention a few.

"These systematic disturbances affect the biochemical structure of various tissues of the body. One of the pioneer practitioners of biological medicine in the United States, Dr. R.P Watterson, calls the result of such systemic disturbance a 'biochemical suffocation'. One of the most characteristic pathological changes observed in rheumatoid arthritis is the degenerative changes in collagen. The changes in collagen — the connective tissues of the body, the inter-cellular cement — are affected by biochemical changes brought about by metabolic disorders or nutritional deficiencies. The resultant accumulation of the fibrous tissue in the joints and the accumulation of toxic wastes and mineral deposits completes the picture of a fully developed arthritis" (Airola 1968:30).

It has been said that nutrition is one of the most important causative factors in the development of arthritis. This is to say that an unbalanced diet will cause a general deterioration of health, biochemical imbalance and systemic disturbances. These conditions eventually lead to a great deal of metabolic disorder, thus allowing pathological changes in other body tissues and systems to occur (Airola 1968:57).

PERIODONTAL DISEASE

"The effect of systematic factors has been implicated primarily in the form of periodontal disease designated as 'periodontosis' " (Kerr 1962:302.) There is somewhat of an enigma concerning periodontosis because it is usually defined as a form of periodontal disease resulting from systemic factors even though the systemic factors are not recognizable. Also, there is no consistent rela-

tionship between the development of periodontosis and the existence or development of a specific disease.

Many systemic factors and even some specific diseases have been suggested in the etiology of both periodontosis and periodontitis (Kerr 1962:302). Both specific and general nutritional factors have been frequently suggested. For example, in laboratory animals, a deficiency in protein has been shown to produce periodontal changes and others have suggested from clinical investigation that protein deprivation plays an important part in the progress of periodontal disease.

"If we consider periodontal disease a 'chronic destructive process' of the periodontium, which progresses at various rates, depending on the individual's ability to repair the damaged tissue, one would expect the progress to be more rapid in any condition which influences repair" (Kerr 1962:302).

Clinical evidence has shown that a deficiency of protein delays body repairs. Therefore, if a person is unable to perform the normal procedures of repair because of a deficiency in protein, periodontal disease will progress more rapidly than in a person whose reparative procedures are normal. However, it is important to remember that no evidence has shown that dietary deficiency is the initiation point of periodontal disease (Kerr 1962:302-303).

Vitamin C deficiency is a nutritional factor which is of primary consideration to researchers. A deficiency of Vitamin C does not initiate periodontal disease. Rather Vitamin C is a necessary component in the normal metabolism of endothelium and connective tissues and it could influence the course of periodontal disease. In the face of chronically destructive periodontal disease, a Vitamin C deficiency will prevent repair of the periodontium so that the disease may proceed much faster in an individual with this condition (Kerr 1962:303).

Although periodontal diseases vary, they can simply and satisfactorily be classified as:

1. Gingivitis
 - a. Chronic
 - b. Infective
 - c. Hyperplastic
 - d. Hormonal
 - e. Desquamative
 - f. Necrotizing
 - g. Allergic
2. Periodontitis
3. Peridontosis
4. Occlusal trauma (Bhaskar 1972)

Usually, the disease begins in the marginal and intergingivae and continues apically. The only exceptions are periodontosis and lesions which accompany occlusal trauma. These two conditions are abnormal changes beginning in deep structures. Today, investigators believe that gingivitis and periodontitis start as a sticky bacterial mass (plaque) on the tooth surface.

Gingivitis is an inflammation of the gingiva. Several types of this inflammation exist.

Chronic gingivitis is the most common form and is universal. The cause is from local irritation from conditions such as plaque (Bhaskar 1973: 182-183).

Infective gingivitis occurs by means of an infective agent such as streptococcus. Inflammation causes the gingiva to be swollen, red and painful. Sometimes the mucosa is affected by the inflammation (Bhaskar 1973:184).

Hyperplastic gingivitis Under certain conditions, abundant growth occurs in the gingiva which covers varying amounts of the crowns of the teeth. If the gingiva covers the teeth entirely it may form a pseudopocket. This is different from a true pocket which is associated with loss of bone and periodontitis. The major part

of the swollen gingiva is composed of collagen bundles. This condition can be caused by use of certain drugs or from hereditary factors (Bhaskar 1973:186).

Hormonal Gingivitis occurs during the adjustment or alteration of sex hormones. This includes times such as pregnancy and puberty. The gingiva at these times becomes swollen, red or bluish red, puffy and bleeds easily. Pseudopockets may form here. Lesions usually begin in the interdental papilla and later involves the marginal gingiva (Bhaskar 1973:188-189).

Desquamative Gingivitis usually occurs in women about the time of menopause. Here the epithelium lifts and leaves raw areas which bleed easily (Bhaskar 1973:190).

"Necrotizing Gingivitis is also referred to as ulcerative gingivitis, ulcero-membranous gingivitis, Vincent's gingivitis, fusospirochetal gingivitis, and oral fusospirochetosis" (Bhaskar 1973:191). This condition is caused by a vibrio organism, a bacillus (*Bacillus fusiformis*) and a spirochete (*Spirochaeta vincenti*). The clinical features include swollen, red gingiva, fever, malaise, cervical lymphadenopathy, and necrosis of the interdental papillae (Bhaskar 1973:191).

Allergic Gingivitis occurs in the marginal and attached gingiva. Most cases include red, swollen gingiva with a granular appearance. However, there is not a great deal of discomfort accompanying this condition (Bhaskar 1973:194).

Periodontitis is any condition where the inflammatory process extends from the gingiva to the bone underneath. Bone destruction and pocket formation are a common condition which follows this process. The cause can be any number of local irritations such as faulty restorations, poor oral hygiene, calculus and malocclusion.

"Clinically, periodontitis is characterized by changes in gingival color, loss of stippling, edema, hyperplasia or recession, formation of clefts, presence of true pockets, which may exude pus on pressure and tooth mobility" (Bhaskar 1973:194-195).

Periodontosis is a degenerative disease which is common in the Mid East, Near East, and Far East in women of child-bearing age. In the beginning there is migration of teeth that leads to diastemas, to extrusion and to malocclusion. At first, a crevice may be shallow and free of inflammation. Later stages see the appearance of deep pockets (Bhaskar 1973:203-204).

Occlusal Trauma This results in changes in supporting tissues. The causes range from malocclusion to some pathology of the supporting tissues:

"Microscopic changes in the areas of pressure are narrowing of the periodontal space and compression and necrosis of collagen fibers, thrombosis of the blood vessels of the periodontal membrane, and differentiation of osteoclasts on the bone surface and resorption of bone. Bone resorption occurs until pressure is relieved, and then a small amount of new bone is formed to re-embed the fibers of the periodontal membrane" (Bhaskar 1973:207).

"Terms such as gingivitis, periodontal pocket, and alveolar bone loss describe clinically detectable, pathologic results rather than etiologic components or early developmental stages" (Grigsby and Sabiston 1975: 175-176). A helpful mechanism for the prevention of periodontal disease would be to describe in specific terms the initiation and development of periodontal disease. This is exactly what Grigsby and Sabiston did in their investigation on the Periodontal Disease Process. They formulated this process by analyzing the chain of events which result in detectable periodontal lesions.

Periodontal disease is initiated and sustained by the interaction of several oral environmental components.

These components which are essential are (1) the periodontal tissues, (2) bacteria, and (3) nutrient materials. Although the existence of these three components simultaneously does not insure the periodontal disease to erupt, they do have to be present if the disease is to initiate at all. There are three reasons for this.

1. Periodontal Tissues -- Here the simple fact exists that unless there is a periodontium there can be no periodontal disease.

2. Bacteria -- Bacteria is essential to the progress of periodontal disease. This statement comes from observation of experiments done on humans and animals.

"Miller (1890) wrote that 'as regards the participation of bacteria in phorrhoea alveolaris, our present knowledge of suppurative inflammations compels us to consider the former as the cause of the suppurations incident to this disease'. Keyes and Jordan (1964) and Jordan and Keyes (1964) described an infectious agent, Actinomyces viscosus, that induced periodontal lesions in hamsters. Rovin et al. (1966) demonstrated histologically that conventional microbe-bearing rats exhibited significantly greater periodontal inflammatory responses than germ free rats. . . . Kelstrup and Gibbons (1970) isolated a Streptococcus salivarius designated 1 A from dental plaque in a human oral cavity. This organism induced gingival inflammation and alveolar bone loss in monoinfected, sucrose-fed rats while sucrose-fed, germ free rats did not exhibit detectable periodontal lesions. Loe et al. (1965) observed that humans who suspended all oral hygiene activities exhibited increased dental plaque and subsequently exhibited detectable inflammatory changes in their gingival tissues. Human experimental subjects not performing mechanical cleaning procedures eliminated their gingival inflammation by daily rinses with an antibacterial agent, 0.2% chlorhexidine gluconate. Loe and Schoitt (1970). Loesche et al. (1971) and Loesche and Nafe (1973) found that topical application of a 5% kanamycin paste to the gingival environment of humans reduced the gingival index scores for these patients" (Grigsby and Sabiston 1976:176-177).

3. Nutrients -- If indeed bacteria is a necessary component of the periodontal disease process, then a supply of nutrients must be available to meet the metabolic requirements of the disease producing organism.

PHASES OF THE PERIODONTAL DISEASE PROCESS

The interactions of the three components necessary for the periodontal disease process can be divided into five phases:

1. The Inoculative Phase
2. The Proliferative Phase
3. The Stationary Phase
4. The Destructive Phase
5. The Iterative Phase

"The term phase is used to describe the interactions of the essential components of the periodontal disease process and to emphasize the transitory nature of these states" (Grigsby and Sabiston 1976:177).

THE INOCULATIVE OR BACTERIAL ATTACHMENT PHASE

The beginning of periodontal disease is the bacterial colonization or infection of the dentogingival environment. Mechanisms to explain this phase are categorized using interaction between acquired pellicle and bacteria, interaction between tooth substance and the bacteria, and the interaction between the gingival tissue and the bacteria.

THE PROLIFERATIVE OR INCREASING CELL NUMBERS PHASE

Solcransky et al (1971) showed that in a clean, intact tooth the inoculative phase can occur at the rate of 10^6 bacterial organisms attached per square centimeter of tooth surface in about five minutes. However, this rapid rate of colonization does not continue after the first five minutes. This implies that the bacterial attachment phase does not last more than a matter of minutes or hours at the most. However, this first phase itself will not induce a detectable periodontal lesion (Loe et al. 1965 from Grigsby and Sabiston 1976:180).

The second stage, the proliferative phase, is the point at which the bacteria increase their numbers in the dentogingival

environment. This increase can be brought about by cell division or by a secondary attachment of bacteria that colonize the bacteria already present (Grigsby and Sabiston 1976:180). The time that is necessary for this phase is probably a matter of days.

"However, the proliferative phase is limited since a specific dentogingival site can support only a finite number of bacterial cells due to factors such as nutrient supply, forces of attachment, and displacement forces generated during mastication" (Grigsby and Sabiston 1976:181).

The proliferative phase is probably not a continuous activity in the human mouth. This point of view is substantiated by the fact that

"plaque functions more like a non-growing bacterial culture than a rapidly growing one. This suggestion is strengthened by the observation that only 4% of the coccoidal organisms in plaque exhibited signs of cell division, in contrast to 35% of the organisms in exponential growth cultures" (Saxton and Critchley, 1970, from Grigsby and Sabiston 1976:181).

Hence, the stationary phase of the periodontal disease process is that one in which there is no net increase in the number of bacterial cells in the dentogingival environment. Observations have shown that each of the preceding three states (inoculative, proliferative and stationary) can occur at the same time within different microenvironments on the same tooth. These phases must also be recurring.

THE DESTRUCTIVE PHASE

The proliferative phase and the stationary phase usually come before the destructive phase, but it can also accompany them in the periodontal disease process. "This phase occurs when the pathologic changes such as widened intercellular spaces between adjacent sulcular epithelial cells develop within the periodontal tissues" (Thilander 1968, from Grigsby and Sabiston 1976:182).

THE ITERATIVE PHASE

Lastly, there is the iterative or repetitive phase. Here the first four phases are repeated each time that bacterial plaque is moved from its dentogingival environment (Grigsby and Sabiston 1976:184).

"The oral cavity is analagous to other body cavities in that they are all governed by the same physiochemical laws and physiologic principles" (Sukin 1975:26). All of them have the same source of nutrition and are protected by the same antipathologic system. Because the mouth benefits from the general defense mechanism of the body, one can take a look at the mouth and get a pretty dependable picture of the state of systemic health. Many times, the first sign of disease process in other parts of the body will manifest themselves in noticeable changes in the oral cavity. This is especially true in relation to nutritional deficiencies, endocrine and gastro-intestinal disturbances and anemia (Sukin 1975:26).

Years ago when one went to a physician complaining of an illness, neither a cause nor a cure could be found in many instances. Hence, the doctor would recommend the teeth be removed. The reasoning for this was that the physician believed that the primary focus of infection in the mouth was the cause of the medical problem. Today, removal of the teeth is not necessary in order to cure the oral infection and eliminate or severely reduce the medical problem.

"A focus of infection may be defined as a circumscribed area of tissue infected with pathogenic organisms. Focal infection connotes the invasion of the body from a focus of pathogenic organisms having the power of reproduction and multiplication within its host" (Sukin 1975:27).

There are four pathways by which infection and passage of the primary focus to the secondary area can take place. These are (1) transmission by means of the bloodstream, (2) transmission by means

of the lymph system, (3) direct contact with the tissues, and (4) direct contiguity between the gastro-intestinal and pulmonary tracts -- by means of swallowing and breathing infectious material.

TRANSMISSION THROUGH THE BLOOD STREAM

The gingiva is supplied with blood from a rich supply of vessels within the periodontal membrane, alveolar bone, and connective tissue. This permits a ready exit for infectious organisms and toxins from the periodontal pocket (Sukin 1975:27). If a tooth is loose, it may act as a piston to force the organisms into the blood stream (Comroe 1947:371). Once they have made their way into the blood stream, the organism can attack anywhere there is lowered resistance to a predisposing factor.

TRANSMISSION THROUGH THE LYMPHATIC SYSTEM

The gingiva is also supplied with an abundant supply of lymph. There are many connections between the various lymph nodes. Hence, the infection could spread from any part of the head to the neck and through the body cavity very easily.

TRANSMISSION BY DIRECT EXTENSION WITHIN THE TISSUES

Infection by this means can occur three ways. The first is the area which is limited within the bone itself. This creates a true osteomyelitis. More frequently this condition occurs in the lower jaw.

The second is similar to the first case except the infection is not localized. Infection passes out of the bone into surrounding soft tissue forming abscesses which may extend to the eye (Sukin 1975:27-28).

Lastly .

"transmission along the fascial planes is important because they enclose the various muscles, glands, blood vessels and nerves and

it is by way of the interfacial spaces which are filled with loose connective tissue, that infection descends. It is important to remember that there are fascial connections between head, neck, and mediastinum and that infection may spread from the head down to the chest. It spreads along the facial planes because they are resistant and encompass the pus in these areas" (Sukin 1975:28).

TRANSMISSION INTO THE GASTROINTESTINAL AND PULMONARY TRACTS

The mouth is a direct access to the rest of the alimentary canal, the nasal cavity and the respiratory system. Free pus moving back from the infected periodontal tissues may be swallowed, or inhaled into the bronchi or lungs. There is the possibility of it becoming lodged in the nasopharynx. The result of this being nasopharyngitis, pharyngeal infections and lung abscesses. Swallowing of this material may cause gastrointestinal disturbances (Sukin 1975:28). A good example theory to fit this type of infectious movement would be arthritis. The exact causes of arthritis are not easy to pinpoint. The most popular explanation has been metabolic disturbances (Airola 1968:115). However, most researchers now feel that the main villain is a disturbed balance of intestinal bacteria. Under normal circumstances, only beneficial substances can pass through the intestinal walls. However, when a bacterial imbalance impairs the permeability, toxins damage the mucous membranes and allow toxic substances to penetrate the walls. Thus, toxins can infect the whole organism, not excluding the areas of connective tissue around the joints. All investigations show that pathological changes leading to arthritis are associated with a general breakdown in the resistance of the body (Airola 1968:115).

The toxins now deposited in the joints cause infection. The infection is a defensive mechanism against the toxins which have entered the system. This infection results in pain and swelling. Joints surfaces are damaged by decalcification, and the joints are either repaired or replaced by calcification. This calcification will eventually cause a total fusion of the joints and deformation,

and immobility soon will follow (Airola 1968:116).

This theory of periodontal tissues being an important foci of infection is of great significance to paleopathological literature. Ruffer (1921) noticed that a significant number of instances of dental disease and spondylitis deformans and chronic arthritis occurred in ancient Egyptian and Nubian skeletons. Moodie (1928) found an association between arthritis and dental diseases he studied. However, at about the same time Keith (1925) found situations quite different. Some of the skeletons had both teeth and joints affected by pathological change. Others had potential oral foci and healthy joints and still others had no oral foci but possessed pathological joints.

"A possible relationship between osteoarthritic changes in the temporomandibular joints and dental disease has been suggested by Strauss and Cave (1959), Poitrat-Targowla (1962) and Köttschke (1960). Taylor (1963) studied collections of Moriori and Maori skulls in which, according to him, oral sepsis was prevalent as a result of excess attrition with pulp exposure and periapical osteitic foci as sequelae. In skeletons of the Moriori Duckworth (1900) was struck by the frequency of osteoarthritic changes. Taylor interprets this as lending support to the hypothesis that the two conditions are related" (Brothwell 1967:585).

Møeller-Christensen and Brinch (1948) and later Inglemark, Møeller-Christensen and Brinch (1958) examined later Mediaeval Aebelholt skeletal material in search of a possible correlation between oral foci in the form of periapical osteitis and changes in the spinal joints. The skeletons studied were all of mature or senile individuals. Degenerative changes were found in the spinal joints.

"Møeller-Christensen and Brinch found that 17% of the individuals had joint changes in the cervical part of the vertebral column and oral foci as determined by the authors in dentitions without ante- or post-mortem loss of teeth" (Brothwell 1967:585-586).

In addition, skeletons where oral foci were present, the incidence of individuals also having joint changes was 93%. This led to the

conclusion that a causal relationship existed between pathological changes in the cervical part of the column and certain foci of dental infection in this Mediaeval skeletal population (Brothwell 1967:586).

Some investigators have suggested that arthritis in the temporo-mandibular joints and periodontal disease were somehow related in European Neandertal Man. This theory extends as far back as Choquet (1909) and Boule (1911-1913).

"Choquet and Boule found pathological alterations in the dentition belonging to the La Chapelle-aux-Saints skeleton and morphologically changed articular surfaces of joints at several places in the skeleton, including the temporo-mandibular joints. The alveolar changes were called polyarthritis alveolar-dentalis, which was then the usual term for periodontitis" (Brothwell 1967:573).

As far as can be determined, the pathological joint changes were osteoarthritic in nature.

Given information that we know today, little can be used as evidence of a causal relationship between periodontitis and the temporo-mandibular joint. One cannot say from evidence of periodontal disease that arthritis was present in the temporo-mandibular joint, thus explaining alveolar changes in the mandible of the adult Ehringsdorf skeleton as the result of a common infection of the periodontium and joints (Virchow 1920). When Virchow made his investigation, it is very possible that he misinterpreted Choquet's term polyarthritis alveolar-dentalis. Recently, Köttschke (1960) studied alveolar and joint changes in Neandertals. Using the Krapina mandible in addition to the skull from La Chapelle-aux-Saints, he stated these as evidence that the theory that the periodontal disease succeeded a joint disorder (Brothwell 1967:573).

In 1966 an expedition from the University of Michigan headed by Dr. Harris, went to Egypt to study the mummified remains of Pharaohs and ancient nobles held in exhibit by the Egyptian Museum. The

project was conducted by the University of Michigan's School of Dentistry in conjunction with members of the museum staff. Five seasons of work were conducted on the mummies. The purpose of the study was to X-ray these remains so that further knowledge of their physical appearance, their diseases, life spans, techniques of mummification and family relationships could be ascertained. In the late 1800's and early 1900's Gaston Maspero and Elliot Smith made an anatomical examination of the mummies. Now, with the new investigation, a radiographic survey could be made of the Pharaohs and nobles who had never been unwrapped for one reason or another. These X-rays were very informative and much has been learned since the examinations (Harris and Weeks 1973:6).

The Pharaohs and nobles were not without their physical ailments when they were alive. Many of them had to suffer with the pain accompanying arthritis (Harris and Weeks 1973:130-131). Examinations showed Rameses II to have been one of those unfortunate suffering rulers. Rameses II suffered from severe dental wear and a great number of abscesses also. Many of the teeth were loose in the sockets and heavy pitting of the bone in this area indicates this to have been a condition which developed during his life. Also, the X-rays show that Rameses II suffered from severe degenerative arthritis and arteriosclerosis of all major arteries in the legs (Harris and Weeks 1973:155).

Rameses II outlived twelve of his one hundred sons. The thirteenth son, Merenptah succeeded him to the throne. His mummy was found in the tomb of Amenhotep II. The tomb of the latter was used as a cache to protect the mummies of rulers whose own tombs had already been violated by tomb robbers. Merenptah's mummy shows him as an old man. "Few mummies have shown so clearly the desira-

bility of X-ray studies, for few rulers suffer so many pathological conditions, almost all of which would be invisible without careful dissection" (Harris and Weeks 1973:157). The cervical vertebrae indicate severe degenerative arthritis and soft tissues show that Merenptah also suffered from arteriosclerosis.

Like Rameses II, his father, Merenptah had extremely bad dentition. What few teeth he did have at death show only moderate wear, but all molars and premolars were lost and evidence shows considerable bone loss and periodontal disease. Close examination of the areas of missing teeth show that the teeth may have been deliberately removed. If this was the case, this is the only evidence of dental surgery from dynastic Egypt. Many of the rulers suffered from conditions much like these two, when they lived long enough for the diseases to develop (Harris and Weeks 1973:158).

As has been established, no evidence has conclusively stated that the dental condition of patients who suffer from rheumatoid diseases is any different from those who have no type of rheumatoid affliction. More recent investigations have dealt with the relationship between rheumatoid diseases and periodontal disease. Some investigatory believe that the rheumatoid disease plays an etiologic role in the periodontal disease while others contend that a common factor may account for a predisposition to both diseases (Malestrom and Calonijs 1975:49).

In a study done by Malestrom and Calonijs, the dental condition of patients suffering from rheumatoid disease was compared, on the basis of roentgenologic examination, with dental condition of otherwise healthy outpatients. The data revealed that rheumatoid patients suffered teeth loss more often than control patients with a frequency that was statistically highly significant. Such findings cannot be regarded as other than a positive correlation between poor dental health and rheumatoid disease. The reason for the disproportionately greater loss of teeth among rheumatoid patients is not known (Malestrom and Calonijs 1975:52-53).

A possible explanation for the difference in tooth loss might

be the genetically determined hyperractive immunity system of patients suffering with rheumatoid disease. This results in the greater destructiveness in the periodontal disease process (Malestrom and Calonius 1975: 54).

Malestrom and Jokinen (1975) conducted an experiment closely related to the previous investigation.

To determine whether evidence of rheumatoid inflammation in the form of free rheumatoid factor might be found in the teeth-supporting tissues of patients with known rheumatoid disease, tissues from the dental periapical lesions of one group of 50 rheumatoid and 23 control patients and from the marginal gingiva of a second group of 58 rheumatoid patients were examined. Both dental periapical lesions and gingivitis occur in otherwise clinically healthy persons as well as in patients who suffer from rheumatoid disease. The etiology of gingivitis as well as of dental caries . . . is considered to be a bacterial infection (Malestrom and Jokinen 1975:121).

At the Rheumatism Foundation Hospital, Heinola, Finland, a total of 59 biopsies were taken from 50 patients suffering from periapical lesions. Of the 50 patients, 38 were inflicted with rheumatoid arthritis, 8 with ankylosing spondylitis and 4 from rheumatoid disease "post infectionem". Thirty-three of these patients were found to be seronegative as regards the rheumatoid factor. Out patients (25 total) from the Institute of Dentistry, University of Helsinki, comprised the control group (Malestrom and Jokinen 1975:121).

Between 60 and 90% of patients with rheumatoid arthritis have rheumatoid factor in their sera. In the present study, 38 of 50 rheumatoid patients suffered from rheumatoid arthritis. Of these 38, 43.7% were seropositive as regards rheumatoid factor. The remaining 12 patients of whom 8 had rheumatoid disease 'post infectionem', were all seronegative. The patients were selected at random and the sample contained by chance more seronegative than seropositive patients in the group whose dental lesions were studied. The results do not, however, provide any information about whether rheumatoid patients who are seronegative might have poorer dental health than patients who are seropositive (Malestrom and Jokinen 1975:123).

Thus the results from this study show that a free rheumatoid factor can be detected in the periapical lesions of patients suffering from rheumatoid conditions, but this is only slightly more than control

patients (Malestrom and Jokinen 1975:124).

CONCLUSION

The literature shows that some type of relationship or link between arthritis and periodontal disease exists "Medical researchers admit that they do not know what causes arthritis and consequently, do not know how to cure it" (Airola 1968:29). From evidence already known, if the two conditions are related, the focus of infection probably first exists in the mouth and manifests itself as periodontal disease. With such available pathways such as the bloodstream, lymph system, and the gastrointestinal tract, transmission of bacteria and/or toxins could easily make their way into any area of the body where there is a low resistance to infection. It is not unusual for both arthritis and periodontal disease to be associated with infectious organisms such as streptococcus. Arthritis and rheumatism have been considered as exerting an influence on the periodontium by way of impairment of nutrition and general and local resistance. The periodontium can actually be looked upon as a joint that can be affected by arthritis and rheumatism (Citron 1931; Arnett and Ennis 1933; Messing 1959). Since the joints are so low in protein and reparative procedures are slow, any increase in destructive processes would cause a degeneration in that area. It could be that the amount of protein is the predisposing factor which allows the infectious organisms to inflict their damage. However, a genetic predisposing factor could be responsible for this process. In addition to periodontal diseases and arthritis having some type of link, there is also implied a relationship between both the conditions and a general deterioration in the systemic health of the individual. Evidence has already shown that systemic disturbances affect the biochemical structure of various tissues in the body and this certainly

includes collagen, which is an important structure in both joints and the periodontium. At any rate, arthritis does not cause periodontal disease and periodontal disease does not cause arthritis. At most there is a general condition which allows for both to occur, or a predisposing factor is responsible for their appearance.

Actually, there has not been a great deal of extensive research which has been published on the relationship between arthritis and periodontal disease. Most of the material available indicates that there is indeed some type of link between the two conditions which might be helpful in finding a cure for each. There is still much research to be done in regards to these diseases. Everyone is aware of the effects of arthritis because it is a much publicized disease. To a lesser extent, the general public knows about periodontal disease. A probable area of productive research would be the intense investigation of the processes and relationships between arthritis and periodontal disease. Perhaps through this kind of parallel research, valuable information on cures could be reached and two painful conditions either reduced or eliminated in most cases.

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