THE PATHOGENESIS OF DENTAL DISEASE IN CARNIVORES

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I have several objectives in preparing this paper. The first is to present the general principals of the cause and effect relationships which lead to oral disease and dysfunction. The second is to demonstrate that the etiological agents of oral disease in exotic animals are so common, and so widespread, that among captive populations, the incidence of oral disease has become epidemic.

Oral and dental disease is one the most studied of all human ailments. For the sake of clarity, it is necessary to accept a nomenclature which accurately describes oral disease in animals. Although one would assume that to be a relatively simple task, there is a difference of opinion. In the late 1800 's dental disease was lumped into four categories: (1) caries, (2) pulp disease, (3) periodontal ligament disease, and; (4) diseases of the gums and supporting structures. By the mid 1950's, from a radiographic point of view, 23 categories of oral disease were described, and from a physiological prospective, one author described 26 distinct pathologic pathways to oral disease.

From a practical point-of-view, we should be familiar with this diversity. But, from a clinical point of view, we can adequately describe the oral disease found in animals with four basic categories, which are: (1) developmental and congenital defects, (2) maxilla facial trauma, (3) periodontal disease, and (4) trauma of the teeth. Oral disease results from a wide variety of circumstances, and the clinical appearances of the disease process vary considerably within a single species, and can vary enormously from one species to another.

In all animals, the inflammatory process is the bodies response to disease, and is always characterized by the five classic Greek signs of: rubor-red; calor-heat; tumor-swelling; dolor-pain; and functio lasis - loss of function. Howevert, the pathways of viral, fungal, bacterial, allergic or neoplasic disease may be

remarkably dissimilar.

The first category to be considered is DEVELOPMENTAL AND CONGENITAL DEFECTS. Tooth decay is not a major problem in exotic animals, but it does occur. For simplicity's sake, I've included dental caries within the category of developmental disorders, and will dismiss it with the observation that aside from gross enamel dysplasia, and the like, caries is essentially a result of significant, localized, diet-induced or developmental environmental change--a local rusty spot on a tooth. If the decay hasn't progressed to the point of causing irreversible pulpal necrosis, it can be restored from a functional point of view, and should present a relatively minor problem for the clinician.

Other developmental and congenital oral defects number in the hundreds. When they become clinically significant, all are readily identifiable by the loss of function of the masticatory apparatus in one way or another. When they occur, look for genetic patterns, environmental patterns like poisons, or toxic chemicals, and/or major nutritional imbalances.

The resolution of developmental or genetic defects is usually extremely difficult and represents an administrative, or genetic management problem as much as a clinical issue. As we become more successful with our efforts at captive breeding, I expect to see more of these types of problems, and would encourage the clinician to look for them, and to seriously consider bold and aggressive culling decisions whenever genetic defects are encountered.

The second category of oral disease in exotic animals that I'll consider is MAXILLA FACIAL TRAUMA. Essentially this group differs very little from the general orthopedic problems encountered by the clinician, except for the generalizations that: **first**, there are more essential and/or vital items per square inch of tissue in the head and neck than anywhere else in the body; **second**, nonunion of fractures is a major problem, particularly of the mandible; and traumatic edema causes considerable secondary tissue injury that can easily result in a compression obstruction of the trachea leading to further complications. Although it is a category unto itself, I don't believe it requires a great deal of separate discussion, and should be considered as just another branch of orthopedic problems.

The third category from an etiological point of view is PERIODONTAL DISEASE. Just about everything that happens in and around the oral cavity, either

directly or indirectly results in, or contributes to, the development of periodontal disease. The disease process is very well described in the literature, and any of the larger, bulkier text books should prove to be most helpful.

Periodontal disease is defined to include all of those disorders associated with the tissues surrounding and supporting the dentition, which include the gingiva, the periodontal ligament, and the alveolar bone. Although there has been innumerable volumes written on the subject, from a clinical point-of-view there still remains a good deal of confusion and misunderstanding. As a practical matter, the majority of dentists treating human patients delegate the problem to the dental hygienist, or periodontist.

The dentist can correct the problem once it is recognized, but he really can not do much about its recurrence, aside from patient education. Everyone has had, no doubt, the personal experience with the individual who never does anything with his teeth and never has any problems, or the individual who does everything possible with his mouth and continually has all of the problems in the book. This state of affairs leads to a good deal of ignoring of the problems in the hopes that they will go away, which it doesn't, and which results instead in a good deal of litigation relating to malpractice issues. If this is the state of affairs with human patients, it is quite apparent then, that the state of affairs with exotic animals is no better, and is no doubt, somewhat worse, particularly with captive groups.

Goldman, in his text on Periodontal Therapy, describes "**observation**" as the "hallmark of the resourceful, imaginative therapist," and he points out that:

"one cannot treat what does not exist, and for the casual observer there is much that does not appear to exist which is in reality present and waiting to be discovered."[1]

In order to recognize and diagnose the disease process the clinician must have an intimate and complete familiarization with the normal oral cavity and that will come only with experience and continuous observation. On the assumption that I can do little more than encourage one to become an observer, I'll devote my attention to etiology and pathogenesis in an attempt to aid in the understanding of the disease process.

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[1]Goldman, H. H., (Ed): Periodontal Therapy, Third Edition, St. Louis, C. U. Mosby Co., 1964, p. 67.

As an interesting historical aside, periodontal disease did not become common in humans until after the appearance of cooking of food. It has long been considered a preventible disease, whose etiology is well recognized as "attributable to injury of the gums caused by food of either a physical or chemical nature or of both simultaneously."[2] Both hard and soft foods contribute to the disease process. Hard foods by physical trauma, obstruction of free flow of the saliva after impaction, and as a vehicle to aid the process of fermentation. Soft foods, by sticking directly onto or into the irregularities in or about the teeth, and by providing the convenient source of sugars, starches, and cellulose which are necessary for the process of fermentation.

Periodontal disease is divided into two groups, based upon the etiological origin of the disease. The first is INFLAMMATORY and includes three sub-headings: 1) gingivitis, 2) marginal periodontitis, and a special acute exacerbation form known as, 3) ANUG, acute necrotic ulcerative gingivitis. The second group is DYSTROPHIC which includes: 1) disuse atrophy, 2) occlusal trauma, 3) gingivosis, and 4) periodontosis.

Gingivitis is the first step of periodontal disease, and is defined as inflammation of the fibrous tissues which cover the alveolar process of the upper and lower jaw and surround the necks of the teeth. The etiological factors which contribute to inflammatory gingivitis are: 1) abrasion, 2) local factors, 3) drug reaction/allergies, 4) hormonal, 5) systemic, 6) idiopathic.

Particular attention should be paid to the local etiological factors which are: (a) materia alba (plaque), (b) calculus, (c) irritating restorations, (d) food impaction, (e) infections, and (f) other causes.

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[2]Colyer, J. F.: Dental Disease in its Relation to General Medicine. Longhans, Green & Co., London, 1911, p. 60-67.

TABLE 1

DIAGNOSTIC SIGNS AND SYMPTOMS
FOR GINGIVITIS

1. Change in color
2. Change in form, position, surface texture
3. Retraction
4. Pocket formation
5. Bleeding
6. Presence of exudate
7. Osseous changes
8. Mobility
9. Migration
10. Malocclusion

Although described as a discrete clinical entity, MARGINAL PERIODONTITIS is more clearly understood as the logical extension of untreated gingivitis. Although it is described as an initial lesion, marginal periodontitis represents the clinical fact that the gingivitis stage has passed unnoticed, and the disease process has progressed through the protective layering of the gingival tissues and into the bony structures which support the teeth.

The term marginal denotes that the inflammatory lesion has extended to involve the marginal bony area of the teeth. This develops as a sequela to a chronic gingivitis and has the identical etiology. Clinical marginal periodontitis is an inflamation of the soft tissues with radiographically identifiable resorption of the

crest of the alveolar bone. Severe local trauma, chronic gingivitis, peristent occlusal trauma, and chronic noninflammatory or degenerative destruction of the periodontium usually all result in marginal periodontitis, which if untreated, will advance to local or generalized destructive periodontitis with osteomyelitis.

TABLE 2

ETIOLOGY OF MARGINAL PERIODONTITIS

1. Initial Lesion

2. Secondary Lesion

    a. chronic gingivitis
    b. occlusal trauma
    c. periodontosis

TABLE 3

ETIOLOGY OF PERIODONTAL DISTROPHY

1. Disuse

2. Occlusal trauma

    a. Bruxism
    b. Clenching
    c. Cribbing, etc.

3. Degenerative disease of the attachment apparatus

The second major group of periodontal diseases are the DYSTROPHIC lesions. These are characterized by a gradual wasting away of the size, or function of a component part, tissue, or cell – ie: a progressive degeneration or the site. A form of defective nutrition of a component part leading to its atrophy. For all practical purposes, there is very little recognizable clinical difference between inflammatory and dystrophic forms of periodontal disease. It is a rare case which will manifest a clear cut, text book set of signs and symptoms. But, it is important to understand the clinical entity, however, because the underlying etiological is degeneration from either: 1) no use of the dentition, 2) too much use of a tooth, or 3) one of a whole host of systemic degenerative diseases which embody oral manifestations either as primary or secondary signs.

Dystrophic periodontal disease has a particular significance with respect to captive groups of animals. Dorlan's Medical Dictionary defines the GENERAL ADAPTATION SYNDROME (G.A.S.) as "the total of all nonspecific systemic reactions of the body to the long, continued exposure to systemic stress." I believe this term adequately defines a state of affairs seen in captive groups of exotic animals, particularly carnivores, living in situations which create stress induced nervous displacement activities.

In humans, these symptoms manifest themselves in a wide variety of psychological circumstances. But in exotic animals, I see a primary manifestation as a chewing, biting, clenching and general gnashing of the teeth -- all leading to the increased tendency toward dystrophic periodontal disease. Gnashing and biting type nervous displacement activity is commonly associated the capture, confinement, and transport of exotic animals. When combined with the diet induced inclination toward inflammatory periodontal disease, particularly in older members of a breeding group, the animal’s GENERAL ADAPTATION SYNDROME is certainly contributing to an increase in the incidence of oral disease in that animal.

An investigator named Sorrin, in 1935, described in an article entitled, *"Habit: An Etiological Factor of Periodontal Disease,*" over fifty common occupational or neurosis induced habits which lead to periodontal disease. I suspect that dystrophic periodontal disease is on the increase, and an increase in observation of oral problems will reveal a much more accurate measure of its true incidence.

The fourth and final classification of oral pathology from an etiological

point of view is TRAUMATIZED TEETH. A little bit of trauma leads to chronic degenerative disease, a whole lot of trauma, results in fracture of the teeth. Although Ellis' text, *The Classification and Treatment of Injuries of the Teeth of Children*, describes 9 classes of trauma from simple fracture of enamel, to complete luxation of the tooth. For all practical purposes, we need really be familiar with only classes 1 through 3.

Class 1 and Class 2 fractures may not require intervention or treatment from a clinical point of view, but should be documented in the individuals medical record and re-evaluated radiographically in the future. Class 3 requires clinical intervention of either an endodontic or surgical nature. As a general rule of thumb, if a syringe needle will catch or stick into the discolored spot at the end of a fractured tooth, the case is a Class 3 problem, and treatment should be when discovered.

The bacteria in saliva continuously bathes the teeth, and, with time, will invade the pulpal tissues within the tooth and lead to an irreversible necrotic pulpitis. This will eventually result in an apical inflammatory response which results in the spread of infection along the peridontal ligament spaces, or through the blood supply to the adjacent teeth and tissues, and in time results in further oral problems. The solution is endodontic therapy which is described in detail in the literature.

TABLE 4

CLASSIFICATION OF TRAUMATIZED TEETH

Class 1	Simple fracture of the crown of the tooth involving little or no dentin.
Class 2	Extensive fracture of the crown--involving considerable dentin, but not the dental pulp tissue.
Class 3	Extensive fracture of the crown--involving considerable dentin and exposing the dental pulp tissue.
Class 4	The traumatized tooth which becomes non-vital -- with or without the loss of crown structure.

Class 5 Teeth lost as a result of trauma - - subluxation complete or partial.

Class 6 Fracture of the root-with or without loss of crown structure.

Class 7 Displacement of a tooth-without fracture of the crown or the root.

Class 8 Fracture of the crown in mass and its replacement.

Class 9 Traumatic injuries to the deciduous teeth, or primary teeth.

Oral disease is a subject, which has sustained an enormous amount of investigation, attention and documentation. It is a field which can occupy a lifetime of effort and study. I believe, that with respect to exotic animal medicine, oral disease must assume the posture of, "just another arrow in the quiver of the clinician."

From a practical point of view of providing the clinician with a readily accessible handle on this rather overwhelming problem area, I have chosen to classify oral disease from a functional point of view. I think it is correct to describe it as a cause and effect relationship, and I think there's a good deal that can be done to prevent or minimize the insidence of oral disease in any animal population. I am inclined to agree with Goldman that "observation" is the clinicians single most important tool, and it is impossible to treat or recognize something if it is not seen. Clinical evidence suggests that oral disease does have a major impact on the systemic health of the individual animal.

The pathogenesis of periodontal disease begins with gingivitis which is initiated by a combination of local chemical irritants from toxins, enzymes of dental plaque bacteria, the mechanical irritations of calculus and food impactions. If the gingivitis is untreated, then purulent infection and toxins develop, which invade the adjacent tissues, whose resistance may be low. The products of inflammation plus lytic enzymes destroy the peridontal ligament and the bone. The connective tissue elements and bone are replaced by chronic inflammatory tissue and in short, the inflammatory lesion causes a break down of the connective tissue elements of the periodontal ligament, thus detaching the tooth from the bone; the bone is resorbed; the tooth loosens and may be exfoliated.

Periodontal disease is a cause and effect relationship. Although the prognosis is poor once the disease process has established a foothold, it is a preventable disease.

REFERENCES

Colyer, J. F.: Dental Disease in its Relation to General Medicine. Longhans, Green & Co., London, 1911.

Ellis, R. G.: The Classification and Treatment of Injuries to the Teeth of Children. Year Book Publishers, Chicago, 1960.

Goldman, H. M., (Ed.): Periodontal Therapy, St. Louis, Mosby Co, 1964.

Nizel, A. E.: Nutrition in Preventive Dentistry: Science and Practice. Saunders, Philadelphia, 1972.

Stafne, E. C.: Oral Roentgenographic Diagnosis. Saunders Co., Philadelphia, 1963.

Sorrin, S.: Habit: An Etiological Factor of Periodontal Disease. Dental Digest, 41:291, 1935.

Tiecke, R. W., et al: Pathologic Physiology of Oral Disease. C. V. Mosley Co., St. Louis 1959.