

DIET CONSISTENCY AND PERIODONTAL DISEASE IN EXOTIC CARNIVORES

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During the thirties, in London, a scientist named Sir Frank Colyer wrote an exhaustive 700 page book about dental disease in animals. While addressing the subject of progressive destruction of the masticatory apparatus, he said, "In my opinion there is abundant evidence to show that, in animals, periodontal (sic) disease is attributable to injury of the gums caused by food."¹ Colyer then proceeded to analyze the problem at length in domestic animals, in wild animals in captivity, and in wild animals in their natural habitat. He observed that the relocation of the wild animal from its natural habitat into a captive environment brought about a radical change in the environment of its oral cavity² and this change resulted in the development of oral pathology. After examining thousands upon thousands of individual cases of oral pathology in exotic animals, he came to the following conclusions:

1. "The disease starts as an injury to the gum margins caused by food. This injury may be of a traumatic or of a chemical nature; when traumatic, the food in the process of mastication penetrates the soft tissues; when chemical, the products of stagnant food injure the epithelial surface and thus expose the deeper tissues to infection."
2. "The bone lesion is a progressive, rarefying osteitis commencing at the margin of the bone."
3. "The disease is caused by an alteration in the character of the diet of the animal of either a physical or of a chemical nature--in other words, by a departure from natural diet and conditions."³

During the 50 years since Colyer's exhaustive study, there have been untold millions of

¹Colyer, F.: *Variations and Diseases of the Teeth of Animals*, p. 677. John Bale, London, 1936.

²*Id.*, p. 680.

³*Id.*, p. 690.

dollars and man hours spent investigating the etiology, pathogenesis, treatment, and prevention of oral pathology in mammals. The American Dental Association tells us that oral pathology is the single most common pathologic problem in humans. They estimate that 99% of the population have oral pathology in one form or another.⁴ Analysis of the mechanisms of periodontal disease, one of the most common and destructive types of oral pathology, suggests that it is predominantly a bacterial problem and that the bacteria in the oral cavity affect the periodontium in four ways: (1) by invading the tissues directly, (2) by releasing harmful toxins, (3) by producing destructive enzymes, and (4) by initiating antigen/antibody reactions."⁵ The bacteria accomplish this by forming amorphous colonial microbial masses known as dental plaque. Since the pathogenesis of oral pathology has been described in detail elsewhere, I won't discuss it here.

Much is known about the relationship between dental plaque, nutrition, diet, and oral pathology. What is well known but not well accepted is that:

1. The consistency or texture of food has an affect upon the composition and the rate of dental plaque⁶ formation.
2. Soft diets tend to produce more bacterial plaque than firm diets.⁷
3. Excessively course, granular diets can produce periodontal disease through overuse and by direct injury of the supporting tissues.^{8 9 10 11}

⁴Nizel, A.: *Nutrition in Preventive Dentistry: Science and Practice*. W. B. Saunders Co., Philadelphia, 1972. p. 387.

⁵*Id.*, p. 400.

⁶*Id.*, p. 401.

⁷Egelberg, J.: Local effect of diet on plaque formation and development of gingivitis in dogs. I. Effect of Hard and Soft Diets. *Odont. Rev.* 16:31, 1965.

⁸ Auskaps, A. M., Gupta, O. P., and Shaw, J. H.: Periodontal disease in the rice rat. III. Survey of dietary influences. *J. Nutr.* 63:325, 1957.

⁹Cohen, B.: Comparative studies in periodontal disease. *Proc. Roy. Soc. Med* 53:275, 1960.

¹⁰Stahl, S. S., Miller, C. S., and Goldsmith, E. C.: Effect of various diets on the periodontal structure of hamsters. *J. Periodont.* 29:7, 1958.

¹¹Person, P.: Diet consistency and peridontal disease in old albino rats. *J. Periodont.* 32:308, 1961.

4. Foods of firm consistency will increase the number, distribution, and tone of the capillaries in the gingival tissue;^{12 13} which in turn, improves the metabolism and vitality of all of the supporting and surrounding structures.

5. The degree of keratinization of the stratified squamous epithelium which affords protection against trauma and other injurious agents is affected by the frictional qualities of the diet.^{14 15}

6. Chewing, by its mechanical action produces a compression and expansion of the periodontal ligament space around the teeth which, in turn, promotes formation of a dense fibrous suspensory structure by increasing both circulation and fibroblastic activity.¹⁶

7. The width of the periodontal ligament, a measure of its health, is directly related to the intensity of the mastication function.¹⁷

8. Regarding the maintenance of the alveolar bone itself, the proper balance between bone resorption and new bone formation is materially aided by hard foods and that inadequate masticatory function induced by soft foods will produce atrophic changes and lower the threshold of bone activity.¹⁸

9. And, finally, although there is poor evidence that oral pathology can be initiated by diet consistency and/or texture alone, texture is a major secondary contributor or modifier of the disease process.¹⁹

¹²Burwasser, P., and Hill, T. J.: The effect of hard and soft diets on the gingival tissues of dogs. *J. Dent. Res.* 18:398, 1939.

¹³Pelzer, R.: A Study of the local oral effects of diet on the periodontal tissues and the gingival capillary structure. *J. Amer. Dent. Ass.* 27:13, 1940.

¹⁴O'Rourke, J. T.: The relation of the physical character of the diet to the health of the periodontal tissues. *Amer. J. Orth. Oral Surg.* 33:687, 1947.

¹⁵Weinmann, J.: Keratinization of the human oral mucosa. *J. Dent. Res.* 19:57, 1940.

¹⁶Brekhus, P. J., Armstrong, W. D., and Simon, W. J.: Stimulation of muscles of mastication. *J. Dent. Res.* 20:87, 1941.

¹⁷Coolidge, E. D.: The thickness of the human periodontal membrane. *J. Amer. Dent. Ass.* 24:1260, 1937.

¹⁸*Supra*, at note #4, p. 403.

¹⁹*Id.*

In short, diet consistency and texture more than likely play a regulatory role in the etiology of oral pathology.

It is clearly difficult, and in fact, quite often misleading to extrapolate the data developed in human medicine directly to a similar problem in animals. This is particularly true with exotic animals. But, it is equally true that such results cannot be completely ignored. Nothing short of actual clinical trials to determine the relationship between diet, texture and oral pathology will be able to document the issue as it relates to a specific species. However, the test that will meet with universal approval has not yet been devised. Moreover our problems are urgent, and solutions are needed now. The weight of the evidence of the observations made in my clinical practice with exotic animals is overwhelming. There is sufficient evidence at this point to declare that oral pathology is a major problem, particularly in carnivores. I have treated numerous cases from the Order Carnivora: Dingo, Dhole, Cape Hunting Dog, South American Maned Wolf; Hyenas; Wolverine, Binturong; Bears; Serval; Caracal; Cheetah, Leopard, Jaguar, Lion, Tiger; Otters; Lesser Panda, and on and on. The etiology is diet related, and there are sufficient data to cause alarm among concerned clinicians.

In man, the incidence of spontaneous bacteremia appears to be directly related to the severity of existing oral pathology, as well as to the amount of tissue trauma during mastication. It is well known that transient bacteremias occur spontaneously in normal, healthy persons in day-to-day living. For example, 24% of 305 persons while brushing their teeth and 17% of 225 persons chewing hard candy produced bacteremia.²⁰ Procedures such as gastrointestinal tract endoscopy, per cutaneous liver biopsy, and urethral catheterization also cause significant transient bacteremias in man.^{21 22 23} Numerous studies have shown that almost all dental procedures produce bacteremia, and that the very common oral streptococcus viridans group of organisms are responsible for over 50% of the infective endocarditis reported in the literature.²⁴ Of the two factors responsible, trauma and infection, one study described infection as the more important, and noted that bacteremia occurred in 86% of the patients with marked gingivitis with

²⁰Cobe, H. M.: Transitory bacteremia, *Oral Surg.* 7:609, 1954.

²¹Shull, H. J.; Green, B. M.; et al.: Bacteremia with upper gastrointestinal tract. *Endoscopy, Ann Intern Med* 83:212, 1975.

²²LeFrock, J. L.; Ellis, C. A.; et al.: Transient bacteremia associated with per cutaneous liver biopsy. *Clin. Res.* 21:843, 1973.

²³Sullivan, N.M.; Sutter, V. L.; et al.: Bacteremia following genio-urinary tract manipulation. *Bacteriol ProcM.* 210:98 (Abstract), 1971.

²⁴Black, A. P.; Crichlow, A. M.; et al.: Bacteremia during ultrasonic teeth cleaning and extraction in the dog, *16:4, p. 611-616, 1980.*

such slight trauma as moving of the tooth or biting on a tooth.²⁵ Therefore, it is probably true that exotic animals with oral pathology--particularly periodontal disease, experience transient bacteremias during their daily routine as well.²⁶ Although there is not good evidence in animals, there is sufficient data from analysis of a variety of zoo-maintained mammalian species, to show that periodontal disease is plaque related, and from the bacterial analysis of the plaque of zoo animals, it has been shown that diet does influence the diversity of the bacterial content and the composition of the plaque.²⁷

I want to make it clear that I am not proposing that diet is the sole cause of periodontal disease, or of all oral pathology. What I am proposing, is that from my perspective of 10 years of investigative analysis and treatment of oral pathology in a wide variety of exotic animals, I see a direct relationship between the texture of the diet, oral problems, and systemic health. I also see a cause and effect relationship which is a relatively easily controllable factor in the maintenance of our animals.

Diet related oral pathology affects the body of an animal in one of three ways:²⁸

1. Through immediate contact from adjacent tissues, the infection spreads from one local area to another, i.e. cellulitis to local osteomyelitis.
2. Via natural pathways, it spreads through the trachea, into the lungs, through the esophagus into the stomach; through the eustachian tubes to the inner ear; etc.
3. Through the lymphatic and circulatory system it spreads to the entire body by continuous passage of bacteria, toxins, and harmful and metabolic byproducts.

I have repeatedly treated severe oral pathology in association with arthritic problems, acute renal failure, pancreatic, and hepatic infections, etc.

I believe our task as medical personnel caring for exotic animals can be defined as maintaining a healthy, active, and reproducing group of animals. That is no small task, and there are enormous voids in our knowledge. As a consequence, I propose we do more to eliminate those variables that we know something about. The etiological relationship between diet, oral

²⁵Dent, V. E.: The bacteriology of dental plaque from a variety of zoo-maintained mammalian species. *Archs Oral Biol*, Vol. 24. 1979. p. 277-282.

²⁶Quie, P. G.: *Streptococcus Viridans* and *Streptococcus Faecalis* Endocarditis. *Zbl. Bakt., I. Abt. Orig.*, 214:298.

²⁷Hall, R. K.: Dental management of the chronically ill child. *Aust. Dent J.*; Vol. 4, No. 5, Oct. 1979.

²⁸Colyer, F.: *Dental disease in its relation to general medicine*, p. 130. Longmans, Green & Co., London, 1911.

pathology and systemic health is well known and understood.

The veterinary medical staff caring for any group of captive exotic animals must of necessity examine diet from a variety of viewpoints, including:

- 1) the reported food preferences in the wild,
- 2) known nutritional requirements of related domestic species,
- 3) occupational therapy value of food items not necessarily related to nutritional value,
- 4) practical considerations relating to the foodstuff's availability, perishability, and/or economy.

In reality, zoo diets tend to remain rather variable to accommodate changes in foodstuff availability, additions or deletions to the collection, and newly available nutritional information. Of course the diet should provide suitable nutrients for growth, maintenance, reproduction, lactation, and the changing needs of the individual animal or group.

From my perspective, the major emphasis appears to be on the nutritional composition of the diet, and the economy of acquisition, storage, and transport. But captive animals need wear and tear on the dentition. The masticatory apparatus of carnivores was designed to be used, and used aggressively and ferociously. If the animals don't use their dentition and masticatory apparatus, they are going to lose it, and the systemic health of any individual animal will not be adequately maintained with the loss of the primary entry mechanism to the digestive system. Moreover, as the average age of our captive breeding groups increases, which it certainly should as our reproduction research programs prove successful — we will be faced with the oral pathology problems of the geriatric individual, which can be considerably more complex.

Oral pathology should be recognized as a serious problem in the care and maintenance of exotic animals. Human dental technology has sufficient solutions to manage the problem. But, the application of those solutions to animals represents a time consuming, and expensive effort. I propose that a great deal can be done to eliminate oral pathology as a consideration in exotic animal medicine by the means of prevention. It is possible to do something immediately and significantly to minimize oral problems in captive exotic carnivores. That "something" is to reevaluate their diet. Animals need more "hassle factor" per mouthful of nutrients. The best kept secret of the last fifty years is that we must eliminate the pre-processed, the over-cooked, the smashed, the blended and the pureed foods, and feed our animals a more appropriate diet duplicating the feeding habits of feral conditions. The literature contains hundreds of references to the food habits of feral carnivores²⁹ and, therefore, the appropriate menu is readily available. Convenient prepared diets, those without sufficient "hassle factor," are ruining the mouths and

²⁹Landry, S. M., Van Kruinengen, H. J.: Food habits of feral carnivores: A review of stomach content analysis, Jol. Am. Ani. Hosp. Assoc. 15:6, 1979. p. 775-782.

compromising the health of our animals.

Carnivores, in their natural habitat, eat rabbits, mice, rodents, birds, etc., in toto: i.e., toenails, eyeballs, and intestines. This is the diet we must reproduce, not predigested TV dinners. I have attempted, with this presentation to outline a health problem from a clinical perspective. My primary purpose has been to stimulate more discussion to better define the problem, and then to encourage group effort to resolve the matter by establishing norms for more appropriate diets.

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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 cause and effect relation which lead to oral pathology. The second is to demonstrate that the etiological agents of oral pathology in exotic animals are so common and so widespread that among captive populations, the incidence of oral pathology can become epidemic.

Oral pathology 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 pathology 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 pathology 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 we can adequately describe oral pathology 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 pathology 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. The inflammatory process is always characterized by the five classic Greek signs of rubor-red; calor-heat; tumor-swelling; dolor-pain; et functio, lasis-loss of function. But, the pathways of viral, fungal, bacterial, allergic, neoplastic, or therapeutic pathology 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 are 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 pathology 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.

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 preventable 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 cellulosis 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 restoring, (d) food impactions, (e) infections, and (f) other causes.

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

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

²Colyer, 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
-

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 inflammation of the soft tissues with radiographically identifiable resorption of the crest of the alveolar bone. Severe local trauma, chronic gingivitis, persistent 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
 - a. chronic gingivitis
 - b. occlusal trauma
 - c. periodontosis
-
-

TABLE 3

ETIOLOGY OF PERIODONTAL DYSTROPHY

1. Disuse
 2. Occlusal trauma
 - a. Bruxism
 - b. Clenching
 - c. Cribbing, etc.
 3. Degenerative disease of the attachment apparatus (periodontosis)
-

The second major group of periodontal diseases, **DYSTROPHIC**, are characterized by a gradual wasting away of the size, or function, of a component part, tissue, or cell--a progressive degeneration--Defective nutrition of the component leading to 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, GENERAL ADAPTATION SYNDROME is certainly contributing to an increase in the incidence of oral pathology in animals.

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 peridontal disease. I suspect that dystrophic peridontal 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 require no intervention or treatment from a clinical point of view. 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 initiated.

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 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 loss of crown structure.
Class 5	Teeth lost as a result of trauma - - subluxation complete, subluxation or luxation.
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 pathology 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 pathology 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've classified oral pathology 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 oral pathology. I am inclined to agree with Goldman that "observation" is the single most important tool, and it is impossible to treat or recognize something if it is not going to be seen. Clinical evidence is tending to suggest that oral pathology 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 and 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 periodontal 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.

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