From: **The Colyer Institute**,(Director: Dr. D.A.Fagan) **[** Corresponding author - Post Office Box 26118, San Diego, California, USA, 92196-0118, e-mail: colyer@pacbell.net**];** and From: **The Zoological Society of San Diego**, California. (Director of Veterinary Services - San Diego Wild Animal Park: Dr.J.E.Oosterhuis), and (Director of Elephant Programs-Zoo & WAP: A.Roocroft)

SIGNIFICANT DENTAL DISEASE IN ELEPHANTS

D.A. Fagan, J.E. Oosterhuis, and A. Roocroft

INTRODUCTION

The association of elephants with humans and human activities has been well documented for hundreds of years. It has also been well documented that elephants in captivity are predisposed to a variety of medical ailments, which are also found, but with lesser frequency, in their species in their wild state. These medical problems include, but are not limited to, various skin infections, dental difficulties and specific foot problems.

The two most common dental disorders of elephants in captivity are impacted or mal-positioned molar teeth - more common in the Asian elephant; and abraded, split or fractured tusks - commonly observed in both species. These two dental problems occur specifically because of the uniqueness of the elephant's dentition; and are exacerbated when combined with elements customarily found in the human's environment - ie: steel, concrete and a lack of abrasivity in the diet. It is also this same species specific uniqueness which makes these two relatively common dental disorders - so problematic to diagnose, and difficult to treat. Their diagnosis and treatment is the focus of this paper.

MOLAR TOOTH FORMATION

One of the other most unusual characteristics of the elephant's molar dentition is the manner in which the individual molar teeth are created by the fusion of a dozen or more successive *dental plates*. A predetermined number of dental plates are formed as *embryonic denticles* developing from a replicating *dental-laminae*. These enamel plates or *tooth buds* are shaped like small human hands. They are composed of cells which differentiate into the basic odontoblastic components of mammalian teeth, and in due course, manufacture the enamel, dentin and cementum necessary to create a single dental plate. As this succession of individual plates mature they fuse together to create a " package of dental plates", which becomes the molar tooth. Many of the common developmental dental maladies such as *gemination, supernumerary teeth*, and *root dilaceration* can and do occur at this stage of development resulting in the eventual malocclusion of a deformed or nonfunctional tooth.

The molars are composed of a number of parallel plates or vertical laminae of enamel surrounded by dentin and cementum. The enamel, by virtue of its superior hardness, is less susceptible to abrasive attrition during mastication, and eventually remains standing above the more easily worn dentin and cementum, resulting in an uneven occlusal grinding surface. Each of the dental plates is joined throughout the body of the molar tooth, which thus creates a tooth with 15 to 30 functional apical openings. On the *occluding* or grinding surface, the pattern formed by the worn enamel plates distinguishes the Asian elephants molars with a squashed ovoid pattern, from the African elephant with a squashed diamond pattern. In fact, it is the pattern of the molars which provides the origin of the scientific name Loxodonta.

With any abnormal molar positioning, food impaction may become a problem. The impacted food debris decomposes and results in localized infection of the surrounding structures. If not resolved, this initial infection spreads locally into the adjacent tissues, and eventually, it will spread systemically through the circulatory and lymphatic systems, but it is not reflected in the animal's blood profile making it difficult to evaluate the severity of the condition. Systemic infection in the elephant is always poorly monitored by analysis of the blood. If the offending tooth is not removed in a timely fashion, the masticatory function of the individual animal will eventually become impaired leading to poor nutrition in an animal already compromised by the presence of a developing infection. The inability to properly masticate feed eventually leads to dietary deficiency, malnutrition with weight loss, and possibly an impaction colic. The combination of transient, intermittent septicemia with local infection and poor nutrition will in time have fatal consequences. Progressive masticatory malfunction is fairly easily monitored by routine inspection and/or analysis of the individuals fecal mass.

WHY MOLAR MAL-POSITION ?

An elephant's molar dentition is unique in a variety of ways. These includes: basic design, dental formula, process of development, eruptive sequence, consequence of wear upon replacement, impact of diet upon development and eventual loss of dentition.   During their normal life span, elephants form six sets of teeth in each quadrant for a total of 24 molars, in a sequentially "as needed" pattern. Ideally, this would provide four functional molars present in the mouth at any one time; i.e., one per quadrant. However, since the development, wear, loss and replacement rate within each quadrant is such an environmentally sensitive dynamic process, dependant upon so many unpredictable factors, this is usually not what happens. As the molar teeth are methodically abraded away and finally lost, their replacements have already taken shape and are developing directly behind and below them. Properly referred to as a "*succedaneus tooth*", this next developing molar is slowly moving forward within the body of the *alveolar process* into position to substitute for, or replace it's predecessor.   The rate of growth and replacement of these molars has obviously evolved over time as has the elephant to approximate the customary rate of tooth attrition found within a specific habitat.

Although it is rarely visualized in the patient without the help of general anesthesia, the elephants molar malocclusion disorder tends to originate uni-laterally in the mandible, when one of the "in-use" molars does not abrade away at the anticipated rate.   However with the passage of time, the most commonly visualized first clinical sign of malocclusion is a mal-positioning of the opposing maxillary molar.   Even though a molar may fail to exfoliate as planned, it's developing successor or replacement tooth continues to develop, grow and mineralize at a genetically pre-determined normal rate. Normal abrasive wear and tear erodes away the 'in-use" molar while the eruptive forces produced by the growth of it's *succedaneous* tooth continues to push the "in-use" molar forward until it is exfoliated or finally comes into contact with the cortical plate of bone shaping the anterior mandible. When the "in-use" tooth is no longer able to drift or erode away, the cortical bony contour of the *alveolar process* then begins to direct the "in-use" tooth aside until it finally becomes *impacted* or wedged into a position from which it can not move. At this point, the forces necessary to cause necrosis of the bony cortex, exceed the eruptive forces of the developing tooth, and complete impaction has occurred. This impaction is usually inclined medially, and often is accompanied by *supra-eruption* of the tooth. This process is much like the drifting of logs or icebergs down river with the flow of the current until they become obstructed into a log jam or ice pack. This impacted or mal-positioned molar now tends to become *periodontally* infected following the impaction of food matter, and in time will become periodontally and/or peri-apically abscessed requiring extraction therapy.

Aside from the visually obvious mal-positioning of the teeth, which may or may not be apparent by casually looking into the animals mouth, the first clinical sign of a functionally significant *molar malocclusion* is a change in the texture and courseness of the animals fecal mass associated with weight loss. These subtle changes will almost always occur slowly over a period of time, and unless one is experienced at closely observing the individual and the fecal consequence of well masticated feed, it is easy to overlook. A change in fecal consistency is a direct and absolute indication of a change in the *masticatory efficiency* of the animal. These changes are always an early indicator that more diagnostic analysis is indicated, and the most common sign that the mal-positioned molar should be removed.

Although the removal of an elephant's molar tooth is always a major undertaking, it has been done successfully on numerous occasions. The successful technique follows the proven principles of *oral & maxillofacial surgical extraction theory* with a major adjustment to accommodate the extraordinary size of the tooth. The soft tissue is detached and protected, a moat is created around the tooth in the alveolar process, elevating and loosening forces are applied as indicated, the tooth is removed, the socket debrided and cleaned, and treated as an open wound to be flushed twice a day with water using a modified "water-pic". The earliest published report of a similar extraction procedure occurs in the British literature in 1884 by Robert A. Sterndale in "Natural History of the Mammalia of India and Ceylon." Sterndale also provides a description molar dental treatment by sawing off a portion of the offending tooth in a fully conscious, physically restrained adult elephant in "Seonee" in 1887.

ABRASIVITY OF THE DIET

The young elephant's first molar tooth erupts into it's oral cavity before it is sufficiently calcified to function normally. This animal's mouth has been designed to consume a diet of milk from its mother for the first six to twelve months. As the youngster learns to use it's trunk to begin feeding itself, soft grass is gradually added to the milk diet for another six months to a year. The elephant's eventual mixed cellulose based diet will be quite abrasive. When the rate of abrasive wear of the "in-use" molar exceeds it's rate of mineralization, exposure of the pulp tissue is sure to follow. The abrasivity factor of the young elephant's diet must not exceed the mineralization rate of it's continually calcifying developing dentition or irreversible pulpal necrosis will develop, as occurred in a group of African juveniles imported into the United States. Eventually an osteomyelitis of the alveolar process will result, and in time the tooth will be prematurely exfoliated or must be extracted. In captivity, if the young elephant must be hand reared, care should be taken to provide a non-abrasive diet until the molar teeth are fully mineralized at about two years of age.

Once the calcification process of the molar is completely underway however, abrasivity in the diet is an absolute requirement in order to begin the life long process of eroding away the chewing surface of the tooth at a uniform rate to facilitate the anticipated normal dental wear, growth and replacement. Fortunately cellulose is generally quite abrasive, unless its sole source is a pelletized feed, delivered in the manner of a 'pre-digested TV dinner". Elephants are individuals, and they can be very selective consumers. The actual variety of food stuffs consumed by each and every individual should be considered in the determination of that individuals dietary coefficient of abrasivity. Browse, if and when available, should be offered in addition to the grass based diet. Elephants spend a good deal of time and effort striping bark off of trees and branches, and then chewing on and playing with these branches of various sizes. In fact Ian Redmond has documented elephants digging and chewing rocks in the Elgon Caves which on occasion results in over abrasion of their dentition. The wear and tear of this course cellulosic based material provides an important additional source of necessary abrasivity to the molar dentition. Elephants in captivity on occasion require more hassle factor per mouthful of nutrients than is provided. This can be added in a variety of ways. The addition of a large root ball of a freshly harvested tree, complete with feeder roots and dirt clods, not only provides this "hassle factor" but adds a great deal of occupational time and therapy to the process of "eating".

It was first noted in 1884 that "In the wild state, sand and grit, entangled in the roots of plants, help in the work of attrition." Others have suggested that "the tame animal, getting cleaner food, and not having such wear and tear of the teeth, gets a deformity by the piling over of the plates of which the grinder is composed." In captivity, the diet as presently formulated may be nutritionally adequate; however, with some individuals there is growing evidence which suggests that there may be insufficient abrasivity in this diet. Some have suggested that this abrasivity can be supplied by the addition of an inexpensive coarse dental pumice to the diet. However, a sand impaction colic is never out of the picture if too much abrasive is added to any diet. It may be wiser to consider the addition of a more naturally abrasive feed than that presently used if it is determined that additional abrasive wear is indicated in order to maintain the normal wear of the animals dentition, and thereby avoid the complications of invasive dental therapy. Clearly in this situation, an ounce of prevention is worth a great deal more than a pound of cure.

THE PROBLEM WITH TUSKS

The elephant's tusk is another one of those marvelously designed mammalian body parts, which functions wonderfully well in the environment within which it was designed, but can be a disaster for all concerned within the confines of captivity. Of course, this circumstance is not unique to elephants. It is well known that walrus do not fare any better when their tusks are used to retrieve mollusks at the bottom of a concrete swimming pool. Nor does the Babirusa's tusks survive long when used to persuade concrete or steel to move aside. But, since there are now, and will no doubt continue to be for a few more decades at least, tusked elephants in captive environments it is important to understand something of the uniqueness of this protrusive anterior denticle.

The elephant's tusk is essentially just another large example of an extremely well vascularized continuously growing tooth. Like all of the other examples it actually has two centers of growth. The first is *axial* growth which originates solely around the apex of the tooth, and which results in all of the eruptive force of the tusk - much like the 'nail bed' is the sole source of eruptive growth of a toe nail. The second center of growth is properly referred to as *radial* growth, and represents the thickening of the internal axial wall of dentin directed toward the axial center line of the developing tusk. Tusks, like all denticles are "outside" structures like hairs and toe-nails. They are embedded into an indentation in the alveolar process and cantilevered out of the body in order to function. The teeth of mammals are actually held in place like it's joints are held together. They are attached to the bone of the animal jaws by a ligament known as the *periodontal ligament* (PDL), and have more in common with the elephants knee joint than with it's toe nail.

Tusks have two blood supplies as do all mammalian teeth. The first enters the tooth through it's open apex in a complex multi-vascular pattern. The gelatinous textured soft tissue within the tusk is properly referred to as *pulp tissue*, and in the elephant it is extremely well vascularized all the way to the internal tip of the tusk - where it is known as the *pulp horn*. The second vascular source supplies the periodontal ligament through the surrounding bone, and does not actually enter the tooth, although intracellular fluids can flow osmotically through a healthy periodontal ligament into the dentin tubules system of the tusk under certain circumstances. This dual blood supply is the basis for the success of traditional endodontic or root canal therapy. This accounts for the fact that the blood supply of the tooth can be obliterated by endodontic procedures, and yet the tooth will stay in the mouth and continue to function relatively normally.

The significant of all of this is that when the an elephants tusk is rapidly abraded, fractured, sawed off to shorten it or split in such a fashion that the pulp is exposed, the "inside / outside" barrier of the animals body is breached and microbes invade and infect the pulp tissue immediately. Even if the pulp is not exposed directly, it can be over heated by a mechanical cutting device and suffer a burn type injury. In the work camps in India, tusks are traditionally cut under water. The tusk reacts to any injury with exactly the same inflammatory response that occurs elsewhere in the animal's body. In cases where the tusk fractures below the gingival crest, there can be a great deal of pain and discomfort associated with the sharp edge of the underlying tusk erupting through surrounding overgrown hyper-plastic gingival tissue. The unresolved question is why doesn't the elephant seem to notice immediately the exposed pulpal injury to it's tusk, and why does it behave as if there is no pulp tissue pain involved? Part of the answer, according to evidence from current research underway by these authors, is that there is no nerve tissue within the dental pulp of the elephant. Another portion of the answer is that the dental pulp tissue is so well vascularized that the animal's natural immune system mediated defensive mechanisms defeat the infection, and isolate the injury by forming a *secondary dentin bridge* in a process similar to the classic foreign body reaction seen through the remainder of the body. This self repairing mechanism is well documented in all healthy mammalian teeth. Obviously it's success depends upon such issues as the virulence of the invading organism, the severity of the exposure, and the health of the tissue, and clearly it does not always succeed. There are many broken tusks known to us all locked in the battle of chronic, suppurative necrotic pulpitis, eventually to be lost.

The management of this potentially chronic dental infection can be time consuming, expensive and frustrating. It is however, possible to resolve the problem quickly by taking advantage of the tooths natural self-repair capability - the secondary dentin bridge formation mechanism. With the help of a little well placed *dental first aid*, the bold clinician can dramatically influence the outcome of this all to common tusk injury. This often utilized traditional dental therapy is none other than that described for the *vital partial pulpotomy*. It has been well described in the literature, has a good history of success in many exotic animals with continuously growing teeth, and has been successfully applied to elephant tusk injury. Here again, a well timed ounce of prevention, in the form of the prompt bold application of proven dental therapy, is worth much more than a pound of cure.

CONCLUSION

Both molar malocclusion and traumatic injury of the tusk are relatively common problems associated with the management of elephants in captive environments. The common understanding of these two problems comes have a history of difficult treatment options, poor success rates, and expensive, time consuming, labor intensive involvement which becomes less resolvable with the passage of time. Recent advances in the clinical practice of Veterinary Dentistry have provided numerous well documented solutions to common dental problems which only a few years ago frequently remained undiagnosed and untreated. The same can now be said concerning molar malocclusion and tusk injury. It is now possible to apply fairly straight forward, predicatable clinical solutions to these two problems. Moreover, early diagnosis facilitates the application of both proven preventative measures, and treatment alternatives which now also have a history of success. Early diagnosis, preventive measures, and proven treatments have been discussed. These can effectively reduce the negative impact of two common dental problems upon the resources dedicated to the management of elephants in captivity.

REFERENCES

Allen, J. L., Welsch, B. et al (1984b) Medical and Surgical Management of a Fractured Tusk in an African Elephant. J. Am. Vet. Med. Assoc. 185 ; 1447-1449

Bland-Sutton, Jr. 1910. The Diseases of Elephants' Tusks in Relation to Billiard Balls. Lancet, 2:1534-1537.

Bush, M., Heese, D.W., et al (1988) Surgical Repair of Tusk Injury (pulpectomy) in an Adult, Male Forest Elephant (Lonodonta cyclotis). J. Am. Dental Assoc.

93(2):372-375

Busch, F. 1890. Ueber Verletzungen, Abscesse und Dentikel am Stosszahn des Elephanten. Dtsch. Mschr. Zahnheilk., 8:62-65.

Colyer, Sir F. 1936. Variations and Diseases of the Teeth of Animals. John Bale, Sons and Danielsson, Ltd.

Driver, M. (1988) Extraction of an Infected Tusk in an Adult African Elephant. J. Am. Vet. Med. Assoc. 192(10): 1455-1456

Finn, F. Revised Edition of Sterndale's Mammalia of India.

Haagner, A. 1920. South African Mammals.

Howard, W. E. and Smith, M. E. 1952. Rate of Extrusive Growth of Incisors of Pocket Gophers. Jour. Mamm., 33:485-487.

Humphreys, H. F. 1926. Particulars Relating to the Broken Tusk of Wild Indian Elephant. Brit. Dent. Jour., 47:1400-1401.

Kapustin, N., Proudfoot, J., et al. (1996)

Kertesz, P (1990) The principles of Elephant Tusks and Their Extraction. Proceedings of the Fourth Elephant Keepers Workshop, Port Lympne Zoo Park, England, pgs: 18-20

Kingdon, J. 1979. East African Mammals, An Atlas of Evolution in Africa.

Laws, R. M., Parker, I. S. C., and Johnston, R. C. B. 1975. Elephants and Their Habitats.

McGavin, M.D., Walker, R.D., et al. (1983) Death of an African Elephant from probable Toxemia attributed to Chronic Pulpitis. J. Am. Vet. Med. Assoc. 183(11): 1269-1273

Miller, W. D. 1890-91. Studies on the Anatomy and Pathology of the Tusks of the Elephant. Dent. Cosmos, 32:337, 421, 505, 673; 33:169, 421.

Mitchell, W. D. 1903. Some Notes upon the Dentition of the Elephant and Injuries Thereto. Dent. Rev., London, 17:83-110.

Perry, J. S. 1954. Some Observations on Growth and Tusk Weight in Male and Female African Elephants. Proc. Zool. Soc. London, 124:97-104.

Pitman, C. R. S. 1953. The Elephant in East Central Africa. Edited and published by Rowland Ward Ltd., London.

Prater, S. H. 1971. The Book of Indian Animals.

Roberts, A. 1951. The Mammals of South Africa.

Sanderson, G. P. 1907. Thirteen Years Among the Wild Beasts of India.

Shadle, A. R. 1936. The Attrition and Extrusive Growth of the Four Major Incisor Teeth of Domestic Rabbits. Jour. Mamm., 17:15-21.

Shadle, A. R., Ploss, W. R., and Markus, E. M. 1944. The Extrusive Growth and Attrition of Incisor Teeth of *Erethizon dorsatum*. Anat. Rec., 90:337-341

Shadle, A. R., Wagner, L. G., and Jacobs, T. 1936. The Extrusive Growth and Attrition of the Incisors of Albino and Hybrid *Rattus norvegicus* (Erxleben). Anat. Rec., 64:321-325.

Short, J. 1981. Diet and Feeding Behavior of the Forest Elephant.

Sikes, S. K. 1971. The Natural History of the African Elephant

Stannus, H. S. 1911. Diseases of Elephant’s Tusks. Lancet, 1:617.

Sterndale, R. A. 1884. Natural History of the Mammalian of India and Ceylon.

Sterndale, R. A. 1887. Seonee, or Camp Life on the Satpura Range.

Weatherford, H. F. (1940) Some Observations on the Tusks of the Wild Indian Elephant. Brit. Dent. J. 47: 1400-1407