Osseous Surgery to Augment Treatment of Chronic Periodontitis of Canine Teeth in a Cat

A 10-year-old neutered/male Manx cat was referred to the dentistry/oral surgery service for periodontal evaluation and treatment. The cat did not receive regular home dental care. The patient's diet was a balanced, high fiber, dry cat food recommended by the referring veterinarian for weight control and promotion of gastrointestinal function.[a] A professional teeth cleaning procedure with scaling and polishing had been performed by the referring veterinarian 2-months prior to presentation based on clinical signs of oral discomfort. At that time, oral examination showed periodontal pocketing and root exposure of the left maxillary canine tooth (204) and the left mandibular canine tooth (304). An additional tooth, distal to the right mandibular molar tooth (409), was also noted. The referring veterinarian had extracted the right mandibular canine tooth (404) for periodontal abscessation 2-months before presentation. The cat's owners desired periodontal treatment in order to avoid extraction of 204 and 304.

The cat appeared bright and alert with no obvious indication of oral discomfort. Conscious oral examination was limited due to the cat's fractious nature. The extraction site at 404 appeared to be healing without complication. The 204 appeared slightly extruded. A firm buccal alveolar enlargement was noted at both 204 and 304. The tentative diagnosis was periodontitis with chronic alveolar osteitis. The diagnostic plan included pre-anesthetic laboratory tests, general anesthesia, appropriate analgesia, oral charting, maxillofacial examination, and radiographic evaluation. Treatment options included extraction, root planing, osseous resection, osteoplasty, bone replacement, or a combination of these treatments.

Pre-anesthetic laboratory tests, including serum chemistries, electrolytes, complete blood count, and urinalysis were within normal limits. The cat was premedicated with buprenorphine[b] (0.01mg/kg IM) to aid pre-emptive and postoperative analgesia.[1] In order to minimize risk to staff and decrease risk to the patient (stress, incidental injury), induction was accomplished through chamber administration of an inhalant anesthetic. Sevoflurane.[c] was administered to achieve profound sedation and a 4.0-cuffed endotracheal tube was placed with the patient maintained on oxygen and sevoflurane. A 24-gauge intravenous cephalic catheter[d] was placed and an isotonic balanced electrolyte solution[e] was administered at 10ml/kg/hr. The following parameters were monitored: indirect arterial blood pressure, heart rate, respiratory rate, pulse quality, jaw tone, mucous membrane color, capillary refill time, eye position and reflexes. Body temperature was maintained with a warm water circulating heating pad[f] and a warm air-blown body covering[g]. Clindamycin[h] (5.0 mg/kg IV) was administered. Carprofen[i] (1.1mg/kg SQ) was given to provide additional pain control.[2] The mouth was lavaged with a 0.12% chlorhexidine solution[j] to decrease the amount of aerosolized bacteria and the degree of bacteremia induced by the periodontal treatment procedures.[3] Intraoral regional nerve blocks were administered to provide pre-emptive analgesia, reduce the general anesthesia requirement, and to provide post-operative pain relief.[4] Bupivacaine[k] (0.5 mg each) was injected at the right

Figure 1

Oral photographs in a 10-year-old neutered/male Manx cat with chronic periodontitis of the left maxillary canine tooth (204) and left mandibular canine tooth (304). Note the pronounced expansive contour of the 204 (A) buccal mucosa, and attachment loss with root exposure. Bony attachment loss is present at 304 (B) with inflammation and irregularity of the marginal gingiva.

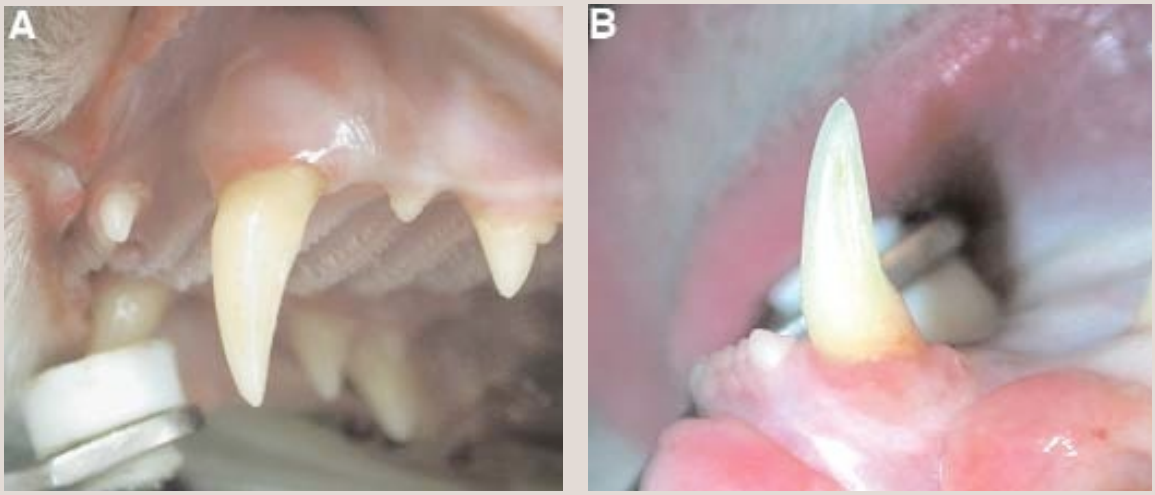
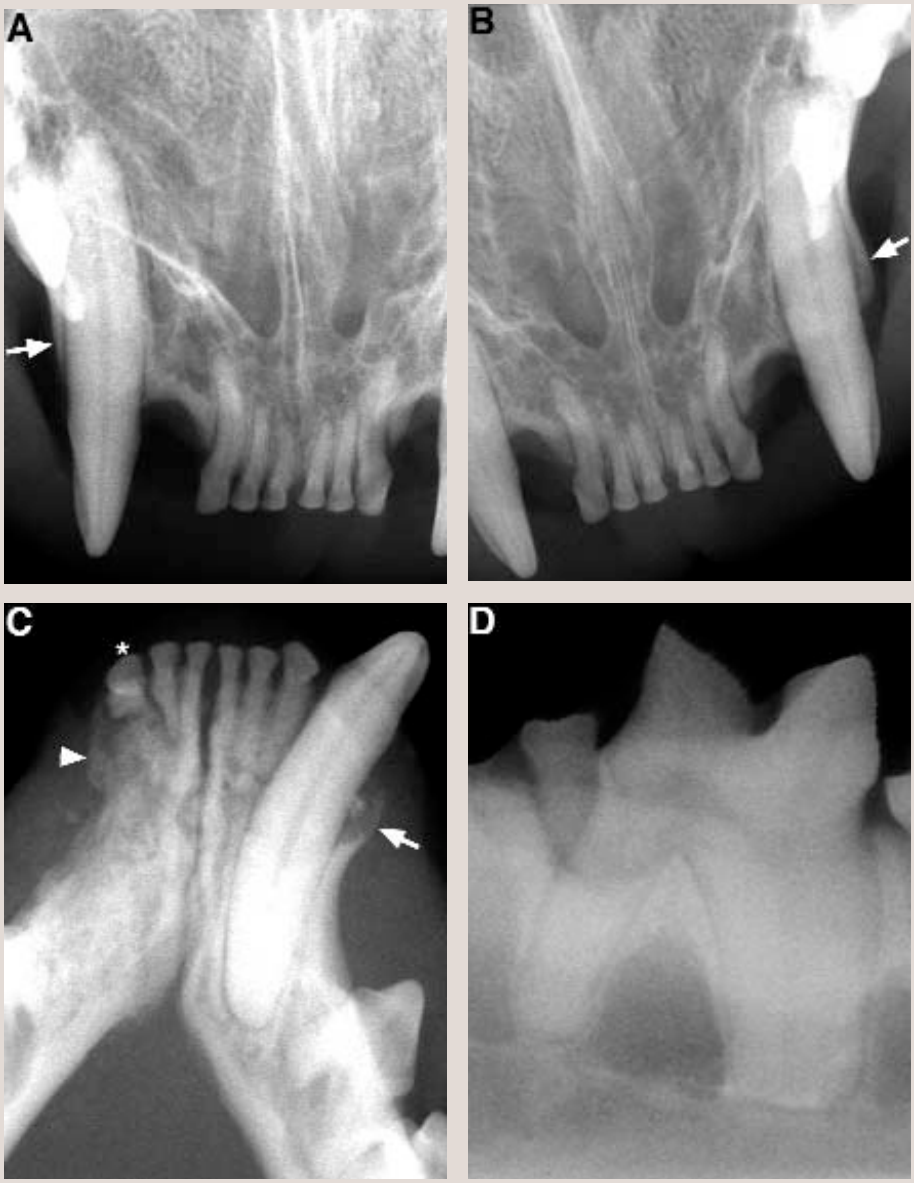


Figure 2

Dental radiographs in a 10-year-old neutered/male Manx cat with chronic periodontitis of the left maxillary canine tooth (204) and left mandibular canine tooth (304). A comparison radiograph (A) of the right maxillary canine tooth (104) shows the normal knife edge tapered contour of crestal bone (arrow). The preoperative radiograph (B) of 204 shows irregular crestal bone (arrow). The preoperative radiograph (C) of 304 shows flaring of the buccal crestal bone margin (arrow). Bony remodeling is present at the left mandibular canine tooth (404) extraction site (arrowhead). A transverse root fracture of the left mandibular incisor tooth (403) is present (*). A supernumerary, single-rooted molar is located distal to the left mandibular first molar tooth (409) [D].



and left mandibular foramina and at the entrance to the left infraorbital canal using a 1-cc syringe and 25-gauge needle. Care was taken to avoid intravascular administration by aspirating prior to injection. Digital pressure for 60-seconds was applied following injection at each site to provide diffusion of the medication and prevent hematoma formation.

A complete oral examination with dental charting was performed and revealed generalized stage 1 periodontal disease characterized by mild to moderate gingival inflammation and no changes in normal sulcus depth or periodontal attachment level.[6] Periodontal attachment level (PAL) is a more accurate measurement of periodontal destruction compared with periodontal probing depth because it measures from the base of the gingival sulcus/periodontal pocket to a fixed point on the tooth crown (cementoenamel junction).[5] Focal periodontal disease stage 3 was present at 204 and 304, as evidenced by tooth extrusion (supererruption) with 25-50 % bony support loss. Periodontal pockets of 1 to 2-mm were measured (Fig. 1).[5] The 204 and 304 had chronic alveolar osteitis (buccal bone expansion) characterized by a pronounced, bulbous shape. The 104 appeared normal. The 404 extraction site appeared healed with mild bony enlargement. The adjacent 403 had a mobility index of 1.[5] A supernumerary, single-rooted molar was located just distal and crowded to 409. Dental radiographs demonstrated a transverse root fracture of 403.[5] The 204 and 304 displayed widened periodontal spaces and buccal alveolar bulging, with flaring of the buccal crestal bone margin (Fig. 2). The supernumerary molar was shown to be distal to 409. Mild bony proliferation was noted at the site of the extracted 404. No other significant radiographic findings were present.

The teeth were scaled with an ultrasonic scaler[m]. Open root planing and osteoplasty of the buccal alveolar bone was performed at 204 and 304 (Fig. 3). Circumferential horizontal releasing incisions were made by inserting a # 11 scalpel blade

apically into the gingival sulcus, and cutting down to the level of the crestal bone. At the palatal/lingual surfaces, envelope flaps were created by gently reflecting the gingiva with a # 11 Miller surgical curette[n] to expose root surfaces and alveolar bone. Vertical, divergent releasing incisions were made at the buccal mesial and distal line angles of 204 to create a full-thickness mucoperiosteal flap. A similar flap was made at 304 by making a single full-thickness vertical releasing incision at the buccal mesial line angle. The vertical incisions were extended to the level of the mucogingival margin to allow adequate exposure of the abnormally expansive buccal alveolar bone. The buccal flaps were mobilized with a periosteal elevator[o] and reflected to provide access to root surfaces and expose underlying alveolar bone. A fine bone chisel[p] was used to remove all the crestal alveolar bone flaring away from the teeth. The remaining buccal bone was recontoured with a high-speed handpiece and water-cooled, tapered diamond bur[q] to reestablish the proper physiologic relationship between the bone and root. Care was taken to remove a minimal amount of bone. Using a universal curette[r], root scaling and planing were performed to remove all plaque and calculus from the root. Any remaining fibrotic pocket epithelium was carefully debrided by gentle application of the high-speed handpiece and water-cooled, diamond bur. The buccal gingival flaps were repositioned tightly against the tooth surfaces, with care to establish a normal sulcus depth, and sutured interproximally to the palatal/lingual tissue with an absorbable monofilament material[s]. The vertical releasing incisions were sutured closed in a simple interrupted pattern using the same suture material (Fig. 4). Gentle digital compression was applied for several minutes to encourage readaptation of the mucogingival tissues to bone and control hemorrhage. Imbricating retention sutures were not necessary since minimal bone was removed and flap placement was only slightly repositioned apically. Simple extractions of the fractured 403 and right mandibular supernumerary molar tooth were performed without complication. Extraction sites were sutured using the same absorbable suture material used for flap apposition. Finally, the teeth were polished with a fluoridated prophy paste[t].

The patient recovered from anesthesia without complication. Clindamycin[u] (5.0 mg/kg BID) was prescribed for 14-days. Buprenorphine[b] (0.01mg/kg BID) was administered for 3-days postoperatively. The patient was also sent home with an oral hygiene rinse containing 0.12 % chlorhexidine and zinc gluconate[j] to be applied BID to help control plaque accumulation.[6,7] The owners were instructed to handle the cat's mouth minimally until the 2-week postoperative examination. Hard food and chew items were prohibited.

Conscious oral examination revealed good healing of the mucogingival flaps at 204 and 304, with minimal inflammation 2-weeks postoperatively. The 403 extraction site had healed, whereas the supernumerary molar extraction site could not be assessed on conscious examination due to the patient's fractious nature. The cat's owners reported apparent good oral comfort levels observed at home, a good appetite for softened food, and maintenance of body weight. The owners were instructed to continue daily application of the oral hygiene rinse. A recommendation to begin brushing the patient's teeth with a flavored, pet-formulated toothpaste[w] was emphasized, with instruction to perform as often as possible, preferably once daily. The patient was placed on a prescription diet formulated specifically for the nutritional management of cats with dental disease.[x]

Figure 3

Intraoperative photographs in a 10-year-old neutered/male Manx cat with chronic periodontitis of the left maxillary canine tooth (204) and left mandibular canine tooth (304). Elevation of the buccal mucoperiosteal flap has exposed the irregular crestal alveolar bone at 304 (A). The irregular crestal alveolar bone at 304 has been removed with a fine bone chisel (B). Finally, the buccal bone at 304 has been contoured with a water-cooled, high-speed handpiece and diamond bur (C).

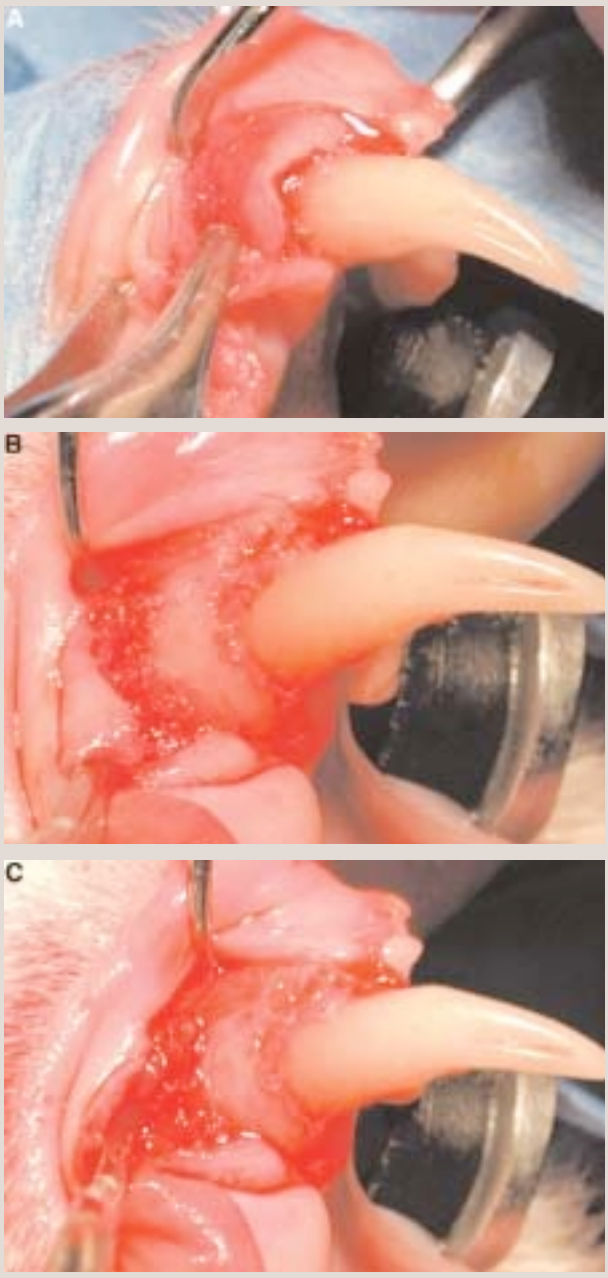
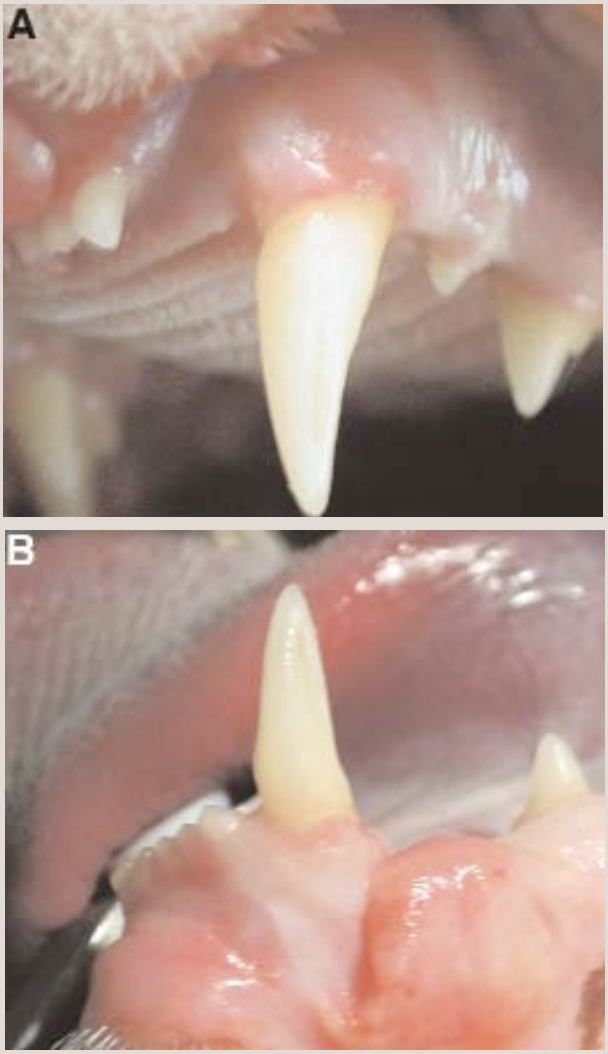


Figure 4

Intraoperative photographs in a 10-year-old neutered/male Manx cat with chronic periodontitis of the left maxillary canine tooth (204) and left mandibular canine tooth (304). Flaps have been repositioned and sutured at 304 (A) and 204 (B) following the same procedure. Care was taken to avoid placement of sutures into the sulcus.

Figure 5

Oral photographs in a 10-year-old neutered/male Manx cat 3-months following osseous surgery for chronic periodontitis of the left maxillary canine tooth (204) and left mandibular canine tooth (304). Note improved buccal alveolar bone contour and satisfactory soft tissue healing at 204 (A) and 304 (B).



The patient was returned for examination 3-months postoperatively. The cat was receiving daily chlorhexidine oral rinse application, twice-weekly tooth brushing, and the dental formulated diet. Pre-anesthetic parameters were within normal limits. General anesthesia was provided as described previously. Oral examination revealed generalized low-grade plaque (PI 0-1), calculus (CI 0-1), and gingivitis (G 0-1).[5] The extrusive changes noted at 204 and 304 appeared stable. Further, the attached gingiva at 204 and 304 appeared normal (Fig. 5). Intraoral dental radiographs demonstrated a physiologically normal knife-edge tapered contour to the crestal bone at 204 and 304 (Fig. 6). Extraction sites at 403 and the right mandibular supernumerary molar tooth appeared healed. Mild to moderate bony proliferation was noted at the site of the previously extracted 404. There was a widened periodontal space at the distal aspect of 402, perhaps indicating periodontitis. Since this tooth appeared

Figure 6

Dental radiographs in a 10-year-old neutered/male Manx cat 3-months following osseous surgery for chronic periodontitis of the left maxillary canine tooth (204) and left mandibular canine tooth (304). A comparison radiograph (A) of the right maxillary canine tooth (104) shows the normal knife edge tapered contour to crestal bone. Distolateral (B) and mesiodistal (C) radiograph of 204 shows more normal knife edge crestal alveolar bone (arrows). The radiograph of 304 demonstrates improved crestal bone contour (arrow). Note bony remodeling at the extraction sites (D) of the left third incisor (403) and mandibular canine (404) teeth (arrowhead), and the widened periodontal ligament space at the distal aspect of the left mandibular second incisor tooth (402).

otherwise stable, with no pocketing, recession, or mobility, no treatment was performed. Examination to assess for gross and radiographic evidence of devitalization was recommended in 3-months, coinciding with the next scheduled recheck of 204 and 304. At discharge the owners were complimented on the excellent homecare provided thus far, and the need for continued dental maintenance care. Recommended dental homecare included daily chlorhexidine rinse application, tooth brushing as often as possible, and a dental formulated diet.

The owner's presented the cat for examination 6.5-months following periodontal surgery. A routine of twice-weekly teeth brushing protocol had been established without difficulty, in addition to the daily chlorhexidine rinse and dental formulated diet. The cat had gained 0.25 kg, with all other extraoral physical examination findings within normal limits. CBC and serum chemistry values were normal. General sevoflurane anesthesia was provided without complication using the protocols described previously. Oral examination revealed similar findings as noted previously including mild plaque, calculus, and gingivitis; no progression of cuspid extrusion; and stability of the periodontium at 204 and 304. Intraoral dental radiographs demonstrated no further progression of the earlier periodontal abnormalities (Fig. 7). The recontoured crestal bone edge at 204 and 304 maintained a normal shape. The bony proliferation previously noted at extraction site 404 had not progressed. Likewise, 402 remained stable, with no signs of periodontal pocketing or bone loss. The widened periodontal space along the distal aspect was considered to be a result of the altered anatomy of the region, secondary to the extraction of 403 and 404.[8] Mild vertical bone loss was noted at the supernumerary molar extraction site, but no pocketing was present. At hospital discharge, the cat's owners were again commended for providing good oral care. Recommendations were made to continue the established oral homecare protocol and plans to return for a professional teeth cleaning procedure every 6-months.

Figure 7

Dental radiographs in a 10-year-old neutered/male Manx cat 6.5-months following osseous surgery for chronic periodontitis of the left maxillary canine tooth (204) and left mandibular canine tooth (304). Radiographs of 204 (A) and 304 (B) shows normal knife edge crestal alveolar bone (arrows). Continued bony remodeling is present at the extraction sites of the left mandibular third incisor (403) and canine (404) teeth (arrowhead), and the widened periodontal ligament space at the left mandibular second incisor (402) tooth that appears similar to the 3-month radiograph. A 6.5 month follow-up radiograph of 409 (C) demonstrates mild vertical bone loss (arrow) at the location of the extracted supernumerary tooth.

Discussion

Periodontal disease may be the most common disease in the cat.[10, 11] It is reported to occur in 60 % of client-owned, domestic breed cats over 3-years of age and up to 85 % of cats > 6-years of age.[11,12] Periodontal disease includes inflammation of the periodontium (gingiva, periodontal ligament, cementum, and alveolar bone) resulting in recessive alteration of the periodontium, with or without active disease. Periodontal disease begins as gingivitis and may advance to periodontitis. Periodontitis is distinguished from gingivitis by clinically detectable attachment loss. It is characterized by active destruction of the periodontal ligament and alveolar bone with pocket formation, recession, or both.

Destruction of the periodontium eventually leads to tooth loss. In cats, chronic periodontal disease can be uniquely expressed as canine tooth extrusion (supereruption). Feline chronic alveolar osteitis is another distinguishing clinical finding of the canine teeth in cats. It is characterized by bulging alveolar bone around one or more canine teeth. Radiographically, these lesions appear as bone loss around the root and expansive buccal alveolar bone.[14, 15]

Periodontal disease in the cat is a debilitating condition and severely affected cats are reluctant to eat or drink.[16] Untreated periodontal disease may result in chronic rhinitis and ophthalmic manifestations.[17] It can also progress to endodontic disease if alveolar bone loss advances to the root apex or lateral canals, resulting in pulpitis and pulp necrosis.[18] In dogs, there is evidence that periodontal disease may affect systemic health based on a positive correlation between the severity of periodontal disease and histopathologic changes in the kidney, myocardium, and liver.[19] Studies suggest an association between human periodontal disease and certain systemic disorders such as diabetes mellitus, pneumonia, heart disease, and preterm birth.[20]

Periodontal disease is initiated primarily by the accumulation of bacterial plaque on the teeth and periodontium.[10] Bacteria induce tissue destruction through direct action and indirectly by activating host defense cells, which in turn produce and release mediators that stimulate the effectors of connective tissue breakdown.[21, 22] In cats, as well as dogs and humans, genetic makeup appears to be the most important determinant in the development of periodontal disease.[23] Other factors such as diet, chewing habits, age, home care, and general oral health status play a role as well.[24,25] As plaque accumulates, gingivitis develops. Gingivitis is reversible if plaque is removed from the tooth surface.[26] Periodontitis may develop in patients with untreated gingivitis. Periodontitis is progressive and involves the destruction of the periodontal ligament and alveolar bone, with eventual tooth loss.[10] The goals of periodontal disease treatment[27] include: reestablishment of normal anatomic/ physiologic form and function (pocket reduction and elimination of soft/bony lesions); eradication or arrest of the periodontal lesion, with correction or cure of the deformity created by it; and, alteration in the mouth of the predisposing factors that lead to periodontal disease.

For the patient in this case, our goal was to employ a professional dental technique to eliminate periodontal pocket depth and improve tissue contour. In cats, an expansive, bulbous

appearance to the buccal alveolar bone plate at the canine teeth is common. In a recent study, 78 of 147 (53 %) cats displayed expansion of the buccal bone radiographically at one or more canine teeth.[14] Most cases are mild with buccal bone width less than 2-mm.[14] Buccal bone expansion at the canine teeth represents a condition of osteitis.[28] It has been called feline chronic alveolar osteitis.[15] In severe cases, tooth extraction is usually recommended.[28] The closest comparable findings in humans are small or large nodular, ridge, or spike-like outgrowth projections of bone described as exostoses.[29] Because the patient in this case demonstrated alveolar bone deformity as well as periodontal pocketing, a surgical periodontal plan was elected to allow osteoplasty of the bulging bony contour at the time of pocket treatment, including open root planing. Closed root planing (without surgical flap) is considered appropriate in feline patients with pocket depths of 1 to 2-mm and lacking bony abnormalities.[27] Osseous surgery in periodontal therapy often provides the most reliable method for reducing pockets and remains one of the mainstays of periodontal treatment based on its long-term success and predictability.[30]

Osseous surgery has been classified as additive or subtractive.[30] Additive osseous surgery is the restoration of the alveolar ridge to its original level, as in guided tissue regeneration. It produces the ideal outcome of periodontal therapy, regeneration of lost bone, and reestablishment of the periodontal ligament and gingiva at a more coronal height.[30] Conversely, subtractive or resective osseous surgery restores the form of alveolar bone to the position present at the time of surgery or to a level slightly more apical.[30] The treatment plan in this case included osseous resective surgery since it is considered the most predictable pocket reduction technique and there was sufficient bone present for tooth stability.[30]

In order to perform osseous surgery, creation of periodontal flaps is required for adequate exposure of alveolar marginal bone. Periodontal flaps also enhance accessibility to root deposits, and facilitate elimination or reduction of pocket depth by resection of the pocket wall lining.[31,32] Open curettage allows for direct visualization in order to perform satisfactory debridement of subgingival calculus, plaque, necrotic epithelium, and pocket lining epithelium as well as osteoplasty of alveolar bone.[6] Osteoplasty of the marginal bone can be performed to return the area to a more normal topography. Osteoplasty is the reshaping of bone without removal of tooth-supporting bone.[30,32] Conversely, ostectomy is the removal of tooth-supporting bone.[30,32] There is often overlap between osteoplasty and ostectomy during osseous resective surgery. The goal is to remove the least amount of bone required to produce a satisfactory form.[30,32] The objective of osteoplasty is to reshape marginal bone to produce a normal anatomic/physiologic contour, closely resembling the alveolar process undamaged by periodontal disease.[30,32] Osteoplasty achieves a physiologic contour of marginal alveolar bone favorable to gingival flap adaptation with minimal probing depth by eliminating or modifying bony ledges, irregular alveolar bone, and bony exostoses.[30] Osseous surgery also results in pocket reduction that can enhance oral hygiene and periodic professional maintenance.[30] The width of attached gingiva is preserved and access for granulomatous tissue debridement and root surface planing is provided.[30]

For a successful outcome to periodontal treatment, surgical therapy must be followed with appropriate postoperative management, as well as suitable long-term home care maintenance. Appropriate pain management is considered an essential component to professional periodontal therapy. Inadequate control of pain may result in delayed wound healing, increased risk of infection, reduced food and water intake, and change from normal behavior patterns.[33] Pre-emptive analgesia in the form of preoperative buprenorphine administration and regional bupivacaine blocks were provided to the patient in this case report. Pre-emptive analgesia decreases the intensity and duration of postoperative pain and minimizes the likelihood of a chronic pain state being established by preventing peripheral and central nervous system sensitization during the surgical procedure.[33] The use of local anesthetics and opioids in cats is well established.[2,34]

This patient also benefited from the administration of a non-steroidal anti-inflammatory agent (NSAID) in the form of carprofen. Carprofen use in cats has been shown to provide good analgesic effect.[2] Meloxicam, ketoprofen, tolfenamic acid, and vedaprofen have also been used in cats.[2] Feline NSAID use is considered extra-label in the United States. In addition to analgesia, NSAIDs may help control the connective tissue destruction that occurs in periodontal disease.[35] Plaque bacteria induce tissue destruction indirectly by activating host defense cells, which subsequently produce and release mediators that stimulate the effectors of connective tissue breakdown. A wide array of molecules are produced including the cytokines interleukin-1 (IL-1) and tumor-necrosis factor-alpha (TNF-alpha); prostaglandins, especially PGE2; and hydrolytic enzymes.[21,22] Gingival fibroblasts produce the bone-resorbing cytokine IL-6.[21] Cyclooxygenase-2 inhibition, provided by carprofen, may be useful in helping to control fibroblast production of IL-6 in patients with severe periodontitis.[36] Human clinical trials and animal model studies support the theory that inhibition of local arachidonic acid metabolites with NSAIDs slows periodontal disease progression.[35,37]

The patient in this case report also received perioperative and postoperative clindamycin hydrochloride. The role of antibiotics for treatment of periodontitis is not well defined. In human dentistry, it is recognized that antimicrobial treatment should not be used as a monotherapy, but should be incorporated as part of the comprehensive periodontal treatment plan.[29] There is evidence in dogs that short-term use of clindamycin as part of the treatment regimen can help control dental plaque accumulation and oral malodor and maintain gingival health immediately before and after a professional teeth cleaning procedure.[39,40] In a recent study in cats, treatment with clindamycin, spiramycin-metronidazole, or doxycycline produced a substantial reduction in the number of *Porphyromonas spp.,* and resulted in substantial clinical improvement.[41] This may indicate that these antimicrobial agents are useful adjunctive therapy to mechanical debridement in domestic cats.

Professional periodontal procedures are considered an adjunct to the overall periodontal treatment plan. Daily plaque control provided at home will determine oral health as much as the professional dental care provided.[23] An appropriate homecare plan should take into account the individual requirements and restrictions of the patient and client. Tooth brushing is ideal, and is generally considered the superior technique for daily plaque control.[6,10] After plaque has formed, its biofilm characteristics resist most passive control methods, but brushing will mechanically disrupt plaque accumulation.[42] Recommended frequency of brushing is unclear.[43] For patients with periodontal disease, daily tooth brushing is optimal, yet a frequency of three times weekly may still be beneficial.[6] Ultimately, how often a pet's teeth are brushed will depend on the compliance and motivation of the owner and the acceptance or tolerance by the pet.

In addition to regular tooth brushing, the patient in this case also received daily chlorhexidine and zinc gluconate administration in the form of an oral rinse.[44] Chlorhexidine is a diguanidohexane that is considered the optimal oral antiseptic.[45] Chlorhexidine can help reduce anaerobic subgingival microflora in humans when used as an oral rinse.[45] Zinc ascorbate used as an oral antiseptic has been shown to decrease development and progression of gingivitis in cats.[8] Chemical plaque control is an adjunct to, not a replacement of, mechanical plaque control.[44] Because periodontal disease may be influenced by diet texture, this patient was placed on a diet formulated to meet the needs of cats with dental disease. Feeding a food that promotes prolonged tooth surface contact helps remove plaque.[46]

Many other agents could be considered in the design of the home care dental maintenance plan, including chew toys, dental chews, gels, inert polymer barrier sealants, and other chemical agents like triclosan.[44] An alternative periodontal disease management technique includes the feeding of diets or treats containing polyphosphates. Polyphosphates chelate salivary calcium, thereby preventing plaque from mineralizing into calculus.[47] Application of a dental barrier sealant[y] is another possible addition to the periodontal therapy. Short-term studies in dogs show decreased plaque and calculus accumulation on sealant treated teeth.[48] Ultimately, the decision of what agents to incorporate into the periodontal therapeutic plan depends on current knowledge of the available products, ideally based on rigorous assessment of evidence (evidence-based dentistry).[43]

Successful treatment and control of periodontal disease requires a multidimensional approach.[43] Periodontal health is achieved through a combination of professional dental treatment and daily home care management. This case demonstrates a successful outcome through the combined use of periodontal surgery, postoperative management, and the establishment of appropriate long-term daily home care.

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[a] Feline w/d Prescription Diet, Hills Pet Nutrition, Topeka, KS

[b] Buprenex, Reckitt Benckiser Pharmaceuticals, Richmond, VA

[c] SevoFlo, Abbott Laboratories, North Chicago, IL

[d] B D Insyte, Becton Dickinson, Sandy, UT

[e] Lactated Ringer's, Baxter, Deerfield, IL

[f] K-Mod 100 Heat Therapy Pump, Baxter, Deerfield, IL

[g] Life-Air 1000 Hypothermic Therapy System, Progressive Dynamics Inc., Marshall, MI

[h] Cleocin, Upjohn & Pharmacia, Kalamazoo, MI

[i] Rimadyl, Pfizer Animal Health, Exton, PA

[j] C.E.T. Oral Hygiene Rinse, Virbac AH Inc., Fort Worth, TX

[k] Marcaine, Abbott Laboratories, North Chicago, IL

[l] Lightyear Digital Radiography System, Lightyear Technologies, Roswell, GA

[m] Odontoson-M, Goof, Denmark

[n] #11 Miller surgical curette, Henry Schein, Port Washington, NY

[o] EX-9 feline periosteal elevator, Cislak Manufacturing Inc., Niles, IL

[p] Fedi 2, Cislak Mfg. Inc / Zoll-Dental, Niles, IL

[q] Taper diamond bur, Henry Schein, Port Washington, NY

[r] Columbia 13/14, Henry Schein, Port Washington, NY

[s] Monocryl, Ethicon Inc., Somerville, NJ

[t] Zircon-F prophy paste, Henry Schein, Port Washington, NY

[u] Antirobe Aquadrops, Pharmacia & Upjohn, Kalamazoo, MI

[v] Torbugesic, Fort Dodge Animal Health, Fort Dodge, IA

[w] C.E.T. Toothpaste, Virbac AH Inc., Fort Worth, TX

[x] Feline t/d Prescription Diet, Hills Pet Nutrition, Topeka, KS

[y] Oravet, Merial, Duluth, GA

Contributed by:

Donald E. Beebe, DVM
William R. Gengler, DVM
University of Wisconsin-Madison, School of Veterinary Medicine, 2015 Linden Drive, Madison, WI 53706.
Dr. Beebe's present address is the Animal Dental Clinic, 987 Laurel Street, San Carlos, CA 94070. Email: HealthyK9s@mac.com

References

1. Robertson SA, Taylor PM, Sear JW. Systemic uptake of buprenorphine by cats after oral mucosal administration. *Vet Rec* 2003; 152: 675-8.
2. Robertson SA, Taylor PM. Pain management in cats--past, present and future. Part 2. Treatment of pain--clinical pharmacology. *J Fel Med Surg* 2004; 6: 321-333.
3. Witzenberger T, O'Leary TJ, Gillette WB. Effect of a local germicide on the occurrence of bacteremia during subgingival scaling. *J Periodontol* 1982; 53: 172-179.
4. Lantz GC. Regional anesthesia for dentistry and oral surgery. *J Vet Dent* 2003; 20: 181-186.
5. Wiggs RB, Lobprise HB. *Veterinary dentistry, principles & practice.* Philadelphia: Lippincott-Raven, 1997: 114.
6. Jones CG. Chlorhexidine: is it still the gold standard? *Periodontol 2000* 1997; 1: 55-62.
7. Clarke DE. Clinical and microbiological effects of oral zinc ascorbate gel in cats. *J Vet Dent* 2001; 18: 177-183.
8. Wood BC. Management of rostral mandibular fracture including lateral luxation of a mandibular canine tooth in a dog. *J Vet Dent* 2003; 20: 91-94.
9. Harvey CE, Emily P. *Small animal dentistry.* St. Louis: Mosby-Year Book, 1993: 89-144.
10. Lund EM, Armstrong JP, Kirk CA, et al. Health status and population characteristics of dogs and cats examined at private veterinary practices in the United States. *J Am Vet Med Assoc* 1999; 214: 1336-1341.
11. Maretta SM. Current concepts in canine and feline dentistry. In: Kirk RW, Bonagura JD, eds. *Current veterinary therapy XII.* Philadelphia: WB Saunders, 1994; 685-691.
12. Tholen M, Hoyt RF. Oral pathology. In: Bojrab MJ, Tholen M, eds. *Small animal oral medicine and surgery.* Philadelphia: Lea & Febiger, 1990; 42.
13. Wiggs RB, Lobprise HB. *Veterinary dentistry, principles & practice.* Philadelphia: Lippincott-Raven, 1997: 186-231.
14. Wiggs RB, Lobprise HB. *Veterinary dentistry, principles & practice.* Philadelphia: Lippincott-Raven, 1997: 482-517.

[15] Lommer MJ, Verstraete FJ. Radiographic patterns of periodontitis in cats: 147 cases (1998-1999). *J Am Vet Med Assoc* 2001; 218: 230-234.

[16] Bellows J. Interpreting dental radiographs for periodontal disease. *DVM Newsmagazine* July 1, 2001.

[17] Mallonee DH, Harvey CE, Venner M, et al. Bacteriology of periodontal disease in the cat. *Arch Oral Biol* 1988; 33: 677-683.

[18] Reiter AM, Brady CA, Harvey CE. Local and systemic complications in a cat after poorly performed dental extractions. *J Vet Dent* 2004; 21: 215-221.

[19] Ammons WF, Harrington GW. The periodontic-endodontic continuum. In: Newman MG, Takei HH, Carranza FA, eds. *Clinical periodontology.* 9th ed. Philadelphia: WB Saunders, 2002; 840-850.

[20] DeBowes LJ, Mosier D, Logan EI, et al. Association of periodontal disease and histologic lesions in multiple organs from 45 dogs. *J Vet Dent* 1996; 13: 57-60.

[21] Rose LF, Steinberg BJ, Minsk L. The relationship between periodontal disease and systemic conditions. *Compend Contin Educ Dent* 2000; 21: 870-877.

[22] Page RC. The role of inflammatory mediators in the pathogenesis of periodontal disease. *J Periodontal Res* 1991; 26: 230-242.

[23] Graves DT, Cochran D. The contribution of interleukin-1 and tumor necrosis factor to periodontal tissue destruction. *J Periodontol* 2003; 74: 391-401.

[24] Hale FA. Home care for the veterinary dental patient. *J Vet Dent* 2003; 20: 52-54.

[25] Beck JD, Arbes Jr SJ. Epidemiology of gingival and periodontal diseases. In: Newman MG, Takei HH, Carranza FA, eds. *Clinical periodontology.* 9th ed. Philadelphia: WB Saunders, 2002; 74-94.

[26] Gorrel C, Inskeep G, Inskeep T. Benefits of a dental hygiene chew on the periodontal health of cats. *J Vet Dent* 1998; 15:135-138.

[27] Ingham KE, Gorrel C, Blackburn JM, et al. The effect of toothbrushing on periodontal disease in cats. *J Nutr* 2002; 132: 1740S-1741S.

[28] Holmstrom SE, Frost P, Eisner ER. Periodontal therapy and surgery. In: Holmstrom SE, Frost P, Eisner ER, eds. *Veterinary dental techniques.* 3rd ed, Philadelphia: WB Saunders, 2004: 233-290.

[29] Manfra Marretta S. Recognition of feline oral lesions. *Proceedings of the Atlantic Coast Veterinary Conference,* 2001.

[30] Carranza FA. Bone loss and patterns of bone destruction. In: Newman MG, Takei HH, Carranza FA, eds. *Clinical periodontology.* 9th ed. Philadelphia: WB Saunders, 2002; 354-370.

[31] Sims TN, Ammons W. Resective osseous surgery. In: Newman MG, Takei HH, Carranza FA, eds. *Clinical periodontology.* 9th ed. Philadelphia: WB Saunders, 2002; 786-803.

[32] Carranza FA, Takei HH. The flap technique for pocket therapy. In: Newman MG, Takei HH, Carranza FA, eds. *Clinical periodontology.* 9th ed. Philadelphia: WB Saunders, 2002; 774-785.

[33] Lindhe J. *Textbook of clinical periodontology.* 2nd ed. Copenhagen: Munksgaard, 1993: 386-421.

[34] Tranquilli WJ, Grimm KA, Lamont LA. *Pain management for the small animal practitioner.* 2nd ed. Jackson, WY: Teton NewMedia, 2004: 1-12.

[35] Flecknell P, Waterman-Pearson A. *Pain management in animals.* London: WB Saunders, 2000.

[36] Golub LM, Ryan ME, Williams RC. Modulation of the host response in the treatment of periodontitis. *Dent Today* 1998; 17: 102-109.

[37] Tipton DA, Flynn JC, Stein SH, et al. Cyclooxygenase-2 inhibitors decrease interleukin-1beta-stimulated prostaglandin E2 and IL-6 production by human gingival fibroblasts. *J Periodontol* 2003; 74: 1754-1763.

[38] Holzhausen M, Rossa Jr C, Marcantonio Jr E, et al. Effect of selective cyclooxygenase-2 inhibition on the development of ligature-induced periodontitis in rats. *J Periodontol* 2002; 73: 1030-1036.

[39] Jolkovsky DL, Ciancio SG. Chemotherapeutic agents in the treatment of periodontal diseases. In: Newman MG, Takei HH, Carranza FA, eds. *Clinical periodontology.* 9th ed. Philadelphia: WB Saunders, 2002; 675-687.

[40] Zetner K, Plum G, Wolfe-Dieter R. Effect of clindamycin hydrochloride on gingival crevicular fluid and immune mediators in beagles. *Vet Ther* 2002; 3: 177-188.

[41] Bowersock TL, Wu CC, Inskeep GA, et al. Prevention of bacteremia in dogs undergoing dental scaling by prior administration of oral clindamycin or chlorhexidine oral rinse. *J Vet Dent* 2000; 17: 11-16.

[42] Norris JM, Love DN. In vitro antimicrobial susceptibilities of three Porphyromonas spp and in vivo responses in the oral cavity of cats to selected antimicrobial agents. *Aust Vet J* 2000; 78: 533-537.

[43] DuPont GA. Understanding dental plaque; biofilm dynamics. *J Vet Dent* 1997; 14: 91-94.

[44] Roudebush P, Logan E, Hale FA. Evidence-based veterinary dentistry: a systematic review of homecare for prevention of periodontal disease in dogs and cats. *J Vet Dent* 2005; 22: 6-15.

[45] Perry DA. Plaque control for the periodontal patient. In: Newman MG, Takei HH, Carranza FA, eds. *Clinical periodontology.* 9th ed. Philadelphia: WB Saunders, 2002; 651-674.

[46] Santos S, Herrera D, Lopez E, et al. A randomized clinical trial on the short-term clinical and microbiological effects of the adjunctive use of a 0.05% chlorhexidine mouth rinse for patients in supportive periodontal care. *J Clin Periodontol* 2004; 31: 45-51.

[47] Logan EI, Berg ML, Coffman L, et al. Dietary control of feline gingivitis: results of a six month study. *Proceedings of the 13th Veterinary Dental Forum,* 1999; 54.

[48] Warrick JM, Stookey GK. Overview of clinical trials using sodium hexametaphosphate for the prevention of dental calculus. *Proceedings of the 18th Veterinary Dental Forum,* 2004; 272-276.

[49] Gengler W. A study to assess efficacy of a prophylactic dental product in dogs. *Proceedings of the 18th Veterinary Dental Forum,* 2004; 152.