Chapter 17: Feline Odontoclastic Resorptive Lesions

Etiology?

Over the past many years there have been a number of papers presented and published examining the problem of feline odontoclastic resorptive lesions (RLs), and though the body of knowledge has been growing, the pieces of the puzzle have not been adding up to a clear understanding of the condition.

Historically, some of the theories as to the cause have included:

- Nutritional hyperparathyroidism leading to resorption of calcium from sub-gingival dentin. This suggestion was based on the higher incidence in domesticated cats but has been proven groundless.
- Chronic calici virus in the sulcar tissues may be a factor but has not been a consistent finding. Infecting SPF cats with calici virus failed to cause RLs.
- Viral infection at the time of tooth development leading to a defective and susceptible tooth. (Previous) studies had failed to find any differences, chemically or microstructurally, between affected and normal teeth.
- Chronic regurgitation of hair balls with gastric hydrochloric acid may pre-dispose. This theory is based on similar lesions in human bulimic patients.
- Low pH diets causing a chronic acid environment in the sulcus may be a factor, but no evidence to support this notion exists at this time. A study of post-prandial dental surface pH found no difference between cats eating an acidifying diet and cats on a control diet.
- Genetics do seem to play a role. The incidence is highest in Persians, Abyssinians, Siamese, Russian Blue, Scottish Fold, and Oriental Shorthairs suggesting that over-breeding has led to a predisposition. This may be a function of a weak or inappropriate immune response to periodontal disease.

Any condition that is poorly understood tends to accumulate a number of names (each an attempt to more accurately describe the unknown). Neck lesions, cervical line lesions, cervical erosions, feline external odontoclastic resorptive lesions… are just some of the names given to this enigmatic and frustrating condition. At the time writing, there is a trend to just refer to them as resorptive lesions (RLs).

In the past few years, there have been a number of new studies presented that show the promise of bringing the bits together so that a plausible picture may be forming. What follows was current when I wrote it but may well be dated information by the time it reaches your hands, as there is lots of research ongoing and lots of new information coming to light.

Before I embark on this discussion, we need to review a bit of anatomy to make sure we are all speaking the same language.

The crown of the tooth is that part covered by enamel and the root is covered by cementum. Where the crown and root meet is the neck, cervix or cementoenamel junction (CEJ). The bulk of the tooth is composed of dentin and inside the tooth is a hollow chamber containing the dental pulp.

Most of the root is situated in a depression in the bone known as the alveolus. The most coronal portion of the root extends above the margin of the alveolar bone. Gingiva is a tough tissue that lies against the tooth and alveolar bone. The free-gingival margin is above the CEJ, but gingiva does not attach to enamel, so the gingiva above the CEJ is known as the free gingiva (‘cause it ain’t attached) and the space between gingiva and enamel is known as the gingival sulcus. From the CEJ to the crest of the alveolar

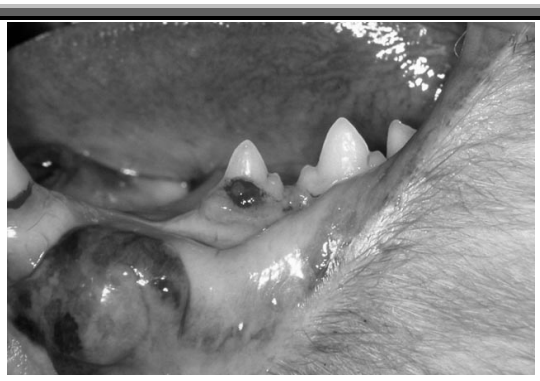

Figure #17.1. The dark area over the furcation of the left mandibular third premolar is the inflamed (and sensitive) soft tissue filling an RL that has advanced to involve the crown of the tooth.

bone, the gingiva should be firmly attached to the root cementum in a tight collar completely encircling the tooth. It is this collar of gingival attachment that isolates the periodontal ligament space from oral bacteria.

Within the alveolus, there is a space between the root and bone (the periodontal ligament space), occupied by the periodontal ligament, which attaches to the bone and cementum. The periodontal ligament space appears radiographically as a fine radiolucent line following the contour of the root.

I need to quote an axiom that must be kept in mind. *A little knowledge is a dangerous thing*. What follows will be a condensed review of some recent papers. Much of this information is very new and needs further study before we can change our approach to treating resorptive lesions. I do not want you going off half-cocked.

One more introductory note (which will be repeated because it is soooo important).

INTRA-ORAL DENTAL RADIOGRAPHY IS ESSENTIAL FOR THE ACCURATE ASSESSMENT AND PROPER TREATMENT OF RESORPTIVE LESIONS.

I am not just saying this to encourage you to refer (though that would be nice) – it just makes sense. I doubt any of you would even consider treating a fractured limb without diagnostic radiographs. Nor would you do an exploratory laparotomy without radiographs. Without dental radiographs, you cannot know what you are getting into or when you have gotten out.

Two Types of RLs?

Gregg DuPont has described what he believes to be two distinct types of resorptive lesions based on clinical, radiographic and histologic appearance. According to Dr. DuPont:

Type I lesions are the less common ones associated with moderate to severe gingivitis and/or periodontitis. They typically start at the CEJ and can extend in all directions from that starting point. There is typically little or no root resorption evident beyond the confines of the lesion. The cause of these lesions might well be local inflammatory reaction due to gingivitis or periodontitis.

As type I lesions are often associated with periodontitis and endodontic disease, these teeth

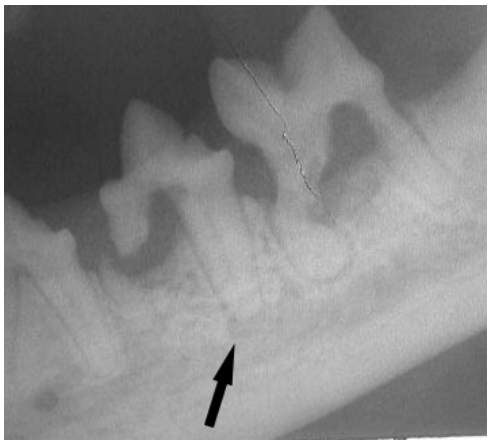

Figure #17.2. Intra-oral radiograph of Type I resorptive lesions affecting the left mandibular fourth premolar and molar teeth. The defects are centered on the cervical region of the teeth and are associated with periodontal bone loss. Beyond the confines of the lesion, the roots look perfectly normal with normal radiodensity, clearly visible endodontic chambers and a clearly visible periodontal ligament space between root and bone. Once the pulp chamber is breached by the lesion, these teeth often develop endodontic disease. The original film showed an obvious radiolucency around the apex of the distal root of the fourth premolar (black arrow).

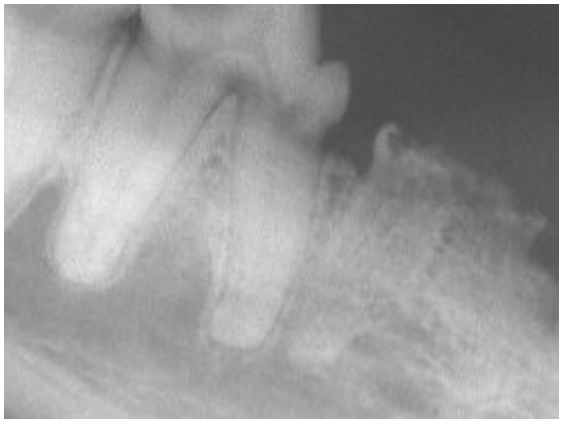

Figure #17.3. A grade 5, type II lesion of the right mandibular third premolar tooth. Most of the crown has fallen away, though some remains. The roots have been largely resorbed and replaced by bone-cementum tissue. There is no periodontal ligament space visible. Frequently the gingiva will appear to have healed over these lesions, though there is often a fistula and persistent inflammation. In this film, there is no periodontal bone loss and no evidence of endodontic disease. This lesion seems particularly common on the canine teeth of cats.

must be entirely extracted. It is not permissible to leave any remnants of these roots behind. The good news is that the presence of a periodontal ligament (i.e., the lack of ankylosis) makes these roots fairly easy to extract. The bad news is that the resorptive process usually creates serious weak spots on the roots, so the roots break and then it is necessary to surgically remove the root remnants. Having a pre-operative dental x-ray lets you know where the weak spots are.

Type II lesions are not only more common, they are also more frustrating to deal with. However, these are the ones that we may be getting closer to understanding.

Type II lesions are not generally associated with endodontic disease or periodontitis, though there may be localized gingivitis or granulation tissue in and around the lesion itself. These lesions seem to be able to arise at any location on the root and are associated with extensive root resorption, loss of periodontal ligament and ankylosis.

One Etiology – Two Presentations?

It should be noted the Dr. Colin Harvey is not convinced that there are two distinct types of RLs but that the differences Dr. DuPont describes are just different stages or presentations of the same process. Consider the following.

If a lesion begins deep within the alveolus, it is isolated from oral bacteria and so the non-inflammatory process can proceed gradually and the physiologic replacement of lost dental tissue with bone-cementum tissue can also occur. As the lesion progresses up the root and breaks through the gingival attachment, it becomes contaminated with oral bacteria and becomes an inflamed and painful situation. This would give the clinical and radiographic appearance of a Type II lesion.

If a lesion begins near the alveolar crest, it may breach the gingival attachment and become contaminated with oral bacteria early on in it's evolution. The ensuing infection and inflammation would prevent the physiologic repair of the defect. Instead, there would be inflammatory resorption with further dental tissue loss as well as bone loss. This would appear clinically and radiographically like a Type I lesion.

Back to Etiology

New Histologic Studies

Steinberg looked at 80 clinically normal teeth taken from cats that had at least one clinical RL and found that 100% of these teeth had microscopic evidence of resorption and these lesions were found primarily in the non-cervical region of the root. This suggests that if these cats live long enough, every single tooth will eventually succumb to resorptive lesions. Taken further, it could be said that if they live long enough, every cat will develop type II RLs.

Gorrel and Larsson also did a study looking for microscopic lesions on the roots of cat teeth. They harvested 56 teeth that appeared clinically and radiographically free of RLs. Of these, 43 teeth (group A) were taken from cats that had a clinical RL on at least one tooth and 13 teeth (group B) were taken from cats with no clinical or radiographic evidence of RLs on any teeth. Twenty six teeth from group A and one tooth from group B showed microscopic evidence of external root resorption.

Among the salient observations of this study were the following:

- the cervical cementum on the roots of teeth from RL-cats was thicker and more irregular than that found on the roots of teeth taken from RL-free cats.
- The periodontal ligament around non-ankylosed resorptive lesions was narrow and the fibers arranged vertically (rather than the normal horizontal or oblique arrangement). The ligament around ankylosed lesions was grossly abnormal with edematous vascularized tissue.
- Teeth from RL-cats were much more likely (60%) to have microscopic resorptive lesions than teeth from RL-free cats (8%). These microscopic lesions were all located at the mid root or apical portion of the root and were not associated with inflammation.
- Healed cemental lesions covered by intact periodontal tissue were seen in some cases.
- Ankylosis was associated with formation of reparative bone-cementum tissue filling in the space vacated by the disappearing root tissue.

If we accept that there is an etiologic distinction between Type I and Type II RLs, these findings suggest that type II lesions start within the cementum of the root and in some cases they may heal spontaneously. The other big surprise was the finding that these early lesions are not associated with inflammation. It seems that as long as the lesion remains on the root **below the level of gingival attachment and protected from oral bacterial contamination**, there is no inflammation. Once the lesion extends through the gingival attachment and comes in contact with oral bacteria, inflammation develops as a result of the lesion, not its cause.

I will now offer my own speculation. From these and other histologic studies, it appears that RLs can start anywhere on the root of the tooth. If the lesion starts on the root deep within the alveolus below an intact collar of gingival attachment and so isolated from oral bacteria, a non-inflammatory lesion which Dr. DuPont would call a Type II lesion would result. If the lesion starts near the neck of the tooth and quickly becomes contaminated with oral bacteria, inflammation will develop and a DuPont-Type I lesion develops. So the distinction between Type I and Type II may merely be a function of where on the root the lesion starts and how early in its progression it becomes contaminated with oral bacteria.

New Biochemical Studies

Alex Reiter has done a study which may prove to be the key to unlocking the mystery. He looked at the serum concentration of calciotropic hormones in domestic cats. What he found was that cats with RLs had significantly higher serum levels of 25 hydroxyvitamin D than cats without RLs.

As he points out, rather than asking "why do some cat teeth undergo resorption?", we should ask "why do some cat's teeth not undergo resorption?". After all, teeth are made of tissues very similar to bone which is constantly undergoing remodeling. Normally, the roots of permanent teeth are considered to be resistant to resorption because of a protective organic matrix on their external surfaces. If this matrix is lost or becomes calcified, then odontoclasts (which are virtually identical in every way to osteotoclasts) can attack the root surface.

Reiter proposes that hypercementosis, osteoid production along the socket wall and gradual calcification of the periodontal ligament may result in fusion of the tooth root(s) and alveolar bone (ankylosis) and gradual resorption of the roots. He further postulates that this may be caused by chronic dietary intake of excess vitamin D. Several other studies found that excess administration of vitamin D or its metabolites to experimental animals resulted in dental and periodontal changes very similar to those seen around teeth affected by RLs.

Looking at the vitamin D content in canned cat foods, it was found that 20 of 49 brands (41%) had in excess of 30 times the vitamin D requirements of 250 IU/kg diet dry matter and 31% actually exceeded the AAFCO maximum level of 10 000 IU/kg diet dry matter.

Colin Harvey and others have done some work looking at a biphosphonate drug (alendronate) that is used in humans to prevent osteoporosis. One study found that the drug bound very heavily to alveolar bone and root cementum – that's good news as it means we can get this drug right were we want it.

The next study looked at the effect this drug might have on established lesions over time. Using colony cats at research facilities, he quantified the size of lesions found. The cats then received alendronate for a year and the lesions were re-measured. In the vast majority of cases, the lesions were no larger a year later and in some cases they were actually smaller. In the control cats, the lesions increased in size as predicted. These findings suggest that alendronate may someday be useful to prevent the progression of very early lesion and even in preventing them completely.

What does all this add up to?

It is too early to be certain and we should not get overly excited as yet, but here is what seems to be coming into focus.

- Type I lesions are possibly the result of gingival and periodontal inflammation and arise at the cervical region of the tooth. From there, they may extend up the crown and/or down the root. Prevention would be based on maintaining good oral hygiene. Treatment of detectable lesions is complete extraction of the tooth and its roots (leave nothing behind).
- Type II lesions are a non-inflammatory resorption that begins within the socket or at

least below the level of gingival attachment and may be triggered by vitamin D toxicity. Only after the lesion enlarges sufficiently to break through the gingival attachment to become contaminated with oral bacteria does inflammation become a factor. Treatment is still extraction and in my view it should be complete removal of all dental tissues (more on this later).

- The only way to distinguish between a type I lesion and a type II lesion is with intra-oral dental radiography.
- It is possible that we may find a way of preventing the more common type II lesions either by reducing dietary intake of vitamin D and its metabolites or by administration of a biphosphonate drug.
- It is also possible that Type I and Type II lesions have the same etiology and only differ in appearance due to their site of origin and how early or late in the process they become contaminated with oral bacteria. If this is the case, all RLs may be the result of hypervitaminosis D and restricting dietary vitamin D may prevent RLs

Progression of RLs

The life of an RL depends largely on where it begins. What follows is more speculation on my part, based on my current understanding of the pathogenesis of RLs.

For those that start on the root surface deep in the alveolus, the non-inflammatory lesion can see the resorption of most of the root and its replacement with bone-like tissue. In these cases, the periodontal ligament is obliteratated and the tooth becomes ankylosed to the bone. To our knowledge, this process is not painful.

As the lesion progresses coronally and breaks thorough the gingival attachment, it becomes contaminated with oral bacteria and inflammation develops. Now the lesion is detectable with a dental explorer and at this point the lesion also becomes quite painful.

As the lesion continues to absorb the coronal dentin, the overlying enamel, having lost its foundation, caves in exposing an area of inflamed granulation tissue filling a coronal defect. As the lesion within the crown is not surrounded by bone, it is not backfilled with bone-like tissue. Rather, the coronal tissue is gradually lost until all that remains is a bump in the gingiva overlying the ghosts of the roots and any subgingival coronal remnants (as in figure 17.3).

If the lesion begins near the cementoenamel junction, it very quickly becomes contaminated with oral bacteria and so becomes inflamed and painful early on. As the lesion extends down the root, periodontal infection follows, and so the physiolgic replacement of lost dental tissue with bone-like tissue is prevented. The result is a combination of dental tissue and bone loss. In time, the loss of dental tissue may become severe enough to allow the crown to break free, leaving the infected roots behind.

In both scenarios, after the crown is gone, the gingiva may appear to be healing over the site. However, on close inspection, I almost always find a fistula in the gingiva that extends down to the retained tooth remnants. It is this frequent observation that makes me very reluctant to leave any dental tissue in place when treating these lesions.

Grading

Grading RLs was used to be important when we thought we could save teeth with early lesions by restoring them with glass ionomers. Retrospective studies and clinical experience have shown that restoring these lesions only delays the inevitable and condemns the animal to live in pain longer. The current thinking now is that any detectable lesion requires extraction regardless of the stage. For historical perspective, the stages are as follows:

Grade I: Early lesions extending less than 0.5 mm into the tooth and involving enamel only. I do not know where this came from, but it is obviously outdated now, because these lesions do not attach the enamel at all. They occur on the root cementum, therefore, there technically can never be a Grade I RL. I would say a lesion that extends less than 0.5 mm into the cementum or dentin could be considered a Grade I lesion

Grade II: Lesions with significant tooth destruction, not yet into the pulp but extending into dentin.

Grade III: Lesions into the pulp but not involving significant crown loss.

Grade IV: Lesions with extensive loss of tooth structure including loss of large portions of the crown.

Grade V: Hidden lesions where the crown is gone but the roots remain buried under inflamed gingiva.

I tend to use this five Grade system and will be doing so for the remainder of this discussion. Some people combine Grade IV and V and so end up with a four Grade system.

Radiology

Cat's teeth are small. RLs are even smaller. Therefore, to properly radiograph cat's teeth for RLs, you should use small, intra-oral dental film. The materials and techniques have been covered already in Chapter 10.

When looking at dental X-rays, there are a number of things to assess. Check the height of the alveolar crestal bone for evidence of bone loss. Look carefully at the roots. Can you see a distinct periodontal ligament (seen as a lucency between the tooth and the lamina dura of the alveolar bone)? Is the pulp chamber visible and distinct? Are there any irregularities in the shape or density of the root or crown? Is there a lucent halo around the apex of the root? Does the alveolar bone look normal or moth eaten?

If you would like to see some examples of normal and abnormal dental radiographs, I would suggest the *Atlas of Canine and Feline Dental Radiography* by Mulligan, Aller and Williams and published by Veterinary Learning System in 1997 or *An Atlas of Veterinary Dental Radiology* by DeForge and Colmery, published by Iowa State Press in 2000. All other veterinary dental texts also include radiographs of these lesions.

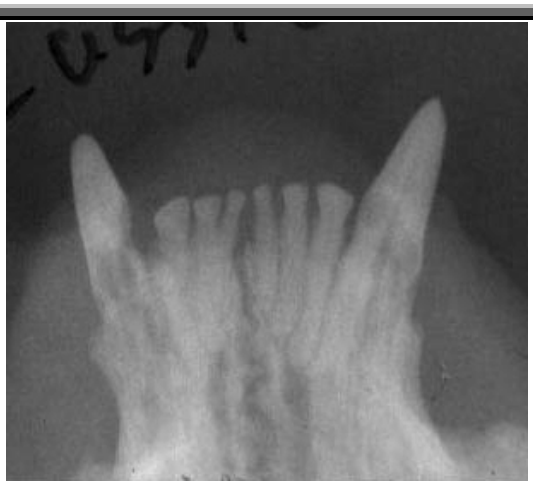

Figure #17.4. Type II RLs often affect the roots of canine teeth. Only in advanced cases do the lesions extend above the gingival attachment to become detectable on probing. Intra-oral radiographs are the key to detecting these.

More on the treatment of Type II RLs

About ten years ago, **Gregg. DuPont** published a paper in the Journal of Veterinary Dentistry suggesting that *in very particular circumstances*, it **might** be permissible to leave some root remnants in place when extracting teeth with resorptive lesions. Unfortunately, this message got bastardized until the word on the street was that is was okay to just snap off the crowns and leave the rest to sort itself out. This gets back to the axiom about a little bit of knowledge being dangerous.

I will be the first to admit that veterinary dentists do not have all the answers with regard to RLs yet, however, we do always take dental radiographs as part of our evaluation of RLs and that is absolutely essential.

As I have stated, type I lesions should be completely extracted. Also, teeth with any radiographic or clinical evidence of deep periodontal or endodontic disease should be extracted completely. Teeth in cats who are positive for FeLV, FIV or who have a history of chronic oral inflammatory disease (lymphplasmacytic gingivitis/stomatitis) should be completely removed.

If a tooth has a type II lesion without periodontal or endodontic disease (as confirmed radiographically) and the root appears to have been largely replaced by new bone-cementum tissue, then it might be permissible to leave a little of this tissue in the socket. The theory goes that the lesions are non-inflammatory and the tissue is healing the defect. My worry is that the lesion has been chronically exposed to oral bacteria by the time the lesion is detected and so I expect that bacterially induced inflammation must extend at least part way down the “root”. Therefore, my approach is still to remove all identifiable dental tissue and then suture the wound closed.

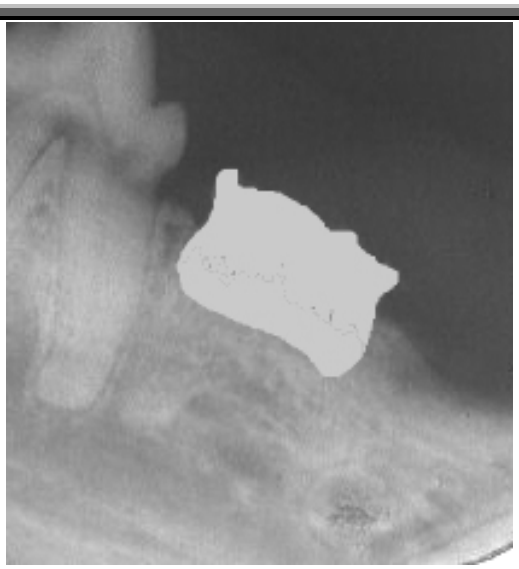

Figure # 17.5. The shaded portion of the stage 5, type II lesion is the minimum which must be removed after reflecting gingival flaps and prior to suturing the gingiva over the site.

Even those who advocate the more conservative approach of "intentional root retention" agree that removing just the crown is not enough. In Figure #17.3, we can see that there is still some coronal enamel at the most distal aspect of the tooth and there are also portions of the root projecting above the alveolar crest. Much of the root tissue has been replaced by bone-cementum tissue and so the roots appear as ghosts. The gingiva overlying this tooth typically would have a fistula and so there is ongoing bacterial invasion. Left on its own, the rest of this tooth will likely resorb, but in the meantime, there is an open and contaminated wound with inflammation and pain. At the risk of being repetitive, my approach would be to reflect gingival flaps, remove all visible or radiographically detectable dental tissues, smooth the bone and suture the wound with 5-0 monofilament absorbable.

The other way some people would manage this lesion would be to reflect the gingival flaps and then just remove the portion I have shaded in Figure #17.5 while leaving the buccal and lingual alveolar plates in tact. Next, they would smooth the alveolar margins and suture. They would always document this radiographically and fully disclose to the owners that this has been done, that it is still a "new" technique and that radiographic and clinical follow-up is required.

Keep in mind a study of the causes of mandibular swellings in cats (Kapatkin A et al. Mandibular swellings in cats: Retrospective study of 24 cats. JAAHA, 1991; 27(6):575-580) that found that of the non-malignant causes, retained root fragments was the number one cause accounting for 9 of the 12 cases. In no way can Dr. DuPont's trial be construed as license to leave roots behind simply because removing them is challenging. Unless you have good quality, intra-oral radiographs and know how to interpret them properly, there is no way to determine if a tooth is a candidate for root retention. As Dr. DuPont says, "If you bury pathology, it will come back to haunt you."

How do I want you to manage RLs?

In my fantasy world, you would just refer them all to me.

If you are going to treat cats with RLs here is what you have to do:

- Radiograph every tooth in the head (even those that appear to be missing) and examine each tooth subgingivally with a dental explorer. If you are not going to radiograph, then do not try to treat these cats, just refer them to someone who will radiograph them.
- For type I lesions, extract the entire tooth and root, smooth off the alveolar bone and suture the wound closed with a fine absorbable monofilament.
- For type II lesions, extract as much of the tooth as you can without causing excessive trauma (that is a judgment call), being sure to at least get everything above the alveolar crest and 1 – 2 millimeters into the socket before smoothing the bone and suturing as above.
- Plan to re-examine and re-radiograph annually as cats that have had one lesion will almost certainly get others sooner or later.
- Send the animals home with analgesics (I like meloxicam) for at least four days and put them on softened food for 14 days.
- Antibiotics are for the treatment of infection, not for the prevention of infection. When I do oral surgery, I remove the

infected tissue and so only rarely do I feel the need to send home antibiotics.

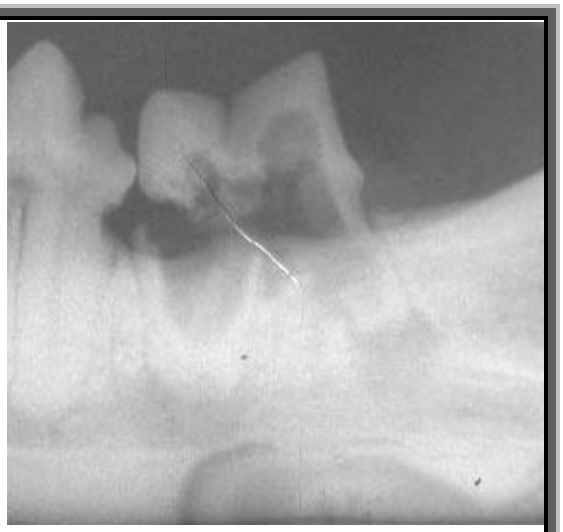

Figure #17.6. Here's a beauty. The crown of the left mandibular molar is hanging on by a sliver of dentin along the distal aspect of the distal root. There are large apical lucencies below both roots of this tooth. Complete extraction of all remnants of this tooth before suturing the gingiva over the site would be necessary. Leaving any portion of these roots behind would ensure persistent infection and pain. Post-operative antibiotics would be indicated in a case like this.

Chapter 18: Lymphocytic Plasmacytic Stomatitis

Another enigmatic problem seen in cats (mostly, though I have seen it in dogs a number of times) is chronic lymphocytic/plasmacytic stomatitis (LPS). The typical history is that of a cat with very inflamed gums, often with only minor calculus accumulation. The cat may have had its teeth cleaned several times and may have had extractions. The mouth often looks better after a cleaning and a course of antibiotics, but the improvement does not last long. If the inflamed gingiva has been biopsied, the pathologist almost always reports lymphocytic-plasmacytic gingivitis.

There have been many theories proposed and published as to the etiology of LPS. Many of these theories have been elevated to the status of fact through repetition but without any scientific evidence to support them. A literature review on the subject was presented by F. J. Verstaete at the Annual Dental Forum in 2000. Dr. Verstraete was looking to separate the speculation from the scientifically verifiable. He asked the question, "what is there in the peer-reviewed journals versus what has been published in proceedings, trade publications and by word-of mouth?" In so doing, he found that the only statement he could make regarding the etiology of LPS is that it is an *atypical immune response of unknown etiology*. Every other statement ever published regarding the cause of LPS he discarded as having no scientific basis.

All We Can Say With Confidence is that it is An Atypical Immune Response of Unknown Etiology

There is no one factor that is common to all cats suffering from chronic gingivitis-stomatitis. Some will have underlying disease of some sort, and a thorough work-up is indicated to assess the general heath status of the animal. Any disease that might interfere with the cat's local immunity in the gingiva may play a part.

Cats should be screened for FeLV, FIV and FIP though they are usually negative for all three. Direct IFA for calicivirus in biopsy samples may reveal a chronic local viral infection, though the significance of this is not clear. Disorders of the circulation such as diabetes mellitus can play a role as can immune disorders such as SLE and food allergies. In fact, almost any severe systemic disease will contribute to gingivitis in a susceptible individual. These other factors should be identified in the interest of being thorough. It does no good to get the mouth in order and then lose the cat in a ketoacidotic crisis.

Another fairly recent suggestion (by Hardy, WD at the Annual Veterinary Dental Forum, 2002) has suggested *Bartonella* infection as a cause. The proponent of this theory has a small credibility issue in that he owns the laboratory that has the serological test for Bartonella. His claim was that in the short term, the cats responded favourably to a dental cleaning with appropriate extractions and treatment with azythromycin. However, we know that, in the short term, almost all cats respond well to a cleaning and extractions alone, so what added benefit was attributable to azythromycin is unclear. Longer-term studies are required to see if there is anything to this theory.

If your work-up has revealed systemic disease, managing it may make managing the gingivitis easier. One factor which is common and cannot be altered is genetics. There seems to be a much higher incidence of gingivitis-stomatitis in highly bred cats such as Siamese, Abyssinian and Himalayans.

To simplify things (always a risky move), these cats act as if they have a complete intolerance to dental plaque. Even a thin, invisible film elicits a dramatic over-reaction of the cat's local immune response. Therefore, one approach to treatment of feline gingivitis-stomatitis depends on getting the mouth plaque free and keeping it that way.

The first step involves a thorough assessment and management of systemic disease. Next comes a *thorough* dental prophylaxis. This procedure must include scrupulous cleaning of the crowns and any root surfaces exposed by loss of gingival attachment. Any anatomical features which would be plaque retentive must be eliminated. This would include 'RLs', exposed furcations, over crowded teeth and retained roots from previous extractions. Once scaled and planed, the teeth should be polished with a fine paste, rinsed, dried and treated with a fluoride

gel. A final coating with a hydrophilic wax polymer (Pro-V-Seal™) may slow down the recolonization of the crowns with plaque bacteria.

Now that the mouth is clean, it must be kept that way. Discharge the patient with a seven-day course of antibiotic and an oral antiseptic such as zinc ascorbate (Maxi/Guard™) to be used daily. If there have been extractions, have the owners feed soft food for a week. Schedule a recheck for seven to 14 days post-operatively at which time you can assess healing. Since the absorbable suture material used to close any extraction sites will retain plaque, you can expect some reaction to persist until these sutures have disappeared.

Once the mouth is comfortable, the owners should start to work on home care. Home care should include daily brushing with an antibacterial dentifrice such as zinc ascorbate gel or an enzymatic paste.

An attempt should be made to get the cat onto a hard, dry diet. Eating kibble does not really clean the teeth, but eating canned and semi-moist will get them dirty much faster. After a meal of wet or semi-moist food, there is a lot of food left in the buccal pouch, in the interdental spaces and at the gingival margin. This retained food feeds oral bacteria and supports the rapid development of plaque.

Hill's Prescription™ Diet feline t/d™ has been shown to aid in plaque control and should be considered if there are no dietary requirements which take precedence.

Have the cat back monthly for rechecks to keep ahead of the problem. If the owners are successful with the home-care program, a prophy every six to twelve months may keep the problem under control.

The problem with the above plan is that these cats often have considerable oral pain and they simply will not allow the owners to brush the teeth to remove plaque and so the problem comes back within months or even weeks of treatment. Keeping the animals comfortable with antibiotics and steroids until the home care program is established may help.

If despite your best efforts (and the client's) at controlling the disease the gingivitis recurs, the next step is extraction of all premolars, molars and retained roots thereof plus a scrupulous cleaning of the canines and incisors with extractions of them as indicated (based on clinical and radiographic examination). With the posterior teeth removed and only the anteriors to worry about, homecare becomes considerably easier. For some cats, the reduced plaque load allows the condition to go into remission.

If the problem still recurs, remove the remaining teeth and be certain there are no retained roots from previous extractions or RLs.

Once all the teeth and retained roots have been completely removed, there is a high cure rate with no need for home-care or further dental work. If there is still inflammation in the mouth then you may have missed a tooth root somewhere or there may be a sequestrum of necrotic bone to be removed.

Depending on the animal and the client, you may opt for extraction sooner rather than later. Just be sure of the diagnosis first.

Unfortunately, some cats fail to respond to even whole-mouth extraction. Cats that present with inflammation around the teeth only tend to respond well to extraction. Cats that have the inflammation extending into the oro-pharynx present a greater challenge.

Another prognostic indicator is response to conservative treatment. If a cat responds well, though transiently, to a routine hygiene procedure and/or antibiotics, this suggests that they will respond well in the long-term to extraction. If there was no response to the initial treatments, then the prognosis with extraction is not as certain.

Many medical regimens have been tried to treat or control LPS and most have shown some promise in some cats for some time. These would include antibiotics, corticosteroids, levamisole, bovine lactoferrin, co-enzyme Q10, cyclosporin and interferon. None of these protocols have given consistent or lasting results and none of them can reverse degenerative changes such as RLs and periodontal disease. If an owner wishes to preserve teeth, the first step *must* be a thorough cleaning with extraction of significantly compromised teeth. Once this is done, medical management has a much better chance of helping as the challenge has been greatly reduced. These medical regimens may also have a place for cats that continue to have trouble following whole-mouth extraction.

In my experience, by the time a cat is referred to me for treatment of LPS, the conservative options have been tried and have failed. Under close examination, I frequently find that many of the significant teeth have RLs and/or extensive periodontal disease (gingival recession, pockets, bone loss) and that these teeth have to come out. Following removal of the teeth that have to be extracted, those that remain are often of little functional value, and since they are prone to be ongoing sources of trouble, I feel it makes sense to remove them as well. Therefore, in my practice, the typical protocol for the treatment of LPS is either extraction of all posterior teeth or extraction of all teeth. Anecdotally it seems that the sooner this is done, the better the response.

When I suggest whole-mouth or posterior tooth extraction to owners, many are concerned how their animal will cope without teeth. My experience is that they cope extremely well. Many are eating hard, dry kibble again within weeks and I have known of some edentulous cats that continued to hunt successfully.

Figure #18.1. One of these cats had every tooth removed due to chronic oral inflammatory disease a few months before this picture was taken. It was the gray tabby on top. He is obviously managing just fine without teeth.

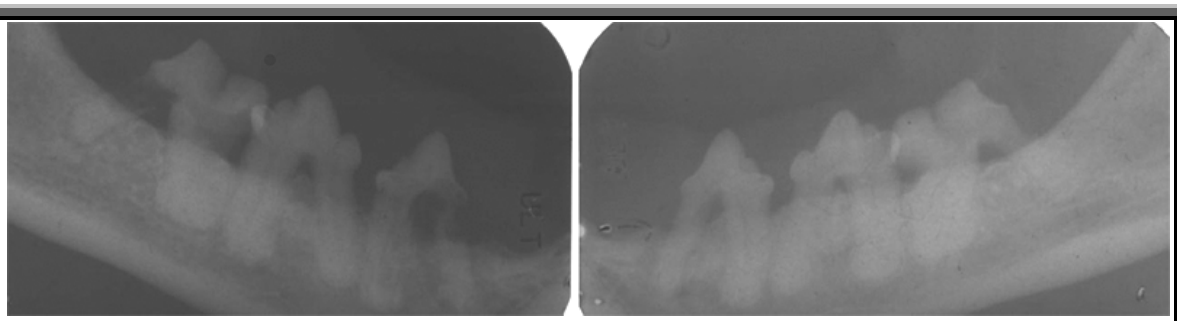

Figure #18.2. The mandibular radiographs of a cat with chronic oral inflammatory disease. The degree of resorptive lesions and periodontal disease would indicate extraction of every one of these teeth even if the gingival inflammatory condition could be magically “turned-off” with the flick of a switch. Removal of these teeth may bring about complete resolution of the signs. If not, extraction will at least make it much more likely that the cat will be able to respond favourably to medication.