BOOK OF PROCEEDINGS
OF THE
25TH EUROPEAN CONGRESS OF VETERINARY DENTISTRY

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Keynote lecture
Insights:
Oral and maxillofacial surgery

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How do oral and maxillofacial surgery and veterinary medicine relate? First of all Vets can operate on people in an emergency and most doctors cannot operate on animals. My first Professor in OMFS was asked to operate with a Vet on a valuable horse’s overshot lower jaw, the operation was a great success but the horse died. Doctors can usually talk to their patients but Vets need to read their patients’ clinical signs. I am not sure who has the advantage. My University career in started in veterinary medicine in TCD in 1971. Having been brought up on a farm, I worked closely with animals, and as a young teenager helped the Vet in his duties. The more, different disciplines, work together, the more we see of how the same diseases are handled in different areas, the more we will be able to do for our patients/animals.

Mouth cancer is my main area of interest, including its excision and reconstruction and this presentation hopes to show you what can be done for humans from cancer diagnosis, to excision and oral rehabilitation. How much is an animal worth? – I have no idea and can never put a price on another person’s emotions.

Facial trauma is a major problem in OMFS and hopefully some of the techniques illustrated will help.

Dental extractions in humans can be difficult but having seen dental extractions in dogs, doctors/dentists are on the right side of the table. Hopefully some of the techniques illustrated, may be useful to you. With the drill in the surgeon’s hand, no tooth can win.
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Presentations
Forget “low T”. What about “low D”?

Barden Greenfield

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AAHA well managed practices can have 7-10% of their gross related to their dental services. The question you may ask yourself is how can this be done, especially in a volatile economic situation and more competition? The answer lies in one general theme, and from this theme everything develops... MINDSET.

If you are not sold on providing high quality dental services for all your patients (from juvenile to super seniors), no implementation of any kind will allow your hospital to reap profits that they should. This lecture will address the most important aspects of increasing dental profits for your practice: Universal team member mindset; Promote dentistry year round, not just in February; Ditch the word ‘dental’ and never mention it again!; Audio-visuals of your dental program; Never leaving a stone unturned; Stop and slow down to maximize profits; Educate yourself and recapitalize your practice with state of the art equipment. Each is independent but directly related to the other!

Universal team member mindset

Marketing your dental practice comes from the top and the ownership must buy into the need to provide comprehensive oral care for all stages of a pet’s life. Essentially, all professional staff must believe that dentistry is important to the patient’s well being. Buy-in will be difficult if doctors perceive dentistry as just an ancillary service that should be offered by groomers or paraprofessional staff. For doctor’s with this ‘old-fashioned’ opinion of dental care, additional dental CE is essential to changing their paradigm. If the owner and associates are not on board any marketing program is doomed to fail. I have spent time at many veterinary practices where the technicians are wanting to market dentistry, but the owner would rather sit in the office and read emails. This is unfortunately the standard instead of the isolated incidence, and dentistry must be discussed for each patient day after day after day after day. Just like marketing heartworm and flea protection, it has to be mentioned at every visit.

Profits from dentistry services have the highest ROI, so why are so many people reluctant to promote and perform dental procedures? The answers lie in emotion and time. Treat marketing dentistry like training and running in a marathon. Don’t sprint out of the gate but rather as my wife says, “start out like you can hold out”.

Your Pet Dentist of Memphis, and Little Rock
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http://www.yourpetdentist.com
I HIGHLY recommend your hospital become members of the AVDS (American Veterinary Dental Society). Not only do you get discounted registration to the Annual Veterinary Dental Forum (this year it is in Atlanta, GA Nov 13-16), but also you receive the Journal of Veterinary Dentistry (JVD) quarterly which is loaded with tremendous information for the dental novice, as well as the dental specialist. (www.avds-online.org)

**Promote dentistry year round, not just in February**

The AVDS and Hill’s Pet Food started National Pet Dental Health Month many years ago to help promote awareness for pet dentistry. While this program has had good intentions, it has been transformed by veterinarians into a discounted ‘dental’ month. Many practices brag on how many ‘dentals’ they performed each day or for the month. Unfortunately, what has happened is shoddy and speedy dental procedures have taken the place of a truly comprehensive dental procedure, which will be discussed below. There is absolutely no way in the world that a practice can perform more than 4-5 dental procedures in a day, no less in a morning as so many practices claim. The general public is therefore hoodwinked into thinking their dog/cat has had a comprehensive dental procedure just by having supragingival and subgingival scaling.

**Solution:**

1. Market dental procedures year round and don’t have clients wait until February to schedule a procedure.
2. Avoid discounted dental procedures. Instead, offer VOHC-approved treats (www.vohc.org) and possible T/D diet (or other VOHC-approved diets) as a to-go home.
3. Schedule no more than 3-4 procedures / day and make sure the DVM (which is YOU!!) is available for all procedures to check behind the technician with dental probing, review of dental X-rays, and possible treatment plans going forward with pathology seen during the exam).
4. Stage procedures if you can’t do everything on the day of the initial dental procedure.
5. ALWAYS have a 10 -14 day recheck for EVERY dental patient. My compliance in private practice during my residency was almost 100% and is 100% in the specialty practice. This is the time you and your team sit and go over the next 6-12 months.
6. Forget the 12-month cycle. If your patient has advanced periodontal disease, don’t wait a full calendar year to re-treat. Many patients require 3, 6, or 9 month sedating rechecks/procedures.

**Ditch the word ‘dental’ and never have your team members use it again!**

There is no such procedure as a ‘dental’. Is there an ‘internal medicine’, ‘cardiology’, or ‘dermatology’ procedure? No, there is not. Veterinarians are KILLING themselves financially by using this term. I will be using examples of why this is hurting the practice and taking away much needed profits.

The proper term should be **Oral ATP (Assessment, Treatment and Prevention) or Professional Teeth Cleaning or Periodontal Therapy**. You and your team must inform the client that you are going to call them after the initial diagnostics and scaling/polishing/
probing/ and YES RADIOGRAPHS, to then move forward on the second part of the procedure.

Most of my dental income comes AFTER the initial workup, not before it. That was true even during the 1st year of my residency. You must inform the client that there may be pathology that you can’t diagnose until you get in the procedure, so don’t shoot yourself in the foot by saying the ‘dental is $XYZ’. Tell them the initial part of the procedure is $XYZ, but ‘I will call you after I get the initial work done to let you know if I find anything that needs attention’.

Audio-visuals of your dental program

Your team should have available in the waiting room picture books of your ‘before and after’ procedures with a picture of the pet and even the client in the book. Have a ‘Wall of Fame’ of pictures of patients that recently had a dental procedure and brag on them to your clients. If you have a TV monitor, find a stock reel of someone discussing the need for dental care for your patients and it is very important to have YOUR NAME(S) mentioned on the reel. “Dr. Smith has learned that 8 of 10 dogs and cats have periodontal disease, so dental care for your pet is paramount for their overall health and well-being. The doctors of ABC Animal Hospital are committed to providing quality dental care for your beloved pet” (or something like that).

Smile Books should be in each exam room. Your On-Hold messages should reflect your awareness and need for dental care.

Examination of the oral cavity should include a lot more than just flipping the lip. Look closely for gingivitis, oral growths, malocclusions, discolored teeth, fractured teeth, and crowding. Your Smile Book should have each of these problems in it with a brief description and treatment plan.

Your Website and Facebook are PRIME areas for marketing your services and bragging on your clients/pets. Everyone loves to see pictures of their own pet on FB, so have a delegated team member available to do just that (EVERY DAY). There is nothing worse than a stale website. It should be active and promote those services that you find most important.

Every Oral ATP patient must have a Discharge Information Sheet with Pictures to describe the procedure. Keep a digital camera in each exam room to be used to take a picture of the pet before sedation and then incorporate that image with the other ‘before and after’ images that are needed. Use a good image stock paper and don’t be cheap on just using paper. Buy heavy glossy paper to be used for your take home images. You can even keep a second copy (with normal paper) in the pet’s record to remind the owner of the procedure being done.

Never leave a stone unturned

Qualified eyes must look at every patient seen in the exam room. Noticing and treating malocclusion at a young age will alleviate a life of pain and suffering, while making a content owner. And it adds profit to the practice.

Start dental education with your clients at an early age. As owner compliance for brushing is roughly 5%, it should be recommended to everyone but understood that very few can succeed at 3 times/week brushing. This is a place for VOHC-approved chews and diets and it is best recommended from you and your team vs. a teenager working at a pet supply store. Your team should be educated on the types of chews and market them heavily. I especially
love Tartar Shield treats (www.tartarshield.com) but Greenies and CET Veggiedents are also VOHC –approved chews. Give them as treats to your patients and have them marketed well in your display area. I also aggressively market Healthy Mouth water additive (www. healthymouth.com). While this product is a lost-leader as far as profits go, it provides an excellent way to provide an anti-plaque product passively to your pet.

Things to look for in the exam room: fractured teeth, discolored teeth, missing teeth, oral masses, gingivitis, halitosis, malocclusions, mobile teeth, etc. Cats especially have a high % of tooth resorptions (up to 70 % of mix breed cats have TR’s) and gingivostomatitis.

Another phrase to eliminate from your vocabulary is “let’s keep an eye on this”. That’s like saying, “Let’s find another way to shoot yourself in the foot!” Keeping an eye on a broken tooth, discolored tooth or anything mentioned above is truly a great reason for your client to go down the street and find a new family vet. If there is pathology, recommend treatment… plain and simple. This is where PASSION must be present, for if you don’t think it needs treating, then it won’t. If you passionately recommend treatment and your costs are fair and reasonable, most pet owners will heed your advise and proceed. Using key words like “painful, uncomfortable, suffering” are justifiable and warranted in most dental cases where gingivitis, tooth fracture, oral mass, or discolored tooth are present.

A new diagnostic tool that can be used in the exam room is OraStrip. This test strip measures volatile sulfurs in the mouth (thiols) created by the black-pigmented anaerobes in the mouth. These anaerobes are present in periodontal disease and are easily quantifiable on the strip. Your team then shows the owner the strip with the results that in many cases convinces them to schedule an ATP. This test costs roughly $10/test so be sure to add this in some fashion in the exam if possible. I recommend treating this as a lost leader and not marking up much as the ROI is very good if the owner sees the data and schedules the appointment. I also recommend OraStrip tests to be administered either by the owner in 4-6 months or having the owner come to the practice to have a follow-up test. This lets you and the owner see how well home care is being done and a new procedure may need to be scheduled. (The test strip is already charged out at the time of the Oral ATP).

Golden opportunities missed by most practitioners that warrant dental diagnostics/treatment:

1. Missing teeth (seen at spay/neuter and Oral ATP). If a tooth is missing, it could be embedded leading to a dentigerous cyst. Dental radiography is vitally important.

2. Malocclusions (seen at puppy visits, sedation for spay/neuter). You or your tech must look at the occlusion PRIOR to intubation to see if there is attrition (tooth on tooth contact). If so, interceptive orthodontics to remove the offending tooth/teeth must be performed.

3. Persistent primary teeth (seen at spay/neuter or 1st annual exam). The best time to prevent malocclusion pathology is early in life. Removal of persistent primary teeth (previously called retained puppy teeth) must be done and not ‘wait and see’.

4. At 1-3 years of age when early gingivitis is present. Gingivitis is the only part of periodontal disease that is reversible. Once Stage 2-4 PD occurs, bone loss occurs and the body can’t reverse bone loss. Get those cases in early and don’t depend just on calculus. That is not what you should be looking at, but rather the gingival margin.
5. Geriatrics. Don’t be afraid to sedate a senior or super-senior pet. If you haven’t already learned, age is not a disease and never will be. A large part of my wellness practice involves Oral ATP’s of aged pets because veterinarians are scared to sedate an aged pet. If you have proper blood work, urine and even thoracic rads (in some instances) as well as the proper anesthetic monitoring devices (HR, BP, EKG, CO2, RR, Oxygen Saturation) then sedation is not difficult. Study up on anesthetic monitoring and trends and don’t be afraid to use drugs such as dobutamine to help hypotension. I would recommend avoiding using Telazol in your Oral ATP’s as an induction agent. I get a lot of complaints from pet owners that had bad anesthetic recoveries when Telazol administered as an induction agent by the previous DVM. Multiple anesthetic agents such as Diazapam, Midazolam, Propofol, Hydromorphone, etc., can make an anesthetic event quite uneventful and owners very happy at discharge.

6. Cats: Tooth resorptions (TR) are prevalent in both mix breeds and pure breed cats. However, if you don’t have dental radiography, performing an Oral ATP in a cat is ill advised due to the % of pathology that is present below the gumline that otherwise appears clinically normal. This may sound high brow but the AVDC and AAHA recommend dental radiography in all annual dental procedures.

But if you do have dental radiography, this is the golden opportunity to market your practice and set yourself apart from neighbors that have do not have x-ray capability

**Stop and slow down to maximize profits**

As I mentioned earlier, performing an abnormal number of procedures daily (>4 in a complete day) only makes for a multitude of procedures performed poorly. We teach quantity over quality with regard to performing oral/dental procedures.

These are the diagnostic and treatment procedures that you should be able to perform at your practice with a moderate amount of dental education:

1. Dental radiography (full mouth in a dog and cat).
2. Closed root planning.
3. Interceptive orthodontics.
4. Perioceutic Application in periodontal pockets.
5. Surgical flaps for extractions (yes, charge for these).
8. Regional nerve blocks.
10. Open root planing (advanced dental knowledge needed).
11. Bone grafts to fill infrabony pockets (advanced dental knowledge needed).

Remember to charge for your anesthetic time. Avoid having a tier for your periodontal cleanings, as it should take roughly the same amount of time to scale every pet. Why would you want to clean a diseased tooth? Therefore, if it needs to be treated otherwise via extraction, root planing, etc., charge for those independently. Don’t tier dental cleanings.
Educate yourself and recapitalize your practice with state of the art equipment

The unfortunate truth is you probably received marginal or no dental education in veterinary school. To be comfortable recommending any diagnostic or therapeutic treatment, veterinarians must be confident they can properly perform the advised treatment. Therefore, it is up to YOU to learn basic dental skills that you should have learned in school. Cleaning and polishing teeth should not constitute the entirety of your dental knowledge.

Many diplomates, including myself, offer intense dental education for DVM’s and technicians. These meetings are more intimate than the larger national meetings that have 30-40 attendees in a wet lab environment. However, national meetings (AAHA, NAVC, Western States, AVMA) do have excellent lecture platforms and laboratories for you to increase your dental IQ. National meetings offer you the chance to talk to vendors that carry dental equipment and look at a variety of instruments/equipment.

These are the basic instruments/equipment that are ESSENTIAL, not recommended:

1. High speed/low speed unit.
2. Weldin Periodontal kit (Miltex).
3. Winged elevator kit (size 2-8).
4. Dental radiography generator.
5. Size 2 digital dental sensor OR Indirect digital system (Scan X or IM3).
6. 4-0 and 5-0 absorbable suture (Monocryl or Gut advised).
7. Composite placement instrument for placement of Doxyrobe (Miltex).
8. Doxyrobe gel.
9. Small size (4 ½” or comparable) needle holders and thumb forceps.
10. Periosteal elevators (Miltex, Cislac, IM3).

In my humble opinion, sectioning a tooth with a Dremel, giggly wire or other primitive method is to me tantamount to malpractice. Leaving a tooth root remnant during an extraction procedure places you and your practice at extreme liability. The only way you can confirm you removed a tooth is via dental radiography, therefore, it is also a legal document you performed that which you charged. Not performing surgical flaps (non-tension) for extractions can lead to oronasal fistula, delayed healing of an extraction site, and undo oral pain for your dental patient. While this sounds very high brow and holier than thou, it really isn’t. It’s just basic dentistry done right and we have to rethink these sub-standard practices that were engrained to generations of veterinarians before us.

Following these guidelines and changing your mindset regarding your dental practice will reap profits up to 8-10% of gross revenues of your practice. The fact is that recommending excellent dental care for your patients is not only practicing ‘good medicine’, but it is also good business. It's not a gimmick or newfangled approach, but solid, tested and true practices.
Dental tips and tricks for the general veterinary practitioner

Paul Cooper

There is no full text for this, it is essentially visual, using a power point presentation of tips and tricks to make dentistry easy, and avoid common pitfalls.
The purpose of anesthesia is to have safe, effective chemical restraint for medical and surgical procedures with minimal stress, pain, and toxic side effects to the patient or anesthetist. Every patient should have pre-anesthetic evaluation and planning. Also, the anesthesia should be individualized for each patient. An IV catheter should be placed in each patient prior to inducing anesthesia if possible and if the animal is too fractious, place the IV catheter immediately after induction.

Anesthesia for the veterinary dental patient has some very specific concerns. The patient needs to be under anesthesia to chart and clean and radiograph the entire oral cavity adequately. Every patient must be intubated to prevent water, calculus and debris from going down the trachea. In many practices the technician is cleaning the teeth by themselves and may have trouble monitoring anesthesia as well. The patients may be on a wet table or a metal table, with their heads getting wet from the ultrasonic scaler and may become so hypothermic that it is life threatening. Often, there is intermittent or no pain stimuli and the procedures can be quite lengthy predisposing the patient to hypotension.

Having a dedicated anesthesia technician: monitoring and recording the anesthetic parameters every 5 to 10 minutes and having sufficient monitors can prevent anesthetic emergencies and death. Anesthesia can be safe for most, if not all, patients: if pre-anesthetic planning and principals of good IV fluid therapy are performed, adequate staff monitoring anesthesia is provided, good anesthetic and monitoring equipment is available (and used) and appropriate responses to anesthetic complications are implemented. Anesthesia should be a team effort, with the veterinarian, support staff, and technicians all involved and communicating continuously with each other.

The immediate post-operative period is a critical one. A patient can become hypothermic quickly, even if the body temperature was normal during the procedure. If they became cold during the procedure, they may not be able to recover without supplemental heat. Remember that serious metabolic issues and even death can occur from hypothermia. Vomiting and aspiration has been known to occur in the post-operative period. This can also lead to critical illness or death.

The most important monitors are human senses. The staff member monitoring anesthesia is the most important monitor of all! The most appropriate method for assessing the depth
of anesthesia in veterinary practice remains the assessment of clinical signs.\textsuperscript{4}

**Normal ranges for anesthetic monitoring:**

- **Temperature:** 99 to 102.5 °F.
- **Blood pressure MAP:** 70-90, systolic 90-180.
- **Heart rate:** small dog or cat 90-180bpm, big dog 70-120bpm (if fit: 50-110bpm).
- **Respiratory rate:** 8-20 respirations per minute.
- **End Tidal CO\textsubscript{2}** 35-45 mmHg.
- **Pulse Ox:** 99-100%.
- **ECG:** Any abnormal wave form inform the veterinarian.

**Temperature:** Temperature monitoring is critical for the veterinary dental patient. They can lose heat through metal tables, wire bars over open air, cool operatory temperature, wet fur, ears and body from the ultrasonic scaler, heat loss through the endotracheal tube during respiration and cool IV fluids. There should always be dry and warm blankets and supplemental heat supplied, especially for small and thin-coated animals. Circulating water blankets, convection heat and warm blanket systems can all help. Avoid hot water bottles and heating pads as burns can result.\textsuperscript{3}

**Blood pressure:** Hypotension is defined as a systolic pressure <90 mmHg or a mean of < 60 mmHg. This can be from inhalant gas anesthesia, hypothermia and positive pressure ventilation. This is usually measured using a Doppler or oscillometric non-invasive methods. The ideal cuff is approximately 40% of the circumference of the limb in dogs and 30-40% in cats. Wider cuffs will lead to underestimation of the true pressure.\textsuperscript{4}

**Heart rate:** Small dog or cat 90-180bpm, large dog: 70-120 bpm (if athletic: 50-110bpm).

**Respiratory rate:** 8-20 respirations per minute.

**End Tidal CO\textsubscript{2} (capnography or capnometry):** The EndTidal CO\textsubscript{2} should be between 35-45mmHg. The measured ETCO\textsubscript{2} is 3-5mmHg less than the actual PaCO\textsubscript{2}. Observing and understanding the wave forms allows assessment of ventilation, the breathing circuit, and ventilation-perfusion function in the lungs. This is also a very important monitor when using a ventilator.\textsuperscript{5,6}

**Oxygenation (pulse oximetry):** Pulse oxygenation monitoring allows non-invasive measurement of the saturation of hemoglobin with oxygen ($S_pO_2$). The percentage reading on the pulse ox monitor should be between 99-100%. If apnea occurs there is no change in the pulse oxygenation reading for 3-5 minutes. Hypovolemia, hypothermia, motion, pigment, placement or use of electrosurgical equipment can all result in inaccurate readings. This is especially important in pets with cardiac or respiratory disease. In conjunction with the ETCO\textsubscript{2} it can give complementary information on ventilation.\textsuperscript{4}

**Blood gases:** An Istat machine can do accurate blood gas analysis during anesthesia. This is important when using a ventilator. The sublingual vein values are quite close to arterial values and are much more accessible than arteries for collecting blood for blood gas analysis.

**ECG:** Any abnormal waveform should be brought to the attention of the veterinarian. VPCs and 1st or 2nd degree heart block are fairly common anesthetic complications.\textsuperscript{4}

**IV fluid rate:** The current recommendations for the IV fluid rate are: 2-6mL/kg/hr for dogs and 2-3mL/kg/hr for cats.\textsuperscript{7}
Use of opioids and local anesthetics to smooth anesthesia during painful procedures: A fentanyl bolus or CRI during painful procedures can eliminate or reduce the need to increase the vaporizer settings.

Post operative monitoring: It is extremely important to continue monitoring your patients after the procedure has ended. Many anesthetic complications and deaths occur after the procedure has ended. These complications can include aspiration, airway obstruction, hypo- or hyperthermia and others.4

There is a Veterinary Anesthesia Support Group at VASG.org. There many informative articles and good information on this website.

Treatment of hypotension2

- Turn down vaporizer setting
- Fluid bolus (unless cardiac disease present) of 10-20 mL/kg.
- Bolus of Hetastarch or a polygelatin solution at 5-10mL/kg.
- Dopamine: 5-10ug/kg/min (with infusion rate of 1-2mL/kg/h).
- Dobutamine: 2.5-5 ug/kg/min (with infusion rate of 0.5-1 mL/kg/h).
- Ephedrine: 50-100ug/kg boluses (with infusion rate of 0.01-0,02 mL/kg per 15-20 minutes).

References

Dental radiography units

Radiographic exposure is controlled by 3 components: kVp (kilovolt peak), MA (milliamperage), and exposure time. KVP controls the “quality” of the x-ray beam. This is the power of each particular x-ray particle which controls the penetration of the beam through tissues.

The quantity of the exposure is controlled by MA and time of exposure. The higher the MA, the more X-rays produced over the time period. Multiply this number by the exposure time and you will get the total number of x-ray units.

Since there is not a significant amount of variation of tissues in oral radiology, the KVP and MA are set constant on dental radiology units. The only variable factor is time. This is measured in seconds or pulses. One pulse is equal to 1/60 of a second. Most standard (human) dental radiology units have a digital control for the exposure and it is set by the operator based on a technique chart. Recently, however, veterinary specific machines have become available which has a computer that sets the exposure based on the size of the patient, the speed of dental film used, and the particular object tooth. This can take a lot of the guesswork out of the exposure setting. However, with a little experience and practice, it is easy to figure out a setting.

Dental radiographic film

Dental film is non-screen film. This means that it is directly exposed by the x-ray and does not require an intensifying screen. This gives much more detail than standard radiographic film, but requires a higher amount of exposure. It is packaged in its own paper or plastic sleeve, to protect it from light and the oral environment.

There are two types of dental film commonly used in dental radiology. These are Ultra-speed “D” and Ektaspeed “E” film. Recently “F” speed film has become popular. The difference is in the size of the silver halide crystals and secondary to this the amount of exposure required to expose the dental film. “E” speed film requires approximately ½ the amount of radiation for exposure than “D” speed film, and “F” speed even less. This
decreases exposure to the patient and staff as well as decreases the wear and tear on the x-ray unit. There is a slight decrease in resolution with faster films due to the larger crystal size, but according to most experts, the difference is negligible. Therefore, it is recommended in human dentistry to use “E or F” speed to decrease exposure time. They are more technique sensitive, however, in both the exposure and development of the image. This may be frustrating for the novice, therefore it is generally recommended that practitioners start with “D” speed and advance to “E or F” speed when they are more comfortable with the settings and positioning.

There are several different sizes of dental film available (4, 3, 2, 1, and 0). The most common sizes used in veterinary medicine are 4, 2, and 1. Size 3 are bite wings and are generally not used in veterinary medicine. Size 4 (occlusal) film is the largest available, it is used mostly in large breed dogs or when taking whole mouth radiographs. For small dogs and cats and most any single tooth radiograph, size 2 (standard) is commonly used. For the mandibular first and second premolars, and very small cats and puppies size 1 (or 0) (periapical) are used.

Another consideration in selecting film size is cost. Size 4 film is about 3 times the cost of size 2. Therefore, if you can use a size 2, it is recommended. However, it is much easier to position size 4 films, allowing for much more latitude in positioning. This will result in less retakes. Therefore, the less experienced may consider practicing with size 4 film and graduating to size 2 when a level of skill is obtained.

**Digital dental radiology**

There are numerous human veterinary digital systems. These are excellent means of obtaining dental radiographs. The only major problem currently is the lack of a number 4 sensor. The major advantages to these systems are the decrease in radiation exposure, rapidity of the development, and that you can reposition the sensor if the view is not correct the first time. There is one company, however which makes a size 4 phosphor plate (CR).

**Taking a dental radiograph**

**Step 1: Patient positioning**

Position the patient so that the area of interest is convenient to the radiographic beam. In general this is where the object is “up”. For maxillary teeth, the patient should be in ventral recumbency. For mandibular canines and incisors the pet should be in dorsal recumbency. Finally, for maxillary cheek teeth, the patient should be in lateral recumbency with the affected side up. This being said, in our practice virtually all radiographs are exposed in lateral recumbancy. This takes some getting used to, but decreases the number of times a patient must be rolled when doing surgical or endodontic procedures.

**Step 2: Film Placement within the patient’s mouth**

There is an embossed dot on the film. The convex side of this should be placed towards the x-ray beam. In most films, this side is pure white. The opposite or “back” side of the film will usually be colored (purple or green). Place the film in the mouth so that the entire tooth
(crown and entire root surface) is covered by the radiograph. Remember, the roots of all teeth are very long. This is especially true of canine teeth, which are longer than you think. Always err on the side of having the film too far in the mouth to ensure you do not cut off the root apexes. The film should be placed as near as possible to the object (generally touching the tooth and gingiva) to minimize distortion.

**Step 3: Positioning the beam head**

There are two major techniques for positioning the beam head in veterinary patients. Both of these techniques are used daily in veterinary practice.

**Parallel technique:** This is where the film is placed parallel to the object being radiographed and perpendicular to the beam. This is how standard (large) films are taken. This gives the most accurate image. Unfortunately this is only useful in the lower cheek teeth in the dog and cat. This is due to the fact that these patients don’t have an arched palate. The film cannot be placed parallel to the tooth roots because of the palate’s interference. Therefore this technique is not always possible.

**Bisecting angle technique:** This is the most common type of dental radiograph taken in veterinary patients. This uses the theory of equilateral triangles to create an image that accurately represents the tooth in question. To utilize this technique, the film is placed as parallel as possible to the tooth root. Then the angle between the tooth root and film is measured. This angle is cut in half (bisected) and the beam placed perpendicular to this angle. This gives the most accurate representation of the root.

If this angle is incorrect, the radiographic image will be distorted. This is because the x-ray beam will create an image that is longer or shorter than the object imaged. The best way to visualize this is to think of a building and the sun. The building will create a 90 degree (right) angle to the ground. The bisecting angle in this case is 45 degrees to the ground.

Early and late in the day, the sun is at an acute angle to the building and casts a long shadow. In radiology this occurs when the angle of the beam to the object is too small and is known as elongation. At some point in the late morning and early afternoon, the sun is at a 45 degree angle to the building, which is the bisecting angle. This gives an accurate representation of the building height. As the sun continues up in the sky, the shadow shortens. This occurs in veterinary radiology when the angle is too great and is known as foreshortening. Finally, at noon, the sun is straight up from the building, which gives no shadow.
The “Simplified Technique” as developed by Dr. Tony Woodward does not utilize direct measurement of any angle, instead relying on approximate angles to create diagnostic images. There are only 3 angles used for all radiographs in this system 20, 45, and 90.

Mandibular premolars and molars are exposed at a 90 degree angle, maxillary premolars and molars at a 45-degree angle, and incisors and canines at a 20 degree angle.

To initiate any radiograph, place the film in the mouth and set the positioning indication device (PID) perpendicular to the film. For mandibular cheek teeth, this is the correct placement. For the maxillary premolars and molars, rotate the beam to a 45 degree angle. For the incisors and mandibular canines rotate 20 degrees. For the maxillary canines an additional rotation 20 degrees lateral is necessary to avoid superimposition of the first and second premolars.

**Step 4: Setting the exposure**

If you are using a machine where you set the exposure manually, you will need to set up a technique chart similar to one for a standard (large) unit. The good news is that there is only one variable that needs to be adjusted.

If you are utilizing the computer controlled system, set the buttons for the species, size of the patient, and tooth to be imaged. If you have correctly set the machine and the image is incorrectly exposed, the easiest way to adjust is to change the f setting. By pressing this button, you will see the numbers go up on both sides. The one on the left is the f number and the one on the right is the exposure time. If you continue to press the button it will continue to increase the exposure until you reach 9 when it will markedly lower and the f number will go back to 1. If the radiograph is overexposed (too dark) lower the f number by 1. If it is underexposed (too light) increase the number by 1. Continue this process until you have the film that you want. Generally, the f number will be the same for all radiographs once you have discovered the correct setting for your machine start at that number in future sessions.

**Step 5: Exposing the radiograph**

Dental radiograph machines have a hand held switch to expose the radiograph. If it is possible, leave the room prior to exposing the radiograph. If it is not, stand at least 6 feet away at a 90 to 130 degree angle to the primary beam (meaning to the side or back of the tube head, not in front or behind). Once everything is set, press the button. It is important to remember, that these switches are “dead man’s”. This means if you let up during the exposure, it will stop the production of x-ray beams. On a standard unit, this will make a light radiograph, on a computer controlled one it will give an error message and you will need to start over. Make sure you hold the button down until the machine stops beeping.

**Step 6: Developing the radiograph**

The most economical way to develop the radiograph is coffee cups filled with dental developing solutions in your darkroom. (Using chemicals other than products for dental radiology will result in inferior film quality.) Although developing films in a darkroom can produce quality films, the use of a chair side developer has several distinct advantages.
1. The chair side developer also allows you to easily judge when development time is correct, and be able to evaluate your films in only 1-2 minutes.
2. The technician does not leave the room and can still monitor the patient.
3. The units take up very little space, minimize chemistry use, clean up easily and store quickly.

To develop films, begin by peeling back the covering layers from the film, taking care to handle the film only by the edges. Use a film clip to grasp the corner of the film and place it in the developer. When developing a size 4 film, make sure to immerse the entire film in the liquid to ensure that the whole film gets developed. Develop the film until an image is just visible (sight developing). Then rinse the film briefly in a water bath, and place the film in the fixer for one minute until partially fixed. The film may be evaluated at this time, but should be placed back in the fixer for an additional 10 minutes to ensure complete fixation (archival quality). When completely fixed, the film becomes clear and will lose all traces of a greenish color. The film should then be thoroughly rinsed in running water or placed in a clean water bath for 10-15 minutes. This is followed by a final rinse to remove all traces of fixer. Be sure to remove the clip and rinse all film surfaces thoroughly. Traces of fixer remaining on a dental film give it a characteristic “slick” feel, therefore rinse the film under running water while gently rubbing the film between your fingers, for a few seconds, until the film does not feel slick. The film is then placed in drying clips overnight to dry. Make sure to dry the film completely to ensure that they do not stick together.

Be sure to change the solutions whenever the developing and fixation times seem to be slowing down. This will occur after you have developed and fixed around 20 smaller (#0 or #2) films, or 10-15 larger (#4) films. Use of exhausted chemistry results in poor image quality and hazy images.
Periodontal disease is commonly encountered in feline practice, with gingivitis and periodontitis being the two commonly encountered forms. This presentation will cover the aetiopathogenesis of periodontal disease in the cat; noting species differences to dogs, in particular implicated pathogens. Gingivitis, followed by inflammation and destruction of the rest of the periodontal tissues (periodontitis) can lead to chronic oral infection, bacteraemia, pain and tooth loss. Local effects of periodontal disease are discussed, including the association of periodontitis with type 1 tooth resorption. Evidence is also mounting that periodontal disease can have significant associations with systemic diseases. The evidence supporting the association between chronic kidney disease and diabetes mellitus with periodontal disease in the cat is examined. The talk will also cover the diagnosis including: conscious oral examination findings, periodontal probing findings under anaesthesia, and also radiographic signs. Management options will be discussed, using an evidence –based approach wherever possible. Gingivitis is a reversible and potentially controllable condition with efficient plaque control. Periodontitis however, is an irreversible and progressive condition. While guided-tissue regeneration techniques are possible, this must not be attempted unless homecare is optimal. Homecare in the cat is notoriously challenging. We will review the products and methods available for homecare in the cat, and discuss ways to communicate effectively with clients.
Options for mandibular fracture repair

Barron P. Hall

The mandible is made up of mirrored left and right halves that are firmly united rostrally at the intermandibular joint, known as the mandibular symphysis. This fibrous joint is a synchondrosis. The body of the mandible is the portion that contains eleven teeth. The mandibular canal lies within body of the mandible containing the neurovascular structures supplying the mandibular bone, teeth, and soft tissues. The ramus of the mandible is the distal portion that does not contain any teeth. The neurovascular complex enters through the mandibular foramen located on the lingual aspect of the caudoventral mandible. Its dorsal extent is the coronoid process and its most distal extent is the condyloid process. The condyloid process is part of the temporomandibular joint, a synovial joint. A thin articular disc within a loose joint capsule completely divides the joint cavity into dorsal and ventral compartments separating the articular cartilage covered surfaces of the condyloid process and the fossa of the temporal bone.

In order to repair a fracture and stabilize the segments, forces placed on the bone need to be mitigated. The muscles of mastication place a variety of forces on the mandible. These forces may help or hinder fracture repair, so these muscles should be understood. There are five muscles of mastication. The four used to open the mouth are innervated by the trigeminal nerve, while the two bellied muscle used to open the mouth is innervated by both the trigeminal and facial nerves. The masseter muscle originates from the ventral border of the zygomatic arch extending caudolaterally to insert on the ventrolateral surface of the mandible (masseteric fossa) with some fibers extending around the ventral and caudal border to insert on its ventromedial surface. The temporalis muscle is the largest and strongest of these muscles. It originates primarily from the parietal bone and to a lesser extent from the temporal, frontal, and occipital bones. The muscle lies within the temporal fossa as fibers extend dorsally and ventrally beneath the zygomatic arch to insert on the coronoid process with some fibers reaching the ventral margin of the massteric fossa were they blend with the masseter muscle. On the medial aspect of the mandible they blend with the pterygoid muscles. The lateral pterygoid muscle is smaller and shorter than the medial pterygoid muscle. It originates from a small fossa on the sphenoid bone extending ventrolaterally and slightly caudal to insert on the medial aspect of the mandibular condyle just ventral to its articular surface. The medial pterygoid muscle originates from the lateral surface of the
pterygoid, palatine, and sphenoid bones extends caudolaterally to insert on the medial and caudal surface of the angular process of the mandible. The digastricus muscle is the only muscle of mastication used to open the mouth. It originates from the paracondylar process of the occiput extending rostrally to attach on the ventral border of the mandible. The muscle appears to be a single bellied muscle, but a tendinous intersection divides the muscle into a rostral (trigeminal) and caudal belly (facial) with their own innervation.

As with other orthopedic injuries, mandibular fracture repair can be very rewarding or very frustrating. There are usually more than one option for fracture stabilization based on your training and the equipment you have. The way you approach a fracture can vary on the way that you were trained. Those trained from the dental/oral surgery side usually opt for intraoral fixation using the teeth, wires, composite, and acrylic. Those surgically trained are more likely to use an extraoral approach with IM pins, External Skeletal Fixation (EXF), or plates and screws.

There are multiple factors that need to be considered when planning on how to repair a fracture: patient, fracture, owner, and veterinarian.

1. The patient’s age, breed, neutered or not, size, skull type, dentition, oral health, overall health, and other health issues all need to be considered.

2. How did the fracture occur? Is the dentition involved? What is the periodontal health? Was the fracture pathologic? What is the bone health? Is there bone missing? Bilateral or unilateral?

3. What can the owner afford? What are the owner’s expectations? Do they comprehend their part in this multiple week process? Where will the pet recuperate? Are there other pets in the home? Will the pet be left alone?

This may be the most difficult. You need to be honest with yourself and your client. Are you qualified? What advanced training have you had in the various methods of fracture repair? How mandibular many fracture repairs have you done? What equipment do you have? What is your comfort level with all of the above?
Dental care for senior veterinary patients

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**Introduction**

To prevent senior and geriatric age pets from suffering with insidious dental and oral disease, preventive professional dental and home care during all life stages is the best medical recommendation. As immunosenescence begins and concurrent systemic and age related disease occurs, the once hidden dental disease may become more obvious, progressive, and require medical intervention to alleviate disease, infection, pain, and suffering. Age categories are arbitrary. However, based on AAHA senior care and life stage guidelines, a senior may be defined as a dog and cat that has lived 75% of a predicted life span. A geriatric patient may be defined at life expectancy and beyond (breed dependent). For cats, the last 25% of their life span (i.e., senior) may defined as 11-14 years of age and a geriatric 15+ years of age.

**Concurrent medical illness**

Anesthesia and dentistry treatment require individualized patient assessment, pre-anesthetic planning, and individualized anesthetic plans. A thorough medical history including all current medical illnesses, past medical illnesses, past dental treatments, and current medications is necessary. A complete physical examination and blood work (complete blood count, chemistry panel, urinalysis, and thyroid evaluation) are necessary for all aged patients. A preoperative blood pressure is recommended. Concurrent disease such as cardiovascular disease (e.g., heart murmurs, cardiac dysfunction), and renal disease are common co-morbidities. Disease such as diabetes mellitus, hyperthyroidism, hyperadrenocorticism, and hepatopathy may be identified. Concurrent morbidities do not preclude general anesthesia. However assessment with thoracic radiographs, echocardiography, and stabilization of concurrent medical co-morbidities, as necessary, should be recommended for management prior to treatment of chronic dental and oral disease. Anesthesia and dentistry treatment require individualized patient assessment, pre-anesthetic planning, and individualized anesthetic plans.
Oral and dental disease

A pain free and infection free oral cavity is a priority. The assessment and treatment requires general anesthesia and intraoral radiographs. The prevalence of periodontal disease increases with age. Periodontal stages (i.e., PD0-PD4) are assigned based on intraoral radiographs and periodontal probing. Oral tumors represent about 6-7% and 3% of all canine and feline tumors, respectively. Biopsy is required for diagnosis. The majority of feline and canine oral tumors occur in middle aged and older patients. The most common malignant canine oral tumors are malignant melanoma (30-40%), squamous cell carcinoma (20-30%), and fibrosarcoma (10-20% and male dogs predisposed). The most common feline tumors are squamous cell carcinoma (70-80%) and fibrosarcoma (13-17%). Odontogenic tumors (e.g., canine acanthomatous ameloblastoma, peripheral odontogenic fibroma) compromise 20-30% of oral tumors. Endodontic disease is commonly the result of fractured and concussive/luxation injuries of teeth. Fractured teeth, non-vital teeth, caries, developmental anomalies, concussed teeth, luxated teeth, and perio-endo lesions result in inflammation and infection of the endodontic system and periapical tissues. All fractured teeth with pulp exposure require endodontic treatment or extraction. Classification of tooth fractures can be found at www.avdc.org (nomenclature). Feline tooth resorption is a common and frustrating dental problem in the feline patient. The prevalence of the disease, in cats, has been reported in the literature as 20-75% and increases with age. The stage of lesions can be classified by the extent of tooth involvement. The lesions can be further divided into radiographic types for treatment planning. Recently, several publications have evaluated tooth resorption in dogs. Peralta, et al. (2010) identified increased frequency of tooth resorption in older and large-breed dogs with no sex predilection. Tooth resorption was detected in 53.6% of the dogs. Malocclusions are deviations from the normal occlusion. In some pets, malocclusions have resulted in unrecognized, chronic soft tissue, hard tissue and dental injuries that are not recognized until much later in life. Malocclusions are classified as symmetrical skeletal malocclusions (e.g., Class 1-3) and asymmetrical skeletal malocclusions.

Anesthetic considerations

Anticipated problems in the senior and geriatric patient include but are not limited to, co-morbidities, hypotension, hypothermia, and increased anxiety. Multimodal anesthesia and analgesia is necessary. The use of regional nerve blocks as part of the anesthesia plan is beneficial to decrease the requirements of inhalant anesthetics. Likewise, constant rate infusions of opioids, such as fentanyl, are commonly used to provided anesthesia and analgesia in senior and geriatric patients with concurrent morbidities so that the depth of anesthesia can be maintained with little impact on the cardiovascular system. Constant rate infusions with dopamine and dobutamine are utilized based on concurrent disease processes such as renal disease and cardiovascular disease to maintain blood pressure as indicated. Thermoregulation with both forced air convection warming devices and conductive polymer fabric heating are both used to maintain normothermia. A dedicated staff member is required to monitor anesthesia. Blood pressure monitoring (e.g., doppler, oscillometric), ECG, end-tidal carbon dioxide, and pulse oximetry are monitored in every patient. A ventilator is beneficial to help control tidal volume and rate of respiration so that anesthetic gas exchange is consistent.
The management of periodontal disease (PD) is fundamental to veterinary dental care and impacts on all the other aspects of dentistry. Veterinary practitioners are dealing with periodontal disease day in and day out as it affects all dog and cat mouths to a variable extent.

Best management is achieved by a marriage of owner’s homecare and professional treatment which need to be integrated and delivered in a realistic, attainable and affordable way.

Periodontal disease is a lifelong battle between plaque and the host - the battleground is the soft tissues (gingiva) in close proximity to mature plaque (at the base of the crown).

**Plaque.** Properties. A biofilm / Soft sticky paste / Tooth coloured / Cause of halitosis (bad breath) / Can’t be rinsed off / Disrupted by mechanical dislodgement – tooth brushing. Plaque becomes mature and also begins to convert to tartar after as little as 48 hours. Mature plaque has a different bacterial composition – a shift to more anaerobes due to a change in the local micro-environment.

**Calculus (Tartar)** is not the cause of PD. Its main role is as a plaque retentive surface which makes plaque control ineffective. There is NO correlation between the amount of calculus deposits and the severity / stage of PD.

**Gingivitis** = inflammation of the gingiva margin from mature plaque. It is not painful unless there is an ulcerative or desquamative component. The presence of gingivitis – inflammation or bleeding on touching – indicates that the periodontal tissues are under duress because of inadequate plaque control.

**Periodontitis (PD)** – inflammation and destruction of the attachment structures around the tooth. PD is preceded by chronic gingivitis. Simplistically the changes are then irreversible. The resultant change in anatomy favours plaque being harboured and so disease establishment. The attachment structures of the tooth are broken down including gum attachment loss, bone loss, attachment migration & periodontal pocket formation. The
periodontal pocket is a reservoir of infection with adjacent inflammation and ulceration. It has not been proved that uncontrolled periodontal disease is the cause of disease or problems elsewhere in the body. However there is a large amount of information to support an association between the multiple foci of inflammation and infection of PD and aggravation of disease at other sites especially when inflammation is a factor.

Periodontal status or prognosis can not be assessed by examination of a conscious animal and it should be remembered that a “Guestimate” is the best that can be achieved.

Tooth mobility (grade 3) and furcation exposure (grade 3) on 3 rooted teeth are indicators that treatment is required. Signs such as gingival recession, gingivitis and missing teeth give an impression but do not tell you the stage of PD. Being able to retrieve the dental chart(s) from previous times gives much valuable information on what may be expected as the current periodontal condition.

Once the patient is under GA, all the clinical periodontal parameters can be determined. The most important, by far, is the maximum (true) clinical periodontal pocket depth. This is combined with all the other factors and risk factors to enable a decision on each tooth’s prognosis and so treatment option. Examples of risk (or interplay) factors are Host Response (influenced by genetic predisposition & general health), ongoing Plaque control, age, chewing activity.

Clinical pocket depth relationship to prognosis

<table>
<thead>
<tr>
<th>Tooth Prognosis</th>
<th>Cat</th>
<th>Dog Miniature/Small</th>
<th>Dog Medium/Large</th>
</tr>
</thead>
<tbody>
<tr>
<td>GOOD</td>
<td>Up to 1</td>
<td>Up to 2</td>
<td>Up to 3</td>
</tr>
<tr>
<td>MEDIUM</td>
<td>1 to 2</td>
<td>2 to 4</td>
<td>3 to 5</td>
</tr>
<tr>
<td>POOR</td>
<td>Over 2</td>
<td>Over 4</td>
<td>Over 5</td>
</tr>
</tbody>
</table>

PD indicating extraction is when the prognosis is poor. Then deterioration is inevitable to a condition when there is harm to the patient. Tooth extraction before late stage disease not only avoids pain and potential knock-on effects (of infection and inflammation) but also major complications such as oro-nasal fistulae and fractured mandibles.

The following findings indicate extraction would be the best option:

- Mobility Grade 3 (>1 mm in any direction)
- True Pocket depth
  - > 5 mm (7 mm canine tooth) – medium dogs
  - > 4 mm (6 mm canine tooth) – small dogs
  - > 2 mm (3 or 4 mm canine tooth) – cats
- F3 furcation involvement on 3 rooted teeth

These would be moderated when other negative factors co-exist – usually leading to earlier extraction.

Periodontal staging classifications are very useful for research and other purposes but do not help in a clinical scenario as the stages do not correlate to prognosis and treatment requirements.
**Lifelong management (treatment)**

There should be a programme of care, from birth to death and not stand alone (single) treatments. PD can not be “cured”.

Treatments should be integrated with homecare. Treatment decisions are influenced by homecare being performed. Homecare is then followed up with periodic treatment as required.

The main aims of the professional periodontal treatment are:

1) To assess all teeth and extract any teeth before they get to the stage of causing harm.
2) To make ongoing homecare more effective and delay future progression of disease.

Dealing with plaque retention and stagnation factors is core to conservative treatments.

**Plaque retention factors** create a rough surface for plaque to latch on to as opposed to a smooth enamel surface. Calculus and exposed root cementum are the main retention factors which are dealt with by thorough scaling and root planing. Enamel defects or tooth fractures are other retention factors which are much more significant when close to or below the gum margin.

**Plaque stagnation factors** are those which harbour plaque so that it is not dislodged in function or more difficult to access in homecare. Periodontal pockets, gingival hyperplasia (causing false pocketing), gum recession (especially leading to furcation exposure), crowded teeth, teeth out of alignment and teeth without a functional opponent all result in increased plaque stagnation. Gingivectomy to eliminate false pocketing and selective extractions to relieve crowding are treatments to reduce plaque stagnation.

All scaling should be performed so as it does not scratch the tooth surface. Tooth polishing is mainly for cosmetic purpose (to remove extrinsic staining) and likely only delivers minor therapeutic benefits. However it has the potential to scour (roughen) the surface if a gritty paste (medium or coarse) is used. Harm due to frictional heat is an immense concern with polishing. Your technique should be checked by polishing a finger nail.

Antibiotics are never to be used as treatment for the periodontal disease itself.

**Homecare (daily plaque control).** Much more still needs to done so owners can deliver more effective homecare. Products and techniques should be tried and tested. Recommendations should be based on good evidence based support or sound science.

Information from a source with a vested financial interest (commercial bias) should be checked and viewed with a healthy degree of cynicism.
Seven unforgivable sins of veterinary dentistry... and how to avoid them

Barden Greefield

This lecture will address seven (7) common mistakes veterinary practitioners make regarding veterinary dentistry.

**Sin #1: Not Using or Underutilizing Dental Radiography in ALL Cases**

JAAHA: 2013 AAHA Dental Care Guidelines for Dogs and Cats* Holmstrom SE. et al.
Radiograph the entire mouth, using either intraoral or digital radiographic systems. Radiographs are necessary for accurate evaluation and diagnosis.

Studied 226 dogs and 115 cats.

*Value of radiographs w/no clinical findings present.*
- Incidental radiographic findings: Dog (41.7%), Cat (4.8%).
- Clinically important findings: Dog (27.8%), Cat (41.7%).
- Radiographs of no value: Dog (30.5%), Cat (53.6%).

*Value of radiographs with clinical findings present.*
- Conformational only: Dog (24.3%), Cat (13.9%).
**Additional findings:** Dog (50.0%), Cat (53.9%).
- Clinical essential findings: Dog (22.6%), Cat (32.2%).
- No value: Dog (3.1%), Cat (0%).

Of 8308 teeth evaluated and abnormal radiographic findings were found in 2458 (29.6%). Findings that were only detected on radiographs, which were not noted on routine oral exam, were more common with older dogs. Full-mouth radiographic evaluation should be performed to obtain important information for making accurate diagnoses.
Sin #2: Not treating discolored and fractured teeth.

Over 93% of all intrinsically stained teeth have irreversible pulpitis (Hale FA. Localized intrinsic staining of teeth due to pulpitis and pulp necrosis in dogs. J Vet Dent 18[1]:14-20 2001 Mar).

Leaving a discolored tooth in the mouth IS NOT an option: Canines, maxillary lateral incisors, maxillary 4th premolars and mandibular 1st molars should be referred for RCT as these are more strategic in nature.

Complicated crown (and crown/root) fractures: (pulp exposure) teeth must have a root canal therapy or extraction.

Leaving a complicated crown fracture in the mouth is like leaving a broken leg untreated. **It is not an option and we as veterinarians must change our bad habits.**

Uncomplicated crown (and crown/root) fractures: (dentin exposure without pulp exposure) need dental radiographs. If 3rd (tertiary) dentin in place and radiograph of tooth normal, then odontoplasty is recommended to smooth rough enamel edges. Dentin bonding is an option for those fractures with no 3rd dentin. Radiograph of tooth shows wide pulp chamber compared to contralateral tooth or has apical pathology, and then root canal therapy or extraction is needed.

Sin #3: Incomplete extractions.

This is especially true for cats with gingivostomatitis. Leaving a tooth remnant in the mouth will not resolve the severe stomatitis and gingivitis and can make things worse.

Pulverization can lead to perforation into the mandibular canal, infraorbital canal, or nasal passages. Pulverization can propulse tooth fragments into the above mentioned canals. Bone necrosis, air embolism, and sublingual/subcutaneous emphysema have been reported with this technique.

This is also true with Tooth Resorptions (TR)

Type I TR
Peridontal ligament is still present.
Usually more diffuse gingivitis present.
Entire tooth must be removed.

Type II TR
Periodontal ligament is not visible and the tooth being replaced by alveolar bone.
Crown amputation is an option for these cases provided you have radiographic evidence to support this.
More localized vs. Type I.

Sin #4: Leaving extraction sites open or not performing a tension-free full coverage flap

An extraction is a surgical procedure. The gingiva should lie flat against the alveolar bone in a tension-free manor. Suturing helps speed extraction site healing, prevent infection,
and reduce post operative pain. Exposed bone can cause pain and leads to delayed wound healing. Sufficient gingiva is elevated thus allowing a tension-free suturing. With tension, the suture will fail and there would be wound dehiscence.

This is especially true for oronasal fistula repair (tooth no longer present) or is evident upon dental probing into the nasal sinus. Failure to remove the downgrowthed epithelialized tissue as well as provide a tension free coverage will lead to wound closure dehiscence.

**Sin #5: Ignoring malocclusions.**

The veterinary clinician has a benefit that most dental specialists do not have in that they see many puppies and kittens as post purchase exams or to see them for their juvenile immunization and examination series. This is a tremendous opportunity to identify the variety of malocclusions that can lead to attrition (tooth on tooth contact), palatal trauma, or mandibular mucosal trauma. Early identification and treatment of the variety of malocclusions can eliminate unnecessary oral pain and discomfort for the life of the pet.

The following are the types of malocclusions and the treatment plan recommended.

*Type I Malocclusion*
- Rostral and caudal crossbites.
- Based-narrow mandibular canine teeth.
- Mesioverted (lanced) canine teeth.

*Type I Treatment*
- Based-narrow canines – acrylic incline plane; crown reduction and vital pulpotomy, extraction of canines (not recommended).
- Mesioverted canines – orthodontic appliance or extraction.
- Rostral crossbite – maxillary expansion device, maxillary arch bar (if no attrition, no tx).

*Type II Malocclusion*
- Mandibular brachnathism.
- Maxillary prognathism.

*Type II Treatment*
- Crown reduction with vital pulpotomy/root canal therapy of mandibular canines.
- Extraction of mandibular incisors.
- Extraction of mandibular canines (not recommended).

*Type III Malocclusion*
- Mandibular prognathism.
- Maxillary brachynathism.

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**Type III Treatment**

Assess attrition (tooth on tooth contact) of mandibular canine tooth and maxillary lateral incisor tooth – Lateral incisor may need to be extracted.
Crown reduction or extraction of maxillary incisor teeth.\(^{1,2,3,4}\)

**Sin #6: Using the antibiotic “Wheel of Misfortune” in periodontal and endodontic disease**

Antibiotics will NOT treat periodontal disease or endodontic disease alone. Antibacterials can cause variable degrees of clinical improvement (none to substantial) during treatment but after cessation of medication, immediate recurrence of clinical signs recur. Without the mechanical removal of the inflamed tissue (periodontal surgery, exodontia, or endodontic therapy), the pathology will return. Antibiotics should never be used as a monotherapy with oral infections or used as preventive management of oral conditions (pulse therapy).\(^5\)

Doxycycline can be used daily in sub-antimicrobial levels for chronic periodontal disease after initial therapy has been performed and is monitored.\(^6\)

Anti-inflammatory properties without causing antibiotic resistance.
Anti-collagenase activity.
Clindamycin injectable + oral suspension or capsules (Antirobe®).
Spectrum against many anaerobic periodontal pathogens. Penetrates bone, walled off abscesses and white blood cells. As PD involves bone, this is a good choice. However, usage in a pulse-therapy regimen is not supported by any published research data.
Amoxicillin tryhydrate + clavulanate potassium (Clavamox®).
This antibiotic has more systemic protection. In Vitro studies in the JVD mentioned that amoxicillin + clavulanate may have more in vitro efficacy than clindamycin against many oral pathogens.
Metronidazole (Flagyl).
Excellent activity against oral anaerobes. Excellent absorption. High dosages can lead to CNS disease.

**Sin #7: Inadequate or incomplete pain management.**

- Pre-operative, intra-operative, and post-operative management is needed.
- Pre-operative management.
  NSAID, Opioids.

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5 American Veterinary Dental College. American Veterinary Dental College- position statement; the use of antibiotics in veterinary dentistry. Available at http://www.avdc.org/position-statements.html#AB.
- Intra-operative management.
  - Opioids, regional nerve blocks, CRI (MLK, LK, HLK, K).
- Post-operative management.
  - Opioids, NSAID, Opioid-like drugs.
Surgical extraction of mandibular and maxillary canines

Paul Theuns

Reasons for extraction

Reasons can be:
- Infection with or without an abscess. Resorption. Trauma that has caused luxation, subluxations of fracture.
- Owners that are unable or unwilling to have a root canal treatment done is a frequent reason for extraction.
- Finally, linguoversion of the mandibular canines, causing damage and possibly an oronasal fistulas can also be a reason for extraction.

Anatomy

The apex of the canines can reach up to the second premolar in the dog.
In the cat the canines are less curved and have a more blunt tip. They tend to have a more vertical direction toward the apex.

Open or closed extraction

Extracting the mandibular and maxillary canine with a simple closed extraction can be tedious. Personally I only extract the canines with a closed extraction if the canines are mobile because of periodontitis and avulsion or luxation.
Under most other circumstances open extraction is preferred. In all instances x-rays are mandatory.

Surgical extraction

Scalpels
The size 15 and 15C or eventually the size 11 are the preferred scalpels.
Flaps

There are 3 types of flaps:
- the envelope flap.
- the pecicle flap.
- the triangular flap.

The envelope flap is created by making a sulcular incision and no vertical incisions. The major benefit of this procedure is the much lower risk of dehiscence compared to the other flap techniques. For the extraction of the canines this flap type will generally be insufficient.

The triangle flap is created by adding a single vertical incision to the sulcular incision that was made for the envelope flap.

The pedicle flap is made by adding a second vertical releasing incision. The two vertical incisions have to be slightly diverging.

To prevent the suture line from resting on the opened alveolus the flaps should be of sufficient width.

After the releasing incisions are made, the flap is further released by the use of a periosteal elevator. The periosteal elevator is used to bluntly dissect the underlaying tissue.

Closing: after the procedure the flap needs to be closed tension free with interrupted sutures 2-3 mm apart.

Maxillary canine tooth

A pedicle or a triangle flap can be used (extraction with an envelope flap can be tedious). For the pedicle flap the extension of the canine until the second premolar needs to be taken into consideration. For this reason the caudal vertical incision could be made starting on the distal line angle of the second premolar.

A suture from the flap attached to the skin can be used to keep the flap out of the surgery field to prevent damage.

The buccal bone is removed with for instance a round diamond. The amount of bone that needs to be removed varies depending on the pathology present. For an ankylosed tooth one needs to remove more bone than for a tooth with periodontal infection.

The luxator can be used to sever the periodontal ligament.

The extraction forceps can be slightly rotated to put tension on the periodontal ligament and severe the blood vessels, this will facilitate the extraction process.

Ideally the surgeon should be able to remove the tooth with 2 fingers without using an extraction forceps.

It should always be prevented to make any movement to the medial side of the canine root by tipping the tip of the tooth in a buccal direction. The luxator should never be put in between the alveolar bone on the palatinal side and the tooth to prevent creating an oro nasal fistel.

After the extraction an alveoloplasty should be performed to prevent any sharp edges.

Mandibular canine tooth

There are different approaches to the mandibular canine tooth.

The tooth can be approached buccal, lingual or by a combination of the 2.
For the buccal and the lingual approach triangular flaps are made. For the combined approach both a buccal and a lingual flap are made.

The lingual approach has the advantage that the the apex is situated more on the lingual side, so it can be reached more easily.

The structures to be taken into consideration are the lip frenulum and the neurovascular structures from the mental foramen.

For closure of the flap after extraction the same points as mentioned above need to be dealt with.

**Complications**

*The most important complications are:*

- Creation of an oro nasal communication after a maxillary canine extraction.
- Fracture of the rostral mandibula after a mandibulary extraction.
Eosinophilic granuloma complex

The true etiology of these conditions is unknown; however a local accumulation of eosinophils is thought to initiate the inflammation and necrosis. The accumulation may result from a local (food) or systemic allergies; although these lesions have been seen in cases where allergic disease has been ruled out. Additional causes include a response to irritation, such as chronic grooming or traumatic malocclusion. There may also be a genetic predisposition.

Indolent Ulcers are the most common oral manifestation, and they will present as brownish-red lesions on the upper lip or around the maxillary canine teeth.

Linear granulomas can be single or multiple; the most common sites are the lips, gingiva, palate and tongue. They are generally non-painful, but can become secondarily infected. The typical presentation is a raised, lobulated yellow-pink mass; however, they can also appear ulcerative causing severe damage to the oral mucosa and underlying bone. This may lead to severe periodontal loss, pathologic fractures, or oronasal fistulas.

Histopathology should be performed to confirm the diagnosis. Following confirmation of the diagnosis, a thorough allergy evaluation should be conducted including food trial, flea treatment, +/- allergy testing.

The acute disease process is best treated with systemic corticosteroids; however corticosteroids should NOT be used for long term disease control due to the significant systemic side effects. The typical initial protocol is prednisone 2 mg/kg q 12 hours for 3-4 weeks. Additional options include intralesional triamcinalone (3 mg weekly) or methyl prednisolone injections. Antibiotic therapy is required occasionally to induce remission and/or treat secondary infection. There are also cases that appear to respond to antibiotic therapy alone. Therefore, we initially treat mild cases with antibiotics alone and more severe cases with a combination of antibiotics and corticosteroids.

Many cases remain idiopathic, requiring lifelong therapy; options for this include antibiotics and cyclosporine. Fewer side effects may be expected with cyclosporine in comparison to steroids. However, there are reports of opportunistic fungal and fatal protozoal infections associated with its chronic use. Use the lowest effective dose, and perform regular therapeutic
levels and routine blood testing.

**Caudal stomatitis**

This is another relatively recent disease process in cats that is frustrating us at present. The best description is a severe immune mediated reaction to dental tissues, but we really don’t know. Some feel that this may actually be a group of disease processes that look the same clinically which is why they can be very frustrating to treat.

The history will generally include anorexia, drooling, gagging, and pain during mastication. Physical exam will typically include a thin pet with unkempt fur. The oral exam will reveal severe stomatitis usually over all teeth. The inflammation will most commonly be worse on cheek teeth than canines and incisors. However, faucitis is the key clinical finding. Severe hyperplastic inflammation to the gingiva can result from periodontal disease, however faucitis will not be present.

A pre-operative blood panel will generally show a marked elevation in globulins (Polyclonal gammopathy) and total protein.

Histopathology is recommended but not required. There have been a few cases with the classic look that were created by another pathology (fungal, Pemphigus). In this case full mouth extractions would be ineffective.

Recently bartonella has been implicated as a possible cause of stomatitis. This is due to the high incidence of bartonella in the domestic feline population. Stomatitis is one clinical sign of bartonella infection; however it is not a typical cause. The other major sign is lymphadenopathy. If you see severe lymphadenopathy with stomatitis, consider testing prior to therapy. Most veterinary dentists do not really think that this is the cause of the vast majority of cases. Treatment is zithromax for 21 consecutive days. In multi-cat households the patient must be isolated or all patients treated. The results are questionable at present and therapy is pricey.

**Medical Therapy:** Most medical therapies will work for a while, however in general resistance will start within a year or less. In addition, most therapies have side effects worse than the disease process in and of itself. In general, medical therapy is very frustrating to the practitioner and client.

Corticosteroids are the mainstay of most medical therapy today. It is generally very effective at first and is relatively inexpensive for the client. In my experience, injectable (depomedrol 10 mg IM) is much more effective than oral preparations in my experience. However, they will typically loose effectiveness after a year or so requiring higher and higher doses at shorter increments. This generally results in significant deleterious effects. About 10% of stomatitis cases we treat are already diabetic!!!

Antibiotics are safer than steroids but much less effective, especially in long term therapy. They are generally disappointing in their success. Metronidazole and clindamycin are the mainstays of therapy; however Clavamox and amoxicillin can be used as well. Metronidazole may be the antibiotic of choice due to its anti-inflammatory effect.

Other immune suppressive such as Imuran, Cytoxan, Gold Salts, Cyclosporine have been used. However, they are all very expensive with numerous adverse side effects (mylosuppression). Cyclosporine is currently the most commonly prescribed immune modulatory drug (other than steroids) for this disease process. However, its chronic use is very expensive and has been implicated in severe fungal and protozoal infections. Starting
dose is 5-10 mg/kg. You need to dose for a trough level of about 500 ng/ml on regular basis. In most dentists opinion it is only really effective AFTER teeth are removed. However, it has shown promise in resistant cases.

Laser therapy is not proven at all, most clients and RDVM’s are very unhappy with the long term results. It is very expensive and short term relief only.

**Surgical Therapy:** Extraction is currently the ONLY effective long term treatment for this disease process in cats. In our experience, the sooner this is done, the better that cats do both post-operatively as well as long term.

For extractions to be successful, the teeth must be COMPLETELY removed. Therefore post-operative radiographic confirmation of complete extraction of the tooth roots is recommended. Following the insurance of complete removal of the teeth, perform aveloplasty to remove the periodontal ligament and smooth rough bony edges. This is typically performed do this with a rough diamond bur.

Studies report a 60% success rate when all teeth caudal to the canines are extracted, however our experience has not been as good. However, whole mouth extractions have a success rate of approximately 90-95% for clinical remission. Slight faucitis may remain, but pets are comfortable. In addition, the rare cases that don’t completely respond are generally much more responsive to medical therapy.

If there is NO inflammation to the canines or incisors (which is rare), then the owner is given the option of leaving the canines. However, if these are inflamed, all teeth should be extracted. If the teeth are ankylosed, complete root pulverization may be necessary.

60-70% of the strength for the rostral mandible is contained in the canine tooth roots. Extracting both mandibular canines at the same time has resulted in jaw fracture on occasion. Therefore, consider extracting all but 2 ipsilateral canines on the first visit. Often this will be sufficient and cats will never have the last two teeth extracted.

Why leave a maxillary canine if the goal is to decrease the inflammation? This is because about 1/3 of cats who have maxillary canines extracted surgically will develop lip trauma from the mandibular canine. In many instances, this has necessitated further therapy in a cat where the stomatitis was resolved.

**Feline juvenile (puberty) gingivitis/periodontitis**

**Definition:** Juvenile periodontal disease is inflammation which occurs soon after permanent tooth eruption. This syndrome can be described in two categories, feline hyperplastic gingivitis and juvenile onset periodontitis.

**Etiology:** The etiology of this condition is unknown. However, in humans there is a period of increased susceptibility to gingivitis during the pubertal period. A genetic predisposition towards feline juvenile onset periodontitis has been reported in Siamese, Somali, and Maine Coon cats.

**Clinical Features:** Hyperplastic gingivitis appears as gingival enlargement and significant inflammation which is confined to the gingiva and begins during the eruptive period of the permanent dentition. Bleeding during mastication and on oral exam are common findings. While occasionally seen in dogs, this condition has a much higher incidence in cats. It is generally a non-painful condition for the patient, and halitosis is a common complaint. If left untreated, it typically proceeds quickly to periodontal disease, which may result in early exfoliation of the teeth. This disease is commonly mistaken for caudal stomatitis. The
A distinguishing clinical sign is the lack of caudal inflammation in this disease process. As the patient matures, susceptibility appears to subside at approximately two years of age.

In contrast, juvenile periodontitis does not involve enlargement of the gingiva and usually leads to the rapid proliferation of plaque and calculus and subsequent inflammation. This in turn results in significant early bone loss, periodontal pocket formation, and furcation exposure. This is generally the worst in around the mandibular first molars. Treatment and effective management of these cases is often exceedingly difficult.

**Diagnostics:** Histopathology (via incisional biopsy) should be considered to rule out other causes of gingival inflammation. Culture and sensitivity testing is generally unrewarding, but may be of value in non-responsive cases. Dental radiographs should be performed to evaluate the quality of the alveolar bone and also for early tooth resorption. Finally, Bartonella testing may be beneficial in some cases, especially in patients who do not respond to traditional management practices.

**Management:** In the management of both of these conditions, early (9 months of age) and frequent (q 6-9 months) dental prophylaxis (even if only minimal plaque is present) along with strict homecare is critical to decrease inflammation. Ideally, homecare consists of daily brushing, as it is the gold standard of plaque control. Other homecare alternatives include chlorhexadine rinses as well as plaque control diets and treats. In cases where gingival hyperplasia is present, early gingivectomy is recommended to remove pseudopockets, decrease inflammation, and facilitate plaque control (both professional and homecare). Finally, extraction of any significantly diseased teeth is warranted to decrease the degree of inflammation.

**Oral neoplasia**

The oral cavity is the fourth most common place to find cancer in cats. It is reported that 7.4% of all feline tumors are located in the oropharynx with 89% being malignant. By far the most common form of feline oral cancer is squamous cell carcinoma (SCC), which make up approximately 60% of all feline oral tumors. However, obviously other types of neoplasia
are possible. While benign tumors are rare, they are still seen and can appear to be more aggressive tumors. In addition, aggressive tumors look like benign lesions early in the course of disease. Finally, non-neoplastic process (e.g. eosinophilic granuloma complex) can mimic aggressive tumors. Therefore dental radiographs and histopathology are always indicated. This chapter will focus on SCC as it is the most common with fibrosarcoma (FSA) a distant second. FSA is so similar in appearance, treatment, and prognosis that separate discussion is not warranted. Finally, a brief discussion of benign feline oral growths will be presented at the end.

**Squamous Cell Carcinoma (SCC)**

The most common feline oral tumor is squamous cell carcinoma (SCC). The most common locations are the premolar/molar regions (maxillary > mandibular). In addition, it can be seen sublingually. It is highly locally aggressive, however rarely metastasizes.

**Etiology:** A true etiology is not known in most cases of this as well as other types of oral neoplasia. However, the inflammation associated with periodontal disease has been linked to oral cancers. There is an increased incidence of SCC in cats infected with feline immuno deficiency virus (FIV), but no link has been established. In addition, some investigators suggest that an increased incidence may be seen with certain flea control products, diet, and environmental tobacco smoke.

**Clinical appearance:** SCC is typically seen in older cats (11-13 years is average age at diagnosis), but has been reported in cats under one year of age. This tumor can appear as an inflammatory soft tissue growth or an area of bony destruction. In addition, it can
present as a bony swelling often with facial asymmetry. Cats with SCC may also be presented for anorexia or ptyalism. Finally, in cases of maxillary SCC the initial clinical sign may be nasal or ocular discharge or exophthalmos. The ocular discharge is caused by the neoplastic obstruction of the nasolacrimal duct. The third eyelid will also be elevated in some cases.

The growth type is typically red and friable fleshy mass which bleeds easily. This type is fairly commonly seen on oral exam and often causes anorexia as it grows. The destructive type will invade bone and result in mobile teeth in an otherwise healthy mouth. Finally, the bony destructive type may present as a swelling on the jaw.

If the lesion starts under the soft tissues and is more bone involved, it is commonly mistaken for dental disease. This is due to the fact that cases of bony destruction create mobile teeth which mimic periodontal disease. Further, proliferative cases result in swelling that is often diagnosed as tooth root abscesses. Finally, upon presentation to referral clinics, a non-healing extraction site is often the complaint.

Finally, sublingual SCC is not uncommon. Clinically this will present as inhibition of tongue movement. The tongue may appear swollen and often feels “firm”. However, early cases may be missed which highlights the importance of a complete oral exam, especially in cases of anorexia.

**Diagnosis:** Since there are numerous benign lesions which can appear neoplastic and on occasion neoplasia (especially early) can appear benign, diagnostic testing is always recommended prior to definitive treatment.

The first step of diagnosis following physical exam is dental radiology. Note that even edentulous arcades can have retained roots which can be infected causing the swelling and therefore radiographs are still indicated. The classic radiographic sign of an abscessed tooth is periapical rarefaction. If this is seen, a tentative diagnosis of a tooth root abscess can be made. However, it does not confirm that it is the cause of the swelling. Periodontal loss is seen as complete loss of the alveolar bone with fairly regular edges. This is important when discerning periodontal disease from neoplasia as the cause of mobility as it is common for cases of oral neoplasia to be presented following dental therapy (extractions) in the area. The other clues are the periodontally diseased teeth should have calculus covered roots (as opposed to neoplasia where the roots will usually be clean) and that in neoplasia the other arcades are relatively healthy with mobile teeth on one quadrant.

Radiographic appearance of malignant feline tumors consists of mottled bone lysis, and in some areas new bone formation. Initially, the bone will have a mottled ‘moth eaten’ appearance, but radiographs late in the disease course will reveal a complete loss of bone in the area. At this time, the teeth will appear to float in space. If the cortex is involved, an irregular periosteal reaction will be seen. In addition, the edges of the tumor will also appear to be ragged, as opposed to smooth contours of cysts and most periodontally induced bone loss. In advanced cases, the root apices may develop a spiked appearance. The extent of tumor seen radiographically is always greater than on clinical examination.

As important as dental radiographs are, they cannot be fully relied upon for a definitive diagnosis. In general, fine needle aspirates (FNAs) are unrewarding, but occasionally it can tell between inflammation and neoplasia. By far the best diagnostic tool is surgical biopsy and histopathology. When harvesting a sample for testing, always ensure that a sufficient sample is taken to ensure an accurate reading. Superficial samples may return erroneous inflammatory diagnoses.

After a histopathologic diagnosis is achieved, staging consisting of:
a) Measuring the size of tumor clinically and radiographically
b) Determining lymph node involvement (palpation, aspirate, excision and biopsy)
c) Distant metastasis (chest radiographs, +/- abdominal ultrasound, CT?)

*Treatment:* Feline SCC is a very difficult condition to treat with overall reported survival times of anywhere from 60 days to 6 months. However, mandibular cases treated with a mandibulectomy had a significantly increased survival time with a 43% survival rate at 2 years.

The ideal therapy is surgical removal with 2 cm margins. Unfortunately, by the time of diagnosis, the tumor is generally inoperable. The one area that carries a decent prognosis is very early lesions on the rostral mandible. In this location, margins may be obtainable, giving the superior numbers above.

Radiation therapy, while somewhat effective in dogs, has minimal to no benefit on increasing survival in cats. Radiation therapy (especially stereotactic) may have a palliative effect in increasing quality of life. However this is not universally reported and most radiation oncologists do not feel it is justified at this point in time. There may some benefit to combining surgery and radiation therapy. One such study found an 11 month disease free interval in mandibular cases. However, this study must be viewed in light of the fact that mandibular SCC has a better prognosis than other forms with surgery alone. Finally, radiation therapy combined with mitoxanthrone gave median remission time of 170 days. However, there were often significant side effects with this treatment. Finally, some studies support gemcitabine as a radiation sensitizer, which may be beneficial in the future. Although other studies have found that this product has significant negative side effects.

As far as medical management is concerned, this has at best a palliative affect. Non-steroidal anti-inflammatories (NSAIDs) have shown some improvement in quality of life and possibly survival time.

Feline SCC is an area of fairly intense research and therefore, we may have better success in the future. This may relate to current research on the various chemical markers and molecular variables seen with this disease as well as other neoplasias (especially head and neck). The practitioner is urged to keep up with current research and collaborate with veterinary dentists or oncologists in treating this disease.
Feline extraction techniques

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Introduction

Dental extractions are a very common surgical procedure, typically performed daily in most veterinary practices, yet they are not a simple undertaking. As such, they should be approached with the same level of preparation as for any other surgical procedure.

A guideline for proper and successful dental extractions is summarized in the following 10 steps. These steps constitute the technique for a single rooted tooth, but also for multirooted teeth which are treated the same way following sectioning, and for large teeth following bone removal.

Obtain Consent

Never extract a tooth without prior owner consent, no matter how obvious it is that extraction is the only option. Consent should preferably be written, but is acceptable verbally via a phone call. Be sure to have valid daytime numbers for the client and inform them they must be available during surgery hours. If the client cannot be reached and prior consent was not obtained, do not pull the tooth.

Pre-operative radiograph

Dental radiographs should be made of all teeth prior to commencing the extraction. Dental radiographs are invaluable resources for guiding the practitioner through the extraction process. Radiographs allow the practitioner to determine the amount of disease present, any root abnormalities or resorption/ankyloses. Approximately 10% of feline maxillary third premolars have a third root. Dentoalveolar ankylosis makes extraction by traditional elevation practically impossible. For this reason, crown amputation and intentional root retention is acceptable for advanced Type 2 feline tooth resorption, but it is important to make this distinction prior to treatment decisions. In summary, dental radiographs provide critical information for treatment planning and the successful outcome of dental extraction procedures. In addition, radiographs provide solid evidence in the medical record.
Pain management

Extractions are surgical procedures which are moderately to severely painful. A multimodal approach typically provides superior analgesia and safety.

Cut the gingival attachment

The gingival attachment can be cut with a needle or small elevator/luxating elevator. The selected instrument is placed into the gingival sulcus with the tip of the blade angled toward the tooth. The blade is then advanced apically to the level of the alveolar bone, and carefully continued around the entire tooth circumference. This step is very helpful in performing extractions as the gingival attachment contributes approximately 15% of the retentive strength of the periodontal apparatus. More importantly however, this step will keep the gingiva from tearing during the extraction procedure.

Elevation

Elevation is the most dangerous step in the extraction procedure. Elevators are sharp surgical instruments which are used in an area of numerous critical and delicate structures. In order to avoid causing iatrogenic trauma in the event of instrument slippage or upon encountering diseased bone, the index finger is placed near the tip of the instrument.

It is important to select an instrument which matches the curvature and size of the root. In general “go small”, as this will result in less pressure and damage being created. Elevators larger than 3-mm are rarely indicated; generally sizes 1-2 mm are most effective.

Elevation is initiated by inserting the instrument firmly yet gently into the periodontal ligament space (between the tooth and the alveolar bone). The insertion should be performed while keeping the instrument at a 10 - 20 degree angle toward the tooth, to avoid slippage.

Once in the space between the bone and the tooth, the instrument is gently twisted with two-finger pressure. Hold the position for 10-30 seconds to fatigue and tear the periodontal ligament. The tooth should move at least slightly during elevation. If the tooth does not move, no damage is being done to the periodontal ligament.

The periodontal ligament is very effective in resisting short, intense forces. It is only by the exertion of prolonged force (i.e. 10-30 seconds) that the ligament will become weakened. Increased pressure will transfer much of the force to the alveolar bone and tooth which can result in the fracture of one of these structures. Therefore, it is important to moderate the force.

After holding for 10 - 30 seconds, reposition the instrument approximately 1/8 of the way around the tooth and repeat the above step.

Another elevation technique is the wheel and axel. To perform it in this fashion, place the instrument perpendicularly under a ridge of tooth that is at (approximately) the level of the alveolar bone and gently twist. If a natural ledge is not present, one can be created with a dental bur.

The key to successful elevation is PATIENCE. Only by slow, consistent elevation will the root loosen without breaking. It is always easier to extract an intact root than to remove fractured root tips.

If the elevation is not resulting in tooth mobility in a fairly short period of time, there is a problem. This may be due to faulty extraction technique, or more likely an area of dentoalveolar ankylosis. Review your technique and make sure the elevator is between the tooth and the bone. If so, review the dental radiographs for signs of ankylosis. If ankylosis
is present, a surgical approach should be employed. Moreover, if the extraction is not going well, a surgical approach is always available.

**Extraction**

Removing the tooth should only be attempted after the tooth is very loose. This is accomplished by grasping the tooth with the extraction forceps and gently pulling the tooth from the socket. Do NOT apply undue pressure as this may result in root fracture. In many cases, especially with premolars, the roots are round in shape and will respond favorably to gentle twisting and holding of the tooth while applying traction. This should not be performed if there are root abnormalities on the pre-operative radiograph.

**Alveoloplasty**

This step is performed to remove diseased tissue or bone, or any rough bony edges that could irritate the gingiva and delay healing. Diseased tissue can be removed by hand with a curette. Bone removal and smoothing is best performed with a coarse diamond bur on a water-cooled high-speed air driven hand-piece. Next, the alveolus should be gently flushed with a physiologic solution to decrease bacterial contamination.

**Post-operative dental radiograph**

Dental radiographs should be exposed post-extraction to document complete removal of the tooth. Retained roots are a very common complication associated with dental extractions. In fact, a recent study reported 92.8% of extracted carnassial teeth in cats have retained roots.

A retained root tip may become infected, or more commonly act as a foreign body creating significant inflammation. There are rarely any clinical signs observed with this complication, but occasionally retained roots do create an abscess.

**Closure**

Closure of the extraction site promotes hemostasis and improves post-operative comfort and aesthetics. It is always indicated in cases of larger teeth, or any time that a gingival flap is utilized. This is best accomplished with size 4/0 to 5/0 absorbable sutures on a reverse cutting needle. Closure is performed with a simple interrupted pattern, placing sutures 2 to 3-mm apart. It is best to utilize one additional throw over manufacturer’s recommendations in order to counteract tongue action.

In regards to flap closure, there are several key points associated with successful healing. The first and most important is there must be no tension on the incision line. If there is any tension on the suture line, it will not heal. Tension can be removed by fenestrating the periosteum and/or extending the gingival incision along the arcade creating an envelope flap or by making vertical releasing incisions creating a full flap.

**Multi-rooted teeth**

All multi-rooted teeth should be sectioned into single rooted pieces. The best tool for sectioning teeth is a bur on a high-speed air driven hand piece. In
addition to being the most efficient tool, it also has air and water coolant to help avoid overheating the tooth and bone. Many different styles of burs are available. This author prefers a cross-cut taper fissure bur (# 699).

When sectioning teeth, start at the furcation and work toward the crown. This method is used for two major reasons. It prevents the possibility of missing the furcation and cutting down into a root and avoids cutting through the tooth and inadvertently damaging the gingiva or alveolar bone.

After the tooth has been properly sectioned, follow the previous outline of steps for each single rooted piece. In some cases, the individual tooth pieces can be carefully elevated against each other to gain purchase.

Surgical extractions

Difficult extractions are best performed via a surgical approach. This is typically the canine and carnassial teeth, but is also beneficial for teeth with root malformations or pathology and for retained roots.

Surgical extractions are initiated by creating a gingival flap. Two options include a horizontal flap made with an incision along the arcade to create an envelope flap or alternatively making vertical releasing incisions to create a full flap.

Following flap elevation, buccal bone can be removed. This author favors a cross cut taper fissure bur for this step. The preferred amount of buccal bone removal is controversial, with some dentists removing the entire buccal covering. This author prefers to maintain as much buccal bone as possible, starting with removal of an amount equal to 1/3 the root length of the subject tooth on the mandible or 1/2 the root length for maxillary teeth. If this does not allow for extraction after an appropriate amount of time, more can be removed. If ankylosis is present, a significant amount of bone removal may be required.

Following bone removal, multirooted teeth should be sectioned, and then removing each piece as described in the steps previously outlined. After the roots are removed and radiographic proof is obtained, the alveolar bone should be smoothed before closure. Flap closure is initiated with an important step called “fenestrating the periosteum”. Fenestration can be performed with a scalpel blade, but a LaGrange scissor allows superior control. After adequate fenestration, the mucogingival flap should stay in desired position without sutures. If the flap does not remain in the desired position, tension is still present and further release is necessary prior to closure. Once adequate release is accomplished, the flap is sutured closed.

Maxillary fourth premolar

Extraction of this tooth requires a gingival flap. Classically, this is done with a full flap with one or two vertical releasing incisions, however this author finds envelope flaps sufficient for cats.

Envelope flaps are created by incising the interdental tissue between the tooth and the adjacent teeth. The flap is then carefully elevated along the arcade.

Following flap creation, buccal bone is removed to a point approximately ½ the length of the root. Next, the tooth is sectioned, separating mesial roots from the distal root by starting at the furcation and cutting coronally. The two mesial roots are then separated by sectioning in the small depression between the palatal and buccal roots. Complete sectioning can be confirmed by placing an elevator between the crown sections and twisting gently.

Following these steps, extraction proceeds as outlined above for single root pieces.
Mandibular first molar

The extraction procedure for the mandibular first molar generally requires a gingival flap. This author finds that an envelope flap is sufficient in virtually all cases.

The extraction of feline mandibular first molars is less complicated than in dogs as the roots are much shorter. An important point for successful extraction of these teeth in cats is the discrepancy in root size. The distal root is very small, about 1/3 the size of the mesial root. Therefore, the sectioning cut should be made about 2/3 of the way distally on the tooth. Creating a small gingival flap in order to start the sectioning at the furcation ensures the position and angle of the cut will always be correct. Furthermore, using small instruments in a very gentle manner will help to avoid fracturing this fragile root.

Maxillary canine

Maxillary canines are very challenging extractions due to the significant length of the tooth root. Furthermore, the plate of bone between the tooth root and the nasal cavity is very thin, creating a high risk of iatrogenic oronasal fistulas.

Mucogingival flaps with vertical incisions are usually necessary for exposure as well as closure. At minimum, a slightly distally divergent distal incision should be performed, but adding a mesial incision creates more tissue available for closure. The major issue with extracting the maxillary canine in cats is lip catching with the mandibular canine. In this authors experience it occurs in approximately 50% of feline maxillary canine extractions. In addition, it is more common when a mesial incision is created.

The releasing incision is typically made at the mesial line angle of the second premolar. Classically, the mesial incision was made at the mesial line angle of the canine tooth or distal line angle of the third incisor. However, in this author’s opinion, the mesial line angle of the canine tooth does not allow sufficient exposure and there is no reason to risk damaging the third incisor and increasing surgical trauma. Therefore, if a mesial incision is performed, it should be made in the diastema between the canine and third incisor. After the incision is made, the interdental gingiva between the canine and second premolar is incised to the bone.

After the incisions are completed, the flap is carefully elevated. The attached gingiva over the maxillary canine in cats is very friable and strongly attached to the underlying bone. If the flap cannot be elevated fairly easily, the interdental tissue may not be fully incised, and this step should be repeated. Once the flap is raised, approximately 1/2 of the buccal bone should be removed. It is important to remove some of the mesial and distal bone, as the tooth widens just under the alveolar margin. After bone removal, the tooth is carefully elevated. Once the tooth is elevated to a point of very loose attachment, it can be carefully extracted with forceps.

Bone is then smoothed with a coarse diamond bur. Gingival closure is initiated with fenestration of the periosteum. After fenestration, the gingival tissue should stay in position over the defect. If it does not, tension is present (and the flap will ultimately dehisce). Closure of the flap is done by placing the initial sutures at the corner(s) to ensure correct placement without tension. This step helps to avoid having to redo the entire closure if it does not place correctly to cover the defect after suturing most of it closed. After placing the corner sutures, the reminder of the incision is closed with simple interrupted sutures 2-3 mm apart.
Mandibular canine

These are quite simply the most difficult extraction in feline dentistry. Therefore, for numerous reasons, it is strongly recommended to avoid extraction of the mandibular canine teeth if possible, making referral for root canal therapy a much better option in many cases.

The best flap for mandibular canine tooth extraction is generally triangular with just one distal vertical incision. A horizontal incision is created along the arcade to just mesial to the mesial line angle of the third premolar. Next, a slightly distally divergent vertical incision is created. The flap is then carefully elevated. When the flap is created in this fashion, the frenulum will be elevated with the flap.

The buccal bone is then removed to a point approximately 1/3 of the way down the root. More bone can be removed if necessary, but caution must be used when creating a larger flap or taking more bone, as the mental nerve and artery exit approximately 3/4 of the way down the root. Next, the tooth is carefully elevated and extracted. Be careful not to damage the 3rd incisor or fracture the bone. It is helpful to use a small/sharp luxating elevator with gentle elevation.

Gingival closure is initiated with fenestration of the periosteum. After this is performed, the gingival tissue should stay in position over the defect. If it does not, tension is present and the flap will ultimately dehisce. The flap should be closed with the initial sutures placed at the corners to avoid having to redo the entire closure if it does not place correctly. Preplace the flap to ensure that there is no tension present, and close with simple interrupted sutures.

Crown amputation technique

Once a suitable candidate has been found and radiographically confirmed (see above), the procedure can begin. Crown amputation is initiated by creating a small gingival flap around the target tooth. This is typically a conservative envelope flap.

Next, a cross cut taper fissure bur on a high-speed handpiece is used to remove the entire crown to the level of the alveolar bone. The bone and remaining tooth should be smoothed with a coarse diamond bur. Following clinical and radiographic confirmation that the tooth is removed to at least the level of the bone, the gingiva is sutured over the defect. Closure may require a small amount of fenestration to relieve tension.

Further reading

Niemiec BA: Dental Extractions Made Easier. Practical Veterinary Publishing. Tustin CA
New from iM3, introducing the Vet-Tome

The Vet-Tome improves the tooth removal process for both vets and their patients by providing greater control during extractions. The Vet-Tome is an automated periotome that offers extremely precise tooth extraction with minimal or no alveolar bone loss.

The surgery is often flapless so the animal experiences reduced pain and swelling. This translates to less time spent extracting teeth and faster recovery time for the animal.
However good you are at dentistry, having poorly maintained equipment, both hand instruments and powered machines will be at best frustrating, and at worst damaging to the patient. This presentation aims to give an overview of keeping dental equipment in the best condition to perform dentistry to the highest standard. It is aimed at the general practitioner, and is suitable for both veterinary surgeons and nurses.
Double your dentistry®. A study of five practices and their keys to success

Cindy Charlier

As the leading provider of capital equipment to companion animal veterinary hospitals, Midmark Animal Health conducted a study to determine the key ingredients necessary to develop a successful dentistry practice to serve the needs of patients in a small animal hospital. Our goal was to develop a ‘recipe’ for success and provide case studies to support our “return on investment” claims when selling equipment. Midmark partnered with Dr. Cindy Charlier, a board certified dentist, to develop and conduct this study. Our hypothesis was that dentistry and oral surgery is the largest untapped source of practice revenue and patient care in most small animal practices. With the proper equipment, training and tools most practices should see a significant return on their investment in the dentistry portion of their practice. What is possible? What is that untapped potential? We will present our findings, which supported our hypothesis and exceeded our expectations.

The method

We selected five well managed multiple doctor practices that had an interest in improving their dentistry. They were all performing at or below the industry benchmarks in terms of dentistry as a percent of practice revenue.

As part of the process, we first determined what each hospital was already doing in terms of veterinary dentistry: how they were trained, what equipment they were utilizing, and how they were promoting dentistry services within the practice. Then we installed state-of-the-art dental equipment, including digital radiography and high speed dental delivery systems. Dr. Charlier provided comprehensive clinical and practice management training for their staff, and they were given templates, materials and support that would help them promote their dentistry practice and educate their clients.

The results

The industry benchmarks utilized for the study were the AAHA Fee Reference Guide 7th Edition and Benchmarks 2011 by Vet Economics, Wutchiett & Tumblin Associates. (referred to as “AAHA” and “Top 100” in the tables below) Both of these sources suggest
that a well managed general practice should be generating 3% of their practice revenue from dentistry services. These sources provide median practice revenue for clinics included in their surveys, data on dentistry as a percentage of practice revenue, and dentistry revenue per full time equivalent veterinarians (FTE). Table 2 compares the pre study performance of the 5 practices (the “Midmark” study clinics) to these industry benchmarks.

Table 2: Pre Study Data: Total dental revenue, dental revenue / FTE and dental revenue as a percent of total practice revenue.

<table>
<thead>
<tr>
<th>Site</th>
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<th>Hospital 3</th>
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<th>Hospital 5</th>
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<tr>
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<td>3</td>
<td>5.5</td>
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<tr>
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<td>31</td>
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<tr>
<td>Patients</td>
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<td>1,965</td>
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The success of the study is evident in the practice numbers in Table 3 which show a dental revenue average of $95,000 more per hospital than the median dental revenue for AAHA accredited hospitals and almost three times the benchmarks in terms of revenue per FTE.

Other data including increase in the number of dentistry procedures and increase in revenue from dental radiographs will be shared during the presentation.

Table 3: Post Study Data: Total dental revenue, dental revenue / FTE and dental revenue as a percent of total practice revenue.

<table>
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<th>Site</th>
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<td>Ann Rev</td>
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Chart 1 demonstrates the improvement in productivity measured by dentistry revenue / FTE on a quarter by quarter basis during the 12 month study period. This quarterly improvement can be attributed to doctors and staff becoming more comfortable with procedures and new protocols, the staff being more comfortable educating clients about the importance of oral healthcare, implementation of more ideas, and continuous monitoring of hospital performance as the study progressed.

Using these results, we can calculate the return on investment for the typical dental suite equipment that includes a high speed dental delivery unit like Midmark’s VetPro 1000 plus a digital dental radiography unit like Midmark’s VetVision Complete. With an initial equipment investment of $20,875 (at full retail), based on the data from the study hospitals, the payback period would have been as little as 2 months, with an average of a 5 month payback period. Using this same scenario, the return on investment averaged 330%.

**Five Keys to Success**

1. Owner buy in
2. Proper Equipment (especially digital dental X-ray)
3. Training for the entire staff
4. Client Education
5. Continuous improvement

The results of this study supported the hypothesis that with the proper equipment and training and with an implementation plan, the dentistry portion of a small animal practice can provide the hospital with a new source of patient care and practice revenue and a significant return on investment.
Ptyalism

Saliva is a viscous protein-based fluid which flows throughout the mouth, playing an important role in support and maintenance of the health of the oral soft and hard tissues. Among the body’s defense mechanisms, saliva works to lubricate and initiate breakdown of ingesta as well as protect the oral soft tissues.1 Ptyalism is defined as a pathologic overproduction of saliva, which may occur from a number of disease states.2 Pseudoptyalism refers to drooling caused by an inability or reluctance to swallow a normal amount of saliva.3 This lecture will cover both forms as they are often interrelated.

Excessive salivation, defined as saliva beyond the margin of the lips, is considered to be a normal finding in some breed of dogs (e.g. Saint Bernard, Dogue de Bordeaux, and Mastiff).4

Etiology and pathogenesis

Saliva is produced in and secreted by the salivary tissues, which are mostly in glands but also occur diffusely throughout the mouth. There are four major pairs of salivary glands in cats and dogs, parotid, zygomatic, mandibular, and sublingual.5 The cat has in addition two small circumscribed glands linguocaudally to each mandibular first molar tooth, which are called the lingual molar gland.6

References

Parasympathetic postganglionic cholinergic nerve fibers control the rate of the salivary secretion, inducing the formation of large amounts of a low-protein, serous saliva. 

Sympathetic stimulation promotes saliva flow through muscle contractions at salivary ducts. In this regard, both parasympathetic and sympathetic stimuli result in an increase in salivary gland secretion. The sympathetic nervous system also affects salivary gland secretions indirectly by innervating the blood vessels that supply the glands.

Ptyalism results from an increase in production by one or all of the salivary glands. Pseudoptyalism results from some disruption of the swallowing mechanism, voluntary or involuntary. Often the voluntary disruption is pain-induced, whereas the involuntary is caused by obstruction.

There are numerous causes for hypersalivation, and numerous locations where the inciting cause may originate.\textsuperscript{1,2} The inciting cause can originate from the oral cavity, esophagus or alimentary tract, or within the salivary glands themselves. Hypersalivation also can occur due to systemic or neurologic conditions.

Excessive salivation is a common clinical finding in patients with diseases of the oral cavity, and it is usually seen as a consequence of pain, inflammation or obstruction. Trauma patients, such as those with mandibular fracture, may have concurrent disruption in the normal mechanisms of swallowing.

Inability to retain saliva within the mouth due to poor head or lip control, constant open mouth, decreased or abnormal tongue mobility, decreased tactile sensation, macroglossia, dental malocclusion and nasal obstruction can all lead to ptyalism.

Patients who have ingested toxins may have both direct noxious effects on the saliva production, and indirect effects through inflammation of the mucosal surfaces. Intoxicated patients can also be seen with ptyalism caused by the central nausea effect of the toxin.

Primary salivary gland disorders (e.g. necrosis, inflammation and cancer) usually provoke an increase in the secretion of saliva. However, other salivary gland diseases may actually result in a decrease in saliva production.

Neoplasia affecting structures of the oral cavity, oropharynx, or esophagus can disrupt the normal swallowing mechanism and cause pseudoptyalism.

True ptyalism is a common clinical sign with gastrointestinal, metabolic, and systemic diseases, and involves activation of the humoral and neural pathways for nausea and vomiting. Infectious diseases, including viral, bacterial, rickettsial, and protozoal infections, can have direct or indirect effects on the saliva production. Central nervous system disorders can either increase salivation or interfere with normal swallowing function.

**Clinical signs\textsuperscript{3,4}**

The classic appearance is saliva dripping or pouring from the oral cavity. The drooling may be categorized as mild to severe as well as intermittent to continuous. Clinical signs


can occur acutely or be seen as gradual or chronic. The saliva may appear clear or it can be mixed with sanguineous or purulent exudates. Other potential clinical signs such as vomiting, regurgitation, anorexia, oral pain, and oral inflammatory lesions are related to the individual cause.

**Differential diagnoses**

- **Neurologic:** trigeminal neuropraxia, megaesophagus, facial paralysis, seizures, nausea, from vestibular diseases; glossopharyngeal, hypoglossal or vagus nerve lesions that result in the inability to swallow.

- **Developmental:** severe brachygnathism, extensive lip fold, long tongue.

- **Trauma:** soft tissue ulceration or laceration, electrical burn, temporomandibular joint (TMJ) luxation or fracture, mandibular fracture.

- **Postsurgical:** anesthesia, mandibulectomy, glossectomy, mandibular canine extraction.

- **Toxic:** organophosphates, caustic ingestion, animal venom.

- **Drug induced:** opiates, medications with a bitter or unpleasant taste.

- **Behavioral:** Pavlovian salivation associated with food, contentment/mood as in cats during

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purring, pain.

**Obstructive:** oral or esophageal foreign body, hematoma, seroma, or neoplasia.¹

**Metabolic:** hepatic encephalopathy, renal failure, exocrine pancreatic insufficiency, hyperthermia.²

**Gastrointestinal:** Nausea, hiatal hernia, megaesophagus, gastric dilatation/volvulus, gastric ulcer, esophageal stricture, esophagitis, neoplasia, or foreign body.³

**Infectious:** acute calici or herpes virus infection, rabies, pseudorabies, tetanus, botulism, upper respiratory infection, candidiasis, severe periodontal disease, spirocercosis.⁴

**Immune mediated:** Chronic ulcerative paradental stomatitis (CUPS) in dogs, caudal stomatitis in cats, pemphigus, bullous pemphigoid, toxic epidermal necrolysis (TEN), Masticatory muscle myositis, myasthenia gravis.⁵

**Salivary:** sailolith, foreign body, neoplasia, hyperplasia, infarction, sialoceles, necrosis, idiopathic.⁶


**Diagnostic steps**

The most important initial step is to obtain a complete history and perform a thorough physical exam.

**History**

Establish possible cause, severity, complications and progression of disease. Important areas of history include: age and mental stage of the patient, chronicity of the condition, associated neurological signs, timing, provoking factors, and estimation of quantity of saliva. Historical questions should include: general history, age, acute vs. chronic clinical signs, other gastrointestinal signs (e.g., vomiting, diarrhoea), toxic exposure, drug administration, or trauma.

**Historical significance**

*Age of the patient:* Young patients are more likely to have toxic exposure, foreign body, acute viral infection, or portosystemic shunts (especially small breed such as Yorkshire Terriers and Maltese). Mature patients are more likely to be affected by metabolic, immune-mediated or neoplastic diseases.

*Acute versus gradual or chronic:* Acute onset of significant pseudo- or true ptyalism is most often associated with a virus, toxic, or oral trauma. Gradual or chronic onset is more likely to be associated with a metabolic or neoplastic process.

*Gastrointestinal (GI) signs:* If ptyalism is associated with nausea or vomiting, a GI, systemic, or neurologic problem is more likely. Regurgitation should prompt an esophageal exam, as this is typically associated with an esophageal condition (i.e. megaesophagus). If ptyalism is noted in combination with difficulty eating or quidding, an oral problem should be suspected and a complete oral and maxillofacial exam performed.

**Physical exam**

The most important part of the physical exam is a complete oral/maxillofacial/esophageal examination. A thorough oral exam evaluates for infection, neoplasia, fractured/abscessed teeth, periodontal status, trauma, inflammatory/ulcerative disease and foreign bodies.

The clinician should check for sores on the lips or chin, tongue control, swallowing ability, nasal airway obstruction, decreased intraoral sensitivity, and anatomical closure of the mouth. The oral examination should include the sublingual area, as this is a common place for masses and string foreign bodies.

Oral ulcerative inflammatory diseases are typically in advanced stages before ptyalism is induced, and thus in these cases abnormal oral examination findings should be readily seen. These conditions include acute calici virus or herpes virus infection, immune mediated diseases, caustic ingestion, and uremia.

The saliva itself should be examined for consistency and any additional components (i.e. blood or pus). Assess severity and frequency of the drooling. The hydration status and head posture of the patient should be evaluated.

It is important to note that a complete oral examination is not possible without general anaesthesia, and that a minimum database should be obtained prior to the anesthetized exam.
Saliva that has a sanguineous, purulent or fetid component us usually secondary to a problem within the oral cavity, such as: oral infections (such as oronasal fistula), trauma, neoplasia, inflammatory disease, or uremic ulcers.

A maxillofacial exam should include evaluation for swellings, asymmetry, TMJ luxation, trauma, cranial nerve function, and size and consistency of the salivary glands.

Inability to close the mouth indicates one of the following causes: traumatic (TMJ/mandibular fracture/luxation), neurologic (botulism or trigeminal neuropraxia), or obstructive (neoplastic or foreign body). Inability to open the mouth is most commonly associated with: tetanus, craniomandibular osteopathy, masticatory muscle myositis, neoplasia, or TMJ issues. The salivary glands should be systematically evaluated. Enlargement could indicate: infection, sialoliths, or neoplasia.

The physical examination of the esophagus is limited to external palpation for masses, pain or foreign bodies. Complete evaluation may require radiographs (+/- contrast), fluoroscopy, cat scan, and/or endoscopy.

**Diagnostic tests**

It is important to start the initial diagnostic testing with a minimum database including complete blood count/chemistry panel/thyroid level, and urinalysis. This will rule out most of the metabolic causes as well as help ensure that the patient is healthy enough to undergo general anesthesia for further evaluation.

For oral mucosal changes that are not obviously associated with a toxic or caustic cause or systemic disease (e.g., uremic ulcers) a surgical biopsy should be performed under general anesthesia and submitted for histopathology. When obtaining the pathology sample, ensure that it is representative and of sufficient size for an accurate assessment. It is worthwhile to note that cytology as well as culture and sensitivity are often insufficient for an accurate assessment of disease processes in the oral cavity.

In cases that clinically appear to have an oral cause but the problem cannot be readily identified on oral exam, dental radiographs should be performed. These radiographs may elucidate a subgingival cause such as a tooth root abscess or dentigerous/radicular cyst. Patients that present with derangements of jaw motion or maxillofacial swellings should be further evaluated with skull radiographs, nuclear scintigraphy, magnetic resonance imaging or cat scan. Finally, tests such as sialography can be of benefit.

Once oral and maxillofacial causes have been ruled out, further diagnostics are indicated, initiating with thoracic and abdominal radiographs. If the cause of the ptyalism has not been identified at this point, more specific testing should be performed, where indicated, such as upper GI studies, fluoroscopy, and endoscopy. The clinician may also consider tests for botulism and rabies.

**Treatment**: is directed at the undelaying cause. Examples of treatments of ptyalism can be:

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• Direct toxic exposure should be treated with dilutional therapy and supportive care.\(^1\)
  Water or milk are considered the liquids of choice for dilution.\(^2\)
• Therapy for oral inflammatory diseases should be directed towards reducing the inflammation. This can be accomplished medically with immunosuppressive agents or surgically with periodontal treatment and/or extractions.\(^3\)
• Oral traumatic diseases are best treated surgically.\(^4\)
• Portosystemic shunts can be managed surgically or medically.
• Metabolic derangements treat as appropriate for the disease process.\(^5\)

In case of idiopathic or incurable conditions such as structural or neurologic diseases, treatment is directed at decreasing the flow of saliva and protecting the epidermis in the chronically wet area. If one salivary gland is responsible for the increased production, surgical excision is the treatment of choice.\(^6\) Cheiloplasty can be performed to help eliminate excessive drooling caused by lip malformation, mandibulectomy, glossectomy, or neurologic disorders of swallowing.\(^7\) Finally, surgical repositioning of the parotid salivary duct may be effective in controlling the excess salvation.\(^8\)

Decreasing the overall flow of the saliva can be attempted with atropine or glycopyrrolate.\(^9\)


In cases of idiopathic ptyalism, phenobarbital may be effective. In these cases it is thought that the ptyalism is a form of epilepsy. In human dentistry, injections of biotoxin or ethanolamine oleate (EO) into the salivary glands, as well as radiotherapy, scopolamine via a transdermal patch, and even acupuncture have been investigated options for long-term salivary control.

**Halitosis**

The origin of the term halitosis comes from the Latin word halitus meaning “breath or exhaled air.” Halitosis is defined as an offensive odor of the breath. It is a common problem in companion animals and constitutes a significant psychosociological problem in the companion animal-owner relationship. There is no sex or breed predilection, but the incidence increases with age.

**Classification of halitosis**

There is no universally accepted, standardisation in terminology and classification of halitosis. Genuine halitosis can be sub-classified as physiologic halitosis or pathologic halitosis. Pathologic halitosis means that the breath odor is a symptom of a disease or a pathologic condition. Physiologic halitosis covers the situations where patient has no disease but has a malodor because of putrefaction processes taking place in the oral cavity, most frequently caused by bacterial plaque. An example of physiologic halitosis is what is known in human as “morning breath.” In most cases, physiologic halitosis can be resolved with improved oral home care. This classification of bad breath is considered to be transient, in the sense that its presence comes and goes, as determined by temporary localized conditions in the mouth, and that it can be relatively easily resolved.

The best classification system for veterinary patients is based on etiology which divides

halitosis into types based on where the offending molecules originate. This formula is broken into oral, airway, gastroesophageal, blood-borne, and subjective.

Halitosis can also be classified according to the character of the odor:1 Sulfurous is caused by volatile sulphur compounds: methyl mercaptan, hydrogen sulphide and dimethyl sulphide. Fruity is caused by acetone. Urine or ammoniacal breath is caused by ammonia, dimethyl amine and trimethylamine. Sweet smelling breath is often associated with ketones. The degree of halitosis can be measured by a subjectively scale from 0 to 3,2 or It can be objectively measured using a commercially available sulfide monitor.3

**Causes of halitosis**

**Oral Halitosis:** In about 90% of human patients with halitosis, the origin of the problem is within the mouth itself.4 The list of possible causes of halitosis originating from the oral cavity itself is long, however by far the most prevalent cause is bacterial growth below the gum line in periodontal pockets created by periodontitis.5 Periodontal disease is generally described in two categories, gingivitis and periodontitis. Gingivitis is the initial, reversible stage of the disease of which the inflammation is confined to the gingiva.6 At this point, there is no inflammation in the periodontal ligament or alveolar bone. The gingival infection is initiated by the plaque bacteria and can be reversed at this stage if a dental prophylaxis is performed and proper home-care maintained.7 Periodontitis is the later stage of the disease process, which is defined as an inflammatory disease of the supporting structures of the teeth (the periodontal ligament and the alveolar bone) caused by microorganisms.8 While it is initiated by plaque, the progression of disease is regulated by the patient’s immune response.9 In fact, it is actually the host response that often damages the periodontal tissues.10

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10 Lang NP, Mombelli A, Attstrom R. Dental Plaque and ZCalculus. In: Clinical Periodontology and implant
Both gingivitis and periodontitis is initiated when oral bacteria adhere to the teeth in a substance called plaque.\(^1\) Plaque is a biofilm almost entirely made up of oral bacteria contained in a matrix composed of salivary glycoproteins and extracellular polysaccharides.\(^2\)

Plaque formation begins with the formation of the pellicle. The pellicle is a thin, saliva-derived layer including numerous proteins, enzymes and other molecules that can act as attachment sites for bacteria.\(^3\) This starts forming nanoseconds after a prophylaxis. Plaque is formed when bacteria attach to the pellicle.

Gingivitis is caused by an increase in the overall numbers of bacteria, which are primarily motile gram-negative rods and anaerobic species.\(^4\) The early colonizers are gram-positive aerobic and generally minimally pathogenic. However, they promote the growth of the secondary and more periodontopathogenic colonizers, such as Porphyromonas. They accomplish this by using oxygen and making products such as lactate, formate, and succinate. The host provides nutrients to the pathogenic species in the form of blood and crevicular fluid.\(^5\)

The whole process of plaque formation takes 24 hours if the plaque is not disturbed, which means that the teeth accumulate plaque one day following a complete dental prophylaxis.\(^6\) After day 4 the plaque does not grow anymore, but the flora changes from gram-positive to gram-negative bacteria. This change in bacterial species is what initiates gingivitis.\(^7\)


5 Niemiec BA. Veterinary periodontology pp. 18-34.


gingiva, but they become approximately 95% of the flora in dogs with periodontitis.\textsuperscript{1} As the virulence of the bacteria increases, so does the effect of bacterial byproducts which elicit inflammation, including chemotoxins, mitogens, antigens, and enzymes such as hyaluronidase, chondroitin sulphate and proteolytic enzymes.\textsuperscript{2}

Oral halitosis mainly originates from volatile sulfide compounds (VSCs), especially hydrogen sulfide (H\textsubscript{2}S), methylmercaptan (CH\textsubscript{3}SH) and dimethylsulfide ((CH\textsubscript{3})\textsubscript{2}S).\textsuperscript{3} These compounds typically result from the proteolytic degradation of peptides by oral microorganisms. These peptides are present in the saliva as well as gingival crevicular fluid, interdental plaque, and blood. In addition, they can come from shed epithelium, food debris, and discharge from the naso-pharynx. It is interesting that only gram-negative anaerobic bacteria possess such proteolytic activity. Wherever the location, the common pathophysiology is tissue destruction and putrefaction of amino acids by bacteria. The bacteria associated with gingivitis and periodontitis are almost all gram negative anaerobes and are all known to produce VSCs.\textsuperscript{4}

The VSC levels in the mouth correlate positively with the depth of periodontal pocket(s).\textsuperscript{5} This is likely due to the fact that deeper pockets will contain more bacteria, including a higher percentage of anaerobic species. The amount of VSCs in breath increases with the number, depth and bleeding tendency of the periodontal pockets.\textsuperscript{6} The VSCs also directly aggravate the periodontitis process. They increase the permeability of the pockets and mucosal epithelium and expose the underlying connective tissue of the periodontium to the bacterial metabolites.\textsuperscript{7} Low oxygen tension in the deep periodontal pockets results in a low pH and activation of the decarboxylation of the amino acids (e.g. lysine, ornithine) to

cadaverine and putrescine, both malodorous diamines. Thus, in the presence of gingivitis or periodontitis, VSCs play a prominent role in halitosis; but it is important to remember that not all patients with gingivitis or periodontitis have halitosis and vice versa.

The association between bad breath and periodontal disease in companion animals poses an important issue because halitosis is often the first clinical sign of periodontal disease noticed by the owner. However, it is important to note that halitosis is typically a sign of advanced periodontal disease. Clients should therefore be counselled that halitosis is not normal and that is an indication for professional therapy.

**Treatment of periodontal diseases**

There are numerous therapeutic options available for periodontal diseases; however, the basis of periodontal therapy remains plaque control. The cornerstone of plaque control and the first step for any periodontal therapy is a thorough dental prophylaxis. A complete dental prophylaxis should include the following steps: pre-surgical exam, 0.12% chlorhexidine lavage, supra and sub-gingival scaling, polishing, sulcal lavage, periodontal probing, oral evaluation, and dental charting, dental radiographs, treatment planning and surgery if necessary.

Pockets over 0.5 mm in cats and 3 mm in dogs are pathologic and require a deeper form of cleaning (+/- perioceutic). Teeth with pockets greater than 6 mm, furcation exposure level II or III, or mobility require periodontal flap surgery or extraction to eradicate the infection.

Bacterial plaque colonizes clean tooth surfaces within 24 hours of cleaning. Therefore without a commitment to home-care, gingival infection and inflammation quickly recurs. In addition, with regards to established disease, a recent study found that periodontal pockets become reinfected within two weeks of a prophylaxis if homecare is not performed. This same study showed that pocket depth returns to pre-treatment depths within 6 weeks of therapy. Furthermore, it was found in a human review that professional cleanings were of little value without home-care.

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Other oral causes: There can be other intraoral courses of halitosis; however, these are all much less common than periodontal disease.1 The list of differential diagnoses within the oral cavity is long but contains the following conditions: infections, ulcerations, tumors, and foreign bodies.

Owners often misdiagnose malodour caused by intertrigo or skin fold pyoderma as halitosis.2 Intertrigo arises from an overgrowth or colonization of skin folds by normal skin bacteria and sometimes yeast. Lip folds are common in brachycephalic breeds, spaniels, and many water dogs and these breeds are often presented due to the smell. The clinical signs are hair loss, redness, and accumulation of debris in the lip folds around the mouth.

Airway

Nose and sinuses: The nasal and sinus passages can also be the origin of halitosis. The smell typically has a stronger odor when coming from the nose compared to oral infection. Nasal halitosis can be caused by foreign bodies, neoplasias, or even chronic rhinosinusitis. Breath analysis techniques have not been applied to this condition, but theoretically there are several possible mechanisms of halitosis caused by infection in the nose or sinuses.3

Tonsils: In the human literature, there is disagreement as to the proportion of halitosis caused by tonsil pathology.4 Tonsillar diseases which may be associated with halitosis include: chronic caseous tonsillitis, tonsillolithiasis and to a lesser extent peritonsillar abscess, actinomycosis, fungating malignancies and different kinds of tumors.5

Bronchi and lungs: Pulmonary causes include chronic bronchitis and bronchiectasis.6 Bronchiectasis is the result of chronic airway disease leading to a dilatation of the bronchi and a collection of mucus and cell debris within the air passages. It is commonly found in those animals that have been suffering from chronic bronchitis or bronchopneumonia.

Gastroesophageal: Megaesophagus is a disorder of the esophagus characterized by dilation and decreased peristalsis. Halitosis is a common clinical sign of megaesophagus along with regurgitation, weight loss, and coughing.7 Halitosis resulting from other extraoral gastrointestinal disorders is considered to be rare. However, it has been reported among the

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signs related to \textit{Helicobacter pylori} infections and gastroesophageal reflux disease.\textsuperscript{1}

Systemic conditions can also occasionally cause halitosis. The list of systemic differential diagnosis for halitosis include: diabetes mellitus, renal infections and failure, liver disease, and carcinoma.\textsuperscript{2} However, patients suffering from such systemic diseases typically show additional and more diagnostically conclusive symptoms than halitosis alone.

Diabetes mellitus is a metabolic disorder characterized by a chronic hyperglycemic condition resulting from defects in insulin secretion, insulin action, or both.\textsuperscript{3} The deficiency in or the lack of action of insulin leads to uncontrolled lipolysis and elevated levels of free fatty acids in the plasma. This loss of proper regulation may result in the formation of ketones, used for energy production. A spontaneous breakdown product of the ketones (acetoacetate) is the acetone that is exhaled by the lungs, which gives a distinctive odor to the breath, which has been described as “rotten apples”.\textsuperscript{4}

Kidney insufficiency will lead to increased uric acid levels in the blood, which is exhaled creating ammonium-like breath.\textsuperscript{5} This is usually described as a “fish odor”.\textsuperscript{6} To fully investigate the underlying cause of kidney damage, imaging, blood tests an often renal biopsy are often used.

Liver insufficiency, such as cirrhosis, will cause ammonium to accumulate in the blood and be exhaled.\textsuperscript{7} Foetor hepaticus is a peculiar odor to the breath in people with severe liver disease, caused by volatile aromatic substances that accumulate in the blood and urine due to defective hepatic metabolism. It is a late sign in liver failure and is one of the clinical features of hepatic encephalopathy. Malodor caused by end stage liver disease has a sweet odor, which some describe as that of “dead mice”.\textsuperscript{8}

\textit{Specific Character of Breath odor.}\textsuperscript{9} A “rotten eggs” smell is indicative of VSCs. Malodor caused by end stage liver disease has a sweet odor, which some describe as that of “dead mice”. The smell of “rotten apples” has been associated with unbalanced insulin-dependent diabetes, which leads to the accumulation of ketones. A “fish odor” can suggest kidney insufficiency characterized by uremia and accumulation of dimethylamine and trimethylamine.

\textsuperscript{1} Kindberg S, Stein M, Zion N, Shaoul R: Can J Gastroenterol. 24(9):552-6, 2010.
Acanthomatous ameloblastoma (canine acanthomatous ameloblastoma) is classified as a non-inductive odontogenic tumour [1,2,3,4,5,6,7,8]. Odontogenic tumours can be classified as either benign or malignant. The benign tumours can be classified as those with little potential for recurrence (odontomas), locally aggressive (central ameloblastomas, AA, amyloid-producing odontogenic tumour, feline inductive odontogenic tumour) and those with some potential for recurrence (peripheral odontogenic fibroma). The malignant tumours include ameloblastic fibro-odontoma, odontogenic myxoma and ameloblastic carcinoma [1,2,3]. Malignant odontogenic tumours are extremely rare in dogs and cats [1,2].

The AA, formerly called acanthomatous epulis or peripheral ameloblastoma, is a benign epithelial tumour but behaves in a locally aggressive way [1,2,3,5,6,7]. It originates from remnants of the odontogenic epithelium located in the gingiva in the tooth-bearing areas of the jaws [1,2,3]. It is a common odontogenic tumour in dogs and arises mostly at the age of 6-12 years [16] or 7-8 years [2], but can also be seen in dogs under 5 years old [2,8]. This tumour shows no signs of distant metastasis. A common area for the tumour is the rostral mandible, but it can also be seen in the maxilla and sometimes in the ramus of the mandible [1,2]. Clinical presentation is of a gingival overgrowth. Radiographically it may show signs of being osteoinvasive and osteodestructive [1,2,3,5,6,9,10].

Different treatment options are described: surgical removal, radiotherapy and chemotherapy [1,2,4,8,9,10,11,12,13,14,15,16]. For chemotherapy, the injection of bleomycin or bleomycin/Interleukin 12 has been described [18,20] in a small group of dogs which was shown to be effective. There are some potential side effects such as tissue reaction, bone exposure, local swelling and infections and failure of second intention healing, but this seems to be a possible palliative treatment for non-resectable AA’s [12,13,14].

Radiotherapy is another option [1,2,15,16]. In smaller tumours, radiotherapy may show good results. In bigger tumours (over 3-5cm), an incomplete remission or recurrence can occur. Therefore, radiotherapy is often used as an adjunct to incomplete removal of the tumour. Radiation therapy alone has a risk of 18% for tumour recurrence or malignant transformation at the irradiated site [1,2,8,9].

The best long term results have been shown with radical surgical resection, with 97% of cases not demonstrating any recurrence [1,2,4,8,9]. In most cases partial mandibulectomy
is adequate. The resection margins of this tumour are described from 0.5 cm – 1 cm [1,2]. Often it depends on the location of the AA, the dimension and the area. A CT scan can help to find the margins for surgery and to plan surgery. After tumour resection biopsies of the tumour bed are indispensable as well as confirmation of the diagnosis by a pathologist [15,16].

Possible complications are suture dehiscence, infection or automutilation by the dog [17]. A collar is required after surgery as well as a good pain medication and antibiotics [18,19, 20]. A swab taken directly from the tumour in the beginning of the surgery or at time of CT scan can save time if change of antibiotic treatment is necessary. Soft tissue swelling can cause problems with salivary glands and ducts. Mandibular drift may be seen after mandibulectomy or partial mandibulectomy. Powerchains or immediately reconstructive treatment with bonegraft and plates are options to prevent problems [21,22, 23].

Literature

Therapy in veterinary medicin ; J Transl Med 10 :234.
Immunohistochemistry markers in canine oral squamous cell carcinoma: Are they useful to the clinical practice?

Lisa A. Mestrinho

Comparative oncology studies in oral squamous cell carcinoma (OSCC) have implicated several markers in its carcinogenesis - markers of cell cycle regulation, apoptosis, angiogenesis, and adhesion, degradation of extracellular matrix among others. Regardless of the direct impact of such markers in differential/early diagnosis and prognosis, they can be used to predict response to specific treatments. The gold is a multimodality approach, especially in advanced stage tumours, when surgery fails to achieve cure.

Immunohistochemistry is one of the techniques used to identify such markers and used routinely in both human and veterinary medicine.

In light of the current knowledge of the molecular nature of such tumours, some of these markers have been included in the routine of some veterinary labs, improving the diagnosis and consequently treatment choices.

Cell cycle regulators such as Ki-67 and PCNA tend to be overexpressed in both human and canine OSCC. These markers are related with an increased growth, in some cases with high-grade tumours (poor differentiation) and with lymph node metastasis. These markers are related with prognosis, being related with a high recurrence rate and thus the necessity to increase surgical margins. Additionally, they can be used as indications in the selection of radiotherapy or chemotherapy protocols. Theoretically radio and chemotherapy act in active cell cycles, consequently improving the initially guarded prognosis of such tumours. In canine OSCC studies histological grade may be an important prognosis factor. A shorter disease-free survival time was observed in those animals with high grade and elevated PCNA index.

Apoptosis markers, such as p53, p63, p21, Bcl-2, etc., were addressed in human OSCC. These markers theoretically could identify resistant cell populations to cytostatic drugs or radiotherapy. Additionally p63 has been demonstrated to be highly specific for epithelial cells, being, in human labs, used for differential diagnosis in undifferentiated tumours along with cytokeratins, or alone in some cases. In the dog p63 expression seems to be reactivated, secondary to gene mutation and/or overexpression and is very specific to epithelial cells. Clinical human retrospective case series have pointed p63 overexpression as a marker of prognosis and treatment resistance, especially to cisplatin, commonly used to treat this cancer.

Angiogenesis markers, such as VEGF, have been determined by immunohistochemistry
in human studies, but molecular biology techniques are more sensitive. In the dog, VEGF was evaluated by ELISA methods, showing highest values for OSCC. This marker triggers lymphatic angiogenesis, which may result in a higher risk of lymph node and haematogeneous metastasis. The combined evaluation of microvessel density and lymphovascular invasion were also assessed in canine OSCC being related with a worst prognosis.

Cell adhesion markers such as cadherins, catenins, etc., are transmembrane glycoproteins involved in cell-to-cell adhesion. E-Cadherin is one of the most studied in humans and also studied in the dog. Under expression in a common event in OSCC, being related with a poor prognosis and increased chance of lymph node or distant metastasis.

With regards to degradation of extracellular matrix, matrix metalloproteinase (MMP) are zinc dependent endopeptidases which are able to degrade all kind of extra-cellular matrix proteins. In the dog MMP-2, tissue inhibitor MMP-2 (TIMP-2) and MMP-2/TIMP-2 ratio were evaluated, being positively related with metastatic disease.

Finally, cyclooxygenase (COX)-2 is an induced enzyme expressed by cells that are involved in inflammation, ischemia and cancer. COX-2 was increase in a large percentage of OSCC in dogs. Although, the studies didn’t address prognosis inhibition of such molecule could be a targeted for anti-tumour therapy.

*Literature available upon request.*
ATP of maxillo-facial lumps and bumps

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Like with many other areas of the body, the practitioner is often confronted with masses or “lumps” that involve the head and the oral cavity. These are often discovered during routine wellness examinations. Most of the time, clients are usually unaware of the lump’s history and existence. This is especially true where the growth is in the oral cavity. By the time the pet shows any specific symptoms related to the lump it usually has been growing for a while changing the pet’s behavior and causing pain. Practitioners therefore must perform a careful evaluation. In order to have a more focused approach when dealing with these lesions, the practitioner needs to adhere to an evaluation sequence which helps develop a rule out diagnosis. This will yield a more accurate understanding of the growth’s origin and development and allow for the subsequent recommendation for treatment that the owners will follow.

Exophytic lesions are a term that encompasses all masses regardless of type and etiology. By definition it is any pathologic growth that projects above the normal contours of the maxillo-facial surfaces.

ATP has been coined as assessment, treatment and preventative. For our purposes, the “TP” will signify “treatment plan” since prevention often depends on the lump’s diagnosis which might not be preventable.

Assessment of the patient’s problem involves a thorough history. Any changes of an animal’s behavior might key in as to when a growth started to be painful. Key questions that need to be asked:

- Does the patient play with toys? If so what types? (Important if pieces can splinter off and create a foreign body reaction) If the pet stopped playing with its toys or only takes it from one side or prefers only soft toys, this needs to be noted and a more thorough evaluation of the opposite side of the mouth which might be favored should be undertaken.
- Does the pet exhibit halitosis. Is there a necrotic smell?
- Is the animal salivating? Is it unilateral? Is it bilateral?
- Is there any blood on the pet’s play toys? Does the animal spontaneously bleed where there is blood in the saliva or on furniture or bedding? Does it occur after eating or...
drinking?

- Is there any facial shyness? Does the pet tilt his head to the side? If so, then the side that is down is usually the affected side.
- Does the animal vocalize or attempt to rub or scratch at the face?
- Does the pet show an interest in food and goes frequently to the bowl but yet does not want to eat? And if he does eat does he swallow quickly and drop food. Does he flip his head to the side when chewing? If he does, then the affected side is opposite to the side that he turns his head to.
- Weight loss? Over what time frame?
- Is there any nasal bleeding? Unilateral or bilateral or in association with respiratory stridor? Is the nasal swelling on the ipsilateral side as the bleeding?

If there is any facial swelling, is there any association with any oral pathology. If there is, is it related to a dental structure, bone or soft tissue? In the case of swelling is it confined to the skin or underlying tissue? Is there any ocular drainage? Is the eye in a normal position? Is one or both eyes affected?

The lump’s qualitative characteristics are important to establish possibly the masse’s origin. The physical characteristics of the mass that need evaluation are the following:

**Color**

- A pink surface color indicates that underlying capillaries are showing through the overlying thin stratified squamous epithelium.
- A white color of the mass indicates either a retention of keratin or fibrosing with diminished vascularity.
- A bluish or black color might indicate an accumulation of melanin, hemosiderin or fluid accumulation

**Consistency**

- Soft texture can indicate adipose type tissue, veins loose connective tissue. Or minor salivary glands.
- Cheesy feel can indicate a caseous necrosis or a sebaceous cyst.
- Rubbery texture can be a relaxed muscle or glandular tissue with a capsule or tissue that is compressed.
- Firm touch can be fibrous tissue, tensed muscle or cartilage. For example gingival hyperplastic tissue.
- Hard consistency would be a lump that has bone or dystrophic mineralization example being is an Osteoma.
- Fluctuance indicates a fluid filled cavity. Cysts, abscesses, mucoceles should be palpated with both hands to feel for an undulation generated between the hands or fingers by pressing the lump from one side and with the other hand feeling the fluid wave.

A mass’s surface can either be smooth indicating that it arises beneath the squamous epithelium or in the mesenchyme; example being a salivary gland, abscess or cyst. If the mass is corrugated it originates within the squamous epithelium like a papilloma or a squamous cell carcinoma.
Mobility indicates that the lump can emanate from the cutaneous, subcutaneous or submucosal tissue. A skin mass might be connected to deeper underlying pathology via a mobile fistulous tract. This is often seen with parula or “gum boil” which exits submandibularly from a mandibular tooth abscess.

If the mass is fixed this can occur either be due to fibrosis secondary to inflammation or if it is involving a malignancy that is either growing up from deeper tissue planes or growing down from more superficial planes. A fixed mass can also involve the attached gingiva or underlying bone as seen with Epulids or benign osteomas.

In addition to the above characteristics, any evidence of either loose teeth in association with the lump or missing teeth can indicate that the growth is down growing into the alveolar bone thereby displacing the teeth as seen often in either malignancies or locally aggressive masses like the acanthomatous ameloblastomas which stem from the periodontal ligaments.

Missing teeth or partially erupted teeth with an associated soft to fluctuant protuberance can indicate an odontogenic cyst. If the swelling is firm to hard in consistency and there is no evident tooth as seen with odontomas a primary tumor of the tooth should be suspected. This is especially true in younger animals that are in the clinic for their spays and neuters at 6 months.

As we can see from the history and the description of the growth we start to narrow down our rule-out diagnosis list. This is important in regards to client education and clearly describing the prognosis and the necessary treatment plan that needs to be followed. Helpful is to try to place the mass into one of the following groups: infections, foreign bodies, cysts, retention phenomena, inflammatory hyperplasias and neoplasias.

**Infections** of underlying dental structures can often present as skin masses. The location of these swellings can be suborbital in the case of a non-vital, abscessed maxillary P4 carnassial tooth or a rostral submandibular swelling from the mandibular canine tooth. Caudal swellings on the ventral aspect of the mandible or intermandibular area can often stem from a malformed, non-vital molar. A high index of suspicion should be maintained for a problem of bacterial dental origin. Often initially the lump responds to antibiotic therapy. Its return could be 1-3 months later. At this time it becomes more resistant to either the same antibiotic or one of different spectrum. It is therefore imperative that if a mass is external on the face and is responsive to AB’s, the practitioner should evaluate all the teeth for any evidence of fractures or discolorations & radiograph.

The treatment plan would be to perform intraoral radiographs to rule out a non-vital tooth and then either extract it or perform endodontic therapy to prevent further bacterial apical drainage into the surrounding bone. Careful curettage of the alveolus would eliminate potential cystic retention secondary to the granuloma.

**Foreign body granulomas** are fairly common. They can be superficial and visible to the unaided eye on routine oral evaluations as material lodged in the surface mucosa or buried deep within the connective tissue of the oral cavity or skin. In the latter presentation the overlying granulomatous lesion can resemble either an inflammatory hyperplasia or neoplasia. Usually the associated inflammatory lump is red with white striations if the lesion starts to develop fibrosis. They have a firm consistency and can be solitary or at multiple sites. The treatment plan initially for deep sites is to perform an incisional or punch biopsy to determine the type of lesion. If it returns as unspecific inflammation then an “enbloc” resections should follow.

**Cysts** belong to the category of non-inflammatory lumps. Anatomically they are composed of three structures: a. central cavity which is filled with fluid, cellular debris, keratin or mucus; b. Epithelial lining which is either keratinized or nonkeratinized stratified...
squamous; c. Cyst wall of fibroblastic connective tissue. So by definition a true cyst has an epithelial lining and is further divided into odontogenic cysts and developmental. Bone cysts contrary to true cysts, lack this epithelial lining and are therefore pseudocysts. Odontogenic cysts contain an epithelial lining derived from tooth development. This epithelium stems from either 1) rests of Malassez epithelium derived from the tooth root’s sheath that is now contained in the periodontal ligaments. 2) enamel epithelium which is a residual tissue surrounding the crowns of the tooth after enamel is complete 3) remnants of the dental lamina which originate from the oral epithelium and remain in the tissue.

The odontogenic cysts are further categorized into:

A. periapical cysts which proliferate in response to inflammation secondary to tooth death. These are also called radicular cysts since they are closely adhered to the root apices. As the cyst enlarges it’s lumen osmotically draws fluid from the wall or capsule capillaries. This expands causing bone resorbing factors to be released. Radiographically the cyst is rounded and well circumscribed. It appears as a radiolucency surrounded by dense bone. This is called cortication. With periapical cysts due to the nature of the etiology, often there is an inflammatory neutrophilia histologically involving the cyst.

It is very important that after extracting the non-vital tooth that the cyst’s epithelial lining is curetted otherwise a residual cyst can develop.

B. Dentigerous cysts develop from the enamel epithelium that surround the impacted tooth’s crown. The cyst encapsulates therefore only the crown and not the tooth’s root. Visually the arch is missing this tooth and there might be a fluctuant swelling seen. Radiographic evidence of displacement of other adjacent teeth can be seen. It is possible that epithelial neoplasias can arise within the dentigerous cysts, such as ameloblastomas, and squamous cell carcinomas.

C. Eruption cysts are odontogenic cysts similar to a dentigerous cyst. The tooth crown has erupted through the bone but not the gingiva. The cyst therefore evolves in the overlying gingiva. Rather than an intrabony lucency like the dentigerous cyst, there is a fluctuant swelling of the alveolar ridge above the non-erupted tooth. This swelling can involve both the deciduous as well as the adult tooth.

D. Odontogenic Keratocyst derives from the epithelial remnants of the dental lamina and behave like benign neoplasms. They can occur at any site in the jaws. It can occur as multiple cysts and has a rapid growth potential and a high recurrence rate similar to an ameloblastomas if the cystic lining is not thoroughly curetted.

Treatment plan for Eruption cysts is to perform an elliptical gingival incision around the tooth’s crown and perform an apical reposition flap thereby exposing the adult tooth and draining the cyst. In the case of dentigerous and keratocyst, exodontia of the affected tooth(teeth) and curetting the cyst’s epithelial lining should be undertaken. If it is a calcifying cyst, an “enbloc” resection of the jaw should be done.

Salivary gland disorders are usually of the reactive type as a result of injury or infection. Primary neoplasia affecting the salivary glands usually are infrequent and represent often adenocarcinoma. Injury, on the other hand, to the gland’s major ducts is common. Obstructive sialadenitis occurs either due to the stricture and narrowing of the ductal lumen or blockage by an object(s) such as a sialolith(s). This stenosis of the duct’s lumen with subsequent obstruction causes the secretory acinar cells to shut down their secretion due to an increase back flow pressure. The gland initially enlarges and is painful. Eventually the
increase pressure causes atrophic degeneration and necrosis of the glandular parenchyma. Eventually the secretory tissue is replaced by fibrous scarring and the gland atrophies and hardens.

When a salivary duct tears the acinar cells will continue to secrete into the surrounding connective tissue. The tear usually occurs in the sublingual salivary gland duct prior to it’s rostral course under the lingual nerve. The extravasation of mucin is called a mucocele. Depending on the mucocele’s location, the names imply position i.e. pharyngeal mucocele, cervical mucocele and ranula (sublingual swelling).

In comparison to a mucocele, a true cystic formation is seen with the formation of a mucus retention cyst or a sialocyst. These stem from an obstruction of a salivary gland excretory duct which results in an epithelial lined cavity with mucus. This is most commonly encountered in the parotid gland.

Treatment plan for all mucoceles is to initially drain the mucin, followed by extirpation of the mandibular-sublingual gland complex. Ranula’s can be marsupialized by creating a fenestration through to the mucocele lumen and then suturing the epithelial lining to the sublingual mucosa. Eventually the fenestration will seal up with time and often these require the salivary glands to subsequently be removed on the ipsilateral side.

Reactive inflammatory hyperplasias are a group of lumps that histologically show chronic inflammatory reactions where there is an endothelial proliferation and an in growth of capillary vessels. Under this category are the following pathologies:

- Fibrous gingival hyperplasia is the most common form in this category whereby the proliferations occurs as a result of chronic irritation which often stems from the presence of subgingival calculus. This causes an increase production of collagen which resembles scar tissue. This is an exuberant overgrowth of collagen and fibroblasts. The clinical appearance is a domelike growth with a smooth mucosal surface of normal coloration.
  
  Treatment plan is to perform a gingivectomy of the hyperplasia either with a scalpel or electroscapecel. This will reestablish the normal gingival contour and sulcus depth. Any calculus on the tooth or root surfaces which were originally covered by the excessive hyperplasia, should be scaled and then polished. Daily home hygiene with brushing and swabbing the tooth surfaces with antimicrobial oral rinses should be undertaken.

- Epulids are the most common benign masses of the oral cavity. They are focal connective tissue proliferations of the attached gingiva surrounding the teeth. They are also called peripheral fibromas. Peripheral ossifying fibromas contain focal areas of mineralization within a connective tissue matrix that involve the attached gingiva. The previously named fibromatous and ossifying epulids are collectively now called peripheral odontogenic fibromas since they stem from dental epithelial origin. The Acanthomatous ameloblastoma is a true localized neoplasia which invades the bone and moves adjacent teeth.

  Treatment plan: Since most clients prefer to have the lump removed with one anesthetic procedure, and the type of mass is often unknown at the time of surgery, performing an enbloc resection of the tooth, bone and at least 1 cm of normal tissue would be a logical approach. Alternatively for a two anesthetic procedure, a 3–5 mm punch biopsy taken from the center of the lesion should be done. Care must be taken to go full depth to bone in order to give the pathologist an adequate sample especially in the case
• Proliferative Periosteitis is a condition that is seen from stimulation by a low-grade infection causing the periostium to become reactive. The bone that is produced is less dense and is deposited in a layered pattern of thin bone which lacks a cortex. This type of bony swelling is seen often in relation to the feline’s canine tooth which is undergoing root resorption. A large subgingival bulge of porotic bone is seen on exam. 
Treatment plan: Surgically extract the affected tooth and remove the bulge of reactive bone which impedes the flap closure of the area.

• Eosinophilic granuloma is a condition of unknown etiology which affects both cats and dogs although in felines it is a fairly common lesion. The eosinophilic granuloma can be single or multiple in its expression. Most common sites are the roof of the mouth, tongue and lips. Sublingual presentations are often seen. They usually are non-painful although they can become infected and ulcerate. They appear as a raised lobulated yellow tinged pink mass.

Since allergic phenomena might be the trigger mechanism for lesion development, flea control, plastic bowl removal, food trials and intradermal testing should be considered. Food allergy and food storage mite allergy can simultaneously be tested for by feeding a hypoallergenic canned diet for four to six weeks. Antibiotic therapy should be initially attempted although only a small percentage of the animals respond in this in the authors experience. If utilized than Clavamox 10 mg/lb bid, cephalexin 10 mg/lb bid or Antirobe 5 mg/lb/day should be trialed for 2-4 weeks.

Treatment plan: If after the above allergic phenomenon has been ruled out and treated Intralesional injections with Depomedrol can bring them under control with subsequent management with methylprednisolone orally to maintain remission. Initially a dosing of 2-4 mg daily should be tapered over a few weeks until resolution is seen. Alternatively 20 mg of Depomedrol i.m. can be given q2weeks until the lesion is in remission. Occasionally resection of the mass followed by corticosteroids orally can maintain remission. Cyclosporine has been proven effective in the treatment of the complex although it is off label. Doses of 2-5 mg/kg daily and then tapered once the response is visible. Chronic use of cyclosporine has been documented to cause a fatal toxoplasmosis.

Malignant neoplasia

The third or 4th most common area to find malignant lumps is the oral cavity. These can originate from either the dental tissue odontogenically (Acanthomatous ameloblastomas) or involve superficial structures of the epithelium like the Squamous Cell Carcinoma (SCC) or of the mesenchymal origin: Fibrosarcoma, Malignant Melanoma, Osteosarcomas. Based on the TNM-classification (primary tumor, regional lymph node, and distant metastasis) a patient can be classified into 4 clinical stages I-IV. Stage IV is prognostically the worst. An attempt at curative therapy during the initial stages might involve en bloc surgical resections with a 1.5 -2cm margin in combination with radiation therapy of 3-5 fractions delivered weekly for 3 weeks as in the case for Fibrosarcomas.

Megavoltage radiation therapy should be considered where surgery alone will not be curative. Specifically where tumor free margins are not obtainable in the caudal oro-pharynx
or maxilla, radiation therapy can extend the margins after surgical debulking. Complications of radiation are usually acute and transient. Caudal mucositis, alopecia, possible dehiscence of the surgical site can be expected with this treatment. In cats and dogs, stomach tube placement and post therapy nutrition is essential post caudal tumor radiation due to the above side effects.

- Fibrosarcomas are frequently encountered in small breed dogs ages 7-10 years and 4-5 years of age in large breeds. German shepherds, Golden Retrievers and Labradors are the most commonly affected breeds. Usually they are a slow growing mass that appears almost benign in it’s growth pattern. However, a distinct entity of this group, is the histologically low-grade biologically high grade fibrosarcoma seen in Golden Retrievers. These have a very rapid growth rate with local aggressive invasion and a higher metastatic potential. Cats are not as commonly affected by the FSA. This tumor is a mesenchymal fibrous connective tissue mass characterized by immature proliferating fibroblasts. They are extremely locally invasive with a high rate of return at the surgical site. They infrequently metastasize. The maxilla is more often affected than the mandible and the rostral maxilla has a higher frequency than the caudal area. Often its characteristics appear benign. A smooth lump that can be mobile in the submucosal area is the most common appearance. When this is detected an early biopsy obtained below the mucosa if using a punch should be taken for evaluation. Treatment plan is for a wide resection including the underlying bone and overlying skin should be undertaken. Depending on margins, adjunctive radiation therapy should be considered to extend the tumor free margin.

- Squamous Cell Carcinoma (SCC) is the most common oral malignancy in cats and is considered the second most common in dogs that are 8-10 years of age. Usually the gingiva and buccal mucosa are the preferred sites for the SCC. In cats and dogs sublingual presentations are not uncommonly seen. The SCC is locally aggressive and regional and distant metastasis is more common later in the progression of the disease. The SCC appears as an erosive corrugated pink to red fleshy mass. As the tumor progresses it invades the bone and causes lysis and tooth mobility and resorption. In general rostral mandibular SCC has a more favorable prognosis than the other affected areas. A variant of the SCC is seen in young dogs as histologically an “invasive papilloma” or other pathologist read it as a papillary SCC. This mass should be considered aggressive and excised with good clean margins. SCC in early cases with a rostral location can be cured with wide local excision. Rostral lingual SCC should be treated with a partial glossectomy. A combination of cisplatin and piroxicam appears to have antitumor effects in canine SCC. The tumor is radiation sensitive.

- Oral Malignant Melanoma (OMM) is considered the most common oral malignancy in older dogs between 9-11 years. Cocker spaniels, German Shepherds and other canines with heavy pigmentation of the oral mucosa are highly represented. The biologic behavior pattern of the OMM is focal invasion of the mucosa and underlying dental and bone tissue with early metastasis to the regional lymphnodes and lungs. It can appear in any area of the oral cavity’s mucosa, mucoperiosteum, or dorsal portion of the tongue. Classic dark pigmentation of the surface as well as amelanotic pink
to red tumor surfaces is often seen. Microscopic determination of the amelanotic melanoma can be challenging for these round cell tumors since their characteristics are shared with other masses like the lymphomas and carcinomas. Often of benefit is a immunohistochemical confirmation.

Treatment plan: Large en bloc surgical resections of 1.5-2 cm margins are considered the first line therapeutic approach. Radiation should be considered in areas where the tumor resection is incomplete or palliative. Cisplatin and Piroxicam has shown antitumor activity against canine OMM. The canine melanoma xenogenic DNA vaccine contains a human tyrosinase which is a glycoprotein essential for melanin synthesis. The canine tyrosinase on the other hand is present on the dog’s melanocytes and therefore not considered by the dog’s body as foreign and therefore incapable of generating an immune response. Using the xenogenic Tyrosinase therefore mounts an antitumor response. This vaccine is considered essential for promoting a longer survival rate. A local remission by surgical excision either followed by the vaccine or the vaccine initially started followed by surgery is considered optimal. Initial treatment requires 4 doses at 2-week intervals followed by booster doses at 6-month intervals.

Osteosarcoma (OSA) of the dog is commonly encountered however in cats it is less common. Usually large breed dogs ages 7-10 years old are more represented. The oral OSA appears locally aggressive but slower to metastasize than the above tumors. Its clinical appearance varies from having a surface that is smooth to that of ulceration. The prognosis for dog’s with oral OSA compared to appendicular OSA is better due to the lower metastatic potential. The mandible involvement has a more favorable median survival rate of 14-18 months versus the maxilla which is 5-10 months. The more rostrally located the mass is, the more favorable the longevity. Usually it is due to local reoccurrence versus metastasis that the animals are euthanized. An aggressive surgical approach with wide margins is the best solution.
Odontogenic tumors: occurrence in a private veterinary dental practice

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Oral tumors are relatively common in dogs and cats, but may not be noticed at an early stage by pet owners. Oral tumors may be benign or malignant. A special group of benign tumors commonly seen on the oral cavity are odontogenic tumors. Essentially, these are tumors that arise from tooth forming tissues. Not all of the embryological tissues that form the tooth bud undergo apoptosis when their job of tooth formation is done. Remnants of odontogenic epithelium remain in the periodontal ligament and gingiva for life. In the gingiva, they are called epithelial rests of Serres and in the periodontal ligament they are known as the rests of Malassez. It is these tissue remnants that contribute to the formation of most odontogenic tumors.

The clinician must remember that not all enlargements of the oral tissues are malignant. Non-neoplastic enlargements include gingival hyperplasia, localized infections, and collections of saliva from damaged ducts of salivary glands. Benign tumors do not spread to other parts of the body and generally grow more slowly than malignant tumors although they can be locally destructive. Malignant tumors invade the adjacent tissue and can spread to other parts of the body. Not all tumors appear like a “typical” visible growth. Some present as non-healing, ulcerated areas. While the visual appearance and location of an oral growth can often give clues as to its identity, confirmation of the diagnosis (and thus an accurate prognosis and identification of appropriate treatment) requires biopsy. All oral growths should be investigated. A “wait and see” approach is not in the patient’s best interest.

Radiography is an essential part of assessing the tumor characteristics, in particular the extent of the tumor and the presence of bone involvement. Intraoral dental radiographs are indicated in cases of suspected oral cancer. Skull radiographs often do not aid in the diagnosis because of super-imposition of the contralateral structures. Other advanced diagnostic imaging techniques may be indicated in some cases. Tumors located in the back of the mouth, or adjacent to the nasal passage often require computed tomography (CT) to determine the extent of the tumor.

The radiologic findings associated with oral tumors are often subtle and non-specific. Careful evaluation of radiographs may make it possible to associate different patterns with certain tumor types, and/or suggest a benign or malignant (aggressive) lesion. Good diagnostic imaging is especially important in correctly planning a surgical procedure. The
type of tumor cannot be determined accurately based on radiographs alone, and biopsy is always required for definitive diagnosis. Proper management of a patient with an oral tumor starts with accurate diagnosis. A definitive diagnosis is made with histopathologic (microscopic) assessment of a biopsy from the tumor. Biopsy may be incisional (taking a small portion for microscopic examination, with no attempt to remove all of the swelling) or excisional (surgery to remove the tumor completely). If there is a palpable abnormality in the regional lymph nodes biopsy or needle aspiration of the abnormal lymph node is recommended.

For most oral tumors, surgical removal offers the best chance of cure. For aggressive oral tumors surgery may need to be radical (removal of part or all of a jaw) in order to provide the best chance for complete removal of the tumor. Dogs tolerate radical surgery very well. Feline patient must be carefully selected if radical excision is planned for oral tumors. Because there are so many types of oral tumors that can occur, each with a different prognosis, management by veterinary specialists with knowledge of the teeth and oral cavity along with a veterinary oncologist will provide optimal outcome. The key to successful treatment is early detection of the growth. Owners that are in the habit of brushing their pet’s teeth every day are more likely to note changes in the mouth earlier than those that do not brush. For pet owners not brushing daily, a weekly oral inspection is recommended. Annual oral examination should be part of the physical examination by the primary care veterinarian.

A recent JAVMA article by Fiani, et al. shed some interesting light on some of the more common “benign” odontogenic tumors. They reported on evaluation of oral tumors that were diagnosed as either: canine acanthomatous ameloblastoma (CAA)(previously called acanthomatous epulis); peripheral odontogenic fibroma (POF)(previously called a fibromatous epulis; and focal fibrous hyperplasia (FFH) (previously called gingival hyperplasia). The study reviewed the breed, age, reproductive status, and location of these lesions in the oral cavity of each case. Other types of odontogenic tumors were not evaluated due to their relative very low incidence in dogs.

The article does try to clarify some terminology. The term “epulis” is simply a descriptive term of any gingival enlargement and does not provide any information with regard to the histological appearance or pathologic nature. The POF is a benign process of odontogenic origin. FFH is non-neoplastic, reactive inflammatory tissue that enlarges in response to chronic irritation.

Clinical appearance of the CAA differs slightly from POF and FFH. CAA is commonly more ulcerated in appearance and as it enlarges will invade bone and displace teeth.

The most common location of the CAA was found to be on the rostral mandible. In general, the CAA is a benign tumor that has never been reported to metastasize, but it is locally invasive into surrounding bone and therefore treatment dictates excision of the mass with at least 1 cm margins of clinically and radiographically normal tissue. Clean surgical margins equates to an excellent prognosis with a very low incidence of recurrence (less than 2%). These tumors are also radiosensitive and those that cannot be resected surgically should respond well to radiation therapy. The POF and FFH behave similarly. Both were more commonly found in the rostral maxilla. The treatment of either is similar, excision and contour to a level of normal gingival attachment. Recurrence is common with incomplete excision, and if POF is the tumor involved, extraction of the involved tooth with alveoloplasty can prevent recurrence. In cases of POF, removal of the inciting cause, such as plaque and calculus can be of benefit. Simple excision is not advised with CAA.
Bell and Soukoup recently (J of Vet Dent Vol. 31, No. 4: 2014) reported further on the nomenclature and classification of odontogenic tumors in dogs and cats. Their review provides an in-depth look at the odontogenic tumors that have been reported animals based on histological appearance:

**Tumors of Odontogenic Epithelium (without odontogenic mesenchyme)**
- Ameloblastoma
- Amyloid-Producing odontogenic tumor
- Canine acanthomatous ameloblastoma

**Tumors of Odontogenic Epithelium (with odontogenic mesenchyme)**
- Ameloblastic fibroma
- Ameloblastic fibro-odontoma
- Feline inductive odontogenic tumor
- Complex odontoma
- Compound odontoma

**Tumors of Odontogenic Ecto-mesenchyme**
- Cementoma
- Cementifying fibroma

**Tumors derived from tissue of periodontal ligament**
- Fibromatous Epulis

**Cysts of the jaw**
- Dentigerous cyst
- Radicular cyst

**Tumor-like lesions**
- Gingival hyperplasia
- Peripheral Giant cell granuloma
- Inflammatory lesions of odontogenic and periodontal structures

**References**


Nomenclature and Classification of Odontogenic Tumors – Part II
Bell CM, Soukup, JW. JVD vol. 31, No. 4 2014, p.234.
Odontogenic cysts are uncommon in the dog and cat, are benign and form within the tooth-bearing regions of the jaws. They cause local destruction of bone and teeth. An inflammatory or developmental stimulus may be what induces islands of odontogenic epithelial remnants to proliferate. A genetic role has been implicated in the etiology of odontogenic cysts in humans as well as the sightings of cytomegalovirus in both inflammatory and non-inflammatory odontogenic cysts. The walls of the cysts have epithelium that resembles ameloblastic epithelium.

The differing types of odontogenic cysts have varying clinical and biological behaviours. Differentiating them is important to effective treatment planning. An epithelial tumour may appear cystic related to central tumour necrosis and fluid accumulation just as a cyst may undergo a malignant transformation. Histological and immunohistochemical markers of cell proliferation exist in providing information on neoplasia versus cyst for humans, which may also prove valuable for use in veterinary medicine. Odontogenic cysts in humans are classified according to their morphological appearance and site of origin, however, some odontogenic cysts in animals can’t be classified in this manner. Because of the rarity of odontogenic cysts in dogs and cats there is confusion in the understanding, study, terminology and classification of them. Improper identification thus occurs. Poulet et al in a 1992 paper recognized the importance of a collaborative effort between veterinary and human pathologists to detail a unified nomenclature for types of odontogenic cysts in order to reduce the confusion in terminology, and thus allow a clearer path to treatment planning for the practitioner.

Poulet et al published the results of a retrospective study from 1980 to 1990 where cases of oral masses were histologically examined. Canine and feline odontogenic cysts were found. Two had malignant transformation of the epithelial lining and invasion into adjacent bone. They were termed basi-squamous carcinomas. One keratocyst was identified which recurred after surgical excision. This study found the most consistent differentiating feature of the keratocyst case to be ortho or parakeratotic keratinization of the cyst lining’s squamous epithelium, producing keratin which filled the cystic lumen.

Wiggs and Lobprise discuss odontogenic cysts to include primordial cysts and gingival cysts. Primordial cysts were stated to be a result of the stellate reticulum of the enamel
organ degrading, are found early in life and in place of a tooth. Gingival cysts are divided into: newborn- occurring from remnants of the dental lamina, are along the alveolar ridge, are multiple and non-painful adult-arising from the dental lamina remnants, enamel organ, or epithelium and are associated with the periodontal ligament.

A more recent summary of odontogenic cyst classification in a table format, “Origin of Odontogenic Cysts”, has been published.

<table>
<thead>
<tr>
<th>Cyst Type</th>
<th>Stimulus</th>
<th>Origin of Epithelial Rests</th>
<th>Name of Rests</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dentigerous Cyst</td>
<td>Unerupted or Malformed Tooth</td>
<td>Enamel Organ</td>
<td>Reduced Enamel Epithelium</td>
</tr>
<tr>
<td>Radicular Cyst</td>
<td>Non-vital Tooth</td>
<td>Epithelial Root Sheath</td>
<td>Rests of Malassez</td>
</tr>
<tr>
<td>Odontogenic Parakeratinized Cyst</td>
<td>Unknown</td>
<td>Epithelial Connection Between the Mucosa and Enamel Organ</td>
<td>Rests of Serres (Dental Lamina)</td>
</tr>
</tbody>
</table>

Verstraete et al published a retrospective study between 1995 to 2010 and found odontogenic cysts in 41 dogs. 29 of these were dentigerous cysts, primarily noted in the brachycephalic breeds, 1 was a radicular cyst, 1 a lateral periodontal cyst, and 9 had similar histological features to the odontogenic keratocyst noted in humans and were found in the maxilla.

Dentigerous cysts are the most common of the odontogenic cysts in the dog and cat. They have been described further into eruption cysts or follicular cysts. Eruption cysts are as result of dilation of the normal follicular space around a tooth during eruption, noted with either a permanent or deciduous tooth. Follicular cysts are a result of a dilation of the follicular space around the crown of an erupted tooth. They are associated with impacted or embedded permanent and deciduous teeth and are most commonly noted with the mandibular first premolar tooth which has no deciduous precursor. Odontogenic reduced enamel epithelium (REE) is attached at the cementoenamel junction and encompasses the crown of the unerupted tooth. The REE is a protective layer for the tooth and normally is eroded from the erupted tooth through mastication. As the cyst expands, bone and teeth are destroyed with thinning of the cortical bone. Pathologic jaw fractures, external root resorption, and pulpitis may also result. Dentigerous cysts are not usually painful unless secondarily infected. There are 3 parts to the cyst: the central fluid-filled cavity, the thin cyst wall of connective tissue and non-keratinized stratified squamous epithelium which is 2-6 cell layers thick and may contain inflammatory cell infiltrates, and a connective tissue capsule. The pathogenesis of cyst formation is not certain however trauma has been considered to be the main etiological factor. One theory is fluid accumulation from pressure by the erupting tooth on an impacted follicle which obstructs venous outflow. This leads to a rapid fluid movement causing a separation of the REE from the crown.

REE cells proliferate and osteoclastic bone resorption mediators are released. Matrix metalloproteinases may be involved in the bone and collagen destruction augmenting the cyst’s damage to the surrounding tissues. Another theory is the spread of inflammation from a non-vital deciduous tooth to the follicle of the permanent successor increasing inflammatory exudates and cyst formation. This of course does not explain cyst formation in the most common location of the mandibular first premolar tooth.

Intraoral radiographic features are considered pathognomonic to include a usually unilocular, smooth-margined radiolucency that contains a tooth-shaped structure.
Dentigerous cysts can undergo malignant transformation to ameloblastoma or squamous cell carcinoma. Immunohistochemical findings suggest that chronic deregulation of cell death in the epithelial lining along with proliferation activity increases the risk of modification or mutation of the epithelial cells allowing the dentigerous cyst to undergo malignant transformation. As example, a case report was published of a 10 year old Staffordshire Terrier who developed a painful swollen mandible. Radiographically and clinically it appeared the swelling was from a dentigerous cyst formation with secondary infection. Histopathology revealed a squamous cell carcinoma. This emphasizes the importance of histopathology in obtaining an accurate diagnosis and thus in case management.

Radicular cysts, also termed by the World Health Organization (WHO) as apical periodontal cysts and periapical cysts, are an inflammatory cyst associated with a non-vital tooth. They occur infrequently in dogs and cats however are the most common odontogenic cyst in humans. A chronically infected, necrotic root canal including bacterial endotoxins, stimulates inflammation and the formation of a periapical granuloma. This induces the epithelial rests of Malassez (odontogenic remnants of Hertwig’s epithelial root sheath), that lie in the periodontal ligament at the tooth apex, to proliferate. This forms the epithelial lining of the cyst. Cellular by-products accumulate in the cyst lumen, and an increasing osmotic pressure draws fluid into the cyst leading to expansion. Radicular cysts are locally aggressive and have the potential for malignant change. Radicular cysts have been classified as:

a) True cysts- lined with epithelium and only an epithelial attachment to the root apex
b) Pocket cysts- sac-like epithelium communicating directly with the root canal.

Radiology cannot differentiate the two classifications thus a biopsy is most important.

Histologically the cyst is keratin rich. There is evidence of a foreign-body granulomatous inflammation with lymphocyte and plasma cell infiltrates into the epithelium.

Odontogenic parakeratinized cysts or odontogenic keratocysts (OKCs) are developmental cysts with an unusual propensity for recurrence. These cysts are the second most common type of odontogenic cyst, with no sex predilection and a wide age range of occurrence in dogs and cats. They span the roots of normally erupted teeth displacing them, however rarely is there root resorption noted. They are locally infiltrative. The cyst grows by bone resorption from fluid osmosis and cellular cytokine production. The epithelial connection between the overlying gingival epithelium and the superficial aspect of the dental follicle is the dental lamina. This gives rise to the rests of Serres. OKCs form from the rests of Serres. Radiographically, a well-circumscribed, usually unilocular radiolucency with smooth opaque margins is noted. The radiology findings are not diagnostic for OKCs and histopathology is always required to obtain a diagnosis. Both the lumen contents and the cell walls should be biopsied to identify free keratin with-in the lumen as the cell walls may only have limited keratin adhering to the cells. The WHO has proposed a name change to Keratocystic Odontogenic Tumour to more accurately reflect their neoplastic nature in man. The markers of biological proliferation indicate this is an aggressive tumour not a benign cyst. Verstraete et al found 9 cases of patients with “OKC-like” histopathological features as noted in humans, ie: some but not all features to be diagnostic of OKC in humans, and proposed the term Canine Odontogenic Parakeratinized Cyst (COPC). The Verstraete paper stated their cases had the following features consistent with OKC in humans:
The cyst walls were of nonkeratinizing-stratified squamous epithelium, of 6-10 cell thickness, with a parakeratotic surface and a thin, flat epithelial connective tissue interface.\textsuperscript{5,2} They did not have these following features however:

A hyperchromatic palisading cuboidal or columnar basal cell layer with high mitotic index and keratinaceous debris in the cyst lumen.\textsuperscript{5,2}

These cases were all found in the maxilla whereas in humans they are found in the mandible. A paper published by Watanabe et al also found an OKC in the maxilla of a canine patient.\textsuperscript{14} LaDouceur et al published a paper discussing an OKC in a feline patient.\textsuperscript{13} Tooth root resorption and mandibular location was noted in this patient.\textsuperscript{13} The histology report stated the cyst was lined by stratified squamous epithelium with luminal parakeratinization and a prominent palisading basal cell layer.\textsuperscript{13} The lumen contained ortho and parakeratotic keratin.\textsuperscript{13} In humans, quantifying the proliferative marker Ki-67, aids in prognosis for recurrence after surgery.\textsuperscript{13} This may have some value for veterinary patients diagnosed with OKC or COPC as Verstraete has proposed for nomenclature.

\textit{Treatment of odontogenic cysts includes:}

- Extraction of affected teeth
- Complete removal of the cyst wall
- Curettage
- Osteoplasty
- Bone regeneration
- Resection
- Monitoring radiographically on a regular basis for recurrence.\textsuperscript{1,2,12}

The owner may not notice oral swellings of odontogenic cyst formation if there are no clinical signs of oral pain and if the owner is not daily brushing the teeth. Thus oral examinations and full mouth radiographs obtained while performing regularly scheduled professional dental cleanings are important to recognize the development of these locally aggressive and destructive cysts. This presentation will discuss the diagnosis and treatment of a COPC of the rostral mandible.

\textbf{References}

Advanced omega wire technique for base narrow canine correction

Peter Stelzer

Base narrow canine teeth can cause severe injuries of soft tissue and neighbouring teeth. Therefore certain techniques (e.g. inclined plane or extension screw) have been established over the last decades. The aim of all those gadgets is to lateralize either one or two base narrow canines. After orthodontic treatment the canine tooth should not be in contact with any soft or hard tissue. Sometimes (e.g. severe overbite) it can be necessary to lateralize a lower canine distally of the upper canine. One of the problems is the hygienic handling of the gadgets. Very often food parts stick to the extension screw etc. Soft tissue inflammation of the palate gum can occur during treatment with an inclined plane device.

The new technique uses a wire made from a Nickel-Titanium alloy a so called memory wire. The preparation of the inside surface of the canine teeth starts with the enamel etching technique using phosphoric acid. Next step is the activation of the Nickel-Titanium wire. The activation starts with bending the wire so it is able to move the canines into the desired destination. After this the first layer of Dual curing PMMA is attached and dried with a light gun to the inside surface of the canines. Shortly later the wire is positioned and fixed in its working position. Several layers of Dual curing PMMA are attached for embedding the wire in plastics. The wire is now fixed on the inside of the canines coming down and crossing the oral floor to the opposite side. The result of the procedure is an activated wire which moves the canine tooth without any further activation. Another advantage is the hygienic situation with little opportunity of food to contaminate the device.

Depending on the orthodontic situation of the base canines the wire and the plastic is removed and the teeth are polished and fluoridated.
Introduction

Periodontal disease is a ubiquitous disease in canine and feline veterinary practice. Periodontal disease is loss of the periodontium (i.e., gingiva, periodontal ligament, cementum, and alveolar bone). General anesthesia and a thorough clinical subgingival evaluation and intraoral radiographs are required to assess, diagnose and treat periodontal disease. Periodontitis is active inflammation of the periodontium caused by the bacterial biofilm (plaque) and the associated host inflammatory response. It begins with the accumulation of the dental pellicle followed by first colonizing oral bacterial within hours of a clean tooth. The plaque biofilm matures within days. Mineralization of the plaque biofilm results in calculus.

Periodontal stages

Treatment plans can be designed based on the individual tooth stage as well as the overall periodontal stage (PD0-PD4) of the oral cavity (www.avdc.org). Periodontal pockets are clinical periodontal probing measurements greater than the normal sulcus (e.g., dog is 0-3 mm, cat is less than 0.5 to 1.0 mm). Periodontitis can result in gingival enlargements, gingival recession, and periodontal pockets. Periodontal pockets are a haven for gram-negative anaerobic bacteria and spirochetes in the subgingival plaque biofilm and planktonic bacteria in the gingival crevicular fluid. There are often combinations of periodontal pocket types as they are not mutually exclusive. Pseudopockets are created when the gingiva enlarges and the marginal bone remains at the appropriate level. Breeds such as Boxers and Collies have a genetic predilection for gingival hyperplasia. Common veterinary medications such as cyclosporine and amlodipine may cause gingival enlargement. Suprabony pockets occur when marginal bone loss exceeds gingival recession (the marginal bone is lost horizontally below the tissue). Intra(Infra)bony pockets occur when bone is lost vertically around a tooth. Infrabony pockets can be classified as one-wall, two-wall, three-wall, and four-walled (cup or crater) defects.
Treatment and management

To fully understand professional treatment options and home care products understanding periodontal disease reduces to two topics: 1) Plaque biofilm inciting host inflammation and 2) Periodontal pockets. Management of periodontal disease is not a once in a lifetime event for the patient but rather an ongoing program throughout continued life stages of the patient. The goals with periodontitis management are to stop the disease, minimize further attachment loss, and treat compromised teeth. A professional dental cleaning followed by daily home care is the gold standard to prevent and control periodontal disease.

Periodontal treatment and surgery

Periodontal pockets greater than 5 mm (with breed variations), periodontal probing depths beyond the mucogingival junction, stage 2 and 3 furcation exposures, intrabony pockets, gingival clefts, mobile incisors, loss of gingiva, and periodontal trauma require periodontal surgery. Stage 4 periodontal disease is sometimes best treated by exodontics.

Periodontal treatments

Periodontal surgery occurs with, and after, the oral cavity has had a thorough assessment, intraoral radiographs, and professional periodontal cleaning. Following periodontal treatment the process is not complete until a home care plan is recommended for the individual patient. 1) Pericuettical treatment – Following periodontal debridement, subgingival scaling, and root planing, commercial antibiotic gels may be placed in appropriate depth suprabony pockets based on manufacturer recommendations. 2) Open periodontal flaps with root planing – mucoperiosteal flaps are created to in order to visualize and expose the root surface. The root surfaces are root planed with curettes and an irregular periodontal bone is contoured. The periodontal flap is meticulously sutured back around the involved teeth. 3) Periodontal Splinting – mobile incisor teeth are open root planed, and a periodontal splint is placed on the teeth in order to anchor the individual single rooted incisors into one multi-rooted unit to stabilize the regional mobility of the incisors. 4) Gingivectomy/Gingivoplasty – Excessive gingival tissue is excised with an external bevel gingivectomy and contoured via gingivoplasty in order to remove pseudopockets. 5) Periodontal pedicle flaps – are created and moved over cleaned root and bone surfaces to treat gingival clefts and festoons by returning a larger band of gingiva on the tooth. 6) Osseous resective/subtractive surgery – some intrabony pockets, where there is still significant periodontal bone attachment, can be treated with open root planing, removing alveolar bone (osteoplasty), and soft tissue re-sutured around the tooth with an apically repositioned periodontal flap. 7) Osseous additive surgery (Guided tissue regeneration) – is the new formation of periodontal tissues (cementum, periodontal ligament, and alveolar bone) that had been destroyed from periodontitis. Regeneration, reconstitution of lost tissue, is differentiated from periodontal repair, healing of the periodontal wound/defect by tissue that does not fully restore the normal histological architecture. GTR involves open periodontal flaps, +/- debated root surface preparations (e.g., tetracycline, citric acid, EDTA), +/- grafting materials, +/- biological modifiers (e.g., growth factors, cytokines), and periodontal barrier membranes.

References available on request
Quantification of dental plaque using quantitative light-induced fluorescence

Corrin Wallis\textsuperscript{a}, Yadvinder Gill\textsuperscript{a}, Alison Colyer\textsuperscript{a}, Ian Davis\textsuperscript{a}, Judi Allsopp\textsuperscript{a}, Gleb Komarov\textsuperscript{b}, Susan Higham\textsuperscript{b} & Stephen Harris\textsuperscript{a}

Periodontal disease is the most widespread oral disease in dogs and dental plaque is an important aetiological factor. Evaluating the quantity of plaque on the tooth surface is therefore essential for determining the efficacy of oral hygiene products. Methods for plaque quantification have been developed for use in both human and animal dental research but many have poor resolution and are subjective. The aim of this work therefore was to evaluate Quantitative Light-induced Fluorescence (QLFTM) as an alternative method for determining plaque coverage on dogs’ teeth.

In a series of studies using both conscious and anaesthetised dogs, QLFTM showed good inter-photographer reproducibility (CV of 3.2\% for undisclosed teeth and 8.5\% for disclosed teeth). The QLFTM software accurately identified areas of plaque as demonstrated by comparison to the results from five human scorers manually marking plaque on QLFTM acquired images (p=0.1). There was good agreement between the results from QLFTM analysis and those from the modified Logan and Boyce method in a product efficacy trial using a clean tooth model. Furthermore, a retrospective power analysis (90\%) established that fewer dogs were required to measure the same difference in plaque accumulation between treatments.

QLFTM is a reliable method for measuring dental plaque in dogs with the added advantage that it is not subjective and requires fewer animals. The use of fewer animals supports one of the guiding principles underpinning the humane use of animals in scientific research; namely reducing the number of animals used to a minimum to improve animal welfare.
Recognition of effectiveness of dental hygiene products requires comparison of the plaque and calculus scores in control and test groups from trials conducted under controlled conditions. These data are used to determine whether the performance of the test group compared with the control group meets or exceeds the pre-set standards established by the Veterinary Oral Health Council. Products that meet or exceed the VOHC standard are awarded the ‘VOHC Accepted’ Seal.

Historically, subjective (non-linear observational) scores have been used.

At the Veterinary Dental Forum in 2015, a series of presentations described the history, current use and limitations of subjective scores. Objective scores using electronically-generated data were also discussed.

This presentation will summarize the Monterey discussions, and suggest how further progress can be made.
Four canine mandibular canine fracture cases will be presented. The repair methods, success, and failure will be discussed.
Perioperative considerations for the dental and oral surgical patient

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Normal versus abnormal

The unpaired incisive papilla immediately caudal to the maxillary first incisors, the apparently ‘swollen’ mucosa of the most rostral hard palate, the small linguomolar salivary gland lingual to the mandibular first molar (in the cat), the distinct lateral frenulum attaching the lower lip to the gingiva immediately caudal to the mandibular canine tooth, and various presentations of normal pigmentation may be confused with oral pathology.

Risk of anesthesia

The overall risk of anesthetic and sedation-related death (occurring within 48 hours of use of sedation or anesthesia) is approximately 0.1-0.2% in healthy and 0.5-2% in sick dogs and cats. This risk can be reduced by means of endotracheal intubation, fluid administration, pulse and pulse oximetry monitoring, and greater patient care in the postoperative period.

Wide mouth opening

Prolonged wide mouth opening in cats reduces maxillary artery blood flow, which can result in temporary or permanent blindness and/or other neurological deficits post-anesthesia. Placing 30 mm or 20 mm plastic gags between maxillary and mandibular canines enables adequate mouth opening for most oral procedures in cats.

Tracheal injury

Overinflation of an endotracheal tube cuff risks tracheal rupture in cats particularly when endotracheal tube movement during oral manipulations and turning over of the patient are to be expected. A secure airway can be accomplished by filling the endotracheal tube cuff with 1.6 +/- SD 0.7 mL.
Oral edema

Oral edema can be due to overzealous pharyngeal packing, rough tongue manipulation, and excessive elevation of alveolar mucosa on the lingual aspect of the mandible. Breathing could be compromised during recovery from anesthesia, and management of this complication varies from a single injection of dexamethasone at the end of the procedure to performing an emergency tracheostomy.

Iatrogenic trauma

Excessive ventral pulling of the tongue in the cat prior to intubation can cause puncture of the sublingual or lingual mucosa by the mandibular canine tooth. Pathologic mandibular fracture can occur in dogs with severe periodontal disease during relatively routine manipulation (when opening the mouth for intubation, placing a mouth prop or gag, and during tooth extraction).

Corneal damage

Corneal damage can occur during prolonged anesthetic episodes. Thus, frequent application of an eye lubricant, while under anesthesia, is recommended. One can also protect the eye by taping a piece of gauze over the closed eye lids when performing procedures near the eye.

Hypothermia and hyperthermia

Hypothermia is of particular concern in dentistry and oral surgery patients when water is used to cool power instruments or to rinse debris from the mouth. On the other hand, opioids, in particular hydromorphone, have been reported to cause an increase in body temperature in cats.

Oral bleeding

Diffuse nasal mucosal bleeding may be stopped by irrigation with a mixture (0.05-0.1 mL/kg in cats; 0.1-0.2 mL/kg in dogs) of 0.25 mL phenylephrine 1% and 50 mL lidocaine 2%. Other means of hemostasis include vessel ligation, digital pressure, refrigerated rinsing solutions, astringents, bone wax, cellulose meshes, gelatin powder/sheets, polysaccharide powder, collagen powder/sheets, thrombin in a gelatin matrix, fibrin sealants, and cyanoacrylate tissue adhesives. A pressure bandages or temporary ligation of an artery is required for uncontrolled bleeding that could lead to hypovolemic shock. Volume replacement is accomplished with crystalloids, colloids or blood products.

Emphysema

Emphysema after use of air-driven equipment is rare and usually resolves within a few days. Blowing air or air/water spray into submucosal tissues, particularly after deep dissection of large mucoperiosteal flaps, should be avoided. Blowing of air or air/water spray into alveolar sockets, onto denuded bone or bleeding tissues can cause air emboli and is strongly discouraged.
Wound breakdown

Wound dehiscence is usually due tension on suture lines. Other causes include tissue infection or necrosis when the surgery resulted in additional trauma or caused loss of vascular supply. An oral wound that does not heal for 7 days or longer following surgery should be biopsied to rule out neoplasia.

Bacteremia

Temporary (short-lived) bacteremia secondary to an oral condition occurs frequently in patients with periodontal disease and has been described in cats and dogs during and after dental cleaning and tooth extraction. This should not be an indication for the perioperative use of systemic antibiotics in the otherwise healthy patient. Systemic infection as a result of poorly performed tooth extraction has only anecdotally been reported.

Use of antibiotics

Inappropriate prescribing of antibiotics may be due to the demand from the patient owner, time pressure on the veterinarian, financial considerations, and diagnostic uncertainty. There is a tendency to use antibiotics as part of the management of any animal with oral disease, although there is no justification for this. One should distinguish between the use of antibiotics for the treatment of specific oral disease (e.g., severe stomatitis, osteomyelitis, etc.) in addition to surgical management of that pathology and the use of antibiotics to prevent or reduce bacteremia during treatment of a dental or oral surgical patient that has a co-morbidity (e.g., organ disease, endocrine pathology, immunodeficiency, etc.).

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Surgical correction of oronasal fistula: When is it a beneficial procedure?

Tomáš Fichtel, Jitka Zimková & Janalík

Introduction

Periodontal disease (PD) is one of the most common conditions in canine and feline dentistry. Late stages of the PD raise the risk of severe local and systemic consequences. Oronasal fistula is a typical local complication. Due to this condition, thorough examination of the oral cavity should not be omitted in patients suffering from chronic rhinitis.

A few therapeutic options were described; the approach should be always surgical. However, there is not an universal technique. Our research project studied two methods of oronasal fistula closure regarding healing of the wound and rate of complications (dehiscence). The principal goal was to make the choice of surgical approach easier.

Material and methods

Two groups of dogs treated for progressive PD were compared. The treatment took place between 1st February 2012 and 1st February 2013 at the small animal clinic of the University of Veterinary and Pharmaceutical Sciences Brno (Czech Republic). All patients were treated by the first author. Weight, age, sex or breed were not exclusion criteria; however, all included dogs were in small breed category. Pre-anaesthetic examination did not reveal any concurrent disease which could affect wound healing. The two groups were:

**Group 1:** Patients diagnosed with PDI 4 in the area of maxillary canine teeth (104, 204), plus oronasal fistula (ONF) along the lingual root surface. The ONF was closed surgically immediately after the extraction of affected teeth.

**Group 2:** Patients, where ONF was already present at the time of first examination and treatment but the surgical correction was postponed for 4 weeks (allowing healing of the surrounding tissues).

Anaesthesia and medications: Premedication with medetomidin and butorphanol (Domitor© and Butomidor©) in standard dose. Induction with propofol (Norofol©), consequent endotracheal intubation. Anaesthesia maintained with air-O2-isoflurane. Doses
of medicaments and percentage of inhalation agent were not evaluated. The dogs were medicated with amoxicillin-clavulanate (Synulox© 17,5 mg/kg s. c.) and NSAID meloxicam (Metacam© 0,02 mg/kg s. c.).

Surgical procedure: No disinfectant or any other agents were applied topically prior to the procedure to avoid any affection of the wound healing. Saline isotonic solution was used to rinse during the procedure.

Group 1 – Affected teeth were extracted using a closed technique without surgical preparation of surrounding tissues. Immediately, the ONF was closed surgically.

Group 2 – Surgical closure of persistent ONF

Surgical technique was the same for all patients – the „single-flap technique“. The initial step was to prepare a mucosal flap. It overlapped the wound by 2-4 mm in every direction. The incisions were divergent to ensure wider base of the flap. The flap was released using a periosteal elevator and Metzenbaum scissors. HM 1SQ 023 bur (Messinger©, Germany) mounted in turbine (300 000 rev. per minute) was used to debride the tissue margins. The wound was rinsed periodically with sterile saline solution. In all cases, surgical closure was provided with single-layer suture; absorbable monofilament material (glycolide-ε-caprolactone, Monolac 5/0, Chirmac©) was used in a single interrupted pattern. Postoperatively, the dogs were medicated with amoxicillin-clavulanate (Synulox© 20 mg/kg q12h p. o.) for 5 days. Check-up was performed after 4 weeks; in case of dehiscence, its percentage in individual cases was recorded.

Statistical assessment included comparison of overall success rate in groups 1 and 2. Chi-quadrate (χ²) test was used to evaluate the difference between groups regarding the presence/absence of wound dehiscence; percentual length of dehiscence was compared using non-parametric Wilcoxon test. Average percentage of dehiscence, its median, and modus were computed for both groups.

Results

29 ONFs in 24 dogs were included in the study. 16 fistulas were assigned to group 1, 13 to group 2. In group 1, dehiscence occurred in 75 % of cases, 25 % healed uneventfully. In group 2, 30,77 % fistulas showed dehiscence, 69,23 % of cases healed without complication (Graph 1).

Percentual length of wound dehiscence was highly significantly different between groups 1 and 2 (p = 0,011) with bigger dehiscence in group 1 (Graph 2).

Discussion

ONF forms due to progressive heavy damage to periodontal tissues in the upper jaw. Treating patients in such state means handling of tissues severely influenced by inflammation. Wound infection significantly alters inflammatory phase of healing, thus endangering the suture as the probability of dehiscence rises (Amsellem 2011). Most authors recommend surgical closure of ONF at the time of the diseased tooth extraction (Smith 2000, Bellows 2004, Pavlica 2006, Niemiec 2010). In contrary, Hedlund a Fossum (2007) suggest to close the fistula several weeks after the tooth extraction. According to Pavlica (2006), immediate ONF closure should be regarded as a generally valid rule. Caiafa (2007) supports the approach of extraction and closure with mucosal flap; in addition, his recommendation is
to medicate the patient with clindamycin 3-5 days prior to the procedure. Patients in our study were given amoxicillin clavulanate which is used routinely in this indication at our workplace. Doxycycline with its combined antimicrobial and anti-inflammatory effect could be considered as an option (Sreenivasan and Gaffar 2007). Its inhibition of collagenase might support the wound healing. Previous study by Fichtel and Vincencová (2012) investigated the influence of doxycycline on periodontal pocket healing in dogs; the results proved that the drug use helped to reduce pocket depth. Diseased tissues are infiltrated with monocytes, lymphocytes, and neutrophils. These produce substances like proteolytic enzymes and reactive forms of oxygen, further damaging the tissues (DeBowes 2010) and inhibiting the collagen synthesis (Caiafa 2007). Therefore, surgical wounds in such tissues may not heal appropriately.

In our study, wound healing got significantly more complicated when treated immediately (group 1). When the closure was delayed (group 2), ONFs healed more easily. Statistical assessment proves that the difference in occurrence of complications was highly significant.

**Graph 1.** ONF dehiscence rate in groups 1 and 2 4 weeks after the surgery.

**Graph 2.** Percentual length of wound dehiscence in groups 1 and 2.
Moreover, even the percentual length of dehiscence was smaller in the second group.

**Conclusion**

Based on our results, it can be stated that it is safer to delay ONF closure until the inflammation is resolved and the tissues heal. This approach seems to be a way to ensure undisturbed healing and minimize the possibility of dehiscence. However, there are certain limitation for practical application – the risk of second anaesthesia may not outweigh the benefits in some cases. Moreover, two-step procedure is more financially demanding from the owner’s perspective.

**Literature**


Flap design: a tension relieving process

Loïc Legendre

Introduction

Flaps are used in dentistry everyday from extraction flaps, lateral sliding flaps, coronally or apically positioned flaps, pedicle flaps. Positioned and pedicle flaps are used for oronasal fistula repair, for palatal trauma correction, for ectomy closure, as part of bone augmentation and crown lengthening procedures. For each of those procedures there exist variations dictated by exact presentation and by individual preferences. Flaps are supposed to improve visualization, to be sutured on top of bone not on top of the defect, to be tension free and to be designed to keep patient morbidity to a minimum. If there are variations, some fulfill the requirements better than others.

Review

One can create an envelope flap, a flap with a single vertical release incision, or a flap with two vertical releasing incisions. This is the first choice to make, the second is to decide where exactly the releasing incisions will be. If the goals of the flap, listed above, are to be kept in mind, the results will vary from tooth to tooth and from procedure to procedure. For example, the maxillary canine tooth requires a buccal osteoplasty thus the flap has to offer good visibility. It follows from this that the flap should have 2 vertical releases and should extend to the apex of the canine in case the area needs to be explored. Thus, the best flap design can be extrapolated for each tooth and each procedure.
Interceptive orthodontics (IO) occurs when the problem has been already manifested. Any procedure that may include minor local tooth movement or extraction which eliminates or reduce the severity of malocclusion in the developing dentition. Interceptive orthodontics is often the first phase of orthodontic treatment. It may not prevent the need for braces, retainers, or other orthodontic techniques in later years but it can minimize the second phase of treatment. The most common indications of IO include: persistent deciduous dentition, crowding of the teeth, uneven development of maxilla and mandible, impaction of the teeth caused by mechanical reasons and supernumerary teeth.

Interceptive orthodontics involves the selective extraction of any deciduous teeth that would impede the development of a proper bite. The general rule is to extract the teeth from the short jaw. For a class 2 malocclusion, extraction of the deciduous mandibular canines and incisors will alleviate the dental interlock. These procedures do not alter the animal’s genetic make-up nor do they make anything happen. Rather, they allow the animal to express its full genetic potential by removing any mechanical impediment to growth. Owners and breeders should be cautioned that, even if the animal undergoes successful treatment, it did require intervention and has the potential to pass the malocclusion on to offspring.¹

To maximize the benefit of interceptive orthodontics, extractions should be performed as early as possible. The hope is that the jaw length relationship will normalize before the permanent teeth erupt and recreate dental interlock. The more time between deciduous tooth extraction and permanent tooth eruption, the better the chances of success. However, the owners should be made aware that most animals with jaw length discrepancies at eight weeks of age will not ‘go normal’, regardless of treatment, and there will very likely be orthodontic problems when the permanent teeth erupt. The removal of the abnormal interlock does not make the short jaw grow fast and catch up, rather it allows the animal to express its full genetic potential. If the programming is faulty, interceptive orthodontics will not change that. A second (and much more predictable) benefit of interceptive orthodontics is that it immediately relieves the oral trauma and pain associated with abnormal tooth-to-tooth or tooth-to-soft tissue contacts. This on its own is sufficient cause to recommend the

In deciding if interceptive orthodontic extraction is required (and if so which tooth or teeth should be extracted), one should be familiar with the normal location of the permanent teeth. Except for the permanent maxillary canines (which erupt mesial to the deciduous), permanent teeth erupt lingual to their deciduous counterparts. Besides relative positioning, other clues to differentiate deciduous teeth from permanent teeth include comparative whiteness, diminutive size, and degree of root development.

When performing pure interceptive orthodontics, the simple rule is to extract the teeth on the jaw that needs to grow. However, recent texts recommend extracting any deciduous tooth that is or is likely to become a hindrance to movement, while not extracting teeth that may be creating a favorable dental interlock. Favorable dental interlocks most commonly occur with class III malocclusions where the mandibular canines are close but still distal to the maxillary lateral incisors.

Among other procedures which may act as interceptive orthodontics are also extractions of the permanent dentition, operculectomy and odontoplasty. All these actions can allow the patient to correct itself during the growth phase. IO by changing the trajectory of permanent teeth eruption can improve relations and position of erupted teeth. Often such procedures are combined with minor teeth movement.

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“We shall be best at animal dentistry and nothing else”
Tricks for the repair of challenging congenital palate defects in dogs

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Introduction

Congenital defects of the formation of lip and palatal structures may be inherited or result from an insult during fetal development (intrauterine trauma or stress). Clefts can occur if the intrauterine insult (trauma, stress, corticosteroids, antimitotic drugs, nutritional, hormonal, viral, and toxic factors) occurs at a very specific time in fetal development (25th to 28th day in dogs). Incomplete fusion of maxillofacial structures during fetal development may result in uni- or bilateral clefts of the upper lip, lateral area of the most rostral hard palate, midline of the hard and soft palate, and the lateral area of the soft palate. Rarely, the soft palate may be markedly reduced in length (hypoplasia). The growth of the palatine portions of facial bones in broad-headed fetuses may not always successfully compete with the growth of the head, and thus brachycephalic breeds tend to be at higher risk of developing defects of the primary and secondary palates.

Cleft lip

This congenital defect of the primary palate appears as defect of the lip +/- most rostral hard palate and could be associated with abnormalities of the secondary palate. Unilateral defects occur more commonly on the left side. Except for being externally visible, cleft lips rarely result in clinical signs beyond mild local rhinitis, and repair may be performed for esthetic reasons. Simple sliding procedures rarely are successful because there is no connective tissue bed to support the flaps. The most rostral hard palate and the floor of the nasal vestibule are reconstructed by creating overlapping and advancement, rotation or transposition flaps of both oral and nasal tissue or flaps that are harvested from oral soft tissue only. Removal of one or more incisors and also the canine tooth on the affected side 6-8 weeks prior to definitive surgery will facilitate flap management. Lip repair is completed by reconstructive cutaneous surgery to provide symmetry.

Cleft palate

This congenital defect of the secondary palate is almost always in the midline of
the hard palate and usually associated with a midline soft palate abnormality. Soft palate defects without hard palate defects may occur in the midline or are unilateral; occasionally, soft palate hypoplasia may occur. An association between congenital unilateral defects or hypoplasia of the soft palate and middle ear pathology was reported in dogs. According to the stage of development and the severity of the cause, other physical or neurological abnormalities may be present. Clinical signs and history of patients with cleft palate include failure to create negative pressure for nursing, nasal discharge (milk coming from the nares during or after nursing), coughing, gagging, sneezing, nasal reflux, tonsillitis, rhinitis, laryngotracheitis, aspiration pneumonia, poor weight gain, and general unthriftiness. The prognosis without surgical repair is guarded because of the continued risk of aspiration.

**Timing of surgery**

Management of patients with defects of the secondary palate usually requires nursing care by the owner, which includes transoral tube feeding to avoid aspiration pneumonia. Most procedures for correction of defects of the secondary palate are performed on animals between 3 to 4 months of age. Surgery prior to 2 months of age is challenging due to the presence of delicate and friable soft tissues in very young animals. Postponing surgery until after 5 months of age may result in a wider cleft, as the animal grows, and in compounded management problems, which are not desirable. Because clefts of the primary palate are associated with less severe clinical signs, the operator could postpone surgical repair until after eruption of the permanent incisors and canines, which may need to be removed if they prevent proper flap management.

**Overlapped flap**

This technique is preferred for repair of congenital primary and secondary hard palate defects. There is less tension on the suture line, which is not located directly over the defect, and the area of opposing connective tissues is larger, which results in a stronger scar. This technique provides more reliable results for repair of hard palate defects compared to the medially positioned flap technique (the latter is more useful for repair of traumatic midline clefts of the hard palate in cats). Incisions are made in the mucoperiosteum to the bone (full-thickness) along the dental arch about 1-2 mm away from the gingiva and to the rostral and caudal margins of the defect on one side, forming an overlapped flap, and at the medial margin of the defect on the other side, forming an envelope flap. Both flaps are carefully undermined with a periosteal elevator. The major palatine artery exits the palatine shelf of the maxilla about 0.5-1 cm palatal to the maxillary fourth premolar (more rostral in the cat than the dog) and must not be transected during flap elevation. When the artery is identified at the connective tissue side of the overlapped flap, careful dissection close to it will release it from surrounding tissue to accommodate the rotation of this flap. The envelope flap also is undermined with a periosteal elevator on its medial margin to create a pocket of space for the overlapped flap. The overlapped flap is inverted at its base, turned and secured under the envelope flap with horizontal mattress sutures so that large connective tissue surfaces are in contact. Granulation and epithelialization of exposed bone generally are completed in 3 to 4 weeks.
Medially positioned flap

The medially positioned flap technique may be utilized for very narrow congenital hard palate defects, but the author usually uses it only for repair of midline clefts of the soft palate. If used for narrow hard palate clefts, incisions are made at the medial edges of the defect and along the upper dental arch about 1-2 mm away from the gingiva on one or both sides. The mucoperiosteum is undermined with a periosteal elevator (avoiding injury to the major palatine arteries), and the now mobile flaps are slid together and sutured over the defect. The exposed bone next to the teeth is left to granulate and epithelialize. If the relieving incisions are long and gape, a lateral oronasal defect may occasionally result, particularly in narrow-nosed dogs. Another disadvantage is that the rostral aspect of the sutured defect has a tendency to break down. In the case of midline clefts of the soft palate, incisions are made along the medial margins of the defect to the level of the caudal end of the tonsils. The palatal tissues are separated with a blunt-ended scissors to form a dorsal (nasopharyngeal) and ventral (oropharyngeal) flap on each side. The two dorsal and the two ventral flaps are sutured separately in a simple interrupted pattern to the midpoint or caudal end of the palatine tonsils. Repair of unilateral soft palate defects is performed with or without removal of the ipsilateral tonsil. The tonsillectomy incisions can be extended rostrally to meet at the most rostral location of the soft palate defect and continued along the medial edge of the soft palate. The pharyngeal and palatal tissues are separated, and two dorsal and two ventral flaps are sutured separately in a simple interrupted pattern to the midpoint or caudal end of the contralateral tonsil. Treatment of congenital hypoplasia of the soft palate is challenging, but may be treated in similar fashion after bilateral tonsillectomy and extension and continuation of incisions into the rudimentary, uvula-like soft palate tissue. Dorsal and ventral flaps are created and sutured separately in a simple interrupted pattern to the midpoint or caudal end of the tonsillectomy sites.

Other techniques and combination of techniques

If an overlapping flap technique cannot be performed for repair of a cleft of the hard palate, a rotation flap made up of palatal mucoperiosteum and supplied by the major palatine artery on one side may provide a better alternative for repair of midline clefts of the hard palate than the medially positioned flap technique. A bilateral overlapping flap technique for a midline soft palate cleft has also been described. Bilateral buccal mucosa flaps (one rotated and the other rotated and overlapped) were also used to repair soft palate hypoplasia in dogs. A combination of bilateral pharyngeal advancement flaps and one overlapping hard palate flap for treatment of hypoplastic soft palate was described in a cat.

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The red-necked wallaby was presented with acute onset of pain, inappetence and mandibular swelling, which could be typical clinical signs of oral necrobacillosis. Oral necrobacillosis could be associated with signs of systemic illness and the prognosis is poor. The case of necrobacillosis very often ends in euthanasia. In this case teeth extraction and treatment were successful, and the patient is without clinical problems or recurrence 6 months after the treatment.

Case report

A 4-year-old male red-necked wallaby was presented to the Animal Clinic (Prague, Czech Republic) with acute onset of pain, inappetence and salivation. He has never had the same or similar problems before. This kangaroo was kept with another female that was without any clinical problems. The owner did not mention any accident. The kangaroo was fed with a commercial food for herbivore mammals, especially hay, and he had free access to grass and water. There was no change or anything new in the diet. The weight of this kangaroo was 34kg and it was not possible to do a good clinical examination without anaesthesia, because the patient was very painful and aggressive. At first sight it was possible to see swelling on the right side of mandible.

We started with intramuscular application of premedication: diazepam (1mg/kg), medetomidin (40ug/kg) and ketamin (3mg/kg). Than the kangaroo was anesthetized with isoflurran by using the facial mask. After this it was possible to do a clinical examination. The clinical examination revealed rigid swelling and edema without the presence of a draining sinus on the right side of mandible under the diastema (between lower incisors and the premolars). There were all teeths and soft tissue without any signs of trauma in the oral cavity.

Blood was collected from intravenous catheter which was placed in v. cephalica antebrachii. All blood tests results, including a complete blood count and a plasma biochemistry, were within reference range.

Computer tomography of the head: There was found alveolar osteomyelitis around the left mandibulary incisor and presence of infection at the tooth root. Other structures were without any evidence of pathological process.
Treatment

The kangaroo was submitted to extraction of the left mandibulary incisor. Patient was monitored during the surgery (thoracic and cardiovascular auscultation, ECG). The oral cavity was flushed with chlorhexidine liquid. The gingival attachment was cut around the whole circumference of the incisor. At first the gingival flap was prepared by using the periostal elevator. The bone overlying the root on the dorsal side was drilled away. The tooth was removed with the rabbits incisors luxator and also with the elevator. The wound was flushed with warmed saline with iodine. Thorough debridement was performed. A collagen cup with gentamycin was placed into the alveolar socket. The gingiva was sutured by resorable material. Samples of pus were submitted for bacteriology and sensitivity testing.

After teeth extraction we started with analgesics and antimicrobial therapy: metronidazol (20mg/kg PO BID), clindamycin (11mg/kg PO BID) and meloxicam (0,2mg/kg PO SID).

The result of bacteriologic examination was Escherichia coli and Enterococcus sp.

Results

Two days after the treatment the kangaroo started to eat grass and hay normally, withouth any help. One week after the treatment the lower jaw was without any signs of swelling. He started to be very friendly to the owner.

Discussion

The dental disease or oral necrobacillosis ( „lumpy jaw“) is common cause of morbidity and mortality affecting captive macropods. This cases very often end in euthanasia. However extraction with or without marsupialization of the absces, or endodontic treatment of the affected teeth could be succesfull. In another article the usage of the locally applied chlorhexidin is mentioned, which shortens the treatment time and may prevent reccurence.

Conclusion

This case report describes a succesfull treatment of periapical abscedation and apical osteomyelitis in red-necked wallaby.

References


In November 2014 a 3-month old seal pup was presented with a history of a non-unionized fracture of the right mandible. The fracture had been treated conservatively for a few weeks. It was comminuted and compound, with an intraoral wound. A few necrotic bone fragments had recently been removed under sedation.

Upon presentation, there was no external swelling or deformation. The patient was premedicated, induced, intubated and maintained on isoflurane inhalation. Forty milliliters of plasma were collected to generate PRF and PRP. X-rays showed a slightly displaced fracture of the right mandible with a gap present.

The mandible was accessed ventrally. Both fragments were exposed. The area was debrided and lavaged. A cortical bone membrane of equine origin\(^1\) was wrapped around the ends of both fragments, in the shape of a taco shell. The inside of the taco was then filled with DFB particles of canine origin\(^2\) mixed with PRF. PRP was poured over the mixture before tacking the top edges of the taco, thus preventing the filling from migrating. PPP was poured over the surgical site while it was being sutured closed in multiple layers with 3-0 Monocryl. The intraoral defect was next repaired before allowing the patient to recover. The patient was returned to the rescue facilities with continued antibiotics and analgesics (Carprophen, Tramadol, Ciprofloxacin). Recommendations were to feed him small pieces that could easily be swallowed.

The patient was monitored, it did well for a couple of months. CT scans were obtained 2 and 8 weeks post operatory showed callus formation, a smaller gap and no sign of further infection. Ten weeks post surgery the primary veterinarian was contacted because the patient was eating slower than before and seemed unable to process bigger pieces of fish. Closer examination revealed a restricted range of motion of the mandible. A third CT scan was planned in order to discover the cause of the problem. In the meantime, the patient was fed minced fish. Four weeks later, the results of the CT scan showed some bony bridging between the caudo-lateral edge of the mandibular fracture and the medial surface of the zygomatic arch.

It was decided to try a second surgery to rectify the problem. It took another 4 weeks

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\(^1\) Ossiflex, Veterinary Transplant Services, 215 E Titus St, Kent, WA 98032, United States.
\(^2\) Periomix, Veterinary Transplant Services, 215 E Titus St, Kent, WA 98032, United States.
before the surgery could be scheduled. By that time, the patient could barely open its mouth. The jaws had to be forced apart with the oral speculum in order to intubate “Caesium”. The mandible was accessed through the incision made previously. Palpation located a cord of ossified tissue between the mandible and the zygoma. The ossified tissue was removed in small fragments, using rongeurs. This process freed the mandible and suddenly full range of motion was back. A fat pad, 3cm long by 1.5 cm wide by 8 to 10mm thick was harvested ventral to the mandible and was sutured between the lateral side of the mandible and the medial side of the zygoma to prevent recurrence of the osseous bridging. The surgical site was closed in a similar manner as in the first surgery. The patient was sent back to the aquarium, for closer monitoring, on a regimen of antibiotics and anti-inflammatory drugs (same as before). He was to be fed small fish in small portions as shorter intervals than previously.

He progressed steadily and was reported to be able to handle progressively larger prey. At a month post second surgery, he had caught up weight wise with the other seals in the group and was processing food normally.

We started with an infected site and placed two xenografts of different origin. Ossiflex serves as a barrier membrane, provides structural support, osteoconductivity, biodegradability and replacement. Periomix provides osteoinductivity, osteoconductivity, biodegradability and replacement. Both show minimal immunogenicity because of the treatment they underwent. PRF and PRP are autogenous. PRF consists of fibrin mixed with platelets, leucocytes and growth factors. The growth factors can be released for at least a week and up to 28 days. The growth factors attract and stimulate mesenchymal cells to form osteoblasts. PRP is similar minus the fibrin. By combining several products, we aimed to create the ideal bone implant. In this case may have progressed too far and we ended up with ossification within the soft tissues.
Iatrogenic incisor pathology in rabbits and guinea pigs

Vladimir Jekl, Minarikova Andrea, Silvia Kohutova & Karel Hauptman

Dental disease is one of the most common diseases in rabbits and guinea pigs. As a result, many veterinary universities offer nowadays at least some training in these more “exotic” companion species. A lot of education centres and veterinary conferences are aimed to educate veterinary practitioners and veterinary students, particularly about rabbit and guinea pig dentistry, European Congresses of Veterinary Dentistry included. The European College of Zoological Medicine (ECZM) and the European School of Advanced Veterinary Studies (ESAVS) offer “cutting edge” education in exotic companion small mammals for residents or for advanced practitioners. Data for self-education are also available in a form of books and journal articles (Capello et al. 2005, Jekl 2009, Quesenberry and Carpenter 2012, Harcourt-Brown and Chitty 2014, Böhmer 2015).

Incisor malocclusion is one of the dental pathologies seen commonly in rabbits and in guinea pigs and it is mostly secondary to acquired syndrome of dental disease (Böhmer 2015). In contrast to proper care, there is a still malpractice to trim the incisors using pliers and scissors (Jekl et al. 2008). The aim of this study was to highlight the necessity of proper care and to show secondary pathological changes associated with this painful and unnecessary procedure.

In a recent article (Minarikova et al. 2015), the iatrogenic incisor malocclusion, typically caused by veterinary general practitioners using unsuitable tools for incisor clinical crown height correction, was seen in 2.7 per cent (27/1000) of guinea pigs and was associated with tooth fracture, uneven occlusal surface, pathological tooth movement, pulpal opening and bleeding. The same pathologies were seen also by the authors in rabbits, but in higher, 6% incidence (60/1000). Moreover, in rabbits it was also associated with tooth torsion and with periapical abscessation. In guinea pigs, trimming the teeth using pliers caused more pronounced anorexia (as the incisors were out of occlusion) and general health status deterioration.

The proper care of the incisor pathology is the exact diagnostics and proper care. The exact diagnostics consists of clinical crowns adspetion, teeth and gingival sulci palpation and incisor radiograph using lateral and intraoral views. Proper treatment of incisor pathology depend on the cause of the disease. In case of incisor malocclusion, coronal height and occlusal surface should be adjusted using diamond dental burrs or diamond discs. In case of
Iatrogenic pulp exposure is another common complication associated with traumatic tooth fracture or overzealous iatrogenic incisor trimming. Treatment of exposed pulp requires aseptic preparation of the site, sterile rinsing, haemostasis, drying, preparation of a small cavity and the use of an intermediate restorative material (e.g. hydroxide cement). Extraction should be considered only where there will be positive definite benefits for the animal. Pre-extraction radiographs are necessary to establish the tooth morphology, curvature, fractures, or adjacent tissue disease. In cases of incisor overgrowth, the authors had good experience with initial coronal height adjustment using diamond disc and extraction in 5-7 days later. This is because the rate of incisor eruption in hypo function (not in occlusion) is more rapid due to lack of wear (approximately 5-6 mm per week), dentoalveolar junction is more delicate and the teeth extraction is easier (Jekl 2009).

In conclusion, all practitioners should be recommended to use proper techniques for incisor height and occlusal adjustments. Trimming the incisors using pliers and/or scissors is a malpractice which should be avoided. Rabbit and guinea pig owners do not recognize, that the incisors were improperly treated, so public education is also important.

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Dental pathology of the bobcat (*Lynx rufus californicus*) and mountain lion (*Puma concolor californica*)

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Dental pathology has been studied in domestic and feral cats, but there has been little study of the dental pathology of wild felines. The goal of this study was to characterize the types of dental lesions found in the California bobcat and California mountain lion, and to compare them to those found in domestic and feral cats. We hypothesized that dental lesions in wild felines may be similar or differ to those found in domestic and feral cats, based upon similarities or differences in disease susceptibility, behavior, and diet.

The study population included 277 bobcat skulls and 91 mountain lion skulls from the collections of the California Academy of Sciences, San Francisco and Museum of Vertebrate Zoology, U.C. Berkeley. Only adult and young adult bobcat and mountain lion specimens were examined. The examination included a detailed macroscopic examination with concurrent radiographic examination.

The bobcat and mountain lion were found to exhibit a range of dental lesions and abnormalities. The prevalence of congenital and developmental abnormalities was relatively low, but acquired lesions were common, especially attrition/abrasion, periodontitis, and tooth fractures. Acquired lesions were found to affect adult bobcats and mountain lions more frequently and severely than young adults. As a result of acquired lesions, some specimens suffered from periapical disease and endodontal disease. It was of particular interest to show that tooth resorption is found in these wild felines, though at a lower prevalence than in domestic cats. Specimens that exhibited severe generalized dental disease would have likely suffered from considerable morbidity while alive, possibly leading to an increase in mortality.
Oral and dental diseases in a population of domestic ferrets (*Mustela putorius furo*)

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**Background:** Domestic ferrets (*Mustela putorius furo* or *Mustela furo*) belong to the family Mustelidae. They have been used for many purposes, but recently their popularity as companion animals has increased greatly. However, data on their oral and dental diseases are rather rare.

**Objective:** To investigate oral and dental diseases in client-owned domestic ferrets.

**Methods:** In this study, a detailed oral exam and full-mouth dental radiographs were performed on 40 client-owned domestic ferrets undergoing general anesthesia.

**Results and conclusion:** Variations in occlusion and number of roots were noted in comparison to published literature on ferrets and polecats. Periodontal disease, attrition/abrasion and dental fractures, especially of canine teeth, were commonly observed. However, periapical disease associated with dental fractures, malocclusion, tooth resorption and neoplasia were uncommon.
The polar bear evolved from the brown bear some 70,000 to 100,000 years ago. It lives in the northern circumpolar regions of the world. It is the Arctic’s top predator carnivore and is completely dependent on sea ice for survival. The diet of the polar bear consists primarily of newborn ringed seal pups and 2 kg of seal fat is required daily for survival. Fifty percent of calories in a seal carcass are fat and the polar bear will consume the fat first and preferably, maximizing their caloric return. Annually, forty-four percent of newborn pups are killed by polar bears. They will also capture bearded seals, harp seals, and harbor seals and will kill walruses, and beluga whales. They will consume alternate food sources during fasting such as berries, other vegetation, birds, fish, caribou, muskox, and reindeer. The fasting period for the polar bear is extensive, lasting from the ice-free period to freeze-up.

Northern peoples rely heavily upon the polar bear as an important resource to their economy, as a food source, a subsistence activity, and it has important social and psychological functions. Twenty per cent of the polar bear population is hunted for sport. A hunt can bring $20,000.00 per bear into the community. These are important dollars to maintain today’s lifestyle of the northern peoples, allowing the purchase of gas and snowmobiles as examples. The hunt is a community affair and a skill taught by the elders to the young. The polar bear is a mentally and culturally powerful spiritual being and is part of the legends told to the young through stories by the elders.

Global warming in the Arctic is twice that found elsewhere in the world. Sea ice has been declining with rising air temperatures, and it is predicted that by the end of the 21st century there may be no sea ice cover during the summer months in the Arctic ocean. Reduced sea ice, warmer air and ocean temperatures, has changed the Arctic ecosystems. For example, subarctic fish have been noted with increased frequency in the Arctic related to reduced sea ice cover over the summer months. Arctic climate changes have been compounded by increased northern transport, tourism and mineral exploration.

Contamination in the Arctic is a major concern. The Arctic has become a trap for contaminants such as lead, cadmium, and zinc from increases in precipitation. Sea ice can accrue, concentrate, and transport radio-contaminated sediments. Mercury has a complex cycle in the Arctic, including a unique scavenging process, biomagnification in food webs,
and chemical transformation such as methylation.\textsuperscript{5}

Global warming and the melting of the permafrost releases mercury from organically bound to free, and this oxidizes to a very toxic form, methyl-mercury.\textsuperscript{5}

Sunlight-induced atmospheric reactions or springtime atmospheric mercury depletion events can load 150-300 tons of mercury to the Canadian Arctic archipelago each spring and these atmospheric mercury depletion events are the ultimate source of mercury to Arctic foodwebs.\textsuperscript{5} Atmospheric mercury depletion events are a recent phenomenon related to the Arctic climate change of temperature warming and increased atmospheric circulation.\textsuperscript{5}

The low ecological diversity, which characterizes the Arctic, also imparts vulnerability. A warming climate leads to remobilization and possible increases of contaminants in the food chain and exposure in the Arctic, and thus an increased risk for Arctic peoples and animals. Subsistence hunting and inadequate food inspection expose the Arctic peoples to contaminants in the food web. For example, Inuit mothers have contaminant levels such as PCBs, dioxins, and mercury, 6-12 times greater than Caucasians, Dene, or Métis peoples.\textsuperscript{6} These contaminants are known to affect the immune system, hepatic system,\textsuperscript{6} endocrine system, reproductive system, bone density,\textsuperscript{7} maternal and newborn health,\textsuperscript{8} and other organs.\textsuperscript{6} Yupik people of St. Lawrence Island Alaska consume primarily marine mammals-whale, walrus, and seals. These people have a higher serum concentration of PCBs than the general U.S. population.\textsuperscript{9} Marine mammals’ blubber and oils, sampled between 2004 and 2009 for PCBs, chlorinated pesticides and 7 heavy metals, were found to have a concentration of PCBs (the highest concentration was found in polar bears) at levels that trigger advisories for severely restricted consumption for humans.\textsuperscript{9} Lead is a contaminant found in the Arctic. Blood lead concentrations are associated with periodontal disease and lead affects the immune system.\textsuperscript{7} Cadmium toxicity affects bone remodeling causing osteoporosis through reduced mineralization and increased bone resorption, and may be a risk factor for periodontal disease and bone loss.\textsuperscript{10}

Persistent organic pollutants, POPs, were measured in 68 traditional foods from 1997-1999 and the concentrations were greatest in caribou liver, ringed seal liver, polar bear meat, and Beluga whale meat. They are causing human health concerns.\textsuperscript{11} POPs disrupt the reproductive, immune and endocrine systems, and decrease bone density.\textsuperscript{7} They are associated with periodontal disease. 1,234 adults from 1999-2002 were studied and found that organochlorine contaminant levels were associated with attachment loss and increased pocket depth seen with periodontal disease.\textsuperscript{6}

Threats to polar bear survival are great. Polar bears are losing sea ice to hunt on, resulting in diminished body size/weight, low reproductivity, and low cub survival rates.\textsuperscript{2,3,12} They are also losing their valuable food source, ice-associated seals, vulnerable to habitat loss from diminished sea ice.\textsuperscript{2,3,12} The growing tourist industry of polar bear watchers and photographers suggests a potential increased occurrence of contact between bears and humans.\textsuperscript{2,3,12} Starving bears will risk death to obtain food, and the numbers of bears that are killed defensively increases when the food source of the polar bear is less available.\textsuperscript{2,3,12} There are increased tourist hunting pressures and the profitability of such to the Inuit community may strain the relationship between subsistence hunting, providing for the community, and government management of polar bear populations.\textsuperscript{2,3,12} Polar bears and humans are upper trophic level consumers. They consume a local diet.

Polar bears are susceptible to contaminant-induced stress that may have an overall sub-clinical impact on their health and population status via impacts on their immune,
endocrine, and reproductive systems. The East Greenland population of polar bears is the most polluted species in the Arctic. The East Greenland population of polar bears and sled dogs had an estimated daily concentration of contaminants of 32-281 times greater than the WHO guidelines for humans. Polar bears bioaccumulate mercury as they eat polluted ringed and bearded seals. Levels of mercury in the liver and kidneys were found to be greater than the known lethal toxic threshold level in terrestrial animals. Bone density reduction and immune system impairment was found in polar bears exposed to organic pollutants.

Oral bacterial infection and inflammation and the destructive forces of the host’s immunological defence mechanisms lead to destruction of the dental supporting tissues to include connective tissues and the supporting bone, and eventually leading to tooth loss. The association between PD and systemic illness has been extensively documented for humans. However, limited information has been gathered as to oral health and the oral/systemic association of non-domestic animals, primarily with-in wild animal populations such as polar bears. Polar bear skulls from the East Greenland population were sampled for osteopenia from 2 periods- a considered polluted period from 1966-2002 versus the pre-pollution period 1892-1932. The results suggest that the reduced bone density found in the skulls from the polluted period may have been caused by organochlorine exposure. Organochlorines are known to alter endocrine homeostasis and are associated with reduced levels of IgG.

One study examined 87 polar bear teeth and found mercury, nitrogen, and carbon with increasing amounts as age increased.

A contaminants journey from emission to accumulation in the Arctic ecosystem is a complex series of steps, each of which can be changed by global changes. How contamination causes stress is influenced by environmental, ecological, and physiological stressors to form a complex picture. While oral disease is likely the most common disorder of dogs and cats, might it be that environmental contamination and the oral/systemic connection already noted in our Arctic peoples could also apply to the polar bear and become as destructive to this species?

The “Vulnerable” polar bear, the top predator carnivore of the world’s Arctic, is at risk from human behaviours that may forever alter their environment and their health, and lead us to only know this magnificent animal through exposure whilst housed in zoos. Collaborative efforts, to include governments, polar bear management groups, zoos, dental and medical personelle are of utmost importance to preserve our Arctic environment, peoples and our Ursus maritimus. One World One Health.

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Intraoral radiographs in rabbits: a case study with 13 rabbits

Manfred Schumacher

**Objective:** In this case study we investigated if intraoral digital radiographs are feasible in rabbits.

**Material and methods:** 13 rabbits with a bodyweight between 0.75-2.5kg were intraorally radiographed using the digital computed radiography system CR35/CR7 vet. In the first two rabbits without dental disease we established the feasibility of the technique using small sized photo stimulating phosphor plates to obtain intraoral radiographs. The remaining 11 rabbits presented in our clinic with dental disease. After clinical examination including probing with a periodontal probe, extraoral and intraoral radiographs were taken. We used a similar bisecting angle technique as in dogs and cats. This technique was easy to transfer to rabbits and it was possible to obtain intraoral radiographs for all patients. The radiographs provided clinical information which was important for further treatment. In 6 cases the findings directly influenced which therapy was used for these patients and therefore on the prognosis.

**Conclusion and clinical relevance:** The results support the clinical usefulness of intraoral digital radiographs in rabbits. A combination of extra and intraoral radiographs provides much more diagnostic information and has a good cost performance ratio compared to Computed Tomography.

**Intraoral radiographs in rabbits:** A case study with 13 rabbits.

**Literature**

Introduction

A thorough history of current and past medical problems can provide valuable information about small mammal health status. Physical examination may reveal pale mucosal surface, abnormalities of the heart or lungs, abdominal masses or other pathologies that may require further evaluation such as a haematological and plasma chemistry examination, urinalysis, radiography, electrocardiogram, echocardiography or any other diagnostic method prior to performing general anaesthesia. Thorough physical examination is therefore very important in establishing further diagnostic and anaesthetic plan. Based on clinical examination, laboratory analyses and results of imaging methods is patient placed in appropriate ASA physical status scale (class I-V). In dependence of the ASA-group an additional examination should be performed and/or patient life functions must be stabilised before any anaesthesia or sedation. Even when a rabbit is placed in ASA I-II, author recommends ensuring intravenous access.

Exotic pet mammals should be properly weight and all drug dosages should be calculated carefully. In case of intramuscular injectable drugs with potential pain effect (e.g. ketamin) or in case of larger volumes, the agent should be administered to more places to avoid any necessary pain. Intraperitoneal anaesthetic administration is not recommended. The anaesthesiologist should monitor anaesthetic depth and all the vital signs to keep the rabbit safe and in appropriate surgical plane. The objectives of surgical anaesthesia (stage 3, plane 2) are that patient does not feel pain, is not moving, is not aware, have no memory of the procedure afterward and all the vital signs are not dangerously depressed. Anaesthesia is achieved when there is a loss of sensation to either a part or all of the body. General anaesthesia also renders the patient unconscious. Sedation is similar to general anaesthesia but the patient remains semi-conscious. Careful attention to intraoperative care is an important contributory factor to successful anaesthesia.

Respiration

Due to small thoracic cavity, rabbits and rodents should be placed in position in which is ensured the ability of the lungs to be fully expanded. Respiration is monitored
by observing chest movements or by monitors detecting inspiration and expiration. A rise in respiratory rate during anaesthesia is mainly caused by a reduction of a depth of anaesthesia. A fall of respiratory rate below 40% of the patient’s normal rate (30-60 breaths/minute for rabbits) indicates respiratory failure. If the animal is not intubated, it is necessary to intubate him when anaesthetic monitoring showing signs of cardiovascular or respiratory failure. The airways should be inspected for obstruction and oxygenation together with assisted ventilation should be started. Doxapram is additionally administered. The pressure of assisted ventilation should never be greater than 8 mmHg.

Capnography measure the amount of carbon dioxide in the air that is breath in and out. The patient “end-tidal” CO$_2$ refers to the amount of CO$_2$ measured at the end of expiration and it is used for the estimation of arterial carbon dioxide partial pressure. A side stream capnograph is located between the breathing circuit and endotracheal tube to minimise the dead air space.

Supplemental oxygen, which maximises cardio and pulmonary efficiency, should be provided in all cases of sedation or anaesthesia. In general a flow rate of 200 ml/kg/min will provide an inspired oxygen concentration of at least 40%. Double flow rate could increase inspired oxygen concentration up to 80%. Humidifying and warming the oxygen is optimal.

Monitoring of cardiovascular function and circulation includes palpation of peripheral pulse to determine rate, rhythm and quality, and evaluation of mucous membrane colour and capillary refill time (CRT). Auscultation of heart beat, pulse oximetry and ECG are used to grossly assess of peripheral perfusion and heart function. The indirect blood pressure measurement is most commonly used. The ultrasonic Doppler flow detector makes audible blood flow in an artery distal to the blood pressure cuff. The mean arterial pressure (MAP) should be kept above 60 mmHg and systolic pressure above 90 mmHg to ensure adequate organ perfusion. A pneumatic cuff is usually placed on the shaved area above the elbow in a bed of ultrasonic gel and taped in place. Conjunctiva and gingiva should have a pink colour. CRT is best to measure on the gingiva dorsal to the incisors and should be no more than 1-1.5 sec.

Pulse oximetry is a non-invasive continuous measurement of oxygen saturation in tissues. A SpO2 greater than 91% usually indicates adequate oxygenation, however pulse oximetry may be inaccurate by as much as 5%, so the SpO2 level should be kept above 95%. Pulse oximetry fails to detect hypoventilation, hyperventilation or other respiratory problems; capnography is necessary to detect these changes. Pigmented skin could make the reading variable. A quality signal may be unobtainable in patients in hypothermia and hypotension.

Inhalant agents are naturally hypotensive, and untoward effects are dose dependant. The use of inhalant agents as sole anaesthetics necessitates higher doses with increasing the risk of potential adverse effects. Therefore, the use of analgesia and injectable premedication is recommended not only in rabbits, but also in rodents and ferrets. It was reported, that use of medetomidine often demonstrates a profound negative impact on the cardiovascular and respiratory system and should be used with caution. If hypotension occurs during the surgery, the inhalant anaesthesia is reduced first, while the continual rate infusion is increased.

Intravenous (IV) catheter is placed in lateral ear vein, cephalic or saphenous vein.
Before catheter placement a layer of EMLA is applied to provide local anaesthesia. Administering perioperative fluids subcutaneously is unreliable due to peripheral and visceral vasoconstriction. Intraosseous administration is an alternative to IV access.

Fluid therapy is in exotic pet mammals an important component of haemodynamic stabilisation minimising drug-exacerbated hypotension and risks related to anaesthesia. Perioperative fluids are given via precise syringe pumps. A dosage rate for perioperative IV fluid therapy is 10-15 ml/kg/hour. In case of gastric dilatation, fluid administration into saphenous vein should be avoided.

Recommended first line therapy for hypotension are IV boluses of polyionic fluids of 5-15 ml/kg. Overhydration should be avoided. If a rabbit or another exotic pet mammal is refractory to isotonic crystalloid therapy, the use of synthetic colloids should be considered. Hetastarch is administered at 5 ml/kg IV over 5 to 10 minutes. Another possibility is to use 7.5% hypertonic saline in 1-3 ml boluses over 10 minutes until normal heart rate and blood pressure are obtained. Dopamine or norepinephrine can be used to treat refractory hypotension. Checking glycaemia, PCV, total protein and blood gas analysis intraoperatively is recommended.

Blood loss

Blood losses less than 10 % of blood volume in a patient with normal PCV could be corrected with isotonic fluids at a rate of three times the estimated blood loss amount. In case of chronic (PCV <20-25%) or acute blood loss, transfusion of whole blood is recommended. Synthetic colloids should be also added to the crystalloid therapy in rabbits with hypoproteinaemia. Rabbit clinical examination and laboratory analyses are important in determination whether is animal suitable as a donor and for calculation proper amount of blood needed for a recipient. Rabbits have four blood groups; however no problems in the first transfusion were noted by the author. The major cross-match is optimal. The estimation of the amount of whole blood required by the recipient can be calculated as weight (lbs) × 30 × desired PCV − recipient PCV/donor PCV. Whole blood is administered by syringe pump or in boluses into the IV catheter or intraosseously. The haematocrit and total plasma protein concentration should be evaluated 1, 2 and 24 hours post-transfusion. Blood transfusions should be administered within 4-6 hours to prevent the bacterial overgrowth.

The most common method of monitoring temperature is with the use of a rectal thermometer. Heat loss occurs by convection, radiation, conduction and evaporation. Rabbits lose heat rapidly because of their high surface area relatively to their bodyweight. Use of heating blankets/pads set at specific temperature (35-37°C) is strongly recommended. Also fluid and oxygen should be pre-warmed. Using heating infusion devices is recommended.

### Table 1. Diazepam and midazolam dosages used for sedation of selected small mammals.

<table>
<thead>
<tr>
<th></th>
<th>Ferret</th>
<th>Rabbit</th>
<th>Guinea pig</th>
<th>Chinchilla</th>
<th>Rat</th>
<th>Prairie dog</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diazepam</strong></td>
<td>mg/kg</td>
<td>mg/kg</td>
<td>mg/kg</td>
<td>mg/kg</td>
<td>mg/kg</td>
<td>mg/kg</td>
</tr>
<tr>
<td>Ferret</td>
<td>0.05-0.1</td>
<td>0.2-0.5</td>
<td>0.3-1.0</td>
<td>0.2-0.4</td>
<td>0.5-1.5</td>
<td>0.4-0.6</td>
</tr>
<tr>
<td>Rabbit</td>
<td>0.05-0.15</td>
<td>0.2-0.5</td>
<td>0.3-1.0</td>
<td>0.2-0.4</td>
<td>1.0-2.0</td>
<td>0.3-0.5</td>
</tr>
<tr>
<td>Guinea pig</td>
<td></td>
<td></td>
<td>0.3-1.0</td>
<td>0.2-0.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chinchilla</td>
<td></td>
<td></td>
<td>0.2-0.4</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rat</td>
<td></td>
<td></td>
<td>0.5-1.5</td>
<td>0.4-0.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prairie dog</td>
<td></td>
<td></td>
<td>0.4-0.6</td>
<td>0.3-0.5</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 2. Acetylcholine receptor antagonists used in small mammals.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Ferret</th>
<th>Rabbit</th>
<th>Guinea pig</th>
<th>Chinchilla</th>
<th>Rat</th>
<th>Prairie dog</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atropine</td>
<td>mg/kg</td>
<td>0.05</td>
<td>0.8-1.0</td>
<td>0.05</td>
<td>0.05</td>
<td>0.05</td>
</tr>
<tr>
<td>Glycopyrrolate</td>
<td>mg/kg</td>
<td>0.01</td>
<td>0.01-0.02</td>
<td>0.01-0.02</td>
<td>0.01-0.02</td>
<td>0.01-0.02</td>
</tr>
</tbody>
</table>

Table 3. Recommended intramuscular premedication and induction for selected small exotic pet mammals followed by isoflurane.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Ferret</th>
<th>Rabbit</th>
<th>Guinea pig</th>
<th>Chinchilla</th>
<th>Rat</th>
<th>Prairie dog</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atropine</td>
<td>mcg/kg</td>
<td>0.01-0.02</td>
<td>0.02-0.05</td>
<td>0.05-0.1</td>
<td>0.02-0.05</td>
<td>0.05-0.2</td>
</tr>
<tr>
<td>Ketamine</td>
<td>mg/kg</td>
<td>2-5</td>
<td>5-15</td>
<td>5-15</td>
<td>1-10</td>
<td>1-15</td>
</tr>
<tr>
<td>Midazolam</td>
<td>mg/kg</td>
<td>0.1</td>
<td>0.2-0.3</td>
<td>0.2-0.3</td>
<td>0.1-0.2</td>
<td>0.2-0.5</td>
</tr>
<tr>
<td>Butorphanol</td>
<td>mg/kg</td>
<td>0.1</td>
<td>0.3</td>
<td>0.3</td>
<td>0.3</td>
<td>0.3</td>
</tr>
</tbody>
</table>

For induction, intravenous drugs of propofol, midazolam and/or ketamine could be administered to the effect. General anaesthesia is then maintained using isoflurane/sevoflurane.

Table 4. Recommended total intramuscular anaesthesia for selected small exotic pet mammals.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Ferret</th>
<th>Rabbit</th>
<th>Guinea pig</th>
<th>Chinchilla</th>
<th>Rat</th>
<th>Prairie dog</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medetomidin</td>
<td>mcg/kg</td>
<td>0.04-0.06</td>
<td>0.04-0.15</td>
<td>0.08-0.2</td>
<td>0.05-0.08</td>
<td>0.08-0.2</td>
</tr>
<tr>
<td>Ketamine</td>
<td>mg/kg</td>
<td>3-10</td>
<td>5-15</td>
<td>5-15</td>
<td>5-10</td>
<td>5-20</td>
</tr>
<tr>
<td>Midazolam</td>
<td>mg/kg</td>
<td>0.15-0.25</td>
<td>0.2-0.4</td>
<td>0.3-0.6</td>
<td>0.3-0.4</td>
<td>0.2-0.3</td>
</tr>
<tr>
<td>Butorphanol</td>
<td>mg/kg</td>
<td>0.1</td>
<td>0.3-0.4</td>
<td>0.3-0.4</td>
<td>0.3-0.4</td>
<td>0.5</td>
</tr>
</tbody>
</table>

Table 5. Analgesic drugs used in selected small exotic pet mammals.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Ferret</th>
<th>Rabbit</th>
<th>Guinea pig</th>
<th>Chinchilla</th>
<th>Rat</th>
<th>Prairie dog</th>
</tr>
</thead>
<tbody>
<tr>
<td>Butorphanol</td>
<td>mcg/kg</td>
<td>0.05-0.1</td>
<td>0.2-0.6</td>
<td>0.08-0.2</td>
<td>0.05-0.08</td>
<td>0.08-0.2</td>
</tr>
<tr>
<td>Buprenorphine</td>
<td>mg/kg</td>
<td>0.01-0.03</td>
<td>0.01-0.05</td>
<td>5-15</td>
<td>5-10</td>
<td>5-20</td>
</tr>
<tr>
<td>Meloxicam</td>
<td>mg/kg</td>
<td>0.2-0.3</td>
<td>0.2-0.8</td>
<td>0.3-0.6</td>
<td>0.3-0.4</td>
<td>0.2-0.3</td>
</tr>
<tr>
<td>Carprofen</td>
<td>mg/kg</td>
<td>2-5</td>
<td>2-5</td>
<td>0.3-0.4</td>
<td>0.3-0.4</td>
<td>0.5</td>
</tr>
<tr>
<td>Tramadol</td>
<td>mg/kg</td>
<td>5</td>
<td>5-10</td>
<td>5-10</td>
<td>5-10</td>
<td>5-10</td>
</tr>
</tbody>
</table>

Drug dosages used by article authors in selected small exotic pet mammals. Please note, that the optimal dosages need to be adjusted based on a current clinical status of the animal.

Acknowledgements

This paper was supported by specific research of the Faculty of Veterinary Medicine, University of Veterinary and Pharmaceutical Sciences Brno, Czech Republic (2015/2016) and by the project IGA No. 114/2013/FVL.

Further reading

Hauptman K., Jekl V., Knotek Z. 2003 Use of Medetomidine for Sedation in the
Histopathological characteristics and immunohistochemical expression of Ki-67 and VEGFR-2 in canine inflammatory, benign and malignant oral conditions

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E-mail: anna.rejec@gmail.com

Background and objective

Differences in the biologic behaviour and in the prevalence of local recurrences and/or distant metastatic disease of malignant oral tumours may develop as a consequence of abnormalities in intracellular signalling pathways where both proliferation of tumour cells and angiogenesis play an important role. Our aim was to investigate and compare histopathological parameters (grade, presence of necrosis, mitotic index) and immunohistochemical biomarkers Ki-67 and vascular endothelial growth factor receptor 2 (VEGFR-2) in canine inflammatory (stomatitis), benign (epulides) and malignant oral (carcinomas, sarcomas, melanomas) conditions and correlate the measured biomarkers with the overall survival. Better understanding of the biology elucidates new aspects of canine head and neck neoplasia biological and clinical behaviour, which is essential as it might help to guide the therapy and develop new, more effective treatment strategies.

Materials and Methods

Tissue samples of oral inflammatory, benign and malignant canine conditions were collected from the clinical cases presented to Animal Hospital Postojna, Slovenia where the cases were diagnosed and treated. A total of 54 samples were identified. There were 9 stomatitis samples, 11 benign samples consisting of 6 samples of acantomatous epulis and 5 fibromatous epulis samples, 17 epithelial tumours (SCC) and 17 other samples consisting of 7 malignant oral melanoma and 10 oral sarcoma samples. The medical records of dogs were reviewed and data regarding the clinico-pathological variables and survival were abstracted. Immunohistochemical analysis was performed using monoclonal mouse anti-human VEGFR-2 antibody (clone A3, Santa Cruz Biotechnology, Santa Cruz, INC, CA, dilution 1:50) and a mouse monoclonal anti-human Ki-67 antibody (clone MIB-1, Dakocyтомation, UK, dilution 1:50). Data were analysed by parametric and non-parametric statistical analyses. Mann-Whitney test (Wilcoxon rank sum test) was used to detect differences in the Ki-67 and VEGFR-2 between different types of conditions investigated. Univariable statistical analysis was performed to identify the presence of potential prognostic factors. Curves for overall survival were generated by the Kaplan-Meier product limit method (log-rank, Cox model).
Associations between the variables investigated (age, gender, grade, histotype, mitotic activity, Ki-67, VEGFR-2) were assessed using Sparman’s rank correlation method tests.

Results

Percentage of expression and labelling intensity of Ki-67 and VEGFR-2 determined immunohistochemical (IHC) score. The Ki-67 IHC score in dogs with benign (epulides) and inflammatory (stomatitis) oral lesions in comparison with malignant tumours was significantly lower (p<0.001 and p=0.008, respectively). The expression of VEGFR-2 was not detected in inflammatory (stomatitis) and benign oral tumours (epulides) of dogs while malignant tumours expressed VEGFR-2. The significant difference in VEGFR-2 IHC score when comparing benign tumours (epulides) or inflammatory oral conditions (stomatitis) with malignant tumours was detected as well (p<0.001 and p=0.002, respectively). Dogs with oral tumours expressing higher percentage of VEGFR-2 had worse prognosis and shorter overall survival in comparison with those that did not express or expressed lower percentage of VEGFR-2.

Conclusion

We have shown that VEGFR-2 status has the potential to emerge as an important biomarker in diagnosis, prognosis and treatment of malignant oral tumours in dogs as we confirmed the involvement of VEGFR-2 signalling pathway in canine malignant head and neck tumours by detecting the protein expression in 59% of cases. On the contrary, inflammatory (stomatitis) and benign oral tissues (epulides) were negative for VEGFR-2. Therefore, the potential use of VEGFR-2 as a supplemental diagnostic biomarker of malignancy affecting head and neck region in dogs can be suggested. It was found that oral SCC tumours were negative or expressed lower levels of VEGFR-2 in comparison with other malignant tumours. The concomitant high expression of Ki-67 and VEGFR-2 in indicated on their more aggressive biologic behaviour. Malignant melanoma and oral sarcoma tumours expressed higher values of VEGFR-2 in comparison with oral SCC. Dogs with head and neck tumours of malignant origin expressing VEGFR-2 may benefit from using the biologically target drugs inhibiting angiogenesis as monotherapy or as a part of multimodal treatment approach. Although several histopathological variables investigated including histological type, grade, mitotic rate, invasiveness and/or degree of differentiation, differed between different head and neck histotypes, the variables did not prove as predictors of survival of dogs with neoplastic head and neck tumours investigated. There was a trend of increased overall survival of dogs with low number of mitosis and dogs with well-differentiated tumours but the differences did not reach statistical significance. In contrast, site (location), stage, grade of the head and neck tumours and the presence of necrosis affected the overall survival of dogs investigated. Advanced stages of the head and neck tumours and the presence of necrosis in the pathohistological specimens correlated with shorter overall survival. Furthermore, the negative prognostic significance of VEGFR-2 but not Ki-67 in dogs with malignant tumours was confirmed. As the role of VEGFR-2 as unfavourable prognostic biomarker in dogs with malignant oral tumours was confirmed, further work is focused to ascertain how different expression levels of VEGFR-2 can be used in screening, diagnosis, monitoring and prognosis of dogs with different types of head and neck neoplasia.
References


Cultivable root canal microbiota of fractured teeth in dogs

Ana Nemec\textsuperscript{1}, Špela Srečnik, Urška Javoršek, Tina Pirš\textsuperscript{2} & Irena Zdovc\textsuperscript{2}

\textbf{Background:} Untreated, dental pulp exposure leads to pulpitis, pulp necrosis and periapical disease due to infection. However, data on composition of endodontic microflora in dogs lack.

\textbf{Objective:} To investigate microbial composition in root canals of fractured teeth in dogs.

\textbf{Methods:} Systemically healthy client-owned dogs, routinely treated for complicated crown or crown-root fractures were included in this study. Twenty-five teeth were evaluated. Before any dental treatment was performed, each tooth was radiographed and a sample taken from the gingival sulcus/pocket with a sterile paper point. The tooth was then scaled above and below the gumline and 5.25\% NaOCl solution placed on the crown for at least 1 minute, before rinsed with sterile saline. A sample was obtained from the crown with a sterile swab and the root canal then accessed under aseptic conditions to obtain a sample. All samples were immediately inoculated onto blood agar plates and incubated at 37°C in aerobic and anaerobic conditions. Isolates were identified by MALDI-TOF technology.

\textbf{Results and conclusions:} Anaerobes were predominant bacterial species in the root canals and the most common bacteria isolated belonged to the genera \textit{Bacteroides}, \textit{Porphyromonas}, \textit{Propionibacterium} and \textit{Streptococcus}. The same type of bacteria were also most commonly found in gingival sulci/pockets, suggesting that the sulcus/pocket is likely the source of bacteria in root canals, similar to humans. Limitations of this study are related to lack of data on the duration of pulp exposure, the ability of the isolation of all of the bacterial species, and limitations of techniques for bacterial identification.
Systemic levels of regulatory T cells and other lymphocyte subpopulations investigated by flow cytometry in dogs with periodontal disease

Ana Rejec  
Animal Hospital Postojna, Postojna, Slovenia  
E-mail: anna.rejec@gmail.com

Background and objective

The immune background of periodontal disease (PD) might be elucidated through investigation of the immune mechanisms, which help to maintain a balance between activation and suppression of the immune responses. In these processes, both innate and adaptive immunity play a role as they provide the defence against invading periodontopathogens responsible for destruction of the tooth-supporting structures. The balanced immune regulatory mechanisms between different T cell sets and subsets (CD4+ helper T cells, CD8+ cytotoxic T cells, regulatory T cells) and B cells in PD help to keep periodontium in a healthy state. An unbalanced host response to periodontopathogens is also determining the disease outcome. Sufficient number and function of regulatory T cells is essential for maintaining peripheral tolerance, preventing autoimmune diseases and limiting chronic inflammatory diseases, also PD. The role of systemic levels of regulatory T cells in PD is limited. As PD might also result from the failure of peripheral tolerance mechanisms including regulatory T cells it was of our interest to investigate whether there are differences between dogs with stage 1 PD (gingivitis) and dogs with stage 3,4 PD (moderate to advanced periodontitis) with respect to systemic levels of regulatory T cells (CD4+CD25+ and CD4+CD25+FOXP3+). Other T cell subpopulations (CD4+, CD8+, CD5+) and CD21+ B cells were followed as well.

Materials and Methods

Dogs with stage 1 PD and stage 3,4 PD presented and treated in Animal Hospital Postojna, Slovenia were included in the study. Flow cytometric immunophenotyping of peripheral blood lymphocytes included evaluation of cell surface expression of T cell (CD5, CD4 and CD8) and B cell (CD21) markers. Negative controls were controls using an appropriate isotype matched controls. Immunostaining for FOXP3 was done with a cross-reactive murine PE-conjugated FOXP3 antibody. A directly conjugated rat IgG2A antibody was used as the isotype control. The differences in the parameters investigated between groups of dogs investigated were compared by Mann-Whitney U test (Bonferroni adjustment, p-values <0.005). The analysis was performed using R software (Core Team, 2013).
Results

No significant differences in the absolute number of white blood cells and the relative numbers of lymphocytes were detected between dogs with stage 1 PD and dogs with stage 3,4 PD \( (p=0.009; \ p=0.012, \text{ respectively}). \) Significant decrease of absolute numbers of lymphocytes in dogs with stage 3,4 PD in comparison with dogs with stage 1 PD was detected \( (p<0.001). \) Dogs with stage 3,4 PD had significantly lower values of peripheral blood CD5+, CD4+, CD8+ T cells and CD21+ B cells compared to dogs with stage 1 PD \( (p<0.001). \) The mean ratio of CD4/CD8 in dogs with stage 3,4 PD versus dogs with stage 1 PD was not significantly altered \( (P=0.052). \) Dogs with stage 3,4 PD had significantly lower percentages of regulatory T cells \( (\text{CD}4+\text{CD}25+ \text{ and CD}4+\text{CD}25+\text{FOXP}3+) \) in the blood as compared to dogs with stage 1 PD \( (p<0.001). \) High positive correlation between the frequency of CD4+CD25+ and CD4+CD25+FOXP3+ regulatory T cells in dogs with stage 3,4 PD was demonstrated \( (r=0.71). \) Positive correlations between CD4+, CD8+, CD21+, CD5+ and CD4+CD25+ and CD4+CD25+FOXP3+ were demonstrated as well. The differences were statistically significant.

Conclusion

The results presented indicate that lower systemic levels of regulatory T cells \( (\text{CD}4+\text{CD}25+, \text{CD}4+\text{CD}25+\text{FOXP}3+) \) and lower levels of other immunophenotype markers investigated \( (\text{CD}5+, \text{CD}4+, \text{CD}8+, \text{CD}21+) \) correlate with advanced stages of PD in dogs and might thus contribute to the pathology processes responsible for multiplication, activity and persistence of periodontopathogens. As regulatory T cells might represent an interesting target for new treatment strategies of PD not only in dogs but also in humans, further studies including assessment of biomarkers investigated in different clinical presentations of PD with following in a longer period of time are needed to understand the exact relationship among regulatory T cells and effector T cells regulating the balance between protective and destructive immune responses involved in the pathogenesis of periodontal diseases.

References


It has been previously shown that oral calicivirus carriage is strongly associated with caudal stomatitis in cats. Progress in molecular biology has led to the development of quantitative polymerase chain reaction techniques, which enable to evaluate the viral load. The main purposes of this study are to assess the relationship between the oral calicivirus viral load and 1. the severity of the lesions at the time of presentation for cats suffering from chronic caudal stomatitis, 2. the treatment outcome after dental extractions.

The dental records of cats referred to our speciality practice between 2011-2015 for management of chronic stomatitis were retrieved. Inclusion criteria were: cats with clinically confirmed caudal stomatitis lesions, recorded grading of caudal stomatitis lesions, positivity for oral calicivirus carriage using a real-time quantitative PCR test, primary management of the cases with no previous extractions. Seventy cats met the requirements. Data collected from patient’s record included patient history (breed, age, presence of respiratory disorders, duration of clinical signs), blood tests, viral testing (Herpesvirus, FeLV, FIV), extent and severity of oral lesions, teeth extracted and treatment outcome. Multivariate statistical analysis is performed to assess the relationship between the different parameters. Results of this preliminary study will be presented.
Is *Leishmania infantum* an etiologic factor in feline chronic stomatitis?

Ignacio Velázquez-Urgel\(^a\), Maria Flaminia Persichetti\(^b\), Antonella Migliazzo\(^c\) & Maria Grazia Pennisi\(^d\)

Feline Stomatitis is an oral disease, characterized by a chronic mucosa inflammation with a lymphoplasmocytic\(^7\) histological pattern. Its etiology is unknown. Many theories have speculated about several causative agents. *Leishmania infantum* is a parasite and one of its clinical signs is chronic gingivitis and stomatitis in cats\(^1\). The histological pattern of these lesions is pyogranulomatous to granulomatous\(^1,2,3,10\), with a predominantly diffuse infiltration of macrophages\(^3\).

In several reported cases and clinical studies leishmaniosis has been associated with chronic stomatitis\(^2,8,9,10\), speculating on the possibility that *Leishmania infantum* may be one of the causative agents of Feline Chronic Stomatitis in endemic areas\(^1,6\).

This prospective study aims to advance knowledge about Feline Chronic Stomatitis, trying to determine whether, in the geographic area of Barcelona (leishmaniosis endemic area\(^4,5,9\)), this parasite is present or not in patients with chronic stomatitis.

Twenty-four cats with clinical signs of Feline Chronic Stomatitis were tested for leishmaniosis. The samples were collected from conjunctiva (swabs), oral mucosa (swab and biopsy), blood (serum and plasma) and urine.

This presentation will discuss the results and conclusions of the study.

**References**


Matrix metalloproteinases and their inhibitors in feline gingiva

Isabelle Ritz¹, Cetina Thiel¹, Martin Kramer¹ & Susanne Alldinger²

¹Department of Veterinary Clinical Science, Small Animal Clinic, Surgery, Justus-Liebig-University, Frankfurter Straße 108, Giessen 35392, Germany; ²German Veterinary Medical Society, Friedrichstraße 17, Giessen 35392, Germany

The aim of this study was to immunohistochemically evaluate the presence of matrix-metalloproteinases (MMPs) and their tissue inhibitors (TIMPs) in feline gingival tissue and to find a relationship between these enzymes and the occurrence of resorptive lesions (RL) or gingivitis.

Gingival punch biopsies were taken from 55 cats after clinical and radiological examinations of their oral cavity for histological and immunohistochemical evaluation. From patients that suffered from RL the biopsies were taken at the gingival margin of affected teeth. These samples were histologically evaluated with hematoxylin-eosine and by immunohistochemistry to detect MMP-1, -2, -8 and TIMP-1 and -2. The results were semiquantitatively evaluated for statistical analysis.

The grading of gingivitis based on clinical and histological observations. These grades showed a significant correlation. The clinical evaluation showed in 29 % of cats a moderate to severe gingivitis, histologically moderate to severe inflammatory signs were observed in 35 % of the samples. Thus, the majority of cats examined showed none or moderate signs of gingival inflammation.

The enzymes examined were highly expressed in epithelial and endothelial cells and moderately in fibrocytes and fibroblasts of feline gingiva, except MMP-8, which was generally expressed slightly. The inflammatory cells were producing MMP-1 and -2, but also TIMP-1. Remarkably, the MMPs and TIMP-2 were showing nearly no presence in mast cells.

Statistically significant results were demonstrated for mast cells and fibrocytes or fibroblasts related to the enzymes. In detail the examined MMPs showed, in comparison to other structures or cells, a very low expression with statistical significance in mast cells. The fibrocytes and –blasts were showing statistically significant expression values compared to other structures and cells for all enzymes except for MMP 1. The expression of MMP-2 was significantly higher in fibrocytes and –blasts than the expression of other examined enzymes in these cells.

Particularly MMP-1 was showing significant results in relation to the grading of gingivitis. This MMP was upregulated in all cells and structures examined except for mast cells. Similarly, MMP-2 was showing an increased expression with higher degree of inflammation in the lamina propria, the inflammatory cells and the perivascular inflammatory infiltrates.
The degree of inflammation negatively correlated to TIMP-2 expression in the lamina propria and fibrocytes and blasts. For MMP-8 and TIMP-1 no similar relationships were detected.

The results regarding fibrocytes/-blasts and mast cells are suggestive of an interaction between these cells by mast cell mediators. These mediators might lead to an overexpression of MMP-1 in fibrocytes/-blasts and the remodeling of gingival and periodontal tissues.

The simultaneous inverted downregulation of TIMP-2 supports this hypothesis. Exhaustion or lower production of this enzyme might trigger the inflammatory process. Similar results are available for MMP-2. Assuminngly, TIMP-1 is not involved in the remodeling of feline gingiva. Regarding the ambiguous immunohistochemical reaction of MMP-8 in some parts of the positive control tissue, it is possible that the reaction of this antibody, perhaps because of the high species specificity, was not consistently successful.

No significant results of MMP and TIMP expression in gingiva were shown concerning the resorptive lesions, indicating that a correlation between resorptive lesions and the presence of MMP and TIMP in gingiva is unlikely. Further investigations are required in dental hard tissues.

Overall, it was possible to detect MMP-1 and -2 and also TIMP-1 and -2 plus MMP-8 irregularly in different cell types of feline gingival tissue. A distribution profile of these enzymes in feline gingiva was created, particularly mast cells and fibrocytes/-blasts showed significant differences in comparison to other cell types. MMP-1 and -2 and also TIMP-2 showed significant results with regard to gingivitis grading. These results indicate an interaction between these enzymes in the pathogenesis of gingivitis.

Literature

metalloproteinase-8 (MMP-8) and MMP-9 are increased in chronic periodontal disease and decrease after non-surgical periodontal therapy. Clinica Chimica Acta 2009;409:117-122


The effect of feline chronic gingivostomatitis on the esophageal and gastric mucosa – preliminary results

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Feline chronic gingivostomatitis (FCGS) is characterized by severe inflammation of the oral cavity, severe pain, dysphagia and debilitation which may occasionally lead to euthanasia. The disease is characterized by ulcerative or/ and hyperplastic lesions of the oral cavity and pharynx.

Studies concerning the effects of FCGS have only been limited to the oral cavity and the pharynx. However, the digestive system as a whole extends from the oral cavity to the anus. Various inflammatory conditions can affect every part of it. Feline chronic gingivostomatitis is an inflammatory disease of unclear cause. The objective of the current prospective clinical study is to investigate the potential effect of FCGS on the esophagus and stomach. For this purpose, 34 cats suffering from FCGS have undergone endoscopy of the esophagus and stomach. Moreover, the pH of the saliva and esophagus has been estimated with a portable ph meter. Ten cats undergoing anesthesia for elective surgeries have been used as witnesses.

Preliminary results of the current study reveal esophagitis of various degrees in all cases of FCGS. Esophagitis appears to affect the anterior and posterior parts of the organ in most cases. Gastritis was observed in a limited number of animals. In addition, differences in the saliva and esophageal pH have been recorded between the study group and the witnesses.

It seems that FCGS affects a great part of the digestive system. The clinician should consider investigating and perhaps treating cats with FCGS for esophagitis and gastritis at the same time.
Mandibular hypertrophic osteodystrophy in a six-months old German Shepherd dog

Elham A. Hassan & Faisal A. Torad

Hypertrophic Osteodystrophy (HOD) is a developmental disease process of unknown etiology affects primarily young rapidly growing giant breed dogs. Very few literatures have been published reporting Mandibular HOD. The present study is a report of a case of Mandibular HOD in a 6 months old German shepherd dog admitted with decreased appetite since one month, difficulty in chewing, opened mouth with continuous drooling of saliva. Physical examination revealed thickened mandible with filling of the intermandibular space with hard material. By opening the mouth, the tooth was loose. The intermandibular groove could not be identified. Radiography revealed thickened mandible when compared with mandible of the same age and breed. The right half of the intermandibular space was filled with radiodense bone-like structure when compared with the left half. The case first gave an impression of bone neoplasm. So, a bone biopsy from thickened mandible was performed and histopathological examination was carried out. Histopathological study revealed hypertrophic Osteodystrophy (Osteodystrophy Fibrosa) where multiple hypertrophied chondrocytes, calcified cartilage specules devoid of surface osteoid surrounded by hemorrhage and massive cellular infiltration (neutrophils and mononuclear cells). Few osteoclasts were also noticed. In hematologic examination, serum alkaline phosphatase, and serum calcium and phosphorus values were within normal range. Trial to manage this case
included administration of ascorbic acid (1 gm daily) for one month but a marked thickening in fore limbs (distal extremity of radius and ulna) and hind limbs (distal extremity of tibia and fibula) was noticed and radiography revealed large osteophyte formation that could be differentiated from bone neoplasms. Euthanasia was advised.
Temporary palatal prosthesis in a puppy with cleft palate

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Introduction

A cleft palate is a congenital craniofacial defect. Surgical treatment can be performed, but most puppies die or are euthanized before. Puppies which survive are hand raised by tube feeding. This case shows an alternative way of raising a puppy with a cleft palate.

Clinical case

A three day old boxer puppy was presented at the clinic due to low body weight gain and nasal discharge. During examination of the oral cavity a cleft palate was diagnosed. The puppy showed no signs of pneumonia. It was treated with amoxicillin - clavulanic acid and bromhexin and was inhaled with ambroxol. Bottle feeding was additioned by tube feeding. With ten days of age a temporary palatal prosthesis was made while the puppy was asleep. For the prosthesis a mouth guard for children (normally used for protecting the teeth during sport) was used. The mouth guard consisted of thermoplastic silicon, which one could reform by heating it in water. Only a small piece of the mouth guard was used and after softening, a plate which had the size of the upper jaw, was produced. When the optimal size and thickness was achieved, the plate was softened again and was pressed against the upper jaw of the puppy.

The obturator was only inserted into the mouth for bottle feeding. The puppy showed a fast and normal milk intake. Small amounts of nasal discharge were occasionally observed after drinking, but there were no signs of milk aspiration. Because of the fast growth of the puppy a new prosthesis had to be made about every two weeks. At an age of six weeks the milk started to be mixed with royal canine starter mousse. The hole of the feeding teat was enlarged and the puppy was able to chew the starter mousse out of the feeding teat, while wearing the prosthesis. With three month of age the food was changed to dry dog food and water, both of which the puppy was able to take in without wearing the prosthesis. Corrective surgery by using the „von Langenbeck“ technique was done at the age of five month and a weight of 14 kg.
Conclusion

Hand raising a puppy by tube feeding is time consuming and needs veterinarian monitoring and the medical treatment and the surgical coverage of a cleft palate are expensive. In our case the manufacture of the palate prosthesis was cheap and easy but effective and no anaesthesia was needed. These are important aspects when it comes to the willingness of owners to raise a puppy with a cleft palate. Furthermore the manufacture of the palate prosthesis can be once shown to the owners and later be done by the owners themselves. This allows the puppy to grow up in a normal social environment. Thus suckling is possible which is important for the physiological jaw morphology and the proper development of the muscular forces during orofacial growth. At the same time the risk of milk aspiration and pneumonia are minimized.

Literature


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Objective: The diagnostic yield of cone-beam computed tomography (CBCT) was compared to standard intraoral radiographs. Two patient groups were evaluated: (A) small brachycephalic dogs with dental disease and (B) small dogs with maxillofacial trauma.

Study design: Prospective studies

Methods: Eighteen small brachycephalic dogs with dental disease and 23 small dogs with maxillofacial trauma were included in this study. Both groups were evaluated by full-mouth dental radiographs and CBCT, including a panoramic view and a tridimensional (3D) reconstruction. Twenty-one predefined anatomic landmarks and 31 predefined dental/maxillofacial disorders were evaluated for each patient by all means of diagnostic imaging. Pairs of tests on the same teeth for all diagnostic methods (intraoral radiographs, CBCT, panoramic and 3D views) were evaluated using the McNemar’s Chi-square test.

Results: In part (A) the benefits of CBCT images compared to dental radiographs in brachycephalic dogs, especially in areas of crowding and rotation of teeth, will be discussed. Part (B) will focus on the importance of comprehensive diagnostic imaging in patients with maxillofacial trauma.

Conclusion: The diagnostic benefits of CBCT in small brachycephalic dogs with dental disease and small dogs with maxillofacial trauma will be discussed.

Clinical relevance: Small brachycephalic dogs with dental disease and small dogs with maxillofacial trauma are two indications for CBCT where this modality can greatly contribute to improved patient care.
Surgical treatment of bilateral TMJ ankylosis in a cat

Introduction

Temporomandibular joint (TMJ) ankylosis, is a disorder that causes a restriction in the mouth opening, by a partial or complete immobilization of chewing movements, due to the formation of a fibrous tissue or bone, following trauma, joint disease, neoplasia or previous surgeries.

Clinical symptoms are: weight loss, malnutrition, poor feeding, dehydration, poor and dull hair, ptyalism, masticatory muscle atrophy, facial deformity, lateral mandibular deviation and possible obstruction airway.

They are two types of TMJ ankyloses described:

• True or intra-articular ankylosis. The cause is always in the joint, by any disease that produces inflammation and formation of fibrous tissue as a result of dysplasia, condylar fractures, luxations/subluxations, osteoarthritis and joint’s neoplasia.

• False or extra-articular ankylosis. The etiology is diverse, and is caused by any condition that produces a restriction in mandibular movement, the formation of fibrous or bone adhesions (aberrant callus or exuberant) between the zygomatic arch and the coronoid process and/or branch of the mandible, as a result of a fracture, infection (otitis media, retrobulbar abscesses or cysts), craniomandibular osteopathy and tumours (chondromas, osteomas and osteosarcomas).

The recommended treatment for these diseases, except in cases of luxations and subluxations, is surgical and involves resection of bone and fibrous adhesions that block the TMJ and prevent the opening of the mouth.

Clinical case

A seven years old cat, female, with locked mouth since six months ago, came to dentistry and Maxillofacial Surgery Service of the University of Madrid. In the clinical examination we realize an important weight loss.
The oral exploration showed:

- Complicated crown fracture of teeth 204, 403 and 404.
- It was not possible to make a more complete examination due to poor mouth opening (1 cm).
- The TMJ palpation, was painful, appreciating inflammation and severe atrophy of the temporal and masseter muscles.

A CT imaging was proposed, but due to economic problems it was not accepted. The radiographic study showed new bone formation between TMJ and the zygomatic arch, the left side being most affected.

We decided to do a bilateral mandibular condylectomy and removing all the bony adherences and aberrant tissue.

It was necessary to make a tracheostomy for intubation. After the skin incision along the ventral caudal edge of the zygomatic arch. After exposure of the condyle and delimited the exuberant bone, we resected the lateral portion of the condyle with a rongeur, then we made a cut along the osteotomy line with a hight-speed burr\(^7\). he proceeded to milling for the release of the ATM.

Subsequently we placed sterile autologous fat graft and bone wax (Bone Wax®, B.Braun Vetcare) on the osteotomy surfaces, to prevent the formation of a fibrous tissue or bone callous, that may lock the mouth opening again.

Finally, the extraction of fractured teeth was performed.

After surgery, the excellent mobility of the jaw was found.

In the postoperative radiological study, the correct exuberant bone remodeling and no remains of the TMJ condyle was observed.

The patient remained 24 hours in the Intensive Care Unit of the Hospital, where they proceeded to place a esophagostomy tube to ensure proper nutrition.

Postoperative treatment consisted of the administration of antibiotics, analgesics, opioids and and gastric protector.

Reviews at 2 days were scheduled, and every two weeks. The outcome was positive, with no unexpected complications.
Discussion

The mandibular condylar fractures and TMJ ankylosis, are rare in veterinary medicine and particularly cats. They are usually caused by trauma or malignancy. They can lead to osteoarthritis, ankylosis and finally need a condilectomy.

Unilateral TMJ ankyloses clinical features is facial asymmetry, but in the bilateral, this change are not clear and the animal has more difficulty eating.

The radiological diagnosis is very important and essential for the study of changes in the TMJ, but in some cases complex has its limitations and is preferred by TC study.

In almost all cases of TMJ ankylosis, due to the minimal opening of the mouth, intubation is necessary throught a temporary tracheostomy.

There are many treatments for this pathology described, as are the opening (general anesthesia) forced, progressive and repeated the mouth alone or combined with corticosteroid administration. These treatments are ineffective, and ankylosis reappears, and do further trauma to the soft tissues of the area, which aggravate the pathology.

In the case described, due to the severity of the injury, it was decided to remove all of the aberrant bone tissue with surgical drill, resulting in the release of blocked joints.

There are many complications of this technique, all described in the literature. The most common are: opened jaw, malocclusions, facial nerve injuries, injuries of the maxillary artery, exophthalmos and ankylosis recurrence.

Findings

The surgical management by condilectomy and abnormal bony tissue resection is an effective technique to allow rapid mouth opening and rehabilitation of chewing movements. It is recommended to prevent recurrences, the interposition of a material between the ostectomy surfaces (bone wax and/or autologous fat graft).
References

Canine transmissible venereal tumour (TVT) is the only known naturally occurring tumor that can be transplanted as an allograft across major histocompatibility barriers within the same species, and even to other members of the canine family, such as foxes, coyotes and wolves. It is transmitted from one dog to another via direct transplantation of tumor cells onto abraded mucosa, either by sexual or oral contact. It appears to be more common in temperate climates and large cities. Intact, free-roaming dogs are at a greater risk.

The tumor begins as small hyperemic papules that enlarge to become lobulated or verrucous, red, friable masses that may bleed, ulcerate or become necrotic. It occurs primarily on the genitalia and is found on the penis, prepuce, scrotum, or perineal area in the male. In the female it may be found in the vagina, vulva, on the labia, or the internal genital tract. Although these tumors frequently regress spontaneously, they may cause death due to metastasis to vital locations. Metastasis can occur to the regional lymph nodes, eye, skin, lips, oral mucosa, tonsils orbit, liver, lungs, nasal cavity, and brain.

Immune suppression may play a role in the severity of metastatic lesions. Transmissible venereal tumors may be located in extragenital areas (e.g. eyelid, orbital space, nasal passage, and skin) without genital presentation. Ocular tumors may be flesh colored and protrude into the anterior chamber attached to the iris.

Impression smears or aspiration cytology will show round to oval cells with prominent nucleoli, scant cytoplasm, and multiple clear cytoplasmic vacuoles. There are numerous mitotic figures and the nuclear:cytoplasmic ratio is less than one.

Immunocytochemistry supports a histiocytic origin, although they appear not to be of canine origin having an abnormal karyotype with 59 chromosomes compared with a normal of 78 in dogs.
Management of submandibular and cervical necrotising fasciitis in a 4 month-old Leonberg dog

Florian Boutoille

Case history: Infection localised to the gingiva and the alveolar bone around the right mandibular decidual canine tooth (804) of a 4 month-old Leonberg dog progressed over 4 days to extensive necrosis of the full-thickness of skin and subcutaneous tissue in submandibular and cervical area. A successful outcome was achieved using large surgical debridement, osseous curetage and intensive supportive care, followed by long-term (5 weeks) wound management with silver-containing hydrofiber dressing using tie-over technique and surgical closure using advancement or rotational flaps performed in three steps.

Clinical findings: Clinical findings included severe pain, pyrexia and full-thickness loss of skin from the mental area below 804 to the sternal manubrium. CT-scan performed at the second day of the infectious process showed bone lysis on the site of 804 and around 404 tooth germ, loss of density between the mandibular bone and soft tissue and no visualization of the cutaneous vascularisation in right mental and submandibular area after contrast enhancement. Surgical debridment revealed extensive necrosis of the skin, adipose and muscle tissues. Escherichia coli was cultured from deep tissue samples.

Diagnosis: Necrotising fasciitis associated with E. coli infection.

Clinical relevance: Necrotising fasciitis is a rare, rapidly spreading, life-threatening, bacterial, soft tissue infection. It is a rapidly spreading inflammation resulting in necrosis of the fascial planes and the surrounding tissue. When not diagnosed and treated quickly and aggressively, it can be fatal. The wound management by silver-containing hydrofiber dressing using tie-over technique was particularly effective to enable the success of the reconstructive skin surgeries.
Inability to fully close the mouth in cats is a common emergency scenario in dentistry. It is very important not to make premature decisions prior to having performed a thorough oral examination. Sometimes this examination should be done under sedation or anesthesia because the patient may be painful and not allow you to fully assess the situation. Emergency clinicians may refer the patient with a presumptive diagnosis of TMJ luxation. Although this may be one of the possible causes, the list of differential diagnoses should include other etiologies as open-mouth jaw locking due to TMJ dysplasia and advanced periodontal disease with luxation of the carnassials and/or canines. Even though diagnostic imaging will definitely rule out a majority of differential diagnoses, all three conditions above can essentially be diagnosed by doing a good oral examination and accurate interpretation of the presenting clinical signs.

Three cases for three different etiologies:

**Case 1: TMJ Luxation**

Six year old spayed female Bengal, presented to the Emergency Service at Matthew J. Ryan Veterinary Hospital for a possible mandibular fracture. On awake oral exam, a small laceration was present on the left side of her face, and her right eye did not retpulse as easily as the left. Her mandible was displaced to the left and complete occlusion was impeded by the left canines making contact with one another. Matsa was able to open her mouth completely. No obvious mandibular fractures were palpated and the mandibular symphysis was intact. The patient was transferred to the Dentistry & Oral Surgery Service. Cat’s malocclusion was assessed and a right temporomandibular joint (TMJ) luxation was suspected to be the cause of the patient’s lower jaw deviation based on her clinical signs, but a left mandibular fracture could not be ruled out at this point through physical exam alone. Diagnostic imaging was required to elucidate the definitive cause of the jaw deviation. Blood work (CBC) and a NOVA screen were performed to assess patient’s overall health, and no significant abnormalities were detected. The rest of her physical exam was within normal limits. The cat was then placed under general anesthesia and dental radiographs were obtained. The dental radiographs revealed that right TMJ was luxated, thereby causing the deviation of her
jaw. No fracture of either mandible or visible upper jaw/palate was noted. Right TMJ was then reduced by placing a pencil between her upper right fourth premolar and lower right first molar (the carnassial teeth). Her jaws were then gently closed while the pencil was rotated in a counterclockwise direction, which resulted in the reduction of the luxation. Non-dissolvable sutures (with soft rubber tubing to reduce the pressure on her skin) were placed at the corners of her mouth to prevent patient from opening her mouth too widely and potentially causing another luxation of the TMJ while the connective tissue around the joint heals. The non-dissolvable sutures remained in place for 2-3 weeks because they decreased the range of motion of her jaw and helped reduce the chance of recurrence.

In the recheck exam the area around the sutures was slightly red and swollen indicating mild inflammation from the sutures. The patient did not have any oral discomfort and her range of motion of her jaw appeared normal. The remainder of her physical and oral examination was normal.

**Case 2: Open-Jaw Locking**

One year old female intact Persian, presented to Matthew J. Ryan Veterinary Hospital’s Dentistry and Oral Surgery Service for a history of an intermittent right open-mouth jaw locking. The patient first had an episode of lock jaw 4 month before, which was replaced by her primary care veterinarian. No recurrence of lock jaw occurred until one week before the visit to Matthew J. Ryan Veterinary Hospital’s Dentistry and Oral Surgery Service, when patient’s jaw locked three times. Each time, she was taken to the primary care veterinarian where she was sedated and the jaw repositioned. The lock jaw episodes generally occurred while yawning, grooming her neck, or after eating. The patient otherwise had a good appetite and eat normally. The patient had a CT scan performed at one Hospital in Ottawa. The scan showed no abnormalities, which is consistent with early stage open mouth jaw locking. On an awake oral exam, there was no evidence of TMJ laxity or jaw lock. She had mild gingivitis of her upper teeth and inflammation in the back right region of her mouth. The patient was offered a can of salmon wet food; she had a good appetite and atereadably, and she did not have an episode of jaw locking in the exam room. She was placed under general anesthesia. The jaw and temporomandibular joints were evaluated. While cat presented for lock jaw of her right side, the left side also was able to lock. It was therefore recommended to also perform the surgery on both sides. In order to address cat’s locking jaw, a coronoidectomy and a partial zygomeectomy of the right and left coronoid processes and zygomatic arches were performed. Oral radiographs after her procedures were taken to evaluate the extent of the bone excision. A complete oral exam was performed and revealed mild gingivitis and calculus accumulation of her teeth, and a professional dental cleaning was performed.

**Case 3: Advance periodontal disease with tooth extrusion and luxation**

Five year old spayed female domestic short hair, was presented to Matthew J. Ryan Veterinary Hospital’s Dentistry and Oral Surgery Service because she could not close her mouth. There was no known history of trauma, but she was not always supervised while outside. On oral exam, her left mandibular canine tooth was extruded and luxated, causing it to contact the left maxillary canine and prevent closing of the mouth. Her left
maxillary canine tooth was extruded. There was evidence of periodontal disease and tooth resorption in several teeth. Radiographs of her temporomandibular joints (TMJs) were performed. Radiographs identified tooth resorption in the left maxillary third incisor, left and right mandibular third premolars, and left mandibular first molar. Both right and left temporomandibular joints appeared radiographically normal. Teeth that were extracted with an open surgical technique include left maxillary and mandibular canines, the left maxillary third incisor, the right mandibular third premolars, and the left mandibular first molar. A crown amputation was performed on the left mandibular third premolar. The gingiva over all extraction sites was closed with absorbable sutures. The rest of Bella’s teeth were scaled and polished.

In the recheck exam her extraction site were healed.
Importance of correct radiographic labeling and advanced imaging in the case of a fused vertical ramus to the zygomatic arch

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Dogs or cats with an inability to open the mouth may present in poor bodily condition and/or anxious. Differentials include temporomandibular joint ankylosis, zygomaticomandibular ankylosis, craniomandibular osteopathy, tetanus, or masticatory myositis.

A rescued 1-year old male Samoyed-Cross dog was presented in lean bodily condition with an inability to open his mouth more than a few mm for several months. Skull radiographs and 2M antibody titers had been obtained, and the pet examined with further radiographs from 2 different veterinary hospitals. At one point force was utilized to try to open the mouth- with no success. The 2-M antibody titer was negative. One set of radiographs was incorrectly labeled right and left compared to the previous set. The pet was referred to the author and a CT Scan to fully evaluate all injuries, including evaluation of the TMJs was obtained. Diplomate Dr. Philippe Hennet reconstructed a 3-D image from the CT images. This image clearly illustrated the right-sided fusion of the vertical ramus to the zygomatic arch, the extent of such, as well as resulting changes to the right TMJ.

The plan was to, and the plan followed, to make a skin incision over the zyzomatic arch, full thickness to the bone, from below the orbit rostrally to the level of the TMJ caudally. Periosteal elevators would reflect the periosteum lying over the zygomatic arch dorsally and ventrally. Once exposed, the attachment of the temporal muscle dorsal to the zygomatic
arch and of the masseter ventral to the zygomatic arch would be visualized.

The attachments to totally separate them from the bone would be made, and the ramus exposed by reflecting dorsally the temporal muscle ventrally the masseter.

Osteotomy could now proceed. The procedure firstly to remove the zygomatic arch from just caudal to M1, full height from the ventro-caudal aspect of the maxilla to the orbital rim dorsally, to just rostral of the TMJ. The bone cut would just be leveled to the caudal aspect of the ramus. Secondly, the ramus would be amputated just dorsal to the TMJ and removed with the zygomatic arch. As this author does not own a piezoelectric bone surgery unit, a surgical bone bur would be utilized on a high-speed handpiece with water coolant.

The temporalis and masseter muscles would be sutured together and the fascia sutured as well using 4-0 Monocryl® simple continuous suture pattern. The skin was to be sutured with non-absorbable simple interrupted sutures.

Anesthetic protocol would include premedication with 0.01 mg/kg acepromazine with 0.4 mg/kg butorphenol, induction with propofol, and a propofol CRI for maintenance until the mouth could be opened and then the dog would be intubated and maintained on oxygen and isoflurane gas anesthetic. The neck would be shaved and prepped for an emergency tracheotomy if required. A surgical plane of anesthesia was maintained well with this protocol and all monitoring parameters illustrated a stable anesthesia.

Post-operative radiographs would be obtained.

The owner was asked to encourage the patient to eat, gradually taking it from soft to kibble food. He was also to be encouraged to play daily with balls and Kong toys to exercise his jaw muscles.

The owner was to apply an ice pack wrapped in a towel to the incision for 10 min every hour for 8 hours and the skin sutures were to be removed in 12 days. The patient was placed on antibiotics and pain management. The following day the owner reported that the patient
was eating well, did not appear painful, and was energetic.

This case report demonstrates the importance of differential diagnosis, correct radiographic labeling, advanced imaging, professional collaboration, preparation for emergency airway access, and diligent anesthetic monitoring.

The EVDSFORUM is an e-bulletin entirely dedicated to Veterinary Dentistry. Besides covering news about the European Veterinary Dental Society (EVDS) and its members—including congresses, programs, members publications, etc.—it also publishes clinical studies and reviews in oral medicine and surgery with interest to EVDS members.

Introduction

Canine oral malodour is a common complaint from dog owners\(^1\). It arises mainly from the microbial metabolism of exogenous and endogenous protein substrates in the oral cavity and is exacerbated by periodontal disease and poor oral hygiene. Anaerobic bacteria present in dental plaque and periodontal pockets are responsible for the production of malodorous volatile organic sulphur compounds, such as hydrogen sulphide and methyl mercaptan\(^2-5\). A significant, positive correlation between the concentration of volatile organic sulphur compounds in breath and the perception of halitosis by people has been proven. Thus, measuring the salivary concentration of volatile organic sulphur compounds seems to be an appropriate method for determining oral malodour in dogs\(^6\).

Other volatile organic compounds (VOCs) produced by oral putrefaction processes, such as organic acids, ammonia and amines may also be involved\(^7,8\). Professional periodontal therapy can significantly reduce halitosis, but has to be associated with suitable homecare follow-up in order to prevent the re-establishment of the microbial load\(^4,5\). However, there may be some periods when dogs do not have professional cleaning, for example due to the inability of the dog to undergo general anaesthesia, and some pet-owners lack the compliance and dexterity required for appropriate tooth brushing. This can lead to the inadequate maintenance of gingival health. Consequently, there has been an increase in the interest in alternative solutions based on natural products and plant extracts to help improve canine breath\(^9\).

Cooldent\(^\circledR\) (Virbac SA, France) is a complementary feed which has been proven to be safe, palatable and effective in dogs for freshening breath\(^10,11\). The palatable and bi-divisible tablets contain pomegranate concentrate (0.1%), parsley essential oil (2%), peppermint essential oil (2%) and inulin (3.6%), the effects of which have been shown to be of great interest in dental care in dogs\(^12-15\).

The first objective of this study was to quantitatively and qualitatively assess the persistence in the saliva of the pleasant VOCs originating from the essential oils present in the finished Cooldent\(^\circledR\) product, after its ingestion by dogs known for their unpleasant breath. The second objective was to evaluate the impact of the Cooldent\(^\circledR\) product on the malodorous VOCs naturally present in the saliva of these dogs by measuring these
Materials and Methods

Five adult beagle dogs were included in this study. In each dog, gums were rubbed successively with 2 swabs for 30 seconds each in order to collect saliva before (T0), 5 minutes (T5m) and 1 hour (T1h) after the administration of Cooldent®. All the samples were stored between 2 and 8 degrees Celsius, before being analysed. More than 50 reference substances were screened for, mainly from the following families: alcohols, aldehydes, ketones and sulphur derivatives.

Time, incubation temperature, sorbent type, volume, rate of adsorption on the sorbent, as well as parameters related to desorption were adapted and optimised according to the nature of VOCs.

A statistical analysis was performed to assess the evolution of VOC levels over time (ANOVA test).

Results

Among all the substances screened for, 23 compounds (Table 1) were identified during the biochemical exploration conducted on the VOCs present in the saliva of the dogs. The VOCs present in the highest concentration were dimethyl disulphide, trichloromethane, acetic acid and ethylbenzene.

<table>
<thead>
<tr>
<th>VOCs identified</th>
<th>Substance family</th>
<th>VOCs identified</th>
<th>Substance family</th>
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<tbody>
<tr>
<td>Trichloromethane</td>
<td>Other</td>
<td>15-Crown 5</td>
<td>Other</td>
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<tr>
<td>Disulfide dimethyl</td>
<td>Sulphur derivatives</td>
<td>Phenol</td>
<td>Alcohols</td>
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<tr>
<td>Ethylbenzene</td>
<td>Other</td>
<td>Ethanol 2-phenoxy</td>
<td>Alcohols</td>
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<td>Benzene (1-methylethyl)</td>
<td>Other</td>
<td>Benzene</td>
<td>Other</td>
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<td>Nonanal</td>
<td>Aldehydes</td>
<td>2-pentanone</td>
<td>Ketones</td>
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<tr>
<td>Disulfide trimethyl</td>
<td>Sulphur derivatives</td>
<td>Cyclopentasiloxane, decamethyl</td>
<td>Other</td>
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<td>1-hexanol, 2 ethyl</td>
<td>Alcohols</td>
<td>Toluene</td>
<td>Other</td>
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<tr>
<td>Acetic acid</td>
<td>Acids</td>
<td>Benzene, propyl</td>
<td>Other</td>
</tr>
<tr>
<td>Benzaldehyde</td>
<td>Aldehydes</td>
<td>1-pentanol</td>
<td>Alcohols</td>
</tr>
<tr>
<td>2 propanol, 1 (2-methoxypropoxy)</td>
<td>Alcohols</td>
<td>1-octen-3-ol</td>
<td>Alcohols</td>
</tr>
<tr>
<td>Acetophenone</td>
<td>Ketones</td>
<td>Propanoic acid</td>
<td>Acids</td>
</tr>
<tr>
<td>Cyclobultane 1-butyl-2-ethyl</td>
<td>Other</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Total levels of these VOCs before and after product intake are presented in Table 2, as well as the ratio between the VOCs originating from the saliva of dogs and those from the Cooldent® product.

Discussion

The highest VOC levels originating from the essential oils present in the finished product were detected on the swabs that were rubbed on the gums of the dogs at T5m. These results may be consistent with the results published in another study performed by Navarro, showing
an improvement of oral odour as soon as 5 minutes after the dogs received the Cooldent® product\textsuperscript{11}. In fact, when chewing the product, the oral cavity functions like a bellow, forcing volatile flavour compounds from the essential oils of the product into the exhaling air to the nasal compartment. The processes involved in the release of pleasant flavour include an initial release from the product into saliva, partitioning from saliva to the air phase, and subsequently flavour removal via the retronasal route or through the mouth\textsuperscript{14}. This mechanism could explain the rapid (within 5 minutes) breath-freshening effect observed in the dogs having received a tablet of Cooldent® in the study done by Navarro\textsuperscript{11}.

In Dog 1, Dog 2 and Dog 4, the VOC levels significantly decreased from T0 to T1h. This observation can be explained by the fact that the essential oils not only cover up bad breath, but they can also help to neutralise malodorous VOCs. Indeed, the phenolic content from the essential oils of Cooldent® has antioxidant and antibacterial activity that can neutralise volatile sulphur compounds\textsuperscript{16}.

In Dog 3 and Dog 5, malodorous VOC levels were basically stable over time (no statistical difference between the different time points). In Dog 3, a lower level of molecules from the product had been detected than for other dogs at the same time point. However, we do not have information regarding the chewing behaviour of the enrolled dogs. One hypothesis might be that Dog 3 may have gulped the product rather than chewing it, leading to a reduced contact time in the buccal cavity and, thus a reduced neutralisation of the malodorous VOCs. In a future clinical study, it would be useful to collect all the clinical data, including the chewing behaviour and organoleptic assessment of the breath of the animals enrolled.

One of the limitations of this proof-of-concept study is the small number of dogs (5) included. The next step would be to perform the same analysis on a greater number of animals.

Another interesting point to explore via this method would have been the cumulative effect of the product. Indeed, in another study, it has been proven that the Cooldent® product is even more effective in freshening dogs’ breath after repeated use, probably due to the presence in this product of pomegranate and inulin, which act on the oral and non-oral causes of bad breath, respectively\textsuperscript{11}. Therefore, in a future study, it could be useful to repeat this experiment daily for at least one week.

**Conclusion**

To conclude, this preliminary study has shown that pleasant flavoured VOCs can be detected in the saliva of all dogs 5 minutes after product intake. This may lead to a rapid improvement of oral odour. Moreover, these VOCs are still detected in most dogs after one hour, showing a persistence of the flavoured compounds originating from the essential oils of the Cooldent® product, which is consistent with a prolonged breath-freshening effect.

The gradual decrease in the level of malodorous VOCs in the saliva of most dogs after the ingestion of Cooldent® could be due to the neutralisation of these components by the product.

---

**Table 2. VOCs measured in the saliva of dogs at different time points (ND = Not Determined).**

<table>
<thead>
<tr>
<th>Dog</th>
<th>T0</th>
<th>T5m</th>
<th>T1h</th>
<th>T0</th>
<th>T5m</th>
<th>T1h</th>
<th>T0</th>
<th>T5m</th>
<th>T1h</th>
<th>T0</th>
<th>T5m</th>
<th>T1h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dog 1</td>
<td>107.4</td>
<td>15.7</td>
<td>ND</td>
<td>41.3</td>
<td>ND</td>
<td>145.8</td>
<td>10.3</td>
<td>ND</td>
<td>49.9</td>
<td>2.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dog 2</td>
<td>37.2</td>
<td>13.8</td>
<td>2.5</td>
<td>35.5</td>
<td>2.4</td>
<td>33.7</td>
<td>1.4</td>
<td>41.1</td>
<td>1.4</td>
<td>141.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dog 3</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dog 4</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dog 5</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

$\sum$ VOCs (ng/swab): 1841.7, 1090.9, 790.9, 1576.3, 1574.6, 836.5, 1872.5, 1746.5, 1832.4, 696.7, 634.7, 425.3, 773.6, 733.1, 737.2
References

Objective: Bone grafts have the disadvantage of eliciting an immunologic response with subsequent graft rejection and infection of periodontal defects. The ability of a bone implant with nanosilver as an antimicrobial agent that has anti-inflammatory and anti-infection effects to promote wound healing has been chosen as the subject of this investigation. In this study, human bone strip and nanosilver particles were applied to periodontal fenestration defects in rats to elucidate the effects of nanosilver on periodontal wound healing.

Materials and Methods: Following surgery to create a bony window on the buccal aspects of mandibular molar roots, 24 male Wistar rats were divided into three groups according to bone graft and nanosilver application (group 1: no treatment; group 2: bone graft only with dosage of 5 microgram; group 3: bone graft and nanosilver with dosage of 20 ppm). Animals were killed after 28 days and the mandible taken for histological examination. New bone and cementum formation (including area and thickness) in the defect were analyzed and compared.

Results: In general, minimal new bone was observed in the surgically created defects in group 1, whereas a complete osseous healing occurred in all treated animals. New bone formation (both in area and thickness) was significantly influenced by the bone graft and the examining level, whereas new cementum formation was affected nanosilver. An increase in bone and cementum formation was noted in all animals in the third group when compared with the control and bone graft (second) groups. Among the test groups, the greatest new bone and cementum formation was noted in the second group. New cementum thickness increased on the cementum surfaces of the defects compared with the dentinal surfaces in all study groups.

Conclusion: An increase in new bone and cementum formation was noted after applying a bone strip xenograft to a periodontal fenestration defect in rats. Therefore, we suggest that BMP-6 may play a certain role in periodontal regeneration. The results showed that nanosilver has the capacity to absorb fluids and swell. It also showed enhanced antibacterial activity, bioactivity and controlled degradation in comparison to the control group. Cell viability studies proved the non-toxic nature of the nanosilver particles. Cell attachment and cell proliferation studies revealed the attachment and spreading nature.
of cells. All these studies revealed that these antibacterial nanocomposites could be a promising approach for the management of periodontal defects.

The electronic Veterinary Dental Scoring (e-VDS) is an online dental charting software presented by the European Veterinary Dental Society and available for all veterinarians. It is a valuable tool to improve communication with your clients. The core part is a printout with your clinic data and logo, which the clients receive after dental services (in E, D, F, I, Sp, PT). While the basic version is a tool which is mainly aimed to support the general practitioner in his daily work, the advanced programme provides a wide variety of scoring options for more advanced veterinarians. The scorings can be kept as patient’s history sheets.

The results are scored with mouse clicks on dental charts (created and with the kind permission of David Crossley) for dogs and cats, for primary and permanent dentition. Clinical findings are scored in coloured marginal markings, while treatments and missing teeth in coloured full markings of the tooth. Currently there are only English, German and French available as screen language.

Have a try on http://scoring.evds.org
Presence of heart murmurs due to myxomatous mitral valve disease (MMVD) in dogs may influence decisions of veterinarians and dog owners regarding dental procedures involving anesthesia due to unknown risk of cardiac complications. Knowledge regarding changes in heart rhythm during and after anesthesia in dogs with MMVD is sparse.

The aim of the study is to evaluate the influence of sevoflurane anesthesia on the presence and number of heart arrhythmias in Cavalier King Charles Spaniels (CKCS) with different degrees of mitral regurgitation due to MMVD.

Twenty age- and sex-matched CKCS with and without mitral regurgitation murmurs due to MMVD, and in addition 20 CKCS with different degrees of mitral regurgitation due to MMVD will be evaluated during elective dental procedures. A baseline cardiac examination including auscultation, echocardiography and continuous 24 hour electrocardiography (ECG) is performed at least 3 to 7 days before the dental procedure. In addition, continuous ECG is recorded during sevoflurane anesthesia and 24 hours following anesthesia. The anesthetic protocol used is standardized to include administration of methadone and diazepam prior to induction of anesthesia with propofol and ketamine. Sevoflurane is used to maintain anesthesia.

Presence and number of ventricular premature complexes (VPC) and second degree atrioventricular (AV) blocks are registered from continuous ECG recordings. Results will be presented at the meeting. Preliminary findings show no associations between degree of mitral regurgitation due to MMVD and number of heart arrhythmias in CKCS during and 24 hour following sevoflurane anesthesia.
The relation between chronological stage of salivary mucoceles and their ultrasonographic appearance confirmed by histopathological findings

Faisal A. Torad & Elham A. Hassan

Salivary mucoceles are reported to be the most common disease affecting the salivary glands, mainly affecting the sublingual glands and rarely the zygomatic gland. The present study was performed on 19 dogs of different breeds (7 German shepherd, 6 Pitbull, 3 Rottweiler and 3 mixed breed). These dogs admitted with salivary mucoceles (12 submandibular, 4 sublingual, 3 zygomatic mucoceles). Time of onset of these affections till admission was varied (2 cases admitted within 3 days, 4 cases admitted after 2 weeks, 5 cases admitted after one month, 3 cases admitted after 2 months and 2 cases admitted after 3 months). Cases were diagnosed based on physical, radiographic and ultrasonographic examinations. The chronological stage of salivary mucoceles was correlated to their ultrasonographic appearance and confirmed by histopathological findings that performed in cases underwent surgical interferences (9 cases). In dogs admitted after 2 weeks from onset, mucocele appeared as a round echogenic structure with a large volume of anechoic content. The histopathological findings showed, necrosis and hypertrophy of the acini of some lobules with multiple inflammatory cells infiltration. In dogs admitted after one month, ultrasound images showed more hypoechoic content (glandular tissue) with a decrease in the volume of anechoic content. The histopathological findings included increased connective tissue stroma between the lobules. Fibrosis was detected between the acini as well as between
the lobules. In dogs admitted after 2 months, ultrasonography showed a grainy or mottled, heterogenous appearance with a further decrease in the volume of anechoic content. In dogs admitted after 3 months, ultrasonography revealed marked hyperechoic appearance with distal acoustic shadowing consistent with osseous metaplasia or dystrophic calcification. The histopathological findings revealed metaplastic changes in the duct system.
Biological evaluation of a new pulp capping material developed from Portland cement in dogs

Ehab E. Hassanien¹, Ashraf M. Abu-Seida², Ahmed M. Negm¹ & Mohamed M. Nagy¹

Introduction

Pulp capping is defined as the treatment of exposed vital pulp by the application of capping materials to induce the dentinogenic potential of pulp cells (Schröder 1985). An ideal pulp capping material must be capable of inducing the formation of reparative dentin as well as acceptable biocompatibility and strong antibacterial activity (Mjör et al. 1991)

Calcium hydroxide is considered the gold standard of pulp capping agents. However, the resultant incomplete dentin bridge with tunnel defects may lead to the failure of pulp capping (Al-Hezaimi et al. 2011).

Mineral trioxide aggregate (MTA) has been recommended as a pulp capping material due to its high biocompatibility and sealing ability than calcium hydroxide (Parirokh and Torabinejad 2010). Moreover, MTA induces the differentiation of dental pulp cells to odontoblast-like cells and produces thicker dentin bridges (Al-Hezaimi et al. 2011)

The success rate of MTA as a pulp capping material is higher than calcium hydroxide. However, MTA still has some limitations, including difficult handling characteristics, long setting time and relatively high cost.

Recently, the use of Portland cement as an alternative to MTA is gaining much popularity because of its lower cost, biocompatibility and ample availability (De Deus et al. 2006).

Taking the setting time, push out bond strength and pH value into account, the part I of this study concluded that addition of 10 wt% calcium hydroxide to Portland cement associated with 20% bismuth oxide produces a new pulp capping material (Port Cal I) with acceptable physical and adhesive properties. Therefore, the aim of the present part of this study was to compare the biological properties of the new material, MTA and Portland cement as pulp capping materials.

Materials and methods

Animals: This study was approved by the Ethics Committee of Faculty of Dentistry, Ain Shams University (2013/03END), and Animal Use and Care Committee at Faculty of Veterinary Medicine, Cairo University, Egypt. All surgery was performed under general anesthesia, and all efforts were made to minimize animal suffering and to reduce the number
of animals used.

A total of four male mongrel dogs aged approximately 4-6 months were selected for this study. Four teeth in three quadrants of each dog were included in the study summing up 48 teeth (12 teeth / dog). The dogs were classified into two equal groups according to the evaluation period (n=24); group I (3 weeks) and group II (3 months).

Each group was further subdivided into three equal subgroups (n=8) according to the used capping material including; subgroup (a): MTA, subgroup (b): Port Cal I (Portland cement + 20% bismuth oxide + 10% calcium hydroxide) and subgroup (c): Portland cement + 20% bismuth oxide.

Formation of the new material: Bismuth oxide (Loba Chemie, India) was incorporated into Portland cement (ASEC Helwan cement, Egypt) in the ratio of 20% by weight. The calcium hydroxide powder (ANALAR, Oxford laboratory. Mumbai, India) was then mixed with Portland cement in a ratio of 10% by weight.

The ingredients of the powder were blended together in a vibratory mixer for one hour. The resultant mixture was mixed with distilled water with a powder/water ratio 3:1 and the newly formed cement was designated Portland Cal I (Port Cal I).

Procedure of pulp capping: The anesthetic regimen for each dog included subcutaneous injection of Atropine sulphate (Atropine® ADWIA, Egypt) at a dose of 0.05mg/kg body weight and intravenous injection of Xylazine HCl (Xylaject® ADWIA, Egypt) 1mg/kg body weight as a premedication. The anesthesia was induced by using Ketamine HCl (Ketamine® EPICO, Egypt) 5mg/kg body weight given I.V. via a 20 gauge cannula. The anesthesia was maintained by 25mg/kg incremental doses of 2.5% solution of Thiopental sodium (Thiopental Sodium® EPICO, Egypt).

After general anesthesia, the teeth were disinfected by 0.5% Povidone iodine solution (Betadine® Nile company, Egypt). A class V buccal cavity was prepared approximately 1 mm coronal to the gingival margin with No. 2 Rose head carbide bur under copious normal saline irrigation in each tooth. Deepening of the pulpal floor for each cavity was done until the color of pulp tissue was reflected through the remaining dentin layer. Sterile sharp probe was used mechanically to expose the pulp. The capping materials were obtained by mixing the powder designated by each subgroup with distilled water on sterile glass slab using metal spatula to obtain a putty-like consistency; subgroup (a) “MTA” (Endocem Maruchi, Korea), subgroup (b) “Port Cal I” and subgroup (c) “Portland cement + Bismuth oxide”. This mix was placed on the exposure sites by a fine amalgam carrier and condensed lightly with a moistened cotton pellet. Final restorations were done by insertion of glass ionomer filling (Riva, SDI. Australia).

For pain and infection control, the dogs were given intra-muscular cefotaxime sodium at a dose of 10 mg kg -1 and diclofenac sodium at a dose of 1.1 mg kg -1 once/day for 5 days after surgery (Abu-Seida 2012).

Histopathological examination: Dogs were sacrificed after each observation period by using 20 ml of 5% Thiopental sodium solution rapidly injected through the cephalic vein. Blocks containing a single tooth with its surrounding bone were obtained. The teeth were fixed in 10% neutral buffered formalin for 72 hours. Specimens were then decalcified in 17% EDTA solution for 120 days. After decalcification, specimens were prepared as usual for histopathology. Serial sections (5μm thickness) that showed the deepest part of the cavity and the underlying pulp were selected for histological evaluation. These sections were stained with H&E for evaluation of the following:
Inflammatory cell count: For each slide, three representative fields were analyzed at X200 magnification as described by Tawfik et al. (2013). The total number of cells was then counted as a factor of 103.

Dentin bridge formation: Dentin bridge formation was graded by the scoring system reported by Min et al. (2009) as follow:

- Score 0: No dentin bridge.
- Score 1: Partial dentin bridge formation.
- Score 2: Complete dentin bridge formation.

Dentin bridge thickness was assessed through the H&E stained sections using the image analysis software (Leica Queen 500).

Statistical analysis: Data were analysed using SPSS (Statistical Packages for the Social Sciences 22, IBM, Armonk, NY, USA). All data were tested for statistical significance using ANOVA, Duncan’s test, Mann–Whitney U and Kruskal–Wallis non-parametric tests. Significance was established at P < 0.05.

Results

Inflammatory cell count

In group I (3 weeks), MTA showed the least number of inflammatory cell count (486.±42.6) followed by Port Cal I (1.009±24.0). While Portland cement + bismuth oxide showed the highest number of inflammatory cell count (1.256±68.4). The difference in the mean inflammatory cell count between MTA and the other two materials was significantly high (P<0.001).

In group II (3 months), the mean inflammatory cell count increased in all subgroups. However, MTA showed lower cell count (1.233±92.3), than Port Cal I (1.333±52.8) and Portland cement + bismuth oxide (1.566±50.4) with no significant difference.

Dentin bridge formation

In group I, MTA showed higher score of dentine bridge formation (0.25±0.46), than Port Cal I (0±0) and Portland cement + bismuth oxide (0.125±0.35) with no significant difference.

In group II (3 months), MTA subgroup exhibited significantly higher scores for dentin bridge formation (1.875±0.35) than Port Cal I (1.125±0.83) and Portland cement + bismuth oxide (1.375±0.74).

Discussion

The objective of the present study was to develop and evaluate a new material for pulp capping in dogs by mixing bismuth oxide and calcium hydroxide to the powder of Portland cement.

In the present study 20% bismuth oxide, with 10% calcium hydroxide were added to Portland cement powder. The addition of bismuth oxide was done for its radiopacifying effect in order to simulate MTA (Bueno et al. 2009).

Calcium hydroxide was added in attempt to modify the biological characteristics of
Portland cement. Also its mineralization effect which can be attributed to the hydroxyl ions released that induce an alkaline pH. This pH induces liquefaction necrosis in the superficial portion of the pulp, deeper portions of the pulp witness neutralization so stimulates hard tissue formation (Revathi et al. 2014).

Pulp exposure was performed by mechanical perforation of the cavity floor with a sharp probe to avoid extensive pulp damage and also to create a pulp exposure of uniform size. This approach was recorded before (Tusenda et al. 1995)

In order to assess the biological capability of the tested materials, histopathological evaluation was included to determine the inflammatory reaction of pulp tissues and dentin bridge formation.

During 3 weeks and 3 months periods, MTA showed lower number of inflammatory cell count than PortCal I while Portland cement+bismuth oxide showed the highest number of inflammatory cell infiltrate. These findings could be due to difference in particle size, lack of quality control; in case of Portland cement; increased lead and arsenic content, lower calcium release and carbonation reaction accompanied with Portland cement (Camilleri 2008). This finding contradicts Hwang et al. (2009) who found similar inflammatory reactions between MTA and Portland cement. In this respect the statistical value of PortCal I was better than Portland cement+bismuth oxide which might be due to the higher calcium release provided by calcium hydroxide addition.

The thickness and homogenesity of the dentin bridge formed by MTA might be due to its fine and homogenous particles. This finding disagreed with Razmi et al. (2006) who found no statistical difference between dentin bridge formed by MTA and Portland cement.

In PortCal I subgroup, the presence of calcium hydroxide might be interfered with the hydration reaction of Portland cement giving rise to a weaker and porous structure that did not stimulate more hard tissue formation than other subgroups (Yoshiba et al. 1995). This finding disagreed with Accortine et al. (2008) who postulated that calcium hydroxide has a faster dentin bridge formation despite of inflammation in human. The reason for this contradiction could be related to the differences in the experimental subjects. Also the amount of calcium hydroxide added in the present study was only 10% which might not be sufficient for hard tissue stimulation.

**Conclusion**

The addition of 10 wt% calcium hydroxide to Portland cement improves the possibility of dentin bridge formation.

**References**


bismuth oxide concentration required to provide Portland cement with adequate radio-
Effect of Ethylenediaminetetraacetic Acid (EDTA) on regenerative potential following revascularization of immature permanent non-vital teeth in dogs

Salma H. El Ashry1, Ashraf M. Abu-Seida2, Amr A. Bayoumi1 & Ahmed A. Hashem1

Introduction

Pulp necrosis of an immature tooth as a result of caries or trauma could arrest further development of the root, leaving the tooth with thin and weak walls that are prone to fracture. Endodontic treatment of such a tooth is difficult because thin walls do not allow much instrumentation, and the obturation might not provide predictable apical seal due to the large opening of the apex. Conventional treatment of such cases was multiple-visit apexification using calcium hydroxide (Cvek 1992). Although it was successful, it had several inherent disadvantages including unpredictability of apical closure, multiple visits, and probability of canal contamination between visits, difficulty in follow-up and subsequent treatment. In addition, long-term root canal dressing by using calcium hydroxide weakens the root structure and may lead to future fracture of the root (Doyon et al. 2005).

Apical barrier technique was introduced as a replacement for apexification with calcium hydroxide. In the apical barrier technique a barrier material is placed at the apex to facilitate obturation procedure. Mineral trioxide aggregate (MTA) is the material of choice for this technique considering its sealing ability, biocompatibility, hard-tissue deposition potential, and the ability to set in the presence of moisture. Reducing the number of visits, higher patient compliance and high success rate are the main advantages of this technique (Holden et al. 2008). However, the risk of future fracture may still exist because the root width will not increase in MTA apexification treated teeth.

Recently, regenerative endodontic procedures have gained much attention as it allows root maturation to continue by the generated vital tissue (Nagy et al., 2014). Presence of a smear layer is considered an important issue in regenerative endodontics because it inhibits the adherence of implanted pulp stem cells, potentially causing failure of the regenerative endodontic treatment. Its removal provides better sealing ability of the endodontic filling material to dentin, and prevents the leakage of microorganisms into oral tissues. The most commonly used chemical chelating agent to remove the smear layer from root canal walls is 17% solution of EDTA that is used as a final flush. Therefore, the aim of the present study was to investigate the impact of surface modification of
Materials and methods

A total of six healthy male mongrel dogs aged approximately 4-6 months with complete set of permanent dentition were selected for this study. Three premolars in each quadrant were included to sum 74 teeth and 144 root canals. This study was conducted according to the ethical committee protocol at the Faculty of Dentistry, Ain Shams University, Egypt. These teeth were divided into 3 equal groups (24 teeth each) according to post-treatment evaluation period including; group I (2 weeks), group II (6 weeks) and group III (12 weeks).

Each group was further subdivided into two experimental and two control subgroups according to the treatment protocol.

Anesthesia of the dogs

All dogs were premedicated with subcutaneous injection of atropine sulphate 0.05 mg kg-1 body weight and intramuscular Xylazine HCl 1.1mg kg-1 body weight. The anesthesia was induced by intravenous Ketamine HCl 5mg kg-1 body weight. The anesthesia was maintained during operation by using 25 mg kg-1 incremental doses of 2.5% solution of Thiopental sodium given intravenously.

Induction of periapical pathosis

Preoperative radiographs were carried out for all dogs. In all experimental and positive control teeth, an endodontic access cavity was done using size no. 2 diamond stone with conventional speed hand piece mounted on electric micro motor. A sterile file size no. 25 was used to disrupt the pulp tissue. The access cavity was left open for four weeks. Dogs were monitored radiographically for the evidence of development of periapical pathosis. The operated dogs were given oral carprofen tablets at a dose of 4.4 mg kg-1 per once daily for 15 days as a pain killer.

Root canal disinfection

After the infection period, dogs were anesthetized. Under complete aseptic conditions, the previously infected experimental teeth were re-entered. The canals were irrigated using 20 ml of 2.6% sodium hypochlorite solution and dried with paper points. The root canals were filled with 1-2 ml of the triple antibiotic paste. The triple antibiotic paste was prepared by mixing metronidazole 500 mg tablet, ciprofloxacin 250 mg tablet and doxycyclin 250 mg capsule. The access cavity was then sealed using temporary restoration for three weeks.

Treatment protocols

Under the same general anesthesia and aseptic techniques, the teeth were re-entered; the antibiotic paste was removed by copious irrigation using 20 ml of sodium hypochlorite 2.6%. The root canals were dried and treated according to different treatment protocols.
as follows:

Subgroup a (Blood Clot or revascularization): Hand file size no. 35 was inserted past to the canal terminus until bleeding was induced to fill the canal space just below the cement-enamel junction. MTA orifice plug was applied to seal the canal orifice. The access cavity was then sealed using glass ionomer filling.

Subgroup b (Blood clot and EDTA): Each canal was irrigated with 1ml of 17% EDTA solution which was kept for 2 minutes inside the canal. The canal was rinsed with another 1ml of 17% EDTA, flushed with normal saline solution and dried by paper points. Similar blood clot was induced as in subgroup (a). The canal orifice and access cavity were sealed as in subgroup (a).

Subgroup c (Positive control): This subgroup represented the pulp exposed teeth. Cotton pellets were inserted into the canal space and no temporary filling was applied.

Subgroup d (Negative control): This subgroup represented the untouched normal teeth.

Radiographic evaluation

Periapical radiographs were taken before and after induction of the periapical lesion and compared with follow up radiographs taken according to the group. Periapical radiographs were digitized using a transparency scanner then converted to 32-bitt TIFF files using Image-J analysis software. TurboReg plug-in software was used to standardize these radiographs. The following measurements were calculated in millimeter:

Increase in the root length

The root length was measured as a straight line between the cement-enamel junction and the apex of tooth.

Increase in the root thickness

The level of apical third was determined and fixed from the cement-enamel junction. The pulp space and root thickness were calculated at this level. Dentin thickness was measured by subtracting the pulp space from the root thickness.

Decrease in apical diameter

The diameter of apical foramen was measured.

The difference in root length, thickness and apical diameter was calculated and the percentage of increase in root length and thickness and apical closure were also calculated.

Histopathological evaluation

After each evaluation period, the dogs were sacrificed using overdose of intravenous thiopental sodium. Bone segments including the experimental and control teeth were dissected, fixed, decalcified and prepared for histopathology. Bucco-lingual sections of 6µm thickness were stained by hematoxylin and eosin dye for assessment of the following:
Periapical inflammatory cell scores

It was evaluated according to Holland et al. (2007) as 4 scores (0-3).

Periapical inflammatory cell count

For each slide, three representative fields were analyzed at X200 magnification. Total inflammatory cell number was counted as a factor of 103 using image analysis software (Image J, 1.41, NIH, USA).

Tissues in-growth in the pulp space

It was evaluated according to Tawfik et al. (2013) as 4 scores (0-3).

New hard tissue formation

Qualitative analysis: Criteria for histologic identification of hard structure (Huang 2009) included; dentin, cementum, bone and periodontal ligament

Quantitative analysis: This was evaluated according to Bezerra da Silva et al. (2010) as 3 scores (0-2)

Bone/root resorption

It was evaluated according to Bezerra da Silva et al. (2010) as two scores (0-1)
Table 1. Mean percentage increase in the root length and thickness and apical closure among different groups and subgroups. *Significant at P ≤ 0.05, Different letters in the same column are statistically significantly different according to Tukey’s test.

<table>
<thead>
<tr>
<th>Subgroups</th>
<th>Group I</th>
<th>Group II</th>
<th>Group III</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Increase in the root length</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subgroup (a)</td>
<td>4.9±1.2a</td>
<td>13.9±1.8a</td>
<td>16.3±1.3a</td>
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<tr>
<td>Subgroup (b)</td>
<td>5.1±1.1b</td>
<td>14.1±1.6b</td>
<td>16.5±1.4a</td>
</tr>
<tr>
<td>Subgroup (c)</td>
<td>0±0b</td>
<td>0±0b</td>
<td>0±0b</td>
</tr>
<tr>
<td>Subgroup (d)</td>
<td>6.2±0.5a</td>
<td>15.5±0.6a</td>
<td>19.7±0.4a</td>
</tr>
<tr>
<td>P-value</td>
<td>&lt;0.001*</td>
<td>&lt;0.001*</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td><strong>Increase in the root thickness</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subgroup (a)</td>
<td>4±0.6a</td>
<td>11.4±0.9a</td>
<td>13.4±0.8a</td>
</tr>
<tr>
<td>Subgroup (b)</td>
<td>4.7±1a</td>
<td>11.6±0.9a</td>
<td>13.6±1.3a</td>
</tr>
<tr>
<td>Subgroup (c)</td>
<td>0±0b</td>
<td>0±0b</td>
<td>0±0b</td>
</tr>
<tr>
<td>Subgroup (d)</td>
<td>6.1±0.7a</td>
<td>12.4±1.3a</td>
<td>15.8±1.4a</td>
</tr>
<tr>
<td>P-value</td>
<td>&lt;0.001*</td>
<td>&lt;0.001*</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td><strong>Apical Closure</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subgroup (a)</td>
<td>2.9±0.9b</td>
<td>16.4±1.4b</td>
<td>30.2±1.9b</td>
</tr>
<tr>
<td>Subgroup (b)</td>
<td>3.1±0.9b</td>
<td>16.5±1.1b</td>
<td>30.8±1.7b</td>
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<tr>
<td>Subgroup (c)</td>
<td>0±0c</td>
<td>0±0c</td>
<td>0±0c</td>
</tr>
<tr>
<td>Subgroup (d)</td>
<td>6.2±0.6a</td>
<td>20.5±1.2a</td>
<td>47±1a</td>
</tr>
<tr>
<td>P-value</td>
<td>&lt;0.001*</td>
<td>&lt;0.001*</td>
<td>&lt;0.001*</td>
</tr>
</tbody>
</table>

Table 2. Mean inflammatory cell scores and count among different groups and subgroups. *Significant at P ≤ 0.05, Different letters in the same column are statistically significantly different.

<table>
<thead>
<tr>
<th>Subgroups</th>
<th>Group I</th>
<th>Group II</th>
<th>Group III</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Inflammatory scores</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subgroup (a)</td>
<td>2.1±0.9a</td>
<td>1.3±0.5b</td>
<td>0±0.5b</td>
</tr>
<tr>
<td>Subgroup (b)</td>
<td>2.3±0.9a</td>
<td>1.4±0.5b</td>
<td>0.6±0.5b</td>
</tr>
<tr>
<td>Subgroup (c)</td>
<td>2.7±0.5a</td>
<td>2.9±0.4a</td>
<td>3±0c</td>
</tr>
<tr>
<td>Subgroup (d)</td>
<td>0±0c</td>
<td>0±0c</td>
<td>0±0c</td>
</tr>
<tr>
<td>P-value</td>
<td>0.001*</td>
<td>&lt;0.001*</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td><strong>Inflammatory cell count</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subgroup (a)</td>
<td>25±1.6c</td>
<td>16.4±2b</td>
<td>10.2±1.4b</td>
</tr>
<tr>
<td>Subgroup (b)</td>
<td>25.5±2a</td>
<td>16.6±1.8b</td>
<td>10.4±1.7b</td>
</tr>
<tr>
<td>Subgroup (c)</td>
<td>28.4±1.3a</td>
<td>36.7±1.9a</td>
<td>38.9±2.9a</td>
</tr>
<tr>
<td>Subgroup (d)</td>
<td>2.5±0.9c</td>
<td>2.3±1.2c</td>
<td>2.1±0.9c</td>
</tr>
<tr>
<td>P-value</td>
<td>&lt;0.001*</td>
<td>&lt;0.001*</td>
<td>&lt;0.001*</td>
</tr>
</tbody>
</table>

Apical closure

It was evaluated according to Thibodeau et al. (2007) as 2 scores (0-1).

Statistical analysis

Numerical data were presented as mean and standard deviation (SD) values. ANOVA, Tukey’s post-hoc, Kruskal-Wallis, Mann-Whitney U and Chi-square (x²) tests were used. The significance level was set at P ≤ 0.05. Statistical analysis was performed with IBM®SPSS®.
Results

Radiographic evaluation

Representative radiographs are shown in (Figure 1). All data are collected in (table 1).

Histopathological evaluation

Periapical inflammatory cell score and count

All data are collected in (Table 2)

Tissue in-growth in the pulp space

Connective tissue in-growth was recorded in some samples (Table 3). This tissue resembled the periodontal connective tissue with various amounts of inflammatory cells and noticeable angiogenesis (Figure 2a&b).

New hard tissue formation in the canal space

Data are collected in (Table 3). Cementoid like tissue of various shapes and thicknesses was seen in several samples (Figure 2c).

Bone/root resorption and Apical closure

Data are collected in (Table 4)

Discussion

Management of young permanent teeth with necrotic pulps and incompletely developed root presents a great challenge for clinicians. Recently, regenerative endodontic treatment was introduced.
Table 3. Mean tissue-in-growth and mineralization score among different groups and subgroups. *Significant at P ≤ 0.05, Different letters in the same column are statistically significantly different according to Mann-Whitney U test.

<table>
<thead>
<tr>
<th>Subgroups</th>
<th>Group I</th>
<th>Group II</th>
<th>Group III</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tissue in-growth</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subgroup (a)</td>
<td>0.6±0.5a</td>
<td>1.3±0.8b</td>
<td>1.9±0.4b</td>
</tr>
<tr>
<td>Subgroup (b)</td>
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<td>1.9±0.7c</td>
<td>2.6±0.5c</td>
</tr>
<tr>
<td>Subgroup (c)</td>
<td>0.3±0.5b</td>
<td>0.9±0.4b</td>
<td>0.9±0.4c</td>
</tr>
<tr>
<td>P-value</td>
<td>0.099</td>
<td>0.001*</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Mineralization</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>score</td>
<td></td>
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</tr>
<tr>
<td>Subgroup (a)</td>
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<td>0.7±0.5a</td>
<td>1.3±0.5a</td>
</tr>
<tr>
<td>Subgroup (b)</td>
<td>0.6±0.5a</td>
<td>0.9±0.4c</td>
<td>1.4±0.5a</td>
</tr>
<tr>
<td>Subgroup (c)</td>
<td>0±0b</td>
<td>0±0b</td>
<td>0±0b</td>
</tr>
<tr>
<td>P-value</td>
<td>0.166</td>
<td>0.003*</td>
<td>&lt;0.001*</td>
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</tbody>
</table>

Table 4. Results of Chi-square test for prevalence of bone resorption and apical closure among different groups and subgroups. *Significant at P ≤ 0.05, NC** Not computed because the variable is constant.

<table>
<thead>
<tr>
<th>Subgroups</th>
<th>Group 1</th>
<th></th>
<th></th>
<th></th>
<th></th>
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<th>Group 2</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>Subgroup (a)</td>
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<td>75</td>
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<td>41.6</td>
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<td>16.6</td>
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<tr>
<td>Subgroup (b)</td>
<td>9</td>
<td>75</td>
<td>5</td>
<td>41.6</td>
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<tr>
<td>Subgroup (c)</td>
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<td>83.3</td>
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<td>100</td>
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<td>100</td>
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</tr>
<tr>
<td>Subgroup (d)</td>
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<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
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<tr>
<td>P-value</td>
<td>0.007*</td>
<td></td>
<td>0.038*</td>
<td></td>
<td>0.002*</td>
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</tr>
<tr>
<td>Subgroup (a)</td>
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<td>0</td>
<td>4</td>
<td>33.3</td>
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<td>50</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Subgroup (b)</td>
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<td>0</td>
<td>4</td>
<td>33.3</td>
<td>6</td>
<td>50</td>
<td></td>
<td></td>
<td></td>
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<tr>
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<td>0</td>
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<td>P-value</td>
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<td></td>
<td></td>
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</tr>
</tbody>
</table>

The selected age of dogs was 4-6 months because the dogs have immature permanent premolars and can withstand general anesthesia at this age range (El-Ashry et al., 2013 and Hassanien et al., 2015).

In this study, the choice of EDTA to remove the smear layer was based on its ability to withdraw calcium from the inorganic calcium phosphate crystal lattice, resulting in demineralization of the superficial dentin layer. Also EDTA removes the loosely attached smear layer, leaving clean dentin surface with open dentinal tubules (Verdelis et al. 1999). The time of EDTA solution (17%) application was limited to 2 minutes to avoid the adverse effect on the stem cells of apical papilla as mentioned before (Galler et al. 2011).

In the present study, triple antibiotic paste was used for 3 weeks to disinfect the root canals before initiating regenerative procedures due to the polymicrobial nature of infected root canals (Windley et al. 2005).

Regarding radiographic findings, all samples of group I showed very minute changes in root length, thickness and apical closure with no significant difference between different
subgroups due to the short assessment time (2 weeks). In groups II and III, there was an increase in length, thickness and apical closure with no significant difference between different subgroups. This increase can be attributed to apical deposition of cementum-like tissue.

Regarding histopathological evaluation, all subgroups of group I showed mild to moderate inflammation. These findings are logically attributed to the immediate inflammatory reaction of the periradicular tissues to all of the performed treatment protocols.

In group II, most samples showed mild inflammation with no significant difference between subgroups (a and b). This is an indication for progressed healing of the periapical lesion due to eradication of the etiological factors (Cooper and Smith 2013). Positive control subgroup (c) showed significantly higher inflammatory reaction due to progression of the untreated infection.

In group III, most of samples were devoid of inflammation with no significant difference was found between both experimental subgroups a & b indicating progression of healing of the periapical lesion. Additionally, subgroup (c) positive control showed persistent inflammatory reaction due to continued bacterial irritation from infected root canal. These findings were in agreement with Wang et al. (2010) who recorded mild inflammatory reaction regardless of the new tissue in-growth following revascularization protocol.

Bone resorption after two weeks was evident in most samples of all subgroups due to the inflammatory mediators which favor the bone resorption (Li Kuo et al. 1998). After six weeks bone resorption score declined due to subsided inflammation in all subgroups. After three months the majority of experimental samples showed no signs of bone resorption due to the healing of the periapical lesion.

Histologic evaluation of the new soft tissue revealed that it resembled periodontal tissue and the newly deposited hard tissue resembled cementum. These results agree with Tawfik et al. (2013). Tissue in-growth and new hard tissue was minimal after two weeks without significant difference between both experimental subgroups. This may be due to the relatively short time, which was not enough for new tissue regeneration.

At six weeks, most of samples of subgroups (a) and (b) showed tissue in-growth within the apical third and reaching middle third of the canal, respectively. Hard tissue deposition was reported in some samples in this stage.

In groups II and III, higher significant level of tissue in-growth was noticed in subgroup (b), where EDTA was used, than in subgroup (a). This could be attributed to the partial demineralization of dentin wall induced by EDTA solution which helps the adherence of new tissues to inner dentin walls. This was in agreement with Galler et al. (2011) who reported an increased adherence of newly formed tissue to the root walls after exposure of dentin matrix using 17% EDTA solution.

Apical closure was evident in most of samples of experimental subgroups without significant differences between all groups due to approximation of newly deposited hard tissue at the apex. Similar findings were recorded before (Thibodeau et al., 2007 and Bezerra da silva et al., 2010).

Conclusion

Final rinse using 17% EDTA solution before revascularization has a positive impact on tissue interaction along dentinal walls without modification of the cell type.
References


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Complete blood count indices (N/L, P/L, MPV/PLT and PLCRi) in dogs with stage 3,4 periodontal disease and dogs with oropharyngeal tumours: evaluation and comparison with complete blood count indices of healthy dogs

Ana Rejec

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E-mail: anna.rejec@gmail.com

Background and objective

Systemic inflammatory response in dogs and humans with both oral inflammatory and neoplastic head and neck conditions has been described although with variable extent and in different patterns. Malignancy-associated systemic inflammatory response, assessed by complete blood count indices has been already shown to correlate with the pathogenesis and prognosis of certain cancers in humans while the data regarding the evaluation of the systemic inflammatory response due to periodontal disease by complete blood count indices are limited. Therefore, it was of our interest to investigate the differences between complete blood count (CBC) parameters and CBC indices including neutrophil/lymphocyte ratio (N/L), platelet/lymphocyte ratio (P/L), mean platelet to lymphocyte ratio (MPV/PLT) and platelet large cell ratio index (PLCRi) in one of the most prevalent canine chronic inflammatory diseases, periodontal disease (PD), and neoplastic conditions (benign and malignant) affecting dog’s oropharyngeal region in comparison with CBC indices of healthy dogs and to elucidate the importance of these biomarkers in assessing the degree of systemic inflammatory response, which is being closely associated with the development of both conditions. According to the author’s knowledge, there are no literature data estimating CBC indices (N/L, P/L, MPV/PLT, PLCRi) in a correlation with head and neck inflammatory and neoplastic conditions (benign and malignant) in dogs.

Materials and methods

The medical records of 236 dogs who had undergone medical examination and received treatment at Animal Hospital Postojna, Slovenia were retrospectively reviewed. There were 71 clinically healthy dogs, 73 dogs with stage 3,4 periodontal disease (moderate to advanced periodontitis) and 92 dogs with oropharyngeal tumours (31 dogs with carcinomas, 19 dogs with melanomas, 24 dogs with sarcomas, 18 dogs with epulides). The complete blood counts (CBC), obtained at the time of the presentation before performing any diagnostic steps or treatments, were included and retrospectively reviewed. N/L and P/L ratios were calculated as ratio of the neutrophils and platelets...
to lymphocytes. MPV to PLT ratio was also calculated. The adjusted P/L ratio for large platelets (PLCRi) was calculated after multiplying the conventional P/L ratio with the platelet-large cell ratio (PLCR), as calculated by the automatic analyser in the general blood test result. Statistically significant differences between the groups investigated (healthy control dogs vs dogs with stage 3,4 periodontal disease (moderate to advanced periodontitis) vs dogs with oropharyngeal tumours) were preliminary explored by Kruskal-Wallis test. Multivariable analysis were performed according to the polytomous logistic regression model and linear regression model in the case of predicting overall survival investigated in a group of dogs with malignant oropharyngeal tumours. P values <0.05 were considered statistically significant.

Results

Significant differences in N/L, P/L, MPV/PLT, PLCRi indices between groups of dogs investigated were determined (p<0.05). Healthy dogs exhibited the lowest values of CBC indices while dogs with oropharyngeal tumours were demonstrated with the highest values of parameters investigated. The values of N/L, P/L, MPV/PLT ratios and PLCRi of dogs with stage 3,4 periodontal disease (moderate to advanced periodontitis) were always between the values of healthy controls and the values of CBC indices of dogs with oropharyngeal tumours. The differences in CBC indices of dogs with different types of oropharyngeal (malignant vs benign tumours) were determined as well (p<0.05).

Conclusion

We have demonstrated the significant differences in the systemic inflammatory immune responses provoked by stage 3,4 periodontal disease and benign and malignant oropharyngeal tumours in dogs assessed by CBC indices (N/L, P/L, MPV/PLT, PLCRi). The results of our study confirmed that the systemic inflammatory response driven by host inflammatory cells is present in both groups of dogs but is more pronounced in dogs with malignant oropharyngeal tumours than in dogs with stage 3,4 periodontal disease (moderate to advanced periodontitis). Further prospective studies investigating CBC indices in different types of both early-stage and advanced stage malignant oropharyngeal tumours also in comparison with other clinicopathological, histopathological and immunohistochemical biomarkers are in progress. In these cases, not only pre-treatment but also post-treatment assessments of CBC indices may serve as cost-effective therapeutic decision-making and prognostic biomarkers.

References


Sialography versus ultrasonography in imaging the mandibular salivary gland in dogs

Faisal A. Torad

Sialography is an invasive technique that allows imaging of the salivary gland and its associated duct while ultrasonography is a non-invasive technique allows imaging of the salivary gland. Differential diagnoses of salivary gland pathological conditions include foreign bodies, salivary calculi (sialoliths) causing obstruction, inflammation and neoplasm. The purpose of the present study is to compare between the efficacy of sialography and ultrasonography in visualizing the mandibular salivary gland and its associated duct. The study was performed on 3 adult mixed breed dogs. For sialography of the mandibular gland, dogs were anesthetized and injected with 3-5 ml Iohexil 300 mg/ml (Omnipaque)® through catheterization of the mandibular duct at the level of sublingual caruncle (Fig. 1). Radiography was performed immediately after injection and revealed that, the mandibular gland could be identified caudal to the mandibular angle and has multilobulated appearance (3.5×2.5 cm). The mandibular duct leaves the gland on the medial surface, passes over the cranial part of digastricus and styloglossus muscles, opens into the mouth on the sublingual caruncle near the frenulum linguae (Fig. 2). Ultrasonographic examination of normal mandibular salivary glands revealed a superficial ovoid structure of medium echogenicity positioned between the skin and common carotid artery. Normal glands had well-defined, smooth, echogenic walls and contained a series of hyperechoic linear streaks representing the fibrous duct system. The mandibular duct could not be identified. In conclusion, sialography appeared superior to ultrasonography in visualizing the mandibular duct. So, it could be used for diagnosing, foreign bodies, duct rupture, salivary calculi and obstructive inflammation.
Endodontia is the teaching of morphology, physiology, pathology and therapy of dental pulp and the periapical region. The goal of an endodontic treatment is conservation of a vital pulp. If this is not achievable pulp removal is performed to reserve a non-vital tooth in the dental arcade.

The pulp is located in the pulp cavity and the pulp horns of a tooth. The function is producing secondary and tertiary dentin, sensory innervation and infection defense. Because of producing secondary dentin the pulp chamber the diameter of pulp horns and root canals become smaller with advancing age.

Etiology of pulpitis

If a thermal, infectious, osmotic, mechanical or chemical-toxic noxa exceeds a certain level and a certain time, the pulp reacts with pain, acute or chronic inflammation.

A common cause of pulpitis in incisors is exposed pulp related to traumatic fractures and fissures. Incisor reduction also has a certain risk of pulp exposure or thermal pulp damage.

In cheek teeth different pathways are discussed. A Study from Ian Dacre, S. Kempson, P.M. Dixon (The Veterinary Journal 178 (2008) 352–36) documented etiopathological findings in 57 apically infected maxillary cheek teeth. The etiology of apical infection in this study was anachoresis in 51%, crown fractures in 9%, infundibular caries in 16%, occlusal pulp exposure in 9% and periodontal disease in 17% of the documented cases. A study of White and Dixon (2008) documented a variation of distance between the occlusal surface and pulp in a 16 year old horse from 2 to 33mm! This distance is not predictable and explains the risk of pulp exposure / thermal pulp damage from dental equilibration or odontoplasty. Careful occlusal adjustment is recommended and water cooling should be considered. Even if the pulp is not exposed, a thin dentin layer allows pulp irritation because dentin tubules can be penetrated from bacteria and noxa. A high risk to expose pulp is associated with using molar cutters. All instruments and gags creating a focused force to a single tooth can lead to tooth fracture with pulp exposure.
A consequence of pulpitis can be either a healing process with bacterial elimination and obturation of a pulp exposure with tertiary dentin or pulp necrosis with increasing bacterial growth and continuous tooth decay.

**Endodontic examination**

Other than acute pulp exposure, pulpitis in an early stage is hard to detect. In the course of disease symptoms like gingiva retraction, open pulp horns, oral-, cutane-, sinuidal-, conchal fistula and inflammation are often present.

Because a necrotic pulp cannot produce secondary dentin, dental abrasion will lead to open pulp horns at the occlusal surface.

**Radiographic examination**

Radiographs of the apical region often show periapical lucency with surrounding sclerotic bone in case of pulpitis. Insertion of contrast medium allows information about a hollow and connection to fistulae.

In a CT scan pulpitis is often seen with gas accumulation in the pulp chamber.

**Endodontic therapies**

Depending on the tooth condition one has to evaluate if endodontic treatment is reasonable or extraction is indicated. If the dentin is infected for a longer time the tooth becomes weak and extraction is indicated. Also if the periodontal situation is not good or the crown is fractured below the gingival margin, endodontic therapy is not reasonable.

Aseptic operation and sterile instruments are the requirements for endodontic therapies. Following endodontic therapies are available for different conditions:

1. Pulp capping, vital pulp amputation
2. Total pulp removal (root canal therapy), orthograde endodontic therapy
3. Root resection with retrograde endodontic therapy
4. Replantation after extraction and retrograde endodontic therapy

1. *Pulp capping, vital pulp amputation*: Acute pulp exposure from a dental injury has the risk of an infectious pulpitis because of a high number of bacteria in the oral cavity. The goal is to protect the vital pulp as soon as possible to reduce the risk of infection. Treatment is recommended 24-48h after pulp exposure.

To reduce the number of bacteria in the oral cavity, the mouth is washed with a chlorhexidine solution 0.05%. The exposed pulp can be cleaned with hydrogen peroxide solution 3%. Usually it is necessary to amputate about 5-10mm of pulp to create a cavity that allows a good fitting for pulp capping. This removal of contaminated pulp increases the prognosis of saving a vital pulp. Hemostasis of the pulp surface can be achieved with hydrogen peroxide (3% to 10%), calcium hydroxide powder or using a diode laser. If bleeding doesn’t stop, this can be a sign for pulpitis and complete pulp removal has to be considered. In the ambulatory practice the easiest way is to condense some calcium hydroxide powder with a plugger or endo condenser to the pulp surface. To protect the calcium hydroxide, one layer self-adhesive resin cement (Embrace™) is applied and light
cured. If the cavity is big enough a second layer cement or composite can be applied. Calcium hydroxide is a disinfectant; it has hemostatic properties and induces dentin production of the pulp to seal the pulp. Similar activities are known from MTA (mineral trioxide aggregate). It can be used alternatively to calcium hydroxide.
Endodontic procedures 2 (Total pulp removal)

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Total pulp removal (root canal therapy), orthograde endodontic therapy

In cases of pulpitis or necrotic pulp, total pulp removal is one option to save a non vital tooth.

It is necessary to get a good acces into the pulp system for ortograde instrumentation. The crown of the diseased incisor or cheek tooth becomes reduced 2-5mm if necessary.

**Pulp accesses:** In incisors, one or two holes or in case of a cheek tooth may be more positions of pulphorns are drilled with a round shaped burr in apical direction to open up the pulp horns.

After the round shaped burr has reached the pulp horn, a Lindemann burr is used to enlarge the access hole to the pulp horn.

**Pulpotomy:** Infected pulp, necrotic pulp or other organic material are removed from the pulp horns and pulp chamber with different sizes of hedstom files or similar instruments. Adequate lengths of instruments are required and working length has to be controlled radiographically. Over preparation through the apical foramen induces bleeding and periapical irritation. The working length of the instruments can be marked with rubber stops. In some cases it is only possible to prepare a pulp canal to a certain depth because it is obturated with a dentin bridge. It is possible that apically to the dentin bridge the pulp is still vital.

To soften the organic material the cavity is frequently flushed with sodium hypochlorite solution 3-6%. Infected dentin can be softened with EDTA solution to make it easier to remove. One has to make sure not to use pressure during flushing the pulp cavity to prevent perfusion of the periapical region with hypochlorite solution! A very good technique for flushing is the Endo - Vac™ system because the hypochlorite is soaked into the cavity without pressure. In between the pulp cavity is flushed with high volume of sterile saline solution. This can be performed with an arthroscopy pump and an adequate cannula. During flushing with sodium hypochlorite, suction has to be used at the tooth surface to aspirate the returning fluid to prevent contamination of the mucous membranes. Sodium hypochlorite is highly irritant to the mucous membranes. After this cleaning process the pulp cavity has to be dried with suction through a cannula and paper points. Final disinfection and drying with a diode laser seems to be very useful.
**Temporary filling/medical treatment:** It is simply impossible to clean a highly infected pulp canal in one session thoroughly. A medical treatment of the pulp canal for 3-4 weeks can help to soak the dentin walls with antimicrobial agents for further reduction of bacteria remained in the tooth.

In human endodontia for medical treatment of root canals a mixture of ciprofloxacin, metronidazole and tetracycline is recommended. For medical treatment of pulp cavities in horses usually a calcium hydroxide paste is used. The medication is injected into the pulp canal and transported apically with lentulo filler. If a lentulo is not available in the required length, a Hedstrom or K-file can be used counter clockwise with the same function. Elastic cement is used to cover the medication and to seal it against the oral cavity (Provicol™, VOCO). Because the elastic cement is not bonded to the tooth, chewing forces compress the filling and medication apically during the treatment period.

**Permanent filling:** After 3-4 weeks the medical treatment is removed and the pulp canal is cleaned and filed again like described. If the situation is still not reasonable for permanent filling a second period of medical treatment is indicated. If the cavity is sufficient clean a permanent filling can be applied. In humans and small animals root canal filling is usually done with an endodontic cement and gutta-percha points. Especially in equine cheek teeth this is not useful. An adapted procedure for equine hypsodont teeth is a filling of the apical part of the root canal and pulp canal with calcium hydroxide paste or MTA by injection and using lentulo filler. This technique allows a distribution of calcium hydroxide or MTA in the pulp chamber and the preparations have a chance to reach pulp communications. After condensation with lentulo filler and with cotton swabs, this filling is covered with a layer of elastic cement (Provicol™, VOCO). After 5 minutes of setting time the elastic cement is condensed with a ball shaped plugger or similar instrument apically. Before the next layer of filling is applied, the dentin walls are cleaned with an excavator or burr to achieve a good bonding of the filling to a clean dentin wall. The next layer can be done with self-bonding resin cement (Embrace™, Pulpdent). Depending on the cavity size, the cement can be placed up to the occlusal surface or a composite material can be used to do the occlusal filling after etching and bonding (Futura Bond™, VOCO). Because deep cavities are not easy to access with a curing light, the materials should have either self-curing or dual curing properties. This means they have the ability to cure without a curing light.
Root resection with retrograde endodontic therapy

To achieve a good access to the periapical region and to remove the apex of an infected tooth, root resection with retrograde endodontic therapy can be performed.

A sinus flap or bone flap has to be done to get access to the roots of cheek teeth. After retrograde cleaning and filling the bone flap and wound has to be closed in a routine matter. Because of the bloody environment it is not easy to perform a clean and dry endodontic therapy and filling.

Replantation after extraction and retrograde endodontic therapy

This technique is indicated if the affected tooth is very long and hard to treat with orthograde endodontic therapy.

References

Tumours of the equine head – incidence, diagnostics and treatment

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\textbf{Introduction}

Neoplasms of the head and nasal discharge are a relatively frequent symptom in equine medicine. Very rarely, these symptoms are caused by tumours with autonomous and unpredictable growth behaviour.

The published literature provides only single case studies and case series describing the clinical course as well as diagnostics, treatment and prognosis in equine tumourous diseases. The average incidence of tumours in equines is given as 1-3\%. The head is described as one of the most frequent localisations for tumours in equines.

\textbf{Which tumours can be found on the equine head?}

Tumours are classified as benign, semi-malignant or malignant according to their behaviour. They can be derived from bone (sarcoma, osteoma, ossifying fibroma, myxoma), teeth (cementoma, (complex) odontoma, ameloblastoma) or soft tissues (equine sarcoïds, squamous-cell carcinoma, papilloma, melanoma, fibroma). This list is by no means exhaustive but includes the most common neoplasms of the equine head.

\textbf{Symptoms}

The symptoms are usually unspecific and allow for a range of possible diseases.

\textbf{Diagnostics}

\textbf{Medical history}

The medical history can give the examiner valuable pointers indicating a tumourous disease and the resulting options for treatment:

\begin{itemize}
  \item How long have the symptoms been present, and what has been the clinical course so far?
  \item Did the disease begin slowly and progressively or rather fast and suddenly?
  \item These questions are important because they can help to give an estimate regarding the progression of disease and the urgency of further work-up.
\end{itemize}
The question of a potentially reduced general condition and impaired feeding behaviour is also important to weigh up treatment options.

What are the symptoms? This is important because it helps to draw a first conclusion regarding the affected structures (e.g. unilateral nasal discharge in cases of sinus involvement).

How old is the horse? Some tumours are more common in (very) young horses (e.g. ossifying fibroma) whereas others (such as squamous-cell carcinoma) tend to be found in older patients.

**Clinical examination**

The clinical examination should consider tumours as a differential diagnosis especially in cases of swellings of the lower jaw (Fig. 1) and the facial skeleton as well as slow-healing wounds of gingiva or oral mucosa. Furthermore, attention should be paid to ulcers or necrosis of gingiva or oral mucosa in unusual locations or of unusual sizes.

Any deviation from the norm should be documented, and photographed if possible. Further diagnostic measures (esp. radiography, biopsy, computed tomography) should be discussed with the owner.

![Figure 1. Equine juvenile ossifying fibroma in a 6-month-old Zebra.](image)

**X-Ray**

The X-ray image is part of the basic examinations and helps to rule out many diseases. However, the changes detected in X-ray images are hardly ever typical enough to arrive at a definite diagnosis. The sheer number of osseous tumours and the great variety of their radiographic appearance allows for many other diseases (Fig. 2 and 3).

**Computed tomography**

Computed tomography without any overlappings is considered to be the gold standard
Figures 2 (left) and 3 (right). Complex odontoma in an 8-month-old Hannoverian filly.

Figures 4. Computed tomographic image (sagittal and 3D reconstruction) of a complex odontoma in a 4-year-old warmblood.

in displaying neoplasms on the equine head. Size and extent, demarcation from and involvement of surrounding tissues are easy to assess (Fig. 4). Owners of affected horses can be given a better prognostic evaluation of the case, and a potential surgical treatment can be planned more accurately.

**Magnetic resonance imaging**

Magnetic resonance imaging does not yet feature significantly in tumour diagnostics on the horse’s head. In part, this is due to the fact that MRI scanners are not as widespread as CT scanners. Also, many neoplasms of the equine head involve hard substances such as bone and teeth which are easier to interpret in CT scans.
**Histopathological examination**

Biopsy and histopathological examination of tissue changes play a vital role in avoiding misdiagnosis. Care must be taken to send representative samples for examination, and not to just scrape off superficial tissues that might only be subject to secondary changes due to inflammation. This might lead to false-negative results. These can be avoided by collecting a minimum of three samples, each 6-10 mm in diameter, from different parts of the tumour, fixing them in 4-10% formalin and sending them on their way to the pathologists by normal post, together with a detailed report of prior work-up.

**Treatment and prognosis**

Benign tumours can be surgically removed (e.g. osteoma), or whole parts of the jaw including the tumour can be cut off (e.g. rostral mandibulectomy in cases of ossifying fibroma). Prognosis for patients is good if the neoplasm can be removed in its entirety.

In cases where a malignant tumour has been diagnosed, there are hardly any sensible treatment options at the moment. Therefore, if the animal’s general condition is reduced, euthanasia is indicated in most cases since prognosis is poor to hopeless. One aggravating circumstance lies in the fact that many neoplasms are only detected in a late, far-progressed state. In these cases, the complete surgical removal is difficult to impossible due to the large dimensions of the neoplasm.

In spite of extensive diagnostics using clinical signs, imaging techniques and a final histological examination, it can be still difficult to arrive at a definite diagnosis. This may be due to the fact that the tissues sent for examination were not representative, or that malignant neoplasms can develop from primarily inflammatory processes. This has been described for the squamous-cell carcinoma. Transitional forms often overlap. Therefore, it is not always possible to clearly distinguish between “dangerous” and “safe”.

**Conclusion**

Tumours of the head still rank among the rarer equine diseases. Nevertheless, a neoplasm should be considered when diagnosing patients with nasal discharge, non-healing alveoli after tooth extraction, aggressively proliferating foul-smelling soft tissues in the oral cavity or sinuses and fast growing swellings of the face.

A premature diagnosis after clinical examination and medical imaging should be avoided since in most cases only histopathological examination together with all information from the medical history and the findings made with imaging techniques permit a definite diagnosis.

**References**

Available from the authors on request.
A reflection on “Dental materials in equine dentistry”

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The progress in equine dentistry along with advances in sedative and analgesic techniques during recent decades has made it possible to perform more advanced dental treatments. The consequence is the need to utilize dental materials, such as restoratives, root canal sealers and so forth. The special development of dental materials for equine purposes does not exist, at least as far as the author knows. Subsequently we have to apply dental materials available on the market. The products are developed to meet the demands in the human oral cavity, which differ in quite a number of aspects to the equid oral cavity. For example, the anatomy of the hypsodont tooth is very different to the brachyodont tooth, found in humans which all are covered with enamel when healthy. The hypsodont tooth is designed to withstand considerable attrition, which it accounts for by an almost continuous period of eruption. Another factor, which may seem too obvious to mention, is the size of the equine hypsodont tooth. Importantly, the biochemistry and biology of the saliva of horses differs also in several aspects; for instance a higher pH level, higher content of bicarbonate and larger volume.

Is there a problem with the differences? Well, all the properties of dental materials on the market are tested to cope with the human oral condition which counts for everything from size of syringes and needles for application, to the size of a “normal” filling. Consider the difficulties associated with the shrinkage component during curing for a posterior composite material. The effect on the volume of a “normal” human cheek tooth filling, about 0.13-0.15 cm$^3$, is almost negligible but when looking at the effect on an infundibular restoration of an equine cheek tooth, a volume of maybe 4-5cm$^3$, the shrinkage component will be of importance, just because of the size. This may lead to loss of micromechanical bonding or fracture of tooth substance.

Shortly after coming into occlusion all dental hard tissues of the equine tooth are exposed. Thus the choice of dental material should be related to which part of the occlusal surface is intended to be replaced. If an orthograde endodontic treatment is performed it is the secondary dentine that is intended to be replaced. Hence a composite material with properties similar to dentine should be used. Otherwise, there will be an imbalance according to normal attrition and the risk for fracture of either tooth substance or filling is increased.
Another problem that may affect the choice of dental materials used in equine dentistry, at least in some European countries, is that horses are look upon as potential food for humans. There are no MRL (maximum residue level) values for dental materials nor do we know anything about which chemicals may be absorbed by the body. For instance it is well known that there are filling materials that contains BP-A (bisphenol-A).

Some, and in a sense maybe all, of the dental materials are toxic, especially during curing, and other are allergenic. These facts must be taken into consideration during the everyday work because of the risks that the staff will be exposed to a level that can jeopardize health. The risk is probably much higher for the staff compared to the individual patient.

Thus, using dental materials in equine dentistry must be followed up very carefully both in the short and long term, and from the aspect of animal welfare and common health of the veterinary staff.

Further reading

Endodontic treatment of human teeth has a long history and is today a well-established academic specialty. Endodontic treatment in humans as in horses has the potential to save teeth and thereby maintain oral function. Typical indications in the horse are periapical infections (including those resulting in sinusitis), and traumatic pulp exposure. An important advantage of doing endodontic therapy in hypsodont teeth is to minimize the risk for dental drift, a common, and in most cases unavoidable post extraction sequelae.

The differences between brachyodont teeth and hypsodont teeth are important. However, the endodontic cases published have been performed using a retrograde technique. At the Animal Dental Clinic in Sweden and the University of Agriculture Sweden an orthograde technique has been developed with good long-term results.

The orthograde approach has some important advantages compared to the retrograde technique. Root canal preparation, obturation and sealing demands a very clean and proper working area and this is more achievable at the occlusal aspect. An orthograde approach takes advantage of the occlusal forces present each time the animal chews. The occlusal forces compress and force the sealant in an apical direction optimizing their function. One of the advantages of the apical movement of sealant/intra canal medicant is that it provides room at the occlusal aspect for additional sealant to be placed over time. Importantly, an orthograde approach easily allows for the endodontic treatment to be performed over multiple visits. Multiple visits are essential to ensure appropriate sterilization of the root canal, monitoring of the biological response to the endodontic treatment, and breaking the procedure into working times well tolerated by most animals under sedation.

There is one crucial question to be answered before making the decision to perform endodontic treatment. The question is; “Is it possible to access the affected parts of the tooth?” If not, do not try endodontic treatment.

The orthograde technique is similar to the technique used in brachyodont teeth. In the first step access to the pulp cavity is created using a low speed round shaped low speed carbide bur. Before going further into the pulp horn the occlusal aspect is widened with a Lindeman bur (low-speed). Next step is to clean out the pulp cavity thoroughly using endodontic files (Hedström) of different length and diameter depending on the anatomy of the tooth. Indicator radiographs are taken during the procedure for guidance. The pulp cavity is
irrigated with high volume of saline solution using an electric pump to facilitate removal of bacteria, debris and tooth material. Once the pulp cavity is clean and appropriately shaped it is dried using a suction system and small cotton pellets. Before the pulp cavity is sealed off, it is obturated with an emulsion of Ca(OH)$_2$. The Ca(OH)$_2$ must be thoroughly condensed so the water part of the emulsion is minimized. Finally the access sites are sealed with dental cement in at least two different layers that are not chemically attached to each other. This is for safety reasons if the outer layer is lost it will not interfere with the second layer.

On the initial visit “temporary” fillings are used to allow easy access to the pulp cavity at the second visit. The second visit is recommended after one month. If the pulp cavity is clean and dry on investigation at the one month follow-up a more resistant seal is applied in at least two different layers. If there is liquid in the pulp cavity or other signs of failure the choice of therapy needs to be re considered. The whole procedure may need to be repeated. In cases where the initial treatment appears to be successful, follow up is recommended at least once a year to make sure the restorative is complete. Follow-up radiographs are recommended no earlier than in one 1 year’s time. At the yearly examination the normal tooth eruption should be noted provided that the endodontic therapy is still successful.

The described orthograde technique has been used in Sweden for more than 10 years. The long-term follow up shows a high frequency of success. Treatment failures, when registered, are most often close in time to when the primary treatment was performed. The most common causes of failure are new dental fractures, and persistence of the endodontic infection resulting in fistula formation.

It is proposed that the cause of subsequent dental fracture of treated teeth is largely due to a combination of analgesia administered as part of the initial endodontic treatment and undetected microscopic fractures/infractions. The analgesia results in normal occlusal loading by the horse when chewing, which then causes non displaced crown fracures to progress and ultimately become displaced. Most new fractures are seen within the first three month after primary treatment. Fistula formation is explained by failure to eradicate the endodontic infection as part of the initial endodontic therapy. When fistula is develops subsequent to initial endodontic therapy, repeat endodontic treatment often includes both orthograde and retrograde approaches in the same session.

In our opinion endodontic treatment is a good alternative in many cases to the more common treatment of extraction. There are many advantages to being able to save a tooth. One advantage of endodontic therapy is that initial failure still allows retreatment and the possibility of successful outcome. Tooth extraction is final and cannot be reversed. Endodontic therapy performed at a level which results in successful outcomes is technically challenging. Endodontic therapy and case management should only be performed by appropriately trained and equipped professionals.

Further reading


van Foreest AW, Wiemer P. Apex resection in the horse Tijdschr Diergeneesk. 1997 Dec 1;122(23):670-9
Precision of maxillary nerve anesthesia in the horse for surgical or diagnostic purposes is moderate and a variety of complications can be encountered. Our aim was to establish an ultrasound-guided technique with the needle inserted perpendicular to the skin (UGPI) and compare its accuracy and the incidence of adverse effects to the same insertion technique without ultrasound guidance (PI).

A 3.5inch needle was inserted perpendicular to the skin just ventral to the zygomatic arch, leveled with the lateral canthus of the eye and advanced to the sphenopalatine bone (PI) either with or without the presence of a microconvex 5-8Mhz ultrasound probe, placed ventral to the insertion site, perpendicular to the skin with its beam orthogonal to the forehead (UGPI). Twelve live horses were randomly assigned to injection of 0.15ml methylene blue dye (MB) by either technique. Interventional complications were observed and recorded. One hour after injection the horses were euthanized and dissection of the pterygopalatine fossa performed.

Mean times to injection were 67.25sec (± 13.9sec) for PI and 298.5sec (± 117.3sec) for UGPI. All (n=12; 100%) UGPI-injections resulted in successful deposition of MB-dye in contact with the nerve, but only 7 out of 12 PI-injections (58.3%). Complications were 4.75 times less common in the UGPI-group than in the PI-group. Pathological changes were twice as common in the PI-group than in the UGPI-group.

Our study showed that a maxillary nerve block with the needle inserted perpendicular to the skin can be achieved more accurately and with less complications using ultrasound-guidance.
Effect of butorphanol, midazolam or ketamine on romifidine based sedation in horses during standing cheek tooth removal

T.M. Müller, A. Bienert-Zeit, K. Hopster, K. Rohn & S.B.R. Kästner

Reasons for performing the study: The study was performed to improve sedation protocols in horses for cheek tooth removal.

Materials and methods: Forty horses presented for cheek tooth extraction were divided into four groups (n=10).

- **Group R:** Romifidine (0.03 mg/kg i.v.) followed by constant rate infusion (CRI) of romifidine (0.05 mg/kg/h).
- **Group RB:** Romifidine (0.03 mg/kg i.v.) and butorphanol (0.02 mg/kg i.v.) followed by CRI of romifidine (0.05 mg/kg bw/h) and butorphanol (0.04 mg/kg/h).
- **Group RM:** Romifidine (0.03 mg/kg i.v.) and midazolam (0.02 mg/kg i.v.) followed by a CRI of romifidine (0.05 mg/kg/h) and midazolam (0.06 mg/kg/h).
- **Group RK:** Romifidine (0.03 mg/kg i.v.) and ketamine (0.5 mg/kg i.v.) followed by a CRI of romifidine (0.05 mg/kg/h) and ketamine (1.2 mg/kg/h).

Ataxia, chewing, tongue activity and head movement were graded using a scoring system (score 1-5). If sedation was not deep enough, an additional romifidine bolus (0.01 mg/kg i.v.) was administered. At the end of procedure quality of sedation and extraction were evaluated by the surgeon (visual analogue scale 1-10).

Degree of ataxia and head parameters were analyzed with permutation test and Kruskal-Wallis test, respectively. Quality of sedation and extraction were determined by ANOVA (p<0.05).

Results: Horses in group RM showed significantly less chewing and tongue movement compared to groups R and RB, but had significantly higher levels of ataxia compared to groups RK and RB. Sedation quality was significantly better in group RK compared to group R. Furthermore, horses of group RK needed less additional romifidine bolus compared to all other groups.

Conclusion: The combination of romifidine and midazolam for standing sedation facilitates oral manipulations in the horse even though higher levels of ataxia are to be expected. Improved sedation quality and thereby better surgical conditions for successful cheek tooth extraction was obtained by the combination of romifidine and ketamine.
Periodontal disease with valve diastemata in horses has been described as a very painful condition already more than 150 years ago. Different approaches to the treatment have since been described.

Diastema widening is one of the published options for treatment. It is explained as a procedure where the diastema will be widened up to 4.5 -6 mm down to the gingival margin.

The author suggests a less invasive method. Additionally to the necessary cleaning of the diastema from food a superficial grooving with only a 3 mm burr is recommended. The width of the groove will be 3mm and the depth ranges from 3-5mm. This does not destabilize the teeth but seemingly reduces the reentering of food into the IDS. By this means the inflammation can heal and the generated loss of periodontal tissue can be replaced by the body itself. Quite a few times a restitutio ad integrum has been seen. This happened in cases that were heavily inflamed and a lot of bleeding was seen during cleaning.

Over a period of 2,5 years more than 150 clinical cases have been treated this way. At the current point the results are very promising.

In that lecture the method, typical clinical cases and the preliminary results will be described. In depth statistical evaluation has not been done yet.
Management of equine head shaking by widening of diastemata for the treatment of associated periodontal disease in two horses

Tim Barnett

Not available.
Objectives Equine periodontitis is a common and painful condition. Despite this, the disease often goes unnoticed by owners and is thus a major welfare concern. The aetiopathogenesis of the condition remains relatively poorly understood with few recent studies performed. The innate immune system is known to play an important role in human periodontitis where chronic production of inflammatory cytokines in response to oral bacteria results in periodontal tissue destruction. The aim of this investigation was to characterise the innate immune response to oral bacteria in equine periodontitis and oral health.

Methods: Post-mortem gingival tissue samples were taken from 12 orally healthy horses and 20 horses with periodontitis. mRNA expression of TLRs 2, 4 and 9 and cytokines IL-1β, TNFα, IL-4, IL-6, IL-10, IL-12, IL-17 and IFN-γ was determined using qPCR. Statistical significance of results was assessed using f-tests and paired t-tests. Equine specific cell culture assays were created to assess the innate immune response of equine gingival cells to oral bacteria and putative periodontal pathogens.

Results: A marked inflammatory response was noted in diseased gingival tissue with significant increases in expression of TLR 2, TLR 4, IL-4, IL-10, IL-12 (p≤0.05) and IFN-γ (p≤0.01) in periodontitis. Cell assay work is currently ongoing.

Conclusion: Increased expression of TLR 2, TLR 4 and TLR 9 indicated an increased need for recognition and response to bacteria in periodontitis. A mixed Th1/Th2 inflammatory response was noted and increased expression of IFN-γ may contribute to tissue destruction in equine periodontitis.
Infundibula of equine cheek teeth: odontogenic development and malformations

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Background

The infundibula of equine maxillary cheek teeth usually contain a complete filling of infundibular cementum. However, incomplete cemental fillings are frequently reported and this condition is recognized as an etiological factor in the development of both endodontic diseases (Dacre et al., 2008, Van den Enden et al., 2008, Veraa et al., 2009) and tooth fractures (Baker, 1979, Dacre et al., 2007, Dixon et al., 2014).

The aim of the present study was to document the infundibular development and structure with special regards to infundibular blood vessels and infundibular cementogenesis.

Materials and methods

Forty-one deciduous maxillary premolars were investigated using routine histological and immunohistological methods to visualize blood vessels and the enamel organ. A subset of the specimens was scanned by micro-computed tomography to analyze and visualize the three-dimensional anatomy of the developing infundibulum.

Results

During odontogenic development, the infundibulum is supplied by a central infundibular artery (entering the infundibulum from an occlusal direction) and by a lateral artery (entering the mesial infundibulum from the mesial aspect, and the distal infundibulum from the distal aspect). The lateral artery of the distal infundibulum is located in a more apical position than its counterpart in the mesial infundibulum. No blood vessels entering the infundibulum from an apical direction by penetrating the dentin and the enamel organ at the bottom of the infundibulum were found. Cementogenesis starts first at pronounced enamel in-folding in the occlusal part of the infundibulum, advancing in an apical direction. Infrequently, hypoplastic / aplastic enamel areas were observed. In these areas infundibular cementum was directly attached to periinfundibular dentin.
Discussion

The central infundibular artery becomes destroyed shortly after tooth eruption but the lateral arteries remain vital for an undetermined time after eruption. As the lateral artery of the distal infundibulum is located in a more apical position, blood supply is preserved for a longer time compared with the mesial infundibulum (Suske et al. 2015). These results suggest a distinct asymmetry between the infundibula with the mesial infundibulum prone to incomplete cementogenesis due to early blood supply cessation. This conclusion is supported by clinical observations which documented a higher prevalence of infundibular cemental hypoplasia in the mesial infundibulum (Veraa et al., 2009; Windley et al., 2009). Focal areas of enamel hypoplasia / aplasia might play a significant role for the transmigration of microorganisms form the infundibulum into the perinfundibular dentin and further into the dental pulp (Dacre, 2005).

Take home message

Infundibular cementogenesis is based on a sufficient blood supply which is provided by a central and a lateral artery. After an undetermined time post eruption blood supply ceases, potentially prior to the complete cemental filling of the infundibulum. This condition, termed infundibular cemental hypoplasia, should be regarded as a malformation rather than a disease.

References


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Basics of equine cheek teeth dental extractions

Extraction of long crowned equine cheek teeth is a major surgical procedure with great mechanical force required to break down the periodontal ligament. With the advancement of constant rate infusion sedation and local anaesthesia techniques of maxillary and mandibular nerves in horses, oral extraction in the standing horse is the preferred technique for the extraction of cheek teeth. Oral extraction has been shown to have less postoperative complications than other surgical extraction techniques such as repulsion or lateral buccotomy (Dixon et al. 2005). However, in cases where there is no clinical crown to grasp with molar extractor forceps (e.g. fractured or brittle crown, unerupted tooth), other methods of oral or surgical extraction are required (repulsion, lateral buccotomy, segmentation, minimally invasive trans-buccal technique). It is important to first understand and get experience in the basic oral extraction technique, before more advanced techniques are attempted.

The basic principles of dental extractions are to obtain adequate access to the periodontium, to create an unimpeded pathway for the removal of the tooth and to use controlled force to remove the tooth (Shira 1968). Extraction of equine cheek teeth is technically difficult and requires careful preparation and use of specialized equipment. The minimum equipment required includes a full mouth speculum, oral mirror, excellent light source, and an array of periodontal elevators, molar spreaders and molar forceps. Potential complications such as laceration of the palatine artery, fracturing of alveolar bone and fracturing of tooth crown or tooth roots, need to be considered, so that appropriate action can be taken to manage them.

Prior to oral dental extraction, the horse should be sedated, local anaesthesia of the appropriate nerve should be given, and systemic analgesia and antibiosis should be started. If extracting maxillary cheek teeth, it must be remembered that there may be some sensory innervation on the buccal aspect of the gingiva that branches from the facial nerve and local infiltration of anaesthetic on the buccal mucosa and gingiva may be required in addition to the maxillary nerve block. The mouth should be flushed out and there should good visualization of the affected tooth. The gingiva on the buccal and lingual/ palatal aspect of the tooth should be elevated with a right-angled pick or periodontal elevator. The molar spreaders or separators are applied firstly to the rostral
and then the caudal interproximal interdental space and left there for 3-5 minutes. Care should be taken when applying the molar spreaders in the rostral interdental space for extraction of the third premolars (07s) as it may loosen the second premolar (06). Thus it is recommended to apply the molar spreaders in the caudal interdental space first when extracting the 07. This will allow for some haemorrhage into the periodontal ligament and aid loosening of the periodontium at the mesial and distal aspect of the tooth.

An appropriately sized molar extractor forceps should be applied and the placement of the extractors should be carefully checked to ensure that it is placed only on the affected tooth crown. The forceps handles should then be fixed in place with a locking mechanism or rubber band, and then slowly moved in a horizontal plane from side to side along the longitudinal axis of the tooth. The forceps placement and tooth crown should be inspected at regular intervals to ensure that the forceps have not moved or that they are not just wearing the crown away without loosening the tooth. Excessive force too early on could result in fracturing the crown. Once the periodontal ligament starts to loosen, a ‘squelching’ sound will start to be heard and foamy haemorrhage may be seen around the gingival margin. This indicates loosening of the periodontal ligaments. Repetitive movement of the tooth may also stretch the alveolus, also facilitating extraction. Sufficient loosening of the periodontium may take anywhere from 20–60 minutes or even longer.

Once the tooth is sufficiently loosened, a dental fulcrum may be placed on the occlusal surface at the level of the boxed-hinge of the molar forceps and the tooth carefully levered out. Once the tooth is partially extracted, the forceps may need to be re-applied further apically to further lever the tooth out. In younger horses with longer reserve crowns the tooth may have to be twisted axially to facilitate extraction from the oral cavity. After extraction of the tooth, the tooth should be carefully inspected to ensure the whole tooth has been extracted. Similarly the alveolar socket should be inspected and palpated for tooth remnants or bone fractures/fragments. If there are dental or osseus fragments, the socket can be curetted. If there are larger fragments, the use of right-angled elevators may be useful in extracting these remaining fragments. This may be facilitated by endoscopic guidance. The socket should then be irrigated vigorously and post-operative radiographs taken to ensure that there are no remaining dental fragments.

The alveolus may then be packed with a temporary polysiloxane plug, dental wax or antibiotic impregnated swabs. This is especially important in cases of maxillary cheek teeth extractions that have resulted in an oro-sinus communication. The plug should only fill the proximal third of the dental alveolus as complete filling may impede the healing and filling of the socket with granulation tissue. The horse should then be given a course of anti-inflammatories and antibiotics (if clinically indicated). If there is secondary sinusitis, the sinuses may need to be flushed for 3–5 days postoperatively. The dental plug should then be removed in approximately 14 days and the alveolus inspected for healing. In some cases, bone sequestra or small dental fragments may be apparent at this stage and can then be extracted with elevators. In the long term, regular rasping will be required of the opposing cheek tooth, as it will continue to erupt without dental wear from the extracted tooth.
**References**


Extractions of equine incisors, canines and wolf teeth

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Incisors

The more common indications for incisor extractions include: marked developmental displacements; certain displaced supernumerary incisors; retained deciduous incisors; traumatic fractures with death of tooth and EOTRH affected teeth with secondary fractures or periodontal disease. Needless to say, any dental extraction should only be performed after thorough clinical and radiographic oral examination of the patient. For example, a horse with a traumatic loss of part of clinical crown, may also have a further subgingival fracture of that tooth or indeed a subgingival fracture of another tooth or its alveolar bone, that will totally dictate the correct therapy. Ensure that tetanus vaccination is current before all extractions.

All equine incisor extractions should be performed under standing sedation, with a local nerve block required in most instances; i.e. an ipsilateral mental block for lower incisors and infraorbital nerve block for upper incisors. Regional nerve blocks such as maxillary or mandibular nerve blocks are usually not required. Depending on the age of the horse and the degree of periodontal disease present, extensive surgical alveolar exposure is necessary to extract the curved reserve crowns that can be up to 7cm in length. Shorter incisors or those with diseased periodontal ligaments can be extracted by use of small animal dental elevators and forceps using standard extraction techniques.

If a surgical extraction is to be performed; having decided how much of the rostral alveolar wall that needs to be resected, a vertical midline incision is made over the gingiva overlying this alveolus, and this is continued down onto the underlying periosteum until alveolar bone is reached. In younger horses with heathy periodontal attachments, this exposure will need to be approximately two thirds of the length of the reserve crown, i.e. up to 5cm long. The alveolar bone can most precisely be removed using a water-cooled, high speed burr or less accurately, using an osteotome or an appropriate-sized curved gouge along with a small mallet. The alveolus is elevated off the labial aspect (almost 180 degrees) of the tooth beginning at the gingival margin.

The remaining periodontal ligament is then disrupted using a dental elevator or more efficiently in horses using a curved osteotome or gouge and gentle use of a mallet. The osteotome should be gradually tapped into the remaining periodontal ligaments at the
surgically exposed aspect of the tooth, and also into the unexposed more apical aspect of
the periodontal ligament, taking great care at all times not to disrupt the adjacent alveolar
bone that can be very thin in young horses. After the incisor is sufficiently loosened, it is
grasped with suitable sized forceps and gradually rotated until sufficiently further loosened,
at which stage it can additionally be avulsed in a curved rostral direction during continuing
rotation. Visual examination of the extracted apex will usually reveal if it is has been fully
extracted. With very diseased teeth such as EOTRH and in cases with a marked alveolar
infection, postoperative radiographs should be taken to ensure no dental or diseased osseous
fragments remain. Following their removal, the alveolus should be lavaged with sterile saline
and a blood clot allowed to form in the alveolus.

Shorter alveoli that have had a non-surgical extraction can be left to heal by primary
intention and usually heal quickly if no dead calcified material remains. If a surgical
extraction has been performed, attempts should be made to close the surgical incision,
bearing in mind that some incisions will partially or fully dehisce due to the almost constant
irritation of this wound by the ingestion of forage for up to 18 hours day. All protruding areas
of alveolar bone should be removed; optimally using a high speed drill and the alveolectomy
site can now be smoothed with a bone file. If following the above procedure, the overlying
mucosa will still not join together without tension, midline over the alveolus, the underlying
periosteum can be sectioned in a sagittal plane close to the adjacent alveolus, to allow further
expansion of the mucoperiosteal flaps. The author prefers to close the mucoperiosteum
using simple interrupted sutures with Vicryl® due to its excellent knotting characteristics
and because these knots will not cause labial ulcers, unlike Monocril®. If the occlusal aspect
of the empty alveolus cannot be closed, it can be packed with gauze that is removed a couple
of weeks later, if still present.

**Canine teeth**

Canine teeth are not commonly extracted in horses – the main indications being
EOTRH, traumatic fracture, partial avulsion and marked developmental displacements. It
must be borne in mind that most canine teeth fractures in older horses are due to underlying
EOTRH lesions - further emphasising that radiographic examination is essential in all
potential canine teeth extractions. The techniques for canine extraction are very similar to
those described for surgical incisor extraction, but because the canine teeth have very limited
eruption, they all have long reserve crowns and so surgical extraction is invariably required.
The labial aspect of the affected reserve crown (thicker bone than that overlying incisors)
is exposed for about 2/3rds of its length and the reserve crown is then loosened using an
appropriately-sized osteotome or gouge and mallet as described for incisors. Because of the
absence of adjacent alveoli, it is always possible and desirable to perform complete primary
closure of the alveolus after having ascertained that all dental tissue and diseased alveoli
have been removed. Protruding alveolar bone is removed and the gingival or mucoperiosteal
flap is fully repaired as earlier described.

**Wolf teeth**

The brachydont 1st premolar teeth are usually extracted because of perceived or real
bitting problems, rather than due to any obvious dental pathology. Wolf teeth are usually
found in the maxilla, very seldom in the mandible. Most wolf teeth are spontaneously shed
at about 30 months when the deciduous 06s are shed. There remains little scientific evidence for their routine extraction – but due to tradition and client pressure, many or most wolf teeth are extracted. Wolf teeth that are very large and/or rostrally or buccally displaced are more likely to interfere with the bit and thus cause problem when the horse is ridden than normal sized and positioned wolf teeth. For routine wolf tooth extraction, radiography is not usually indicated. However, all swellings of the intermandibular space (“bars of mouth”) should be radiographed (using a low exposure for these often poorly calcified structures) for the presence of displaced, unerupted wolf teeth. In younger unbitted horses, the wolf teeth usually have very weak periodontal attachments are readily extracted, and as noted, many are made loose in these young horses by the erupting adjacent permanent 06. Some racetrack veterinarians extract wolf teeth in young horses without any sedation or local anaesthesia, by simply placing a bone rongeurs or other suitable forceps on the wolf teeth and quickly pulling down on them. They claim that this procedure that just takes a couple of minutes is less painful to the horse than the injection of local anaesthesia. However, in older horses that have been bitted, wolf teeth can develop very strong periodontal attachments, especially if they are large. Horses should be sedated and depending on the age of horse, and size and position of the wolf teeth, local anaesthesia by adjacent subgingival local anaesthesia infiltration or with firmer and larger teeth, by using a local nerve block (invariably an infraorbital block) should be performed.

The Burgess (apple corer!) instrument is commonly used to extract wolf teeth– but has many limitations. It is designed to cut the surrounding gingival attachments but not to fatigue the periodontal ligaments. If used to lever out wolf teeth – it will fracture longer, well-attached roots (they have no reserve crowns because they are brachydont teeth). If appropriate in size and shape to the wolf tooth being extracted, they can be usefully used to cut a circular attached piece of the adjacent gingiva, but a dental elevator should then be used to complete the extraction. The author prefers to use long handled offset dental elevators to both reflect the adjacent gingiva and to break down the periodontal attachments, using either manual pressure, or a small mallet for better attached or larger wolf teeth.

Disruption of the periodontal ligaments should be performed slowly, because some wolf teeth have long, narrow roots that may break during the extraction. Because these fractured remnants are always healthy, some operators leave them in situ provided they are not loose and do not protrude above the alveolus. The author prefers to extract all, except very fine root tips – and using smaller diameter offset elevators and a small mallet, most retained roots are readily removed. No aftercare, other than possibly post-operative non-steroidal anti-inflammatory drugs are indicated after wolf teeth extraction.

**Recommend reading**


Intra-oral segmentation of cheek teeth in standing horses

Michael Nowak

Not available.
Equine dental radiography and interpretation

Neil Townsend

Knowledge of the normal anatomy of the equine skull and diagnostic images are a prerequisite for radiographic interpretation, which can be assessed with the clinical signs to give a diagnosis. Whilst the most common indication for dental radiography is for the diagnosis of apical infection (facial swelling or unilateral nasal discharge), other indications include severe periodontal disease, polydontia, anodontia, dysplasia and neoplastic or neoplastic-like lesions involving the teeth.

The sensitivity and specificity of radiography for diagnosis of cheek tooth apical infection has been investigated in several studies (Weller et al. 2001; Barakzai 2005; Casey et al. 2009; Townsend et al. 2011). Conventional film radiography has been shown to have sensitivity of 52-69% with specificity of 70-95% (Weller et al. 2001; Barakzai 2005) whereas computed radiography (CR) appears to have an improved sensitivity of 76-80% and similar specificity of 80-90% (Casey et al. 2009; Townsend et al. 2011). This improvement in sensitivity is probably due to the greater dynamic range and improved contrast resolution that can accentuate differences in opacity between tissue types (Armbrust 2000). These values are likely to be greater in a clinical setting where more information such as clinical exam findings and results of ancillary tests are likely to be available (Weller et al. 2001).

A recent study looked at the sensitivity and specificity of specific radiographic signs in the diagnosis of cheek tooth apical infection (Townsend et al. 2011). This study identified that radiographic signs present in the early stages of apical infection such as loss of lamina dura (LD) were less reliable as, although this radiographic sign was highly sensitive for picking up apical infections, it was poorly specific which would lead to a high number of false positive diagnoses which is not desirable in the clinical situation (Townsend et al. 2011). Advanced signs of cheek tooth apical infection may occur if infection has been present for many weeks, and the apex may develop lytic changes that manifest as periapical radiolucent halos or clubbed appearance of the tooth roots (Gibbs and Lane 1987). Increased radio-opacity or sclerosis of the bone supporting the tooth is often also noted in more chronic cases (Gibbs and Lane 1987). Other more advanced signs of apical infection include abnormal apical deposition of cementum (Gibbs and Lane 1987) and dystrophic calcification of the nasal conchae in chronic maxillary cheek tooth infections (Gibbs and Lane 1987). A multivariable model revealed moderate/extensive periapical sclerosis along with mild and moderate/
extensive periapical halo formation to be most significantly associated with apical infection (Townsend et al. 2011).

There is conflicting evidence in the literature as to whether detection of apical infection of the rostral 2 or 3 cheek teeth are more readily identified than the caudal cheek teeth (Wyn-Jones 1985; Gibbs and Lane 1987; Dixon et al. 2000; Tremaine and Dixon 2001; Casey et al. 2009; Townsend et al. 2011). It is likely that these differences result from variations in the chronicity of individual cases and study design.

Radiography is limited in its ability to image the internal structure of a tooth, particularly the pulp and infundibulum. Given the moderate sensitivity and specificity for detection of apical infection there will be cases that will have equivocal radiographic findings. In cases with facial swellings, repeat radiography after 2-3 weeks may help detection of the affected tooth as the changes may become more advanced. In cases with concurrent sinusitis, flushing of the affected sinuses and repeat radiography may make determining an underlying dental problem easier. Alternatively these cases may be referred for nuclear scintigraphy or computed tomography. Nuclear scintigraphy is an extremely sensitive and specific imaging modality for detection of apical infection (Weller et al. 2001; Barakzai 2005) whereas computed tomography provides superior anatomical detail of the paranasal sinuses and dental structures.

References

The use of computed tomography is gaining increasing acceptance in the field of equine dentistry. The demand by customers for precise diagnostics along with goal-directed treatment is being taken into account in many places. Therefore, the patient has not always to be examined using general anesthesia. Many facilities offer this technology while the horse is only being sedated. In order to do so, the patient is either being lowered or the gantry is being lifted to keep the horse’s head on the level of the examining table. Finally, the gantry is being moved on a special rail system over the horse’s head or the patient is being slid through the stationary gantry. With the aid of modern CTs the scan time of the head was reduced to a minimum. However, during sedation blurring caused by the horse has to be expected. Thus sometimes some of the scans have to be repeated to obtain evaluable results. Depending on the diameter of the gantry, the system, the length of the horse’s neck and the cooperation of the patient, the scan can extend from the incisors to the third or fourth cervical vertebra.

Figure 1. Sedated horse in standing CT unit.
In equine dentistry, there are the following indications for the use of computed tomography:

- Dental diseases with uncertain clinical and/or radiological findings
- Exclusion of a dental involvement
- Planning of a tooth-conserving measure
- Unilateral nasal discharge and/or odor
- Sinus diseases
- Head traumas or jaw fractures with possible tooth involvement
- Tumour diseases
- Head shaking and riding problems
• Recurrence of already treated dental, jaw or sinus diseases
• Follow-up or final exams after therapy

With the aid of CT, results can be obtained that allow a correct diagnosis in most cases. For example, in case of possible dental involvement in sinus disease. Moreover, surgical interventions at the teeth, the cranial bones or paranasal sinuses can be precisely planned.

Remnants of teeth in paranasal sinuses, for instance, demonstrate the value of computed tomography. In the beginning, the fragment is located. Thereafter, the ideal access just above the fragment is made out. Sensible nearby structures that have to be protected such as the infraorbital canal, the lacrimal duct, vessels and nerves, as far as they are presentable, have to be considered. Other helpful details are the distance between the bone and the object as well as the size of it. If the success of the surgical procedure has to be confirmed by a radiograph, it is advisable to have a preoperative one available, as they are easier to compare. This spares the necessity of a control CT. The possibility to obtain a three-dimensional reconstruction facilitates the orientation during surgery significantly. Finally, the findings can be easily shown and explained to the owner.

The following criteria are used for the assessment of teeth in CT scans:

• Integrity of the tooth
• Location of the tooth
• Shape of the reserve crown
• Formation of the roots
• Length of the tooth
• Internal structure of the tooth
  o Enamel
  o Cementum
  o Dentine
  o Pulp
  o Infundibula
• Periodontal space
• Alveolar bone
• Adjacent structures
  o Teeth
  o Sinus
  o Bone
  o Nerve canals

The following findings can be seen at the teeth themselves:

• Fracture or fissure
• Dislocation
• Torsion
• Lumps at the reserve crown or the root
• Spread roots
• Crooked teeth
- Deformity or malformation of dental hard tissue
- Inclusions of gas
  - In the pulp or the pulp chamber
  - In the infundibulum
  - In the periodontal space
- Densifications
  - Pulp stones
  - Sclerotic areas
- Widening of the periodontal space
- Thickenings/sclerosis/opening of the alveolar bone
- Condition of the alveolar bone
- Condition of adjacent teeth
- Altered shape of the cranial bone
- Swelling of the sinus mucosa
- Constriction of nerve canals

In the future, the number of facilities, that are equipped with such a diagnostic unit, will increase. Demand and continuous quality improvement in this area will accelerate this development. New fields of application concerning preventive or conservative measures are easily conceivable.
Complications of dental extraction

Henry Tremaine

The majority of surgical dental procedures performed in horses are exodontia techniques, although more recently occlusal restoration and endodontic treatments are being attempted more widely. The aim of exodontia is to detach the diseased tooth from the surrounding dental alveolus, by disrupting the periodontium with minimal trauma. In order for the alveolus to heal by fibrosis all dental tissue must be removed, since enamel has minimal reparative and dentine has limited regenerative potential. Teeth may be intact where the disease is limited to the endodontic pulp, or they may be carious and therefore structurally weakened, or fractured. In many instances the periodontium is mechanically stronger than the tooth and in both carious and intact teeth dental fracture can precede complete periodontal dehiscence. This can be avoided in many cases by appropriate selection of exodontia technique. Published techniques include extraction per os, minimally invasive transbuccal extraction, transbuccal screw insertion, retrograde repulsion (including minimally invasive) or removal of the tooth via a buccotomy. After removal of the tooth the alveolus is appraised clinically and radiographically. Dental or bone sequestra are identified by palpation of the dental alveolus, reconstruction and inspection of the tooth and post-operative imaging (radiography or CT). Dental sequestra are removed by careful precise elevation per os that is greatly assisted by oroscopic guidance, and with guiding radiographs or occasionally via the paranasal sinuses under endoscopic guidance. Identification and removal of sequestra can be challenging. Where possible all dental fragments should be removed at the time of extraction. In aged horses, with intact alveoli, root fragments left in situ may be clinically asymptomatic, and although they will obstruct alveolar granulation the trauma for their removal may be clinically and economically unappealing.

Young horses undergoing exodontia occasionally suffer from alveolar osteomyelitis (“dry socket” in humans). This probably results in the trauma of periodontal separation inadvertently traumatising the alveolar vasculature irreversibly ultimately leading to devitalisation and sequestration of the dental alveolus. Such cases are not detectable clinically or radiographically at the time of exodontia, but show signs of sequestration post operatively and can be confirmed by the presence of dental sequestration alveolar palpation from 14 days post operatively. The full extent of the sequestration may not be apparent immediately and these devitalised alveolar fragments are more easily removed after 4-6
weeks with rapid improvement in clinical signs.

Oro-antral or oronasal (orosinus fistulation) fistulation occurs when the dental alveolus is penetrated by suppuration from a peri-apical pulpitis, dental fracture or as a consequence of retrograde exodontia techniques. The alveolus is prevented from sealing by the presence of dental sequestra, non-vital bone or soft tissue or the passage of food from the oral cavity. Careful debridement and irrigation, followed by temporary alveolar sealing with a PVS implant will usually enable alveolar granulation to commence and eliminate food leakage. Chronic fistulae can be troublesome. Careful identification of the cause is obtained using diagnostic imaging, sinus endoscopy and oroscopic examination. Surgical debridement of diseased or non-vital soft tissue, and removal of any foreign material passing through the fistula is essential for long term healing. Where ongoing patency exists, curettage of the epithelium of the fistula followed by sealing can lead to granulation and healing. However where larger fistulae exist, more permanent protection using repeated PVS or acrylic dental implants, will enable granulation and sealing of the fistulae. Chronic fistula that are intransigent to granulation can be sealed using nasal or palatal transposition flaps, or levator nasolabialis transposition. These techniques have a high failure rate, and can be technically challenging.

Soft tissue complications that can be encountered especially with buccotomy and repulsion techniques include palatine arterial puncture, facial nerve branch paralysis, parotid duct puncture, incisional dehiscence and subcutaneous haematoma. Careful preoperative planning and precise techniques help to limit the likelihood of these.

References

Exodontia of equine cheek teeth using oral dissection with rotating power instruments (burs): 4 cases

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Introduction

Oral exodontia of equine cheek teeth using intra-alveolar dissection with dental burs is a novel, minimally-invasive technique. This method for tooth removal can be employed when dental extraction with forceps is not feasible because of a fractured or missing clinical crown, dental impaction or advanced dental decay. Despite being practiced by a limited number of equine veterinarians, to our knowledge this method has only been described in congress abstracts yet. In contrast to other methods of exodontia like minimally invasive buccotomy, trephination and subsequent dental repulsion or Steinmann pin repulsion, surgical access through soft tissues or bone is not required for the oral dissection of equine cheek teeth.

Material and methods

Exodontia of equine cheek teeth using oral dissection with burs was performed in 6 cases at the equine clinic of the first author between 2014 and 2015. Complete medical records of 4 cases were available and are presented subsequently.

Case 1. Quarter horse (male), 3 years. Using oral endoscopy, a fistulous tract adjacent to the bucco-mesial corner of 508 was diagnosed. A probe was inserted into the orifice and a tract extending about 70 mm in disto-apical direction, reaching the apical area of 108, could be probed. The granulation tissue at the position of the formerly extracted 107 appeared irregular and inflamed. Radiography revealed an irregular radiolucency around the apical area of 108 and the presence of a 508 cap.

Treatment was initiated with the removal of the deciduous 508 tooth cap. Sectioning of the unerupted 108 started at the occlusal surface. The tooth was dissected in mesio-distal, sagittal and midline planes. Subsequently the tooth was fractured along the dissection planes with root elevators. The fracture fragments were separated from their periodontal attachments and removed.

Case 2. Warmblood (female), 13 years. The owner complained about compromised mastication. Oral endoscopy revealed a buccal slab fracture, patent pulp horns and mesio-distal impaction of the 209. On radiographs a crown fracture, reserve crown
inhomogeneity, a distended periodontal space with an irregular lamina dura and loss of apical lamina dura, severe tooth impaction and dental malalignment were diagnosed.

Treatment was initiated by (limited) spreading. The remaining clinical crown fractured during loosening and was removed. The reserve crown and roots were sectioned in sagittal and transvers directions. All dental fragments were elevated and the alveolus was debrided.

**Case 3.** Icelandic horse (male), 18 years. The horse presented with a history of chewing problems. Oral endoscopy revealed severe infundibular caries (grade 5) resulting in sagittal fractures of 109 and 209. On radiographs decreased density of 109, irregularities of the mesial root, a lucent inhomogenic area dorso-mesial to the mesial apex and moderate tooth impaction were visible.

Treatment included the loosening and extraction of fragments, cutting of the reserve crown and roots in appropriate planes and elevation of all fragments with root elevators.

**Case 4.** Shagya-arabian (gelding), 11 years. The major complaint was apparent weight loss in short time. Oral endoscopy revealed a fracture of the clinical crown of 309 involving 3 pulp, diastemata-formation between 308/309 and 309/310 and a lingual slab fracture of 308. Radiographically the crown fractures and diastemata (described above) could be detected. Calcinosis of the mesial lamina dura, severe distention and irregularity of the periodontal space, apex resorption and remodelling as well as suspected root impaction were also diagnosed.

Treatment started as routine extraction. Spreading was only possible to a limited extent. The clinical crown of 308 was extremely fragile that normal elevation with forceps was impossible. Consequently the clinical crown was removed and the reserve crown sectioned with burs as described previously.

**Results**

Following complete tooth removal, the alveoli were packed with 0.2% chlorhexidine infiltrated gauze plugs. The alveolar packing was changed every second day until day 10. Medical treatment consisted of NSAIDs and penicillin for 5 days. All horses recovered completely within two months except case 4. In this horse mandibular sequestration occurred, which might have been caused by excessive drilling. The sequestrum could be identified poorly with radiographs and surgically removed under general anaesthesia. The adjacent bone was thoroughly curetted. Following sequestrum removal, the horse completely recovered.

**Discussion**

A common feature of all cases was the lack of sufficient clinical crown for the placement of extraction forceps to successfully complete oral dental extraction. Minimal invasive buccotomy and subsequent screw extraction as described by Stoll (2007) was considered; however, dental impaction (crown or root in cases 2 and 4) or the age of the horse (case 1) rendered this technique impracticable.

Reliable sedation (detomidin-hydrochloride, 0.01 mg/kg BW and butorphanol, 0.01 – 0.02 mg/kg BW), stable head positioning, perineural blocks and endoscopic guidance are mandatory for the successful performance of this procedure. Dust elimination and/
or suction devices are helpful, especially during mandibular cheek teeth sectioning. Sharp
burs of variable lengths (2-12 cm) with different tips (flame-, ball- or cylindric- shaped, 
3 mm diameter) are required. Individual planning of the dissection planes prior to the
procedure is advisable to facilitate subsequent fragment removal. Frequent cleaning of
the burs during sectioning might prevent heat injury of the adjacent tissues. Loosening
of the tooth prior to sectioning facilitates subsequent fragment removal and is also
helpful before deep sectioning to prevent drilling into the alveolar bone. If the tooth
has been loosened prior to sectioning, it is possible to “feel” the tip of the bur entering
the periodontal space. Excessive and deep drilling might result in inadvertent trauma of
the adjacent alveolar bone which might cause bone sequestration as described in case 4.

Conclusion

Exodontia of equine cheek teeth using oral dissection with rotating instruments is a
viable technique of exodontia for the removal of non- or partially erupted cheek teeth,
teeth displaying severe fractures or loss of the clinical crown and impacted or decayed
cheek teeth without the need for a surgical approach. However, this technique requires a
skilled and experienced operator. Endoscopic and specialised dental equipment, precise
planning of the procedure and the possibility to obtain high quality radiographs are
mandatory preconditions to successfully perform this technique.

References

Available upon request.
The equine temporomandibular joint: collagenous fibrous texture of the articular surfaces

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Background

The temporomandibular joint (TMJ) gained much attention in the field of equine dentistry as TMJ disorders are considered to play an important role in the aetiopathology of masticatory and dental diseases. In order to allow a precise diagnosis of pathological changes within the structures of the equine TMJ, the gross anatomical features of this complex joint were described in detail (Baker 2002, Rodriguez et al. 2006). Concomitantly, several studies were performed to describe advanced imaging techniques of the TMJ, i.e. radiography, computed tomography, ultrasonography, magnetic resonance imaging, scintigraphy and TMJ-arthroscopy (e.g. Weller et al. 2001, May et al. 2001, Rodriguez et al. 2007, Rodriguez et al. 2008, Ebling et al. 2009, Rodriguez et al. 2010). However, the microscopical anatomy of the equine TMJ remains widely undescribed (Ramzan 2006), although histological features might be of significant importance with regard to the understanding of TMJ pathologies, regenerative capabilities and biomechanical considerations.

The aim of the present study was to analyze the architecture of the collagen fiber apparatus in the articular surfaces of the equine TMJ to reveal typical morphological features indicating biomechanical adaptions.

Materials and methods

Ten adult horses without any history of TMJ disorders or dental diseases were included in this study. After euthanasia for other reasons, the TMJ was dissected and the articular surface bearing components of the temporal bone, the condylar process of the mandible as well as the articular disc were removed. The collagen fiber alignment in the articular surfaces was visualized by means of the so called split line technique. Subsequently, the articular surfaces were processed for histological evaluation. Paraffin wax sections were stained with Masson-Goldner Trichrome, Van Gieson’s and Safranin-O solution.

Results

The split lines on the different articular surfaces show a corresponding alignment. Within
the central two-thirds of the articular surfaces, split lines run in a rostrocaudal direction. In the lateral and medial aspects of the articular surfaces, the split line pattern varies, displaying curved arrangements as well as short or punctual split lines.

The articular surfaces of the bones as well as the articular disc are composed of thick collagen fiber bundles and fibrocartilaginous tissue. Fibroblast-like cells are present in almost all areas. Occasionally, isolated clusters of hyaline-like cartilage are intermingled between the fibrocartilaginous tissue.

The alignment of collagen fiber bundles as visualized by the split line technique was confirmed by the histological investigations. Moreover, collagen bundles running in subsurface layers were identified.

**Discussion**

The split line technique allows reliable identification of superficial collagen fiber bundles (Petersen and Tillman 1998, Bae et al. 2008). The direction of split lines is correlated with tensile properties and therefore indicates the predominant direction of joint movements (Mononen 2013).

The split line pattern in the equine TMJ reflects a relative movement of the joint components in rostrocaudal direction. However, this relative movement might result in a rotational movement of the mandible (opening of the mouth) or in a translation of the mandible (rostrocaudal movement). The present data does not allow to assign these movements to the different joint compartments (dorsal or ventral joint cavity). Therefore, it is still unclear whether the two joint compartments of the equine TMJ provide different types of motion as described for the human TMJ (Okeson 2012). The lateral and medial aspects of the articular surfaces provide split line patterns that indicate limited movements or movements around a perpendicular axis presumably associated with laterolateral movements during the chewing cycle.

**Take home message**

The complex movements during the equine chewing cycle are likely assigned to different areas of the TMJ. The predominant tissue components in the articular surfaces (cellular fibrocartilage) propose a pronounced capacity for adaptational changes and regenerative processes.

**References**


How to employ classical and transnasal sinoscopy in the postoperative treatment of primary and secondary (dental) sinusitis

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Introduction

Endoscopy of the equine paranasal sinus system has been described in several publications [1-6]. However all these studies have in common that the sinuses were accessed through surgically created portals. A non-invasive, trans-nasal approach was recently described [7].

An accurate knowledge of the anatomy of the nasal passages, the conchal recesses and bullae, the nasomaxillary aperture and finally of the paranasal sinuses is imperative for the clinical application of transnasal sinus endoscopy (TSE). Anatomical [8,9], computed tomographic [9,10] and clinical studies [11] describing key landmarks of the nasal, conchal and sinusoidal anatomy facilitate the comprehension of the complicated three-dimensional structures and are recommended readings prior to implementing traditional and/or transnasal sinus endoscopy in daily routine.

Endoscopic equipment

Flexible video- or fiber-endoscopes of 3-8 mm diameter may be utilized [7]. For visualization of the narrow recess of the dorsal concha and the sinus systems however, endoscopes with a diameter of less than 5 mm are required [7].

Conventional sinoscopy

Caudal sinus system

Following endoscope-insertion through a frontal trephination port, the tip of the endoscope is located in the concho-frontal sinus. The large (approx. 3x4 cm), oval-shaped frontomaxillary aperture is visible ventrally. The bulla of the maxillary septum (BMS) is located directly ventral to the rostral rim of the frontomaxillary aperture. The caudal sinonasal canal opens slit-like between the rostral edge of the frontomaxillary opening and the BMS.

The infraorbital canal can be identified as a rounded pipe-like bony structure (7-10
mm in diameter) aligned in a rostro-caudal direction. The ethmoid, the aperture into the sphenopalatine sinus, the alveoli of the two last maxillary molars and the medial wall of the bony orbit can also be visualized.

**Rostral sinus system**

The rostral maxillary sinus (RMS) and the ventral conchal sinus (VCS) can be examined by passing the endoscope through the perforation of the BMS. The infraorbital canal resembles a bony duct located in caudo-rostral direction which separates the rostral maxillary sinus (RMS, lateral) and the ventral conchal sinus (VCS, medial)

**Transnasal conchoscopy and sinoscopy**

Four separate conchal and paranasal sinus compartments can be examined using a transnasal approach. The paranasal sinus compartments can be accesses via the nasomaxillary aperture and the respective rostral and caudal sinonasal canals.

1. Rostral recess of the ventral concha.
2. Rostral recess of the dorsal concha.
   Nasomaxillary aperture, rostral and caudal sinonasal canal.
3. Rostral sinus system.
4. Caudal sinus system.

**1. Rostral recess of the ventral concha**

The endoscope is inserted into the middle nasal meatus and directed caudo-laterally along the dorsal and lateral aspects of the ventral concha. It is then directed into the space between the dorsal aspect of the ventral concha and the medial wall of the maxillary bone. Bending the tip of the endoscope in ventral direction, it is guided into the recess of the ventral concha. The elastic properties of the conchal lamella in this area facilitate the insertion of flexible endoscopes of up to 8 mm diameter [7].

**2. Rostral recess of the dorsal concha**

In contrast to the rostral aspect of the ventral concha, the bony spiral lamella of the dorsal concha cannot be compressed as easily during endoscope insertion [7].

A low-diameter endoscope (< 5mm) is inserted into the middle nasal meatus and advanced about 6-8 cm caudal to the nostrils. At this location the gap between the dorsal concha and the medial wall of the maxilla expands over a distance of 2-3 cm which allows the insertion of a small endoscope into the conchal recess [7].

**Nasomaxillary aperture, rostral and caudal sinonasal canal**

To reach the nasomaxillary aperture, a low-diameter endoscope (< 5mm) is positioned on the dorsal aspect of the ventral concha in the middle nasal meatus and inserted about 20-25 cm, depending on the horse’s size [7].
3. **Rostral sinus system**

The rostral sinonasal canal resembles a narrow, horizontally-orientated slit located laterally between the ventral conchal lamella and the medial wall of the maxilla. It is hardly possible in normal horses to pass an endoscope through this narrow orifice. However, the rostral sinonasal canal significantly widens in chronically sinusitis-affected horses, allowing the insertion of a small endoscope into the rostral maxillary or ventral conchal sinus [7].

4. **Caudal sinus system**

From its position on the dorsal crest of the ventral concha, the endoscope can be advanced caudally along the crest. The sickle-shaped, 3-5 mm wide caudal opening can be visualized. Passing a small-diameter endoscope through the caudal sinonasal canal into the caudal maxillary sinus is frequently possible also in healthy animals.

**Postoperative sinoscopic findings**

Despite the fact that gross accumulations of purulent material are usually removed during sinus surgery and flushing of the sinuses with sterile solutions is routinely performed in almost all horses, remnants of disrupted tissues, necrotic bone, blood clots and inspissated purulent exudates frequently remain. The chance of reoccurrence of sinusitis is extremely high in these cases.

Postoperative sinoscopy is routinely performed in all horses subjected to sinus surgery at the reporting clinic every other day until no remnants of gross necrotic tissues, sequestrated bone or infectious inspissated material can be visualised endoscopically.

Sinoscopy has also proven useful for the detection and removal of remnants and/or regrowth of PEH-masses located in the paranasal sinuses. Trans-endoscopic intralesional injection of formaldehyde or endoscopy guided laser ablation can be utilized as treatment strategies for PEH [9,10].

**Take home message**

Postoperative (conventional and transnasal) sinoscopy is an effective method to detect and remove debris, necrotic tissues, inspissated exudates, foreign material and remnants or regrowth of PEH.

This technique might help to decrease postoperative hospitalisation time and further increase the success rates of surgical procedures in the equine paranasal sinuses.

**References**

Surgical Extension of the Nasomaxillary Aperture (SENMAP): A surgical technique to improve sinus drainage in standing, sedated horses

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Introduction

As described in man, thickening of the respiratory epithelium or inspissated pus may also impede drainage of the paranasal sinuses in horses. Surgical or minimal invasive procedures like balloon-catheterisation of the nasomaxillary opening are required to re-establish sinus drainage in these animals. Two surgical techniques to improve sinus drainage in standing, sedated horses are described.

Material and methods

Surgical enlargement of the nasomaxillary aperture (SENMAP) was performed in 8 standing, sedated horses. All horses were restrained in stocks and initially sedated with a bolus of detomidine (0.015 mg/kg, i.v. and butorphanol tartrate (0.015 mg/kg i.v.). Constant rate infusion of the same drugs was used during the procedure. Local anaesthesia consisted of a maxillary nerve block (EPFBI technique) and subcutaneous infiltration of the trephination site (5-10 ml of mepivacaine 2%).

Surgical enlargement of the nasomaxillary aperture (SENMAP)

The procedure was performed in 8 horses (2 mares, 6 geldings; age range: 4-20 years) in which obstruction of the caudal sinonasal channel impeded sinus drainage.

The surgical field was prepared according to standard protocol and surgical access to the conchofrontal sinus was created with a 25 mm Galt trephine.

Fenestration of the Bulla septi sinuum maxillarium (BSSM) was performed as described by Perkins et al. Following sinoscopy, the sinuses were flushed with Ringers solution. In horses with compromised sinus drainage, a portion of the ventral lamella of the dorsal concha (approximate size: 3-4 cm x 3 cm) located rostro-medial of the frontomaxillary aperture was surgically removed under endoscopic control using small chisels, scissors and crocodile forceps. Bleeding was controlled using electro cautery and sinus packing if needed. Subsequently a portion of the rostro-medial lamella of the ventral concha was removed starting at the fenestration site of the BSSM. Resection of
conchal lamella was continued until unobstructed sinus lavage could be performed. A Foley catheter (20 G) was inserted into the sinuses through a stab incision in the centre of the skin flap. Wound closure was performed in a single layer with monofilament non-absorbable suture material (USP 0) in simple interrupted pattern.

**Postoperative treatment**

All horses received antibiotic treatment following sensitivity testing. Sinoscopy and trans-endoscopic sinus lavage was performed in all horses starting on the second postoperative day and repeated every second day until all sinus compartments were cleared from foreign content (coagulated blood, inspissated pus) and signs of sinusitis decreased.

**Results**

Uncompromised drainage could be established in all horses. Marked haemorrhage (> 4 l) occurred in 2 horses. Postoperative transnasal sinoscopy of the caudal maxillary and frontal sinus was possible in all horses using a 5.5 mm flexible endoscope. Sinusitis resolved in 7 of 8 patients. In one horse occasional low grade nasal discharge was reported.

**Conclusions and take home messages**

1. SENMAP is a surgical procedure to effectively restore drainage of the paranasal sinus systems in sinusitis-affected horses with obstructed sinonasal passages.
2. The described technique can be combined with the technique described by Perkins et al.4 to approach the paranasal sinus system endoscopically and facilitate postoperative sinoscopy and trans-endoscopic sinus lavage.
3. SENMAP additionally facilitates postoperative transnasal sinoscopy via the surgically enlarged nasomaxillary aperture.

**References**

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Osteonecrosis of unclear etiology in the maxilla of a dog: a case report

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Osteonecrosis of the jaws, characterised by chronically exposed necrotic bone in maxilla or mandible, is considered an infrequent clinical finding in dogs that is commonly associated with radiation therapy, previous dental extractions, jaw fractures and with a history of dental disease or systemic antibiotic use. Cocker spaniels and scottish terriers are reportedly overrepresented.

We report a non-radiation-related osteonecrosis with a bony sequestrum in the right upper distal maxillary area of a 4 year old labrador retriever with no previous documented history of dental treatment, trauma within last 3 years or long-term antibiotic use, no clinical or laboratory signs of systemic disease and with only mild to moderate dental disease (old complicated crown fractures of three teeth and two teeth with attachment loss due to periodontal disease, those teeth not being directly associated with the osteonecrosis area).
Clinical research on the use of an antlerogenic stem cell homogenate on oral mucosa and periodontal tissue in dogs

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In a clinical study over 75% of dogs older than two years have a varying degree of disease of the oral mucosa and periodontal tissue. Despite wide access to a number of specimens and drugs used in veterinary dentistry for treatment and prophylaxis, new active substances are constantly sought.

Numerous studies on the biology of stem cells have been carried out in recent years. The obtained results laid the foundations for a new approach in regenerative medicine. To date, autologous tissues have been the main source of stem cells in veterinary medicine. The study was carried out as part of a project that used xenogenic stem cells acquired from the Cervus elaphus deer antlers. The use of MIC-1 stem cells that are derived from the antlers of the red deer (Cervus elaphus) and are extracted into cell cultures gives new regenerative possibilities.

The aim of the study was to apply and assess a medical product containing an antlerogenic stem cell homogenate on the mucous membranes and periodontal tissue in dogs.

This study was carried out as part of a larger project and enabled the assessment of the usefulness of the antlerogenic stem cell homogenate in veterinary medicine.

The study in canine dentistry was carried out on 25 Faculty kennel dogs. The medical product with and without the antlerogenic stem cell homogenate was applied twice a day for ten days. The clinical and dental condition of the dogs and histological image, haematological and biochemical blood parameters in venous blood were assessed at two time points.

The study demonstrated that the gel containing an antlerogenic stem cell homogenate can be safely used on mucous membranes and periodontal tissue in dogs. No statistically significant differences in the clinical condition of the dogs and blood parameters were found in the course of the study.

Evaluation of the results dental examinations will be presented in Scientific report Poster in 25th European Congress of Veterinary Dentistry in Dublin.

This study was conducted as a part of the UOD-DEM-1-352/001 project sponsored by the National Center for Research and Development.
References


Periodontal disease (PD) is an inflammatory process of the periodontium highly prevalent in dogs and associated with a chronic systemic inflammatory status. Appropriate treatment for PD can cause a shift of this status. C reactive protein (CRP) is an acute phase molecule used as a diagnostic indicator of systemic disease in humans. In dogs, CRP values have already been assessed in periodontal disease.

The present study aimed to determine the systemic effects of periodontal disease through the measurement of serum CRP and to evaluate the impact of periodontal treatment in diseased animals comparing CRP levels with healthy dogs without periodontal disease.

Thirty-nine dogs from different breeds were subjected to a detailed physical examination, haematological analysis, dental charting and intra-oral radiography. The control group comprised 28 animals under the age of 2 years and without any inflammatory condition including periodontal disease. The diseased group comprised 11 animals over 2 years old diagnosed with periodontal disease. Serum CRP measurement was performed using the kit CRP-Canine (Randox, Ireland) once in the control group and twice in the diseased group, pre and post operatively, with a 30-day interval.

Significant differences (p=0.001) were found between the control (2.947 ± 1.428 mg/L, 95% CI) and pre-treatment diseased group (13.824 ± 17.273 mg/L, 95% CI) but not between the control and post-treatment diseased group (3.376 ± 1.235 mg/L, 95% CI).

CRP measurement has been previously assessed in the dog with periodontal disease, including pre and post periodontal treatment. However, the CRP values reported were always in the presence of periodontal disease and never have been compared with a control group. This study allowed a more accurate establishment of the association between periodontal disease and systemic inflammatory status by comparing the values with a normal control group. CRP values of the control group were significantly lower than those of the diseased pre-treatment group and that CRP values of the diseased post-treatment were significantly similar to those of the control groups. Additionally, the specific analytical sort used in this study was highly specific for this species, allowing a more accurate determination of all values, not being, as for the author’s knowledge,
previously used in clinical studies of periodontal disease in dogs.

In conclusion, CRP levels are increased in dogs with periodontal disease and there is a measurable effect on systemic inflammatory impact of periodontal treatment, when these measurements are compared with a control group.

References

Objective: To determine the prevalence of the most common dental abnormalities seen in a population of brachycephalic cats from different geographic origins.

Design (type of study): Prospective case series multicentre study.

Animals: 50 purebred brachycephalic cats.

Procedures: Routine oral exam, charting, three-view photographs and full mouth radiography under general anaesthesia.

Results: Neutrocclusion was seen in 64%, but of those, 36% presented with some kind of tooth malposition. Mandibular mesiocclusion was present in 28%. Crowding was present in 56%, with the incisors being the most affected teeth. Position change was diagnosed in 64% of the cats; where tipping was the most common abnormality, followed by rotation and impaction. Numerical abnormalities were present in 76% of the cats, 12% polyodontia and 64% oligodontia respectively. Periodontal disease was reported in 72% of the cats. Age differences observed were not statistically significant.

Tooth resorption was present in 70% of the cats. When assessing teeth individually, 8.7% were affected with external inflammatory resorptions, and 5.8% with external replacement resorptions. The results suggest a significant relationship between resorption type and affected tooth.

Conclusions and clinical relevance: Brachycephalic cats show specific anatomical features that predispose them to dental disease. Early awareness is crucial to diagnosing dental anomalies and preventing potential complications.
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