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Emergency

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ABCs of Veterinary Dentistry

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Editor's Note

Lots of emergency tips in this edition. Quick techniques using ultrasonography which doesn't require moving the patient around - rather bringing the ultrasound to the bedside than taking the patient to radiography.

Additionally a nice few case studies for the dentistry article which show just how aesthetic the results of some quite dramatic maxillary and mandibular resections can be.

Also included is an article on salivary gland function - which is quite detailed on the neuroanatomy, but is quite informative if you persist. As an add-on to this article I have included two short papers on salivary gland enlargement and sialoadenosis with hypersialosis as a secondary condition, especially as a result of canine spirocecosis as well as phenobarbitone responsive hypersialosis (limbic epilepsy).

I frequently see these cases as second or third opinions where the diagnosis has been missed. The presentation is really typical, as described in the articles, and is a dysphagia rather than a nausea, which these patients are usually treated for, and the correct treatment is really rapidly effective.

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PREVIOUS EDITION: JULY 2018

Feline CKD: Therapeutic Goals
ABCs of Veterinary Dentistry: "N" is for No
Gastrointestinal Therapy, Anti-emetics and Antacids

NEXT EDITION: NOVEMBER 2018

Pyoderma in Bull Terriers
TFAST - Thoracic scanning
ABC's in Feline CKD

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We welcome any comments, contributions, topic suggestions and letters for publication. Send them to:

The Editor, PO Box 232, GROENKLOOF, 0027
Tel: (012) 346 1590, 082 575 6479. Fax: 086 671 9907
Email: lieselvdmvet@gmail.com
(Dr Liesel van der Merwe)

Advertising Enquiries: The Publisher. Vetlink.
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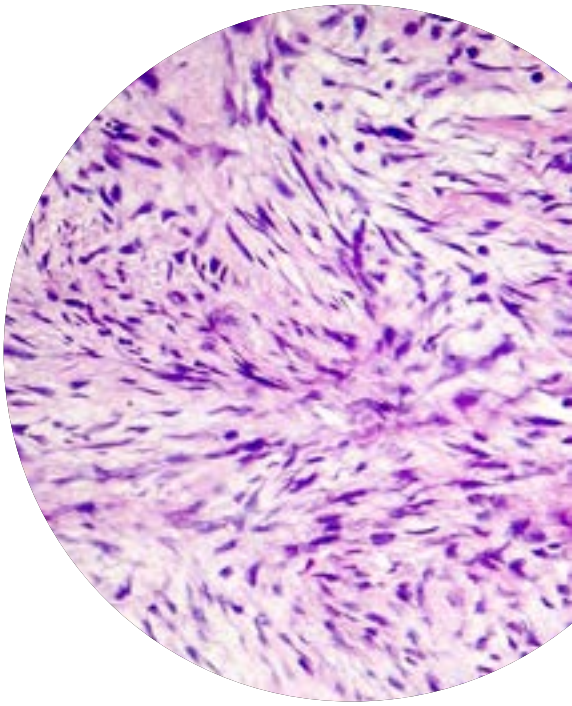
Maximising Histopathology in Oncology

The 3 Pillars for Diagnostic Success and Effective Therapy



Dr Rick Last (BVSc; M.Med.Vet(Path); MRCVS)
Specialist Veterinary Pathologist

Although surgical pathology forms only a portion of veterinary oncology, if used correctly within the framework of the veterinary team (clinician, veterinary nurse, owner, pathologist), it provides the opportunity for rapid accurate diagnosis and immediate implementation of relevant therapy. The pathologist reading the biopsy is removed from the patient and outside the mainstream flow of medical information, pertaining to the particular patient circulating within the practice. The pathologist must therefore rely on members of the clinical team to provide important information which is almost always essential to reaching a diagnostic conclusion or diagnostic rule out's.

Effective veterinary oncology requires the import of different skills of two different but related groups of veterinarians.

What is a surgical biopsy?

This procedure is a temporary partnership between a clinician and his support team of practice staff and the pathologist acting on behalf of the patient to aid the clinician in:

1. Making or confirming a diagnosis.
2. Assisting in the prognosis of the case by assessing the progress of therapy, grading and staging of neoplasms and determining the completeness of surgical excision.

Surgical pathology is a prospective disease investigation on a live patient under medical care and whose disease course can theoretically be altered by therapy. Therefore, a premium is placed on speed and accuracy. However, there are numerous and significant problems in surgical pathology that negatively impact the desired result of a speedy and accurate specific tumour diagnosis.

The upside of surgical pathology is that there is usually no or very minimal autolysis which facilitates accurate identification of morphological detail and accuracy of tumour marker stains. On the downside one needs to consider the following:

1. The pathologist does not see the gross lesion. This can be circumvented to some degree by more efficient use of digital photography by clinicians.
2. There is often a poor or no history provided.
3. The pathologist does not select the tissue and lesions to be examined.
4. Specimens submitted are often very small, distorted and marginally adequate for a thorough evaluation.
5. Tissues are sometimes improperly fixed and partially autolyzed.
6. In many instances the pathologist cannot get additional tissue to examine so they must make a diagnosis with what they have before them.
7. The clinician needs an answer quickly so there is minimal time for reflection.

8. The pathologist usually lacks corroborative data.
9. The clinician will believe the diagnosis is accurate and act on it.
10. The patient is still alive, so the diagnosis better be correct.

The World of Veterinary Oncology

Clinicians and pathologists inhabit very different parts of the oncology world and have become isolated from one another geographically as well as scientifically. Such barriers need to be broken down to achieve the best possible outcome for patients and this requires careful, timely and accurate communication among all the parties involved in the biopsy process. This breakdown in communication accounts for most of the problems, mistakes and complaints.

The objective of the biopsy

The objective is to obtain a sample of tissue from a living patient to identify or characterize a neoplastic process. The surgeon or clinician is involved in the decision to biopsy, the sampling of the tissue, preparation of the tissue and submission of the samples to the laboratory. The pathologist and the laboratory team continue the process, create the microscopic slides, evaluate the specimens and communicate the information back to the clinician.

New and minimally invasive techniques are being applied in surgical pathology and to justify the cost of these techniques, one must maximise the diagnostic and therapeutic yields from the procedure. As a result, clinicians are wanting more useful information from each sample and with the trend toward smaller and smaller pieces of tissue, increases the pressure for an accurate diagnosis on the first samples submitted. With the cost involved with these new procedures it is unlikely that a second biopsy would be an option if the first one did not yield a useful diagnosis.

Total patient evaluation

Clinicians practice the principle of total patient evaluation when making judgments about diagnosis and treatment. They consider all of the data and facts at hand in reaching their conclusions. Likewise, pathologists should not base their diagnosis just on what they see on the slide. Unfortunately, in many scenarios none of the data facts are provided to the pathologists and all that they are left with is a slide to evaluate in isolation. Pathologists should describe what they see in the biopsy but make a final interpretation or diagnosis in context of all the relevant information about the case. This ability for pathologists to perform a total patient evaluation depends almost entirely on the clinician.

- Total patient evaluation for pathologists: read a slide but interpret a patient.
- Total patient evaluation for clinicians: give the pathologists what they need to make a patient interpretation.

Outcomes of the biopsy procedure

- Best outcome: rapid, accurate diagnosis of a specific neoplasia and timely application of appropriate therapy.
- Worst outcome: no diagnosis made and therapy is delayed or inappropriate.
- The middle route: a partial diagnosis is made but does not translate into a specific entity. The non-specific nature does not meet the clinicians expectations or the patient's requirements.

The particular outcome category depends largely on total patient evaluation information being provided pathologist.

Getting a diagnostic biopsy

This remains the most important task of the clinician in his workup of the tumour case. So, what constitutes a diagnostic biopsy?

1. Adequate amount of tissue.
2. Representative of the neoplastic process.
3. Sufficiently free of artefacts to permit a definitive evaluation.
4. Signalment, history, description of the lesion.
5. The clinicians differential diagnoses, rule out's, thoughts.

Adequate amount of tissue

Clinicians had a wide array of techniques at their disposable with which to obtain surgical biopsies for tumour diagnosis. Pathologists should never question first technique used by the clinician, as this is often a 1st rule of medicine (do no harm) decision. However, clinicians should be fully aware of the limitations and potential problems associated with their choice of biopsy technique.

A) Fine needle aspirates

- Advantage: easy, minimally invasive and inexpensive.
- Use: when significant conclusions can be drawn by evaluating individual dissociated cells and is largely employed to distinguish between inflammation and neoplasia.
- Disadvantage: it is extremely limited as no architectural detail can be examined.

B) Tru cut biopsies

- Advantage: permits some evaluation of architecture, are minimally invasive and multiple samples can be obtained.
- Disadvantage: a small amount of tissue is collected, and so only limited architecture can be evaluated, which may lead to an incorrect interpretation. Like looking through a porthole.

C) Punch biopsies.

- These biopsies were designed specifically for skin biopsies in dermatology pathology (and should not be used for other system purpose).

D) Endoscopic and laparoscopic biopsies.

- Advantage: it is a minimally invasive procedure to observe and sample tissues and organs otherwise not available without open laparotomy, thoracotomy or cystotomy. Corrective or therapeutic surgery can be performed at the same time.
- Disadvantage: small tissue sample size, although larger than the other needle biopsy techniques. Only muccousal biopsies taken, the muscular layers are inaccessible. General anaesthesia is required drives up the costs. There are a unique set of issues related to specimen orientation and induced artefacts, as a consequence of the small size and friable nature of the samples.

E) Incisional biopsies.

- Advantage: provides a far greater amount of tissue in the other techniques. No special tools are required beyond scalpel and forceps. Cytology can be performed on the same specimen. As the sample is larger there is less likelihood of handling artefacts which distort the tissue.
- Indications: performed when an excisional biopsy cannot be obtained.

F) Excisional biopsies.

- Advantage: 100% of the lesion can pre-provided for examination. Diagnosis and treatment can be incorporated in the same procedure.
- Contraindications: when the lesion is too large or in an inoperable location.

Representative of the pathological process

Pathologists frequently make the fundamental error of assuming that the biopsy they receive contains the lesion. Neoplastic processes differ remarkably in their distribution through tissues and organs. Therefore, biopsies that do not provide the entire lesion often do not produce tissue sections that are representative of the neoplastic process.

In uniform lesions the neoplastic process is diffused and involves similar cells, such that sampling at any site yields the same diagnostic tissue, as might be observed with hepatoid adenoma or hepatoma etc.

Non-uniform lesions are punctuated or even dominated by non-specific secondary or reactive changes which obscure diagnostic lesions. Osteosarcoma, mammary gland neoplasia, splenic haemangiosarcoma, soft tissue sarcomas etc. frequently induce necrosis, inflammation or stromal reaction scattered throughout the lesion and in very small biopsies may not be accompanied by the underlying neoplastic condition.

The lesion may not be present or included in the biopsy because either it was not sampled or due to the complication of necrosis, inflammation and reactive tissue. Always use techniques that permit direct visualization of the tissue being sampled (ultrasound-guided, CT guided etc.), take multiple tissue samples, take the interface with normal as well as areas that look different.

Sufficiently free of artifacts

Artefacts are structures or substances not normally present but produced by some external agent. Artefacts which prevent interpretation are termed fatal. The most common artefacts encountered are related to the collection and handling of specimens both during and immediately following the biopsy procedure.

- Surgical crush artefact.
- Cautery artefact or thermal injury.
- Freezing artefact.

Fixation

Fixation is the singularly most important step in producing good histopathology slides.

1. No tissue placed in formalin should ever be thicker than 0.5-1.0 cm in thickness.
2. The amount of formalin should be at least 10 – 15 times the volume of tissue placed in it.

Large specimens such as spleens and large masses should be bread sliced into 1 cm slices to ensure adequate penetration of the formalin fixation. Large pieces of tissue contain sufficient extracellular fluid that they can dilute the formalin to substantially less than 10%, resulting in the tissue in the jar undergoing autolysis. Autolysis not only interferes with diagnosis, but impairs accurate mitotic index counts and frequently compromises tumour marker immunohistochemistry.

Size

The single most important variable affecting the likelihood of getting a diagnostic biopsy is specimen size. Small specimens (endoscopic, rhinoscopic, Tru-cut etc.) are highly susceptible to crush artefact. Because such small pieces of tissue may not include diagnostic tissue and in addition can be a challenge to properly orientate in the cassette, very small pieces often produce non-diagnostic biopsies.

Large specimens such as amputations, splenectomy's, large subcutaneous masses etc. resist crush artefacts, but are susceptible to poor fixation. It is important to bread slice such large specimens and then select the interface between abnormal and normal tissue and then subsample different looking areas of tissue for submission. As a general rule the larger the mass the more subsample should be taken. Splenic masses should include at least 4 to 6 pieces.

The margin principal

Surgeons should always tag what margin is important and note this on the submission form. A common problem is when multiple masses are included in the same jar without being marked.

Signalment, history and description of the lesions

Histopathology evaluates only a tiny fraction of the affected tissue. In order to fully orientate pathologist and facilitate a total patient evaluation, clinicians and the practice staff must describe what they saw. Descriptive pathology is a skill that veterinarians obtained from vet school, but these skills are frequently lost due to disuse. Again, this comes back to poor communication between pathologists and clinicians, which ignores the important principles that descriptive pathology provides to both parties. With the emergence of sophisticated imaging techniques, clinicians are starting to see the value of descriptive pathology skills. What clinicians see is extremely important in case management and of enormous value to the pathologist in interpreting their biopsies. Pathology is not just for autopsies and it's not just for pathologists anymore either.

Descriptive pathology for biopsy specimens

The purpose of the gross description in surgical pathology is to orientate the pathologist reading the histopathology slide and to verify what was received at the laboratory. Description should be brief and to the point and include information pertaining to size, colour, location and distribution. Remember to describe what you see as though you were speaking to a blind person, because effectively the pathologist is unsighted to what you are seeing on the patient. Take photographs and attach to the request. If you do not share this information with them, you may lead them into making the wrong diagnosis. Digital pathology has provided clinicians with a very useful and helpful tool to assist with gross description of the lesions.

Signalment **and history**

Many clinicians are still under the wrong impression that if they tell the pathologist anything it will bias him or her in their decisions. Such an approach frequently leads to serious errors in medical judgment and mis-diagnosis. If the goal of the biopsy is to fool the pathologist, then tell them nothing. If the goal of the biopsy is to get an accurate diagnosis quickly then tell the pathologists what you know or think.

The **submission form**

This is a critical document in solving many of the communication problems of surgical pathology. A good form is one that is 100% dedicated to the biopsy.

Remember on this form to give your pathologists the what 4!!

- What did you see?
- What did you do?
- What do you think?
- What do you want?

The 5 important descriptive elements that should be included on every oncology submission form are the following:

1. Signalment – species, age, breed, sex (intact or not).
2. Clinical or historical daughter – pertinent to the case, brief and to the point (1 to 2 lines).
3. Precise location of the lesion or origin of the sample.
4. Descriptive characteristics – size, colour, shape, distribution.
5. Clinicians thoughts – differential diagnoses, rule out's.

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CPD Questions

Maximising Histapathology in Oncology Patients AC/1986/18

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Question 1
In oncology which one of the following cannot be achieved with a surgical biopsy?

a. Confirming a diagnosis.
b. Assessing the progress of therapy.
c. Grading of the tumour.
d. Determining the completeness of excision.
e. Determining the cause of death.

Question 2
Which one of the following features is considered the upside of surgical pathology?

a. The pathologist does not see the gross lesion.
b. They is usually minimal or no autolysis.
c. There is time for reflection on the pathology.
d. Minimal history is required.
e. Clinical data is not needed.

Question 3
Which one of the factors listed below does NOT constitute a diagnostic biopsy?

a. An adequate amount of tissue.
b. Is representative of the neoplastic process.
c. Artifacts are not an issue.
d. Signalment, history and description of the lesion.
e. Differential diagnoses for the lesion.

Question 4
Which one of the factors listed below is an advantage of Tru-cut biopsies?

a. They permit some evaluation of architecture and all minimally invasive.
b. Significant conclusions can be drawn from histopathology in 100% of cases.
c. Surgical margins can be deceased.
d. Metastatic spread can be evaluated.
e. Mitotic index can be considered reliable.

Question 5
Which one of the factors listed below is a disadvantage of endoscopic/laparoscopic biopsies?

a. It is an invasive procedure.
b. Tissues cannot be observed for sampling.
c. Corrective surgery cannot be performed at the same time.
d. General anaesthesia is required.
e. Open laparotomy, thoracotomy or cystectomy is required.

Question 6
Which one of the following are critical to obtaining a biopsy representative of the neoplastic process?

a. Collecting biopsies from the center of the lesion.
b. Necrotic areas provide highly diagnostic information.
c. Always use techniques which allow for direct visualization of the lesion being sampled.
d. Areas with haemorrhage and/all inflammation provide diagnostic information.
e. Conflict biopsies from the age of the lesion.

Question 7
What one of the measurements listed below is the ideal thickness of tissue slices to ensure that adequate formalin fixation?

a. 0.5 – 1 cm.
b. 1.5 – 2 cm.
c. 2.5 – 3 cm.
d. 3.5 – 4 cm.
e. 4.5 – 5 cm.

Question 8
Which one of the following biopsies would be the most susceptible to crush artifacts?

a. Incisional biopsies of solid masses.
b. Punch biopsies of skin masses.
c. Incisional biopsies of skin masses.
d. Tru cut biopsies.
e. Excision will biopsies.

Question 9
Which one of the following is not essential in the clinicians gross description of the tumour?

a. Size.
b. Colour.
c. Location.
d. Distribution.
e. Smell.

Question 10
Which one of the following actions by clinicians is most likely to result in misdiagnosis?

a. Describe what you see at gross examination.
b. Explain what treatment you employed.
c. Tell the pathologist nothing to avoid bias.
d. Provide the pathologist with your clinical differential diagnoses.
e. Tell the pathologist what you want him/her to look for.

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8 Practical Facebook Tips and Ideas for Vets - Johan Mouton
10 Instagram Best Practices for Vets - Johan Mouton
Advertising rules - SAVC - Myths and Facts - John Adam

DAY 2

Help - Veterinary staff are different! ***Managing vet employees*** - Andrew Butters

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Prof. Andrew Leisewitz

Companion Animal Clinical Studies
Andrew.leisewitz@up.ac.za

Andrew Leisewitz graduated with his BVSc degree in 1987. This was followed by an Hons in 1991, an MMedVet(Med) in 1995 and a PhD in 2006. Andrew was appointed as a senior lecturer in the then Department of Medicine at Onderstepoort in June 1990. He is been a full professor of Companion Animal Clinical Studies since 1995 and is a diplomat of the European College of Veterinary Internal Medicine – Companion Animals. He has worked as a specialist clinician in the Onderstepoort Veterinary Academic Hospital for nearly 30 years. He runs a dermatology referral clinic within the Onderstepoort Veterinary Hospital and in a Johannesburg based specialist referral hospital. He has initiated a dermatology specific internship at the Faculty which will run for the first time in 2019. He is an active teacher of undergraduate and post graduate veterinary students, and is a frequently invited speaker and presenter of continuing education for the profession in the sub-region. His research focusses on understanding the mechanisms of infectious disease making use of canine babesiosis and distemper virus infection as models for this work.

SOUTH AFRICAN VET DERMATOLOGY INTEREST GROUP

Autoimmune Skin Disease in Dogs and Cats

This is a unique and less common group of disease in which the body's own immune system turns on self – which in this case are components of the skin. We will discuss the diseases in two broad categories, namely those diseases that are vesicular or pustular in nature and those diseases that are not characterised be vesicles or pustules. We will spend most of discussion time on the most common of these disease.

Discoid lupus is probably the most common autoimmunity seen in dogs. It is not a vesicular disease but is characterised most commonly by nasal depigmentation. Pemphigus foliaceus is probably the second most common autoimmune skin disease in dogs and the most common in cats. It is characterised by superficial cleft formation (resulting in vesicles and then pustules and then crusts) in the epidermis and typically affects the face and feet but may involve the trunk as well.

Other forms of pemphigus are significantly less common and result in cleft formation deeper in the epidermis and have a more severe disease presentation and poorer outcome. Pemphigus may also be triggered by idiosyncratic reactions to drugs as well as by neoplasia. Other even less common dermal auoimmunities include lupoid onychodystrophy, erythema multiforme and toxic epidermal necrolysis.

Systemic autoimmunities such as systemic lupus erythematosus may also result in skin lesions. The corner stone of diagnosis rests on histology of well chosen, properly timed and atraumatically collected skin biopsies. Treatment usually depends on glucocorticoid induced immunosuppression. This treatment is frequently augmented through the addition of other systemic immunosuppressive agents. Prognosis varies widely depending on the specific disease diagnosed.

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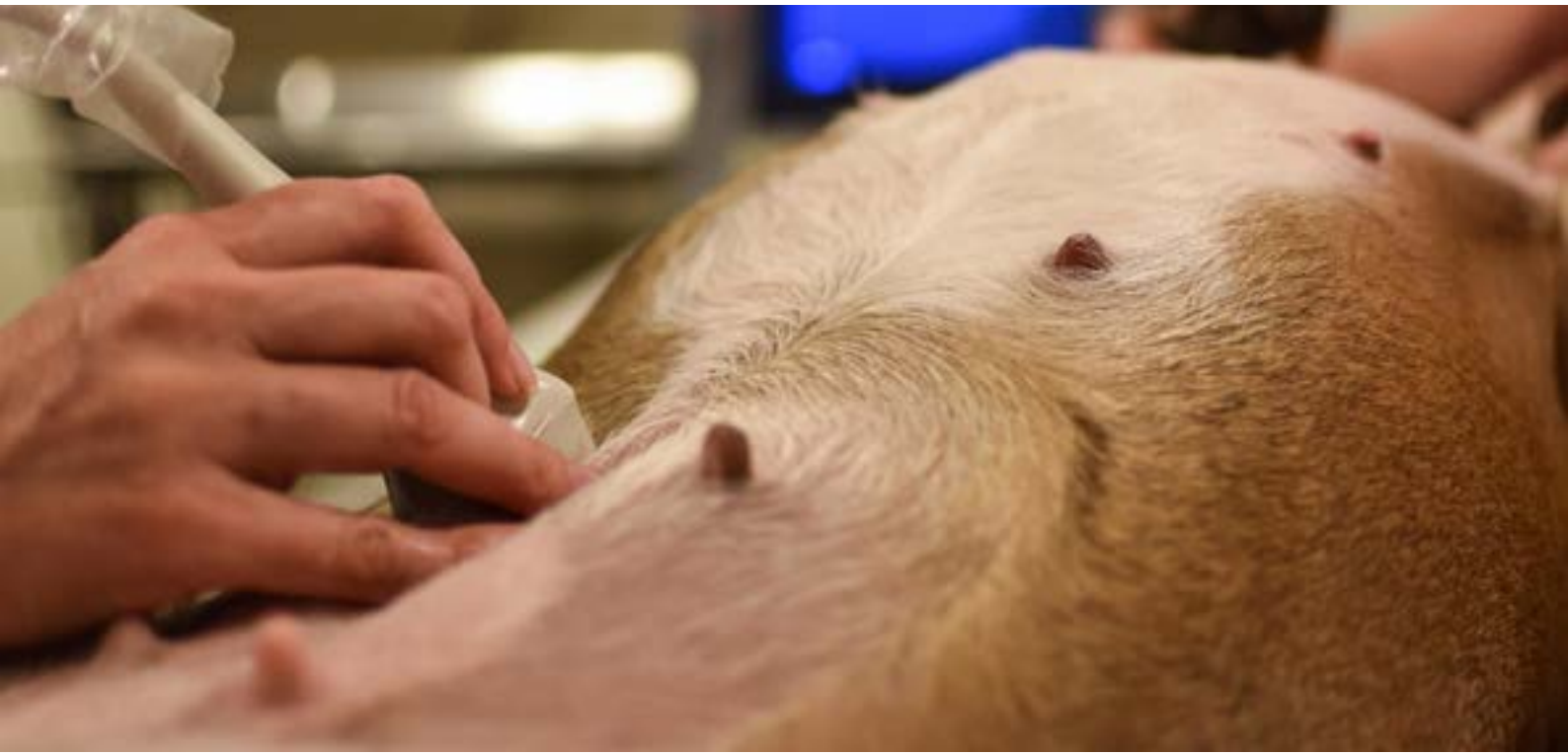

Since ultrasonography is becoming more accessible to all veterinary practitioners, learn all about this ultrasonographic examination protocol for trauma and start saving lives.

By Garret Pachtinger, VMD, DACVECC

Most veterinarians are schooled on ARDS, SIRS and other common medical acronyms. But are you ready for FAST³?

FAST, which stands for focused assessment with sonography for trauma, is a rapid bedside ultrasonographic examination and screening test for blood—or other abnormalities—after trauma. More specifically, there are the abdominal FAST (AFAST), thoracic FAST (TFAST) and Vet BLUE (bedside lung ultrasound exam) techniques that have evolved to the FAST3 techniques—focused assessment with sonography in triage, tracking and trauma.

Why is it important?

Why all the fuss with name changes and the fancy superscript 3? As clinicians, we can't just "set it and forget it" in medicine. We need not only rapid initial triage and assessment, but also constant reassessment and tracking of our patients. We understand that the hypotensive, shocky patient may not have a definitive haemoabdomen on presentation. Rather it may take some time after fluid resuscitation for haemorrhage to present within a body cavity.

Focusing on the AFAST³ technique, why has this

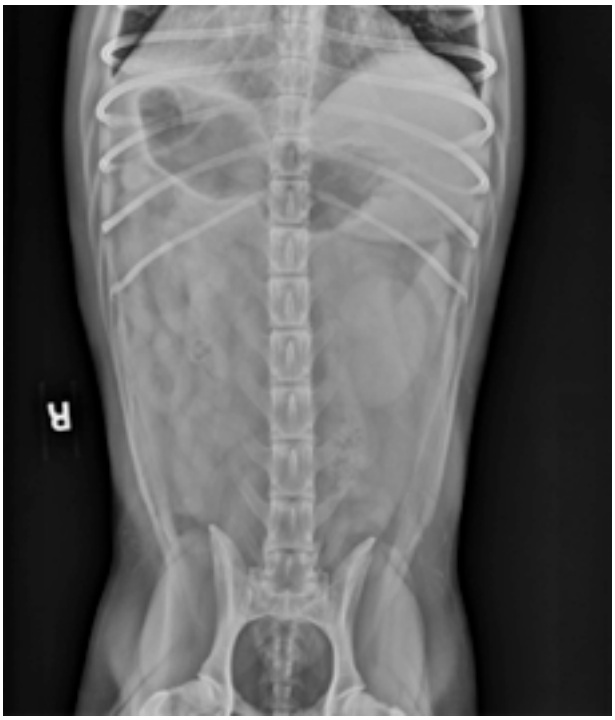

Figure 1: A radiograph from a 8.5-year-old spayed female English springer spaniel. Note the mild gas and fluid dilation of the stomach. The small bowel appears empty to mildly thickened or mildly fluid dilated. There is moderate remodeling of both hips with thickened femoral necks. Diagnostic impressions: Increased fluid in the stomach may be due to gastritis or pancreatitis. There is no evidence of foreign material or obstruction. Incidental moderate chronic hip arthritis is present.

become so important in both human and veterinary medicine? There are clear limitations and concerns when moving a critically ill patient to radiology where it needs to be taped, sand-bagged or manually restrained to obtain diagnostic imaging results.

Human and veterinary studies have demonstrated that radiographic serosal detail is not sensitive or specific at detecting abdominal fluid after blunt trauma (Figure 1).[1,2]

Ultrasonography is thought to be a more ideal initial imaging modality as it can be performed simultaneously while other assessments are being performed and while resuscitative measures are provided. Importantly, there is no requirement to be a fancy board-certified radiologist to perform the ultrasound procedure. Non-radiologist veterinarians can be quickly trained to performed the AFAST[3] ultrasound to make life-saving clinical decisions.

How does it work?

Now that I have your attention and before you head to eBay for an ultrasound machine, let's discuss how to perform the AFAST3. The patient is placed in right

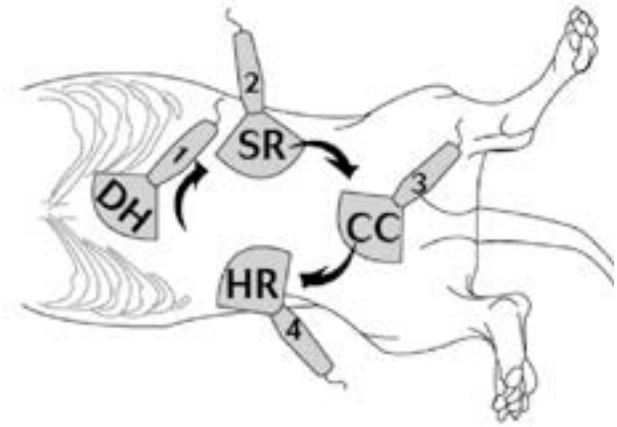

Figure 2: The four areas evaluated on the FAST technique: diaphragmatic-hepatic (DH), spleno-renal (SR), cysto-colic (CC) and hepato-renal (HR).

lateral recumbency to evaluate four specific sites within the abdomen (Figure 2):

1. Diaphragmatic-hepatic (DH)
2. Spleno-renal (SR)
3. Cysto-colic (CC)
4. Hepato-renal (HR)

The sites are evaluated in a clockwise motion as you look for evidence of effusion, which is typically identified as anechoic (black) areas around the identified organ structures (Figure 3). Remember, this is a quick and dirty evaluation. Shaving the patient is not required. A little alcohol or ultrasound gel is sufficient, although it is important to remember to avoid alcohol if the patient is critical and may require defibrillation (or has injuries including abrasions or lacerations).

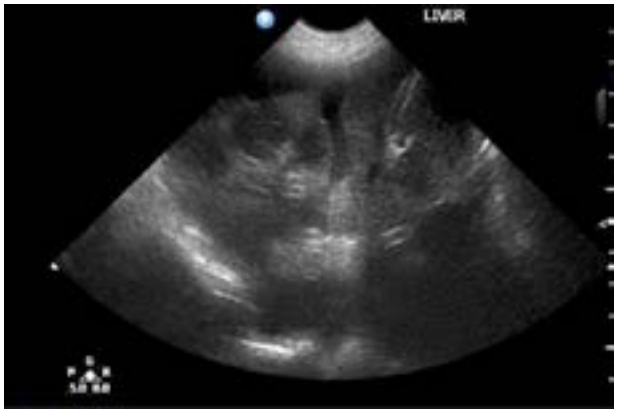

Figure 3: An ultrasonogram from a 10-year-old spayed female American bulldog. Multiple hypoechoic masses are seen throughout the liver measuring 1 to 4 cm. Mild effusion is present, seen here with an anechoic appearance.

Does it work?

I can see you are questioning these statements with the appropriate critical eye. Is this really better? Does this improve my patient evaluation? The answer—yes! In a prospective study of 100 dogs presenting for motor vehicle trauma, a FAST examination was found to have 96% sensitivity and 100% specificity for the detection of free abdominal fluid.[4]

Further studies were performed to determine the likelihood of the traumatic haemoperitoneum patient requiring a transfusion.[1] A fluid scoring system was described for the AFAST3 procedure, with one point given for haemorrhage at each of the four locations described above. An abdominal fluid score (AFS) of 0 means there is no effusion at any of the sites. An AFS of 4 means fluid was present at all four sites. Dogs with traumatic haemoperitoneum with an AFS of 1 or 2 are considered major-injury, small-volume bleeders and rarely become anaemic from the intra-abdominal haemorrhage. Alternatively, dogs that had an AFS of 3 or 4 are considered to be major-injury, large-volume bleeders and are more likely to become anaemic. In this study, about 25% of patients with AFS scores 3 or 4 developed anaemia severe enough to require transfusion therapy.

Serial examinations (tracking) cannot be overemphasized. It's standard of care to repeat FAST examinations four hours after admission in all stable cases to make certain an AFS of 1 or 2 is not developing into a 3 or 4. The take-home message: If a patient has an AFS of 3 or 4, be prepared with blood products, a blood donor or the possibility to transfer the patient to another hospital to provide transfusion therapy.

Are you ready for ultrasound?

Yes! If you are intimidated by technology and ultrasonography, there are numerous courses available for practicing veterinarians, not only as wet and dry labs at conferences but as online courses such as fastvet.com and textbooks with images and protocols.[5]

Although ultrasonography is not 100% sensitive for intra-abdominal haemorrhage, it is practically perfect for recognizing intra-abdominal haemorrhage after trauma. An ultrasonographic examination provides a rapid assessment, which may not only improve patient care, but can be life-saving.

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6 Mistakes to Avoid in the Veterinary ER

And if you can't remember any of them in the heat of the moment, focus on this: perfusion, perfusion, perfusion.

By Justine Lee, DVM; DACVECC; DABT; CEO, VETgirl

Take a deep breath, doc. You got this. Does the idea of a dog presenting with pale mucous membranes, a weak pulse and a heart rate of 190 beats/min make your knees weak? Do you get tachypnoeic when you see a dyspnoeic cat fish-mouth breathing in front of you?

If you don't see emergency cases every day, you're in the right place—this article discusses how to avoid common errors in emergency patients and save your patients' lives. Having practiced everywhere from a busy inner-city emergency room to the ivory tower of academia, I've seen these mistakes made and I've made them myself. Now you don't have to — and your patients (or at least their owners) will thank you.

Here's what to avoid.

1. Not doing chest radiographs

I can't tell you how many times I've had a case referred to the emergency clinic for abdominal ultrasound or postoperative supportive care, only to find a chest full of metastatic lesions. Chest radiographs need to be part of routine geriatric diagnostics.

Dogs older than 6 or 7 years of age and cats older than 12 with, for example, hepatosplenomegaly, icterus, haemoabdomen, immune-mediated disease or fever of unknown origin should have chest radiographs done at the same time as abdominal radiographs. Typically, a three-view chest set is the method of choice, but a right- and left-lateral chest radiograph is also an effective way to screen for metastasis.

While a met check is a "low-yield test" (the likelihood of identifying chest metastasis is relatively low), it's an important screening tool that can help you counsel pet owners on end-of-life decision-making and overall prognosis. However, keep in mind that doing chest radiographs may be too stressful for dyspnoeic cats—stabilise them first

2. Using the shock dose of fluids

The "shock dose" of fluids is extrapolated from a patient's blood volume (60-90 ml/kg for dogs; 60 ml/kg for cats). But recently, emergency and critical care specialists have moved away from using the entire shock dose when trying to stabilise hypovolemic patients—smaller aliquots (one-quarter to one-third of a shock dose) of intravenous (IV) crystalloid fluids are preferred. Better to give small amounts frequently while monitoring!

3. Using the wrong dose of corticosteroids

Traditionally, emergency books have included "shock doses" of corticosteroids—for example, dexamethasone sodium phosphate (DexSP) 4-6 mg/kg. However, criticalists have moved away from giving corticosteroids with trauma because of potential deleterious effects, including gastric ulceration in a poorly perfused "shock gut" in the dog, exacerbation of hyperglycemia, and delayed wound healing.

Recently we have moved to administering different doses of DexSP. An anti-inflammatory dose of DexSP is generally considered 0.1 mg/kg, whereas immunosuppressive doses are as low as 0.25 mg/kg IV every 12 to 24 hours. For that reason, the 4-6 mg/kg dose for shock is no longer indicated. Remember that DexSP is approximately eight to 15 times stronger than prednisone.

Perfusion, perfusion, perfusion

In the first six hours of a critical care case, I focus on perfusion—and avoid corticosteroids and NSAIDs.
The definition of shock is considered cellular hypoxia—the patient's cells are starving for oxygen. Do corticosteroids increase oxygen to cells? No; IV fluids do (with the exception of cardiogenic shock!). The reason I've moved away from using NSAIDs immediately in the shocky patient is that the dog's "shock organ" is the gastrointestinal tract. When an

animal is in shock, it's vasoconstricting to shunt blood to its most important organs—the heart and lungs. If the gut and kidneys aren't getting appropriate blood flow during a shocky state, we ideally want to avoid NSAIDs or steroids until the patient is more stable. When in doubt, perfuse the patient first.

Again, it's not corticosteroids or NSAIDs that save these patients. It's perfusion, perfusion, perfusion.

4. Giving corticosteroids to head trauma patients

In the patient with head trauma, we ideally want to avoid the use of corticosteroids due to the potential for hyperglycaemia. Recent studies have shown that human patients with head trauma and hyperglycaemia have a poorer return to cognitive function than do euglycaemic patients. Why is hyperglycaemia dangerous in these cases? Because elevated glucose concentrations provide a substrate for anaerobic metabolism and glycolysis in the brain. Hyperglycaemia is also associated with proconvulsant effects due to increased neuronal excitability.

Instead of reaching for corticosteroids in the head trauma patient, consider these treatments instead:

- Osmotic agents such as mannitol, which have been found helpful in decreasing intracranial pressure (ICP)
- IV fluid resuscitation to help normalize or maintain blood pressure and maximize perfusion
- Oxygen therapy
- Elevation of the head 15 to 30 degrees (to lower ICP) (*Elevate without kinking the neck as kinking will actually increase ICP. Ed)*
- Minimal jugular restraint or pressure (to prevent increased ICP) *(Preferably use cephalic veins for IV access and blood collection - Ed)*
- Tight glycemic control

5. Not doing FAST ultrasounds

The focused assessment with sonography for trauma (FAST) ultrasound is a two-minute procedure that detects the presence of fluid in the abdominal cavity to allow for rapid therapeutic intervention, such as fluid resuscitation, abdominocentesis, cytology or clinicopathologic testing.[1] This quick ultrasound method to determine "yes fluid/no fluid" is designed to be used by healthcare professionals with limited ultrasonographic training and is not designed for extensive examination of the abdomen.

One of the benefits of the FAST examination is its ability to detect very small amounts of fluid. Typically, 5 to 25 ml/kg of fluid needs to be present to be removed by blind abdominocentesis; 10 to 20 ml/kg of fluid has to be present before it can be detected by fluid-wave assessment on physical examination; and approximately 9 ml/kg of fluid needs to be present before it can be detected radiographically. But as little as 2 ml/kg of fluid can be detected on a FAST examination, allowing for rapid diagnosis and identification of underlying pathology.

The FAST examination typically involves assessment of four sites of the abdomen: caudal to the xiphoid, cranial to the bladder, and the right- and left-dependent flank.[1] The presence of fluid at any of these sites is considered positive. Evaluation of the xiphoid region allows you to check for fluid between the liver and diaphragm and the liver lobes, as well as for pericardial or pleural effusion.[1] The bladder view evaluates for fluid cranial to the bladder and for the presence of a bladder.[1] The right-dependent flank allows for fluid detection between the intestines and the body wall, whereas the left-dependent flank view allows for identification of the spleen and any abdominal effusion near the spleen and body wall, the kidney and spleen, and the liver and spleen.[1]

6. Reluctance to penetrate body cavities

I believe every veterinarian should be comfortable doing an abdominocentesis and a thoracocentesis. These benign procedures are both diagnostic and therapeutic.

Moving quickly but maintaining aseptic protocol, shave and surgically prep a wide area near the umbilicus (for abdominocentesis) or thorax (for thoracocentesis). Thoracocentesis should be performed either dorsally (for air) or ventrally (for effusion) at the seventh to ninth intercostal space (ICS). An imaginary line can be drawn from the end of the xiphoid to the lateral body wall, which is approximately the eighth ICS. This allows for rapid identification of where to perform an emergency thoracocentesis. For an abdominocentesis, a four-quadrant tap should be aseptically performed at the periumbilical region.

ABDOMINOCENTESIS TECHNIQUE

An abdominocentesis should be performed using sterile technique. The abdomen should be clipped, shaved and prepared aseptically. A four quadrant tap around the umbilicus should be performed. If you obtain fluid from one location, there is no reason to tap the other remaining 3 regions. Gently, but briskly, insert a sterile 22-ga. needle into these locations. One can use either a closed technique (e.g., needle attached to 3 ml syringe) or open technique (e.g., needle alone).

If no fluid is obtained, a "2-tap technique" can be used; a second needle can be inserted several millimeters away from the first sterile needle insertion - this will often allow fluid to gently flow out. Once fluid is seen, gentle suction can be used with an attached 1-, 3- or 6-ml syringe to aspirate the ascites. *Editor*

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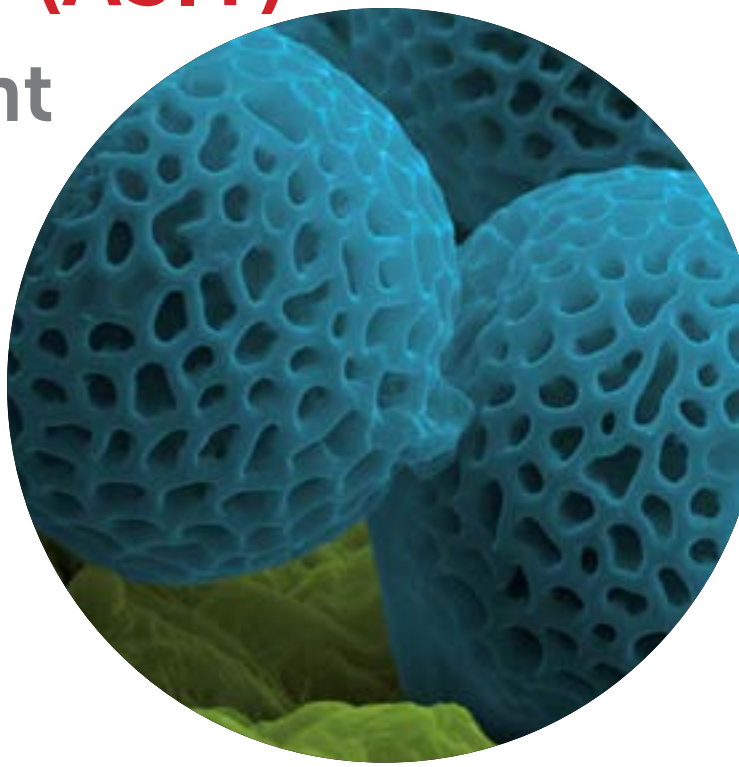
The Use of Allergy Serology and Allergen Specific Immunotherapy (ASIT)

in the Management of Canine Atopic Dermatitis



Dr Rick Last (BVSc; M.Med.Vet (Path); MRCVS)
Specialist Veterinary Pathologist

At the outset it is important to remember that allergy serology is not a diagnostic test used for the confirmation of a diagnosis of canine atopic dermatitis (CAD). Diagnosis of atopy is based on meeting specific historical and clinical criteria (Favrot's criteria) and ruling out other possible causes of similar dermatological and clinical signs. Strict control of ectoparasites, exclusion of bacterial and/or fungal dermatitis and ruling out cutaneous lymphoma, running of elimination diet trials and/or performing the food reaction test (FRT) forms part of the clinical workup of any atopic patient.

In an animal with these historical and clinical criteria, the presence of allergen specific IgE is considered highly significant. Only once the clinical diagnosis of canine atopic dermatitis has been made, is allergy serology considered and then only as a test to identify potential triggering allergens to include in an allergen specific immunotherapy vaccine.
The first detailed description on the use of allergen specific immunotherapy in dogs, was described by Wittich in 1941. He demonstrated allergic sensitization to ragweed pollen and response to allergen specific immunotherapy (ASIT). So ASIT has been known as a therapeutic tool for canine atopic dermatitis for over 75 years and has become an important and foundational treatment for atopic dermatitis in dogs, cats and horses.

There are a growing number of studies that have documented the effectiveness of ASIT in allergic disease in animals. It remains currently the only therapy that can modify, or reverse part of the pathogenesis of CAD, both alleviating clinical signs and preventing progression of the condition. In addition, ASIT has minimal adverse effects with lifelong treatment and provides the possibility for long-lasting effectiveness.

The International Committee for Allergic Diseases of Animals co-ordinates and reviews scientific and clinical research into the following areas of atopic dermatitis

- Pathogenesis.

- Clinical Diagnosis.
- Allergy testing.
- Allergen Specific Immunotherapy.
- Evidence based treatment guidelines.

This highlights the scientific and clinical importance of allergy testing and ASIT in the effective management of canine atopic dermatitis.

Allergy Testing

Atopic dermatitis is a common diagnosis in veterinary dermatology affecting as much as 10% of the canine population. A key factor in the pathogenesis of the clinical manifestations of atopy is the presence of high levels of allergen specific IgE. However, it should be appreciated that canine atopic dermatitis is a complex and multifactorial disease involving immune dysregulation, allergic sensitization, skin barrier defects, microbial colonization and environmental factors.

Only once the clinical diagnosis of canine atopic dermatitis has been made, is it decided whether an allergy test is required or not. Allergy serology should not be used as a front line diagnostic test for atopy, the clinical diagnosis of atopic dermatitis should have been made prior to testing.

The following situations would warrant that allergy testing (allergy serology or intradermal skin testing) be performed.

- Severe clinical signs.
- Prolonged clinical signs that last for more than 3 months of the year.
- Poor control of atopic skin disease with symptomatic therapy.
- Side effects to drugs being used in the therapy program.
- Poor owner compliance.

The results of the allergy test are used to identify offending allergens to enable formulation of allergen specific immunotherapy vaccines. Allergy serology has several advantages over intradermal skin testing including no patient risk (as no sedation or general anaesthetic required), it is more convenient (for owner and dog), does not require repeated injections, serology is objective and reproducible and there is lower risk of drug interactions interfering with test results.

Evidence based studies have provided no evidence of drug withdrawal prior to allergen-specific IgE serological tests for oral cyclosporine or prednisone / prednisolone. For intra-dermal skin testing on the other hand optimal withdrawal times are antihistamines (7 days), oral glucocorticoids (14 days), topical/ otic glucocorticoids (14 days) and cyclosporine (0) days. It has been shown that the success rate of allergen specific immunotherapy (ASIT) based on allergy serology versus intradermal skin testing is not statistically different.

Various allergen specific IgE serology testing assays are available including monoclonal, mixed monoclonal and polyclonal anti-canine IgE assays plus the high affinity IgE receptor alpha subunit assay (Fc-epsilon receptor test/mast cell receptor test). Seum IgE levels are miniscule when compared to IgG, in fact for every IgE antibody there are more than 10 000 IgG antibodies.

It should be appreciated that IgG also binds with allergens and therefore, there is huge opportunity for cross reaction. Hence, serological assays based on the use of monoclonal or polyclonal anti-IgE antibodies are complicated by cross reaction with IgG producing false positive results.

To specifically identify only IgE in serum, one needs to make use of IgE's unique affinity for binding to mast cells and basophils, no other class of immunoglobulin is able to perform this.

This Fc-epsilon receptor test (mast cell receptor test), shows a strong and highly specific affinity for canine IgE and a complete lack of cross reaction with IgG. This system is specific for the detection of IgE only, it will not identify any other Ig class. Due to this higher sensitivity and specificity plus the absence of IgG cross reaction of the Fc-epsilon receptor test, the use of the monoclonal, mixed monoclonal and polyclonal anti-canine IgE assays have decreased. The complete absence of any IgG cross-reaction in the Fc-epsilon receptor test enables this test to detect IgE in serum down to extremely low concentrations, making it ideally suited for the purpose of allergen selection in an immunotherapy vaccine.

Allergy serology only measures circulating allergen specific IgE, it does not measure other allergic pathways and positive reactions are documented in non-allergic dogs. Thus the use of allergy serology should be restricted to only patients with confirmed clinical atopic dermatitis.

Quantitative assessment of serum IgE levels can only be achieved with the IgE specific Fc-epsilon receptor test. A test is considered positive if it is above a certain cut-off level. These positive results are then correlated to

- History of exposure of the patient to the allergen in question.
- Cross reactivity of allergens within botanical groups of related weed, tree or grass pollens.
- Level of IgE if more than 12 allergens are positive.

There is still a lack of standardisation of the currently

employed allergy tests (allergy serology, intradermal skin testing) and it is suspected that false negative and false positive results do occur. Allergy testing is also unable to detect dogs with atopic like dermatitis, which is a condition clinically identical to canine atopy, but in which an IgE response to environmental or other allergens cannot be demonstrated.

Allergen prescription formulation

Selection of allergens for inclusion in an allergen specific immunotherapy vaccine involves interpretation of the medical records of the particular dog, especially as regards seasonality and animal's habitat (i.e. presence of specific allergens in the animals environment) in conjunction with IgE levels of individual allergens achieved with allergy serology.

- Non-seasonal – environmental allergens, indoor moulds.
- Seasonal – spring (trees/ectoparasites), summer (grasses/outdoor moulds/ectoparasites), autumn (weeds/outdoor moulds)
- Seasonally non-seasonal – seasonal and non-seasonal allergens involved concurrently. (Seasonal atopy complicating adverse food reaction/Ectoparasites complicating non-seasonal atopy).

Mould extracts should not be mixed in the same vials as pollen extracts, as the pollen allergens will be degraded by the mould proteases during storage. Therefore, mould extracts should be in a separate vial and administered as a separate injection.

Allergen Specific Immunotherapy (ASIT)

Although numerous treatments exist for atopic dermatitis, many have significant side effects and drawbacks and not all are universally effective. Allergen specific immunotherapy (ASIT) is the only proven treatment for atopic dermatitis that works through reversing the underlying immunopathogenesis of the disease with the added advantages of being virtually free of serious adverse effects, even with prolonged use, offering substantial, long-lasting relief in many patients.

ASIT vaccines are available in two forms namely an injectable form given by subcutaneous injection every 2-4 weeks (SCIT) and a sub-lingual immunotherapy (SLIT) vaccine, which is applied orally under the tongue on a daily basis.

ASIT has emerged as an important and useful tool in the long-term management of skin disease in atopic patients. Allergen avoidance, prevention of allergen contact, antimicrobial therapy, pharmacotherapy and/immunotherapy are crucial in the therapeutic management of atopic patients. Pharmacotherapy is frequently needed when a rapid and short-term response is required or when allergen avoidance is difficult or impossible to implement, while ASIT is utilised as a long-term therapy that reduces or eliminates the need for pharmacotherapy.

Mechanisms of action of ASIT demonstrated in humans include early reduction in effective cell (eosinophils, basophils, mast cells) activity, followed by a long-term immunological shift from a lymphoid T helper 2 (Th2) cell to a T helper 1 (Th1) cell response and the development of immunological tolerance. These changes are accompanied by increases in immune regulator cells and certain cytokines. This all results in an increase in allergen specific IgG (especially IgG4) which impairs the effector functions of IgE and with extended application initiates a decrease in allergen specific IgE.

In canine atopic patients a shift from Th2 to Th1 cell response, increases in immune regulator cells and certain cytokines plus increases in IgG levels, have all been demonstrated and suggests that mechanism of action in dogs is similar to that in humans.

Subcutaneous immunotherapy (SCIT) is available as two methods based on availability and regulatory approvals in North America versus Europe. SCIT vaccines produced in North America are aqueous, saline phenol preserved extracts. The protocol begins with frequent injections of dilute extract, progressing to less frequent injections of concentrated extract as a maintenance therapy. In Europe alum precipitated allergen extracts are utilised, with absorption of the allergen molecules to an aluminum hydroxide adjuvant, which provides a slower release product which has the advantage of less frequent injections.

Time to efficacy with SCIT varies from up to 8 months with aqueous allergens and 9 months with alum-precipitated allergens. Those dogs which have not responded by this time are unlikely to.

These sublingual immunotherapy (SLIT) vaccines have an excellent safety record due to the unique nature of antigen capture at this sublingual site. This oral immune site comprises various antigen presenting cells (Langerhans cells, myeloid and plasmacytoid dendritic cells) with a distinct location in the mucosa and sub-epithelial lamina propria. These dendritic cells are tolerogenic being key in the induction of immune tolerance, resulting in induction of peripheral (skin, respiratory tract) tolerance to allergens. The oral mucosa also contains limited numbers of mast cells and eosinophils mainly located in the deep submucosa explaining the good safety profile (lack of adverse reactions) of SLIT.

SLIT shows a far more rapid clinical response with some dogs showing significant improvement in 3 months, while many have substantial improvement by 6 months.

Modifications of the standard SCIT protocol are emerging and include rush immunotherapy and intra-lymphatic immunotherapy.

Rush immunotherapy has the advantage of limiting the number of injections that an owner must apply during the initiation phase of ASIT as maintenance levels are achieved rapidly. Dogs must be hospitalised and increasing doses of allergen extract or injected subcutaneously every 30 minutes for 7 hours. Animals are then discharged and continue on maintenance therapy. Therefore, with rush immunotherapy maintenance doses are achieved within one day compared with weeks or months with conventional immunotherapy.

Intra-lymphatic immunotherapy is a recent modification of ASIT vaccine application, is reported to be associated with fewer and less severe adverse reactions then encountered with SCIT and to be effective for several years after only 3 intra-lymphatic injections. Alum precipitated ASIT vaccines are administered monthly into 1 of the popliteal nodes (alternating sides with each subsequent injection), under ultrasound guidance over 3 to 5 months. The number of intra-lymphatic injections (4 to 6) is based on the clinical improvement of the individual dog. In most instances, no sedation is required. In various studies complete remission was documented in 13% to 24% of dogs.

Trouble shooting allergen specific immunotherapy (ASIT).

Subcutaneous immunotherapy (SCIT)

- Be alert to anaphylactic reactions, most likely during the initial loading phase.
- Monitor for increase in pruritis and flare ups of otitis or pyoderma.
- If pruritis initially decreases after each injection but then slowly increases prior to the next injection, the interval between injections should be decreased.
- If pruritis initially increases after each injection, followed by improvement prior to the next injection, the allergen dose is too high. Decrease the dose by 25%. If pruritis still spikes after injection, reduce dose by a further 25%.

Sublingual immunotherapy (SLIT)

- A few dogs rub or scratch at their mouth following application and individual dogs will vomit. However, these effects are short lived and usually disappear after a few applications.
- If signs persist or worsen, lowering of the allergen dose may be required.
- Monitor for increase in pruritis and flare ups of otitis or pyoderma.

Some highly effective drugs such as cyclosporin (Atopico®, Elanco) and oclacitinib (Apoquel®, Zoetis) and biologicals (anti-IL-31 therapeutic monoclonal antibody), control clinical signs over a long period of time and may obviate some of the "need" for ASIT. However, these drugs and biologicals still require lifelong treatment and they only reduce clinical signs rather than reversing the pathogenesis of the condition as observed with ASIT. Long-term safety of these agents is not always known, and they carry no hope of permanent cure that can **sometimes** be achieved with ASIT. Use of these drugs and biologicals in conjunction with ASIT provides a template effective disease control in many instances.

Historically, allergen specific immunotherapy has been viewed in general clinical practice as a last resort treatment option. With the growing knowledge of how and when to use ASIT, it has taken its place as a foundation treatment for the long-term management of canine atopic dermatitis.

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Salivation Abnormalities of Neurogenic Origin

Max presented with unilateral temporalis muscle atrophy but also with changes to his saliva. Let's investigate what's behind it.

By Rachel B. Song, VMD, MS, DACVIM (Neurology), Eric N. Glass, MS, DVM, DACVIM (Neurology), Marc Kent, DVM, DACVIM

It's Monday morning. You walk into the office, back from a long weekend. You run through the list of the day's appointments and see that one of your favorite patients, Max, is on the day's schedule with the chief complaint being that the "head looks funny. "Later that day, you enter the exam room and ask, "What's going on with Max today?"

His owner replies, "Well, doc, the left side of his head completely sunk in overnight!"

You take one look at Max's head and your heart sinks—you quickly recognize that the left temporalis and masseter muscles have atrophied completely. Without the muscles, the left zygomatic arch is clearly visible to the naked eye. The left eye also has become enophthalmic with an accumulation of mucoid discharge and third eyelid elevation (Figure 1). Additionally, enophthalmos of the left eye and secondary third eyelid elevation have resulted from atrophy of the pterygoid muscles. (All photos courtesy of Drs. Rachel B. Song, Eric N. Glass and Marc Kent).

When you turn Max's head to examine the right side, you see how well-muscled his head is on the unaffected side. He's still able to close his eyelids when you perform the palpebral reflex and the menace response. You remember that to close the eyelids, cranial nerve VII (the facial nerve) has to be functioning normally. Likewise, all the other cranial nerves appear to have normal function on your examination. When you do an oral exam on Max, you see an accumulation of thick, foamy saliva in the

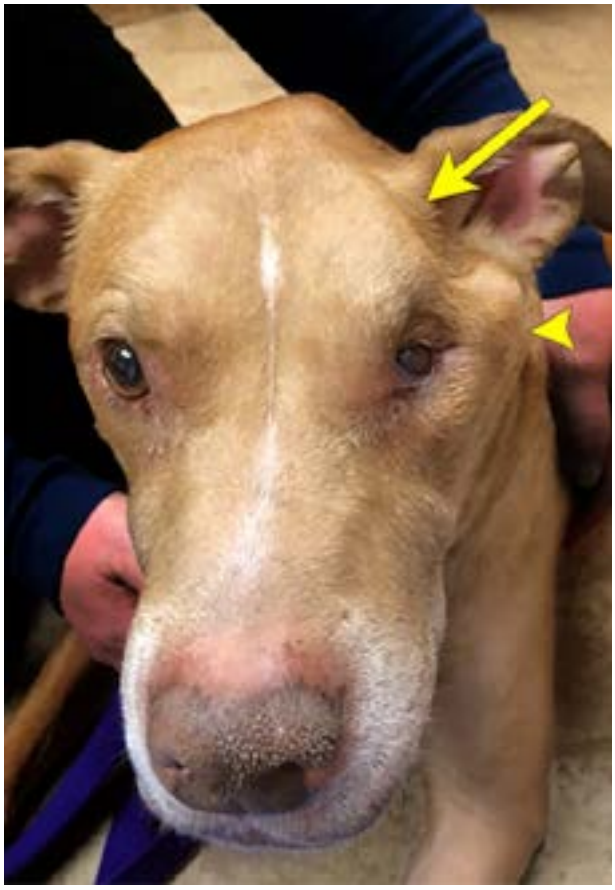

Figure 1: Max has unilateral dysfunction of the mandibular branch of cranial nerve V (trigeminal). Atrophy of the temporalis muscle (large arrow) is evident. Loss of the masseter and temporalis muscles makes the zygomatic arch more clearly defined (arrowhead).

caudal oropharynx only on the side of the muscle atrophy. You are puzzled and take a second and more thorough look to confirm your findings. Why is there foamy saliva in the back of his mouth, you wonder?

Thinking through the causes of muscle atrophy—disuse versus neurogenic atrophy—you think that it must be neurogenic atrophy, given how quickly the owner reported the onset of the atrophy. But then which nerve? Ah, but of course! You remember your vet school neurology training and recall that cranial nerve V, the trigeminal nerve, innervates the muscles of mastication. All those mnemonics you memorized to learn the cranial nerves and their functions are paying off. But then how to account for the abnormal saliva accumulation in Max's oropharynx?

A quick trigeminal nerve review

The trigeminal nerve has three main branches—the ophthalmic, maxillary and mandibular nerves. All three branches relay sensory information from the head and face to the brain.[1] Only the mandibular branch is responsible for providing motor function to the muscles of mastication—the masseter, temporalis, lateral and medial pterygoids, rostral portion of the digastricus, and mylohyoideus muscles—as well as some lesser-known muscles such as the tensor tympani muscle (involved in modulating the ossicles in the middle ear) and the tensor veli palatani muscle (involved in opening the pharyngeal orifice of the auditory tube).

Clinically, unilateral dysfunction of the mandibular branch of the trigeminal nerve is easy to recognize, often because of the dramatic muscle atrophy of the masseter and temporalis muscles. Pterygoid muscle atrophy is inferred by the enophthalmos, as the pterygoid muscles provide ventral and medial support of the eye's position in the orbit. The third eyelid will elevate passively with the enophthalmia.

The salivary link

What probably was not a focus in your veterinary curriculum was the role that the trigeminal nerve serves in the autonomic nervous system. Branches of the trigeminal nerve act as a conduit for the distribution of parasympathetic innervation to target organs such as the lacrimal and salivary glands.

Anyone who has suffered from dry eye (keratoconjunctivitis sicca, or KCS), dry mouth (xerostomia) or dry nose (xeromycteria) can understand the life-altering effects such conditions can cause. Since veterinary patients can't self-report symptoms, most owners don't recognize altered autonomic function until severe end-stage consequences are present. To appreciate this, one only need examine a dog with untreated KCS. The lack of tear production leads to corneal opacification from corneal oedema, neovascularisation, pigment deposits, squamous metaplasia and hyperkeratinisation of the cornea, which results in visual deficits.[2] People with xerostomia suffer from severe oral discomfort, dental caries, speech problems and difficulty eating and swallowing.[3]

In dogs, there are four major salivary glands—the parotid, zygomatic, sublingual and submandibular glands.[4] Although structural diseases of the salivary glands (i.e. sialocoele, sialadenitis, salivary gland neoplasms and sialadenosis) are well-known in veterinary medicine, pure functional disturbances of the salivary glands have yet to be well-described.

Quite detailed Neurological Anatomy. Important points in bold for ease of reading...ed

Each of the salivary glands is innervated by the sympathetic and parasympathetic nervous system. As a whole, the autonomic nervous system has both afferent (sensory) and efferent (motor) components. **Efferents are composed of a two-neuron system—preganglionic and ganglionic neurons.** Sometimes these are referred to as first-order and second-order neurons, respectively. **The preganglionic neuron is located in the central nervous system. Preganglionic axons synapse with the ganglionic neurons in various ganglia. Postganglionic axons innervate target organs** such as glands, the heart, the lungs and many other organs.

For sympathetic innervation, preganglionic neurons are located in the intermediate gray matter of the thoracic and cranial lumbar spinal cord. For most of the body, **the ganglionic neurons are located in the paired sympathetic trunk, which runs bilaterally along the ventrolateral aspect of the vertebral column. For structures of the head, ganglionic neurons are located in the cranial cervical ganglia** near the ventral and caudal aspect of the skull. **Long postganglionic axons course to their target organs. Sympathetic postganglionic axons use norepinephrine as their neurotransmitter.**

For parasympathetic innervation, preganglionic neurons are located in the brain stem, adjacent to the motor neurons for cranial nerves III (oculomotor), VII (facial), XI (glossopharyngeal) and X (vagus), as well as **sacral spinal cord segments. The ganglionic neurons are often located in ganglia close to their intended target organs. Short parasympathetic postganglionic axons course to their target organs and use acetylcholine as their neurotransmitter**.

For the salivary glands, cranial nerves VII (facial) and IX (glossopharyngeal) provide for parasympathetic innervation.[5] Specifically, preganglionic neurons for the parasympathetic innervation of the zygomatic and parotid glands are provided by the parasympathetic nuclei of the glossopharyngeal

nerve in the medulla.[5] Preganglionic axons travel in the glossopharyngeal nerve and synapse at the otic ganglion.[5] The postganglionic axons course with the auriculotemporal nerve, a branch of the mandibular nerve, to arrive at the zygomatic and parotid salivary glands.[5] Preganglionic neurons for the parasympathetic innervation of the sublingual and mandibular glands are provided by the parasympathetic nuclei of the facial nerve in the medulla.[5] The preganglionic axons course in the facial nerve, through the tympanic cavity to join the lingual nerve, a branch of the mandibular nerve, at the level of the oval foramen. The oval foramen, located just medial to the temporomandibular joint, is also the foramen through which the mandibular nerve exits the cranial cavity. After the preganglionic axons join the mandibular nerve, they course to the mandibular and sublingual ganglia and synapse with parasympathetic ganglionic neurons. Parasympathetic postganglionic axon coursing with branches of the mandibular nerve ultimately innervate the mandibular and sublingual glands.[5]

Parasympathetic stimulation of the salivary glands mediated through acetylcholine is responsible for salivary production and flow. The sympathetic nervous system can modulate salivary secretion and composition through norepinephrine by stimulating the blood vessels and the acinar cells of the salivary glands.[6] In general, sympathetic stimulation to the salivary glands results in vasoconstriction of the blood vessels, which reduces aqueous saliva and exocytosis of the acinar glandular cells. Overall, this results in a more proteinaceous and less voluminous saliva flow.[6]

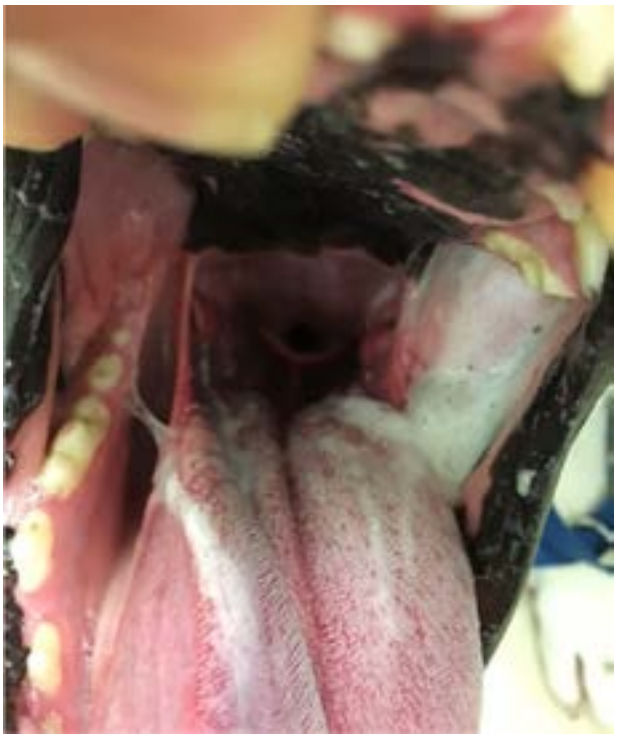

Figure 2; View of Max's oral cavity reveals the accumulation of thick, foamy saliva in the left caudal oropharynx.

An analysis of Max's salivation

In patients like Max with dysfunction of the trigeminal or the mandibular nerve, abnormal saliva that appears foamy, ropy and stringy accumulates in the oropharynx on the side ipsilateral to the nerve deficit (Figure 2). The saliva buildup is located just caudal to the maxillary fourth premolar, which is where the openings to the oral cavity are for the parotid gland and zygomatic gland.[7].

How can one explain the accumulation of abnormal saliva?

Given the normal function of cranial nerve VII (ability to blink the eye) and cranial nerve IX (ability to swallow normally), the preganglionic parasympathetic axons to the salivary glands are likely intact. Therefore, the abnormal composition and flow of saliva produced by the parotid and zygomatic salivary glands, as evidenced by accumulation of the thickened saliva in the caudal oropharynx, is most likely due to dysfunction of the postganglionic axons that course with the branches of mandibular nerve.

In cases like Max's, we hypothesize that the dysfunction of the trigeminal or mandibular nerve also affects the postganglionic parasympathetic axons that course alongside the axons of the trigeminal or mandibular nerve to reach the salivary glands. In other words, the loss of the conduit for the postganglionic axon provided by the trigeminal or mandibular nerve results in dysfunction of the parasympathetic stimulation to the salivary glands, which alters salivary flow and composition.

Normally, the parasympathetic nervous system is responsible for the production of voluminous amounts of aqueous saliva. Parasympathetic denervation results in proteinaceous saliva that is more viscous than normal. This is appreciated clinically as an accumulation of thickened, ropy saliva that accumulates in the oropharynx on the side ipsilateral to trigeminal or mandibular nerve dysfunction.

The essentiality of salivation, and why it might go wrong

Normal salivation provides an important role in day-to-day life. Not only does it aid in food mastication and digestion but also in lubrication of the oral cavity, maintenance of normal pH of the mouth, and prevention of dental caries.[8] Lack of salivation can cause significant oral discomfort and ulcerations as well as a detrimental decline in oral health.[9] It's possible that with unilateral trigeminal nerve dysfunction and accumulation of abnormal saliva, there may be an increase in dental tartar on the side of the nerve dysfunction.[7]

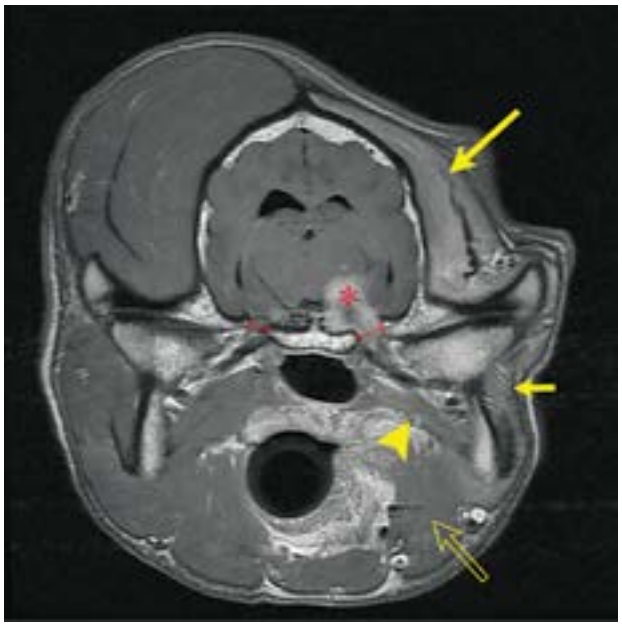

Figure 3. A transverse T1-weighted post-contrast MRI of Max's brain. A trigeminal nerve sheath neoplasm is compressing the brain stem (red asterisk) and is continuing along the mandibular branch as it exits via the oval foramen. Note the enlargement of the left oval foramen in comparison to the right. There is pronounced atrophy of the temporalis (large yellow arrow), masseter (small yellow arrow), digastricus (open yellow arrow) and pterygoid (yellow arrowhead)

A common cause of unilateral trigeminal nerve dysfunction is a nerve sheath neoplasm. Other causes of trigeminal nerve dysfunction include trauma, infectious or noninfectious neuritis, and other forms of neoplasia such as lymphoma. A complete physical and neurological examination may help narrow the differential diagnoses. Magnetic resonance imaging (MRI) of the head is the best way to establish a definitive diagnosis. With trigeminal nerve sheath neoplasms, MRI findings include an enlargement of the trigeminal nerve or its main branches, which display contrast enhancement, enlargement of the oval foramen from pressure necrosis from the expansile growth of the mandibular nerve, compression or invasion of the pons by the neoplasm, denervation atrophy of the masticatory muscles, and effusion in the tympanic cavity (middle ear) (Figure 3).

What to do next

Treatment options for nerve sheath neoplasms include palliative therapy with anti-inflammatory drugs or definitive therapy with radiation therapy.[10,11]

Radiation therapy can provide long-term control.[10] Given the potential for long-term survival after radiation therapy in dogs with trigeminal nerve sheath neoplasms, it's possible that the alteration in salivation may have a significant impact on the overall health and the quality of life of affected patients. It may be prudent to perform more frequent and thorough oral examinations to evaluate for pharyngeal function and salivary function in affected dogs. As the lack of autonomic functions to the head and face can lead to discomfort and secondary complications in our human patient counterparts, veterinarians should be aware of the potential for similar issues in dogs and cats.

Current accepted practices in dental care may provide a starting point for monitoring and planning dental procedures in affected patients.[12] In the future, more tailored evaluation protocols, preventive care measures and therapeutic interventions may be developed to minimize the negative impact abnormal autonomic function has on oral and overall health and to maintain an excellent quality of life in affected patients.

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Sialoadenosis has a major clinical impact and is often misdiagnosed as nausea. The following two articles aim to demystify the condition

Sialoadenosis and Limbic Epilepsy

Liesel van der Merwe BVSc MMed Vet (Med) Small Animals
Senior Lecturer, Outpatients OVAH
lieselvdmvet@gmail.com

Sialoadenosis is a bilateral, persistent, painless, soft, non-neoplastic, non-inflammatory swelling usually involving the parotid and sometimes also the mandibular salivary glands.

Salivary gland enlargement is an uncommon condition in dogs. In a review of 245 salivary gland biopsies from animals, salivary glands accounted for only 0.3% of all biopsies received. The majority of these biopsies (65%) were obtained from dogs and demonstrated neoplasia (30%), inflammation (29%), sialocoele formation (11%), infarction (9%), with the remainder showing miscellaneous changes. Hypersialosis or ptyalism is excessive secretion of saliva (true hypersialosis) or inability to contain saliva within the oral cavity, as may occur with dysphagia. Ptyalism also occurs secondary to both stimulation of salivary secretion caused by other disorders (e.g. dysphagia) and primary disease of the salivary gland (e.g. sialadenosis). Sialorrhoea, ptyalism or hypersialosis are synonyms used to describe the excessive production of saliva. Sialoadenosis causes clinical signs of retching, gulping, anorexia, nausea, vomiting and sialorrhoea with firm palpable, often enlarged salivary glands in dogs.

If endoscopic evaluation of the stomach and oesophagus reveals no lesions and there is no histological evidence of gastritis or oesophagitis, and thoracic imaging reveal no extraluminal oesophageal pathology, a diagnosis of idiopathic phenobarbitone-responsive hypersialosis can be made. The terrier breeds, especially fox terriers and Jack Russell terriers, are over-represented in the literature for both secondary and idiopathic sialorrhoea.

It is suggested that all forms of human sialoadenosis are caused by degenerative changes demyelinating polyneuropathy,in the autonomic nervous system. Salivary gland enlargement may be a secondary reactive change because of over-stimulation by the parasympathetic system. Three publications reported that surgical resection of the affected salivary glands, failed to stop the hypersalivation and dysphagia until phenobarbitone was administered to the patients. This finding supports the theory that the sialorrhoea is part of a multifactorial parasympathetic response to an underlying stimulus or condition.

It has been suggested that the idiopathic form of the disease may be a form of epilepsy, namely limbic epilepsy.The sialorrhoea and oral discomfort (odynophagia) exhibited by these patients responds well to phenobarbitone. There is no direct evidence linking phenobarbitone to saliva production but it does reduce small intestinal motility and could have local effects on the salivary glands The effectiveness of the phenobarbitone may indicate that the primary cause of the condition is parasympathetic activation of salivary gland secretion.

The exact mechanism of action of phenobarbitone is unknown, but is likely to involve inhibition of excitatory neurotransmitters, such as glutamate, and a gamma aminobutyric acid mimetic effect. Successful therapeutic intervention using anticonvulsants for sialoadenosis in dogs was first demonstrated in 1979 by Kelly *et al.* using phenytoin and primidone and subsequently, phenobarbitone was successfully used in several small case series. Most dogs improved within 48 h and all improved within 4 days. In the largest case series described, a starting oral dose of 1 mg/kg PO q12h was effective, although others have used higher doses (2–3 mg/ kg PO q12h), suitable for control of idiopathic epilepsy, with and without a loading dose. In one-quarter of the cases, tapering and cessation of phenobarbitone therapy was possible after a variable length of time (6–12 months until cessation).

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Sialoadenosis and Sialorrhoea in Canine Spirocecosis

Liesel van der Merwe BVSc MMed Vet (Med) Small Animals
Senior Lecturer, Outpatients OVAH
lieselvdmvet@gmail.com

The typical clinical signs of canine spirocercosis include regurgitation and/or vomiting and weight loss. Other frequently encountered clinical signs include coughing, dysphagia, pyrexia, melaena and sialorrhoea.

An article evaluating sialorrhoea in dogs with spirocercosis calculated the incidence at a conservative (some data was retrospective) 11%, 33 of 298 patients. The fox terrier and Jack Russell terrier breeds accounted for 36.4% (12/33) of the population of dogs with spirocercosis with sialorrhoea, compared with only 4/265 (1.5%) of the dogs with spirocercosis without sialorrhoea, a significant finding. Sialorrhoea was also more prevalent in smaller dogs with 69% of affected animals having ≤ 12 kg body weight. It is hypothesised that this may be caused by a relatively greater degree of oesophageal distension in these smaller animals caused by the parasitic nodule, as it has been demonstrated that the vago-oesophageal reflex can be stimulated by distension

The number of nodules was statistically different between the groups, with the sialorrhoea group having fewer nodules than the non-sialorrhoea group. This phenomenon cannot be explained but it is theorised that these patients show clinical signs earlier in the disease process because of the hyperresponsiveness of the oesophago-salivary reflex. The mandibular salivary glands were enlarged in 86.6% of cases and the parotid glands were involved in 13.3%.

An analysis of the clinical history of the sialorrhoea group revealed that the duration of clinical signs ranged from 4 days to 3 months and included regurgitation, intermittent to persistent retching, gulping with repeated swallowing movements, coughing, making efforts to 'clear the throat', apparent choking, excessive salivation and anorexia. Clinical signs worsened with excitement or palpation of the pharyngeal area and glands. The choking episodes could be quite severe with the dogs vocalising, scratching at their faces with their forepaws, contorting the forequarters, head and neck, and these symptoms were even on occasion misinterpreted by owners and referring veterinarians as seizures. Retching often terminated in vomition or regurgitation. Where mentioned, the vomitus was generally described as frothy saliva All patients had weight loss and many showed a very "tucked up" abdomen.

In the longstanding cases clinical signs generally progressed to severe dysphagia and a disinclination or a reluctance to eat. Food aversion was specifically noted in three cases. Laboured respiration was noted by owners in two cases and was detected on clinical examination as abdominal respiration in 25% of the dogs.

A variety of anti-emetics including metoclopramide, betaperamide and chlorpromazine had been used to no avail in the majority of the cases referred.

All patients with hypersialosis were treated with doramectin (Dectomax, Pfizer), phenobarbitone (Lethyl, Pharmacare Ltd. SA) and other symptomatic therapies as required. The treatment regimen of doramectin varied amongst clinicians but generally complied with the following: 400 mg/kg per os or by subcutaneous injection every week or every second week for a minimum of six treatments or until regression of the nodules occurred. The initiating dosage of phenobarbitone was generally around 2 mg/kg bid with a duration of 2–6 weeks as required. A marked decrease in clinical signs was generally noted within 48 hours of initiating phenobarbitone treatment at approximately 2mg/kg bid. Three cases took up to 2 weeks to respond and in these cases either an oesophagostomy tube or percutaneous endoscopically placed gastrostomy (PEG) tube was inserted

Patients with spirocercosis-induced dysphagia will, and do, show reduction of clinical signs with treatment of the underlying cause, but in some patients the degree of dysphagia precludes proper food intake and nutrition and the phenobarbitone therapy provides symptomatic relief whilst the nodule regresses.

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The ABCs of Veterinary Dentistry

'O' is for Oral Masses of the Malignant Kind

The histopathology report from the oral biopsy came back as malignant—now what? Surgery? Radiation? Chemotherapy? Watchful waiting?

By Jan Bellows, DVM, DAVDC, DABVP, FAVD

If caught before metastasis, many malignant oral masses can be removed, leaving the patient with little to no facial deformity and a functional bite. Here's an overview of the types of malignant oral masses you're likely to encounter, as well as what to do about them.

Common oral malignancies

Here are the most commonly diagnosed malignant oral tumours in dogs and cats:

Malignant melanoma.
Malignant melanoma is a locally aggressive tumour that's common in dogs and rare in cats and often metastasizes to the lungs (Figures 1A and 1B). Aggressive surgical excision, including partial mandibulectomy or maxillectomy, is the treatment of choice. Tumour sizes less than 2 cm carry a better prognosis than those greater than 4 cm.

However, radiation may have a role in effectively treating the primary tumor. In cases deemed nonresectable, or if an owner is unwilling to pursue surgery, radiation therapy has a high response rate (70% to 90%). Most protocols utilised are classified as hypofractionated in that they require only a few treatments, but each treatment has a high dose, or fraction. This involves three to four weekly fractions of 8 Gray or six weekly fractions of 6 Gray.

Side effects, including oral mucositis, are generally mild and self-limiting with hypofractionated protocols. Similar to surgery, patients treated with radiation therapy may achieve local control but generally succumb to metastatic disease. Survival times of seven to nine months are common.

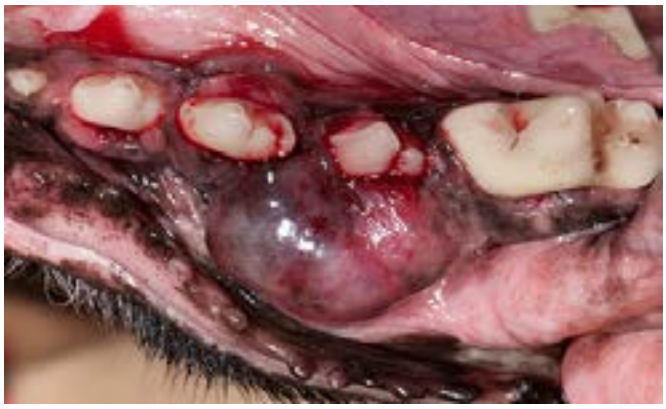

Figure 1A. A large oral mass involving left mandibular premolars and molar. (All images courtesy of Dr. Jan Bellows).

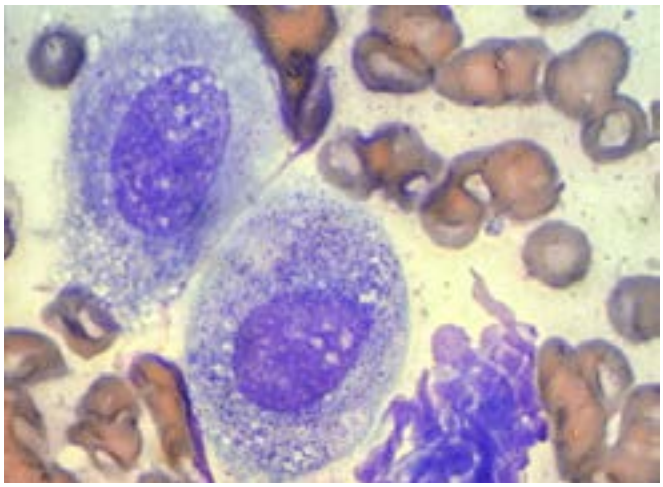

Figure 1B. Cytology of the oral mass in Figure 1A consistent with melanoma.

The Oncept Canine Melanoma Vaccine, DNA (Merial), is administered in stages II and III of canine oral melanoma and can decrease recurrence in some cases after removal or debulking of the primary tumor. The glycoprotein tyrosinase, which produces melanin found in melanoma cells, is the vaccine's targeted antigen. The vaccine appears to be effective in cases of amelanotic melanoma too.

In a 2016 study that retrospectively reviewed the outcome and survival of 32 dogs affected by oral melanoma that were treated with a combination of surgery and the xenogeneic DNA vaccination (with the addition of radiotherapy in some cases), the overall median survival time was 335 days, and the overall median progression-free survival (PFS) was 160 days.[1] Disease stage, surgical margin completeness and vaccine administration delay did not appear to statistically influence survival or PFS, though these results may reflect the study's low statistical power due to small numbers.

Squamous cell carcinoma. Like malignant melanoma, squamous cell carcinoma is a locally aggressive tumor (Figure 2). Oral squamous cell carcinoma in dogs rarely metastasizes unless it affects the tongue or tonsil, and when it does, abdominal ultrasound is indicated as part of staging because distant metastasis is more common to the liver and spleen than to the lungs.

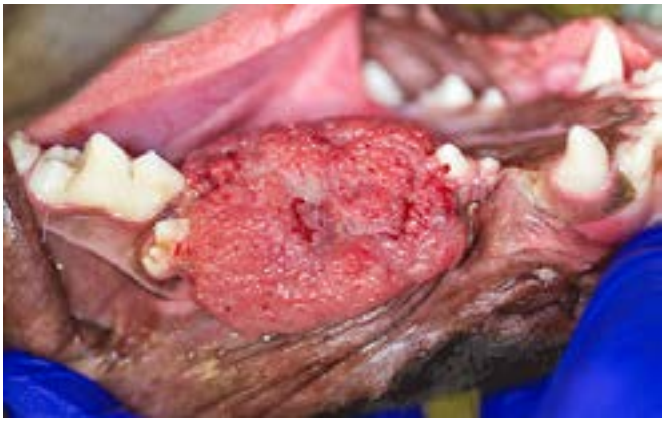

Figure 2. Papillary squamous cell carcinoma in a young dog.

Surgical excision is the primary treatment of choice for macroscopic oral squamous cell carcinoma in dogs, followed by radiation therapy for microscopic disease. Local recurrence is common. Piroxicam, a nonsteroidal anti-inflammatory (NSAID) agent given orally at a dosage of 0.3 mg/kg every other day, can be used for palliation with a median survival time of 180 days in dogs in one monotherapy study.[2] A slightly higher median survival time (272 days) was achieved when piroxicam was combined with cisplatin or carboplatin.[3]

A recent study of 87 dogs affected by nonmelanotic oral malignancies revealed that dogs undergoing postoperative radiotherapy after incomplete excision of oral squamous cell carcinomas had a significantly longer mean survival time (2,051 days) than dogs with incompletely excised tumors and no radiotherapy (181 days).[4]

Oral Malignancy Nomenclature

Adenocarcinoma: An invasive, malignant epithelial neoplasm derived from glandular tissue of either the oral cavity, nasal cavity or salivary tissue (major or accessory) with moderate metastatic potential.

Anaplastic neoplasm: A malignant neoplasm with cells that poorly resemble the normal histologic differentiation pattern; also called a poorly differentiated neoplasm.

Fibrosarcoma: An invasive, malignant mesenchymal neoplasm of fibroblasts with a low metastatic rate; a distinct histologically low-grade, biologically high-grade variant is often found in the oral cavity.

Haemangiosarcoma: A malignant neoplasm of vascular endothelial origin characterised by extensive metastasis; has been reported in the gingiva, tongue and hard palate.

Lymphosarcoma: A malignant neoplasm defined by a proliferation of lymphocytes within solid organs such as the lymph nodes, tonsils, bone marrow, liver and spleen; the disease may also occur in the eye, skin, nasal cavity, oral cavity and gastrointestinal tract; also known as lymphoma.

Malignant melanoma: An invasive, malignant neoplasm of melanocytes or melanocyte precursors that can be pigmented or amelanotic, with a marked tendency to metastasize.

Multilobular tumor of bone: A locally invasive and potentially malignant neoplasm of bone more commonly affecting the mandible, hard palate and flat bones of the cranium, with a multilobular histological pattern of bony or cartilaginous matrix, surrounded by a thin layer of spindle cells that gives it a near-pathognomonic popcorn-ball appearance on radiographs; also called multilobular osteochondrosarcoma, multilobular osteoma, multilobular chondroma, chondroma rodens and multilobular osteosarcoma.

Osteosarcoma: A locally aggressive, malignant mesenchymal neoplasm of primitive bone cells that have the ability to produce osteoid or immature bone with a high metastatic rate.

Squamous cell carcinoma: An invasive, malignant neoplasm of the oral epithelium with varying degrees of squamous differentiation; tonsillar squamous cell carcinoma has a higher metastatic rate and poorer prognosis than nontonsillar squamous cell carcinoma.

Squamous cell carcinoma is the most common oral tumor in cats and carries a grave prognosis if located caudally or if it affects the base of the tongue. If located rostrally, surgery is the treatment of choice when clean, wide (at least 2-cm) margins can be obtained (Figure 3).

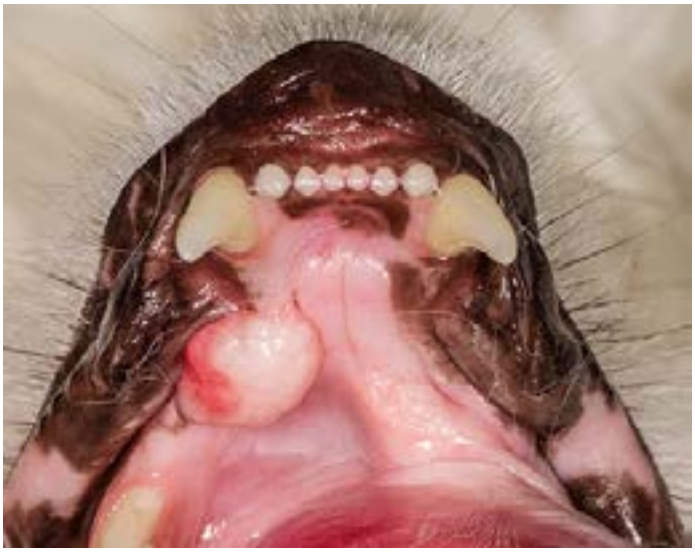

Figure 3. Squamous cell carcinoma in a cat's rostral mandible.

The NSAID meloxicam may have several beneficial effects in treating feline squamous cell carcinoma, including pain relief and reduced inflammation-associated neoplasia and oedema. In the United States, meloxicam is licensed only as a one-time injection for perioperative pain in cats. In Australia and Europe, low doses (0.01-0.03 mg/kg a day) have been used to treat osteoarthritis in cats without significant side effects.

Fibrosarcoma. Diagnosed most commonly in the maxillae of large-breed dogs, fibrosarcoma is locally invasive but infrequently metastasises to the lungs (Figure 4). It is the second most common oral malignancy in cats (after squamous cell carcinoma). A subset of fibrosarcomas has been recognized in which the tumors appear histologically low-grade yet behave as high-grade malignancies biologically (hi-lo).

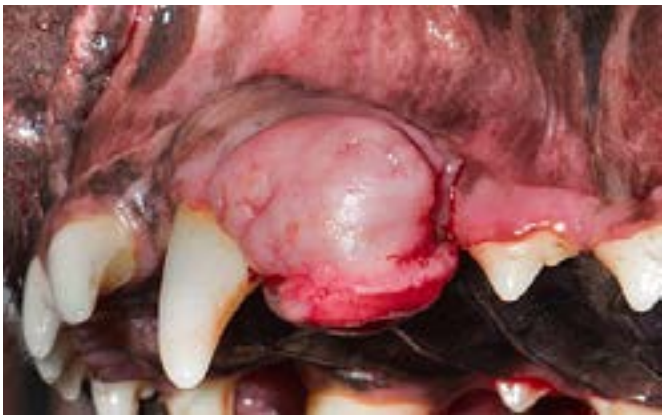

Figure 4. Fibrosarcoma affecting a dog's maxilla

As with squamous cell carcinoma, aggressive surgical excision that includes part of the dog's or cat's mandible or maxilla is necessary. Radiation has been recommended to treat microscopic disease in fibrosarcoma tumors that are too large to obtain adequate margins. Hi-lo fibrosarcomas carry a poor prognosis regardless of surgery or radiation therapy. This may be partly due to the fact that most are associated with the maxilla and are thus more difficult to completely excise.

Osteosarcoma. Although malignant, oral osteosarcoma, which usually occurs in the mandible of dogs, may not be as aggressive as osteosarcoma of the appendicular skeleton in the dog (Figure 5).

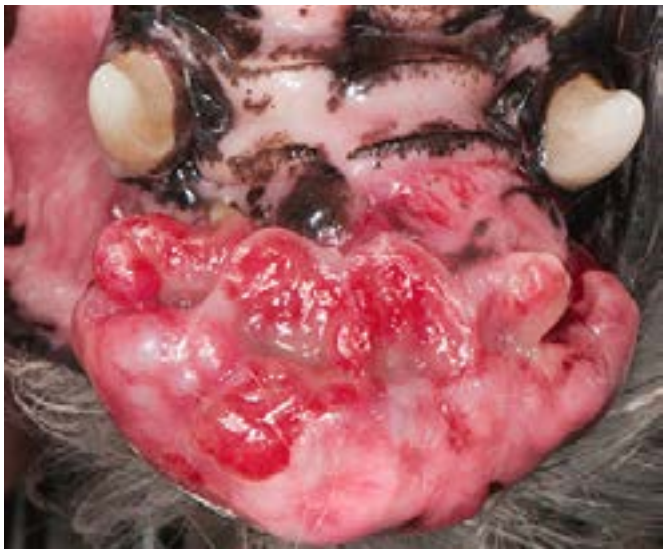

Figure 5. Osteosarcoma affecting a dog's rostral mandible.

Osteosarcoma is rare in cats In a study of 51 dogs with osteosarcoma treated with partial mandibulectomy alone (32 dogs); partial mandibulectomy and chemotherapy (10 dogs); partial mandibulectomy and radiation therapy (3 dogs); partial mandibulectomy, radiation therapy, and chemotherapy (4 dogs); and radiation therapy alone (2 dogs), the group treated with surgery alone achieved a 1-year survival rate of 71%, compared with the entire group's 1-year survival rate of 60%.[5]

In cases of maxillary osteosarcoma in both dogs and cats, recurrence is common after surgery. Chemotherapy and radiation in the treatment of oral osteosarcoma have not been associated with an increase in survival.

Palliative radiation, single exposure every 2-3 months, very effective in controlling pain caused by inoperable maxillary osteosarcomas
- Editor

For a quick reference of all the oral tumors that can affect dogs and cats, see the sidebar, "Oral malignancy nomenclature," on page 27..

How to handle oral malignancies

Diagnosing your canine and feline patients is only the first step. Here's what to consider next: Talk frankly with the client. There can be little benefit from attempting to sugarcoat the diagnosis. If the mass is large, located caudally or has been present for a while, the prognosis is generally poor. Recently diagnosed small masses that are located rostrally generally carry a good prognosis when surgical excision with wide clean margins can be obtained. In some of these cases, radiation therapy combined with surgery adds to the favorable prognosis.

Find out more about the mass and the patient. Three-view thoracic radiographs need to be examined for metastasis. If present, surgery can still be performed, but the prognosis for long-term success is poor.

Further staging should include intraoral radiographs of the mass. The availability of computed tomography and cone beam computed tomography is a game-changer in treatment planning for oral masses in that they allow the practitioner to better view the whole iceberg instead of only the tip. The majority of malignant tumors will have evidence of bony involvement at the time of diagnosis; however, radiographic evidence of lysis does not occur until 50% of the bone has become demineralized (Figure 6).

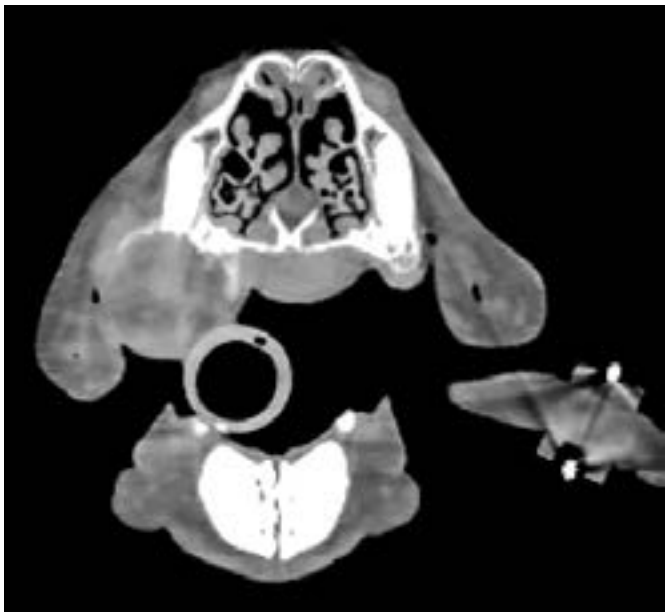

Figure 6. Fibrosarcoma with extension into the bony hard palate

Surgery. In cases where metastasis is not present, the chance to cut is a chance to save - especially if excision can be accomplished with at least 2 cm of clean surgical margins in all directions. (Note: It's the "in all directions" part that often creates a challenge.)

Maxillectomy and mandibulectomy nomenclature

Partial mandibulectomy: The surgical removal (en bloc) of part of the mandible and surrounding soft tissues; also called a unilateral rostral mandibulectomy (Figures 7A-7C).

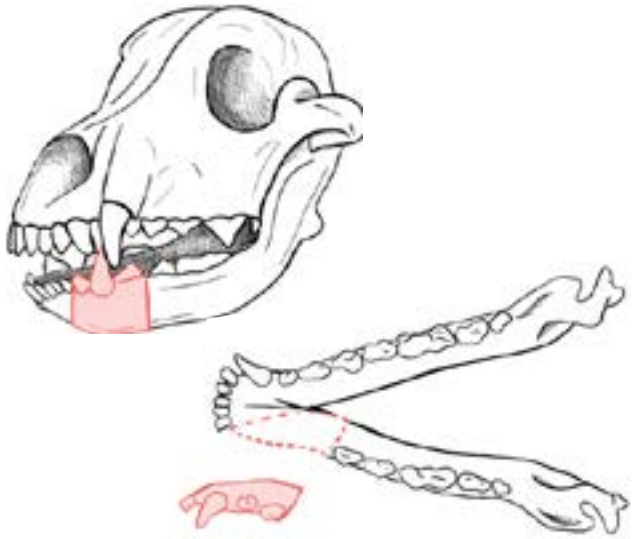

Figure 7A. Partial mandibulectomy diagram. (All illustrations by Roxy Townsend).

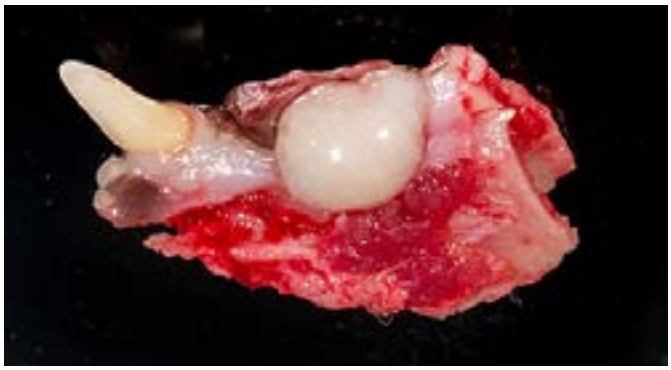

Figure 7B. A surgical specimen from the squamous cell carcinoma patient from Figure 3.

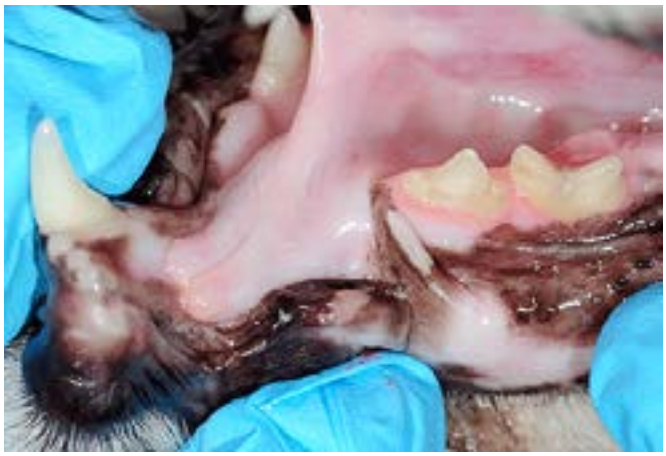

Figure 7C. The healed surgical site with clean margins.

Mandibular rim excision: A form of partial mandibulectomy in which the ventral border of the mandible is maintained (Figures 8A-8C).

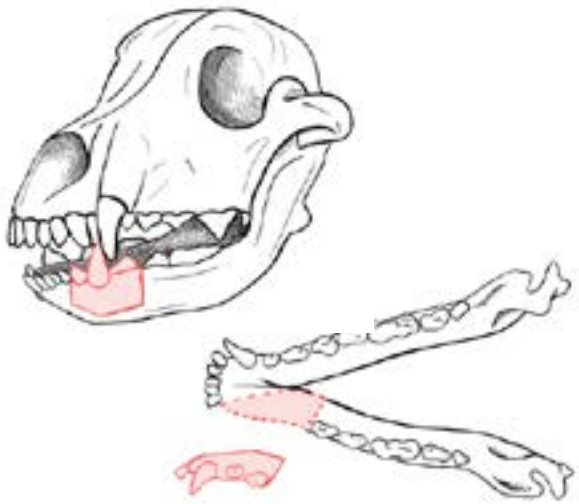

Figure 8A. Mandibular rim excision diagram.

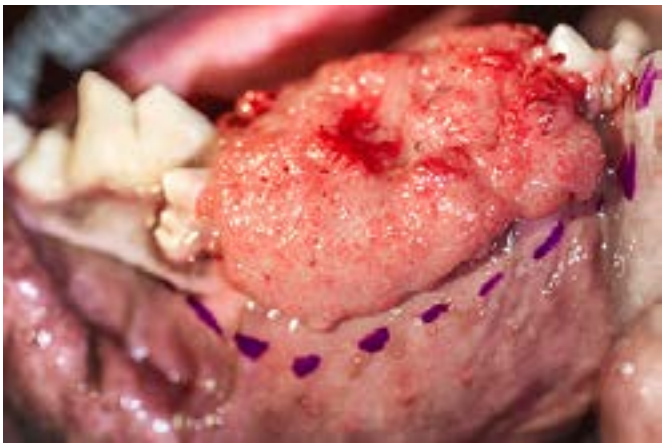

Figure 8B. Surgical markings for a rim excision to debulk the lesion noted in Figure 2. Client would not approve of a rostral mandibulectomy..gif

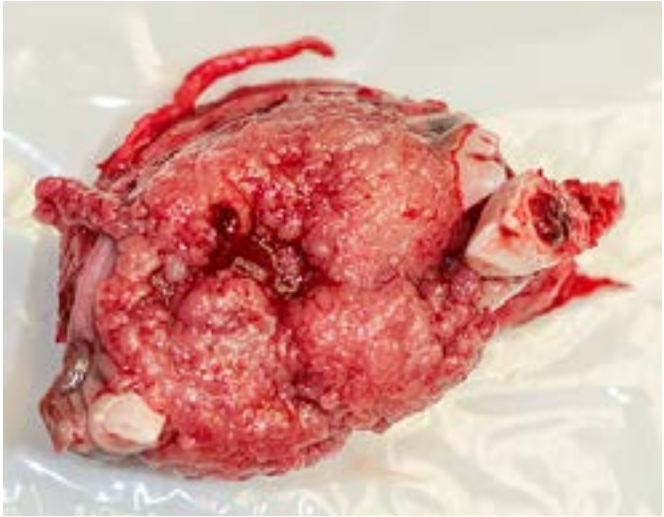

Figure 8C. The surgical specimen without clean histologic margins.

Segmental mandibulectomy: A form of partial mandibulectomy in which a full dorsoventral segment of the mandible is removed; also called a central mandibulectomy (Figures 9A and 9B).

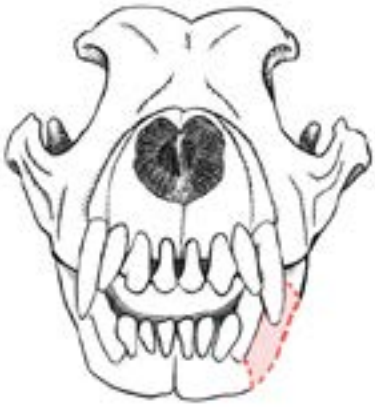

Figure 9A. Segmental mandibulectomy diagram.

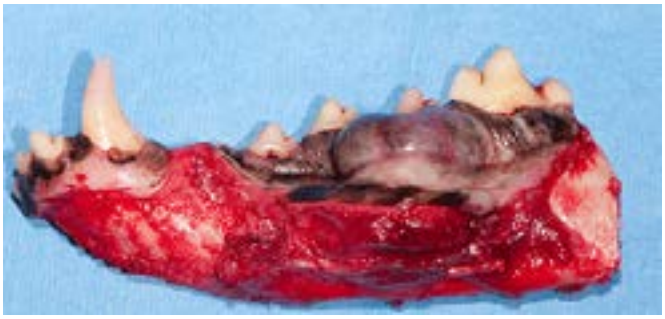

Figure 9B. A segmental mandibulectomy to treat the melanoma noted in Figure 1.

Bilateral partial mandibulectomy: The surgical removal of parts of the left and right mandibles and surrounding soft tissues; also called a bilateral rostral mandibulectomy (Figures 10A-10C).

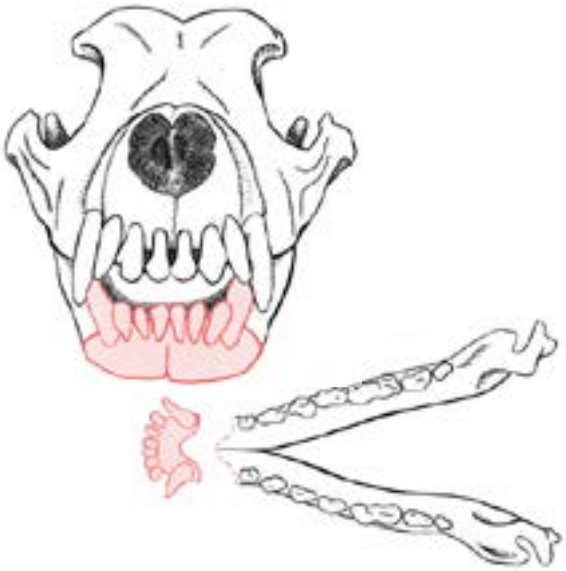

Figure 10A. Bilateral partial mandibulectomy diagram.

Figure 10B. Sarcoma affecting a dog's rostral mandible.

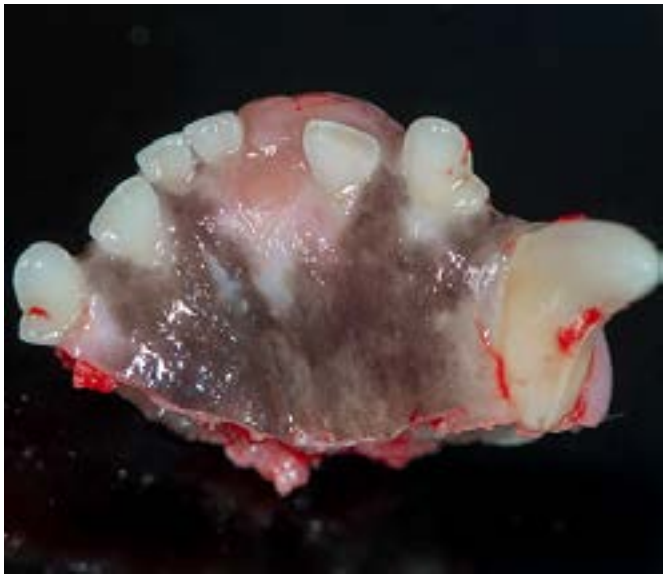

Figure 10C. A bilateral partial mandibulectomy.

Total mandibulectomy: The surgical removal of one mandible and surrounding soft tissues; also called a unilateral mandibulectomy (Figure 11).

Partial maxillectomy: Surgical removal (en bloc) of part of the maxilla and/or other facial bones and surrounding soft tissues; also called a unilateral rostral maxillectomy (Figures 12A-12H).

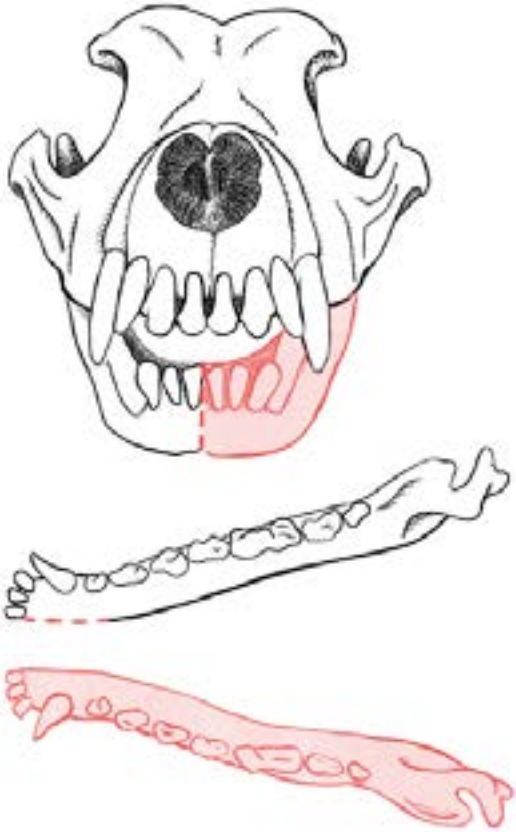

Figure 11. Total mandibulectomy diagram.

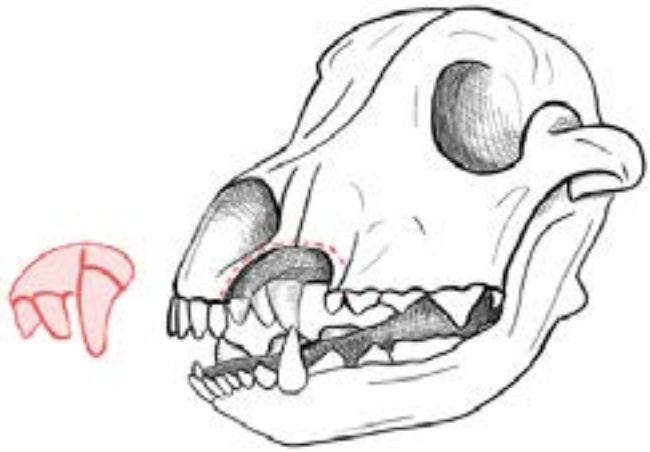

Figure 12A. Partial maxillectomy diagram.

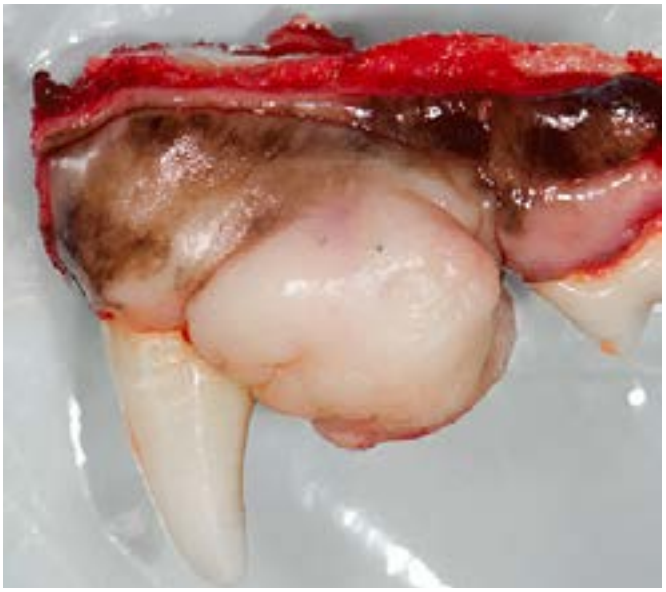

Figure 12B. A palatal view of a fibrosarcoma excision with clean margins of mass noted in Figure 4.

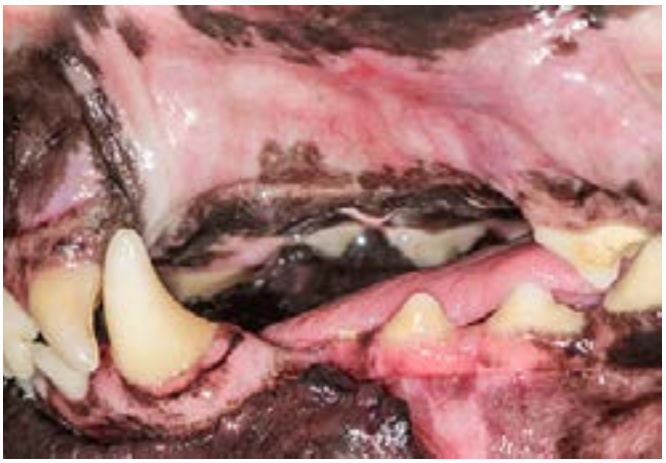

Figure 12C. The patient from Figure 4 one year after surgery in continued remission.

Figure 12D. The patient from Figure 4's clinical appearance.

Figure 12E. Nonfunctional progression of oral osteosarcoma in a different patient.

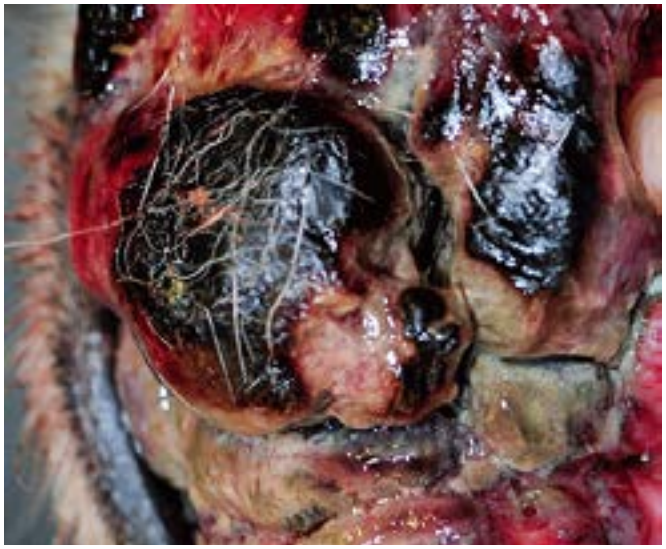

Figure 12F. The surgical appearance of the mass in the patient from Figure 12E.

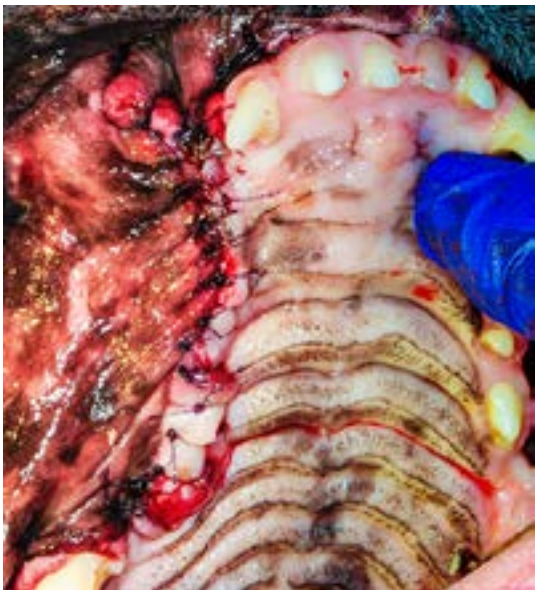

Figure 12G. A partial maxillectomy in the patient from Figure 12E.

Figure 12H. Surgical debulking allowed the dog from Figure 12E to survive seven months after surgery with pain relief and anti-inflammatory medications.

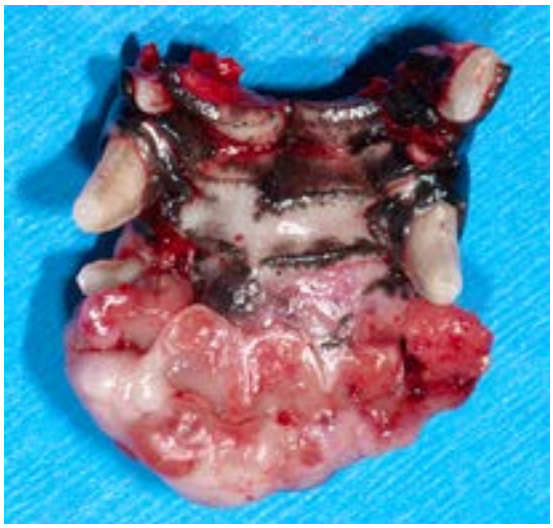

Figure 13A. A bilateral partial maxillectomy to care for osteosarcoma noted in Figure 5.

Bilateral partial maxillectomy: Surgical removal of parts of the left and right maxillae and/or other facial bones and surrounding soft tissues; also called a bilateral rostral maxillectomy (Figures 13A and 13B).

The bottom line? If you can catch and treat malignant masses before metastasis, there's hope for either a cure or at least a happier patient.

Figure 13B. The patient has a normal clinical appearance after surgery.

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