17th Annual

SMALL ANIMAL VETERINARY SYMPOSIUM

Sunday, March 20, 2016

9:00 a.m. – 6:30 p.m.

San Francisco Airport Marriott
1800 Old Bayshore Highway.
Burlingame, CA 94010
(650) 692-9100

SAGE

CENTERS FOR
VETERINARY SPECIALTY
AND EMERGENCY CARE

SAGE

Our Mission

To set the highest standard of compassionate, collaborative veterinary care that improves the lives of pets and people.

Our Values

Compassion
Service
Integrity
Collaboration
Sustainability
Innovation

Sage Philosophy

We believe animals are amazing.

We are here because we appreciate and love animals. We will give pets and their families the best possible care with kindness and respect.

We believe in continual learning.

Our profession is based on scientific knowledge. Our success depends upon creative problem solving, up-to-date training, and the finest skills. We are committed to advancing veterinary medicine and improving pet care throughout the Bay Area.

We believe in collaboration.

We work in partnership with referring veterinarians and one another, knowing it is fundamental to our integrated approach and to continuity of care. All of us are responsible for maintaining the high standards and positive attitude that make Sage a great place to work.

We believe we are part of a greater whole.

In pursuing our mission, we have both opportunities and an obligation to contribute to our communities, to minimize our environmental impact, and to model sustainability.

WELCOME!

We welcome you to the 17th Annual Small Animal Veterinary Symposium. If we can be of any assistance to you, please speak to a SAGE doctor or staff member and we will be happy to help you or answer any questions you may have.

Did You Know…?

- Our practice was **founded in 1992** as Veterinary Surgical Associates (VSA) and has since grown into one of the largest specialty and emergency practices in the country.
- SAGE now has **eight different departments** and **more than 60** doctors on staff.
- We continue to expand the services we can provide at SAGE using a scope through our internal medicine and cardiology departments. This includes video endoscopy and colonoscopy, rhinoscopy, bronchoscopy, and cystoscopy. We perform **fluoroscopy** - including placement of tracheal stents, urinary system stents, pacemaker implantation, minimally invasive PDA correction, and cutting balloon valvuplasty.
- SAGE's team of surgeons and neurologists has a combined total of **277 years of experience**. Surgical services available for companion animals include:

  Orthopedic techniques for fractures and joint reconstruction
  Neurological surgery (including intracranial surgery)
  Thoracic and abdominal surgery
  Reconstructive surgery
  Oncologic surgery
  Minimally invasive surgery

- SAGE offers helical Computed Tomography **(CT scan)** in each of our facilities.
- **Electrochemotherapy**, one of the newest oncologic treatment modalities, involves using electro pulses to increase pores in the cell membrane, making it easier for chemotherapy drugs to enter the cell. It is offered in our Dublin and Concord hospitals.
- Within our Campbell facility, SAGE offers **radiation therapy** with one of the most advanced linear accelerators available in veterinary medicine.
- In Concord, we have a specialist in alternative medicine, offering **acupuncture and herbal support** in an integrative blending of Eastern and Western veterinary medicine.
- SAGE Campbell and SAGE San Mateo have state-of-the-art **physical rehabilitation** units. Treatments include hydrotherapy, laser therapy, joint mobilization, and myofascial release, as well as assistive devices such as carts.
- All four SAGE facilities offer 24/7 emergency care. SAGE has four **criticalists** on staff and can now offer even the most critical patients a chance, including those needing ventilator management.

We hope that you enjoy the educational program offered here today and take the opportunity to continue fostering ties within our regional veterinary community over lunch and at the reception. Please take a moment to fill out our online evaluation of the event and let us know your thoughts. Your suggestions are not only welcome, they are essential in ensuring that this event is the best it can be year after year!

Program Introduction

SAGE Centers for Veterinary Specialty and Emergency Care is pleased to provide a full day of continuing education to Bay Area veterinarians, managers, and nurses. We look forward to spending this enjoyable and informative day with you.

The day will begin with a continental breakfast during registration, with refreshments served throughout the day. We will be offering a seated lunch, and end the day with a great wine and hors d'oeuvres reception.

Here is information about just a few of the noteworthy offerings of the day:

Clinical Tracks:

Internal Medicine? Dentistry? Whatever your interest, there's probably a session in our clinical tracks that covers it. These three tracks will be featuring a number of SAGE Center specialists from each of our departments, including many of our newer doctors. Special guests in our clinical tracks include Dr. Kevin Stepaniuk, a former president of the American Veterinary Dental Society speaking on canine and feline dentistry; Dr. Valerie Fadok, an accomplished lecturer who recently joined Zoetis as the Veterinary Specialist-Dermatology for the U.S. Western Region; and Dr. S. Todd Mitchell, an MD and Principal Investigator in the FDA's open clinical trial studying prevention and treatment of amatoxin mushroom induced hepatic failure.

Nursing Track:

We will begin the morning with a bandage lab, led by doctors and technicians from SAGE's surgery departments. Participants will first hear a presentation about the principles of bandaging, before breaking into groups and practicing. After lunch, guests in the nursing track will hear a series of lectures from David Liss, BA, RVT, VTS (ECC, SAIM), CVPM. David has a rich background in the field of veterinary technology, having spent years working in general practice, referral, and emergency facilities. A contributor to numerous veterinary texts, he was awarded the Veterinary Technician Educator of the Year award in 2012 by Western Veterinary Conference.

Management Track:

Our management track features a variety of compelling topics regarding our industry and the unique issues it faces. Many of the sessions in this track will be led by members of the SAGE management team, with topics including finance, performance reviews, recruitment, and effective communication. The track will close with a management round table discussion, where participants are encouraged to ask questions about issues their practice may be facing. We will also be joined by Byron Farquer, DVM, CVA. Dr. Farquer will be leading an in-depth discussion on increasing profit by understanding what drives client satisfaction and how that affects their purchasing and compliance.

SAGE would like to thank the following sponsors of our 17th Annual Small Animal Symposium

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TRACK 1 — Clinical 1

TRACK 2 – Clinical 2

TRACK 3 – Clinical 3

TRACK 4 – MANAGEMENT

TRACK 5 – NURSING

Track 1

Trends in Diabetes, Hyperthyroidism, and Cystic Calculi Management

Diane Roberts, DVM, DACVIM
Heidi McClain, DVM, DACVIM

Feline hyperthyroidism

Hyperthyroidism is a very common endocrine disease diagnosed in cats. It has been reported that ~10% of geriatric cats will have hyperthyroidism. Due to increased awareness and routine annual laboratory work being performed, we are making the diagnosis much earlier. Many of these cats are being diagnosed with slightly elevated total T4 levels or a normal total T4 and elevated free T4. These patients are typically asymptomatic or subclinical. How should we handle these patients?

In human patients, subclinical hyperthyroidism has been documented. Increased thyroid levels happen over time, and patients in the early stages of the disease do not show clinical signs. The same process is thought to happen in cats. We must be careful in attributing clinical signs (weight loss, vomiting) to hyperthyroidism in a patient with high normal total T4 or just an elevated free T4. If a patient only has mild free T4 elevation and has weight loss or vomiting, it is important to evaluate for other disease processes such as IBD, small cell intestinal lymphoma, chronic kidney disease, or other. The free Total T4 by dialysis is quite sensitive (90%) but is less specific (80%). The clinical issue arises when this patient is referred for I 131. Typically the subclinical patient has no palpable thyroid gland or clinical signs of hyperthyroidism. Many veterinarians mistakenly believe that the finding of a high free T4 concentration alone is enough to diagnose a patient with hyperthyroidism. Dr. Mark Petersen has documented with thyroid scintigraphy that ~20% of these patients are euthyroid. “Over reliance on free T4 testing will lead to the misdiagnosis and inappropriate treatment of many euthyroid cats.” My recommendation is to simply rerun laboratory work in 1 month. Once we have more definitive evidence of hyperthyroidism such as a palpable thyroid gland and a progressive total T4 and free T4, treatment can be reconsidered. If a cat with mild hyperthyroidism is showing clinical signs such as weight loss with a normal appetite, vomiting, diarrhea, muscle atrophy, other diseases should be excluded before treatment for hyperthyroidism. Gastrointestinal disease is the most common disease process misdiagnosed as hyperthyroidism in my practice. In cats suspected to have clinical signs due to hyperthyroidism, a trial therapy of antithyroid drug therapy might be warranted to see if the clinical signs resolve once T4 concentrations are normalized.One other issue we are dealing with in our hyperthyroid patients is the need for increasingly high doses of methimazole over time. Treatment with methimazole does not treat the underlying hyperplasia. The thyroid will continue to hypertrophy and a large goiter is the result. Many of these patients will have escalating doses of methimazole and with time malignant transformation can be seen. If a client would consider definitive therapy, I 131 therapy should be offered earlier in the disease process. As the gland enlarges, thyroid production increases resulting in total T4 levels >15 or even 20 ug/ml in some cases. Our success rate with a one-time treatment decreases in these patients and we warn of the potential of a second dose being necessary.

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Update on Insulin Treatments for Dogs and Cats

Dogs

The most common insulin types that I use in dogs are NPH, and porcine Lente (Vetsulin) or Detemir. Unfortunately, we do not have large, well-controlled studies to compare efficacy of these insulin types. The chronic complaint of NPH is that is has a short duration of activity in some dogs making every 12 hour administration not possible. The recommended starting dose of NPH is 0.25 to 0.5 units/ kg every 12 hours SQ.

Vetsulin (porcine Lente) is the insulin of choice in dogs for some specialists. Porcine and canine insulin are identical in amino acid sequence. The insulin has a duration of action of up to 16 hours in dogs, and the recommended starting dose is the same as NPH (0.25 to 0.5 units/kg every 12 hours). The manufacturer recommends vigorously mixing the Vetsulin due to it being a suspension of two types of insulin. Vetsulin is now available in reusable pens with insulin cartridges in the US.

Detemir (Levemir) is a long acting, recombinant insulin. It has a longer duration of activity than NPH or Vetsulin. It is a very potent insulin and hypoglycemia can occur in dogs. Detemir appears to bind to canine insulin receptors with greater affinity than to human insulin receptors. I recommend careful monitoring of dogs that have been started on this insulin. The starting dose in dogs is quite low due to its potency (0.1 units/kg every 12 hours). The use of Detemir in small dogs is challenging due to this.

Cats

The goal of diabetes mellitus in cats has changed over the last decade. We now hope to obtain remission not just good control. Glargine and Detemir are the two insulins thought to provide the greater chance of remission with PZI close behind. Remission rates are reported to be near 80% with aggressive management. Published data suggests that Glargine or Detemir with a low carbohydrate diet is the initial therapy of choice in cats. The same dosing protocol is used with Glargine and Detemir. Glargine is usually started at 1 to 3 units/ cat every 12 hours. The published dose for Glargine is 0.5 units/ kg every 12 hours with very close monitoring including BG curves daily for the first several days. With this dose, it is very common to have to reduce the insulin in the first two weeks. So, I tend to start slightly lower even though clinical hypoglycemia with Glargine is rare.

Glargine is also now used in initial stabilization of DKA patients in place of Regular insulin. Glargine can be given subcutaneously for a longer duration of action or given IM for a shorter duration of action similar to Regular insulin.

Long term control and maintaining clinical remission will depend on client compliance. At home BG monitoring in cats is much more accurate than curves performed in hospital. A low carbohydrate diet also reduces postprandial hyperglycemia and insulin concentrations in healthy cats. A low carbohydrate diet has been shown to increase remission rates in one study comparing Hills m/d (12% energy from CHO) vs Hills w/d (26% energy from CHO) .

Cystic Calculi Management and Recurrent Urinary tract infection

We often see patients that have recurrent urinary tract infections and the key to managing these is to identify and manage the underlying cause of the infection. One major cause of recurrent infection or stranguria/hematuria is the presence of bladder stones.

STRUVITE STONES

In dogs, 55% of bladder stones are struvite and 40% of kidney and ureteral stones are struvite based. Most struvite stones in dogs occur secondary to a staph or proteus infection. These infections utilize urea which creates ammonia which in turn increases the pH of the urine leading to formation of struvite crystals. Therefore dogs with struvite stones tend to have infection, pH>7.0 and crystals. In dogs, the treatment is a 2 pronged approach for struvite stones.

1) Start a calculolytic diet. NOT A STONE PREVENTION DIET
    a. Acidify urine which increases struvite solubility
    b. Decreased protein so there is less substrate for urease producing bacteria
    c. Increased sodium which induces diuresis
    d. Decreased magnesium and phosphorus
    e. These diets should be used with care in dogs with pancreatitis, heart disease or hypertension
2) Treat the infection
    a. Typically need at least 3 months of treatment as new bacteria will be exposed as each layer of stone is exposed.
    b. Treat for one month past resolution of stone
    c. Kidney stones may require 6 months of treatment

During treatment, monthly rechecks should be done to check urine pH (goal <6.8), urine specific gravity (goal <1.022) and check for crystals and bacteria. If stones are not shrinking by 8 weeks, other courses of therapy should be pursued.

In cats, struvite stones are typically sterile and dissolution is very likely to be successful. In a recent JAVMA paper by Lulich out of Univ of Minnesota, 32 out of 37 cats had complete dissolution of their

stones in less than 1 month. In cats, Hills SD was shown to dissolve stones in an average of 13 days and it drove the pH to 6.0 while Hills CD took average of 25 days. The SD also increases urine volume more and the relative supersaturation rate is much lower than CD.

URATE and CYSTINE stones can also be dissolved.

Urate stones are common in patients with portosystemic shunts, bulldogs and Dalmatians. They are often associated with infection as well so goals of therapy are to alkalinize urine (K citrate), treat infection, dilute urine and treat with allopurinol (xanthine oxidase inhibitor). Unfortunately, dissolution is only effective about 30% of the time. If the stones are going to dissolve, this typically occurs in 8 weeks .

Cystine stones occur in Dachshunds, bulldogs, Mastiffs and Newfoundlands. These can be dissolved by creating alkaline urine and treating with 2MPG (Thiola at 15-20mg/kg q 12hrs). Dissolution tends to take about 10 weeks.

CALCIUM OXALATE STONES:

Calcium oxalate stones are equally as common as struvite stones but unfortunately cannot be dissolved. Males are actually more predisposed than females and certain breeds (Schnauzers, Bichons) have very high likelihood of forming calcium oxalate stones. Several risk factors exist such as increased dietary calcium, increased animal protein in diet, steroid or furosemide administration (increases calcium excretion). There are basically 3 options for stone removal.

1) Surgery: make sure to take x-ray post op!
2) Laser lithotripsy: hoping to have available in the next year. Allows for fragmentation of the stone through a scope and then retrieval
3) Voiding urohydropropulsion
    a. Candidate: female dog with stones <4-5mm, male dog with stones <3mm, female cat with stones <3mm. No male cats!
    b. Procedure: Patient is sedated and the bladder is filled with saline. Patient positioned in vertical standing position so that stones fall into neck of bladder then bladder is expressed. Often done several times. Stones can become lodged in the urethra in male dogs.

With calcium oxalate stones, follow-up after removal is just as important as getting rid of the stones since 40-60% of dogs will reform stones within 3 years! Hypercalcemic patients will reform even faster. Setting up a monitoring program for the high risk patient is critical:

a) Follow up rads or ultrasound every 3 months for first year then every 4-6 months indefinitely to catch early stone formation

b) Monitor urine: goals = urine spec grav < 1.020, urine pH 6.8-7.2.
c) If stone diets are not effective in maintaining urine as above, consider adding potassium citrate at 75-100 mg/kg BID or vitamin B6 2-4 mg/kg q 24-48hr to minimize oxalate formation

Ureteral stones are more common in cats and can serve as a nidus for infection and can often induce severe renal damage by obstruction. These stones cannot be dissolved and should be evaluated for surgical removal or bypass with a stent.

RECESSED VULVA contributing to recurrent infections

In female dogs that have recurrent infections, it is important to always perform an exam of the vulva, especially in younger dogs. Some dogs can have a recessed vulva which then allows for excess moisture to accumulate and create an ideal environment for infection to occur. This can often be managed with weight loss and frequent cleaning but in more severe cases, surgery can be curative.

NEW THOUGHTS ON TREATING UTIs

1) Uncomplicated UTIs can be treated with short course (3 day) antibiotic therapy. A prospective study comparing high dose enrofloxacin (20mg/kg SID) given for 3 days vs standard dose clavamox (13-25mg/kg BID) given for 14 days. Cultures were performed on day 0, 10 and 21 and showed that both methods of treatment were equally effective. Short term therapy is standard of care in human medicine so we will likely trend in this direction. More studies to come.
2) For recurrent UTIs or resistant UTIs, there are studies looking at infusing a nonpathogenic species of e.coli into the bladder to compete with more pathogenic bacteria. It has been shown to decrease pain and reduce bacterial reservoirs and in a small study showed many dogs reverted to negative urine culture. More studies ongoing

Diagnosis and Treatment of Complicated Urinary Tract Infections

BRANDY DUGAS, DVM, DACVIM (INTERNAL MEDICINE)

Overview

- Definitions
- Causes/Anatomy
- Diagnosis
- Treatment
- Prevention

Bacterial Cystitis

- Inflammation of the urinary bladder caused by bacteria
- 14% of dogs during lifetime
- More common in females
- Uncommon in cats, older cats more predisposed (>10 years)

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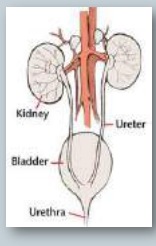
Pathophysiology

- Routes of infection
  - Ascend the urogenital tract
    - Rectal, perineal, and genital bacteria
  - Hematogeneous spread
    - Bacteria
    - Fungus (Candida)
- Must persist and cause an infection
  - Ascend
  - Adhere
  - Multiply and persist

Number & Virulence



Pathophysiology – Host Factors

Local Immunity	Systemic Immunity
• Normal voiding • Anatomic structures • Mucosal defense • Antimicrobial properties of urine	• Local and cell mediated

Pathophysiology – Host Factors

Normal Voiding	• Frequent, complete • Urinary continence
Anatomic factors	• Urethral and ureteral peristalsis, length of urethra, ureterovesical flap valves, urethral high pressure • Prostatic secretions, glomerular mesangial cells, renal blood flow
Mucosal defense	• Antibody production, exfoliation of urothelial cells, surface glycosaminoglycans, competition by commensal bacteria
Urine – antimicrobial properties	• Urine pH, hyperosmolality, urea, Tamm-Horsfall mucoproteins, organic acids, defensins, etc
Systemic immunity	• Humoral and Cell mediated

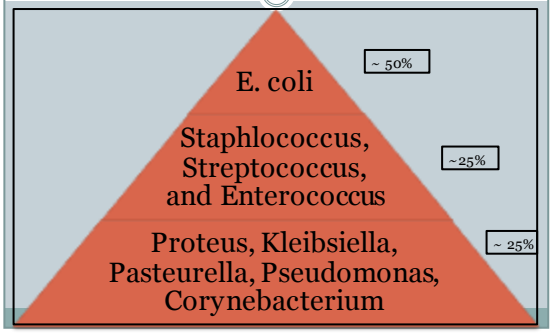
Pathophysiology- Bacteria

- Not all bacteria are pathogenic
  - Escherichia coli, Enterococcus
- Virulence factors (uropathogenicity)
  - E. coli
    - Adhesins (fimbrae, pili)
    - Capsular antigen (K), Somatic antigen (O)
    - Hemolysin
    - Aerobactin
    - R-plasmids
  - Proteus, Staphlococcus, Klebsiella
    - Urease activity
    - R-plasmids

Normal commensal bacteria in lower urinary tract of dogs

Genus	Distal urethra (males)	Prepuce	Vagina
Acinetobacter		+	+
Bacteroides			+
Bacillus		+	+
Citrobacter			+
Corynebacterium	+	+	+
Enterococcus			+
Enterobacter			+
Escherichia	+	+	+
Flavobacterium	+	+	+
Haemophilus	+	+	+
Klebsiella	+	+	+
Micrococcus			+
Moraxella		+	+
Mycoplasma	+	+	+
Neisseria			+
Pasteurella		+	+
Proteus		+	+
Pseudomonas			+
Staphylococcus	+	+	+
Streptococcus	+	+	+

Bacterial Urinary Tract Infections

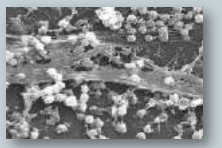


Bacterial Urinary Tract Infections

- My coplasma and Ureaplasma
  - <5%
- Single isolate (75%), two isolates (20%), > two isolates (<5%).

Uropathogenic E. coli

- UPEC
  - Both extracellular and intracellular invasion can occur
  - Failure to clear infection with traditional antibiotics
  - Biofilm also helps protect
    - Biofilm formation associated with E. coli resistance
    - Treating early may prevent formation



Risk Factors

- Exogenous corticosteroids (18-39% of dogs)
- Hyperadrenocorticism (46%)
- Diabetes mellitus (24-37% dogs, 12-15% cats)
- Kidney disease (22-30% of cats)
- Chemotherapy
- Immunosuppressive drugs
- Urinary incontinence
- Anatomic abnormalities

Complicated vs. Uncomplicated UTI

- Uncomplicated: sporadic in healthy individual
- Complicated:
  - anatomic or functional abnormality present or
  - comorbidity that predisposes to recurrent infection/treatment failure

Asymptomatic Bacteriuria

- Bacteriuria ≠ urinary tract infection
- Bacteria present without causing clinical signs
  - Lack virulence factors
- May be protective
  - Complete with pathogenic bacterial strains
- Caution – some patient with underlying immunodeficiency may not show clinical signs
  - Hyperadrenocorticism, diabetes mellitus, hyperthyroidism, chronic kidney disease

Symptomatic Bacteriuria

- Pollakiuria
- Stranguria
- Hematuria
- Dysuria
- Urinary incontinence*

Diagnosis

- Urinalysis and urine culture
  - Cystocentesis
- Urinalysis
  - Normal urine specific gravity
  - Bacteriuria
  - Pyuria
    - >3-5 WBC/hpf if cystocentesis
    - >5-10 WBC/hpf if voiding or catheterization
  - Hematuria
  - Proteinuria

Diagnosis

- Urine culture and susceptibility – gold standard
  - Bacterial identification and antibiotic susceptibility
  - Quantitative

	Significant		Contaminant	
Sample	**Dogs**	**Cats**	**Dogs**	**Cats**
Cystocentesis	>1,000	>1000	<100	<100
Catheterization	>10,000	>1,000	<1,000	<100
Midstream voiding	>100,000	>10,000	<10,000	<1,000

  - Susceptibility testing
    - Susceptible, resistant or intermediate

Imaging

- Abdominal radiographs
  - Uroliths, masses, renal size/shape
- Abdominal ultrasound
  - Thickened urinary bladder wall
  - Mass lesions – polypoid cystitis vs. neoplasia

Imaging

- Contrast cystourethrography, double contrast cystography

Cystoscopy

- Recurrent infections
- Evaluate for underlying cause
- Collect biopsy samples for histopathology and culture

Antimicrobial Selection

- Based on urine susceptibility testing
  - Ease of administration
  - Few side effects
  - Inexpensive
  - Attains high urine concentration
  - Exceeds MIC >4x
- "First line" antimicrobials
  - Amoxicillin
  - Cephalexin
  - Trimethoprim-sulfamethoxazole
- Antimicrobials to avoid
  - Potentiated β-lactams (Clavamox)
  - Fluoroquinolones**
  - Extended release cephalosporins (Convenia)

Antimicrobial Selection

- Uncomplicated UTI
  - 10-14 days
  - Urine culture 5-7 days after AB are finished
- Complicated UTI
  - Intact, predisposing condition, pyelonephritis, prostatitis
  - 4-6 weeks
  - Urine culture after 1-2 weeks of therapy and again 5-7 days after AB are finished

High Dose Short Duration Antibiotics

- J Vet Intern Med 2014;28:818-826

Short and Long Term Cure Rates of Short Duration Trimethoprim-Sulfamethaxazole Treatment in Female Dogs with Uncomplicated Bacterial Cystitis

- No significant difference in resolution of clinical signs or microbiological cure rate at short term (4d) or long term (>30 d) follow up
  - Short term resolution of clinical signs in most (89% TMP, 94% cephalexin), less so for durable clinical remission (50% TMP, 65% cephalexin)
  - LOW microbiological cure rates
    - 4day = 59% TMP, 36% cephalexin
    - >30 day = 44% TMP, 20% cephalexin

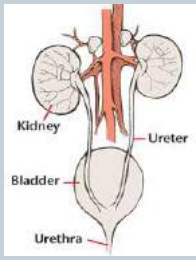
- J Vet Intern Med 2012;26:506-512.

Evaluation of the Efficacy and Safety of High Dose Short Duration Enrofloxacin Treatment Regimen for Uncomplicated Urinary Tract Infections in Dogs.

- Similar microbiological and clinical cure rate for both groups.
  - 7 day follow up only
  - Clinical cure: 77% enrofloxacin, 81% Clavamox
  - Microbiological cure: 81% enrofloxacin, 80% Clavamox
  - No long term follow up

Localization

- Antibiotic penetration into site
  - Prostate: TMS, chloramphenicol, enrofloxacin
- Duration of antibiotic therapy
- Ancillary treatments
  - Neutering



Antimicrobial Resistance

- Widespread problem in human and veterinary medicine
- Indiscriminant antibiotic use:
  - Promote multi-drug resistant bacteria
  - Disturbs normal flora
  - Encourages colonization

Asymptomatic Bacteriuria

- To treat or not to treat….
  - Risks of untreated UTI vs. unnecessary therapy
  - Bacteriuria in a healthy patient – no treatment necessary
  - Bacteriuria in patients with risk of pyelonephritis (CKD, diabetes, immunosuppressive therapy, etc) – treat

Recurrent Urinary Tract Infections

- ***Reinfection***
  - Recurrence occurs with a different organism
  - Suggestive of anatomic abnormality, physiologic dysfunction, or problem with host immunity

- ***Relapse***
  - Recurrence with the same organism
  - Suggests a nidus or reservoir of infection that shields bacteria from antibiotics
    - Kidneys, prostate, thickened bladder wall, mass, urolith, etc
- ***Persistence***
  - Appropriate therapy does not clear the infection
  - Drug factors, bacterial factors, impaired immune system

Treatment of Reinfection

- Treat/remove underlying cause
  - Urolith, hyperadrenocorticism, vulvar hooding, etc
- If months apart treat with 10-14 day course of appropriate antimicrobial based on UC&S

Prophylactic Antimicrobial Use

- Only if a thorough evaluation for underlying causes has been done
- Reinfection >4 x/year or relapse
- Treat with appropriate antimicrobial until culture negative then decrease to once daily at night
- Re-culture monthly
- Minimum of 6 months, re-culture 1 week, then monthly for 3 months, then ever 3 months.
  - Ex: amoxicillin, amoxicillin/clavulanate, nitrofurantoin

Ancillary Therapies

- Cranberry Extract
- Probiotics
- Methenamine hippurate
- Fosfomycin
- Emerging therapies

Cranberry Extract

- Proanthocyanidins
  - Decreases adhesion of E. coli in vitro in humans
  - May also suppress inflammatory response
  - Appears to reduce risk in women

Probiotics

- Most infections due to ascending infection
- Healthy microbial population of vestibule and vagina important in female dogs
- Mixed microbiome
- No clear benefit in human studies

Methenamine Hippurate

- Urinary antiseptic
- pH <6.5 - formaledhyde and ammonia
  - Vitamin C, ammonium chloride, dl-methionine
- Formaldehyde has bacteriocidal effects

- Side effects:
  - Gastrointestinal upset
  - Caution if renal insufficiency as an acidifying agent
  - **Carcinogenic**

Fosfomycin

- Monurol
- Bacteriocidal drug effective against UPEC and MDR E. coli

Emerging Therapies

- Bacteriophages: Bacterial viruses used to kill UPEC strains
- Induction of asymptomatic bacteriuria: specific E. coli strain that cannot express adhesins
- Immunomodulation: Immunostimulant OM-89
- Acupuncture

Prevention

- Correction of underlying cause
  - Ex. hyperadrenocorticism, recessed vulva, ectopic ureters, USMI, urolithiasis, prostatitis, pyelonephritis, etc
- No standard screening consensus
- Patients with risk factors
  - Chronic kidney disease – urinalysis with culture and susceptibility at least every 6 months
  - Initially in all patients with hyperadrenocorticism and diabetes mellitus, repeat if signs of dysregulation occur

Managing Ureteral Calculi in Cats with Novel Therapy

Tim Sellmeyer, DVM, DACVS

One of the most common causes of feline ureteral obstruction is due ureterolithiasis. Most often, cats do not show signs of a unilateral obstruction and are instead presented due to signs of renal failure (>80%), indicating that the unobstructed kidney is also diseased. This eliminates nephrectomy of the obstructed kidney as a treatment option. Traditional ureteral surgeries have 30% complication rates and up to 30% mortality even with operating microscopes and highly trained surgeons. Feline renal and ureteral stones are often composed of CaOx, so dissolution is impossible. Furthermore, 62% of cats do have renaliths that can result in future obstructions even if the initial ureterolith

In the last few years, two techniques have been developed to bypass this obstruction, decompress the kidney, allowing urine flow to the bladder and preserving any remaining renal function. These two techniques are the intraluminal ureteral stent and the subcutaneous ureteral bypass system.

Any ureteral obstruction patient can be a candidate for intralumenal ureteral stenting or subcutaneous urinary bypass (SUB). Candidates are those with an ultrasonographic diagnosed dilated ureter and dilated renal pelvis. Many of the following tests will be performed during the initial work-up of these patients, but a complete list is compiled that can be performed prior to referral.

Pre-operative diagnostics:
CBC, serum chemistry, urinalysis and urine culture.
If there is an active UTI, then IV antibiotics will be administered preoperatively and continued for 6 weeks post-operatively.

Preoperative imaging:
Abdominal radiographs and abdominal ultrasound should be performed on azotemic cats. The renal pelvic dilation must be at least 6mm to accommodate the pigtail coil of the intraluminal ureteral stent. An obstructed ureter with a smaller renal pelvic dilation can be treated with a subcutaneous ureteral bypass system.
If a heart murmur is detected on thoracic auscultation, chest films should be performed as well as an echocardiogram due to the need for high volume fluid diuresis in these patients.

Pre-operative treatment:
It is important to treat these patients with 24 hours of medical management to not only for diuresis in preparation for surgery, but to also allow spontaneous passage of ureteroliths. This is contraindicated if there is hyperkalemia, anuria, or overhydration. Medical management includes IV fluids, prazosin (0.25 to 0.5 mg/cat PO BID), mannitol (0.25 to 0.5 g/kg bolus over 30 min then CRI at 1mg/Kg/min for 24 hr), amitriptyline (0.5 to 1mg/Kg PO SID), antibiotics, H2 antagonists, amlodipine, and phosphate binders. The patient needs to be monitored closely

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for urine output, BUN, Creatinine, and electrolytes during this time. If there is an active UTI, then IV antibiotics will be administered preoperatively.

Surgical placement of either the intraluminal ureteral stent or SUB in cats is performed through a ventral midline celiotomy to facilitate stabilization of the very mobile kidney and to perform a standard cystotomy during the stent procedure. Both procedures require the use of fluoroscopy to confirm appropriate placement of the ends of the product. In the placement of the SUB, fluoroscopy is also used to confirm patency.

Immediate Post-op monitoring:
Body weight TID, Serum chemistry BID, PCV/TS BID, urine output, Antibiotics if indicated for 2 weeks. A feeding tube is going to likely be placed in all cats as most are anorexic pre-operatively and (especially with the subcutaneous ureteral bypass system) are painful post-operatively. Nutritional support through the feeding tube can be started immediately post-operatively and continued as long as needed and even used solely for medication administration.

Long term follow-up:
Recheck at 2 weeks, 6 weeks, 3 months, 6 months, 9 months, 12 months, and then every 3-6 months afterwards. Recheck to include i-stat, blood pressure, urinalysis, abdominal radiographs and abdominal ultrasound. If there was an active UTI identified pre-operatively, then IV antibiotics will be administered for 6 weeks post-operatively.

Long term management:
Potassium citrate supplement (75mg/Kg PO BID) if needed, if persistently azotemic then renal diet with stone neutral diet and phosphate binders if needed.
If dysuria, consider steroids, prazosin, amitriptyline or stent exchange. Dysuria is defined as greater than 4 litter box trips per day.
If a SUB is placed, then flushing of the system under ultrasound visualization is recommended every 3-6 months.

Getting a Grip: Improving Geriatric Dog Mobility

Discussion of Select Geriatric Orthopedic Problems

Lissa Richardson, DVM Dip ACVS

Sage Symposium March 20, 2016

My inspiration for this talk stems from 3 places. I have recently finished a book entitle, Being Mortal by Dr. Atul Gawande, a Boston surgeon, writer and public health researcher. He does a remarkable job in helping guide his reader though the issues that face us in old age by exploring history and patients' stories. He is articulate and unflinching. Reading his book will set your head straight as you approach this for yourself or loved ones. It also opened my clinical eyes to some of the symptomatic ways to help our aging pets. The second source of inspiration is my own aging, I am now 55, feeling great but aware that I have to *work* for the ability to get down on the ground to do those large dog ortho exams….and then get back up. The third has been my patients. I seem to be seeing more frail dogs that can use significant help to get around. Maybe my lens is just focusing here.

The cause of weakness and slipping on wood floors are many and often there are overlapping arthritic, neurological and metabolic causes. These are some of my longest consults. First I will take a history, listening especially as to what is causing the owner the most distress. Then a careful physical, orthopedic and neurological exam needs to be done to generate an often lengthy problem list. Together with the owner, I prioritize the problem list and sketch out the diagnostics that may be needed to come to the root cause so that we can outline a treatment plan and give clear information on expectations. Then the negotiations begin on the cost estimate and this is linked primarily to prognosis and quality of life.

One of the more frequent scenarios is the older large breed dog with some degree of stifle or hips arthritis that develops some neurologic weakness that is seen by ataxia or even just mild scuffing. Owners frequently think it is just the progression of arthritis and it is important to note and educate them on the neurological component. The most common causes are a Hansen type II disc herniation or degenerative myelopathy. More rare are discospondolytis, neoplasia or meningitis. Often, we do a general blood work, thoracic radiographs and plain spinal radiographs. If these are normal, many owners stop short of doing the MRI and are looking for symptomatic care at this point.

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Some of the most effective ways to improve mobility are to strengthen muscles with exercise and physical rehabilitation, reduce pain and inflammation, and weight loss when appropriate. These topics are weighty and worthy of entire lectures. Early recognition and focused conversation about the benefits exercise are critical. Physical rehab can be extremely beneficial in treating chronic slow disc herniations.

An addition, the environment must be modified to provide a surface with texture. It is human nature to resist environmental change as we think it is admission of the frailty of old age. Hand bars at toilets, canes and walkers are synonymous with old geezer. The fact is however, these things prevent falls. A vital aspect of being healthy in old age is to not fall. Falls lead to injuries which lead to hospital visits. Our geriatric dogs are closer to the ground and do not sustain our common injuries of concussion and bone fractures. Yet, slipping leads to iliopsoas strain and ventral hip luxation as well as other injuries. When you have a dog that cannot maintain footing, throw rugs and runners of rug pads even alone are essential. I have made pathways in my house that my old collie would follow like a game piece on a board game using rubber non slip rug mats. I highly recommend Solutions catalog for affordable and attractive floor and stair coverings.

There are also a lot of products or techniques to reduce slipping and gain better traction and paw protection. Some, helpful, some fluff, and some with potential serious downsides. Conflict of interest disclaimer: I have not received any payment to endorse any product. I am not a paid consultant for any company. I have asked for some free samples of certain products but with no strings attached.

Exam of the paws

Pads should be flexible, not hard. Nails should be short. Trim any long interdigital hair. If pads are hard, I recommend balms to help soften. Two that I recommend are Mushers Secret and Burt's Bees paw and nose lotion. I recommend application at night and then placing the E collar to allow for a long lick-free absorption period. Application can also be done prior to a meal so that they are immediately distracted from licking it off. Once a day can often be enough.

Aids to help prevent slipping

Direct on the paw:

Shaws Paw Wax: This is different from most pad lotions; it has more of a sticky feel to it. The ingredients are white oil, paraffin wax, rosin, silicon dioxide, and perfume. I have used it on my own geriatric collie with type II disc. It requires at least a daily application. I found it did not leave marks on the wood floors. It is produced in the UK but is available on Amazon. This product will only serve to slightly improve traction.

Paw Friction: This product uses a thin layer of cyanoacrylate on the pads followed by an application of small rubbery grains to the glue. It provides more traction than the wax and can last 10-14 days. One benefit is that this does not require daily application. It is a two person job however and the cyanocrylate application needs to be done well and avoid the skin or hair. We are currently testing this product.

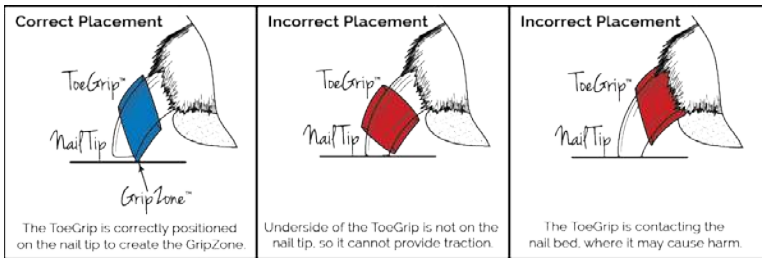
Patches: There are several adhesive patches that stick directly to the dogs pads. The benefits are that one application lasts several days. If you loose one, the others are often enough to help. Clean the pads with rubbing alcohol and let dry completely. The disadvantage is that they often do come off and dogs can ingest them so it is not endorsed.

Puppy Love Paw Pads

Pup Gear

Nail Treatments:

Dr. Buzby's nail grips: These are very interesting and we have been using them for many months in our Concord facility. It works because there is a thick rubber band around the nail that contacts the floor when a dog walks. A single application can last for a month or two. Nails must be trimmed short for these to work and we trim nails and place these in the clinic. The owner must look at these daily to make sure they are not riding up (to loose). They can also be placed to tight and vascular compromise to P3 can happen. Owners should monitor closely for signs of pain. Discoloration in pigmented nails is not possible to see. Click here for the web site. There are several helpful videos for application and trouble shooting. Our physical therapy department has seen severe complications with the riding up of these rings. When dogs are in grass or other compliant surface, the rings can ride up against the skin, even when sized appropriately. Minor irritation can progress to infection or avascular necrosis. Careful daily monitoring of these are essential.



Booties:

These would seem a great option but there are some major drawbacks. The first is that dogs usually hate them. They can cause a dog to high step and try to flip them off. Dogs will often chew at them. Now, I have seen geriatric dogs that suddenly gain traction, tolerate them. Some of the booties that are thin and grippy are not tough enough to use outside and this then necessitates owner taking them on and off. I have many owners who are aged themselves and bending over to place booties on and off is simply not possible.

For dogs with proprioceptive deficits, it is generally better to keep them out of booties for a long as possible. I use them only when they are wearing the skin of the dorsum of the paw and causing injury. Worn nails from scuffing can be capped with Soft Paws to help protect and minimize bleeding.

Here are some products with comments.

Sticky Pawz: These give instant and excellent traction and are thin enough that dogs can feel the floor surface well. They are not very robust when used outside and wear quickly. They are not expensive and can be replaced. They work well in the hospital

for temporary use. The main disadvantage is that they cannot be left on for a long time as they are rubber and do not breath. The second disadvantage is that they are difficult to place and some owners with arthritis or who cannot bend over will not be able to use these.

Naked Socks by Otis: These are thin and comfortable and have a handy Velcro band to keep them on . They have excellent non slip ability but wear quickly when outside on cement. The fabric breaths well so they can be kept on long times.

The website www.baxterboo.com has a very large collection of booties that range from tough to very soft and flexible.

Harnesses and slings:

In the hospital we use the Quick Lift ™ sling and dispense it home with our post op orthopedic and neurological cases. This is helpful to support the rear legs. However dogs that have minimal to no function need more support and we use the Help'EmUp™ dog harness or Walkabout Rear Harness . The additional benefit is that these can stay on all day and just come off at night to allow the belly and groin some rest and to prevent rub sores.

What can your clinic do to improve the experience of the teetery geriatric dog visit?

1. Roll out the carpet. The most cost effective way to lay a path for these dogs is to use a runner of rubber rug mat (like Grip it or Rug Wrench)
2. Apply a pad treatment like paw wax or gripping patches
3. Have a sling ready to help.
4. Include foot pad evaluation with nail and digital hair trim.

Select Geriatric Orthopedic Problems.

Injuries Caused by Slipping

1. Ventral hip luxation. What you see is acute non weight bearing lameness and pain with hip palpation. Radiographs show a ventral hip luxation. Reduction is done under heavy sedation or general anesthesia. While on their back, extend the hip and apply inward rotation (the action is similar to positioning for OFA hip extended films). Sometimes some digital pressure on the proximal medial femur helps. Once reduced, place tape hobbles that allow the dog to walk but

not splay out again. Keep on for about 4 weeks. Hobbles are best placed above the hocks for maximal comfort and minimal tripping. Care is taken to improve footing with nail trim, hair trim and use of above products. It is very rare to ever need surgery to successfully manage these patients.

2. Iliopsoas Strain. What you see is a weight bearing lameness that is worse after activity. Diagnosis is made with finding pain with digital pressure on the lesser trochanter area. Screening radiographs of the pelvis are normal. Treatment is to restrain from explosive activity, jumping and running for 6 weeks. Walks are important but should be of short duration, 10 minutes. Referral to physical therapy is helpful. In dogs with slipping, improve traction as above.

Elbow Callus Infections

As dogs age, the elbow callus often gets thickened and hyperkeratotic. These can crack and become infected. As they are thick areas of skin, the early signs of swelling are masked. Careful palpation of any callus in a febrile dog is essential. If there is any question, a simple aspirate should be done to look for pus.

Once diagnosed, the abscess should be lanced, flushed and drains placed. These are difficult areas to bandage. You cannot safely just bandage the elbow without significant risk of causing swelling distally. Full bandage can be done but will need daily changing. I recommend to place a tie over bandage. Once loops of suture have been placed in the skin around the callus, place lap pads or gauze pads and tie over the gauze which will cover the drains or open areas. We use burn stockinet over this or Hearty Mutts, see picture. The toe can be cut out. Culture the wound and place on appropriate antibiotics until complete resolution, about 2 weeks. Change the bandage daily for 3-5 days, then pull drains.

Prevention involves attention in 2 areas, preventing cracking of the skin and padding if hygromas or pressure wounds are present. I like to treat dry calluses with a balm, see products mentioned earlier for dry paw pads. For the very thick one, best to use a human product for softening calluses, kerasal.

Padding the elbow is best accomplished with using DogLeggs , a well designed padded neoprene bandage that velcros on and off. We have been very happy with the design and the dogs acceptance of them.

Septic Arthritis

What you see is an acute non weight bearing or toe touching lameness of the affected limb. There is marked pain with any range of motion of the joint, the pain mimics fracture pain. There can be swelling but again this is often masked by underlying joint thickening due to arthritis. The infection is blood born and the point of entry can be through GI, respiratory or skin. The inflammation results in leukocytes,

fibrin and fluid into the joint and starts the cycle of profound and irreversible cartilage destruction. [1] Diagnosis is made on cytology of a joint aspirate. A sample should be submitted for culture and the chance of a positive result can be increased with placing the sample in blood culture media. Typical findings are PMN between 40,000 and 100,000/mm3. Treatment should begin as soon as the diagnosis is made (within 24 hours) and ideally is arthrotomy and lavage. An ingress/egress with 14 gauge needles is second best as it often clogs, but can still help to remove the purulent fluid from the joint. A cephalosporin alone or in combination with a fluoroquinolone is started and appropriate antibiotic continued for minimum of 4 weeks. There should be improvement in pain and swelling in 3 days if treatment is effective. Recurrence can happen if the infection is not completely cleared. There can also be a worsening of the underlying arthritis due to cartilage destruction that owners need to know. The key message is to have a high degree of suspicion and make the diagnosis early. This will lead to prompt treatment and critical cartilage sparing.

[1] Textbook of Small Animal Surgery, second edition 1985, Slatter, pp1690-1691

Current Treatments for Chronic Elbow Arthritis

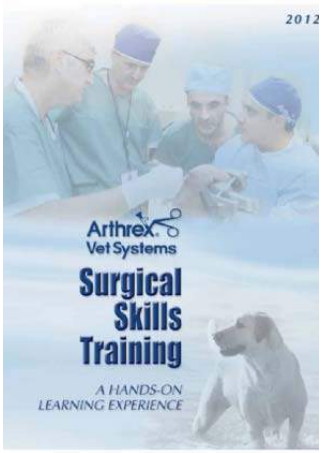
SAGE

An approachable team of specialists providing advanced, collaborative, and compassionate care.

Charles M. Walls DVM, DACVS
March 20, 2016

Disclosure

- Arthrex consultant
- Receive no financial compensation for any Anthrex product.



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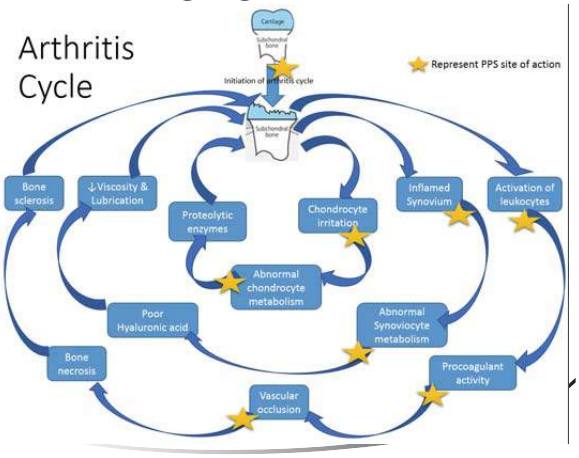
Osteoarthritis (OA)- A brief review



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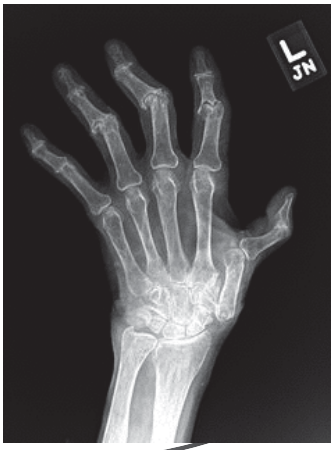
Primary OA

- Is a disease of the entire joint:
  - cartilage
  - joint lining
  - ligaments
  - bone
- *Primarily Human



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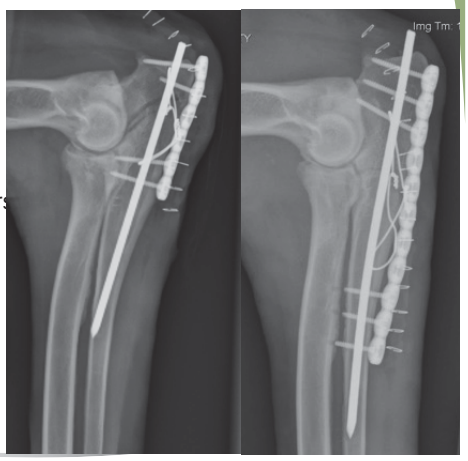
Secondary OA

- Change in the micro-environment of Joint.
  - Trauma
    - Joint fractures
  - Metabolic disorders
    - VWD's
  - Congenital dz.
    - Dysplasia



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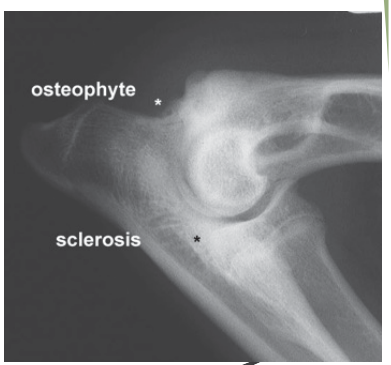
Elbow Dysplasia- OA

- Most common cause of chronic fore limb lameness in dogs
- Inherited, occ. traumatic
- Developmental asymmetry
  - incongruity
- Range of disease pathology and clinical signs
  - FCP, OCD, UAP
- All result in varying degrees of OA



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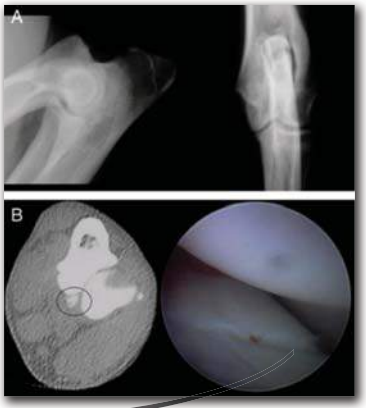
Diagnostic Modalities

- Standard radiographs
  - Lat, AP, Flexed lateral
- CT/ MRI
- Arthroscopy*
  - Mainstay of medial compartment disease diagnosis



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Radiographs

- Typical changes
  - Sclerosis
  - Osteophytosis
  - +/- incongruity
    - humeral/ulnar joint space narrowing
- *Does not correlate with arthroscopic disease/ articular cartilage wear

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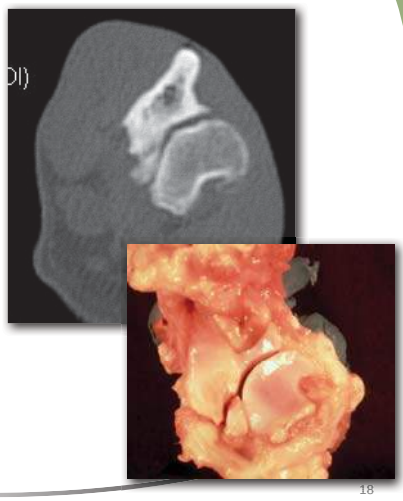
*N. Fitzpatrick, Vet Surg. 38:213-223,2009

CT Scan

- 1mm slices
- Assess elbow incongruity
- Assess medial coronoid changes
  - fissure vs. fracture
  - subchondral bone changes-hypodensity
  - Radial/ulnar incisure



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Arthroscopic Portals of The Elbow

- Caudomedial
  - Craniomedial
  - Caudolateral
- Portal Types
  - scope
  - egress
  - instrument

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Caudomedial Portal

- Anconeal process
- Semilunar notch
- Lateral coronoid & capsule
- Radial head
- Medial coronoid
- Humerus

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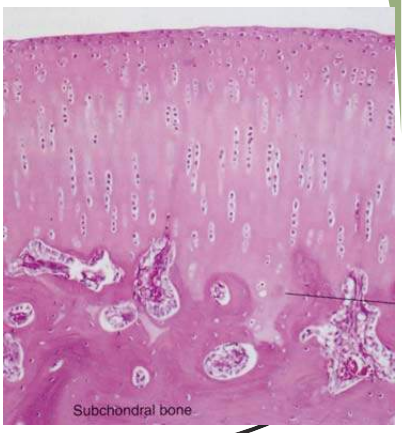
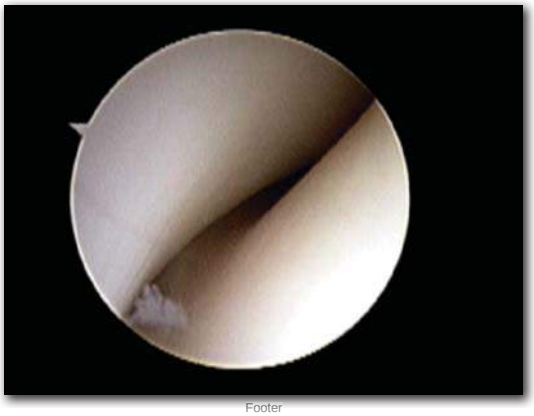
Modified Outerbridge Scale

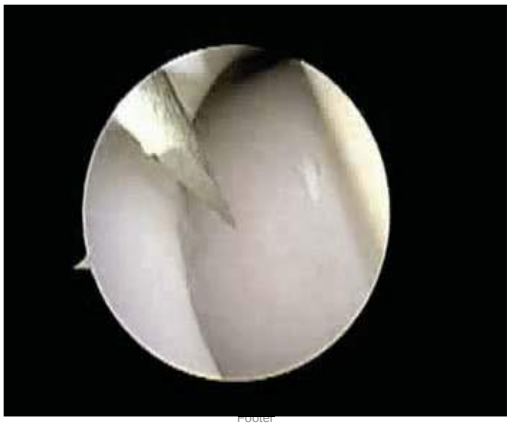
- Grade 0: normal cartilage
- Grade I: cartilage soft spot
- Grade II: minor surface tears- fibrillation
- Grade III: deep crevices-fissures
- Grade IV: full thickness



Grade II & III Art Cartilage Wear



Grade III & IV Art. Cartilage Wear

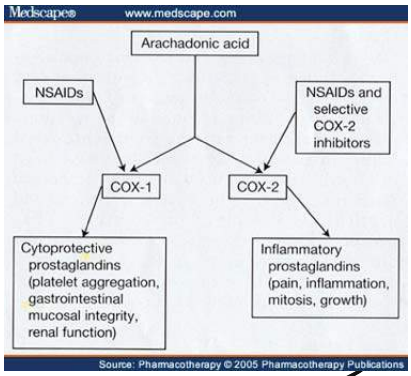


Medical Management of Osteoarthritis

- **Weight loss**
- NSAIDS
- Nutraceuticals
- Biologics/ Regenerative therapy
- Triamcinolone ?
- *Acupuncture/ herbs*
- *Nutrition*
- *Physical therapy*
- *Activity moderation*

NSAIDS
Mainstay of OA treatment

- Cox 1: expressed in GI tract
- Cox 2: sites of inflammation



Non-selective Cox 1 & 2 Inhibitors

- Carprofen (Rimadyl, Novox)
  - Twice daily dosing
  - Safe and effective
  - main side effects
    - Liver, GI

Selective Cox 2

- Deramaxx, Previcox, Metacam
- Once a day dosing
- Theoretically less GI side effects.

- *Anecdotally: We have seen more duodenal perforations with these NSAIDS
- **Avoid Aspirin with Deramaxx

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Non-Selective Cox 1 & 2, plus Lox 5

- Zubrin: Tepoxalin
  - once a day
  - dissolves in second
  - must keep dogs mouth closed x 4 sec.

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Nutraceuticals

- Glycosaminoglycan (GAG) + Chondroitin
- Fish oils (Omega 3)
- Others……
- Uncertain bioavailability
- Better for mild forms of OA

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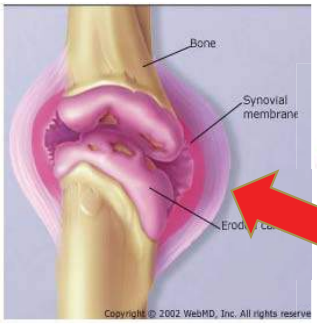
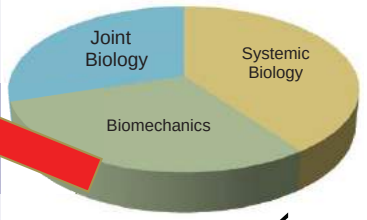
Can we cure osteoarthritis? NO





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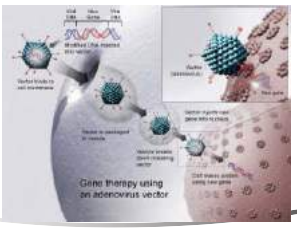
Can we modify osteoarthritis? YES



Biologics/ Regenerative medicine

- Adequan
- Hyaluronic Acid
- Platelet Rich Plasma (PRP)
- Stem Cells
- *Gene Therapy*
- *Cytokine & Growth Factor therapy*

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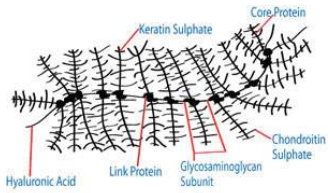
Adequan

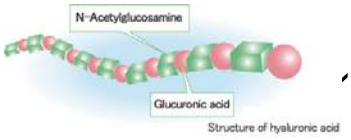
- PS Glycosaminoglycan
- Constituent of joint fluid
- Effect: Anti-inflammatory, joint fluid viscosity
- Dose regime: 5mg/kg
  - q 5-7 days x 4
  - q monthly
- Route: IM vs SQ
- Best for mild and moderate OA



Hyaluronic Acid

Hyaluronic Acid

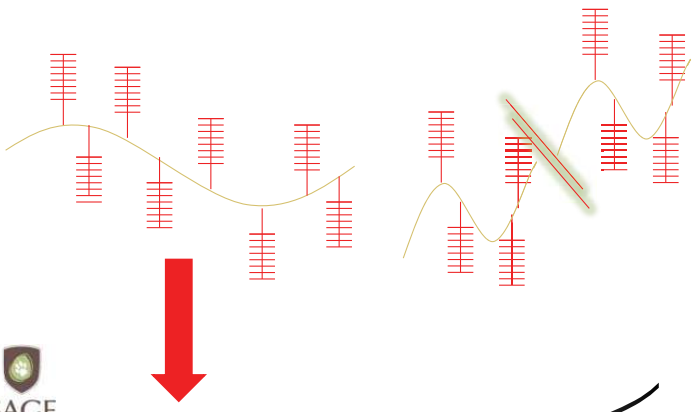
- Structure determined in 1950's
- Repeating disaccharide units
- N-Acetyl-D-glucosamine
- Synthesized in plasma membrane of cell
- Present in every tissue and body fluid



Structure of hyaluronic acid

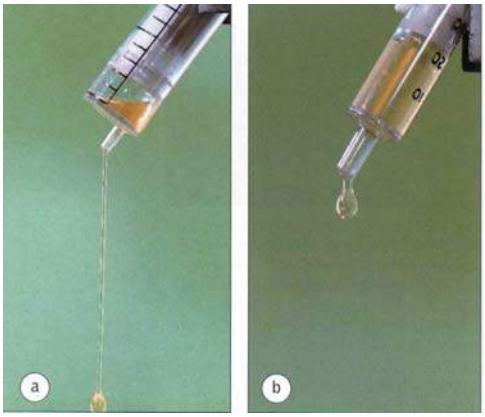
Cartilage

Effects of OA on Hyaluronic Acid

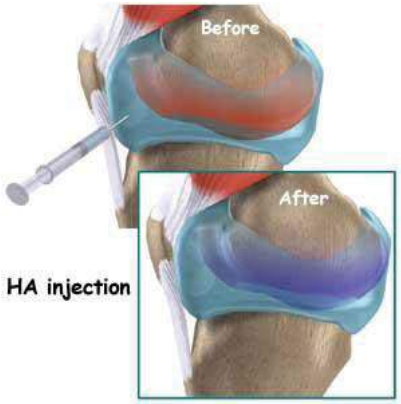


Goldberg 2010

Synovial Fluid

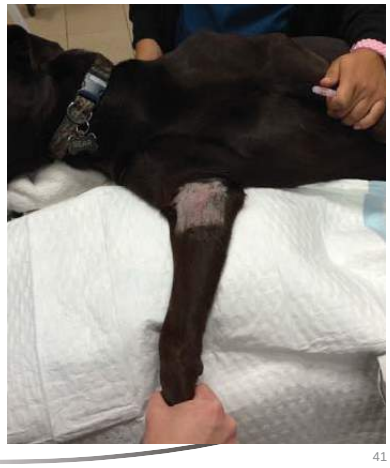


Joint Injection



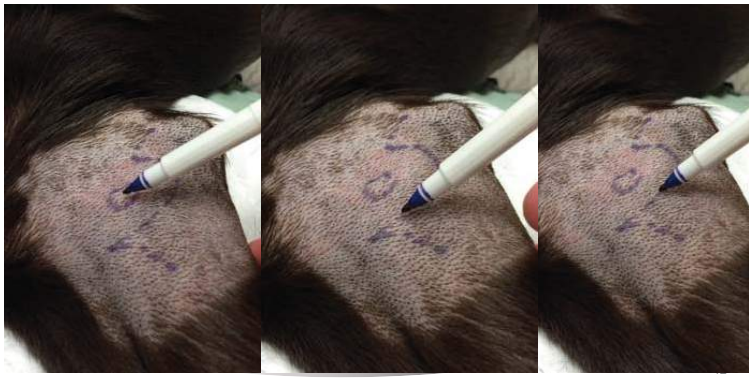
Patient Prep

- Dexdomitor +/- Torb
- Shave medial elbow
- Sterile Sx prep
- Support elbow
- Fulcrum- open jt.



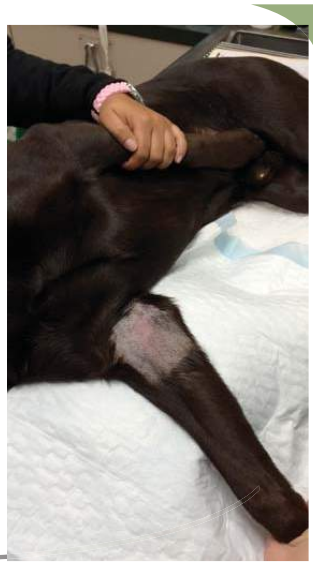
Medial elbow Joint Tap Anatomy

Medial Epicondyle | Medial coronoid | Safe Zone



Medial Elbow Joint Tap

- Palpate medial epicondyle
- Down 7-10mm
- Caudal 5-7 mm
- Introduce needle perpendicular to jt.
- Aspirate jt. fluid
- Injection of biologic agent

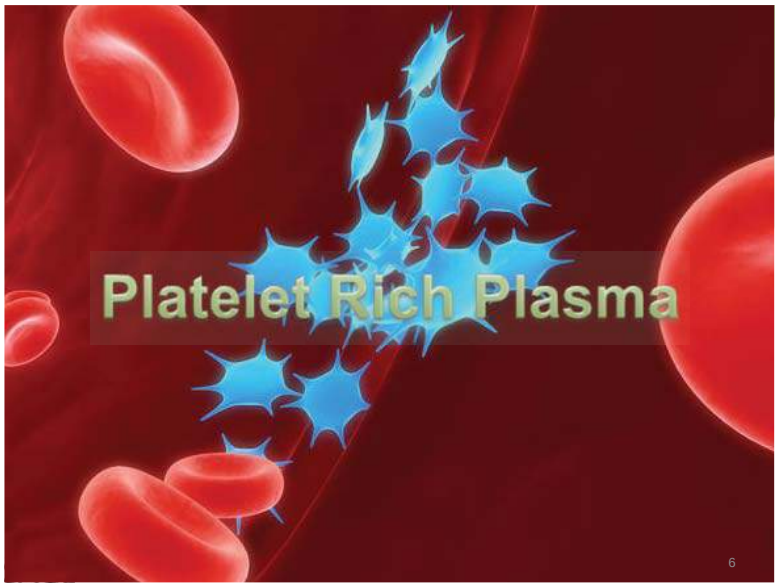


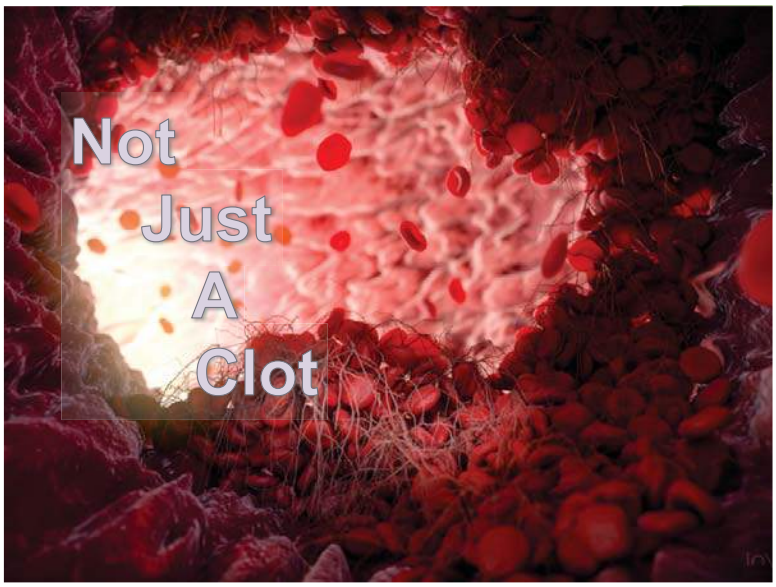
Hyaluronic Acid Modes of Action

- Anti-nociceptive effects
- Cellular effects
  - Enhance synthesis of extracellular matrix proteins
  - Mediates chondrocyte proliferation and function
  - Stimulates HA synthesis
- Inhibits production of inflammatory mediators
- Improved Cartilage health with decreased degradation

Hyaluronic Acid Protocol

- Hylartin V (Zoetis)
  - MW: 3 million Daltons
- Intra-articular injection at day 0, week 2 and week 6
  - Volume: 1-2 mls/ jt.
- Expected duration of effects: up to 3-4 months
- Will combine with triamcinolone depending on presentation (acute synovitis)





What is PRP?

- Platelet concentrated plasma
  - 1.3-3x + platelet concentration of whole blood
  - >1000 x 10_3/ ul
- Low RBC concentration
- Variable WBC concentration

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How PRP Works

- Alpha Granules: cytokines and growth factors
  - Angiogenesis
  - Cell differentiation
  - Cell proliferation
- Dense granules:
  - Serotonin
  - Histamine
  - Dopamine
  - Calcium
  - Adenosine

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Secretory Proteins~ 1500 Unique Proteins

- PDGF- platelet derived growth factor
- TGF - transforming growth factor
- VEGF - your endothelial growth factor
- FGF - fibroblast growth factor
- ECGF - epithelial cell growth factor
- IGF - insulin like growth factor's
- PDAF - platelet derived angiogenesis factor
- PDEGF - platelet derived endothelieal GF
- PF4 - platelet factor 4
- Oc - osteoclacin
- Fg - fibrinogen
- Vn - vitronectin
- Fn - fibronectin
- TSP-1- thrombospondin-1

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J. Cook- AVORE 2014

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Effects of PRP: Intra-Articular

- Inc. Chondrocyte and Matrix proliferation
- Increased Angiogenesis
- Increased HA
- Reduction in IL-1
- Resulting in:
  - Analgesia & Anti-inflammatory Effects
  - Suppression of OA Progression
  - Improved lameness & joint effusion

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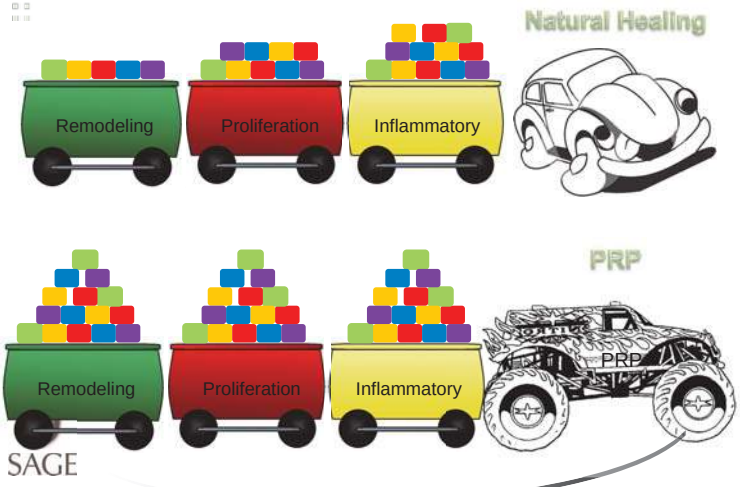

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But…… Is It Safe

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Safety

- No major side effects reported
  - Discomfort noted with elevated leukocyte numbers
- Autologous
  - Minimal reactivity risk
  - Minimal disease transmission risk
    - Non-sterile handling
- Good availability
- Antimicrobial

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How Do We Get Platelet Rich Plasma?

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Two Basic Theories

- Flow Cytometry:
  - analyse the physical and chemical characteristics of particles in a fluid as it passes through at least one laser.
  - Ability to determine WBC conc.

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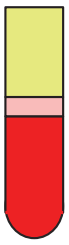
PRP Centrifugation



Platelet Poor Plasma

Platelets and white blood cells

Red Blood Cells

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Preparation Techniques

- Plasma:
  - Blood Volume: 10-15ml
    - jugular v.
  - PRP Volume: 4-7ml
  - Platelet conc.: 1.3-3x
  - WBC conc.: low

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Benefits to Joints

- Poor blood supply to cartilage and synovial fluid
- Self propagating inflammatory cycle
- Limited stem cell source

Limited healing capacity

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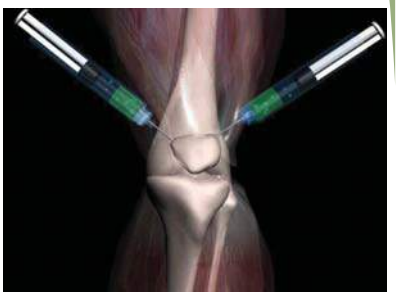
When do we use PRP

- Osteoarthritis- various joints
- Bone healing
- *Soft tissue healing
  - muscle & tendon



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- Protocol: injection at Day 0, 14 & week 6

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Summary

- PRP is an exciting new biologic therapy for orthopedic disease
  - widely used in human athletes
- Natural Autologous Product
- Most anecdotal evidence, need for double blinded, randomized trials in both human and vet med.
- Broad possible applications
  - Full indications unknown

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Stem Cell Therapy

- Promising addition to medical treatment of orthopedic diseases
- Stem Cells:
  - multipotent-mesenchymal (MSC's)
  - differentiate into tendon, ligament, bone, cartilage, cardiac, nerve, blood vessels, fat and liver

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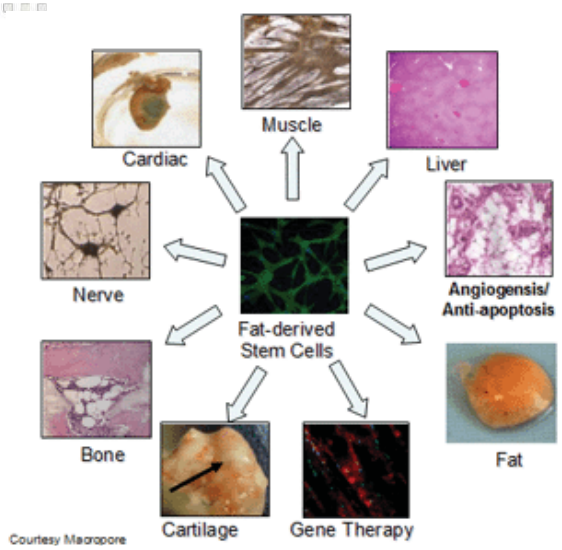

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Types of Stem Cells

» Embryonic Stem Cells

» Adult Stem Cells- Mesenchymal
  – Autologous adipose-mesenchymal stromal cells- Vet Stem
  – Bone marrow derived adult stem cells- Advanced Regenerative Therapies (ART)

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Vet Stem- Adipose Derived

» Readily available source- falciform fat
» Greater number of “cells” collected- not cultured
» Differentiation into multiple lineages
» Heterogeneous mixture of cells:
  – mesenchymal Stromal Cells, endothelial progenitor cells, pericytes, immune cells, fibroblasts
» Cost: ~$1470 plus shipping, collection and administration

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Advanced Regenerative Therapies-Bone Marrow Derived

» Cells cultured from bone marrow- millions of cells
» Regenerate: bone, cartilage, tendon, ligament, meniscus, intervertebral disc, fat, muscle and nerve
  – appears superior to fat-derived for musculoskeletal system (Human literature)
» Ease of collection- bone marrow aspirate
» Cost: $1200 plus shipping, collection and administration.

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Bottom Line on Stem Cell Therapy

» Most evidence with fat derived SC is anecdotal
» Most scientific studies have been performed on bone marrow derived SC (human and equine)
» Probably best used in repair of muscle and tendon
» Intent inflammatory environment within a joint or specific disease process
» Unlikely to regenerate cartilage without differentiation & changing the joint biomechanics
» Number of injections, intervals, duration of effect etc. is unknown

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Triamcilonone Acetonide

» Rapid acting corticosteroid
» Use for joint injections in humans- well documented
» Use for acute synovitis/ inflammatio
» Dose: 3mg/Jt. (10mg/ml)
» effect can last up to 4-6 weeks
» Not > 3-4 times/ year
» Often combine with HA

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Surgical Procedures elbow arthritis

» Sliding Humeral Osteotomhy (SHO)

» Canine Unicompartmental Elbow Arthroplasty (CUE)

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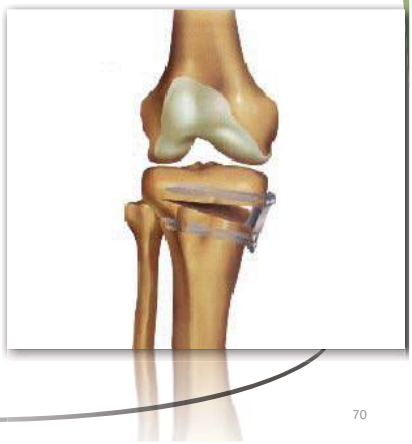
Sliding Humeral Osteotomy

- Novel procedure based on human “High Tibial Osteotomy”
- Translates weight bearing forces to the lateral aspect of the the joint
- Closing wedge vs “Slide”
- Locking plate technology



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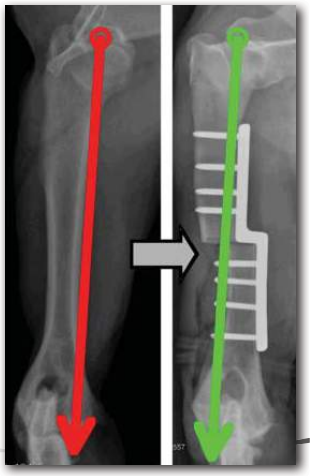
Shifts Mechanical Axis



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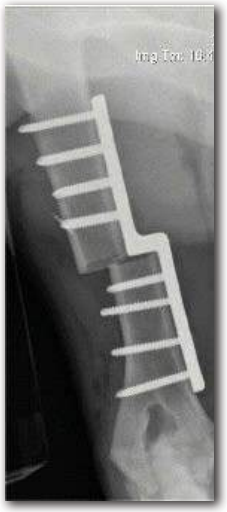

Post op

12 weeks po

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Proof of Principle

Pre-op

6 months post op

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Footer

N. Fitzpatrick

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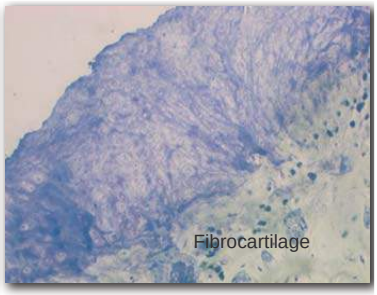
Histology



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Footer

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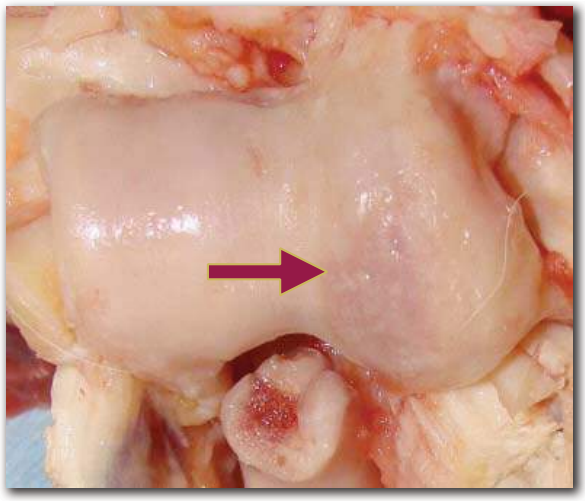

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Coal Roy

- 10 month, mn, lab
- Bilateral elbow dysplasia
- Grade III/V lameness
- Marked elbow effusion
- Bilateral FCP + OCD
- Painful ROM
- Litter mate also affected
- Partial response to NSAIDS

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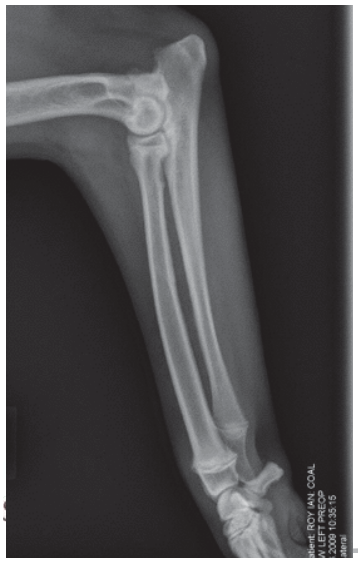

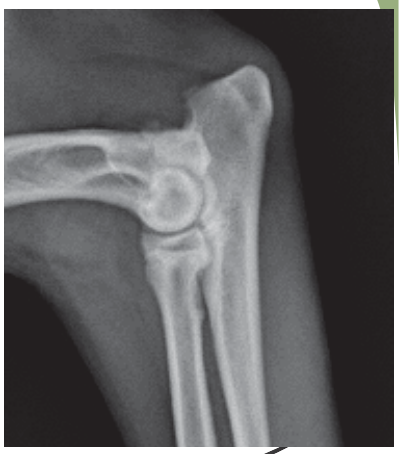

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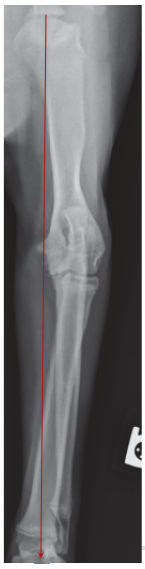

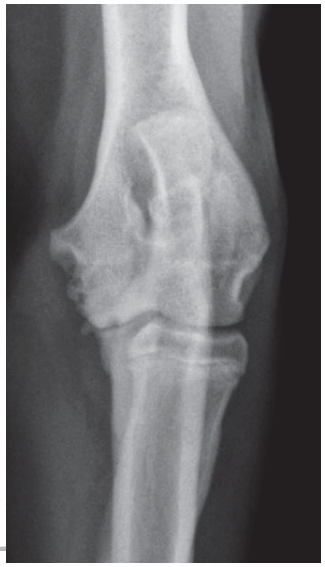

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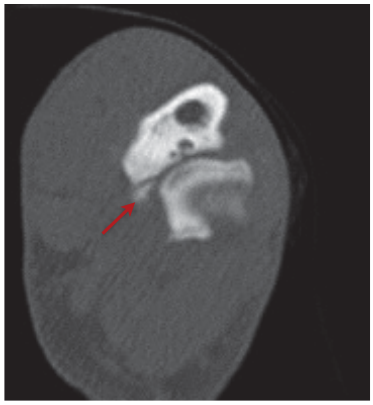
Coal Roy: Pre-op CT scan



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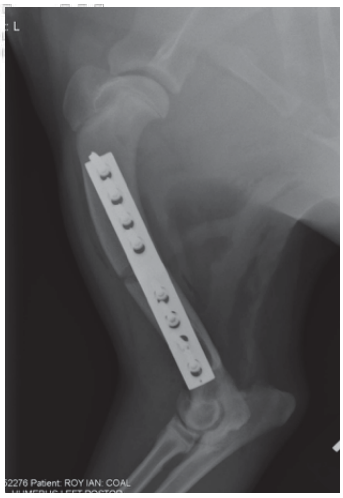

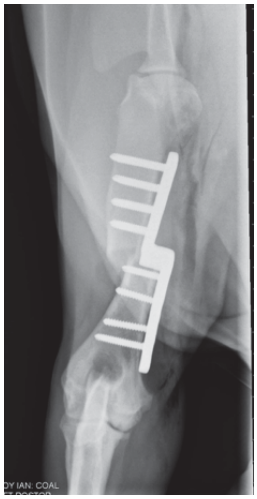

Post op

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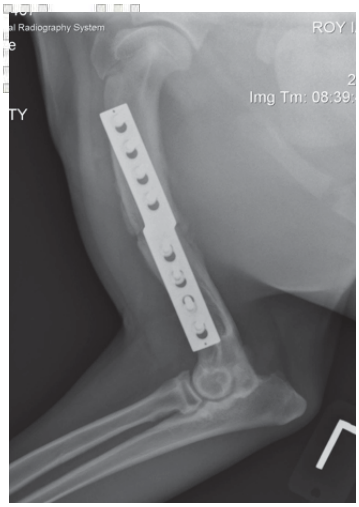
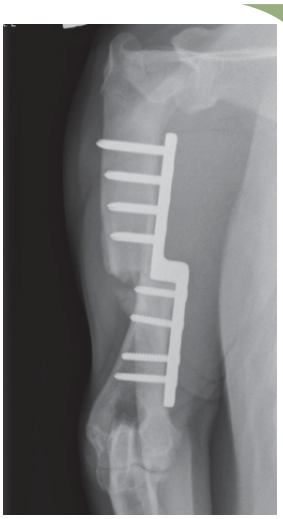
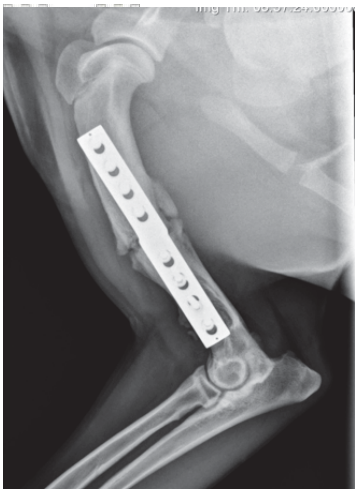
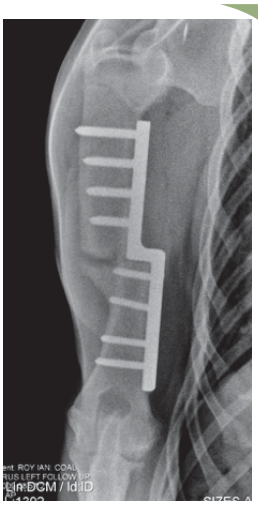




4 wks post op





8 wks post op

SHO- Outcomes

- Fitzpatrick, Noel. Vet Surg. Feb, 2009
- 56 limbs treated with SHO
- Lameness improve in all by 26 weeks
- Lameness resolved 21/32 limbs
- Complications 17.2%, 22.2% & 4.8%
- Two elbows: histology >12 months- fibrocartilage

Canine Unicompartmental Elbow Arthroplasy

CUE

Why?

It's black and white... ok, really RED and white

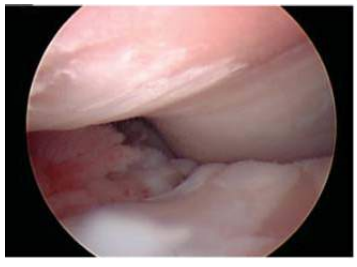
Medial Compartment Disease

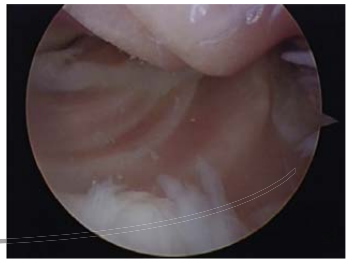
- Pain
- Lameness
- OA

Number of cases

Options & Outcomes

Total elbow failures





The Idea

Just fix what is broken

Human Uni Knees

Uni elbow concept (Schulz)

Total elbow & synthetic osteochondral grafting experience (Cook)

Arthrex expertise, resources & philosophy (Karnes)

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The Principles

Unicompartmental

Bone Sparing

Maintains Stabilizers

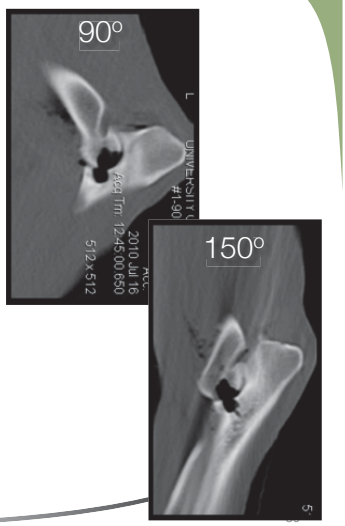
No Luxation

Cementless

Alignment & Kinematics not factors

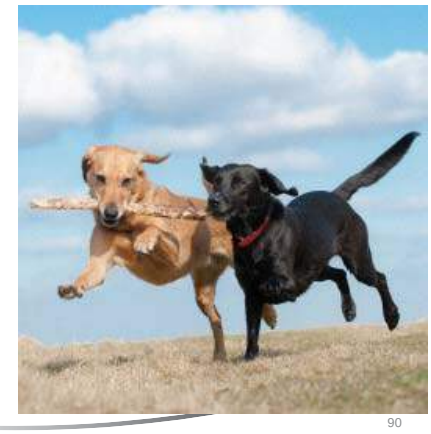
Contact through stance ROM

Relatively Simple & SAFE!!



CUE: Primary Goal

» **Safely** relieve pain and restore <u>full</u> function



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Providing a bearing surface

» Improve pain free range of motion

» Prevent additional joint collapse and cartilage-subchondral bone destruction

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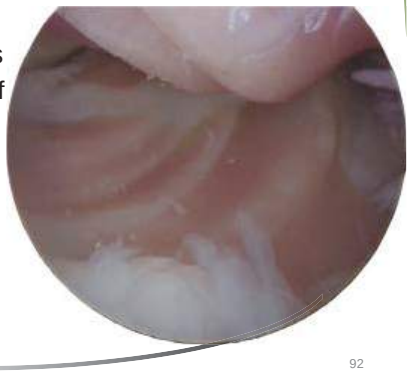
Elimination of bone on bone

» Remove stimulation of subchondral nociceptors

» Diminishes one cause of inflammation

» Enhances the opportunity for fibrocartilage in-growth



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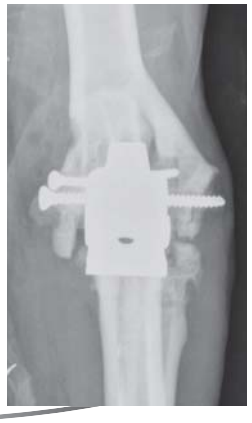
Maintain Natural Joint Stabilizers

» Eliminates the need for design complexity for stability

» Eliminates shear forces that lead to interface failure



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Implants

Humeral implant

- Cobalt-Chrome, CP Titanium bone in-growth surface (BioSync)

Ulnar implant

- UHMWPE
- Barbed/dimpled cylinder
- UHMWPE + CP Titanium bone in-growth surface (BioSync)

Sizes: Med. & Lrg.

Instrumentation

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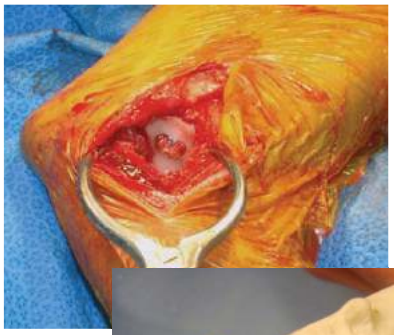

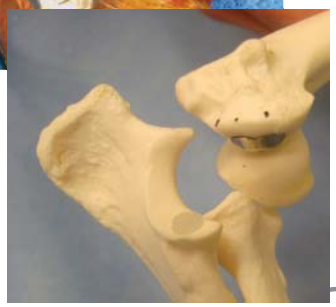

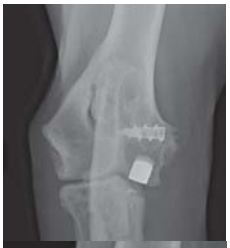

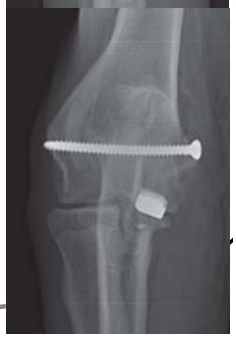

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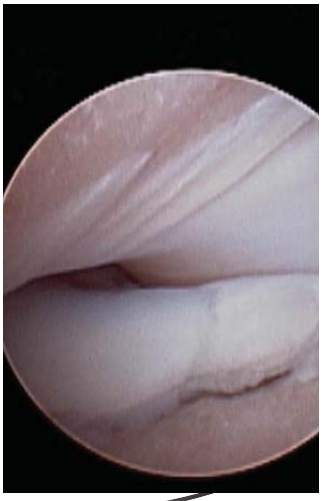
Forest Gilman

- 8 Month, mn, Lab
- Severe bilateral FCP + OCD
- Arthroscopic removal and debridement
- Grade IV art.
- Poor post operative function
  - limited to 5 minute walks
  - chronic medical management



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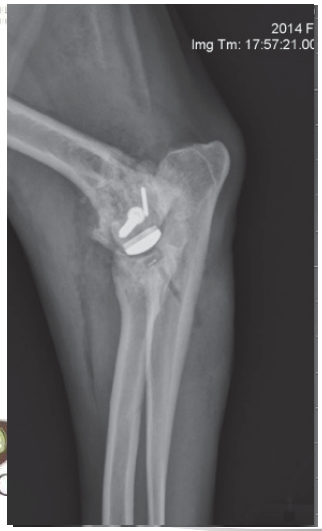

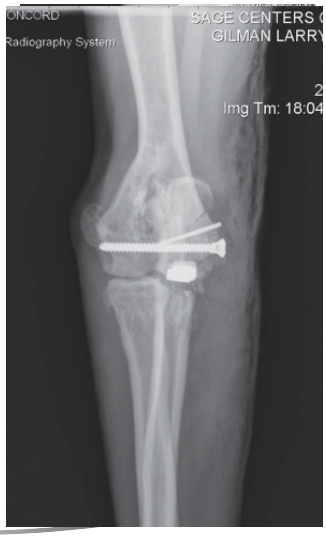

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- "Patsy and I just wanted to let you know how well Forest is doing 6 months post his second CUE. He's been off Carprofen now for 3 weeks after being on it for almost all of his 4 years. It's truly amazing! We would have had to put him down if not for the CUE procedure and your surgical skills. Patsy and I can't thank you enough"
- Patsy and Larry Gilman, DVM

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Clinical outcomes associated with the initial use of the Canine Unicompartmental Elbow (CUE) Arthroplasty System

- Long term outcomes: Function & complications
- 103 cases, 18 surgeons
- Follow-up: 6-47 months, median 10 months
- Complications:
  - 1 catastrophic (1%)
  - 11 major (10.7%)
  - 28 minor (27.2%)
- Oucomes:
  - Full function: 49 (47.6%)
  - Acceptable: 45 (43.7%)
  - Unacceptable: 9 (8.7%)

*Can Vet J 2015;56:971-977

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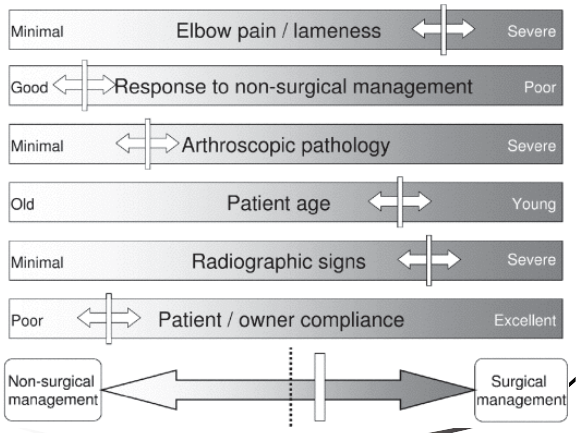
WWCD

Treatment of varying degrees of Elbow OA

101

Treatment Options: Sliding Scale Analogy



102

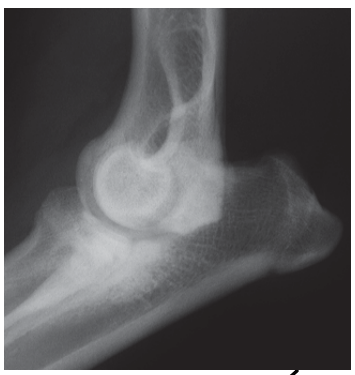
Early FCP, Minimal OA

- Arthroscopic FCP removal + axial SMC
  - NSAIDS + Tramadol- short term
  - +/- Adequan
  - +/- Biologics (HA)
  - Nutrition

103

Moderate OA

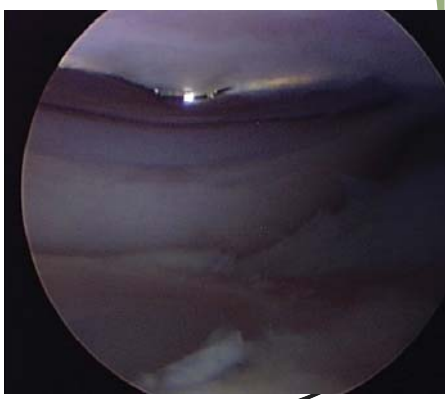
- Lameness & effusion
- Weight management
- Nutrition-meat based or raw
- NSAIDS + Tramadol, Gabapentin etc
- Adequan- very important
- Joint injections:
  - HA
  - PRP
  - Triamcinolone (acute synovitis)



104

Advance or End-stage OA

- Same medical management as moderate OA
- Set realistic expectations
- Consider SHO or CUE procedure



105

Questions???

Antiepileptic Drugs – a panel discussion

Christina Vitale, DVM, Diplomate ACVIM Neurology (Campbell)
David Raczek, DVM, Diplomate ACVIM Neurology (Campbell)
Starr Cameron, BVetMed, Diplomate ACVIM Neurology (San Mateo)

When to start an AED

- Some general guidelines
  - High frequency (varies amongst neurologists, but in general seizures more often than every 1-3 months)
  - Trend towards increasing seizure frequency or severity
  - Known or strongly suspected intracranial disease

Which AED to choose

- **There is no single "best" AED.** The drugs that we use commonly all have good reported efficacy. Each has an individual set of advantages and disadvantages and trying to match these with the individual pet/family is the best way to decide which drug to chose.

Basics of commonly used AEDs

All AEDs have drowsiness and hind end weakness as potential side effects. Some dogs may have a more or less significant reaction to a given drug than other dogs.

- **Phenobarbital**
  - Dose 2-3 mg/kg q. 12 hours
  - Loading dose may be indicated in status epilepticus/severe clusters (takes two weeks to get to steady state with maintenance therapy). Loading protocols vary, usually 16 mg/kg, often divided to avoid severe depression of mentation and respiratory function.
  - Advantages
    - Inexpensive (however the follow up lab work also needs to be taken into consideration)
  - Potential adverse effects/disadvantages
    - Hepatotoxicity
      - Most common at higher doses/blood levels (> 30 mcg/mL) and longer durations of therapy. Recommend evaluation of blood levels +/- liver chemistries 2 weeks after instituting therapy and every 6 months long term.
    - Bone marrow suppression
      - Relatively uncommon. Recommend CBC every 6 months long term +/-2 weeks after instituting therapy.
    - Polyphagia and polydypsia
    - Controlled substance
  - In general, this may not be a good choice for patients that are already overweight, for clients that are not likely to follow up with lab work monitoring, or for very young patients that will likely have a long duration of therapy.
- **Potassium Bromide**

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- Dose 30-40 mg/kg q. 24 hours
- Loading dose often needed (takes 3-4 months to get to steady state with maintenance therapy). Loading dose 100 mg/kg per day (usually divided into BID dosing) for 4-5 days.
- Advantages
  - SID dosing
  - Veterinary approved formulation
  - Available in liquid (easy dosing for small patients and incremental dose changes) chew tabs
  - Inexpensive
- Potential adverse effects/disadvantages
  - Bone marrow suppression
    - Relatively uncommon. Recommend CBC every 6 months long term.
  - Pancreatitis
    - Relatively uncommon. Routine testing for this not recommended, rather only if clinical signs arise.
  - Not to be used in cats due to potentially fatal respiratory effects
  - Bromide induced skin lesions
  - Bromide levels fluctuate based upon chloride intake so need to avoid diet changes and dietary indiscretions (salty foods, ocean water)
  - Long half life means a long time to get to steady state without loading and a long time for dose changes to take effect.
  - Polyphagia and polydypsia
- In general this may not be the best choice for a dog that is already overweight. Not ideal if you want to reach steady state quickly.

- **Levetiracetam**
  - Dose 20 mg/kg PO q. 8 hours
  - Advantages
    - Comes in liquid (easy dosing for small patients and incremental dose changes), tablets, and injectable formulations.
    - No adverse effects to other organ systems
  - Potential adverse effects/disadvantages
    - 8 hour dosing interval makes this impractical for many clients
    - Polyphagia and polydypsia
  - There is an extended release formulation. The dose is 30 mg/kg q. 12 hours. The data on efficacy of this formulation in dogs and cats is still new. Some pharmacokinetic studies indicate it may be effective, however anecdotally this is not always the case. The tablets cannot be split and only come in relatively high doses, so this limits use in all but large breed dogs.
  - In general this is a very good option for clients that can reliably give q. 8 hour medications.
- **Zonisamide**
  - Dose 5-10 mg/kg PO q. 12 hours (start at 10 mg/kg in pets already receiving phenobarbital)
  - Advantages

        - Minimal chance of adverse effects to other organ systems (rare reports of hepatotoxicity)
    - Potential adverse effects/disadvantages
        - Available capsule size (100 mg is largest) makes dosing and costs in large dogs inconvenient
        - Can decrease appetite, especially at higher doses
    - In general this is a good option for many pets, but may not be the best choice in a pet that is already a picky eater or in very large dogs.

My Dog is Walking Wonky

David Raczek, DVM, DACVIM (Neurology)
Andy J. Staatz, MS, DVM, DACVS

My dog walks wonky – a comparative neurologic and orthopedic approach to atypical gaits is a presentation of a series of cases. Each case is presented as it was presented to us. In most cases it was unclear whether the abnormality noted was neurologic or orthopedic in nature. Signalment and history are described. A video of the patient walking is presented at normal and slow motion speeds along with parts of our exam. Our goal is a shared discussion of suspected diagnosis and formulating next steps. The cases are followed out to final diagnosis. Listed below is the starter information for each case. Come enjoy an hour of real life case solving.

CASE #1: Kipper, 6 year f/s Wheaten Terrier

- Two week history of hind limb weakness with splaying on smooth floors
- Neurologic and orthopedic abnormalities on examination
- See video for gait
- Orthopedic or Neurologic??

CASE #2: Taylor, 7 year m/n Sheltie

- Slow to move, Difficulty rising or sitting
- Six months after onset, owner video clarified clinical signs and helped guide towards a diagnosis.

CASE #3: Peggy, 7 year f/s Dachshund

- Prior history of paraplegia and hemilaminectomy with good recovery
- Two month history of pain and difficulty walking
- Watch Peggy walk…

CASE #4: Lilly, 11 year f/s Labrador

- Prior history of elbow dysplasia and TPLO
- Four week history of difficulty on stairs and forelimb stiffness
- Video of Lilly's gait and exam will quickly clarify the nature of her signs

CASE #5: Donald, 10 year m/n Miniature Schnauzer

- Left pelvic limb lameness treated with FHO and poor post-op recovery

CASE #6: Cash, 8 year m/n Pitbull

- Two to three year history of abnormal gait with slow progression
- Likely diagnosis is evident from history and exam…

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Track 2

Subtle Hints of Common Hidden Dental Disease That May be Missed in Practice

SAGE Symposium – March 20, 2016

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INTRODUCTION

It is impossible to describe all the dental and oral pathology and pathophysiology in a single set of notes. Instead, the lecture and notes will focus on a visual tour of common oral pathology and subtle visual and examination clues indicating hidden subgingival disease.

CONSCIOUS EXAM

The oral examination begins in the exam room. A complete medical and oral history, general physical exam, and conscious oral examination are necessary. A complete history and evaluation of the chief complaint is investigated. Questions such as, but not limited to, onset, duration, environment, chew toys, oral health care, current medications, diet, past illness, past anesthetic episodes, behavioral changes, etc. are explored. Many patients with oral disease do not have obvious clinical signs. When the diseases become unbearable for the patient they may exhibit ptyalism, face rubbing, halitosis, partial anorexia, sneezing, nasal discharge, pawing at the mouth, ocular discharge, or nothing at all (suffer quietly in silence).

Temperature, pulse, respiration, body weight, and the organ systems are evaluated. Particular attention to the cardiopulmonary system is made. The maxillofacial skeletal is palpated and the eyes retropulsed. The three basic skull types are brachycephalic (e.g., Pugs, Bulldogs, Persian Cats), mesocephalic (e.g., Labrador, DSH), and dolichocephalic (e.g., Sight hounds, Collies). The regional lymph nodes and salivary glands are palpated. Facial symmetry and occlusion are noted. The range of motion of the temporomandibular joints should be palpated and the patient observed for pain and/or difficulty in opening and closing the mouth. The lips and mucocutaneous junctions should be observed for ulcerations that might indicate an autoimmune disease. Finally, the dentition is evaluated and the teeth counted to determine if all teeth are present. Discolored teeth, persistent deciduous teeth, root and furcation exposure, oral mucosal lesions, sinus tracts, tongue, oral masses, plaque and calculus are noted. Note the symmetry of the head.

The occlusion should be evaluated in the non-anesthetized patient so that the relationships of the teeth and bones can be determined before an endotracheal tube is placed. The normal canine mesocephalic skull has an anisognathic mandible. With orthocclusion, the mandibular incisors occlude on the cingulums of the maxillary incisors, the mandibular canines interdigitate, without

touching, between the maxillary third incisors and canine teeth. The tips of the mandibular 4th premolars will point directly upward between the maxillary 3rd and 4th premolars. The mandibular and maxillary premolars interdigitate, and the tips of the upper and lower second premolars are at the same horizontal level.

Appropriate pre-anesthetic blood testing is obtained based on patient signalment and history. No patient is anesthetized without minimal blood work (packed cell volume, BUN, glucose, and total protein). However, most dentistry patients are older and more extensive bloodwork such CBC / Chemistry Panel / Urinalysis are recommended.

ANESTHESIA AND THE ORAL EXAMINATION

It is impossible to completely examine the oral cavity in a conscious patient. At best, we can estimate the extent of periodontal disease, identify fractured teeth, and see obvious large oral masses. Anesthesia is **required** (www.avdc.org for AVDC position statement) to accurately assess periodontal disease, fractured teeth, and other oral pathology.

MISCELLANEOUS PATHOLOGY WITH SUBTLE HINTS OF HIDDEN DENTAL AND ORAL DISEASE

I. Hidden palatal maxillary canine oronasal fistula

An oronasal fistula (ONF) is a communication between the oral and nasal cavity. The epithelial surfaces of the nasal and oral cavity communicate via the fistula. A loss of the maxillary canine tooth and remaining defect results in a clinically obvious oronasal fistula. However, deep intrabony palatal pockets are missed during clinical examination and due to summation of opaque radiographic structures, is often missed with intraoral dental radiographs. Bacteria and the associated inflammatory response only require microscopic communications between the oral and nasal cavities to create a hidden chronic oronasal fistula resulting in upper respiratory clinical signs. Clinical signs may include chronic nasal discharge (serous, mucopurulent, and/or epistaxis). Sneezing may or may not be present. Oronasal fistulas may be obvious upon clinical examination or may be a pinpoint lesion that requires anesthesia and a thorough oral exam to identify. The teeth may, or may not, be mobile and it is not uncommon to have normal or slightly increased to moderate buccal periodontal probing measurements with very large probing depths palatally. Occasionally, a trickle of blood may be seen exiting from the ipsilateral nares when the probing depths are measured palatally, confirming an ONF. Occasionally, the periodontal probe is inhibited from reaching true probing depths by large accumulations of subgingival calculus and inflammatory tissue.

Surgical extraction and appropriate tension free closure with mucoperiosteal flaps is necessary to correctly treat the oronasal fistula during the surgical procedure.

II. Parulis

A parulis is a raised nodule at the opening of a draining sinus tract. If the parulis is located apical to the mucogingival junction it is often associated with endodontic disease. If the parulis is located at/or coronal to the mucogingival junction, it is often associated with periodontal disease. Intraoral dental radiographs and extraction or endodontic treatment, as indicated, is required.

III. Maxillary 1st and 2nd molars in small breed dogs

The maxillary 1st and 2nd molars in dogs may have minimal clinical probing depths but be mobile during clinical examination. The intraoral radiographs may identify a very wide or absent periodontal ligament space/large palatal root periapical lucency. This is consistent with severe periodontal disease and surgical extraction and closure of the extraction site is required.

IV. Increased probing depths dog mandibular 1st molars and mesial roots maxillary 4th premolars

Large periodontal probing depths may be identified mesial or distal to the mandibular 1st molars in dogs with minimally associated gingival inflammation. Intraoral radiographs will identify large intrabony pockets. Treatment may include extraction of the adjacent non-strategic 4th premolar or 2nd molar, if these teeth are associated with the intrabony pocket, and open root planning and bone augmentation or guided tissue regeneration. If the mandibular molar cannot be saved, then surgical extraction is necessary.

When probing the teeth always probe between them mesial buccal and mesial palatal roots of the maxillary 4th premolars. This is a common place for a hidden intrabony pocket that is not easily identified with intraoral radiographs due to summation and superimposition of radiopaque dental structures and bone. Deep intrabony pockets will require guided tissue regeneration or the tooth will require surgical extraction.

V. Feline mandibular 1st molar periodontal disease

Brachycephalic cats often have a scissors or level occlusion of the incisors. However, the mandibles have bowed laterally during growth. As a result the central cusp of the maxillary 4th premolar contacts the mesial/buccal tooth and periodontium of the mandibular 1st molars resulting in periodontal dehiscence and disease. Likewise, the veterinarian may extract the lower 1st molar and identify the site is not healing and/or identify a mass pre or post extraction that has a histological description such as pyogenic granuloma, lymphoplasmacytic gingivitis, etc. secondary to the trauma created by the maxillary 4th premolars (see feline oral pyogenic granuloma following).

Surgical extraction of a periodontally expired mandibular 1st molar is necessary. The maxillary 4th premolar requires surgical extraction or appropriate crown reduction and endodontic and restorative treatment to remove the offending cusp(s).

VI. Ocular discharge and the nasolacrimal canal

The nasolacrimal canal is located millimeters from the apical aspect of the maxillary canine tooth, particularly in cats. Chronic endodontic disease with the associated periapical infection and inflammation can occlude and damage the nasolacrimal canal resulting in impaired drainage and epiphora.

Assessment, intraoral radiographs, and surgical extraction or endodontic treatment of the maxillary canine tooth is necessary. However, permanent damage may preclude complete resolution of the epiphora.

VII. Cyclosporine related gingival enlargement/hyperplasia

The use of cyclosporine for atopic dermatitis has greatly increased the prevalence of drug induced gingival enlargement in dogs. A combination of the plaque, drug dosage, and individual susceptibility results in the creation of pseudopockets that lead to true periodontal pockets with chronic infection, pain, and tooth loss. Finding the lowest possible dose to maintain control of the dermatological condition but minimize the gingival enlargement is recommended. Annual to semi-annual dental cleanings and daily home care with brushing to control the plaque is recommended. Intraoral radiographs and extractions are necessary for teeth that have progressed to late stages of irreversible periodontal disease.

VIII. Feline sublingual squamous cell carcinoma

Cats will present late in the disease course for partial or complete anorexia, ptyalism, and oral pain. Biopsy and histopathology are necessary for diagnosis because differentials that may appear clinically similar include treatable lesions such as eosinophilic granuloma or a granuloma/infection associated with a sublingual foreign body (e.g., needle, string, plant material).

IX. Feline Oral Pyogenic Granuloma

An inflammatory mass often located in the caudal and buccal caudal aspect of the feline oral cavity secondary to focal infection and trauma can be found in association the mandibular first molar. Traumatic contact from the ipsilateral maxillary 4^{th} premolar and 1^{st} molar cycles continued inflammation in the site. Traumatic occlusion related to breed (i.e., brachycephalic), poor surgical technique (e.g., closure of the molar salivary gland more buccal in position), and focal periodontal disease and inflammation/edema resulting in swelling and occlusal contact may result. The lesions have endothelium lined vascular spaces, inflammatory infiltrates, ulceration, and granulation tissue with proliferating fibroblasts. Surgical excision and debridement with surgical extraction of the ipsilateral contacting dentition (4^{th} premolar/1^{st} molar) or odontoplasty and dental sealant are necessary to treat and prevent recurrence of the lesion. If recurrence occurs despite odontoplasty, then surgical extraction is recommended.

X. Maxillofacial swellings and draining tracts

Maxillary draining tracts should be investigated for odontogenic infections such as periodontal disease or endodontic disease prior to extensive dermatological or neoplastic work ups including advanced imaging and biopsy. Teeth should be the primary differential for the maxillofacial swellings and draining tracts. The pathology is easily diagnosed with an appropriate anesthetized examination and intraoral radiographs, if the veterinarian knows the knowledge of the pathophysiology. If an odontogenic infection is not the cause, then evaluation for neoplasia, etc. can be pursued. Often if it is neoplasia, a tooth is involved and surgical extraction and deep biopsy via the extraction site will provide a histological diagnosis.

XI. Deciduous tooth fractures

Deciduous tooth fractures can lead to endodontic disease, damage to developing tooth buds, and maxillofacial swellings. Complicated crown fractures (exposed pulp) require extraction and a "wait for them to exfoliate with adult tooth eruption" is incorrect and potential malpractice.

XII. Uncomplicated crown fractures

Dentin contains 30 000 – 70 000 tubules/mm^2 allowing oral bacteria to translocate into the endodontic system and result in pulpitis and death of the tooth. The clinical point is that exposed pulp (complicated crown fracture) always leads to endodontic disease but ALSO uncomplicated crown fractures and enamel fractures exposing dentin tubules can lead to endodontic disease.

XIII. Non-healing extraction sites

All extraction sites, except some deciduous tooth extractions, should be sutured closed. If correct surgical closure was performed (e.g., no tension on mucoperiosteal flaps, suture lines over bone) and the surgical site does not heal, the differentials immediately include neoplasia or retained tooth root. Ideally, intraoral radiographs post-extraction would have confirmed the entire tooth was extracted. However, if they were not obtained then anesthesia, intraoral radiographs, remove a tooth root if present, or if not, obtain representative biopsy of the site followed by a large mucoperiosteal flap for closure.

XIV. Linguoversed Mandibular Canine Teeth

Linguoversion of the deciduous mandibular canines (704 and 804) traumatize the palatal tissue and cause an adverse dental interlock. The dental interlock interferes with jaw growth. It is recommended to extract 704 and 804 to allow the patient the best chance of normal mandibular and maxillary growth and normal occlusion.

Adult linguoversed canine teeth are a result of retained corresponding deciduous teeth or a developmental defect. This includes a class 2 malocclusion, brachygnathic mandible, excessive

anisognathism, or retained primary mandibular canines. Linguoversion of 304 and 404 can lead to severe damage to the hard palate, oronasal fistulas, periodontal defects, tooth damage to 103, 104, 203, and 204, and inability to close the mouth. Local periodontal disease and oronasal fistulas can develop in the traumatized maxillary arcade. Treatment options include orthodontic movement, crown reduction and partial coronal pulpectomy with a direct pulp cap, crown reduction and total pulpectomy, or extraction of the offending mandibular canine tooth. Surgical extraction of the adult mandibular canine teeth is rarely performed since the teeth are strategic and the procedure is more traumatic and destabilizing compared to an endodontic procedure. Orthodontic movement is successful. Depending on the severity of the linguoversion, the owner's commitment, and compliance of the patient, various techniques have been discussed for moving 304 and 404. Treatments can consist of removable orthodontic devices (ball), direct acrylic incline planes, indirect acrylic or metal inclined planes, active expansion screws, vital pulpotomy, or extraction.

XV. Dentigerous Cysts

Unerupted teeth (embedded or impacted) can lead to ***dentigerous cysts*** formation and destruction of the bone and adjacent teeth. This condition is preventable so all regions of missing teeth should be evaluated with intraoral radiographs. Unerupted teeth should be extracted. Dentigerous cysts need to be surgically debrided and the cystic lining removed with the offending tooth.

XVI. Complicated Crown Fractures

Intraoral dental radiographs for assessment and treatment are required. **All fractured teeth with pulp exposure (acute or chronic) require endodontic treatment or extraction.** Many teeth with uncomplicated crown fractures and enamel fractures may also have endodontic disease requiring treatment that can only be found via intraoral radiographs.

Classification of tooth fractures can be found at www.avdc.org (nomenclature). Enamel infraction (an incomplete fracture of the enamel without loss of tooth substance), enamel fracture (a fracture with loss of crown substance confined to the enamel), uncomplicated crown fracture (a fracture of the crown that does not expose the pulp), complicated crown fracture (a fracture of the crown that does expose the pulp), uncomplicated crown-root fracture (a fracture of the crown and root that does not expose the pulp), complicated crown root-fracture (a fracture of the crown and root that does expose the pulp), and a root fracture (a fracture involving the root). Uncomplicated crown fractures may lead to the death of the tooth by translocation of bacteria and toxins across exposed dentin tubules or the force that fractured the tooth (concussive pulpitis). Complicated and uncomplicated crown root fractures may lead to periodontal disease since the normal anatomical structures of the subgingival periodontium are altered.

XVII. Non-vital Teeth

Localized intrinsic staining is consistent with a non-vital tooth. Total or partial pulp necrosis was found in 92.2% of intrinsically stained teeth. Radiographic signs consistent with endodontic disease were absent in 42.9% of the teeth. The intrinsic stain is the result of pulpitis and pulp

hemorrhage resulting in hemoglobin and the subsequent breakdown products in the dentin tubules.

Often the patient suffers quietly in silence with only subtle clinical signs of chronic pain being noticed by an astute owner. Clients often remark the improved change in behavior following treatment of a non-vital tooth.

CONCLUSION

Clearly the depth of 1-hour speaker notes cannot cover all the pathology and diseases that may be identified. However, the aforementioned notes highlight some common pathology that is common and frequently missed and not treated in private general practice.

Understanding and Treatment of Periodontal Disease Beyond the Dental Prophylaxis

SAGE Symposium – March 20, 2016

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INTRODUCTION

The tooth is composed of organic and inorganic material. The 3 hard (mineralized) tissues of the tooth include the enamel (crown only), dentin (root and crown) and cementum (root only). The cementum and enamel meet at the ***cementoenamel junction (CEJ)***. A tooth is a living structure and dentin is continually produced throughout the life of an animal in a vital tooth. Odontoblasts produce dentin and are located in the pulp with the blood vessels, lymphatics, and nerves. The pulp (endodontic system) is divided into the root (pulp) canal (in the root), the pulp chamber (in the crown), and the pulp horns (in the cusps of the crown).

The tooth is anchored in the jaws by the periodontium. The incisive bones, maxillary bone, and mandibular bone anchor the teeth. The ***periodontium*** consists of the 1) gingiva, 2) alveolar bone, 3) periodontal ligament, and 4) cementum.

Periodontal disease is the loss of the periodontal attachment; loss of the periodontium. Since 75% of these named structures are identified below the soft tissues of the oral cavity (gingiva, alveolar mucosa, and palatal mucosa), a thorough clinical subgingival evaluation and intraoral radiographs are required to assess, diagnose and treat periodontal disease. General anesthesia is required (www.avdc.org).

PATHOPHYSIOLOGY OF PERIODONTAL DISEASE

Periodontitis is active inflammation of the periodontium. It begins with the accumulation of the dental pellicle (e.g., salivary glycoproteins) that occurs within seconds of a tooth being cleaned. Within hours, first colonizing oral bacterial colonize the pellicle and the plaque biofilm is formed. The plaque biofilm matures within days. Gingivitis (inflammation of the gingiva) is the first clinical sign of the starting inflammatory cascade. Mineralization of the plaque biofilm results in calculus (tarter). Periodontal disease is caused by the bacterial biofilm (plaque) and the associated host inflammatory response. Significant periodontal disease can be present without calculus. Calculus is not the cause of periodontal disease.

As the plaque biofilm matures, early bacterial colonizers, gram-positive aerobic cocci, become less predominant. The biofilm shifts to gram-negative anaerobes and spirochetes located more apical in the periodontal pockets. Bacterial products such as ammonia, volatile sulfur

compounds, and proteolytic enzymes contribute to the destruction of the periodontium. The host inflammatory response, matrix metalloproteinases that degrade collagen of the periodontal ligament, elastase (break down collagen and elastin), and prostaglandins (PGE_2) are directly responsible for tissue damage and/or stimulate osteoclastic bone resorption (PGE_2, IL-1β, TNF-α). The calcium carbonate in the saliva of cats and dogs combines with the plaque to form calculus. Calculus increases surface area for bacterial attachment and can mechanically disrupt and damage the gingiva.

CLINICAL SIGNS OF PERIODONTAL DISEASE

The clinical signs of periodontal disease are often hidden and insidious. Halitosis, gingivitis, supragingival plaque and calculus, reluctance to chew, head shyness, pawing at the mouth, dropping food, sneezing, nasal discharge, are clinical signs. Unfortunately, many of those clinical signs require astute client observation and/or careful questioning from the clinician. Most commonly, there may be no obvious clinical signs to the owner and untrained veterinarian. Almost all the patients are still eating. American Animal Hospital Association Dental Guidelines and Canine and Feline Life Stages Guidelines recommend annual evaluations of the oral cavity. The recommended time to start professional evaluations and cleanings, in order to prevent disease, is in first years of life.
(http://www.aahanet.org/PublicDocuments/Dental_Guidelines.pdf)

DIAGNOSIS OF PERIODONTAL DISEASE

General anesthesia, professional examination, periodontal probing, charting, and intraoral radiographs are all required to successfully diagnose and treat periodontal disease.

Conscious Oral Examination

Periodontal assessment begins in the examination room with the client and the conscious patient. A complete medical and oral history, general physical exam, and conscious oral examination are necessary. Questions such as, but not limited to, onset, duration, environment, chew toys, oral health care, current medications, diet, past illness, past anesthetic episodes, behavioral changes, etc. are explored. Many patients with oral disease do not have obvious clinical signs.

The maxillofacial skeletal is palpated and the eyes retropulsed. The three basic skull types are brachycephalic (e.g., Pugs, Persian Cats), mesocephalic (e.g., Labrador, DSH), and dolichocephalic (e.g., Sight hounds). The regional lymph nodes and salivary glands are palpated. Facial symmetry and occlusion are noted. The range of motion of the temporomandibular joints should be palpated and the patient observed for pain and/or difficulty in opening and closing the mouth. The lips and mucocutaneous junctions should be observed for ulcerations that might indicate an autoimmune disease or pyoderma. Finally, the dentition is evaluated and the teeth counted to determine if all teeth are present. Discolored teeth, persistent deciduous teeth, root and furcation exposure, oral mucosal lesions, sinus tracts, tongue abnormalities, oral masses, plaque and calculus are noted.

Anesthetized Examination

While the patient is under anesthesia a full oral examination and dental charting is performed. For the purpose of this lecture, periodontal indices are discussed (http://www.avdc.org/nomenclature.html). During the periodontal examination, crowded teeth, missing teeth, rotated teeth, mobile teeth, teeth with furcation exposures, gingival recession (root exposure), sinus tracts, gingival enlargements, and periodontal probing depths are noted (The normal gingival sulcus depth in a dog is 0-3 mm and less than 0.5 to 1.0 mm in a cat).

Gingival indice is assessed:

- Gingival indice of 1 - inflammation and swelling of the gingiva with no bleeding during periodontal probing
- Gingival indice of 2 – inflammation and swelling of the gingiva with bleeding during periodontal probing
- Gingival indice of 3 – inflammation and swelling of the gingiva with spontaneously bleeding of the inflamed gingiva prior to periodontal probing

Furcation exposure (involvement) occurs when a periodontal probe can extend between the roots, under the crown, of multi-rooted teeth as a result of attachment loss.

- Stage 1 furcation - the probe extends less than half way (Periodontal Disease Stage 2)
- Stage 2 furcation - the probe extends greater than halfway (Periodontal Disease Stage 3)
- Stage 3 furcation - the probe extends from one side to the other, through and through (Periodontal Disease Stage 4)

Gingival recession (root exposure) is measured from the location of the cementoenamel junction to the free gingival margin. Any probing depths, whether normal or not, are recorded because and additional probing depth is additive to periodontal attachment loss. For example, if there is 3 mm of gingival recession and a 2 mm probing depth, there is a total of 5 mm of periodontal attachment loss.

Periodontal pockets are clinical periodontal probing measurements greater than the normal sulcus. The normal gingival sulcus depth of the dog is 0-3 mm. The normal gingival sulcus in a cat is less than 0.5 to 1.0 mm. The periodontal probe is gently walked 360° around each tooth. A minimum of 6 locations is measured.

Periodontal pockets can be a combination of various types of pockets created by periodontal bone loss and gingival enlargements. Periodontal pockets are a haven for gram-negative anaerobic bacteria and spirochetes in the subgingival plaque biofilm and planktonic bacteria in the pocket gingival crevicular fluid. There are often combinations of periodontal pocket types as they are not mutually exclusive.

However, they may be *simplified* for clinical practice:

- ***Pseudopockets*** are created when the gingiva enlarges (often gingival hyperplasia) and the marginal bone remains at the appropriate level. Breeds such as Boxers and Collies have a genetic predilection for gingival hyperplasia. Common veterinary medications such as cyclosporine and amlodipine may cause gingival enlargement.

- ***Suprabony pockets*** occur when marginal bone loss exceeds gingival recession (the marginal bone is lost horizontally below the tissue).

- ***Intra(Infra)bony pockets*** occur when bone is lost vertically around a tooth. Infrabony pockets can be classified as one-wall, two-wall, three-wall, and four-walled (cup or crater) defects.

  Common locations for intrabony pockets in dog patients include the distal aspect of the mandibular 1st molars, the furcation of the mesial roots of the maxillary 4th premolars, the mesial aspects of the mandibular canine teeth, particularly after the 3rd incisors are lost or are extracted without proper technique, and the palatal aspect of the maxillary canine teeth.

Periodontal Stages

Treatment plans can be designed based on the individual tooth stage as well as the overall periodontal stage of the oral cavity. There are 42 or 30 individual patients (teeth) to diagnose and treat in the dog and cat, respectively.

- ***Stage 0 (PD0)*** - clinically normal oral cavity with no gingival inflammation and periodontitis
- ***Stage 1 (PD1)*** - gingivitis without attachment loss (normal height and architecture of alveolar margin)
- ***Stage 2 (PD2)*** - periodontitis with less than 25% attachment loss and/or a stage-1 furcation in multi-rooted teeth
- ***Stage 3 (PD3)*** - 25-50% attachment loss and/or stage-2 furcation in multi-rooted teeth
- ***Stage 4 (PD4)*** - greater than 50% bone loss and/or stage-3 furcation in multi-rooted teeth

Clinical Findings of Hidden Disease

A ***parulis*** is a raised nodule at the opening of a draining sinus tract. If the parulis is located apical to the mucogingival junction it is often associated with endodontic disease. If the parulis is located near the mucogingival junction, it is often associated with periodontal disease.

Maxillary draining tracts should be investigated for odontogenic infections such as periodontal disease or endodontic disease prior to extensive dermatological or neoplastic work ups including advanced imaging and biopsy. Teeth should be the primary differential for the maxillofacial swellings and draining tracts. If an odontogenic infection is not the cause, then evaluation for

neoplasia, etc. can be pursued. Often if it is neoplasia, a tooth is involved and surgical extraction and deep biopsy via the extraction site will provide a histological diagnosis.

The *maxillary 1st and 2nd molars* in dogs may have minimal clinical probing depths but be mobile during clinical examination. The intraoral radiographs may identify a very wide or absent periodontal ligament space/large palatal root periapical lucency. This is consistent with severe periodontal disease and surgical extraction and closure of the extraction site is required.

Large periodontal probing depths may be identified *mesial or distal to the mandibular 1st molars in dogs* with minimally associated gingival inflammation. Intraoral radiographs will identify large intrabony pockets. Treatment may include selective extraction of non-strategic teeth, open root planning and bone augmentation or guided tissue regeneration. If the mandibular molar cannot be saved, then surgical extraction is necessary.

When probing the teeth always probe between them *mesial buccal and mesial palatal roots of the maxillary 4th premolars*. This is a common place for a hidden intrabony pocket that is not easily identified with intraoral radiographs due to summation and superimposition of radiopaque dental structures and bone. Deep intrabony pockets will require guided tissue regeneration or the tooth will require surgical extraction.

ROENTGEN SINGS of PERIODONTAL DISEASE

Radiographically there will be loss of the marginal bone, loss of the lamina dura, widening of the lamina lucida (periodontal ligament space), and horizontal and vertical bone loss due to the resorption of bone. ***Horizontal bone loss*** occurs when the cortical supporting bone around the tooth and adjacent teeth is lost at a similar rate. If the soft tissue does not recess at a similar rate as the bone, a suprabony periodontal pocket will be formed. ***Vertical bone loss*** occurs when there is one area of bone loss around a tooth with adjacent supporting bone and mineralized tooth structures remaining more coronal. Vertical bone loss results in infrabony pockets (single wall defect, two wall defect, three wall defect, and four wall defect).

TREATMENT AND MANAGEMENT FOR PERIODONTAL DISEASE AND PERIODONTAL POCKETS

To fully understand professional treatment options and home care products understanding periodontal disease reduces to two topics:

1) **Plaque biofilm inciting host inflammation**
2) **Periodontal pockets.**

Management of periodontal disease is not a once in a lifetime event for the patient but rather an ongoing program throughout continued life stages of the patient. Gingivitis is the start of the inflammatory cascade and may progress to periodontitis and loss of periodontal attachment. The

goal with periodontitis is to stop the disease, minimize further attachment loss, and treat compromised teeth (e.g., periodontal surgery, guided tissue regeneration, and extraction as indicated). Therefore, education and prevention of disease (daily brushing, dentifrices, and frequent professional dental care) are the best defenses.

A professional dental cleaning, to return the tooth crown and root to a clean surface, followed by daily home care, to remove the plaque biofilm, is the gold standard to prevent and control periodontal disease. If pockets are eliminated and the plaque biofilm is removed on a daily basis, then the maturation of plaque and further pocket formation can be controlled and minimized. General anesthesia is required for correct periodontal assessment, diagnosis and treatment (http://avdc.org/AFD/).

Periodontal Treatment

Veterinary patients should be scheduled for a periodontal cleaning when there is gingivitis and before irreversible periodontal disease and attachment loss has occurred. Supragingival scaling and subgingival scaling is performed. Subgingival scaling separates a professional dental cleaning from a purely cosmetic procedure. Correct subgingival cleaning is impossible in the non-anesthetized patient.

A professional dental (periodontal) cleaning takes time to assess the oral cavity, obtain intraoral radiographs, and professionally clean the oral cavity. Additional periodontal treatments, periodontal surgery and extractions, as indicated, can easily double the treatment time. Therefore, appropriate time must be scheduled in the surgical schedule to allow unrushed assessment and execution of treatment plans.

Periodontal Cleaning Equipment and Instruments

Equipment necessary for a complete, professional periodontal cleaning includes, but is not limited to, ultrasonic scalers [piezoelectric and magnetostrictive (ferromagnetic stacks and ferrite rods)], hand scalers, universal curettes, gracey curettes (only one working surface offset 70°), slow speed hand piece for polishing, irrigation, dental probes and explorers, and dental charts.

Periodontal Cleaning Steps

Client consent is required prior to the initiation of treatment (be prepared to find more disease then you would expect and prepare the client). Masks, caps, gloves and *protective* eyewear are worn. *General anesthesia* is required. The oral cavity is rinsed with a 0.12% chlorhexidine gluconate oral rinse to decrease help eliminate aerosolized bacteria. *Supragingival scaling* involves removing the calculus and plaque from above the gumline (hand scalers and water cooled ultrasonic scalers – no more than 5-7 seconds per tooth to prevent thermal and concussive injury). *Subgingival scaling* (root planing and subgingival curettage) is crucial for the treatment and prevention of periodontal disease. Hand curettes and some water cooled ultrasonic scalers, with approved periodontal or universal tips, are used to clean subgingivally. *Polishing* involves using a pumice (fine) to smooth out roughness created in the enamel during the periodontal

cleaning. Polishing should be minimized to less than 3 seconds per tooth. The polishing cup should flare 1 to 2 mm subgingivally to polish the subgingival tooth surface cleaned during the subgingival scaling. The air-water syringe is used to irrigate the sulcus and remove debris, plaque, and polishing paste. Intraoral radiographs are obtained.

The periodontal cleaning is not complete until *client education* is presented. If the procedure was a periodontal cleaning without surgery, then the client should be educated on home care at discharge. If surgery was performed, education may be delayed until the recheck appointment to verify the surgical sites are healed (10-14 days) prior to instituting a plaque control home care program. A recall for the next periodontal cleaning and oral exam is set for 6-12 months depending on the stage of periodontal disease, client commitment to home care, and signalment of the patient.

Periodontal Surgery

Periodontal surgery occurs with, and after, the oral cavity has had a thorough assessment, intraoral radiographs, and professional periodontal cleaning. Often, it is best to stage the procedures so that the periodontal surgery is performed several weeks after a periodontal cleaning if periodontal flaps or guided tissue regeneration are being utilized. Soft tissue resection and some osseous subtractive surgeries may be performed during the periodontal cleaning.

When patients have teeth in stage 2 or 3 periodontal disease and/or periodontal pockets, periodontal surgery may be necessary to return periodontal anatomy to a manageable level. Once returned to a manageable status, frequent periodontal cleanings and home care programs can maintain and stabilize the periodontium. Stage 4 periodontal disease is sometimes best treated by exodontics depending on the tooth and signalment of the patient. Be a patient advocate.

Periodontal pockets greater than 5 mm (with breed variations), periodontal probing depths beyond the mucogingival junction (whether 5 mm or not), stage 2 and 3 furcation exposures, intrabony pockets, gingival clefts, mobile incisors, loss of gingiva, and periodontal trauma require periodontal surgery.

Pseudopockets: Gingival enlargement is resected with a complete periodontal cleaning to remove the pseudopocket. External bevel gingivectomy and gingivoplasty are meticulously performed using combinations of scalpel blades, careful use of radiosurgery, diamond burs, 12-fluted burs, and periodontal knives (e.g., Orban and Kirkland) after intraoral radiographs and periodontal probing are used to establish the gingival sulcus and free gingival margin planned positions.

Suprabony Pockets: Suprabony pockets can be treated with open and/or closed root planning depending on the pocket depth. Periceutical treatment can be considered when treating suprabony pockets.

Intrabony Pockets: Intrabony pockets require osseous additive surgery (guided tissue regeneration) or osseous subtractive surgery to eliminate intrabony pockets.

However, if there is not appropriate attached gingiva remaining, not appropriate amounts of periodontal attachment remaining, and the client is not motivated to have future professional periodontal cleanings and provide home care, then surgical extraction may be a better option for the pet to eliminate a lifetime of chronic inflammation, infection, and pain.

Periodontal Pocket Healing

Following periodontal treatment gingival connective tissue, gingival epithelium, periodontal ligament, and alveolar bone compete to create reattachment to the tooth surface. Following subgingival scaling for suprabony pockets the gingiva can re-attach the appropriately cleaned tooth surface via long junctional epithelium.

Following more advanced periodontal surgery (guided tissue regeneration) *four (4) tissues compete for the root surface*: gingival epithelium, gingival connective tissue, periodontal ligament, and alveolar bone. The preferred attachments are the periodontal ligament and alveolar bone for long-term periodontal support. The gingival connective tissue and epithelium colonize the root surface at the fastest rate and exclude, the other more desirable periodontal tissues, periodontal ligament and bone from the root surface.

Advanced periodontal surgery should not be performed if the client is not compliant with home care, the pet is not compliant with home care, or the pet is not medically stable for future anesthetic episodes to recheck and augment periodontal treatments. If there is any doubt, then extraction of an offending tooth or teeth should be executed to alleviate pain and inflammation in the patient. Appropriate training is necessary in order to achieve predictable results when utilizing guided tissue regeneration.

Goals of periodontal therapy include control of the plaque biofilm, prevention of further attachment loss, control and treatment of periodontal pockets (e.g., pseudopockets, suprabony pockets, and intrabony pockets) and preparing the tooth surface for re-attachment of healthy periodontal tissues. Guided tissue regeneration (GTR) can be utilized to increase periodontal attachment of strategic teeth. Before GTR should even be considered, understanding periodontal disease pathophysiology, the periodontal cleaning including subgingival scaling, subgingival curettage, and root planing, in addition to open periodontal flaps, must be understood.

Periodontal surgery includes*:*

1. *Open periodontal flaps with root planing* – mucoperiosteal flaps are created to in order to visualize and expose the root surface. The root surfaces are root planed with curettes and an irregular periodontal bone is contoured. The periodontal flap is meticulously sutured back around the involved teeth. An appropriate collar of gingiva must be secured around each tooth.

2. *Periodontal Splinting* – mobile incisor teeth are open root planed, and a periodontal splint is placed on the lingual aspect of the teeth in order to anchor the individual single rooted incisors into one multi-rooted unit to stabilize the regional mobility of the incisors.

3. *Gingivectomy/Gingivoplasty* – Excessive gingival tissue (e.g., drug induced, breed related) is excised with an external bevel gingivectomy and contoured via gingivoplasty in order to remove pseudopockets.

4. *Periodontal pedicle flaps* – gingival pedicle flaps are created and moved over cleaned root and bone surfaces to treat gingival clefts and festoons by returning a larger band of gingiva on the tooth. Common locations include the buccal aspect of the maxillary canine tooth and the mesial-buccal root of the maxillary 4th premolar.

5. *Osseous resective/subtractive surgery* – some intrabony pockets, where there is still significant periodontal bone attachment, can be treated with open root planing, removing alveolar bone (osteoplasty), and soft tissue re-sutured around the tooth with an apically repositioned periodontal flap. Alveolar osteitis on the buccal aspects of feline canine teeth resulting in intrabony pockets, mesial lingual intrabony pockets of the mandibular canine teeth in the dog, and the mesial and distal aspects of the mandibular 1st molar, after extraction of the mandibular 4th premolar and 1st molar, respectively, are common locations where osseous subtractive surgery may be utilized. Intraoral radiographs and open periodontal flap surgery is required.

6. *Osseous additive surgery (Guided tissue regeneration)* – is the new formation of periodontal tissues (cementum, periodontal ligament, and alveolar bone) that had been destroyed from periodontitis. *Regeneration*, reconstitution of lost tissue, is differentiated from *periodontal repair*, healing of the periodontal wound/defect by tissue that does not fully restore the normal histological architecture. GTR involves open periodontal flaps, +/- debated root surface preparations (e.g., tetracycline, citric acid, EDTA), +/- grafting materials, +/- biological modifiers (e.g., growth factors, cytokines), and periodontal barrier membranes. Intraoral radiographs, in conjunction with general anesthesia, are required for assessment, treatment planning, and treatment execution.

Conclusion

Periodontal treatment always begins with a complete and thorough periodontal cleaning. Periodontal surgery techniques are available to treat periodontal pockets in patients with owners who are committed to home care and future periodontal treatments. Home care products are not discussed or presented today.

Recognition and Treatment of Feline Tooth Resorption

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Introduction

Tooth resorption is a common and frustrating dental problem in the feline patient. It has been known previously by many names. The accepted AVDC nomenclature term is tooth resorption. The prevalence of the disease, in cats, has been reported in the literature as 20-75%.

Pathophysiology

The lesions begin in the cementum, and can occur anywhere on the root surface and not just the cervical region. The resorption progresses into the dentin and enamel of the tooth root and crown. Clinically, these teeth may have localized gingival enlargements of highly vascularized, inflamed granulation tissue covering supragingival defects, small defects at the gingival margin, and/or no supragingival lesions. Intraoral radiographs are required to diagnose and treat the disease. These lesions are painful but, as with all dental and oral disease, the range of clinical signs can vary from partial anorexia, weight loss, halitosis, ptyalism, dysphagia, etc., to no obvious clinical signs at all. If the lesions remain below the gingival attachment, they are often asymptomatic since they dentin tubules and pulp are not exposed to the oral environment. Some cats may exhibit supereruption of involved teeth (particularly the canine teeth) to potentially maintain a normal biological width. These supererupted teeth are associated with hypercementosis. The increased exposure of the tooth root needs to be differentiated from gingival recession/root exposure that is caused by periodontitis.

The cause of tooth resorption is not fully understood. Odontoclasts are derived from hematopoietic stem cells. These multinucleated cells resorb mineralized tooth structure on several regions of the root simultaneously. The stimulation of the odontoclasts is not known. Abfraction, diet, minerals, water sources, periodontal disease and inflammation, and vitamin-D have been speculated as inciting causes. Vasodentin, a type of dentin with tubules positioned randomly and communicating with the pulp canal, has been observed. The microhardness and thickness of feline enamel may be a factor. Furcation canals and lateral canals may or may not play a roll. The initial lesions are non-infectious and non-inflammatory. There may or may not be breed predilections. There is no sex predilection. The lesions may very well be multifactorial. Essentially, feline tooth resorption, not associated with infection (periodontal or endodontic disease) is classified as idiopathic.

Diagnosis

The diagnosis of tooth resorption requires clinical examination and intraoral radiographs, while the patient is anesthetized. Without intraoral radiographs, diagnosis and treatment plans cannot be accomplished.

The stage of lesions can be classified by the extent of tooth involvement (see AVDC Nomenclature www.avdc.org):

- **Stage 1 (TR1)**: Mild dental hard tissue loss (cementum or cementum and enamel)
- **Stage 2 (TR2)**: Moderate dental hard tissue loss (cementum or cementum and enamel with loss of dentin that does not extend into the pulp cavity)
- **Stage 3 (TR3)**: Deep dental hard tissue loss (cementum or cementum and enamel with loss of dentin that does extend into the pulp cavity)
- **Stage 4 (TR4)**: Extensive dental hard tissue loss (cementum or cementum and enamel with loss of dentin that does extend into the pulp cavity; most of the tooth has lost its integrity)
    - **TR4a** (crown = root), **TR4b** (crown>root), **TR4c** (crown<root)
- **Stage 5 (TR5)**: Remnants of dental hard tissue are visible only as irregular radiopacities and gingival covering is complete.

The lesions can be further divided into types. Tooth resorption lesions can be divided into stages and types. **Types** refer to the **radiographic appearance** and are crucial for treatment planning.

Type 1 is generally associated with periodontal disease or apical periodontitis from endodontic disease. There will be a normal root opacity/density with a surrounding lamina lucida and usually a definable root canal. Essentially this is consistent with external inflammatory tooth resorption.

Type 2 (replacement resorption), the teeth have undergone significant resorption and have a different opacity/density. There is loss of the lamina lucida and dentoalveolar ankylosis is present. There may be no discernable root structure present.

Type 3 occurs when one root is Type 1 and one root is Type 2. It is essential that the surgeon use intraoral dental radiographs and understand Type 1, Type 2, and Type 3 lesions for treatment planning.

Treatment

The current treatment recommendation is for extraction of stage 2-4 tooth resorption lesions. All teeth with type 1 lesions must be extracted. Type 2 lesion teeth may be treated with subgingival

crown amputation and intentional root retention (see www.avdc.org position statement) but I extract most teeth and reserve subgingival crown amputation for select cases. Unfortunately, many veterinarians perform subgingival crown amputations incorrectly or when they are *contraindicated*. Examples of incorrect treatment include, but are not limited to, amputating after having difficulty extracting the tooth, performing root "pulverization/atomization", crown amputations without intraoral dental radiographs, performing subgingival crown amputation incorrectly resulting in retained, infected, sequestered roots causing chronic pain and infection. My general rule is to extract, extract, extract, and only perform subgingival crown amputations and intentional root retention when all the following criteria are met. If all the rules are followed correctly, there is a good success rate for subgingival crown amputations.

Subgingival Crown Amputation with Intentional Root Retention Criteria:

1. Intraoral radiographs utilized
2. Roots must show Type 2 replacement resorption
3. There are no periapical lucencies (endodontic disease)
4. There is no periodontal disease
5. The patient does not have "stomatitis"
6. The owner must be advised that subgingival crown amputation was performed and roots were intentionally left
7. It must be recommended to recheck the sites with intraoral radiographs in 6-12 months
8. The procedure is clearly noted in the medical and dental record
9. The tooth was amputated from the start and was not amputated because of a failed extraction attempt

Regardless if the teeth are extracted or crown amputated, patients are treated perioperatively with multimodal analgesia protocols and discharged with analgesics. All surgical sites are sutured closed with absorbable suture such as poliglecaprone-25, chromic gut, or polyglactin 910. Antibiotics are used post-operatively for 7-10 days in cases with significant periodontal disease or extensive surgical flap exposure of the maxillary and mandibular bone.

Subgingival Crown Amputation With Intentional Root Retention

Crown amputations are reserved for clear tooth resorption with a radiographic diagnosis of Type 2 tooth resorption. All type 1 teeth must be extracted. Type 2 lesion teeth may be crown amputated (see www.avdc.org position statement).

An envelope flap is created. A tongue depressor is used to protect the soft tissues of the tongue when treating mandibular teeth. The envelope flap is carefully lifted with a periosteal elevator to expose the furcation and marginal bone. A 330 bur is used parallel to the marginal bone to excise the marginal bone and crown. Once the crown is removed a medium grit football diamond bur is used to continue the subgingival crown amputation +/- 2 mm apically. Smooth, mesially and distally tapering bone margins should be left. An intraoral radiograph is obtained to evaluate the contour of the amputation and to be certain no bone or tooth fragments persist coronally. The site is rinsed with 0.12% chlorhexidine gluconate and sutured closed. All surgical sites are sutured closed with absorbable suture such as poliglecaprone-25, chromic gut, or polyglactin 910.

Crown Amputation Complications

1. Tearing of the gingival tissue preventing closure. Delicate tissue handling and careful elevation are necessary to prevent the complication.

2. Incomplete crown removal irritating the overlying gingiva. Post-op radiographs are necessary to evaluate, identify, and correct this problem.

3. Utilizing a tongue depressor to protect the tissues easily prevents laceration of the lingual tissue with the high-speed bur.

4. Root remnant infections secondary to incorrect diagnosis of tooth resorption type. Intraoral radiographs are necessary for correct diagnosis.

IV Fluid Therapy Review: The Basics and Updates from the Critical Care World

Lindsey Nielsen, DVM, DACVECC

1. Body Fluid Compartments
    a. 60% of our (and patients) bodies are made up of water
        i. 2/3 of this is found in the intracellular space (40% of body weight)
        ii. 1/3 of this is found in the extracellular space (20% of body weight)
            1. ¾ of the extracellular fluid is found in the interstitial space
            2. ¼ of the extracellular fluid is found in the intravascular space- THIS IS PLASMA
    b. The body requires relatively appropriate fluid volumes in each compartment to function normally
    c. The body requires relatively normal ratios of electrolytes and solutes in both the intra and extra cellular spaces in order to function normally
    d. Why does this matter?
        i. When we are giving IV fluids we are mostly targeting the INTRAVASCULAR space, which is only 5% of body weight
        ii. Understanding IV fluid effects and body water distribution will help the clinician understand how to more appropriately chose IV fluid therapies in various clinical situations
2. Hydration vs. Perfusion
    a. Perfusion is the transport of blood to the tissues
        i. Patients with perfusion problems have a lack of fluid in the intravascular space and manifest with SHOCK
    b. Hydration is the role that water plays in tissue support and cellular function
        i. Patients with hydration problems have a lack of fluids in the intracellular and interstitial spaced and manifest with DEHYDRATION
3. Types of Fluids
    a. Crystalloids- the mainstay of fluid therapy
        i. Solutions that contain electrolyte and non electrolyte solutes that are capable of entering all body fluid compartments
            1. These fluids resemble plasma water
            2. Examples= LRS, plasmalyte
        ii. Can be used for replacement to rapidly replace the intravascular space in shock (but require large volumes to do so)
        iii. Can be used to maintain a normal fluid state in patients that are not eating or drinking on their own yet

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iv. Hypertonic Crystalloids (Hypertonic saline)
    1. Yes this is a crystalloid.
    2. This product can be used in low volumes to increase the intravascular space as it's hypertonicity will suck fluid from the interstitial space back in to the intravascular space as well
    3. Typical dose is 4ml/kg over 10-15 minutes
    4. Hypertonic saline as many other beneficial effects as well, like anti-oxidant and anti-inflammatory properties.
    5. It may be very beneficial in cases of brain injury.
    6. Contraindications to its use= dehydration, hypervolemia, hypernatremia

b. Colloids- used when trying to maintain fluid in the intravascular space for longer than with crystalloids
    i. Starling's Law- helps us explain COP, and that the net movement of fluid in and out of a vessel is influenced by the oncotic pressure in both the vessel and directly outside of it.
        1. Albumin in the body is responsible for maintaining at least 80% of COP.
    ii. Albumin- canine and human products exist
        1. Albumin deficit (g)= 10 X (desired albumin-patient albumin) X BW in kg X 0.3
        2. Aim to get albumin to between 2-2.5 in most cases with severe hypoalbuminemia
    iii. Plasma and other blood products
        1. Require 45ml/kg of plasma to raise albumin 1 point
        2. Plasma may help repair damage to the endothelium in illness, helping maintain COP
        3. Blood products are often used as fluid therapy in situations of hemorrhagic shock
    iv. Synthetic colloids
        1. Examples include Vetstarch, Hetastarch, Pentastarch, and Dextrans
        2. Turns out they don't actually stay in the intravascular space like we thought, and tend to leak through the damaged endothelium
        3. They also have a lot of bad side effects like AKI, allergic reactions, coagulopathies, etc.
        4. No longer used in human medicine or most referral veterinary hospitals

4. Questions To Ask About Fluid Therapy For Patients
    a. Is the patient in shock?
    b. Is the patient dehydrated?
    c. Can they drink on their own?
    d. What type of fluids, what route?

    e. How much to give and how fast?
5. How can you maximize your fluid therapy?
    a. Poiseuille's Law

$$\text{Flow} = \pi (P1-P2)r^4 / 8\eta L$$

    b. This translates in to placing LARGER GAUGE and SHORTER IV catheters mean you can give IV fluids faster/more efficiently
6. Monitoring response to IV fluids
    a. Weight gain is a great way to assess for rehydration
    b. If a urinary catheter is in place, urine output is helpful
    c. Central lines can allow you to measure CVPs
7. Complications to IV fluids
    a. Over-hydration; edema or fluid overload
    b. Catheter related infections or complications
    c. Vagal induced bradycardia/hypotension if you give hypertonic saline too fast
8. Contraindications to IV fluids (yes, sometimes the criticalist says DON'T give them)
    a. Cardiogenic shock
    b. Oliguric or anuric renal failure

Antimicrobial therapy: Principles to resist resistance

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Objective: To review rational and judicious antimicrobial use and introduce the topic of antibiotic stewardship

From Boothe's chapter on Principles of Antimicrobial Therapy in Small Animal Clinical Pharmacology and Therapeutics: "Antimicrobial use by veterinarians affects the global medical community; the veterinary hospital, the patient; and, as increasingly being recognized, the pet owner."

What is the purpose of antimicrobial therapy?

To successfully treat bacterial infection while minimizing the development of resistance.

Antimicrobial classes and mechanisms of action

Drug/Class	Target	
Penicillins, cephalosporins, carbapenems, glycopeptides	Cell wall inhibitors	Bactericidal
Fluorinated quinolones, metronidazole	DNA	Bactericidal
Aminoglycosides	Ribosomes	Bactericidal
Polymixin, Colistin	Cell membrane	Bactericidal
Trimethoprim-sulfonamides	Metabolic pathway	Bactericidal
Rifampin	RNA	Bactericidal
Macrolides, lincosamides	Ribosomes	Bacteriostatic, * accumulation in WBC may allow to be cidal
Tetracyclines, phenicols	Ribosomes	Bacteriostatic
Trimethoprim, sulfonamides, ormetoprim	Metabolic pathway	Bacteriostatic

Why should we care about bacterial resistance?

From the limited data available, resistance is on the rise. Multi-drug resistant and pan-drug resistant infections are a major contributor to morbidity and mortality and cost of care in human medicine.

Veterinary drug options are, reasonably so, more limited than in people.

Mechanisms of resistance

- Acquisition of genes that encode enzymes (β-lactamases)
- Acquisition of efflux pumps
- Acquisition of genes that alter the antibiotic target (cell wall, porins)

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Bacteria can acquire resistance via vertical evolution (parent to offspring), or through mutation or selection. Genes can transferred from one bacteria to another via horizontal evolution - through transformation, conjugation or transduction.

Again from Boothe in the chapter Treatment of Bacterial Infections, she advocates that "a three pronged approach is indicated for preventing antimicrobial resistance: escalating hygiene, de-escalating antimicrobial drug use, and optimizing dosing regimens such that the infecting inoculum is eradicated, not simply inhibited."

What is antibiotic stewardship?

From the website of the Infectious Diseases Society of America: Antimicrobial stewardship refers to coordinated interventions designed to improve and measure the appropriate use of antimicrobials by promoting the selection of the optimal antimicrobial drug regimen, dose, duration of therapy, and route of administration.

According to the CDC's website, 20-50% of all antibiotics prescribed in US hospitals are either unnecessary or an inappropriate drug. Antibiotic resistant organisms contribute to the death of 23,000 people per year.

Antibiotic stewardship in human studies has NOT been associated with increases in hospital acquired infections, longer hospital stays or increases in mortality. In fact, a 2013 Cochrane review of interventions to improve antibiotic prescribing practices found that restrictive interventions on prescribing had greater impact than persuasive ones. Interventions that decreased prescribing showed a decrease in a variety of resistant strains (C. difficile, MRSA, VRE). It was also noted that several studies that were specifically aimed at more effective but less excessive antibiotic prescribing for pneumonia identified a reduced mortality rate.

<u>Stewardship programs involve:</u>

- having a leader/s that is monitoring the program
- explicitly documenting drug dose, duration and indication in the medical record
- discussing and potentially implementing facility prescribing guidelines
- having automatic stop orders in place for antibiotics (for example, not routinely continuing prophylactic surgical antibiotics)
- taking an antibiotic history for patients
- re-assessing prescribed antibiotics for de-escalation every 24 hours in hospitalized patients

<u>Important concepts</u>

MIC = minimum inhibitory concentration = lowest concentration which limits bacterial growth (in vitro). This value does not account for any patient factors and is created by a lab standards group.

An MIC that is considered “susceptible” means this is the lowest concentration of drug needed to inhibit the growth of 50% of the bacteria cultured. This is based on data collected by the lab standards group.

In otherwise healthy patients, an MIC 50 is adequate to support the host’s immune system in clearing the infection. However, in systemically ill or immunocompromised patients, this may not be adequate and an MIC 90 is often recommended.

Achieving MIC means something different for different types of antibiotics.

Time dependent antimicrobials = the drug efficacy is dependent on the time spent above the MIC. To be clinically effective this generally means greater than 50% of the time spent above MIC

Concentration dependent antimicrobials = the drug efficacy is dependent on the Cmax achieved and the drugs have a post-antibiotic effect. This is determined by using the inhibitory quotient (IQ) = Cmax/MIC. The IQ should be greater than 4 for healthy patients and greater than 8 for the critically ill.

In an ideal world, treatment is based on culture results however, this is not always possible. Therefore, empiric choices are based on knowledge of the most likely causative organisms, host factors and local resistance patterns.

<u>Designing a therapeutic plan</u>

Step 1.

Are antibiotics warranted?

Fever does not equal infection.

Examples: Viral upper respiratory disease in cats, kennel cough, simple abscesses or wounds where source control is easily achieved

Step 2.

Is there a fluid or tissue that can be cultured? Or, can a gram stain be performed to guide therapy?

2013 JVECC study found that gram staining urine sediment had a sensitivity of 96% and specificity of 100% for identifying bactiuria as compared to a sensitivity of 76% and specificity of 77% when compared to routine sediment evaluation.

Cytology +/- gram stain of airway lavage samples

Step 3.

What are the most likely causative organisms at the site of infection?

What is normal flora? Is this infection likely to be gram negatives, positives, anaerobes, or mixed?

Step 4.

What are the potential host and bacterial factors that may affect the treatment outcome?

Microbial production of materials that impair phagocytosis, facilitates invasion, damage tissues, and biofilm.

Host factors include the overall cardiovascular stability of the patient, local and systemic immunity, infection location and the duration of the infection.

Step 5.

What is the potential risk of failure?

Is the patient immunocompromised? Are they critically ill?

Step 6.

Choose an agent/s with a clearly defined dose and duration of therapy documented.

Combination vs. monotherapy

There is no definitive evidence in people that combination therapy leads to better outcomes. It is recommended that combination therapy be reserved for patients with severe sepsis or other critical illness or when resistance is likely.

For time-dependent antibiotics (particularly β-lactams) extended or continuous dosing can improve their pharmacodynamics. The same dose of the antibiotic given over 4-6 hours or continuously can increase the % of time above MIC and thus the efficacy.

How are critically ill patients different?

Up to 65% of critically ill people have increases in their renal function which is of great concern for renally cleared antibiotics such as penicillins, cephalosporins and carbapenems.

Capillary leak syndromes can increase the volume of distribution of hydrophilic drugs thus decreasing plasma drug concentration

High rates of IV fluids may increase renal perfusion and increase the clearance of hydrophilic antibiotics

Hypoalbuminemic states can result in a higher unbound drug concentration and thus greater volume of distribution and clearance for drugs that are highly protein bound

Decreased organ perfusion secondary to severe disease can lead to decrease clearance and prolonged drug half-life, and thus the potential for toxicity

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Transfusion Medicine- Practices and Consequences

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Sage Concord

Objectives

- Reasons for transfusion
- Blood product types
- Safety and efficacy
- Controversies with blood product administration

Indications for transfusion

- What kind of patients do we see?
- Trauma
- Non traumatic hemoabdomen
- Auto immune disease
- Vitamin K antagonist rodenticides
- Chronic kidney disease
- Bone marrow suppression- drug, toxic, primary
- Von willebrands patients
- Platelet dysfunction patients

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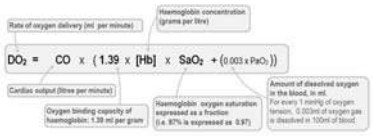
Indications for transfusion

- The trigger
- Hemolytic patients
  - Fuel the fire?
- Acute versus chronic blood loss
  - Acute- increased HR, RR, bounding pulses
  - Chronic- comfortable at rest, but intolerant of handling

Indications for transfusion

- Acute- low 20s
- Chronic- teens
- Will anemia continue? What's the process?



Blood Product Types

- Those with cells
- FWB= fresh whole blood
- SWB= stored whole blood
- RBCs= red blood cell concentrate
- Platelet concentrate
- Platelet rich plasma

Blood product types

- Those without cells
- FFP- processed and frozen within 8 hours of collection
- NFFP or thawed plasma- some loss of factors 5, 8
- Cryoprecipitate- vW factor, 8, 13, fibrinogen and fibronectin

Blood Products

- The elusive platelet product
  - Lyophilized product made briefly
  - Never came to market
  - UCD?
  - Life span, efficacy?

Blood Typing- Dogs

- Typing
  - Donor- YES
    - DEA 1.1 negative desired
    - Of 9 blood types, 1.1 seems to be the most likely to produce a reaction
    - Not exactly a universal donor
  - Recipient
    - 1st transfusion- use DEA 1.1 negative donor
    - Subsequent transfusions- ideally, type specific or 1.1 negative
- Recommended

Cross match- dogs

- Testing for serum antibodies
  - Major
    - antibodies against donor RBCs in recipient serum
  - Minor
    - antibodies against recipient RBCs in donor serum
- First transfusion-
  - May omit, unless past hemolytic reaction
- Second transfusion- no longer optional
- Previously pregnant- ????

Typing and Crossmatch- cats

- Type
- AB cats should get AB or A blood
- Preformed antibodies
- Anti A antibodies in B cats
- Crossmatch
  - should be strongly considered in case of other antibodies

Autoagglutination and Crossmatching

- Hard to interpret the crossmatch
- Alvedia strip test or Rapid Vet gel technique
  - False positive
- Saline slide agglutination
- Washing recipient RBCs, testing against own serum
- Transfuse carefully…

Transfusion Administration

- How much to give?
- Whole blood
  - 2 ml/kg→ 1% increase
- RBCs
  - 1 ml/kg→ 1% increase
- FFP
  - Depends…

Transfusion Administration

- How fast?
  - ASAP
  - 5 ml/kg/h x 30 minutes
  - 10 ml/kg/h

Transfusion Administration

- What tools?
  - Peristaltic pump
    - Controversial- effects on RBC integrity
  - Syringe pump
    - Also controversial
  - Gravity drip
    - Minimal effect on RBCs, but hard to control
  - Hemonate or in line filter

Transfusion Administration

- Avoid transmitting disease
  - Donor screening
- Avoid sepsis
  - Stored 30 days at 1-6 C
  - Supernatant
    - Cloudy, abnormal color, hemolysis
- Once opened…
  - Most literature says use within 4 hours
    - Source?

Transfusion Consequences- TRIM

- Transfusion related immunomodulation
  - Humans, transplant patients
- Down regulation of the immune system following transfusion
  - Plasma components?
    - Leukoreduced products less likely?
  - Increase in post op infections

Transfusion Consequences- TACO

- Transfusion Associated Circulatory Overload
- Volume intolerant patients
- Pulmonary or peripheral edema
  - Diuretic
  - Slowing or stopping the transfusion

Transfusion Consequences- TRALI

- Transfusion related acute lung injury
- Immediate or delayed (1-72 hours)
- Severe bilateral pulmonary edema
  - High protein fluid
  - Oxygen, ventilation
  - Diuretics NOT helpful
- Leukocyte aggregates in pulmonary circulation

Transfusion Reactions

- Febrile non hemolytic
  - Fever, vomiting, tachycardia, tachypnea
  - Inflammatory cytokines in donor blood
  - Slow or stop transfusion
  - Steroids?
- Acute allergic
  - Hives, facial swelling, hypotension
  - IgE hypersensitivity reaction
  - Anti histamines
  - Steroids?

Transfusion Reactions

- Hypocalcemic
  - Muscle weakness, facial itching
  - Citrate chelation
  - Calcium replacement
- Hyperammonemic
  - Hepatic encephalopathy signs, decreased mentation, head pressing
  - Ammonia in stored cells
  - lactulose

To premed or not?

- Effect of premedication and other factors on the occurrence of acute transfusion reactions in dogs
  - Bruce, Kriese-Anderson, et al in JVECC 25(5) 2015
- 558 dogs, 935 transfusions, 144 reactions (14%)
  - Antihistamines, steroids
- Fever (53%)
- Vomiting (18%)
- Facial swelling, hives, itch
- Tachypnea, tachycardia
- 4 had acute hemolysis signs

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Questions?

Amatoxin Mushroom Poisoning: Treatment Guidelines for Poison Control Centers, Emergency Departments, and Hospital Physicians

1) Any patient presenting to the ED with the **delayed onset** of vomiting and diarrhea **six hours or more** following consumption of **foraged mushrooms** must be presumed an **amatoxin poisoning** *until proven otherwise*.[10,18] A careful history will determine a best estimate of the number of hours between ingestion and symptom onset, the number of mushrooms eaten, how they were prepared, and who else may have consumed them.

2) **Treatment** in the ED and hospital admission should be ***neither delayed nor postponed*** pending **mycological specimen identification**. Negative identification of specimens does **not** necessarily rule out amatoxin poisoning. Mycologists can play a helpful role, but their findings should *not* influence or postpone clinical judgment.

3) **Poison Control Center** advice should be *simple and concise*, focusing the treating ED physician on the **single essential task** of **aggressive fluid resuscitation. Preserving renal function** and **restoring a brisk urine output** allows for rapid **clearance and excretion of circulating amatoxin**. Doing so allows more time for the interventions that target amatoxin induced hepatotoxicity and extends the overall timeline of the poisoning.

Amatoxin is *readily absorbed* from the intestine with **~ 40%** rapidly reaching the **general circulation. Unbound to albumin** or other plasma proteins, it is **rapidly cleared** by the **kidneys** and deposited into urine.[2,10]

Amatoxin poisoned patients typically present with prerenal AKI (azotemia) secondary to the hypovolemia and renal hypoperfusion produced by many hours of *cholera like* diarrhea and repetitive vomiting. Despite significant fluid losses, the patient may be *neither* hypotensive *nor* even tachycardic.[1,4,10,15,16,19,22]

Patients who develop ***early*** *oliguric renal failure* (*ARF/Intrinsic AKI*) have an ***exceedingly high mortality rate***. If fluid deficits are not rapidly and aggressively replaced, **ATN** and ***oliguric*** **ARF** typically follow, rapidly devolving into ***irreversible*** **fulminant hepatic** and **multi-system failure.**

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Acute tubular necrosis **(ATN)** can occur simply on the basis of uncorrected hypovolemia and *prolonged* renal hypoperfusion resulting from a very late presentation. ***Hypoperfusion*** *induced* ***urinary stasis*** also gives amatoxin the opportunity to ***penetrate*** *and* ***poison renal tubule epithelial cells*** (*despite the absence of a facilitated entry mechanism similar to that of hepatocytes*), and therefore a second mechanism leading to **ATN** if a brisk urine output is not not rapidly restored (*Amatoxin induced ATN can also be a late sequela of poisoning, occasionally not even manifesting until hepatic recovery is already underway*).[4,10,22]

Elevated serum **lactate** values are the ***best early indication*** of occult **hypovolemia** & **hypoperfusion**. Marked *elevation of hemoglobin and hematocrit* due to **hemoconcentration**, and/or a **BUN to creatinine ratio > 10** are also typical.

Patients with a *very late* presentation following ingestion will have more obvious vital sign and laboratory abnormalities, and are likely to already have a ***diagnostic elevation*** of **transaminases** and a detectable coagulopathy.

4) An initial set of **laboratory tests** (*CBC, PT/INR, CMP, serum lactate*) should be obtained at presentation, followed by serial measurement of CMP, lactate, and PT/INR every 6 hours.

The presence or absence of **rapidly rising transaminases** (***AST, ALT***) over the 24 hours following presentation, will definitively establish or rule out the diagnosis of amatoxin poisoning.[1,10,15,18]
A rapid and specific ***diagnostic urine test*** is **not available** for clinical use.

Serially obtained **lactate and (later) INR** values **predict prognosis** and help guide further treatment decisions.[1,7,14,15,18]

Uncorrected **serial lactate values are the earliest and most** ***sensitive*** **indication of a poor prognosis.**

Serial **lactate** and **osmolality** levels will indicate the ***adequacy of volume and free water replacement***. Lactate levels will drop to normal values with sustained aggressive intravenous fluid administration as urine output is restored and renal function is preserved.

Elevated serum **lipase** values may occur but are *unlikely to indicate bonafide pancreatitis* unless values exceed at least 3x or more the upper limit of normal.

5) **Fluid Resuscitation:** Insert large bore IVs and consider placing a *multi-lumen central line* or *PICC* along with a ***Foley catheter***. Begin aggressive *volume* replacement with **0.9% NS**, bolusing by the liter (*rapid infusion of 3-4 liters of normal saline*) until hypotension and tachycardia resolve, and *satisfactory urine output has been established.* Then convert to **D5 0.45% NS with 20 meq KCL** at an *elevated* maintenance rate (***200-300 ml/hour in adults***) in order to replace *free water* and provide glucose while maintaining a brisk urine output (*100-200 ml/hour*).
The addition of bicarbonate may be considered for a severe metabolic acidosis (*ph < 7.1*), but understand that the ***lactic acidosis of presentation will correct only with sustained aggressive intravenous hydration.***

Sustained aggressive intravenous hydration *alone* will lead to a **full recovery** in most patients with a **mild to moderate ingestion**. Onset of vomiting and diarrhea will be delayed longer (*12 hours or more post ingestion*), but the hypovolemia and hypoperfusion at ED presentation is typically no less severe. ***Such patients may still suffer poor outcomes from a very late presentation or when early aggressive hydration is not provided. Either of these two scenarios can lead to ATN and oliguric ARF*.**

Amatoxin poisoning patients transferred to *Tertiary Care Liver Transplant Programs* have been demonstrated by the **ALFSG** to be at extremely **high risk** (75%) of a poor outcome secondary to the ominous development of ***early oliguric renal failure*** heralded by ***uncorrected*** *serum* ***lactate elevations***.[5]

Discontinuation or restriction of maintenance intravenous hydration has been a frequent occurrence following transfer to Tertiary Care Liver Transplant Programs, invariably leading to early oliguric renal failure. Once admitted onto a tertiary transplant service, amatoxin poisoned patients have typically been *managed like those acquiring acute liver failure from much more commonly encountered etiologies like acetaminophen/paracetamol poisoning and acute fulminant viral hepatitis*. Minimal hydration is provided for fear of inducing *cerebral edema secondary to increased intracranial pressure*, an ominous but *late* complication of fulminant hepatic failure. **Unfortunately, in amatoxin poisoning this strategy results in the *loss of amatoxin clearance from the circulation*, and the rapid development of oliguric renal failure.** Moreover, IV **silibinin** is also rendered **ineffective** when oliguric renal failure occurs.

Failure to convert to D5 0.45%NS from 0.9% NS once vital signs normalize and urine output is reestablished can lead to fluid overload and pulmonary edema. Once vital signs normalize and urine output is reestablished, further volume expansion from Normal Saline is not necessary and can lead to hypoxemia and pulmonary edema, *even in patients with a normal cardiac output and no underlying cardiac disease*.

6) **All strongly suspected or confirmed cases should be referred *immediately*** to the ***Legalon SIL*** **IND Hotline** (*USA toll free*: **866-520-4412;** otherwise **(001)-412-563-1400**) for consultation with the PI and the provision of **Intravenous Silibinin.** The drug is currently *provided and shipped free of charge* by the IND sponsor. Prospective data from the Open IND indicates that silibinin can be safely and effectively initiated within 96 hours of ingestion in patients with preserved renal function. However, *delay is not advantageous* and will result in a more substantial hepatic injury.
Silibinin should be initiated as quickly as possible for any strongly suspected or confirmed amatoxin mushroom poisoning. Earlier initiation will result in a far less severe injury to the liver. Its effect in *arresting the progression of fulminant hepatic failure* is *evident by ~ 36 hours into infusion* when a *dropping INR, heralding recovery,* will be observed.

Silibinin appears to be ***completely ineffective*** when administered to patients who are *oliguric or anuric* due to the development of **early oliguric renal failure.** The **outside window** for silibinin efficacy appears to require drug initiation before ~ **96 hours** post ingestion**.**

The primary mechanism of action for silibinin is the *inhibition of amatoxin re-uptake by hepatocytes,* blocking membrane transport receptors (***NTCP, OATP1B3***) responsible for the enterohepatic recycling of bile salts.[8,13] This blockade results in the *diversion of amatoxin back into the general circulation for renal clearance*; therefore *maintaining renal function and a brisk urine output is crucial* to the success of the drug. Silibinin appears to *attenuate the process of apoptosis* in the amatoxin damaged liver. Silibinin has antioxidant effects and stimulates RNA polymerase I.[15] Silibinin treatment is strongly supported by studies in beagles.[23,33]

Prospective data collected since 2007 indicates that as long as renal function is preserved, a brisk urine output restored, and infusion begun before 96 hours post-ingestion, **silibinin has been associated with an INR reduction by ~ hour 36 of infusion that is sustained, heralding a rapid and complete recovery for *every* patient that has undergone the treatment.**[7,9,15]
To reiterate: ***100%*** *of the patients with preserved renal function from aggressive intravenous hydration treated with intravenous silibinin within 96 hours of mushroom ingestion have made rapid and complete recoveries in the current IND Clinical Trial.*

Silibinin can be *reconstituted in D5W or NS*. It is given as a **5 mg/kg bolus over one hour** followed by a **continuous infusion of 20 mg/kg/day.** Doses up to 50 mg/kg/day have been well tolerated in previous studies. The only commonly observed adverse effect is a flushing sensation during the initial bolus similar to but generally less intense than that of niacin. It is otherwise extremely well tolerated.[9,15]

Once intravenous silibinin is initiated, one can expect INR levels to continue rising for the next 24 hours. The INR will begin dropping in a sustained manner by ~ hour 36 of infusion, a drop that always heralds full recovery in the absence of acute oliguric renal failure. Infusions of more than 72-96 hours are rarely necessary.

7) **Limiting hepatocyte exposure** and preventing further damage to the liver from subsequent rounds of enterohepatic circulation are *vitally important treatment goals*. The means by which bile is accumulated, concentrated, and released presents an opportunity to ***sequester amatoxin in the gallbladder*** and prevent its release into the intestine.

Suspending the enterohepatic circulation of amatoxin in order to prevent the subsequent poisoning of uninvolved hepatocytes can be effectively accomplished by: a) keeping patients **NPO;** and b) administering intravenous **OCTREOTIDE**. Octreotide helps keep amatoxin contained in the **gallbladder** where it can cause no further harm.

Following ingestion amatoxin retains all of its poisonous potency. Amanitins are impervious to the acidic environment of the stomach and unfazed by digestive proteases. They undergo neither metabolic transformation nor detoxification by the liver or kidneys. **~ 60%** of the amatoxin **absorbed** following mushroom ingestion **enters hepatocytes** from the *portal circulation* via sinusoidal membrane transport systems (***NTCP, OATP1B3***) intended for *bile salt recycling*.[8,13] Amanitins poison hepatocytes and are also rapidly excreted into the bile. The long latency between mushroom ingestion and GI symptom onset often allows time for one to have eaten one or more subsequent meals, *often of the very same mushrooms*.

With each meal the gallbladder contracts and releases concentrated bile into the duodenum. Small amounts of bile also enter the duodenum between meals or during the fasting state in a process known as the *migrating motor complex* (*MMC*). **~ 60% of the amatoxin that is released through the Sphincter of Oddi will undergo enterohepatic circulation and *poison hepatocytes previously missed*, while the other 40% enters the general circulation**.[2,10]

Octreotide effectively **inhibits bile outflow** from the common bile duct and gallbladder by **raising pressure at the Sphincter of Oddi**.[28] Octreotide **enhances GB filling** by **reducing GB intraluminal pressure** resulting in the accumulation of a more highly concentrated bile salt content, while also **preventing GB contraction** and release of GB contents.[29, 30, 31] Octreotide **induces *duodenal* Phase III MMC contractions**, a further barrier to interdigestive hepatic bile entry.[29] Octreotide is very *well tolerated* with uncommonly reported adverse effects that include abdominal cramps, nausea, vomiting, diarrhea, and constipation.

Octreotide is dosed with a **200 mcg intravenous bolus** followed by a **continuous infusion of 50 mcg/hour**. Octreotide may be safely discontinued once urine output has been restored, renal function preserved, and intravenous silibinin infusion (*if available*) successfully underway with a dropping INR, and/or following successful removal of amatoxin laden bile via ultrasound guided simple gallbladder aspiration.

8) **Activated charcoal** is ***not*** **recommended** for routine use. It may be helpful if given *very* early (*shortly after ingestio*n) but this is rarely possible because patients usually do not seek medical attention until several hours *after* becoming symptomatic. **Multi-dose activated charcoal** (*mdac*) has **not** been shown to improve outcomes and may be quite difficult for symptomatic patients to tolerate. **Nasogastric** or **nasoduodenal tube** placement *provides no clinical benefit* and may be very difficult for symptomatic patients to tolerate.

There is **no role** for **extracorporeal purification** procedures like hemoperfusion, hemodialysis, plasmapheresis, etc.[1,19] Liver purification systems like *MARS* and *Prometheus* are expensive and not widely available, but may have value as a temporizing measure prior to liver transplant, buying additional time for native liver recovery, especially in the setting of oliguric renal failure and hepatic encephalopathy. However, such measures are unnecessary for amatoxin clearance from the circulation and are unable to remove it from the liver and biliary tract.

9) **NAC** and **Penicillin G** are ***not*** **recommended** for patients presenting with amatoxin poisoning.

NAC has shown *no benefit in animal studies.* Despite near universal use for amatoxin poisoning over the past two decades, NAC has had *no demonstrated effect on outcomes*.[21] The **ALFSG** publication supporting the use of NAC for *non-acetaminophen associated acute liver failure* included no patients with *amatoxin poisonin*g.[12]
NAC consistently *raises* ***PT/INR*** values during intravenous infusion, potentially *clouding the clinical picture* and *adversely affecting clinical decision making*.[11,17]
Penicillin G can be toxic at the very high doses recommended and has also not been shown to improve clinical outcomes.[1,19]

Recommendations to use charcoal or to begin Penicillin G and/or NAC while patients are undergoing treatment in the ED are particularly ***unhelpful*****,** unintentionally serving to ***distrac*****t** the ED from the singularly essential task of undertaking aggressive intravenous fluid replacement and the establishment of a brisk urine output.

10) **All suspected cases should be admitted** for *diagnostic confirmation* via serial transaminases, *sustained aggressive hydration*, and close *monitoring of urine output*. A **multi-lumen central line** or **PICC** is desirable in order to maintain patient comfort during frequent blood draws, assure continuous IV access, and to allow for the easy addition of octreotide and silibinin by pump infusion.
Foley catheter placement allows unimpeded diuresis, precise monitoring of urine output, and prevents medication induced urinary retention. Potentially nephrotoxic medications like Vancomycin should be avoided.

If oliguria and early ARF develop, a poor outcome is virtually certain. Progression to fulminant hepatic and multisystem failure can be a breathtakingly rapid and malignant development.[7,20]

The immediate goals of **treatment in the ED** are the ***rapid correction of hypoperfusion*** to *prevent renal injury* and the ***rapid restoration of urine output*** to allow for the *elimination of amatoxin from the general circulation*.[4,10,15,16,22] **Lactate levels should be corrected quickly** along with the resolution of hemoconcentration and a rapid reduction in BUN and creatinine levels toward goal values of < 10 and < 1.0.

Elderly patients are at *particularly high risk* of developing *early* **ARF.**
Like everyone else with amatoxin poisoning, ***the elderly also require aggressive intravenous hydration to survive.*** Age related reduction in renal reserve and preexisting conditions like diabetes, hypertension, CHF, and intrinsic renal disease can make the process seem much more complicated. If the presentation **BNP** is elevated or **atrial fibrillation** is noted on ECG, an **echocardiogram** may be obtained to measure ejection fraction *in anticipation of potential fluid overload*. Those with an identified risk require close monitoring of vital signs and oxygen saturation along with serial BNP measurements to help assess the need for additional diuretic therapy in case of fluid overload.
Failure to adequately provide sustained aggressive intravenous hydration for an elderly amatoxin patient invariably leads to a poor outcome. *Fluid overload is readily treatable and reversible; ATN and ARF are not.*

Fresh frozen plasma provides *temporary* correction of coagulopathy at the expense of ***losing*** the ***crucial prognostic significance of serial PT/INR values***. Administration in response to serious bleeding complications and prior to invasive procedures makes good sense, but *routine administration in response to an asymptomatic coagulopathy should be avoided.*[1,19]

Poor outcomes have occurred in several cases where an *in charge* treating physician with overall responsibility for decision making could not be easily identified, as in institutions where daily or even more frequent hospitalist turnover is the norm. Having a Clinical Toxicologist or Hepatologist involved may be desirable, but more critical is having a ***physician in charge who will provide continuity*** over several days and nights.

12) **Biliary drainage** by Interventional Radiology (*Simple/Serial Gallbladder Aspiration; Percutaneous Cholecystostomy*), General Surgery (*Open Cholecystostomy*), or GI (*Nasobiliary drainage with suction placed by ERCP*) targets the **gallbladder** where amatoxin laden bile accumulates following its excretion by hepatocytes.[3,6,14,16]

Removing amatoxin laden bile from the gallbladder provides definitive protection to uninvolved hepatocytes by eliminating further enterohepatic circulation of the poison. The bile aspirated from the gallbladder of a patient with amatoxin poisoning will have a characteristic appearance that looks similar to *burnt motor oil.*

Biliary drainage has been demonstrated to be effective in beagles and in a growing number of case reports.[3,6,14,16] The actual amount of amatoxin contained in the bile removed by such methods is currently under active investigation, but published **HPLC** evaluation suggests the yield to be substantial.

Aggressive intravenous hydration combined with biliary drainage appears to be an especially promising strategy in developing countries and other hospital settings when intravenous silibinin is unavailable.[16] We now recommend that **all cases in developing countries** undergo **simple ultrasound guided gallbladder aspiration,** after the patient has been stabilized and the diagnosis has been confirmed, in any hospital setting where SIL is *not* obtainable,

Biliary drainage may also be considered, *in addition to silibinin,* for patients at particularly *high risk* due to short latency (*6-10 hours*) between mushroom ingestion and symptom onset, a very large ingestion, the rapid development of a significant coagulopathy, or the early development of complications like oliguric renal failure.

Abdominal ultrasound should be obtained to evaluate for the presence and condition of the **gallbladder** when considering biliary drainage.

Simple Ultrasound Guided Gallbladder Aspiration appears to be the fastest, safest, easiest, and most efficient means of permanently removing accumulated amatoxin from the biliary tract. It can be accomplished using a *transhepatic* approach early in the clinical course before a significant coagulopathy has developed, or via a *transperitoneal* approach if the INR is above 2. The method can be accomplished at the bedside with local anesthesia using a syringe and an 18-20 gauge long spinal needle with trocar.

Simple GB aspiration should be *performed as soon as possible following presentation,* once the diagnosis is confirmed and the patient has been stabilized with aggressive hydration along with an **octreotide** infusion. The *relative* ***risks*** *of* ***bleeding***, ***infection***, *and* ***bile peritonitis*** *are substantially reduced* in comparison with drain placement. Simple aspiration can be repeated 24-48 hours later once follow up ultrasound has confirmed an accessible re-expanded gallbladder.

Percutaneous Cholecystostomy drain placement by an Interventional Radiologist or a General Surgeon requires that the tube remain in place for *at least* 10-14 days or more until a mature tract has formed, in order to minimize the risk of ***bile peritonitis*** when the drain is removed. Other risks include ***bleeding***, especially if coagulopathy is already present when the procedure is performed, and ***infection***, particularly with a tube that must be left in place for several days to weeks.

If **Nasobiliary Drainage** is performed by **ERCP**, the endoscopist must be *warned beforehand* ***not to cut a sphincterotomy***, as doing so could precipitate a ***coagulopathy mediated hemorrhage***. Wall **suction** must be applied to the drain to assure retrograde emptying of the gallbladder. ***Post-ERCP pancreatitis***, although usually mild and self-limited, may occur in up to 5% of cases. ERCP also requires general anesthesia or at least very heavy sedation.

No matter which method of biliary drainage is utilized, ***all removed bile should be carefully collected, labeled and then frozen for subsequent analysis of amatoxin content*.**

Biliary drainage will not affect a patient's candidacy for liver transplant should the need arise.

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Prevention and Treatment of Amatoxin Induced Hepatic Failure With Intravenous Silibinin (Legalon®SIL): A Nationwide Open Clinical Trial
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"todd mitchell", "mushroom poisoning"

1) Presentation with **delayed onset** of ***vomiting & diarrhea*** **six hours or more** after eating **foraged mushrooms** must be presumed an **amatoxin poisoning**. Treatment in the ED and hospital admission should be **not** be delayed pending identification by a mycologist.

2) **Poison Control Center** advice should focus the ED on the **single essential task** of **sustained aggressive IV hydration.** Preserving renal function & restoring urine output allows rapid clearance and urinary excretion of circulating amatoxin, extending the overall timeline of the poisoning. **Activated charcoal**, **NAC** & **Penicillin G** are **not** effective & **not** recommended.

3) **Labs** (*CBC, PT/INR, CMP, serum lactate*) may be repeated every 6-8 hours. *Rapidly rising* transaminases (***AST, ALT, LDH***) over the next 24 hours will establish or rule out the diagnosis. **Hemoconcentration** & **serum lactate elevation** are **typical** at presentation.

4) **Minimize renal transit time with sustained aggressive IV hydration** to reduce risk of toxin mediated kidney injury (*ATN*) & rapidly eliminate circulating amatoxin. Place 2 large bore IVs (f/b *multi-lumen PICC/central line*) & ***Foley catheter***. Bolus **0.9% NS** (*rapidly infuse 3-4 liters*) to correct vital signs & establish good urine output. Then convert to **D5 0.45% NS with 20 meq KCL** at elevated rate (*200-300 ml/hour in adults*). Goals: sustain brisk urine output (*100-200 ml/hour*); sustain serum lactate correction; BUN < 10 & Creatinine < 1.0.

5) **Suspend enterohepatic circulation:** a) Strict **NPO** status; b) IV **Octreotide** (*200 mcg bolus f/b 50 mcg/hr continuous infusion*) raises Sphincter of Oddi pressure, prevents gallbladder contraction & protects hepatocytes from further amatoxin exposure. Keeps hepatobiliary amatoxin sequestered in the gallbladder. D/C Octreotide & feed once recovery underway.

6) **Refer all** strongly suspected or confirmed cases **asap** to the ***LegalonSIL*** **IND Hotline:** 866-520-4412 or 1-(412)-563-1400. **Intravenous Silibinin** (*5 mg/kg bolus f/b 20 mg/kg/d continuous infusion*) produces sustained INR reduction by the 36th hour of infusion that reliably heralds rapid clinical recovery *as long as renal failure has been avoided*. Hospitalization is < 7 days and requires no ICU time or tertiary care transfer. *Provided and shipped free of charge* in the USA by the IND sponsor. Warmth and flushing are the only observed adverse effects.

7) In hospital settings where **LegalonSIL** is ***not*** **obtainable**, undertake simple needle (*19-21g*) over trocar **Ultrasound Guided Gallbladder Aspiration** *after* the diagnosis is confirmed by rising transaminases. The relative ***risks*** of ***bleeding***, ***infection***, *and* ***bile peritonitis*** are markedly reduced in comparison with percutaneous cholecystostomy drain placement. Transhepatic approach if INR < 2, otherwise transperitoneal. Amatoxin laden bile has the appearance of burnt motor oil. May repeat once after 24-48 hours. Freeze all recovered bile for later analysis.

Track 3

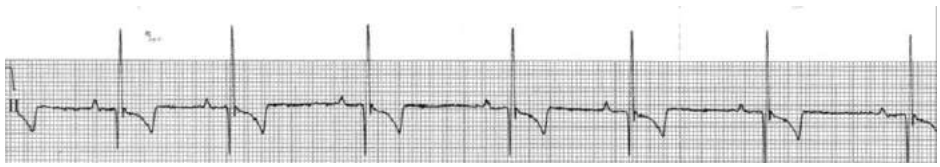
ECG Basics

Sara Johns, DVM
DACVIM (Cardiology)

Sinus rhythm

- 1. Is there a p for every QRS?
- 2. Is there a QRS for every p?
- 3. Is there a consistent P-R interval?

- If yes → this is a sinus rhythm!
  - *Regardless of the appearance of the QRS*



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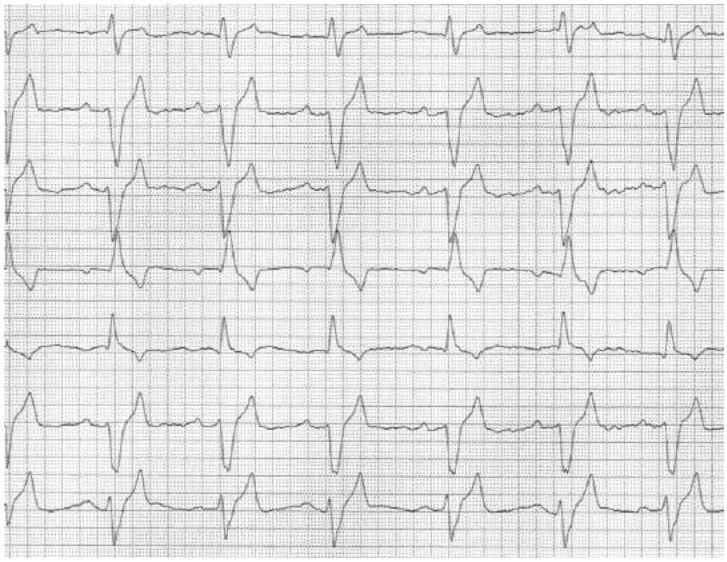
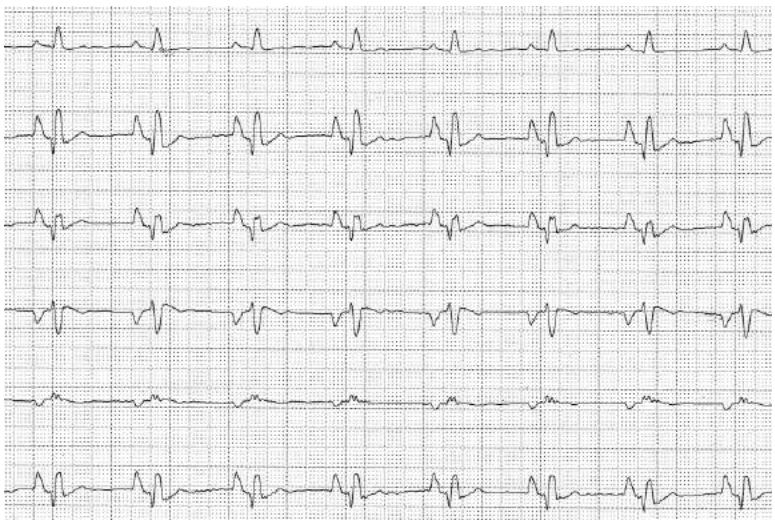
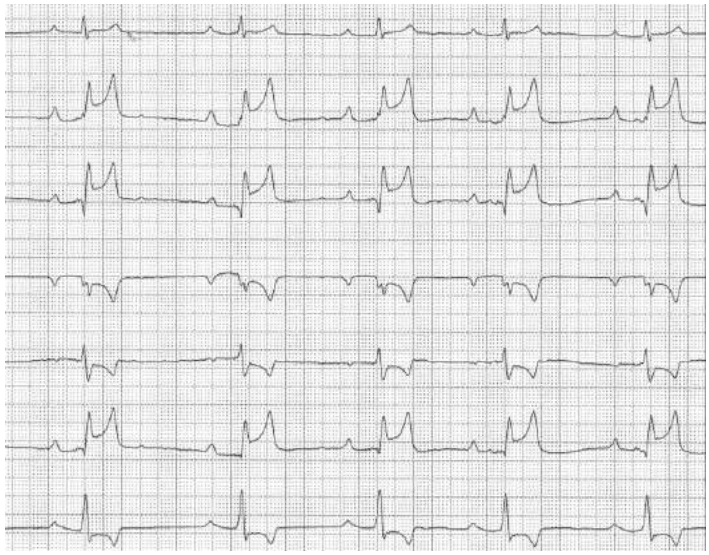
Sinus rhythm



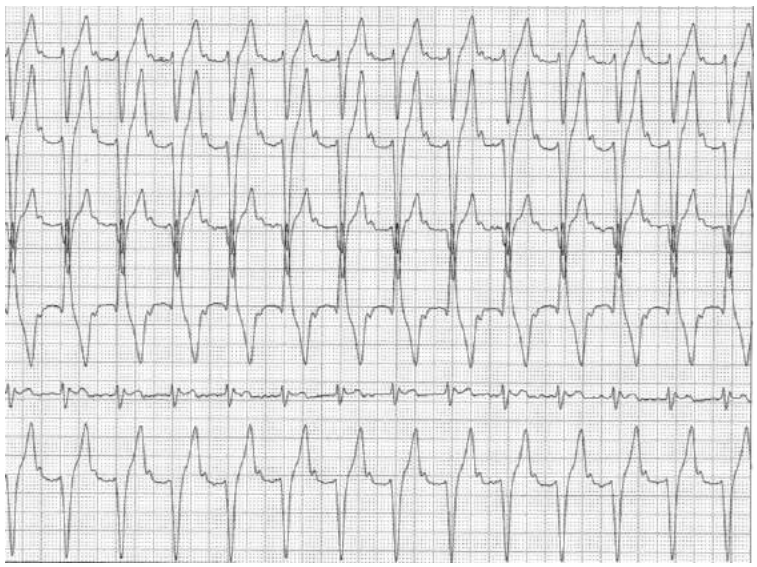
Sinus rhythm



Sinus rhythm



Sinus rhythm



Tachycardias

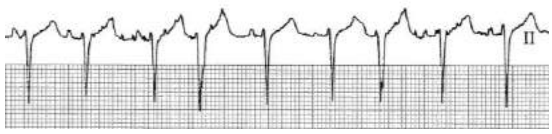
- 4 anatomic locations
  - 1. Sinus
  - 2. Atrial
  - 3. Junctional
  - 4. Ventricular

Tachycardias

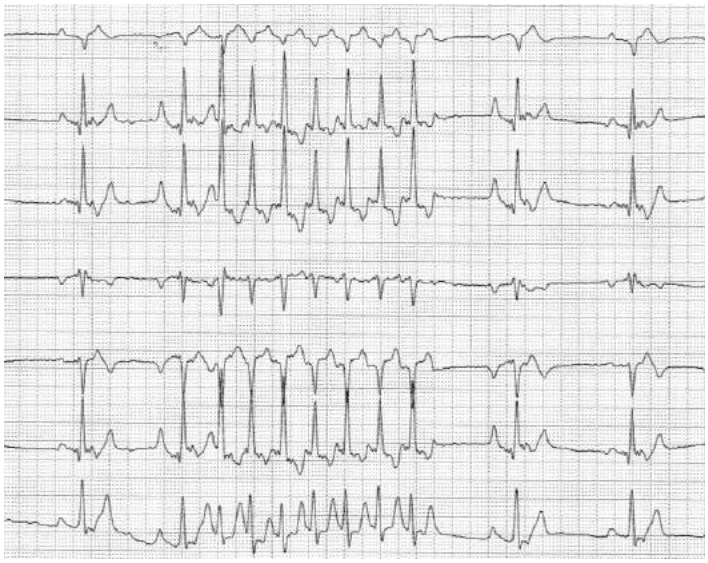
- 4 anatomic locations
  - 1. Sinus
  - 2. Atrial
  - 3. Junctional
  - 4. Ventricular

SVT

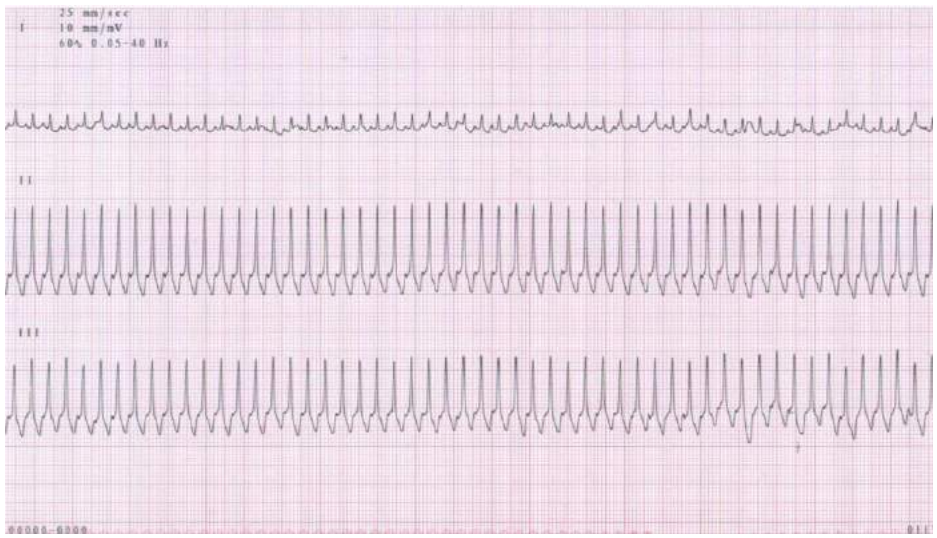
Normal QRS (or same as sinus)
Abrupt onset & termination, or sustained
Can be very fast (300-400bpm)



Supraventricular Tachycardia



Supraventricular Tachycardia



What to do?

- Referral to cardiologist recommended
- Investigate underlying cause
  - Structural heart disease
  - Heart base tumor
  - Primary electrical problem
- Oral antiarrhythmic therapy might be warranted

Tachycardias

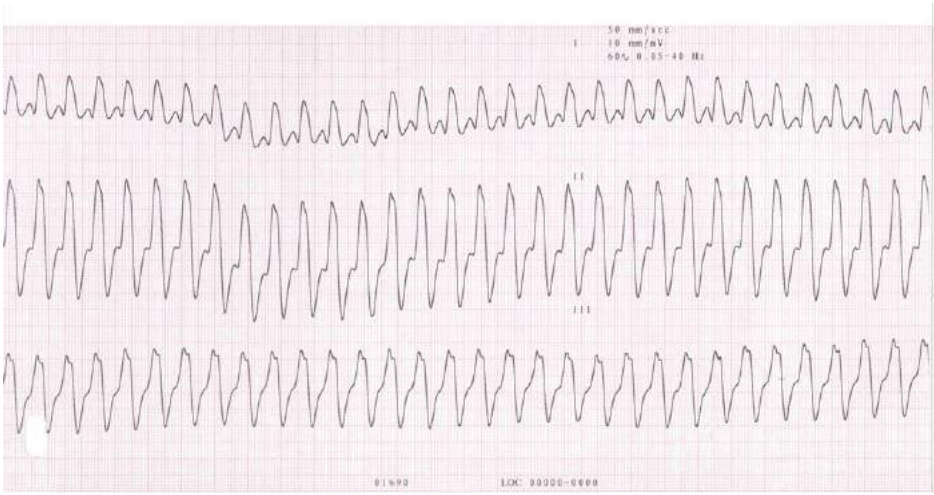
- 4 anatomic locations
  - 1. Sinus
  - 2. Atrial
  - 3. Junctional

  SVT
  - 4. Ventricular
    - P waves are present but dissociated from QRS
    - Wide and bizarre QRS
    - *Ventricular tachycardia is an emergency*

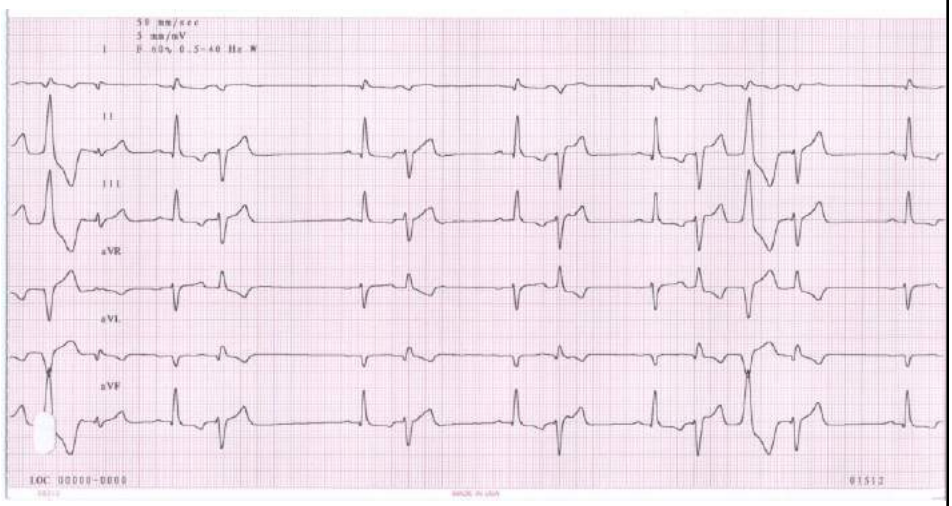
Ventricular rhythms

- Criteria for treatment
  - Rate >160 bpm (tachycardia)
  - Multifocal
  - R on T
  - Clinical signs

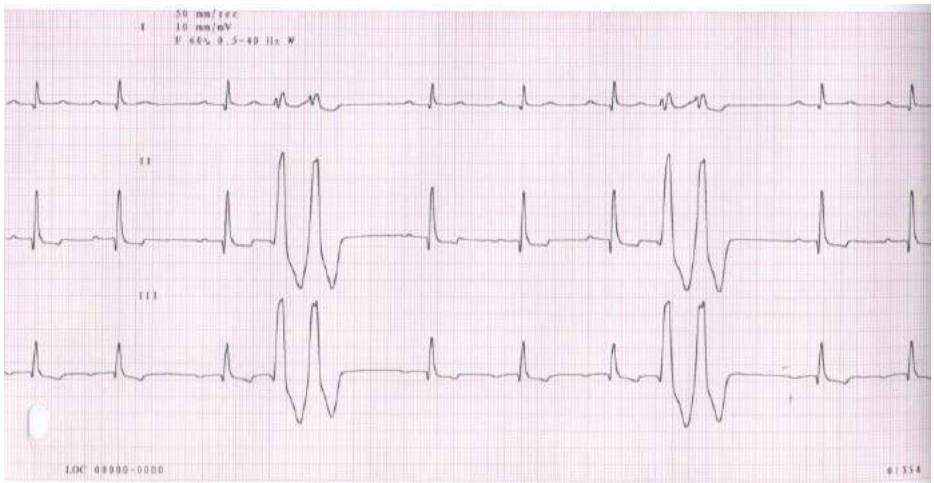
Ventricular tachycardia



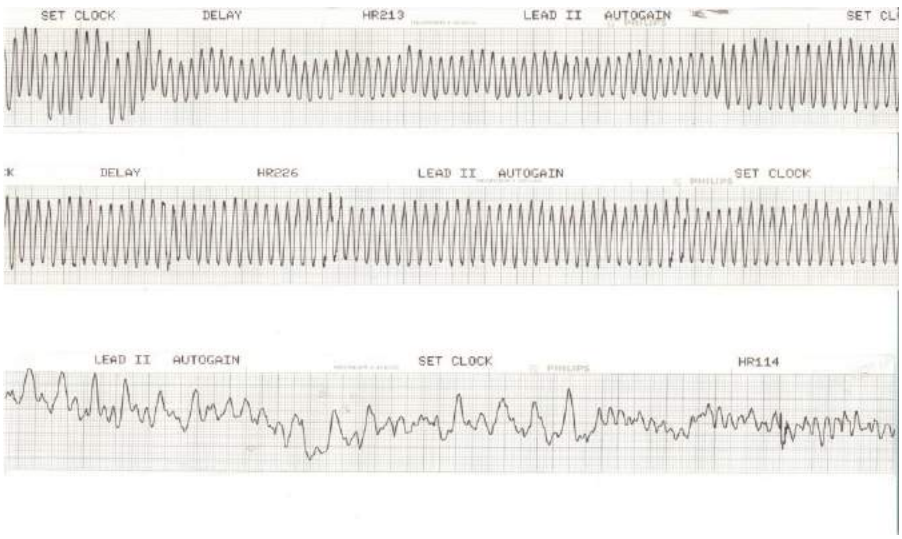
Multifocal VPCs



R on T



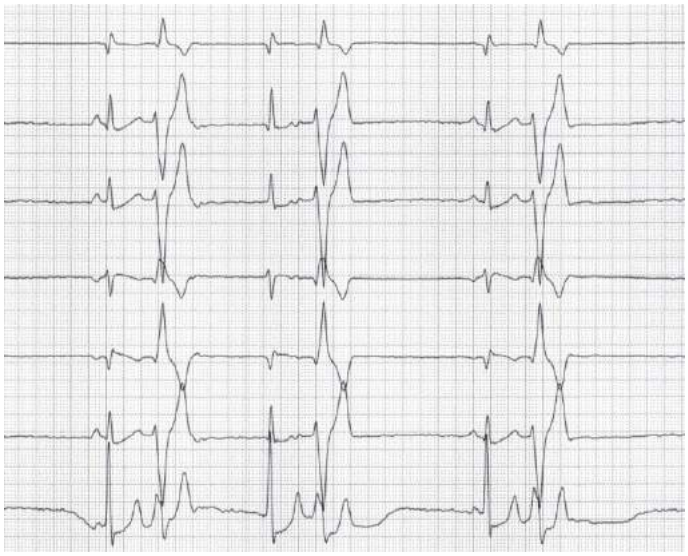
R on T



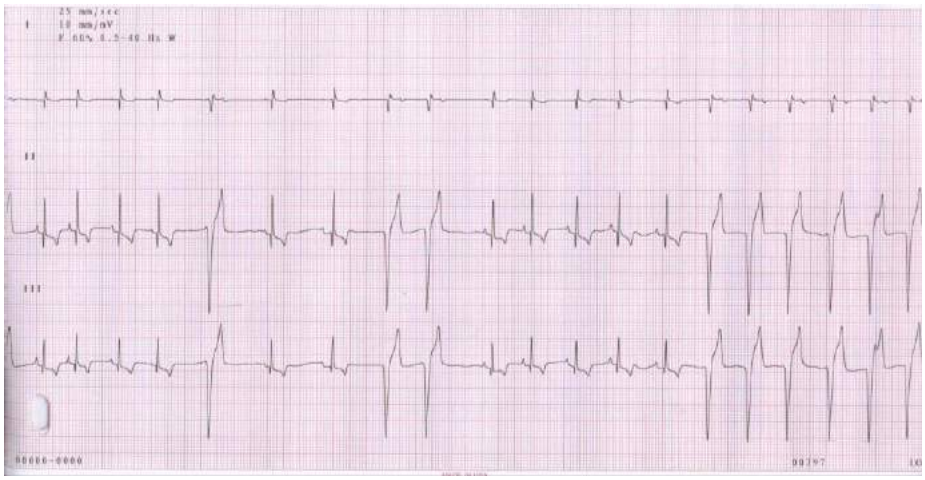
What to do?

- Lidocaine bolus (2mg/kg IV)
  - Dog's weight in kgs/10 = lidocaine volume
    - Ex: 35kg dog needs 3.5 cc lidocaine
- Lidocaine CRI
- Oral antiarrhythmic therapy warranted
- Investigate underlying cause
  - Structural heart disease
  - Primary electrical problem
  - Just about anything

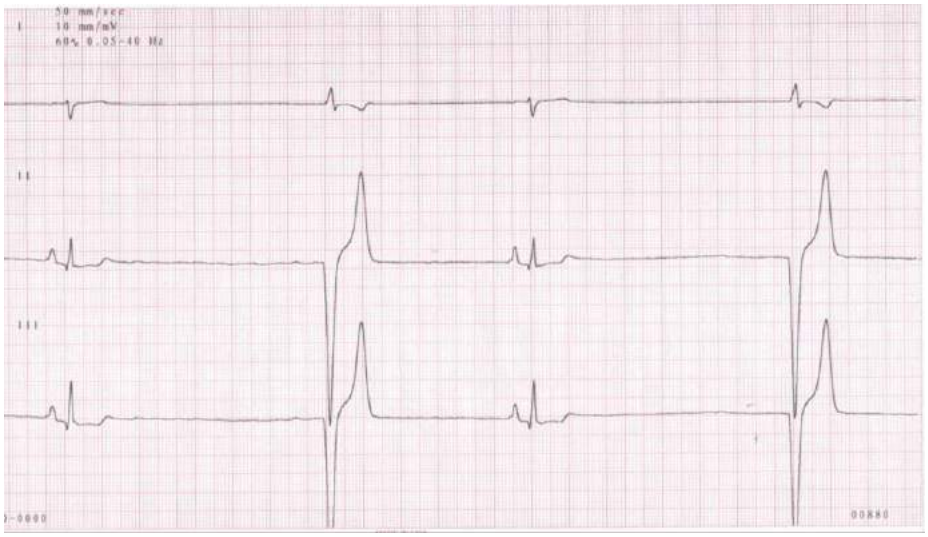
Stable ventricular rhythms



Stable ventricular rhythms



Ventricular escapes

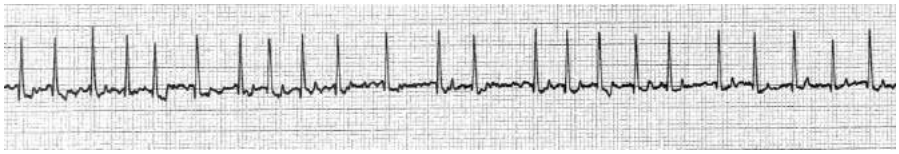


Tachycardias

- Sinus
- Atrial
- Junctional
- Ventricular

Regular

- Irregular tachycardia = Atrial fibrillation

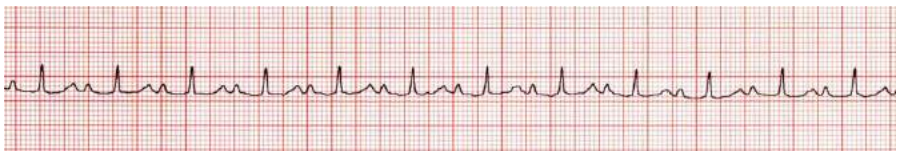


Bradycardia

- Sinus
- AV block
  - 1st degree
  - 2nd degree
  - 3rd degree

Bradycardia

- Sinus
- AV block
  - 1st degree
    - Prolonged p-r interval
    - This is not auscultable



Bradycardia

- Sinus
- AV block
  - 1st degree
  - 2nd degree
    - Type 1
    - Type 2
    - High grade
  - 3rd degree

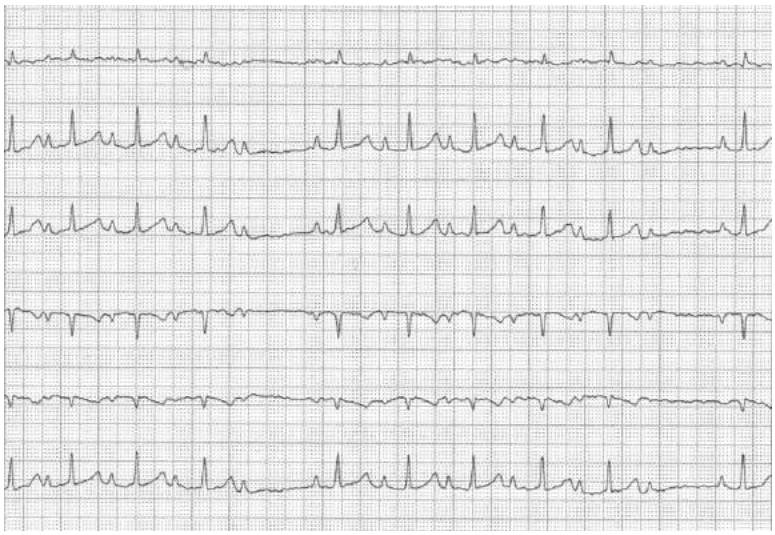
Low grade

Some p waves are conducted, some aren't
-p for every QRS
-but not a QRS for every p

Bradycardia

- Sinus
- AV block
  - 1st degree
  - 2nd degree
    - Type 1
      - Progressively prolonged p-r
      - High vagal tone
    - Type 2
      - Consistent p-r
      - AV nodal disease
    - High grade
      - AV nodal disease
      - These patients can be profoundly bradycardic
  - 3rd degree

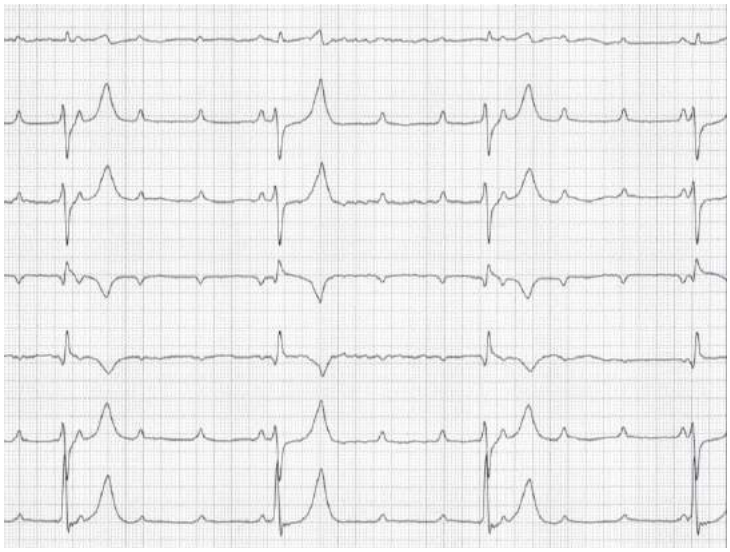
2nd degree AV block



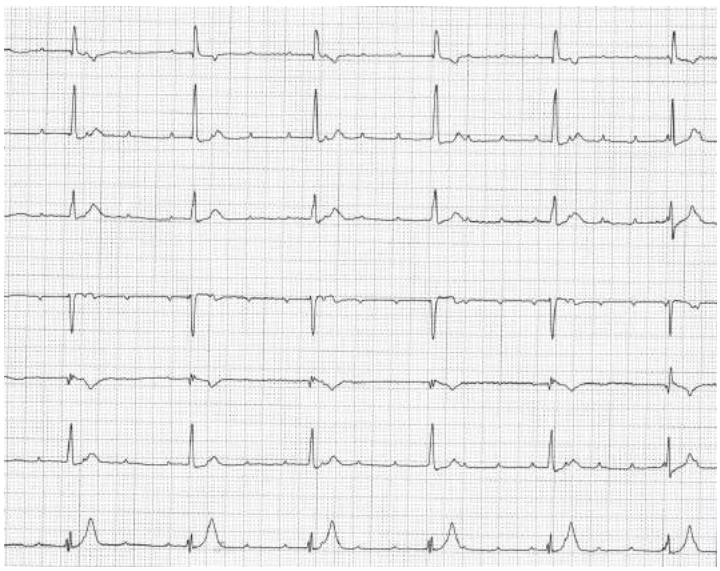
Bradycardia

- Sinus
- AV block
  - 1st degree
  - 2nd degree
  - 3rd degree
    - Complete dissociation
    - Patients are usually profoundly bradycardic
    - This is an urgent referral for pacemaker placement, as patients are at risk for sudden death (even if no signs)

3rd degree AV block

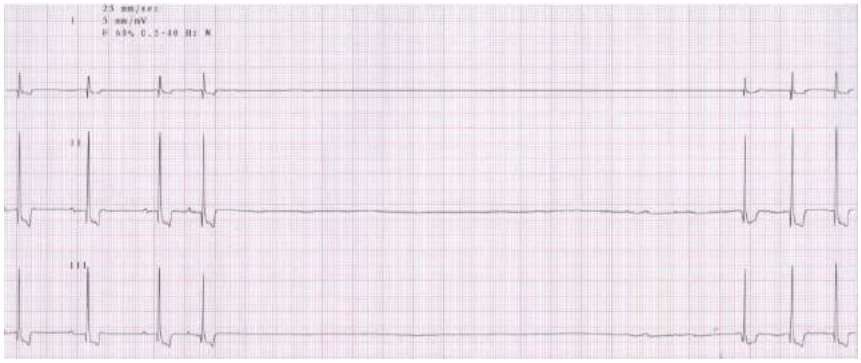


3rd degree AV block



Bradycardia

- Sick sinus syndrome



What to do?

- Sinus bradycardia, low grade 2nd degree AV block

  ➡ Atropine response test (0.04mg/kg)

Positive
(conversion to sinus tach)

What is causing high vagal tone?
-GI, respiratory, CNS disease
-Normal variant, athletic animal

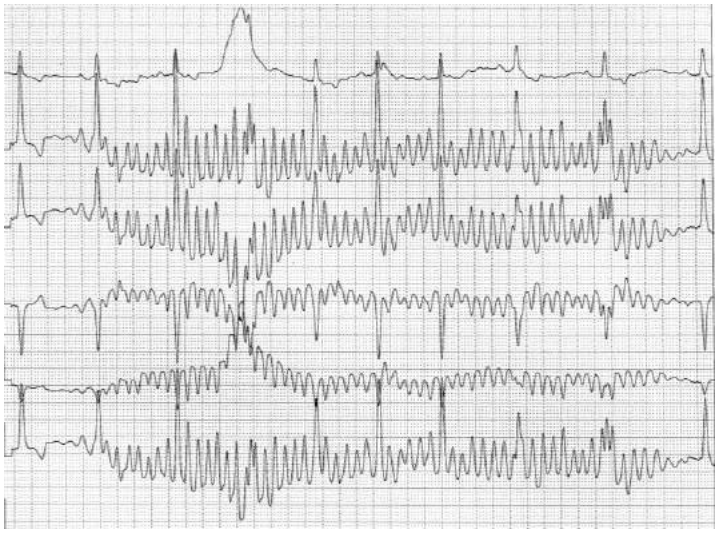
Negative

Referral

What to do?

- High grade 2nd degree, 3rd degree AV block
  - Pacemaker
- Sick sinus syndrome
  - Medical management
  - Pacemaker

Artifact!



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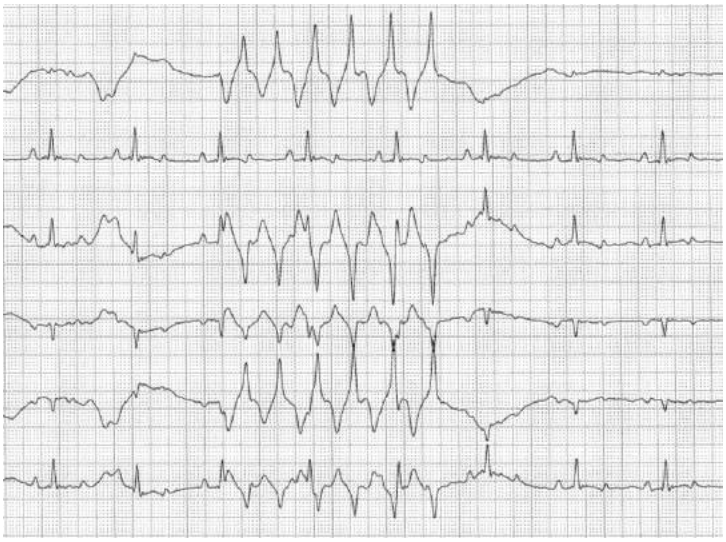

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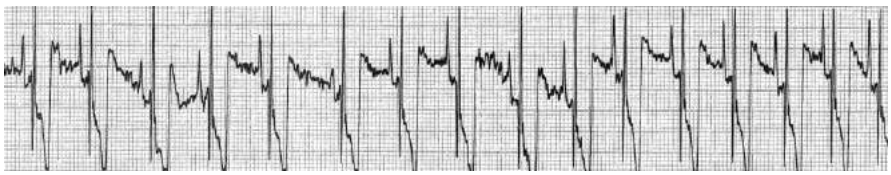

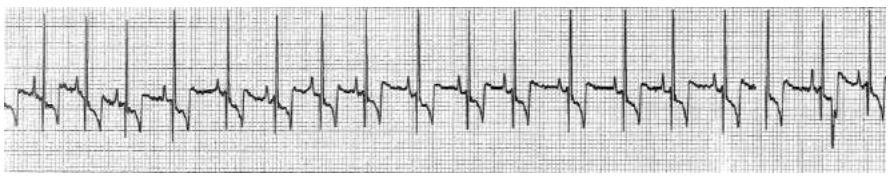

Cardiovascular emergencies: The approach to the patient in respiratory distress

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Definitions

Respiratory difficulty

- Tachypnea: increase in respiratory rate
- Dyspnea: labored breathing
- Hyperpnea: Increased depth of breathing
- Orthopnea: severe respiratory distress where the animal resists dorsal or lateral recumbency

Heart Failure = a physical state in which the cardiac output is inadequate to meet the needs of the metabolizing organs *despite adequate preload*. Heart failure is physically characterized by signs of low cardiac output (hypotension) and/or signs of sodium and water retention (congestive heart failure = "CHF")

Pulmonary hypertension = a persistent, abnormal elevation in pulmonary systolic or diastolic pressure greater than ~31/19 mmHg

Respiratory difficulty – Causes:

- Pulmonary parenchyma
  - Pulmonary edema (LCHF)
  - Pneumonia
  - Fibrosis
  - Heartworm
  - Thromboemboli
- Pleural cavity
  - Chylothorax
  - Pneumothorax
  - Pyothorax
  - Hemothorax
- Pericardial effusion
- PPDH
- Upper airway
- Trachea
- Bronchi
- Trauma
- Ascites
- Neurologic disease

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Recognizing heart failure

- Good history taking
  - Prior history of cardiac disease?
  - Exercise intolerance
  - Difficulty breathing
  - Abdominal distension
  - Lethargic
  - Coughing
  - Collapse/Syncope

- Physical exam findings
  - Weak, depressed
  - Dyspnea
  - Tachycardia
  - Weak pulses
  - Arrhythmia
  - Murmur or gallop
  - Ascites
  - Jugular distension
  - Increased lung sounds/crackles
  - Cyanosis

Hints for coming to a diagnosis of left heart failure versus a suspicion of pulmonary hypertension…

*How many of the clinical signs noted above could be found in *BOTH left heart failure and pulmonary hypertension? Almost all of them!*

Clinical signs

- **Coughing*
- **Difficulty breathing/"raspy" breathing*
- **Lethargic*
- **Collapse/Syncope*
- **Exercise intolerance*
- *Abdominal distension/ascites*
- **Multiple presenting complaints*

*How many of the physical exam findings could be found in *BOTH left heart failure and pulmonary hypertension? Several of them!*

Physical exam findings

- **Murmur*
- *Split/abnormal second heart sounds - think pulmonary hypertension*

- **Pulmonary crackles*
- **Harsh/increased lung sounds*
- **Cyanosis*
- *Tachycardia - think left heart failure*
- *Ascites - right heart failure; with or without pulmonary hypertension as a cause of right heart failure*

Which of all the physical exam findings listed above are you more likely to see in a dog presenting in left heart failure versus pulmonary hypertension? tachycardia

Diagnosing heart failure

- Thoracic radiographs
  - Only if the patient is stable enough!
  - Treat for CHF first, prior to taking radiographs, if the patient is unstable
  - Radiographs in the dyspneic patient
    - Perform if the diagnosis is in question
    - Clinical signs do not add up to heart failure in a dyspneic patient
      - No crackles
      - No murmur, faint murmur
      - Febrile
      - No fluid obtained on thoracocentesis

- How to diagnose left heart failure on thoracic radiographs
  - Perform orthogonal views, remember to obtain a VHS
  - Correct sequence of events: (dog) cardiomegaly with left atrial enlargement, distended pulmonary veins, interstitial-alveolar infiltrates
  - Pulmonary infiltrates (alveolar, interstitial, vascular)
  - Pulmonary infiltrates due to heart failure are NOT nodular or bronchial
  - Exceptions to the rule exists: Ex: chordae tendineae rupture causing pulmonary edema - cardiac silhouette and pulmonary veins may measure normal

Diagnosing pulmonary hypertension

- The diagnosis CANNOT be made on thoracic radiographs
- Diagnosis is made with echocardiography or by cardiac catheterization
- The findings on radiographs can be consistent with or suggestive of PH, however cannot diagnose this disease
  - Enlarged main pulmonary artery or lobar pulmonary arteries
  - Tortuous pulmonary arteries (HW disease)
  - Right heart enlargement

Canine case examples will be discussed including diagnosis and therapy
Case 1: Patient presenting in respiratory distress due to left heart failure
Case 2: Patient presenting in respiratory distress due to pulmonary arterial hypertension

Therapy

- GOALS of heart failure therapy
  Ways to improve the quality of life and increase survival time
  - Relieve congestion and increase blood oxygenation
    - Diuresis
    - Vasodilation
    - Intubation
    - Relieve effusion
  - Improve cardiac pump function
  - Promote the forward flow of blood

- Acute congestive heart failure therapy
  - Oxygen (oxygen cage, nasal oxygen or flow-by oxygen)
  - DO NOT STRESS
  - If severe edema: intubation, suction and postural drainage may be necessary
  - The severity of CHF will determine how aggressive treatment will be in each patient
  - Standard **acute** "triple therapy":
    - Oxygen
    - Furosemide: Initial dose in dogs 2-4 mg/kg IV, IM; cats 1-2mg/kg IV, IM
    - Pimobendan 0.3mg/kg PO BID
  - Do not give ACE-inhibitors in the acute setting/during hospitalization
    - Questions to ask before starting ACE-I
      - Is the dog/cat eating well
      - Are the renal values normal
      - If the answer is no, wait 1 week and recheck prior to starting
  - Aggressive treatment: dobutamine, nitroprusside
  - Hydralazine

- Chronic congestive heart failure therapy
  - Furosemide
  - Pimobendan
  - ACE-i
  - Spironolactone

- GOALS of pulmonary hypertension therapy
  - Improve quality of life and increase survival times (range: days to >2 years)
  - Treat the underlying cause
  - Treat pulmonary hypertension directly
  - Sildenafil 1 mg/kg PO TID
  - If dogs survived the first week of therapy, the probability of survival to 3 months after starting therapy was 95% in one study, 84% survival at 6 months, and 73% survival at 1 year (WITH sildenafil)
  - Prior to sildenafil, median survival time was less than 4 days

Heritable Heart Disease

Andrew S. Waxman, DVM, DACVIM (Cardiology)

SAGE Symposium 2016

- Methods of Screening
    - There is no single/optimal method of screening recognized for most forms of heart disease. The mode of inheritance is often not known form the common forms of canine and feline heart disease. Commercially available genetic screening tests are not 100% sensitive or specific for the commonly screened diseases. Unfortunately echocardiographic and rhythm assessments have some subjectivity and results can be grey/equivocal.
        - Echocardiography
        - Electrocardiography/Holter Monitoring
        - Genetic testing

- Feline Cardiac Disease
    - Congenital-The most common forms of congenital heart disease are related to dysplasia of the mitral and tricuspid valves. Also common are ventricular septal defects. There are no strong breed associations for congenital heart disease in cats.
    - Acquired-Hypertrophic Cardiomyopathy is the prototypical acquired heart disease of cats. There are breed associations with commonality recognized in the Maine coon cat, ragdoll, Persian, and shorthair breeds. Cats being bred or owners with concern are advised to seek an annual auscultation and echocardiogram with a cardiologist. As HCM is adult onset a single screen is not able to completely clear an animal for breeding or risk for development of the disease. It is thought that HCM is a dominant trait in the Maine Coon breed indicating any affected animal should be removed from the breeding pool.
- Canine Cardiac Disease
    - Congenital
        - Patent Ductus arteriosus: The patent ductus arteriosus (PDA) is the most common congenital defect in the dog. Prevalence seems to maximize in several small dog breeds including the Maltese, toy and miniature poodle, and Shetland sheepdog. The PDA has been shown to be inherited in the poodle and suspected in other breeds. Affected animals should not be bred nor those known to have produced affected pups.
        - Pulmonic Stenosis: Pulmonic Stenosis is a common congenital defect in dogs and the cause is typically malformation of the pulmonic valve. Other less common forms involve sub- or supravalvular stenotic lesions and/or hypoplasia of the annulus. Many small to medium breed are commonly affected included terriers and schnauzers. It has been shown to be heritable in the beagle. Bulldogs have a fair showing of pulmonic stenosis, however, their stenosis can be complicated by a

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coronary artery anomaly (abnormal coursing coronary that wraps around rather than under the pulmonary artery).

- Subaortic stenosis: Subaortic stenosis or SAS seems to cluster in larger breed dogs and routine screening is recommended for the Newfoundland, Golden Retriever, Boxer, and Rottweiler. Breeding dogs should be cleared at 1 year of age by at minimum auscultation by a cardiologist or highly experienced veterinarian. Some breeder groups are recommending echo/Doppler studies for clearance.
- Tricuspid Valve Dysplasia: Tricuspid valve dysplasia or TVD is mainly seen in the Labrador retriever with some genetic studies confirming it is a heritable disease. While the disease has characteristic changes to the tricuspid apparatus the echo assessment of mildly affected/greyzone dogs is difficult.

- Congenital screening in the dog: The top 3 congenital defects when severe all carry with them a systolic murmur (also usually severe). For general health purposes the presence of a murmur of any grade warrants investigation by a cardiologist. For breeding purposes subtle abnormalities can be detected by echocardiography/Doppler assessment that may not generate a murmur. Some breeder groups recommend a cardiologist opinion even with the absence of a heart murmur.

- Acquired Heart Disease
  - Endocardiosis: Endocardiosis (Degenerative valve disease, Myxomatous valvular disease, Chronic valve disease) is the most common form of canine heart disease. It is seen in some degree in most senior dogs. Progression and risk for the development of clinical signs is variable per dog. The Cavalier King Charles Spaniel is predisposed to an earlier onset and fast progression than other breeds. As an adult onset process screening for early onset is difficult as early onset can still be after ideal breeding age.
  - Dilated Cardiomyopathy: Dilated Cardiomyopathy is seen in larger breed dogs such as the Doberman Pinscher, Irish Wolfhound, Great Dane, and Boxer dogs. This tends to be an adult onset disease process making screening for the process at younger ages difficult. Yearly screening by echocardiography and Holter monitoring is advised for breeding purposes.
  - Arrhythmogenic Right Ventricular Cardiomyopathy: Arrhythmogenic Right Ventricular Cardiomyopathy or ARVC is almost exclusive to the Boxer breed. It is characterized by specific forms of ventricular arrhythmias. Some affected dogs also have ventricular dysfunction mimicking dilated cardiomyopathy. 24 hour Holter monitoring and echocardiography are indicated as screening tools as well as diagnostics for symptomatic dogs.

A 21st Century Approach to Canine Pyoderma

Valerie A. Fadok, DVM, PhD, Diplomate, ACVD

Pyoderma is a common skin disorder in small animal practice. Yet it remains a frustrating disorder as Staphylococcus tends to be an opportunist which takes advantage of compromised skin. Sorting out the diseases which predispose to pyoderma is a critical feature of infection management, yet many pyodermas become recurrent. We often consider underlying diseases but we may not consider features such as owner and patient compliance, suboptimal dosing, incorrect choice of antibiotic, and an overall reduction in the use of topical therapy. The age of methicillin resistance is now with us in veterinary medicine and we are struggling with how best to deal with this phenomenon which has plagued physicians for some time. The increase in prevalence of multi-drug resistant bacteria is sadly coupled with decreased antibiotic drug discovery and development, so veterinarians and physicians are coming back to old antibiotics for treatment of resistant bacteria as well as nonantibiotic topical therapy, which is particularly suited for dogs with pyoderma. It is important to realize that the best protection against spread of MRS in both humans and dogs is hygiene. Handwashing, the use of alcohol-based hand cleaners, and environmental disinfection do more to reduce the spread of infection than antibiotics can. The next best protection is to use antibiotics wisely. Modern recommendations include using the highest safe dose the pet can tolerate for only the amount of time it takes to clear the infection. The idea is to kill off susceptible bacteria so rapidly that the host's defenses can take over; in this way we prevent selection of resistant strains of bacteria.

Why do dogs get pyoderma? Of all the species with which we work, the dog seems uniquely predisposed to bacterial skin infections. Basic structural features of their skin make dogs more susceptible to skin infections. Unlike human haired skin, dog hair follicles lack the protective lipid plug that blocks the hair follicle opening; in addition dogs have a thin and relatively disordered stratum corneum with less intercellular lipid. In general, the pH of dog skin is believed to be more alkaline that that of human skin and other domestic animals, with acid pH's considered to be more antimicrobial. Dogs with underlying skin disorders, such as atopic dermatitis and other allergic skin conditions, disorders of keratinization, endocrinopathies, and parasitic diseases, are more likely to develop staphylococcal skin infections. We know that the skin barrier, represented by the stratum corneum, is one of the first physical and chemical defenses against microbial infection. This barrier is known to be defective in human patients with atopic dermatitis and disorders of keratinization; we now know that this barrier is defective in dogs with atopic dermatitis as well. In addition, preliminary evidence suggests that dogs with atopic dermatitis may have decreased levels of defensins, cationic antimicrobial proteins that defend against bacterial infections as part of the innate immune system. In spite of this preliminary evidence, we are a long way from truly understanding why staphyloccal infections are so common in dogs.

What bacteria cause pyoderma in dogs? The major canine skin pathogen is S. pseudintermedius; however, S. schleiferi, S. aureus, and Pseudomonas aeruginosa also can be identified from canine patients with pyoderma. S. pseudintermedius binds preferentially to canine skin cells compared to human or feline skin cells, and this binding is enhanced when the corneocytes are derived from atopic dogs. While not considered as virulent as the human pathogen S. aureus, S. pseudintermedius shares many of its virulent characteristics, including enzyme and toxin production, ability to adhere to matrix adhesive proteins, and ability to form biofilms. Each of these features contributes to the ability of the bacterium to colonize and invade the skin. We have learned that many dogs carry this bacteria in their nose, around their mouth, and around the perianal ring; pups are colonized around birth with the strain carried by their mother. S. schleiferi was first identified from human clinical specimens in 1988. It has now been identified as the cause of pyoderma and otitis externa in dogs as well. S. aureus, the human pathogen, has been identified in a low percentage of dogs. Last, while not common, Pseudomonas aeruginosa can also be identified from the skin of dogs, particularly in lip fold pyodermas and post-grooming folliculitis.

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How do we diagnose pyoderma? The clinical appearance of the lesions supports the diagnosis but we nearly always perform cytologies, because staphylococcal infections often coexist with Malassezia infections, and both need to be treated in order to resolve the skin problem. Clear acetate tape can be pressed on the lesions The tape is easily stained with the rapid modified Wright's Giemsa stain Diffquick/Diffquik. The critical feature when using this staining system is to skip the first part, the light blue methanol fix, which will melt the adhesive off the slide or make the tape cloudy and more difficult to evaluate. Once stained, the tape will serve as its own coverslip. It is placed on the slide, immersion oil applied, then the slide examined for microbes, keratinocytes, and white blood cells. Impression smears are useful for moist lesions, and for dry crusts, we mince them in saline on a glass slide. The slide is dried then stained. Culture and sensitivity is recommended for all generalized deep pyodermas and when treatment with two different classes of antibiotic fail to resolve the problem. Methicillin resistance in canine infections is increasing and the sensitivity results are required to pick the correct antibiotic, as we don't have validated methods for empirically picking antibiotics for methicillin resistant staphylococcal infections in dogs. Moreover, identifying the particular Staphylococcus species involved is important so that we can determine whether the dog is infected with methicillin resistant Staph aureus and may be a source of contagion to humans. While there is some risk when dogs have MRSA, the important concept to remember is that these dogs most likely got the infection from a human. MRSP is much less likely to cause human infections, unless a person is very young, very old, or immunocompromised. Even then, the risk is relatively low (see http://www.wormsandgermsblog.com/promo/services/)

How do we treat pyoderma in dogs? Most dermatologists believe that the most appropriate first choice antibiotic for canine pyoderma is a cephalosporin. My preference is for cefovecin (Convenia®, Zoetis) and cefpodoxime (Simplicef®, Zoetis). Most dermatologists will recommend against choosing a fluoroquinolone as a first choice for pyoderma management. There are several reasons for this. First, enrofloxacin in particular has been used extensively in many dogs, often at doses that are suboptimal for Staphylococcus spp. Second, the fluoroquinolones do not seem to be as effective in vivo against S. pseudintermedius as predicted by in vitro sensitivities. This is particularly true for older fluoroquinolones such as ciprofloxacin and enrofloxacin. Third, there is concern that fluoroquinolones may actually increase the risk of selecting for resistance. In humans, the relative risk for developing infections with MRSA were highest for those patients treated with fluoroquinolones compared to other antibiotics. Furthermore, the use of older fluoroquinolones, in particular ciprofloxacin, was highly associated with selection for resistance.

Table of Antibiotic Doses for Canine Pyoderma

Cefovecin (Convenia)®	**8 mg/kg subQ; repeat in 2 weeks if necessary**
Cefpodoxime (Simplicef)®	**5-10 mg/kg QD (higher doses best)**
Cephalexin	**22-30 mg/kg TID**
Lincomycin (Lincocin)®	**20 mg/kg BID**
Clindamycin	**11 mg/kg QD to BID**
Amoxicillin-clavulanate (Clavamox)®	**20 mg/kg BID to TID**
Ormetoprim-sulfadimethoxine (Primor)®	**27.5-30 mg/kg QD**
TMP-sulfa	**20-30 mg/kg BID**
Doxycycline (if sensitive)	**10 mg/kg BID**
Minocycline (if sensitive)	**5-10 mg/kg BID**
Marbofloxacin (Zeniquin)®	**5.5 mg/kg QD**
Enrofloxacin (Baytril)®	**20 mg/kg QD**
Ciprofloxacin (not recommended)	**30 mg/kg QD*** **

Chloramphenicol	**50 mg/kg TID**
Amikacin	**15 mg/kg subQ QD**
Rifampin	**5-10 mg/kg QD******

*** Ciprofloxacin, while inexpensive, is a second generation fluoroquinolone with less activity against gram + bacteria than we would like. It has been shown in 2 different studies to be very inconsistent in absorption. If used, use at the high dose. It may be helpful to crush the tablets to help promote absorption but we really don't know as much about this antibiotic in dogs as we would like (This from data provided by Dr. Mark Papich, NCSU, soon to be published).
****Keep dose at a max of 10 mg/kg/day to reduce risk of hepatic damage, including necrosis and death.

What is methicillin resistance and how do we recognize it? Methicillin resistance in Staph is associated with acquisition of a gene mecA that incorporates into the bacterial genome and is subsequently passed on to all daughter cells. Mec A, codes for a mutated form of penicillin binding protein on the surface of the bacteria. The mutant protein can't bind any beta-lactam antibiotic and thus all penicillins and cephalosporins will be ineffective. The genetic element on which the mec A gene resides can also carry other antibiotic resistance genes and thus some Staph pseudintermedius will be resistant to all antibiotics tested. This genetic element will be retained within the Staph as long as antibiotic pressure is present, but in some cases it can slow bacterial growth. If antibiotic pressure is removed, then the bacteria have the capability of excising the incorporated genetic element and becoming sensitive again. For this reason, it may make the most sense to avoid systemic antibiotic therapy for dogs with superficial pyoderma caused by MRS and focus on aggressive topical therapy. To diagnose methicillin resistance, we must do culture and sensitivity. It is no longer acceptable for your lab to report out coagulase positive Staph spp. You should demand that the species of the Staph be determined, particularly when the infection is reported as methicillin resistant. Also, it is very important to be precise in our terminology. A methicillin resistant Staph. pseudintermedius is not "MRSA" but "MRSP." The term "MRSA" refers specifically to methicillin resistant Staph aureus, the human pathogen.

How do we treat methicillin resistant pyoderma in dogs? Systemic antibiotic therapy for dogs with MRS cannot be picked empirically. Culture and sensitivity is required to know what antibiotic will likely be effective. Given that systemic antibiotic therapy drives the retention of the resistance factors, maybe we need to shift the way we think about superficial pyoderma and use topical antiseptic therapy instead. We hypothesize that topical therapy may give the bacteria time and opportunity to eject the resistance genes and become susceptible again. To test the hypothesis that dogs with methicillin resistant superficial pyodermas could be cleared with topical therapy alone, we recommended daily topical therapy for 10 dogs with methicillin resistant Staph pseudintermedius (MRSP). All dogs were cleared of clinically observable infection within 30 days, and most were substantially improved within 2 weeks.

Clearly not all dogs with MRS will respond to topical therapy, particularly if the infection is a deep pyoderma. For those dogs, systemic antibiotic therapy will be required and culture and sensitivity will be mandatory. If the organism is sensitive, potentiated sulfas offer a great option for dogs with MRS. While side effects are possible, most dogs tolerate these drugs quite well. If reported as sensitive, clindamycin can also be used. A resistance factor termed the clindamycin-inducible resistance factor can be found in Staph spp; one indicator that this gene might be present is reported resistance to erythromycin but sensitivity to clindamycin. Treatment with clindamycin will rapidly induce the resistance factor, and antibiotic therapy will fail. It is only recommended, therefore, to use clindamycin if the bacteria are reported sensitive to all macrolides. Although we have advocated against the use of tetracyclines for most Staph pseudintermedius, MRSP may revert to sensitive. In those cases, doxycycline or minocycline may be helpful. My experience has been that long courses are required, as

these antibiotics are bacteriostatic. The majority of MRSP are sensitive to rifampin and amikacin. Amikacin is well tolerated by most dogs but must be given by injection and does have the risk of renal toxicity. Frequent monitoring of the urine for casts and repeated bloodwork (BUN, creatinine) can make this an expensive option. Rifampin can be used effectively but it has the potential to cause hepatotoxicity. Monitoring liver enzymes every 1-2 weeks is recommended.

Why is topical therapy so important in the treatment of pyoderma? Topical therapy should be part of the regimen for all dogs with pyoderma. Bathing removes scale, grease, and crust, reduces odor, and makes the dog feel and look better. Furthermore, topical therapy will accelerate the rate to cure, and hopefully decrease the length of time oral antibiotic therapy will be required. When used regularly, topical therapy may help reduce the frequency of relapse for dogs with recurrent pyodermas. Several shampoos are available for use, but current evidence suggest that shampoos containing 2-4% chlorhexidine are most effective. If bathing is done as monotherapy for resistant bacterial infections, then it is best done daily with a chlorhexidine –based shampoo, in my opinion. For those clients who cannot bathe every day, sprays and mousses are available. Economical but very effective options can be used topically daily for dogs that cannot be bathed. One of the most effective topical agents for methicillin resistant staphylococci is sodium hypochlorite, the active ingredient in bleach. Even very diluted concentrations of bleach can be very effective. Pediatric human patients are treated with compressed soaked in household bleach 5-6% diluted to ¼ cup per 10 gallons of water. This equates to 60 mls per 10 gallons or 6 mls per gallon. Solutions can be mixed up and put in a sprayer bottle to apply directed to the affected sites. For facial lesions, ocular lubricant can be applied to the eyes, and compresses soaked in the solution applied.

Bacterial pyoderma in dogs is considered a secondary disease and when it recurs, we try to determine whether underlying causes exist. The major considerations for recurrent pyoderma include parasitic diseases such as demodicosis or scabies, allergic skin disease, endocrinopathies such as hypothyroidism, hyperadrenocorticism or diabetes mellitus, and disorders of keratinization.

REFERENCES AND RESOURCES:

An excellent resource for information about methicillin resistant staphylococcal infection is Worms and Germs Blog. http://www.wormsandgermsblog.com/ On this site you can find information sheets for clients that help explain the difference between MRSA and MRSP.

Hillier A[1], Lloyd DH, Weese JS, Blondeau JM, Boothe D, Breitschwerdt E, Guardabassi L, Papich MG, Rankin S, Turnidge JD, Sykes JE.

Guidelines for the diagnosis and antimicrobial therapy of canine superficial bacterial folliculitis (Antimicrobial Guidelines Working Group of the International Society for Companion Animal Infectious Diseases). Vet Dermatol. 2014 Jun;25(3):163-75, e42-3. doi: 10.1111/vde.12118. Epub 2014 Apr 11.

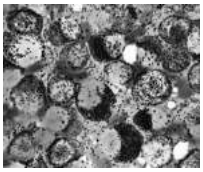
Canine mast cell tumors

Chantal Tu
March 20, 2016
SAGE Sympoisum

SAGE
CENTERS FOR VETERINARY SPECIALTY AND EMERGENCY CARE

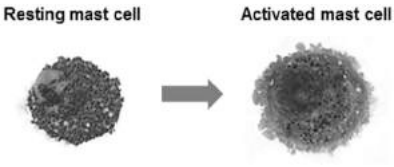
Outline

- Mast cells
- Clinical presentation
- Cytology
- Staging (extent of dz) and grading (associated w/ px)
- Prognostic factors (a challenge!)
- Treatment and prognosis



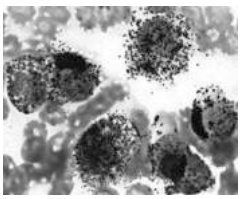
Mast cells
What are they?

- Normal cells involved in the immune system
- "Mastzellen" = well-fed in German



Normal mast cells
Role in inflammation

-
  - responses
-
  - Histamine and serotonin
    - vasodilation
    - increased HCl acid production
  - Heparin and chemotactic factors → inflammation



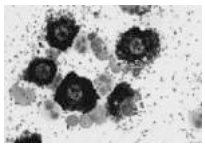
Cancerous Mast Cells
Clinical presentation
Can look like anything!

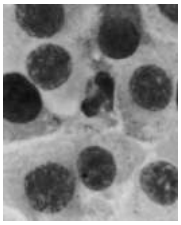
- Most common cutaneous tumor
- More common in caudal half of body
- Older dogs (3 wks-19yrs)
- Any breed but Bostons, Boxers, Schnauzers, Beagles, and Labs over-represented

Cytology
Stains

- How can you tell it's a MCT?
- FNA
- Granules
- Eosinophils

Diff-Quik



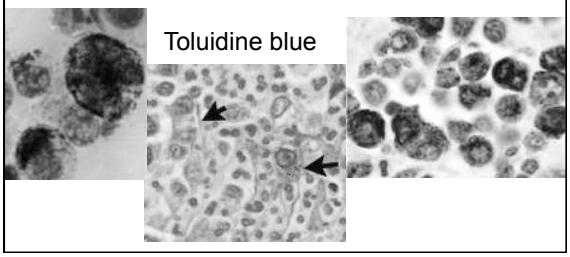


Cytology
Special stains

Wright's stain

Toluidine blue

CD 117 / C-kit

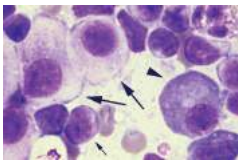


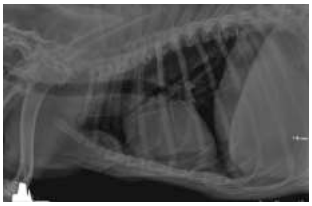
Staging (extent of dz in body)
Modified WHO

Stage 0:
involvement
Stage I: 1 tumor -LN involvement
Stage II: 1 tumor +LN involvement
Stage III: Multiple large, deep skin tumors, +/- LN involvement
Stage IV: Any tumor(s) +LN metastasis
Substage a – no signs
Substage b – signs

Staging
Diagnostics

- Abdominal ultrasound
- LN aspirates?
- Chest x-rays?
- Hepatosplenic aspirates?
- Bone marrow aspirates?
- Buffy coat?





Grading
Patnaik Grading Scheme (1984)

Grade	Patnaik grade	Microscopic description
Anaplastic undifferentiated (high grade)	III	Highly cellular, undifferentiated cytoplasmic boundaries; nuclei irregular in size and shape, frequent mitoses; sparse granules
Intermediate grade	II	Cells closely packed with indistinct cytoplasmic boundaries; nucleus-to-cytoplasm ratio lower than in anaplastic type; infrequent mitoses; more granules than in anaplastic type
Well differentiated (low grade)	I	Clearly defined cytoplasmic boundaries with regular, spherical, or ovoid nuclei; rare or absent mitoses; abundant, large, deep-staining cytoplasmic granule

Grading
Patnaik Grading Scheme (1984)

- Has been the "gold standard" for decades
- Provided a foundation for grading
- BUT…
- Subjective criteria
- Up to 85% of MCTs grade 2 MCTs
  -
  -
  -

Grading
Patnaik Grading Scheme (1984)

- Difficult to reproduce amongst pathologists
  -
  - pathologists
  - Only 62% of MCTs had agreement among at least 80% of pathologists
  - 3 MCTs had all three different grades assigned!

Grading
Two Tier Grading Scheme (Kiupel) (2011)

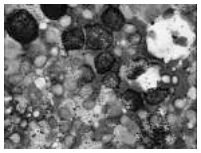
Grade	Microscopic description
High grade	Any one of these criteria: 1) MI > 7/10hpf 2) Multinucleated cells > 3/10hpf 3) Bizarre nuclei > 3/10hpf 4) Karyomegaly (nuclear diameters of >10% of neoplastic cells vary by at least 2-fold) (Using fields w/ highest mitotic activity or highest degree of anisokaryosis)
Low grade	Clearly defined cytoplasmic boundaries with regular, spherical, or ovoid nuclei; rare or absent mitoses; abundant, large, deep-staining cytoplasmic granules

Grading
Two Tier Grading Scheme (Kiupel) (2011)

- Also subjective criteria ("bizarre" nuclei, karyomegaly)
- Little therapeutic literature
- Consensus from VCS Oncology-Pathology Working Group (MCT)
  - Grade is only 1 px factor
  - Evaluate entire clinical picture

Grading
Two Tier Grading Scheme (Kiupel)

- 95 MCTs
- Blindly graded by 28 pathologists from 16 institutions
- 100% agreement for high-grade MCTs
- Better predictor of survival

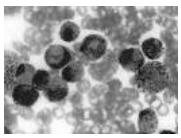


Prognostic Factors

- Histologic grade
- Stage
- Mitotic index
- Breed
- Location
- Growth rate
- Proliferation indices: AgNOR, Ki67, PCNA
- C-kit (mutation and pattern)

Prognostic Factors
Grade

- Most consistent and reliable prognostic factor
- Still will not predict the behavior for every tumor
- Patnaik vs. two-tier



Prognostic Factors
Grade

Grade	Patnaik grade	Microscopic description
High grade (Anaplastic undifferentiated)	III	- **dogs die w/in 1 year** - **1500 day (>4y) survival rate = 6%**
Intermediate grade	II	- **75% long term survival w/ complete excision** - **1500 day survival rate = 44%**
Low grade (Well differentiated)	I	- **80-90% long term survival w/ complete excisions** - **1500 day survival rate = 83%**

Prognostic Factors
Two Tier Grading Scheme (Kiupel)
More consistent and less subjective

Grade	Microscopic description
High grade (10)	**Median survival time was 3.6 months**
Low grade (85)	**Median survival time not reached (>2 years)**

Prognostic Factors
Stage

- Stage
  - 0 and 1 are better
  - 2
    - tx +/- chemo had median DFI = 41 months
  - 3
    - necessarily have a worse prognosis
  - Stage each MCT separately

Prognostic Factors
Mitotic index

- # of mitoses/10hpf
- MI $\leq$ 5
  - Excellent long-term prognosis (MST = 70 months)
- MI > 5
  - Associated with poor survival (MST ~2-3 months)
  - For MCTs of all different grades and all different treatments

Prognostic Factors
Breed

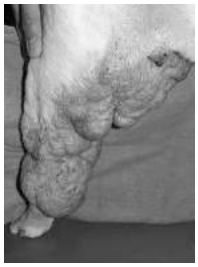
- Boxers and Pugs
  - More commonly low-intermediate grade MCTs
  - 25 purebred Pugs
    - 94% low-intermediate grade MCTs
    - 12% died of mets
- Sharpeis
  - Higher grade MCTs

Prognostic Factors
Location

- Oral mucosa, mucocutaneous junction, or perioral region of the muzzle
  - Negative prognostic factor
- 44 MCTs of oral mucosa, mucocutaneous jxn, perioral region of muzzle
  - MST= 52 months, MST w/ LN mets = 14 months

Prognostic Factors
Growth rate

- Stays localized and stable for a prolonged period (>6 months) = better
- Recent rapid growth = worse

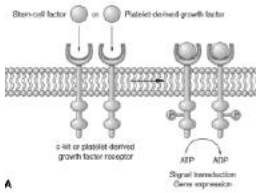


Prognostic Factors
Proliferation Indices

- Ki-67
  - Represents tumor growth fraction
  - Protein necessary for cell cycle progression
- AgNOR (argyrophilic staining nucleolar organizing regions)
  - Represents rate of cell proliferation
  - Involved in RNA transcription
- PCNA (proliferation cell nuclear antigen)
  - Involved in DNA replication and highly expressed in S phase
  - Not as predictive as Ki67 or AgNOR

Prognostic Factors
c-kit mutation

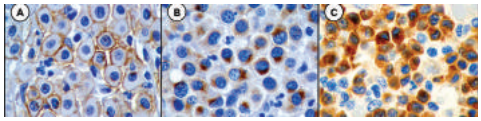
- C-kit
  - Receptor tyrosine kinase
  - Important for normal growth, proliferation, and survival
  - Mutations in c-kit
    - Leads to constitutively active receptor to promote malignancy
  - More aggressive



Prognostic Factors
c-kit pattern

- Normal expression on cell membrane
  - Type 1 (A)
- Increased cytoplasmic and/or perinuclear staining
  - Types 2 and 3 (C&B)
  - Associated w/ decreased survival and increased rates of local recurrence

KIT in Canine Cutaneous Mast Cell Tumors Webster et al. 107

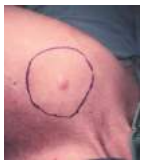


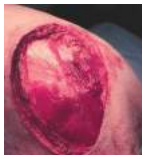
Prognostic Factors
Michigan MCT Prognostic panel

- Asseses all of these factors in combination
  - Ki-67
  - AgNOR
  - PCNA
  - C-kit mutation analysis
  - C-kit pattern expression

Surgery

- If localized and possible, cut it out!
- 2-3cm lateral margins and one fascial plane deep
- Estimate via palpation
- Ultrasound and CT?
  - May change tumor margin estimates b/w 19-65% of cases



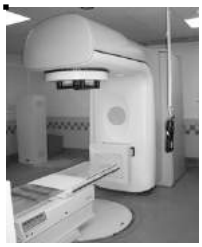


Surgery

- Prednisone?
  - Decrease inflammation
  - Aim for margins of original size of mass
- Margins
  - Ink
  - 10mm lateral and 4mm deep margins revealed no recurrence/mets in one study of 100 dogs
- Prognosis
  - Low-grade: MST > 2 years
  - High-grade: MCT w/ sx have MST ~ 4 months

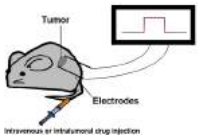
Radiation therapy
SAGE Campbell

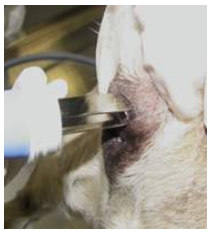
- Brief general anesthesia
- Limited facilities
- Prognosis
  - Median duration of remission = 5-6 years



Electrochemotherapy
SAGE Dublin

- Chemotherapy IV or IT
- Apply electrical pulses for electroporation
- Transient permeant defects
- Molecules can cross the membrane (loading effect)

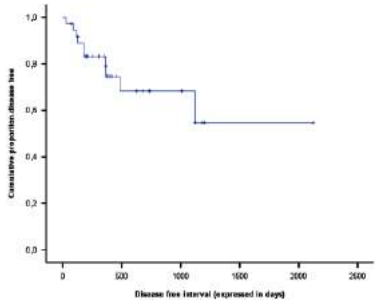




Electrochemotherapy
ECT for K9 MCT

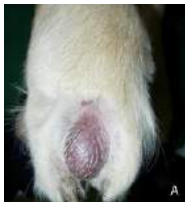
- 37 dogs w/ incompletely resected MCTs of various grades
- 6 year study
  - 78% NED
  - 16% recurrence
    - Median time to recurrence 1200d (>3 years)
  - 3% died from MCT
  - 3% died of unrelated causes
  - 32% had signs of MCT degranulation; self-limiting

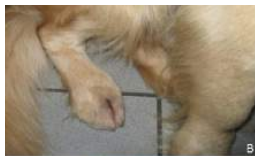
Electrochemotherapy
ECT for K9 MCTs

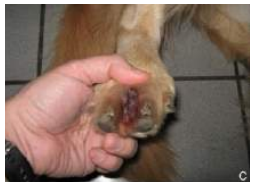


- Median DFI is 1200 days (>3 years)

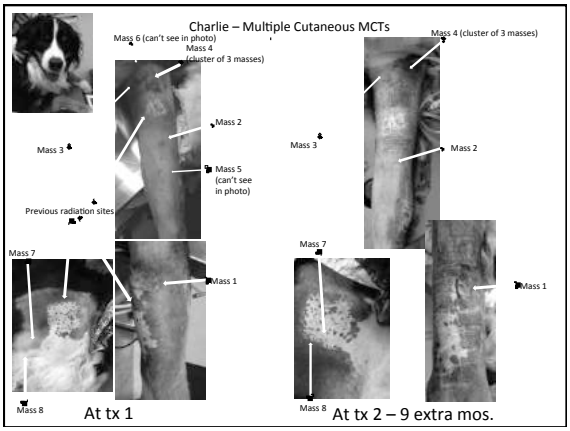
Electrochemotherapy
ECT for K9 MCT





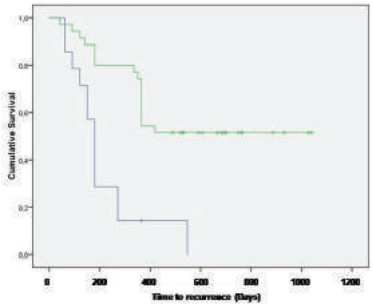


- Right: Mild erythema and edema after 1st treatment



Electrochemotherapy

ECT for other tumors – Feline sarcomas



- Sx alone: median TTR 180d (6 mos)
- Sx + ECT: median TTR 1.8yrs

Electrochemotherapy

ECT for other tumors – feline SCC (nasal, pinnae, canthus)

Presentation

After Tx

- 7/9 CR and 2/9 PR
  - 5 alive at 5y, 2 died of unrelated causes, 2 died of dz

Electrochemotherapy

ECT for other tumors in other species!

- Cutaneous SCC on a yellow-bellied slider tx w/ sx + ECT

Chemotherapy

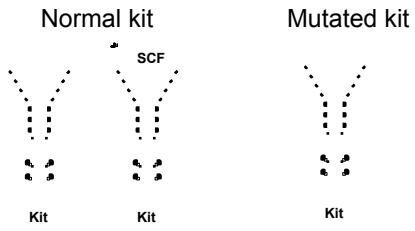
- Indications
  - High-grade
  - Metastasis
  - Location (visceral, mucocutaneous, etc.)
- Prednisone

Chemotherapy

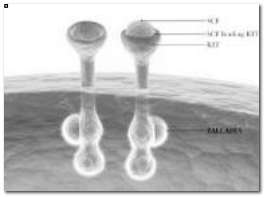
Reported multi-agent response rates in the setting of gross disease

Agent	n	Response Rate	MST
Vinblastine/ Prednisone	15	47%	154 days
Lomustine / Vinblastine	37	57%	Macroscopic dz: 35 weeks Microscopic dz: 48 weeks
Vincristine/ Cytoxan/ Hydroxyurea/Pred	17	59%	97 days in responders
Vinblastine /Cytoxan/ Prednisone	11	64%	Not reported
Prednisolone/ Chlorambucil	21	38%	140 days
Lomustine / Vinblastine /Pred	17	65%	PFS in adjuvant setting 489d

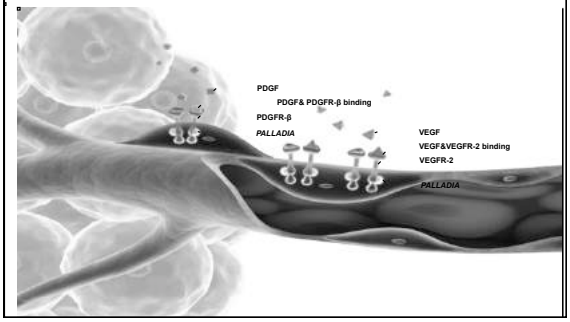
Tyrosine kinase inhibitors (TKIs)
Mechanism of action



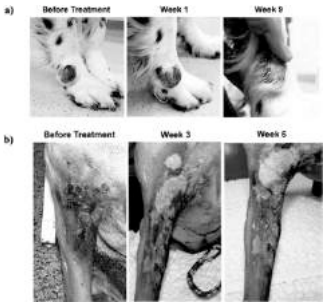
Tyrosine kinase inhibitors (TKIs)
Mechanism of action



TKIs
Palladia



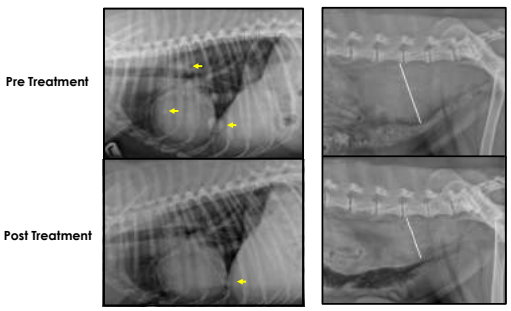
TKIs
Palladia for MCT



TKIs
Palladia for MCT – Tippy pre-treatment

TKIs
Palladia for MCT – Tippy 3 months post-treatment

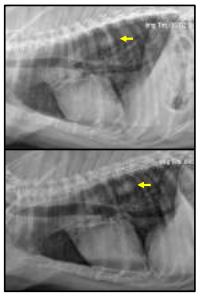
TKIs
Palladia for other tumors - AGASACA



TKIs
Palladia for other tumors – metastatic OSA

Pre Treatment

Post Treatment (4 mths)

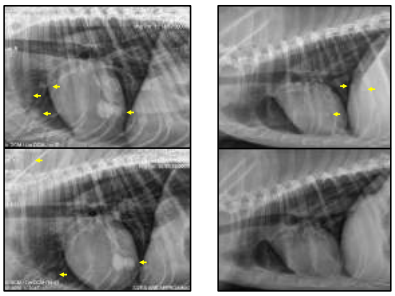


Remained on Palladia for 14 months, eventually developing additional pulmonary metastasis that was very slowly progressive. Euthanized due to metastatic lesion to the humerus with pathologic fracture.

TKIs
Palladia for other tumors – metastatic thyroid carcinoma

Pre Treatment

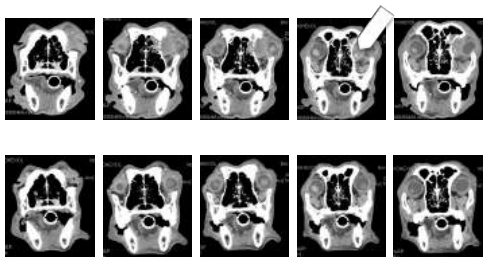
Post Treatment



TKIs
Palladia for other tumors – retrobulbar SCC

Dec 2010

June 2011



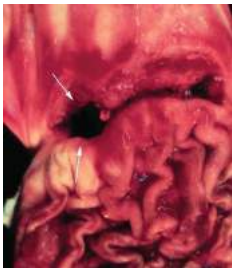
Has been switched to a 1 week on/1 week off regimen due to chronic low-grade lethargy; doing very well at 7 months of therapy.

TKIs
Kinavet/Masitinib

- Currently not available
  - Was conditionally FDA-approved
  - For recurrent or non-resectable Grade 2/3 MCTs not previously treated with chemotherapy or corticosteroids

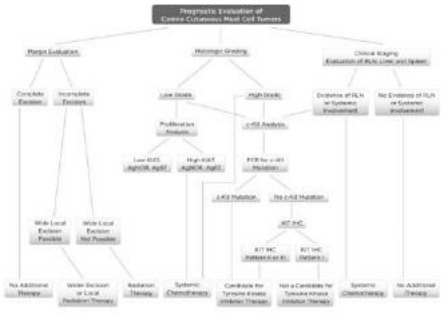
Supportive care

- Benadryl
- Pepcid
- Prilosec



Conclusions

- Many treatment options!



Questions?

SAGE Symposium March 20, 2016
Updates in Veterinary Oncology
Bryan Marker, DVM, DACVIM (Oncology)

New and upcoming chemotherapy agents

Cytotoxic chemotherapy

- Paccal Vet®-CA1 by Oasmia
  - Conditionally approved February 27, 2014
    - Conditional approval process
      - Phase I study
      - New formulation of paclitaxel (Taxol)
  - Taxol
    - Isolated from *Taxus brevifolia*, the Pacific Yew
    - Mechanism of action
      - Stabilizes microtubules and prevents tubulin disassembly
      - Mitosis cannot be completed → apoptosis
    - Ovarian, breast, non-small cell lung cancers in addition to many others
  - Taxol in dogs
    - 5/25 dogs with partial responses to a variety of tumors
      - 2 mammary carcinomas
      - 2 osteosarcomas
      - 1 histiocytic sarcoma
    - Cremophore
      - 64% of dogs experience hypersensitivity reactions
  - Paccal Vet®-CA1
    - No cremophore
      - Micellar technology
    - Treatment administered intravenously every 21 days, over 15-30 minutes
    - Nonresectable stage III, IV or V mammary carcinoma in dogs that have not received previous chemotherapy or radiotherapy
      - 1/8 complete response
      - 2/8 partial response of 50% reduction in tumor longest dimensions
    - Resectable and nonresectable squamous cell carcinoma in dogs that have not received previous chemotherapy or radiotherapy
      - 2/3 had partial response
    - Toxicity
      - Neutropenia 4-7 days post administration
      - Gastrointestinal
- Rabacfosadine (Tanovea™)
  - Background
  - Mechanism of action

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        - Prodrug of nucleotide (guanine) analog
            - 9-(2-phosphonylmethoxyethyl)guanine (PMEG)
                - » Problems with administration of PMEG
            - Interferes with DNA polymerase function, decreased DNA repair and synthesis
        - Preferential action within lymphocytes
        - Limit plasma exposure and thus toxicity
    - Potential applications
        - Non-Hodgkin's lymphoma
            - 77% overall response rate among treatment naïve and resistant lymphomas, stage III-V
            - Cutaneous T-cell lymphoma
                - » Modest clinical activity and remission duration
        - Multiple myeloma
            - Studied in limited fashion
                - » Responses among 9/11 dogs
    - Dose-limiting toxicities
        - Dermatopathy, neutropenia, gastrointestinal
    - Other toxicities
        - Pulmonary
    - Current status/availability

Updates in immunotherapy

- Immunotherapy brief background/history
- Oncept® melanoma vaccine
- Brief background
    - Xenogeneic human tyrosinase
        - Tyrosinase
            - Cellular glycoprotein involved in melanin synthesis
            - Human and canine genes are very similar, only slight difference
        - Administration leads to anti-tyrosinase antibodies
            - Overcoming self-tolerance
            - Immune system attacks tyrosinase expressing cells (cancer)
    - Initial conditional approval from USDA in 2007
- Updates in melanoma vaccine
    - Full licensure in 2010
        - Stage II and III canine oral melanoma
        - Grosenbaugh 2011
            - Dogs treated with local tumor control and followed with vaccination
            - Median survival time not reached during study
            - Compare to historical control group

            - Median survival time 324 days
        - Sarbu 2015
            - Use of the melanoma vaccines in cats
                - Complication rate of 11.4%
- Questioning the vaccine's efficacy
    - Boston et al. 2014
        - Investigated the impact of systemic adjuvant therapies for dogs with oral melanoma
            - No significant improvement in outcome
                - Only 14 dogs received Oncept
    - Ottnod et al. 2013
        - Primarily stage II/III
        - No difference in outcome between vaccinates and non-vaccinates
        - Study flaws
    - Implications for practice? How are we handling this?
- Oncept IL-2
    - For adjuvant treatment of feline injection site sarcomas
    - Background
        - Function of IL-2
    - Jas et al. 2014
    - Current availability
- Canine lymphoma vaccine
    - Proposed mechanism of action
        - CD20
    - Conditional licensure from USDA
    - What we know and don't know
- Monoclonal antibodies for lymphoma
    - Background
        - Mechanisms of action
        - Development
        - Rituximab
            - Ground-breaking development for non-Hodgkin's lymphoma
    - Veterinary medicine
        - T-cell (CD-52)
        - B-cell (CD-20)
        - Availability

Changes in the use of targeted therapies

- Brief review
    - Tyrosine kinases
        - Transmembrane proteins
- Small molecule inhibitors
    - Imatinib (Gleevec)

- Targets
  - Abl, Kit, PDGF-R
- Limited use in veterinary medicine

- Masitinib (Kinavet)
  - Targets
    - Kit, PDGFR, Lin
  - Conditional licensing
    - "For the treatment of nonresectable Grade II or III cutaneous mast cell tumors in dogs that have not previously received radiotherapy and/or chemotherapy except corticosteroids"
    - *Potential* for expanded use
    - Loss of conditional license in 2015
- Toceranib phosphate (Palladia)
  - Targets
    - Kit, VEGF-R, PDGF-R, Flt 3
      - RET and JAK?
  - Labeling
    - "Patnaik grade II or III, recurrent, cutaneous mast cell tumors with or without regional lymph node involvement in dogs. "
  - Update in dosing recommendations
  - How do we monitor patients when using this drug?
    - Effects of VEGF inhibition on renal tubular epithelium
  - How is this drug being used?
    - Preliminary evidence of activity in:
      - Osteosarcoma, anal sac adenocarcinoma, thyroid carcinoma, head and neck carcinoma and nasal carcinomas, GIST, multiple myeloma, heart-based tumors?
      - London et al 2012
        - Objective responses observed in osteosarcoma, anal sac adenocarcinoma, thyroid carcinoma, head and neck carcinoma, and nasal carcinoma
        - Clinical benefit
    - Use in cats
      - Feline injection site sarcomas
        - Safe, but ineffective
      - Feline oral squamous cell carcinoma
    - Combination therapy
      - Protocols
      - Vinblastine/toceranib
      - Lomustine/toceranib

        - Doxorubicin/toceranib
        - Piroxicam/toceranib with or without cyclophosphamide
- Questions yet to be answered
    - Adjuvant therapy
        - Driver vs. no driver
        - Angiogenic conditioning
        - Duration of treatment
            - 6 months?, 1 year?, indefinitely?

A brief review on currently available cancer screening and monitoring assays

Developments in the usage of metronomic chemotherapy

- Background
- Mechanisms of action
    - Anti-angiogenesis
    - Immunomodulation
- Review of recent literature
    - Sole therapy
    - In combination
    - In cats
- Questions yet to be answered
    - Dosing
- Practical aspects
    - Questions surrounding the use of compounded chemotherapeutic agents

Electrochemotherapy

- Background
- Brief review of recent literature
- Current experience, case examples

Future directions/comparative aspects

Principles of Cancer Treatment

Lori Cesario DVM, DACVIM (onc)

Before you can successfully treat a patient with cancer, there are four different categories the must be explored and understood. One must first "know their patient", they must have a detailed understanding of the disease their patient has, they must think about chemotherapy consideration particular to this patient, and they must have read the pathology report, while asking themselves, "does the diagnosis fit the clinical picture", "is there additional information I need from the pathologist", "does this make sense to me, or do I need to discuss the findings further with the pathologist". Once these categories are thought through and satisfied, we can then establish a treatment plan and give a more reliable prognosis for our clients.

First:
Know your patient – breed-related neoplasia, breed-specific concerns
Know your disease – metastatic pattern/incidence, surgery, RT, ECT
Chemotherapy considerations – organ dysfunction, chemo theory and safety
Know your pathologist – interpretation is everything, establish relationship

Then:
Establish treatment plan
Determine prognosis

Know your patient - Breed-related neoplasia

1. Disseminated Histiocytic Sarcoma
25% Bernese mountain dogs affected
    Oligogenic mode of inheritance noted in 800 studied BMD
    78% of affected dogs had relatives with HS
    40% of affected dogs had a relative with a different neoplasia
Rottweilers overrepresented

2. Localized Histiocytic Sarcoma
Soft tissue vs visceral forms
Retrievers
    Flat-coated retrievers, n=180, 57% limb, 26% visceral (splenic)
    Labradors, goldens, etc

3. Transitional Cell Carcinoma
Scottish terriers, 18x increased risk
Beagle, sheltie, wirehaired fox terrier, West highland white terrier
Overweight female exposed to topical flea/tick dip, 28x increased risk

4. Osteosarcoma

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Rottweilers, Saint Bernard, great dane, Irish setter, Doberman pinscher, GSD, golden
Increasing height most predictive for development of disease

5. Mast Cell Tumor
Mixed breeds develop most tumors
Breeds at increased risk
    Bulldog descent: boxer, Boston terrier, English bulldog, pug
    Labrador & golden retriever, cocker spaniel, schnauzer, beagle
    Rhodesian ridgeback, Staffordshire terrier, weimeraner, Shar-pei

Know your patient - Breed-specific concerns

PGP/MDR1 mutations
    ATP-dependent drug transporter
    Mutation leads to toxicity from: ivermectin, vincristine, doxorubicin
        loperamide, moxidectin, digoxin, mexilitine
    Patients receive toxic levels because of delayed drug excretion
    Dose reductions for homo/heterozygous mutants

MDR1 mutant breeds
    One study assessed 5,368 dogs for mutations, the following were noted
    **The first number is homozygous mutant, the second is heterozygous
    Collie, 35% 42%
    Longhaired whippet, 0% 58%
    Austrailian shepherd - standard, 10% 37% mini, 3% 34%
    Shetland sheepdog, 6% 47%
    Old English sheepdog, 0% 1%
    Border collie, 1% 4%
    Silken windhound, 0% 5%
    German shepherd dog, 2% 8%

All possible mutants should be evaluated prior to treatment with vincristine/doxo
    Washington State University performs the test – download form on website
    Cheek swab or blood samples
    The PCR tests are all batched on Mondays and results are available Friday
    Dose reduce vinca alkaloids and doxo pending results, or give different agent
    Normal dose can kill homozygous mutant patient

Know your disease

How difficult is the disease to control locally?
    Do I need 2 or 3 cm margins for a complete excision?
Will surgery benefit this patient at all?
How likely is the tumor to metastasize? And to which organs?
How rapid is this tumor growing? When do we need to treat?

Which treatments are indicated based on behavior?

Surgery – What is needed for local control?
Radiation – Is there a role for palliative or definitive RT for this patient?
Electrochemotherapy - What is this? When can it help?
Chemotherapy – Can chemotherapy help this patient live longer?

In human surgical oncology, complete removal of localized cancer leads to more cures than any other modality. In order to achieve this, we must have local disease (this means the patient must be completely staged to know that the disease is localized), the patient must have early staged disease, with limited potential to metastasize. Sixty percent of human patients cured with cancer are cured by surgery alone. This requires detailed knowledge of tumor behavior prior to surgery, with a carefully planned surgery that obtains adequate margins.

To achieve surgical cure in veterinary medicine, must know…*

Anatomy, physiology, resection, reconstruction options for all organs
Expected tumor behavior and tumor biology
Adjuvants to surgery

As well as…

1. What is the histologic type, stage, & grade of cancer to be treated?
2. What are the expected local and systemic effects of this tumor type, grade, and stage?
3. Is a cure possible and at what cost in terms of cosmetics & function?
4. Is surgery indicated at all?
5. What are the options for alternative or planned combination treatment

The fist surgery is the best chance of cure

How do we achieve a surgical cure?

Use imaging modalities to plan (CT)
Use treatment (chemo, radiation) to reduce tumor first, if needed
Be aggressive

'The aggressiveness of resection should only rarely be tempered by fears of wound closure'

Surgery not typically recommended if metastasis has occurred

Exceptions: removal of the metastatic lymph node (MCT, AGASACA)

These patients need chemotherapy after surgery

What is needed to rule out metastasis?

3-view thoracic radiographs (strongly consider radiologist review)
Abdominal ultrasound +/- aspirates
FNA local lymph node (even if it palpates normally)

No mets? Take wide margins. Measure with calipers and mark tissue with marker
MCT - 2 cm, Sarcoma - 3 cm lateral, FISS – 5 cm

Other:
Biopsies should be performed so tract can be removed with mass
Staples can identify location of excised intra-abdominal and soft tissue masses

Electrochemotherapy

Electrochemotherapy (ECT) is being studied in humans and animals as a means of increasing chemotherapy drug delivery to cutaneous masses in particular, but in some cases, intra-abdominal neoplasia. ECT usually consists of intravenous chemotherapy, intralesional chemotherapy (bleomycin or cisplatin), electropulsation and electroporation. ECT causes reversible changes in the character of the cell membrane, allowing increased uptake of the intralesionally injected chemotherapy agent and increased cell killing. In mast cell tumor patients, ECT has shown a 85% response rate on gross disease, with a time to recurrence of >4 years. ECT is also effective in a microscopic disease setting, similar to radiation therapy (published studies are not yet available). This treatment similarly involves brief periods of general anesthesia, but generally only 2 treatments if microscopic disease is present. If treating gross disease, the recommendation is to treat once past resolution of disease; this typically amounts to 3-4 treatments. SAGE Dublin and SAGE Concord are currently the only facilities in California offering this treatment.

Electrochemotherapy Indications:
- Incompletely excised MCT, STS
- Feline SCC, nasal planum
- Feline injection site sarcoma, incompletely excised
- Epitheliotropic lymphoma
- Hodgkin's-like lymphoma
- Some oral tumors
- Feline OSCC, incompletely excised
- Mammary tumors, incompletely excised

Typically 2 treatments indicated for microscopic disease
Typically 3-4 treatments indicated for gross disease
One treatment for feline nasal planum SCC
Cost $1200-$1400 per treatment

ECT – Mast Cell Tumors
- 85% response rate (gross disease)
  - Time to recurrence >4 years
- 78% no recurrence over 6 years (incomplete excision)
  - 16% recurred, overall time to recurrence 3.3 years

ECT – Feline Fibrosarcoma
- 64 cats with incompletely resected FSA

ECT x 2 = local control with mean of 666 days (median not reached)
1.6% metastatic rate
Versus 180 days in cats with surgery alone

Chemotherapy Considerations

Organ dysfunction

Knowing which organs are responsible for the metabolism & elimination of chemotherapeutics helps determine:
If the drug can be given safely
If a dose reduction is necessary

Hepatic disease

Vinca Alkaloids
Metabolized by cytochrome P450 mixed function oxidase CYP3A
>70% biliary excretion
Dose reduce by 50% if bilirubin >1.5 or 75% if >3mg/dl
Recommended but lack of correlation bt plasma bilirubin and VA clearance

Doxorubicin
Dose reduce if severe hepatic failure, 50% biliary excretion

Renal insufficiency

Carboplatin
90% renal excretion
Clearance directly correlated with creatinine clearance
Dose reduce for renal insufficiency

Also: methotrexate, cisplatin, streptozotocin, bleomycin

Cardiac disease

Doxorubicin
MOA: DNA intercalation, generation of reactive oxygen species (ROS)
Acute cardiotoxicity 11% humans
Chronic dose-dependent cardiotoxicity
Humans: 500 mg/m2 → 20% CHF, DCM
Canine: 180-240 mg/m2 is cut-off
Monitor
Also, epirubicin, cyclophosphamide

Options for "at risk" patient
Add cardioprotectant
Zinecard (dexrazoxane) – iron chelator, reduces superoxide radicals
Alternate chemotherapeutic – epirubicin, actinomycin, mitoxantrone
Administer over 60 minutes – reduces arrhythmias
Frequent echocardiograms

Chemotherapy Theory

Skipper log cell kill

Tumor growth is logarithmic
Each chemo administration kills same percentage of tumor
To cure, must get below 1 surviving cell
Must not allow repopulation between treatments
10^9 cell tumor, need 6 cycles, killing 99% each time = <1 cell
10^9 x .01 alive x .01 x .01 x .01 x .01 x .01 = <1 cell
BUT, watch population recovery! No delays early in LSA treatment

Gompertzian growth

Tumor follows Gompertzian curve, not linear
When larger than 1cc, growth rate slows 5x
Larger tumors hypoxic, not all cells dividing

Chemotherapy Safety

2004
NIOSH recommends CSTD (closed system transfer devices) + PPE + ventilated biologic safety cabinets for chemotherapy

CSTD
Drug transfer device mechanically prohibiting
Environmental contaminants into the system (maintains sterility)
Escape of hazardous drug or vapor out side of the system
Traditional methods of drug prep are associated with high levels of leakage; CSTDs provide near complete containment
Phaseal, Equashield

Know your pathologist

Immunohistochemistry
Bottom line
If diagnosis is different than you suspect
If disease is not responding as you anticipate
…a second opinion is warranted

Establish a relationship, and call
Ask questions for difficult cases
If diagnosis does not match clinical picture, call
Maybe they could use more information
Maybe there was debate about the diagnosis
Maybe they have a suggestion for a "next step"
Maybe there was a typo

Summary

Most cancers require a multi-modality approach for best outcome
This often necessitates conversations early in the course of care between surgery, med onc and rad onc
If anyone has questions about an oncology case, we are happy to help!

Track 4

"That's Not What I Meant!"

Outstanding Communication Skills
When You Need Them The Most

SAGE

An approachable team of specialists providing advanced, collaborative, and compassionate care.

Sonia Davis, RVT
Quality Service Manager

Marc Weinstein, MBA
Chief Operating Officer

Symposium '16

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Agenda

- Communication resources in the veterinary industry
- Communication and hospitality – What is your role?
- Communication skills
- Ways to inspect what you expect
- Closing activity
- Q & A

Our industry has many resources for communication.

- Zoetis Animal Health - ***frank*** communication
- ICCVM - Int'l Conference on Communication in Veterinary Medicine
- IHC - Institute for Healthcare Communication

"We've found that successful communication results in better patient care, as well as greater satisfaction for both the patient [client], and clinician." – Gary Friedlander, M.D., IHC, Yale University School of Medicine

- ICH and Bayer AH Partnership (2002)
- BAH Communication Project (2005)
- Karen Cornell - SAGE Symposium Speaker (2013)
- Hospitality Quotient (hospitalityq.com) – SAGE Symposium Speaker (2015)

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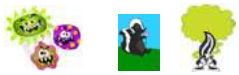
It starts from within…

Communication and hospitality
What is your role?

Staph **Staff**



"The ladies at the desk were friendly, however they did seem to treat the vet technician rudely by not acknowledging her when she brought me to the desk to pay."
2015 survey feedback

…creating an emotional connection (loyalty) begins with taking care of each other first!

"Experience is Everything" Welcome to the experience economy! (AAHA Trends Magazine)

Communication and hospitality
What is your role?

AAHA Trends Nov '16

Veterinary Team Brief 02/2016

5

How do we want to come across? Advocate vs. Gatekeeper

Communication and hospitality
What is your role?

Advocate Mindset

- Remove hurdles
- Confident
- Empowered
- Reading client/co-worker cues/signs

Gatekeeper Mindset

- Hides behind the rules (policy, job description)
- Prefers monologue
- Does not feel empowered

Communication and hospitality
What is your role?

Advocate vs. Gatekeeper

Communication skills

There are nine outstanding communication skills to make every interaction a favorable one.

1. Introduction
2. Open-Ended Inquiry
3. The P-A-U-S-E
4. Reflective Listening
5. Empathy
6. Non-Verbal Behavior/Cues
7. Summarizing
8. Signposting
9. Escorting

Communication skills
Introduction
Open-ended inquiry
The P-A-U-S-E
Reflective listening
Empathy
Non verbal behavior & cues
Summarizing
Signposting
Escorting

1. Introduction – you have one chance to make a lasting first impression.

- Smile!
- Wear nametag
- Direct eye contact
- Introduce yourself by name
- Begin the appointment with a “Welcome”
- Strive to know breed, gender and names
- Reflect back why they are here and who they will be seeing
- Telephone Introduction – say it with a smile!
- Exercise self-awareness as to conversations, tone, volume, and body language

"Welcome!"

Marc and Sonia "Mini"

2. Open-ended inquiry allows you to maintain control of the conversation.

Two Components

1. Open–ended questions yield answers beyond a yes/no
2. Funnel down to targeted yes/no closed-ended questions

"That's not what I meant"

Marc and Sonia "Mini"

3. The speaker must P-A-U-S-E frequently…

Communication skills
Introduction
Open-ended inquiry
The P-A-U-S-E
Reflective listening
Empathy
Non verbal behavior & cues
Summarizing
Signposting
Escorting

People need time to process what has just been said so listeners remain engaged and ready to follow what comes next.

…and the listener must P-A-U-S-E to acknowledge the feeling without necessarily agreeing.

Communication skills
Introduction
Open-ended inquiry
The P-A-U-S-E
Reflective listening
Empathy
Non verbal behavior & cues
Summarizing
Signposting
Escorting

70/30 Formula – Talk less, listen more

1. **DO**
   - Be aware of your non-verbal behavior
   - Nod your head yes
   - Use words like "sure, I see"
2. **REFRAIN FROM**
   - The desire to mentally argue
   - Your assumptions/judgments/bias
   - Forming your next sentence
   - Letting your mind drift
     - **STOP** talking – do not interrupt
     - **DROP** the need to be right
     - **ROLL** (let negative words roll off)

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"Wait for it…"

Marc and Sonia "Mini"

4. Reflective listening is an excellent tool for rapid relationship building.

1. Simple repeat

"So Wrigley seemed lame on his right leg last night after dinner."

2. Summarize

"I'm so pleased you thought to come to us today. It sounds like you and Wrigley had a rough evening."

3. Hypothesis test

"Let me see if I am understanding correctly, given your recent experience with Cooper, I'm wondering if that's what you're worried about with Wrigley today."

"I hear you!"

Marc and Sonia "Mini"

5. Empathy allows others to feel heard, valued, and appreciated.

Empathy - *"the ability to imagine and put yourself in someone else's shoes."*

- Feelings first - there is compliance when others feel heard
- Empathy must be genuine
- "I understand" and "I am sorry, but" are NOT empathy statements

"A walk in your shoes"

Marc and Sonia "Mini"

https://www.youtube.com/watch?v=cDDWvj_q-o8

6. Non verbal behavior provides clues that need to guide your conversation.

Communication skills
Introduction
Open-ended inquiry
The P-A-U-S-E
Reflective listening
Empathy
Non verbal behavior & cues
Summarizing
Signposting
Escorting

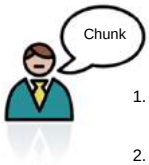
"They were so sweet and moved the rug so my dog could stand on it. He's afraid of tile floors. It made a difference!"
2015 survey feedback

"No mixed messages"

Marc and Sonia "Mini"

7. Summarizing ensures that you and the listener are aligned throughout the conversation.

Communication skills
Introduction
Open-ended inquiry
The P-A-U-S-E
Reflective listening
Empathy
Non verbal behavior & cues
Summarizing
Signposting
Escorting



'n

1. Pause for 1 second between a clause
2. Pause for 2 seconds at the end of a sentence
3. Pause for 3 seconds for a new subject

There are many opportunities to summarize.

Communication skills
Introduction
Open-ended inquiry
The P-A-U-S-E
Reflective listening
Empathy
Non verbal behavior & cues
Summarizing
Signposting
Escorting

- **Front desk reception**
  Initial appointment
  Hospital admission
- **Technician**
  Lab results
  Release appointment
- **Doctor team**
  Physical exam findings
  Treatment plan options
  Rounds

23

"Major download"

Marc and Sonia "Mini"

8. Signposting provides valuable next steps and sets expectations.

Communication skills
Introduction
Open-ended inquiry
The P-A-U-S-E
Reflective listening
Empathy
Non verbal behavior & cues
Summarizing
Signposting
Escorting

- At reception
- Prior to estimate review
- Prior to performing diagnostics
- Prior to performing treatment
- Wait time periods
- Lab results
- Any time in between when you notice something or when someone asks for help

And then what…?

Marc and Sonia "Mini"

9. Escorting is critical to complete the experience.

Communication skills
Introduction
Open-ended inquiry
The P-A-U-S-E
Reflective listening
Empathy
Non verbal behavior & cues
Summarizing
Signposting
Escorting

The Maze Craze!

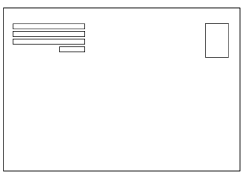
Marc and Sonia "Mini"

There are many ways to inspect what you expect.

- Survey your clients
- On-the-job training
- Monthly or quarterly assessments
- Communication champions

Our closing activity…

You WILL forget 90% of what you learned today.
Reignite the flame!



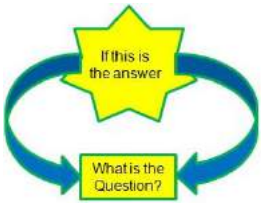
30

Communication skills

There are nine outstanding communication skills to make every interaction a favorable one.

1. Introduction
2. Open-Ended Inquiry
3. The P-A-U-S-E
4. Reflective Listening
5. Empathy
6. Non-Verbal Behavior/Cues
7. Summarizing
8. Signposting
9. Escorting

Q & A!



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Favorable Financial Discussions

SAGE

An approachable team of specialists providing advanced, collaborative, and compassionate care.

Sonia Davis, RVT
Quality Service Manager

Marc Weinstein, MBA
Chief Operating Officer

Symposium '16

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The human – animal bond holds precious value beyond any treatment plan or invoice however…

2

…we must find a way to bridge treatment with reasonable financial options.

Cash!

Check

Credit

CareCredit
Making care possible…today

Third-Party Financing

Pet insurance facilitation

The Bank of
"your practice name here"

3

Back to Table of Contents

Is it ok to review costs in public?

Lobby

Exam room

4

10 steps will help create a favorable financial discussion.

1. Seeking first to understand
2. Expressing empathy
3. Body placement/language, tone of voice
4. Knowing your treatment plan estimate!
5. Benefits to the pet before cost
6. Value for the services we provide
7. Beginning the conversation
8. Handling objections
9. Closing the conversation
10. Gaining buy-in and signature

5

1. Seeking first to understand is critical to establish the relationship.

Seek first to understand
Express empathy
Body placement/language /tone of voice
Know your treatment plan
Benefits to pet before cost
Value for the services
Begin the conversation
Handling objections
Closing the conversation
Buy-in and signature

- Begin with an open – ended question to identify if there any reasons for hesitation
- Provide a safe place to ask questions

6

Identify any possible cause(s) of hesitation which might be fear, sadness or anger.

Seek first to understand
Express empathy
Body placement/language /tone of voice
Know your treatment plan
Benefits to pet before cost
Value for the services
Begin the conversation
Handling objections
Closing the conversation
Buy-in and signature

7

Hesitation shows itself in many ways.

- Fear that their pet will experience pain associated with the procedure
- Fear that testing will reveal a serious or terminal disease
- Sadness about being separated from their hospitalized pet
- Anger about other factors in their life yet expressing anger about our fees

Seek first to understand
Express empathy
Body placement/language /tone of voice
Know your treatment plan
Benefits to pet before cost
Value for the services
Begin the conversation
Handling objections
Closing the conversation
Buy-in and signature

8

2. The client must feel your empathy…or the rest of the conversation doesn't matter.

- Make the charitable assumption
- Inquire using compassionate phrases
- Reflect back any specifics
  - High unexpected costs
  - Wrench in their life, work, pleasure plans
  - Coordinate homecare, pet supervision

Seek first to understand
Express empathy
Body placement/language /tone of voice
Know your treatment plan
Benefits to pet before cost
Value for the services
Begin the conversation
Handling objections
Closing the conversation
Buy-in and signature

9

3. Body placement, language and tone of voice must align with content.

- Body stance
- Posture
- Location in exam room
- Limbs
- Facial expressions

Seek first to understand
Express empathy
Body placement/language /tone of voice
Know your treatment plan
Benefits to pet before cost
Value for the services
Begin the conversation
Handling objections
Closing the conversation
Buy-in and signature

10

"Anxiety mitigated"

Marc and Sonia "Mini"

4. We must know our treatment plan (estimate)!

- Low-end
  - Does not mean lowest level of service offered, or that there were no complications
- High-end
  - Does not mean highest level of service offered, or that there were complications
- Miscellaneous?
- Initial payment = low-end
- What if things change?
- Estimate vs. invoice

Seek first to understand
Express empathy
Body placement/language /tone of voice
Know your treatment plan
Benefits to pet before cost
Value for the services
Begin the conversation
Handling objections
Closing the conversation
Buy-in and signature

12

5. We need to understand the benefits of each line item to the pet.

Be prepared to…

- Communicate benefits to the pet before cost
- Display your house pride
- Reassure with closing statement

Seek first to understand
Express empathy
Body placement/language /tone of voice
Know your treatment plan
Benefits to pet before cost
Value for the services
Begin the conversation
Handling objections
Closing the conversation
Buy-in and signature

13

6. We must know the value for the services we provide.

- Quality of care
- Doctor certification and experience
- Staff availability
- Specialized nursing care

Seek first to understand
Express empathy
Body placement/language /tone of voice
Know your treatment plan
Benefits to pet before cost
Value for the services
Begin the conversation
Handling objections
Closing the conversation
Buy-in and signature

14

"Wow, my baby gets all that!"

Marc and Sonia "Mini"

7. Always begin the conversation with…

- Introductions!
- An open-ended question
- Making quality statements
  - To client
  - About nurse and staff
  - Reassurance to client
- And never rush through the financial discussion unless…

Seek first to understand
Express empathy
Body placement/language /tone of voice
Know your treatment plan
Benefits to pet before cost
Value for the services
Begin the conversation
Handling objections
Closing the conversation
Buy-in and signature

16

"What else before we begin?"

Marc and Sonia "Mini"

8. Handling objections requires excellent listening and observation skills.

Seek first to understand
Express empathy
Body placement/language /tone of voice
Know your treatment plan
Benefits to pet before cost
Value for the services
Begin the conversation
Handling objections
Closing the conversation
Buy-in and signature

18

There may be several reasons behind an objection.

- Asking for a large sum initial payment or deposit
- Financial hardship
- Confusion around described treatment plan

Seek first to understand
Express empathy
Body placement/language /tone of voice
Know your treatment plan
Benefits to pet before cost
Value for the services
Begin the conversation
Handling objections
Closing the conversation
Buy-in and signature

19

"The stall" is one means of demonstrating an objection.

Example: "I would like to think it over."

- Keep calm
- Acknowledge
- Inquire

Seek first to understand
Express empathy
Body placement/language /tone of voice
Know your treatment plan
Benefits to pet before cost
Value for the services
Begin the conversation
Handling objections
Closing the conversation
Buy-in and signature

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"I'll think it over"

Marc and Sonia "Mini"

The "emotional outburst" is another demonstration of an objection.

Example: "What! You want all that now?"

- Keep calm
- Acknowledge
- Act

Seek first to understand
Express empathy
Body placement/language /tone of voice
Know your treatment plan
Benefits to pet before cost
Value for the services
Begin the conversation
Handling objections
Closing the conversation
Buy-in and signature

22

"I don't understand!"

Marc and Sonia "Mini"

9. Closing the conversation begins with…an open-ended question!

"What else do we need to talk about before making a decision?"

Client may need…

- Time to involve family/friend
- Third-party financing processing
- Other

Seek first to understand
Express empathy
Body placement/language /tone of voice
Know your treatment plan
Benefits to pet before cost
Value for the services
Begin the conversation
Handling objections
Closing the conversation
Buy-in and signature

24

10. Gaining buy-in and signature is the final step.

- Initial payment (no “dollars or cents”)
- Signature and best contact number
- Provide copy of signed treatment plan (estimate)
- Expectation of phone call from clinical team
- “Thank you”

Seek first to understand
Express empathy
Body placement/language /tone of voice
Know your treatment plan
Benefits to pet before cost
Value for the services
Begin the conversation
Handling objections
Closing the conversation
Buy-in and signature

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“Let’s do it!”

Marc and Sonia “Mini”

Balancing the Veterinary Ethos and Profitability

SAGE Small Animal Symposium
March 20, 2016

Mike Bilby
CFO

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Hypothesis: Veterinary finance is inconveniently located at the corner of Money and Compassion

Today's Agenda

- The costs of our profession
- The money cycle
- How much profit is enough?
- Profits are good, but cash is king
- The four keys to balance

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3

You are not alone

"Finances overwhelm me." 41%

"I know nothing about finances/investments." 25%

VPI-Veterinary Economics Financial Health Study, 2013

"44% of associates are interested in owning a practice. The most commonly cited reasons for not wanting to own were not wanting to deal with finances and not wanting to manage people."

CVMA Economic Issues Survey, 2013

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Slide 4

Costs

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Slide 5

The Income Statement

Revenues
– Expenses
= Net Income

What you earn
– What you spend
= What you have left

(over a period of time)

Revenue		
Services	100,000	93%
Pharmacy	10,000	9%
OTC	2,500	2%
Discounts	(5,000)	-5%
Net Revenue	**107,500**	**100%**
Cost of Service		
Direct Materials:		
Hospital	5,375	5%
Pharmacy	3,225	3%
Outside Services	2,150	2%
Total Direct Materials	10,750	10%
Direct Labor:		
Veterinarians	26,875	25%
Technicians	17,200	16%
Client Care	3,225	3%
Benefits, Taxes & Insurance	10,750	10%
Total Direct Labor	58,050	54%
Total Cost of Service	68,800	64%
Gross Margin	**38,700**	**36%**
Operating Expense		
Administration	5,375	5%
Rent	7,525	7%
Depreciation	2,150	2%
Repairs & Maintenance, Facilities	1,075	1%
Repairs & Maintenance, Equipment	1,075	1%
Utilities	1,075	1%
Telephone	1,075	1%
Taxes	1,075	1%
Other	7,525	7%
Total Operating Expense	27,950	26%
Net Income	**10,750**	**10%**

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Slide 6

Revenues

Revenue		
Services	100,000	93%
Pharmacy	10,000	9%
OTC	2,500	2%
Discounts	(5,000)	-5%
Net Revenue	**107,500**	**100%**

Revenues (aka Production, the Top Line, *Sales*)

- Gross revenue is before discounts or refunds
- Net revenue: what is earned, and *mostly* what we can expect to receive
  - cash reductions: credit card fees, bad debts
- Discount only when you *need* to

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Slide 7

Cost of Service

COS (aka direct, variable or controllable cost)

- Rise and fall with revenue
- Roughly same % of revenue regardless of top line
- Gross margin is a key financial indicator

Cost of Service		
Direct Materials:		
Hospital	5,375	5%
Pharmacy	3,225	3%
Outside Services	2,150	2%
Total Direct Materials	10,750	10%
Direct Labor:		
Veterinarians	26,875	25%
Technicians	17,200	16%
Client Care	3,225	3%
Benefits, Taxes & Insurance	10,750	10%
Total Direct Labor	58,050	54%
Total Cost of Service	68,800	64%
Gross Margin	**38,700**	**36%**

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Slide 9

Operating Expenses

Operating Expense		
Administration	5,375	5%
Rent	7,525	7%
Depreciation	2,150	2%
Repairs & Maintenance, Facilities	1,075	1%
Repairs & Maintenance, Equipment	1,075	1%
Utilities	1,075	1%
Telephone	1,075	1%
Taxes	1,075	1%
Other	7,525	7%
Total Operating Expense	27,950	26%
Net Income	**10,750**	**10%**

Ops Expense (aka fixed costs, uncontrollables, overhead)

- OE doesn't fluctuate with production
- Cost of business
- Control it, then don't focus on it

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Slide 10

Economies of Scale

			Increase 5%	
Net Revenue	**107,500**	**100%**	**112,875**	**100%**
Direct Materials	10,750	10%	11,288	10%
Direct Labor	58,050	54%	60,953	54%
Cost of Service	68,800	64%	72,240	64%
Gross Margin	**38,700**	**36%**	**40,635**	**36%**
Operating Expense	27,950	26%	27,950	25%
Net Income	**10,750**	**10%**	**12,685**	**11%**

It is generally easier to grow revenue to improve net income than to cut costs.

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Slide 11

The Money Cycle

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Slide 12

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The Money Cycle

Procedure	$500
Revenue	$500
60% Cost of Service	-$300
25% Operating Expenses	-$125
15% Net Income	$75

Annualized:

Practice 1	$ 56,250
Practice 2	$ (37,500)
Practice 3	$ (225,000)

Client 1		
1-Apr	100% deposit	$500
15-Apr	labor, matls, svcs	-$300
30-Apr	operating expenses	-$125
	available cash	$75

Client 2		
1-Apr	50% deposit	$250
15-Apr	labor, matls, svcs	-$300
30-Apr	operating expenses	-$125
30-Apr	50% balance paid	$125
	available cash	-$50

Client 3		
1-Apr	no deposit	$0
15-Apr	labor, matls, svcs	-$300
30-Apr	operating expenses	-$125
1-May	pymt #1	$42
1-Jun	pymt #2	$42
1-Jul	pymt #3	$42
	available cash	-$300

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Profit

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Prof·it (noun) defined

money that is made in a business, through investing, etc., after all the costs and expenses are paid : a financial gain

Merriam-Webster (m-w.com)

Without profits, 1 of 2 things happens:

Investment stops (wage increases, benefits adjustments, clinical equipment)

or

Borrowing begins (and deficits increase)

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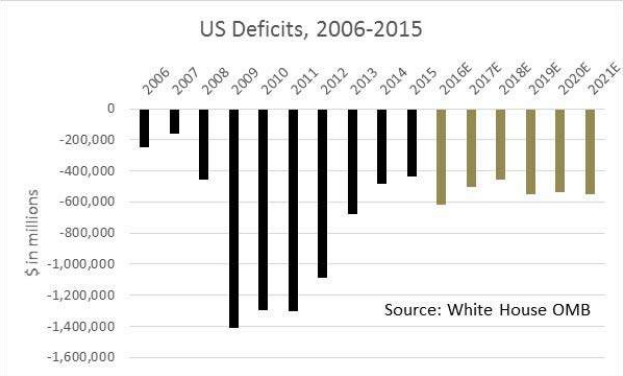
Three economic levels of profit/(loss)

#1: US Government

$7.5 *trillion* dollars spent more than earned in 10 years



$1M in $1,000 bills = 4 in. stack

$1B in $1,000 bills = 364 ft high

$1T in $1,000 bills =

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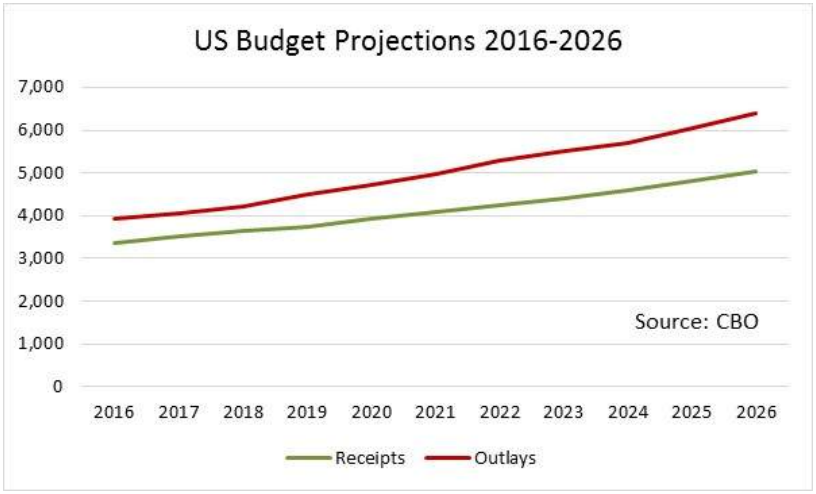
Federal Outlook



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Three levels of profit

#2: Personal

What do you do when expenses exceed revenues?

	Annual	Monthly
Salary	100,000	8,333
Taxes	33,000	2,750
Loans	19,000	1,583
Rent	14,400	1,200
Food	7,200	600
Auto	6,000	500
Insurance	6,000	500
Household	3,600	300
Pets	2,400	200
Utilities	1,800	150
Cable	1,200	100
Phone	1,200	100
Savings	2,400	200
Wine	2,400	200
Expenses	100,600	8,383
Income/(Loss)	(600)	(50)

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Three levels of profit

#3: *Your* practice – it's no different!

Are revenues appropriate? (prices, discounts, ACT?)

Are expenses consistent with industry standards?

Will there be money left over this year to re-invest? To service existing and new debt? What about next year?

The right level of profit for the practice is the level that meets the needs of the owners

Profit ≠ Greed, Profit = Growth!

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Cash

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Three rules of Cash

1. Income ≠ Cash
2. Develop a budget
3. Bankers are good people to know

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Cash Management

Your practice can generate income and go out of business.

Revenue		
Services	100,000	93%
Pharmacy	10,000	9%
OTC	2,500	2%
Discounts	(5,000)	-5%
Net Revenue	**107,500**	**100%**
Cost of Service		
Direct Materials:		
Hospital	5,375	5%
Pharmacy	3,225	3%
Outside Services	2,150	2%
Total Direct Materials	10,750	10%
Direct Labor:		
Veterinarians	26,875	25%
Technicians	17,200	16%
Client Care	3,225	3%
Benefits, Taxes & Insurance	10,750	10%
Total Direct Labor	58,050	54%
Total Cost of Service	68,800	64%
Gross Margin	**38,700**	**36%**
Operating Expense		
Administration	5,375	5%
Rent	7,525	7%
Depreciation	2,150	2%
Repairs & Maintenance, Facilities	1,075	1%
Repairs & Maintenance, Equipment	1,075	1%
Utilities	1,075	1%
Telephone	1,075	1%
Taxes	1,075	1%
Other	7,525	7%
Total Operating Expense	27,950	26%
Net Income	**10,750**	**10%**

Only Interest Expense hits the Income Statement

Debt principal reduction reduces the Balance Sheet liability

Owners take ROI distributions from Equity

Assets	
Cash	100,000
Receivables	50,000
Building	2,000,000
Equipment	500,000
Other Assets	200,000
Total Assets	2,850,000
Liabilities	
Accounts Payable	25,000
Accrued Liabilities	100,000
Mortgage	1,500,000
Loans	400,000
Total Liabilities	2,025,000
Owner's Equity	825,000
Liabilities and OE	2,850,000

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Free Cash Flow

The amount of money left over for re-investment

Net Income	$129,000	$129,000
Plus: Depreciation Expense	25,800	25,800
Less: Debt Service	-60,000	-10,000
Less: Owner Distributions	-75,000	0
Equals: Free Cash Flow	19,800	144,800

Low Debt Service or Low Owner Distributions can strengthen cash, future investment

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Cash Management

Develop a budget or operating plan—and stick to it!

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec	Total Yr
Revenue	50,000	50,000	50,000	50,000	75,000	100,000	100,000	100,000	75,000	50,000	50,000	35,000	785,000
Salaries	22,500	22,500	22,500	22,500	33,750	45,000	45,000	45,000	33,750	22,500	22,500	15,750	353,250
Benefits	3,000	3,000	3,000	3,000	4,500	6,000	6,000	6,000	4,500	3,000	3,000	2,100	47,100
ER Taxes	1,500	1,500	1,500	1,500	2,250	3,000	3,000	3,000	2,250	1,500	1,500	1,050	23,550
Supplies	3,500	3,500	3,500	3,500	5,250	7,000	7,000	7,000	5,250	3,500	3,500	2,450	54,950
Cost of Service	30,500	30,500	30,500	30,500	45,750	61,000	61,000	61,000	45,750	30,500	30,500	21,350	478,850
Gross Margin	19,500	19,500	19,500	19,500	29,250	39,000	39,000	39,000	29,250	19,500	19,500	13,650	306,150
	39%	*39%*	*39%*	*39%*	*39%*	*39%*	*39%*	*39%*	*39%*	*39%*	*39%*	*39%*	*39%*
Operating Costs	15,000	15,000	15,000	15,000	15,000	15,000	15,000	15,000	15,000	15,000	15,000	15,000	180,000
Net Income	4,500	4,500	4,500	4,500	14,250	24,000	24,000	24,000	14,250	4,500	4,500	(1,350)	126,150
	9%	*9%*	*9%*	*9%*	*19%*	*24%*	*24%*	*24%*	*19%*	*9%*	*9%*	*-4%*	*16%*

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Cash Management

Develop a strong relationship with your bank—in many ways they see you as their greatest asset

- They get to know you
- They get to know your business
- They are networked

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Balance

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Slide 27

The Four Keys of Balancing Veterinary Ethos and Profitability

Communication

Talk: here's where we need to be, and why
Profits are not a bad thing

Planning

Is the budget realistic to provide

1) the service we (and our clients) expect
2) at the prices we charge
3) for the profit we need

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The Four Keys of Balancing Veterinary Ethos and Profitability

Flexibility

We can't do everything right now
Guidelines are good, and exceptions happen

Moderation

Singles win more ball games than home runs
Little things add up

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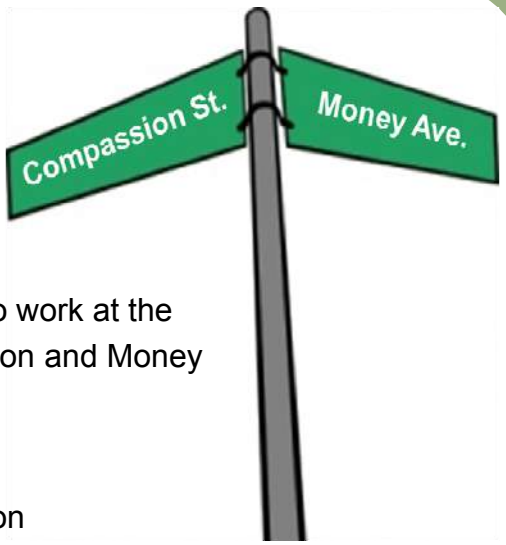
Summary



This isn't a bad corner to hang out at.

Successfully preparing to work at the intersection of Compassion and Money will help create:

- Aligned expectations
- Stronger team cohesion
- Sustainable future for our practices, employees, patients and clients.

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More help?

AVMA
DVM360.com
CVMA
AAHA
mbilby@sagecenters.com

Q&A?

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Products: A Crucial Step to Client Happiness and Practice Profit

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Inventory; Wikipedia defines it as a list of goods and materials, or those items themselves, held available in stock by a business. Many practice managers define it simply as a "pain in the rear". And herein begins our love-hate relationship with bottles, pills and bags. In veterinary practice, inventory is essential to the operation of the practice including the provision of products and services to clientele. It is the second largest expense in the practice, behind payroll, and is one of the only expenses that fluctuate dynamically on a daily basis. Many of the items are small, and difficult to monitor, there are profit differentials between classes, and those items that are primarily for client convenience and preventative health use are now subject to "outside the clinic" competitive pricing pressure from other over-the-counter (OTC) sources.

By the 1980's nearly 10% of all prescriptions are being filled outside the veterinary office. Current estimates range from 20% to 30%. So the question remains, how much product will be sold at locations other than the veterinary hospital in the coming decade? To answer that question, and what impact it has on the veterinary industry, we need to look at what is driving the change, and then what options can be considered to address it. Bigbox retailers, on-line sales sources, and direct-from-manufacturer sales to the client have been a recently trending product sale pathway. In 2008 total Flea and Tick Products sold OTC were 4.6% of total sales, in 2009 that jumped to 17.6%, a 380% year-on-year increase. At the same time sales of flea and tick products through practices were down 14% in 2010 on the West Coast, representing a loss of more than one million doses in this region alone[1]. That's 8,000,000 doses of canine flea and tick products moving through OTC ("other than clinic") channels. The resulting math does not paint a pretty picture. If the average price per dose sold is $13.00 that yields a change of $104,000,000 in lost revenue to veterinary practices just between 2008 and 2009. Searching a single online auction site revealed 2,698 doses of parasiticides available.[2] For the fiscal year of 2009 1800-PetMeds announced record sales including a 17% increase in net sales of $219.4MM, $156.8MM in reorders, and 802,000 new customers (up 13% over 2008).[2] We need to dig a little deeper in order to discover what is driving this change.

Why are people shopping elsewhere? Because they can! And today they demand convenience. 75% of survey respondents indicated they would visit their vet's on-line website and store four (4) times per year if they had one, 76% would sign up for reminders and 91% said they would use a reliable pet health library and purchase history.[3] A Nielsen Online survey of another 1000 shoppers provides additional insight as to why. 81% responded that the primary reason they shop on-line is convenience, yet only 46% shopped specifically for lower prices. Studies at both the University of Missouri and Iowa State University indicated a primary finding regarding keeping customer / brand loyalty intact was making it easier to shop from you than any other competing sources.[4] Still think prices are driving your clients away? Think again. With average client appointment wait times of 17-22 minutes in small animal practices[5] rest assured many practices observe similar delays between the elapsed time when a client enters and subsequently exits a practice solely for the purpose of buying a product. As the graph shows below, clients are concern about cost, but the profession may be focusing on this issue too much.

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REBRANDING YOUR PRACTICE: GOING FROM COW TO WOW! PART 1

Byron S. Farquer, DVM, AVA *– Simmons & Associates*
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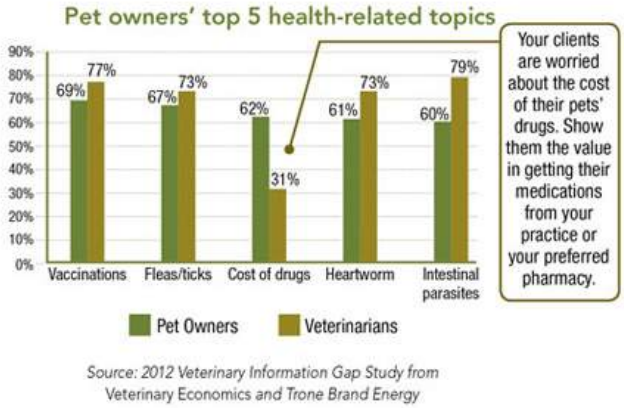
There are 3 components to a successful practice. Success being defined as that magical continuum where doctors get to practice the best medicine possible, can afford to, and the practice grows and grows as a result of clients expressing to others their joy and pleasure of using this particular practice. The senses are delighted, the trust level is strong, behind-the-scenes attributes strengthen the client-practice bond, and word of mouth advocacy is high. These three components are simple to identify, but difficult to define. The more one tries, the deeper one needs to go to adequately grasp their importance. These three components are:

Consumer Experience, Customer Service, Perception of Value

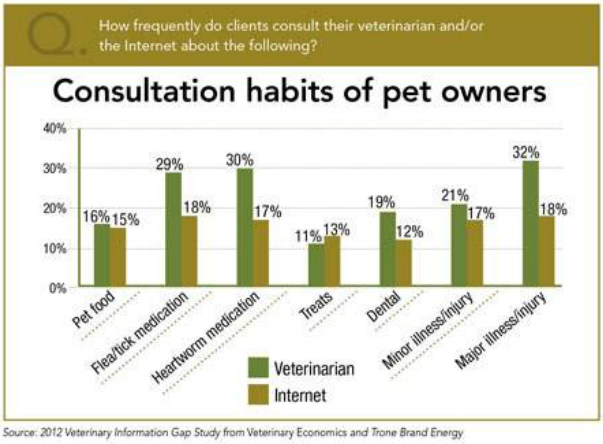
Consumers (your clients) chose to do business with providers that can make the experience pleasurable even in the worst of times, provide exemplary customer service, and convey the true value in relation to price. Give a customer a $10 hamburger that they feel is worth $5 and they go away mad, often never returning. Provide that same customer with a $10 hamburger that, in part due to the whole customer experience, creates a value proposition that the hamburger "dining experience" exceeded expectations and you have a have a happy customer. This isn't something that can be achieved by having a clean restaurant (clinic), polite waiter (your staff) and good pricing. It isn't quite that simple. There is a dynamic psychological interplay between the senses in all aspects beyond the tastebuds and assessment of the waiter's manners and efficiency. The building, the ambiance, the location, the food, the music, the smells, the "everything" all helps shape the customer's perception of value. Those same forces exist when a client comes into a veterinary hospital for services. This whole package effect is representative of the 'atmospherics' a term used to describe the combined ambiance/atmosphere etc… experienced when visiting a location. Its goes beyond paint and politeness, it's everything all working together that creates a hard to define but easily identified "package" that creates a very positive consumer experience. Disneyland is a good example. At its core, we have amusement rides, not un-like other parks or on a smaller scale, county fairs. BUT, and this is a big "but", anyone that has been to Disneyland knows that it's so very different. Everything about the place harmonizes to create an unforgettable experience. And it isn't cheap either, so price alone (being very competitive in pricing veterinary services for example) isn't enough to win the hearts of consumers/clients. Being clean, alone isn't enough, nor is having high quality (high quality rides or high quality medicine). It requires "everything" to be cohesive. Cohesion is partly what creates high quality consumer experiences, whether it be Disneyland, your practice or the restaurant. Provide a great tasting hamburger in a less than pleasing facility or with marginal wait staff and the experience just isn't the same.

Gone are the days when the veterinarian would come to a town that had no doctor, buy an old two story house, tack a shingle to the front of the house that read "Veterinarian" and set up shop on the main floor. There was the doctor and his wife. He did the medicine. She was receptionist, bookkeeper, animal handler and practice manager–a perfect manifestation of the nineteenth century American Mom & Pop small business model. There was a lot to envy in this style of life for the "Doc" and his family. He could take a two hour lunch at home and hang out a gone fishin' sign when trout season hit.



Source: 2012 Veterinary Information Gap Study from Veterinary Economics and Trone Brand Energy

Visiting a website I was greeted with the following message. "Welcome Google Customers, mention 'GoogleClick' at check out and receive $5.00 off plus free shipping on orders over $35.00." The Advantage for Dogs offered on the PetCareRx website listed the price as: "6 month supply ~~$89.99~~ $63.99 Save 29% vs. Vet's price (+ free shipping). When you land on the website you are immediately offered an opportunity to chat with Live Help, if you leave this website without purchasing, another instant-chat box pops up asks you to "Please Wait! We can help. We have a special one day only 20% off any order…." aka a special deal for you. It's perhaps a little too much huckster for my taste, but it certainly prompts me to think about the average practice and then pose the questions "How long did it take to be asked what I needed?", and "Was there any attempt to ask me again if I needed anything before I left?" Retail Industry acknowledges this as customer service 101, and maybe we aren't providing enough of it. Interestingly I did <u>not</u> go to the phone book or window shop to find them. Many of your clients are shopping the same way, on-line. But interestingly many of your clients still want you involved, value your advice and prefer shopping at your practice. If you can offer an excellent shopping experience from your practice (on-line and in-clinic), with excellent customer service AND follow up, clients will "shop at home" with you.



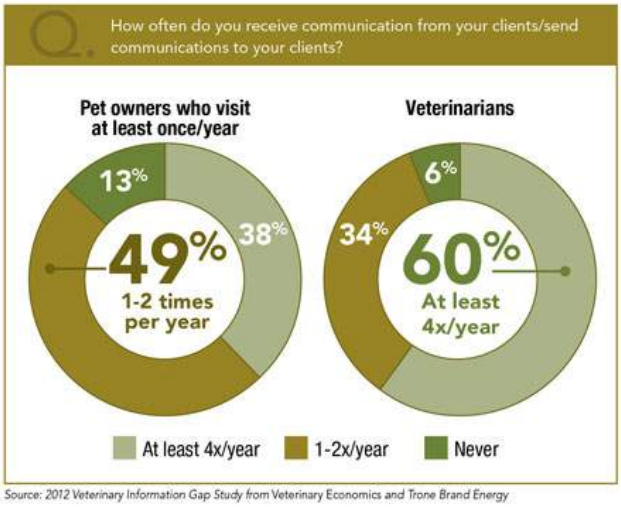
Source: 2012 Veterinary Information Gap Study from Veterinary Economics and Trone Brand Energy

Traveling all over the US these past two years, it's clear veterinarians are feeling economic pains and pressures from loss of product sales. Many practices depend upon the sale of supportive products as a source of revenue for the practice. Parasiticides make up 9% of practice revenue on average.[6] In order to keep these and other sales up we will have to

address the client convenience. We will need to market our products and services using websites, social media, and by improving good old fashion courtesy during in-person sales. We may need to address office hours (Walgreens and Websites are open 24/7), customer service etiquette, telephone hold times, and the "consumer experience" itself.

In order to manage inventory, one must have an inventory manager complete with job description. Simply giving someone the title Inventory Manager, without a clear roadmap of benchmarks and instructions is a sure way to encourage disappointing results. Most practices find that product costs represent approximately 18 to 20% of gross revenues, and is second only to payroll as the most expensive cost of doing business. You need someone who understands the importance of inventory management which includes proper pricing, marketing and profit. Some of the best information on developing an inventory management role comes from Brenda Trassava, a Certified Veterinary Practice Manager. I would recommend you explore her website www.vetmanageradvisor.com for additional useful hospital and inventory management information. Just because you have an Inventory Manager, you cannot "wash your hands" of everything that has anything to do with inventory management. It is important to remember that oversight must be in place. Someone, namely the Practice Manager or Owner must be a part of the checks and balances process. Everyone is responsible for providing products to the client in an educational, efficient AND profitable manner.

Pet owners overwhelmingly reiterate their respect and desire to receive timely and accurate health information from medical professionals. Veterinarians are uniquely qualified to provide pet owners with the best medical advice, including product recommendations. Trust is built between the practice and client when communication remains consistent and frequent. The more clients interact and purchase products from a veterinary practice, the stronger the bond between client and the practice. Empirically, this translates to preservation of revenue from current and future product sales. Interestingly veterinarians may not be communicating frequently enough or effectively with their clients. They may be overestimating their client reach.



Source: 2012 Veterinary Information Gap Study from Veterinary Economics and Trone Brand Energy

You need to manage the inventory, ensure someone is in charge, price correctly and market in ways you never have before. I can only hope private practices are able to remain the client's primary advisor and pet health advocate, while reinventing ourselves as a competitive and

convenient source of pet products. Do not give up your product sales, clients want to shop from you, they trust you, and product sales can be profitable if managed correctly.

How can you keep your pharmacy sales?

1. **First, join a buying group**. Done well these groups can reduce drug costs and benefit from an economy of scale. Use strategic mark ups rather than across the board rules of thumb.

2. **Urgent medications** (antibiotics, pain medication, injections, etc…) are not as subject to price shopping. The urgency of need facilitates a client's tolerance of cost. These items can have strong mark-ups.

3. **Pricing items that are no longer exclusive to veterinarians** (ie. certain flea products, etc.) should be priced considerably differently, usually lower than you previously offered. You don't need to price match but you need to be close. Once the exact same product you sell shows up everywhere else, you are now compared to everyone else. If I can Google a product you sell and find it readily available in many other locations, price becomes an issue. Can't price the product low enough to be competitive and still turn a profit? Maybe it's time to get those price sensitive products off your shelf if you have another good quality substitute.

4. **Dump the dual-marketed products**. If a veterinarian used to be the only source of Product X but now every internet site, big box retailer, feed store and catalog sell the same product, it's time to differentiate yourself. Yes it might have been a big seller for you previously but when you have the exact same product everyone else does, it becomes a commodity. Think about a coach airline seat between Denver and Chicago between Noon and 2pm. If 5 airlines all have flights, and all are reasonable the same regarding service and amenities then price becomes a very important means of choice of airline. But if there is also a chartered jet available with a ticket cost that is slightly higher but not exorbitantly, the choice becomes more difficult. The charter jet won't require a strip search before you board, will generally offer high-end refreshments and excellent customer service, has no baggage claim delay and no yelling children (unless you are bringing your own with you). Having a hard time letting go of certain products because you've used (recommended) them for a long time? Ask yourself how long didit take you to discontinue selling flea collars, powders and home foggers when the new generation flea medications came out. Months? Years? No. Most of us strongly encouraged our clients to switch products in a very short period. They listened to you then, they will listen to you now.

5. **Surround yourself with veterinary exclusive products**. Exclusive products doesn't have to mean expensive, it simply means "access restrictive". When you fill your shelves with the same products clients can buy from Wal Mart, you give up your veterinary-exclusive advantage. You blend in. You are just one of many choices.

6. **Dump the generics**. Despite claims that all generics are equal to the original brand name or legacy drug, many of us have experienced variations in generic drug performance both as veterinarians on the prescribing side as well as general consumers. The public expects generics to be cheaper in part due to competitive pricing but also as a result of their own experiences (perceived or real) they feel generics don't always perform as promised. When you dispense the same generic drug as Costco or Publix

your client expects price to be the most influencing factor. Most of the big box retailers avoid legacy drugs due to lower margins or restrictive sales policies imposed by manufacturers. They are focusing on price alone, and you usually can't win that battle very often. Legacy drugs offer you some level of veterinary exclusivity or differentiation. If you are preferentially handling generics because your are bent on "saving your clients money" in hopes they will like you better, you are dismissing reams of behavior research that indicates cost is not the single most important driver for consumer decisions.

7. **Fight to keep the Prescription.** Make a solid argument why you the veterinary practice, rather than the retailer or internet warehouse is the best source for pet meds including verification of chain of shipping, manufacturer warrantees and adherence to storage and quality control. Don't just sign the fax request. Call that client. Talk to them about their desire to purchase somewhere else. Price will usually be the first comment but dig deeper, there is often more to the story. And don't be surprised if your client just thinks you are priced higher when in reality you are not. Some practices use signage or other client communications to show clients that they are truly price competitive on certain products. If you really want to play the price matching game then make your website's on-line pharmacy a bit more competitive than your in clinic pricing, but impose the same purchase process restrictions as PetMeds, etc… The on line shopper has to place the order, pay in advance, commonly pay shipping, wait for it to be delivered, etc… as opposed to walking into your practice, getting expert advice and receiving the product right on the spot. But if you are not going to offer superior customer service at the front counter then a cheaper price on the internet can easily win over your client.

8. **Use prescription drugs when possible.** Consumers often perceive prescription drugs as more powerful, even when they are not, however that may be true of some formulations. Many newly released drugs (newest pharma-technology) are *only* available by prescription. Prescriptions also require better record keeping than OTC medications. Prescription drug dispensing may require additional doctor examinations, offering benefits beyond additional revenue such as more frequent veterinary-patient-client interactions which translate to improved compliance and continuity of care.

9. **Stay in the loop of treatment.** Dispensing oral pills with instructions to return in two weeks if things don't improve opens the door for client compliance induced treatment failure. Incorporating injectables (ie. Convenia), technician applied treatments (ie. every 5th day otitis flush and deep medication), or other techniques like smaller dispensed volumes for chronic treatment medications (thyroid or NSAIDs) requires more return visits. Client-patient touch points and of course refills.

10. **Re-pricing your Services.** The industry has been lax on seriously structuring its services pricing. Annual "across the board" % mark ups is as sophisticated as many practices get with pricing their services. Many practices have been adjusting fees on services to make up for reduced product mark ups and its corresponding contribution to profit. You can calculate the expected cash flow at a current drug's pricing level and then again at a new lower price. Take that expected lost revenue and spread it over a number of service categories in the form of price increases on service items. Non-shoppable services are ideal. Cashflow is preserved, drug prices remain competitive and services carry more of the load. When prices go up on drugs and you feel you need to freeze your retail price, you need to redistribute the lost cashflow to other sales. That may be other drug sales (ie. prescription drugs in part due to the aforementioned reasons) or they may be redistributed over your services fee schedule.

11. **What Business are you in?** Be realistic with your expectations. Are you in the high volume, low margin product sales business or are you in the veterinary medical health industry? Do you provide prescription drugs because it's a useful tool in your arsenal to fight disease and dispense other products to enhance the pet's well being or are you trying to gain a competitive pricing edge on Wal Mart? It's ok not to be very price competitive when you offer uniqueness. Imagine Denny's trying to sell a cup of diner coffee for $4.95, or even Starbucks trying to sell its unique coffee in a Denny's at the same price schedule?

    Pharmacy and product sales remain an integral part of the current veterinary business model. Until that model dramatically changes, until practices are able to develop a successful sale strategy that is nearly 100% services only dependent, we are going to need to preserve product sales. You just can't blindly mark up drugs 100%, make across the board price % increases, and give away your services anymore. As much as we hate to hear it, we have to run practices like businesses. Luckily most don't require you have a Masters in Marketing and an MBA in order to remain profitable. You just have to be smart, competitive and offer an exceptional sales experience between your staff and your clients.

[1] Combined data and statistics from Elanco, Webster, GfK Kynetec. June, 2010.
[2] Combined research, Hodgkins, SummitVetPharm, July, 2009
[3] VetSTREET Survey, VetSTREET 780 Township Line Road, Yardley, PA 19067.
[4] Nancy Giddens, University of Missouri, 2009. Connie Hardy, Iowa State University Extension, 2009.
[5] Wait Time Survey and Digital Signage Industry Info, Jimmy Schaeffler, Digital Signage - Software, Networks, Advertising and Displays: A Primer for Understanding the Business, pp. 296, Focal Press, 2008.
[5] Dave Titchenal, Wait Times in 120 Veterinary Hospitals Survey, 2008. emebaVet, Sonora, CA.
[6] Christine Merle, DVM, MBA, CVPM, North American Veterinary Conference, Lecture: Power of You, 2010.
[7] Brenda Tassava, CVPM, www.vetmanageradvisor.com, 2012
[8] DVM360.
http://veterinarybusiness.dvm360.com/vetec/article/articleDetail.jsp?id=783973&pageID=8

Competition drives change

The day a second veterinarian moved into town, things changed. The needs of the clients would begin to become more important than those of the doctor. Now the pet owners could choose. What would they base their choice on? Both men were doctors–from the consumer's point of view both were qualified. Convenience became a big issue. Doctor "x" might be open during the lunch hour or after work. Doctor "y" might be closer to the house or easier for driving and parking (location, location, location). After issues of convenience, the consumer had to rely on her senses: which hospital "looked" the best? To the consumer, you are as good as you look.

Your building is your best billboard

As a small business, veterinary hospitals have limited funds for advertising. For the most part, advertising is restricted to the yellow pages, some direct mail, the internet, word of mouth, perhaps some occasional radio and newspaper and, rarely, television. The biggest, most important advertising is the practice building and sign. Location, architecture and design can have a huge impact on the success of the practice. If you are as good as you look, it is very important to not only look your best, but to also look better than the competition.

Our choices are irrational

What happens when a potential client drives by or walks into your hospital? The psychological effect of this experience can be tremendous. The client is a walking litmus test of sensory input. Unconsciously the hospital layout, design, colors, noises and odors make their mark. As humans, we decide whether we like something or someone in one fifth of a second! Many of the factors in this decision are invisible to the conscious mind. In other words, they are irrational. People should choose doctors and hospitals based on a careful examination of their success rates, medical protocols, pain management programs, diagnostic skills, etc. Unfortunately, human beings are human and base their retail choices on their feelings. Over the last fifteen years we have heard countless veterinarians complain that they practice the best medicine in town and still are barely scraping by economically. As if proof were needed, this was a clear demonstration that a medical degree is no guarantee of business and financial success! Business administration is a different skill set. Success in the retail world depends on our ability to please and delight the consumer.

If you build it, will they really come?

Why go to the trouble to give attention to anything but the floor plan and materials? Can I really build a facility that embraces all that the consumer wants, loves and subconsciously responds to, and will it actually make my practice better than if I just do the utilitarian approach?

Dr. Tom Van Meter is a strong case in point, illustrating the progression from traditional functionality into state of the art embracement of consumer psychology.

His original practice, located in a 1600 square foot block construction building typified the classic veterinary facility. Basic, utilitarian, and unassuming. It was clean, but "cold" visually, and portrayed sterility and function. Revenue grew as the practice did until it began to peak. A professional marketing firm re-branded the practice, addressing many of the above issues including signage, color, etc… and created a boutique approach to image. Sales that were flat at $503K or declining once again grew 20 to 25% over the next 22 months, proving that how your building looks, and its match to your practice's self proclaimed image is critical for success. Valuation of the practice changed also, moving from $300K to $450K in the same period. Growth again occurred from a local merger, but soon they were busting at the seams, with carts parked in hall ways and elbows bumping against one another. A new facility was imminent however a divergent path existed. Either choose function only, stay with the traditional approach that the doctor will design the floor plan, chose easy to clean materials, and the spouse will pick the colors, or choose to do it right, embracing all that is described in this seminar. He chose the latter. Today there stands a facility that is beautiful, functional and appealing. Where covered entry ways and courtesy umbrellas are available for rainy days, fresh brewed Starbucks coffee and fresh baked chocolate chip cookies replace the otherwise familiar odors of a practice waiting room, framed art and fresh cut flowers adorn the rooms and small touches like heated exam tables for cats and small dogs, typed visit reports given to each departing client, exercise areas, and bar-less cage doors help round out the "wow" experience. And the "wow" shows up on the P&L too. This practice has consistently posted 20% growth rates, clients drive upwards of an hour past other veterinary providers, new clients numbers reach as high as 200 per month. That's 200 per month, not per year, and support staff and doctors routinely drop off resumes seeking employment. Revenues exceed $3MM and practice value is respectably >$2MM. This practice financially performs well, AND delivers on its customer service promises. It does it more effectively and efficiently than would be possible in the traditional designed practice, or the Taj Mahal that looks pretty but lacks true attention to the very rules that govern successful retail and entertainment architecture.

All of this makes sense only if we understand what business we are in. If we think we are in the business of being really good doctors in the most efficient and practical way possible, this makes no sense at all. If we are in the business of making clients feel good while providing great and compassionate medical care for their companion animals, then it makes a great deal of sense. Then we have to ask ourselves, what kind of facility built in what kind of location will make them feel the best? Every owner will be tempted to make her or himself the center of focus in the practice and will be tempted to accept architecture and design that pleases him or her. The wise owner will endorse the vision of a client-centered practice and will insist on esthetics based on what the client wants. It is the client who is the key to the financial success of every veterinary hospital.

So yes, if you build it they WILL come. And if you do it right, they will come in droves.

REBRANDING YOUR PRACTICE: GOING FROM COW TO WOW! PART 2

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Branding, Marketing, Advertising

These words, although often used interchangeably, aren't. Advertising describes tools you use in marketing which is literally the execution of your marketing plan itself, all of which is designed to create your Brand. There are all types of advertising tools available, and marketing strategies include both inbound and outbound formats, but in the end the single purpose goal is to create Brand for your practice. Not in the sense that Coca-Cola created a brand, yet exactly in the same way that it's brand controls market share. Your market is your immediate community, and establishing your practice as the premier "brand" of veterinary services should be your goal. Wise words come from many sources, some of which are included in this manuscript from veteran marketing experts including Donald Erceg, and commercial marketing sources like HubSpot (www.hubspot.com) and SocialMediaExaminer (www.socialmediaexaminer.com) as well as bestselling authors of books like Tipping Point, Made to Stick, Gung Ho, Good to Great, Juicing the Orange, Raving Fans, and others. It will remain difficult for anyone to really embrace excellence in marketing unless you make a commitment to read these books and others. I can't imagine holding out expectations of being a good veterinarian if I was unwilling to read veterinary books. The same process applies with marketing and brand development.

Lifestyle and Relationship Marketing

Successful veterinary advertising targets the world of the woman who is the primary consumer of small animal veterinary services, paying particular attention to the issues of quality pet care, compassion, convenience and, most importantly, to "getting her" as a person. Health care for her "family" is a job she takes very seriously. She wants quality care and she will pay a premium to get it. Marketing is a way of reaching out to her and telling her that your hospital is the one for her. In deconstructing the success of marketers like Martha Stewart, Oprah Winfrey, Nike and Starbucks, we can see that "lifestyle marketing" is the most powerful form of marketing. If you can reach out and touch her in the core of her lifestyle she will, indeed, beat a path to your door.

To keep her, you'll need to communicate with her continually, not simply sell to her. That means embracing communication pathways she likely uses. Where one-way newsletters gave way to email, today the world is redefining how it communicates, accepts marketing, and acts to purchase goods and services. Marketing experts recognize this modern behavior exhibited through the growing usage of social media. Today social media as well as reputation management (addressing consumers praises, rants and raves on review sites like Yelp!) should be core to your marketing efforts. As if all this branding stuff wasn't already confusing enough, we now have to add "relationship marketing" to your vocabulary.

Building Your Brand

Brand names control market share in the retail world. Again think Nike and Starbucks. Do people prefer their products because of scientific proof of excellence? No. People prefer brand

products because they trust them more. It makes them feel better to buy Nikes. Effective brand marketing for veterinary hospitals tells the family health care giver, that you "get her"--that you understand that her pet is part of her lifestyle and your hospital is totally centered on her and her pet as the center of your universe. Your brand marketing is a promise to deliver to her a client-centered practice that stresses quality, compassionate pet care delivered with high-touch client service. The final part of good marketing is keeping your brand promises.

To develop a home in the public mind, you must first develop a compelling image for your hospital. Many doctors associate Image with the hospital logo. The logo is only the beginning. Your image is everything you look like, smell like, sound like. Everything about your practice that touches the senses of the consumer builds your image. Great care should be paid to your hospital name, logo, colors, choice of typefaces, style of layout, use of photographs. Usually, the final choices are made by the doctor/owner. A strong argument could be made that the doctor/owner is the least qualified of all people to build a company image. “I like it” or “I don’t like it” is a terrible way to make those decisions because he or she has no qualifications or experience to make good decisions regarding the development of corporate image. Just as graphic designers and marketers are not qualified to practice medicine, doctors are not qualified to develop corporate identities. That important job should be entrusted to a professional agency with a proven track history in image development. It is an accepted truth that an attorney who chooses to represent himself has a fool for a client–even though the attorney is trained to practice law. How much more foolish is it for a practice owner to develop his own public image—a job for which he is totally untrained?

Clients are the experts on their own needs and wants. How can we best serve the owner/pet bond? Creating a client-centered hospital has to become the mission of each and every member of the hospital team. This leads to a client-centered culture that always places the client first. A happy, delighted, well-served client is the cornerstone of good practice management and financial success.

Client-Centered Benchmarks

High-touch Service. It is both profitable and socially responsible to provide client-centered, high-touch service. It is the right thing to do.

Happiness. Everyone wants to be happy. Focus your energy on the client’s happiness and the rest will follow.

Relationships. While technical proficiency may be number-one with the doctor, his or her relationship to the client is more important to the client. This makes bedside manner more important to the client’s happiness than the doctor’s medical skills. This is exactly where social media plays a large role.

Customer Satisfaction. Customer satisfaction is no longer the goal. Companies like Nike, Starbucks and Nordstrom have raised customer expectations far beyond “satisfaction.” The new goals are “delight,” “awe” and “surprise.” (Pay special attention to your on-line comments from review sites like Yelp.com and Vetratingz.com. The new buzzword is “reputation management” and it’s something you absolutely must address.)

Committed to excellence. Every hospital says that it is committed to excellence and has a tradition of quality. But is it committed to making the client feel good? For the client-centered practice, client happiness is the definition of excellence.

Feeling Good. Most of us equate "happiness" with "feeling good." Everything the client sees, hears, touches, and smells should have the single purpose of making that client "feel good." Remember, providing great care for the client's pet also makes the client feel good. What are some practical, simple steps that can be taken to create a client-centered-practice?

Customer Service doesn't start with the customer

Hire for attitude and train for skill, a motto coined by Southwest, is generally ignored in the medical profession. Why is it that we tend to define practice positions established basically upon a framework of duties that require certain skills, hire someone to fill that role, only to then be disappointed in the performance? Perhaps too often veterinary staff is hired for skill when what we should be doing is hiring for attitude first. A sign adorns one large corporation that says, "We don't train people to be nice, we simply hire nice people". You can train, and teach skill sets. You can improve and foster advanced application, but it is rare, at least at the adult level that we hire from, that you will be able to instill positive work ethic and attitude.

Southwest Airlines Model

Southwest Airlines has been profitable every year in its 30+ years of history even after 9/11. Why? Great management, service, quick routing? Interestingly a major component of that success relates to having an incredibly dedicated work force, founded upon positive attitude, empowering employees, and garnishing it with respect. As former CEO Herb Kelleher said, "We are not an airline company with great customer service. We are a great customer service organization that happens to be in the airline business." Now, substitute "veterinary" for "airline" into the last sentence. Hiring the right people has been a hallmark of Southwest.

They look for people with an attitude that is positive, creative, and people-oriented. Are you still looking for "5 years experience as a kennel assistant, Acme Vet Tech correspondent graduate, good MS Word skills and familiar with Cornerstone"? Southwest trains for the rest, the skill set, ---and it REALLY works. That's not to say possession of certification (registered technician) should be ignored. It's also a vital component to your staff. But hiring for attitude might really serve you better when it's placed at the top of the criteria list. Customers are NOT #1 at Southwest---employees are, period. If you treat your employees right, they will treat your clients right. And, at Southwest, "outrageous customer service" is commonplace. It takes getting the right people on the bus, then getting them in the right seats. Veterinarians continue to miss the first critical step: the right people. The seats are the skills required. So the second step is ensuring the person you have on the bus (your practice) is matched correctly with their skill set (the right seat), but that first requires gathering great attitudes (the right people).

Southwest Airlines created a great company personality by adhering to the following key points.

- Never give up
- Focus on people
- Be resourceful, willing and able
- Promote esprit de corps
- Know what you do best, and quit doing what you can't do best.
- If push comes to shove choose humor over skill

So, how do you translate great customer service over to a veterinary practice? Answering the phone by the third ring, minimizing on hold times, returning calls promptly, keeping waiting times short, fresh flowers, water and coffee and a snack, current magazines that are right for your clients (popular mechanics vs. Oprah), progress reports, beeper access for critical patients, carrying pet food to a client's car, umbrellas, using the pet's proper gender and the most overlooked of all, eye contact with a big smile.

What is Presumed Excellence?

At one time being a highly qualified and medically competent doctor was all that was expected in practice. You hung a shingle, people brought their pets to you. At one time, there were many animals and very few veterinarians. People patronized practices because they needed pet medical service and the choices were few, often only one. Bestowed upon you, this degree of doctor, comes with great public respect. Your clients consider you an expert already, commanding automatic trust and respect. In fact, studies show your Dr. title caries some degree of automatic trust and general acceptance of your opinion, on any topic, regardless of your expertise. That means that before you open your mouth or pick up your scalpel and prove them otherwise they believe you to be an expert. Look around your office. Very few people in their circle of friends have graduate degrees, certificates and licenses. BUT you are not unique. Your clients have that same feeling about **all** veterinarians. The suffix "DVM" will NOT attract clients. Where once being the 'vet'nary' in town was all it took to collect new clients, today competition is keen. In many areas there are simply too many veterinarians. Looking at a three mile radius you can find 13 practices in Tucson, AZ, nine in Santa Barbara, CA and a whopping 23 in central Los Angeles. Simply having the title DVM isn't enough anymore.

Do clients really care if you're a good doctor? Yes, of course your clients care that you are proficient and qualified. Today, things move very fast, and people rely on mental shortcuts to deal with the amazing amount of information and decisions required to navigate through life. Robert Cialdini describes this process in detail in his book Influence: The psychology of persuasion. For most of us we know the general principle as "first impression". Our minds use specific conditioned responses and shortcuts to quickly evaluate and instruct us how to act or react to any situation. This phenomenon is alive and well in your practice, each and every client is influenced by its principles. Society has also changed. Americans are embracing "Trading Up", a behavioral shift affecting the purchasing habits of buyers. This action refers to the willingness to pay a premium for goods and services that are perceived as higher in quality, performance, value AND remain emotionally significant to the buyer. This is well described by Michael Silverstien and Neil Fiske in the book Trading Up, the New American Luxury. Why does a shipping clerk earning $25,000 annually treat herself to silk sleepwear

from Victoria's Secret, a young professional purchase a $100 specialty wine, and a middle class family purchase a $4000 Viking stainless steel stove when a serviceable generic range was included from the builder? The answer lies in the willingness of consumers to pay a premium for desired products and services that are emotionally important to them. Make no mistake; these same consumers have no tolerance for over-priced, under-valued items or services. Perception and emotion, not reality and logic, drive many purchase decisions.

Therefore, quite frankly, the consumer cares less about what you know than how you look, act, and how much you care about their pet. They already presume you are an expert, well educated, so they immediately lend a critical eye to the rest of the picture. Interestingly, we also live in a unique time when the quality of service (been to the local restaurant lately?), cleanliness, and professionalism are at an all time low. Forty years ago the masses would have marched in the streets demanding a return to chivalry, high moral and ethical standards, and respect. Today, we barely whimper our dissatisfaction with yet one more lousy, cold hamburger.

That presents an enormous opportunity for you to distinguish your practice in the market place. If you give your clients a fabulous experience in sight, sound, smell, and personal interaction, they not only will eat it up, they will pay a premium for it. Are you having trouble getting clients to accept the $1000 invoice? Is it too expensive for the BMW driving client or is it perceived as being overprice or of low value? Look around your practice. Close your eyes and listen, and smell. Is it something else overcoming their acceptance of your expert skills?

Your hospital, staff, and everything about you must within seconds reach inside and grab their hearts, never letting go. By the time you have a chance to work on their minds the opportunity is gone. When we evaluate our own colleagues, we look at their levels of training, diagnostic capabilities, and the overall quality of medicine they practice in determining if they are "good" practitioners. Clients do not have the ability to make these judgments, so they make their decisions on what they do understand, such as how you present yourself, how you act, and, most importantly, **how much you care**. Remember, you have only one chance to make a first impression! The first thing clients see is your sign. This is hugely important and often the ONLY thing the public will ever see. Is it bright, clean, simple, easy to read? Is your parking lot clean and well-striped with easy access for your elderly clients? As clients approach the front door, do you have planters with attractive, healthy plants, or are they dried up and dead? The new client may think, "Hmm, if they can't even keep PLANTS alive, I wonder what else dies here?" Are there spider webs dangling in the doorway? Are there children's chocolate-colored fingerprints all over the windows or are they spotless? They ask to themselves, "If you don't even care about keeping the bathroom clean, I wonder if you keep your surgery suite clean."

Most veterinarians are providing "task-driven service." Your client requests a service, vaccination, surgery, or presents the pet with a medical problem and requests you find a solution. You then provide a standard level of service and the client pays you. There, all done! You assume your client is satisfied. Are you sure? He or she got what was expected perhaps but often nothing more. Picture the client receiving an incredible experience, way beyond expectations? Not only will they pay you happily studies prove they will pay more.

Clients are usually not qualified to choose professionals with a critical eye. If you ask your neighbors for a recommendation for an attorney, they might say, "I just adore Janet Smith. She is so nice and I love her office and her staff. She always calls me right back and she also has a German Shepherd Dog just like mine. She is an excellent attorney. I highly recommend her!" Do any of these qualities really speak to her legal abilities? Not really, but she DOES have an enthusiastic referring client. Your clients will judge you on the things they CAN use to judge you, things that they understand and those that they are absolutely qualified to render an opinion on: cleanliness, odor, attitudes, promptness, creature comforts (coffee, e-mail access, free phone, refreshments, pleasing sounds, etc.) They are not equipped to judge your surgical ability, but they CAN look at the incision and see that it is neat, straight, and not blood soaked. They will notice instantly if Fluffy smells like urine or feces upon release, or if she has just been brushed, nails clipped, and a little bow in her collar.

Like it or not, veterinarians are in the service and retail business. You must adhere to the rules of the game. You must create an environment unlike all others in your industry, and emulate the star performers found in different industries your clients interact with daily. Then, after that important screening function is complied with, you really need to buckle down and provide excellence in medical care. This isn't an about face on the preceding information. Providing excellence in medical and surgical services is critical to your continued success as a veterinary service provider. When you practice good medicine, you increase your case success rates. When that occurs, patients improve and clients are satisfied. The details about understanding that you are first and foremost in a customer service business ensures that you set an environment and hospital culture that embraces that clients trust, and allows the client to "see" just how good a doctor you really are. With a strong positive emotional interaction between practice and client, communication pathways stay open. Instructions get relayed, home therapy and treatments are administered correctly, and pets win.

It's only after you have ensured that you really understand AND buy into the fact that your goal is <u>not</u> to be a veterinary hospital with great customer service, your goal is to be a great customer service organization that happens to be in a veterinary business. In speaking to hundreds of veterinarians over the years, a significant number express dissatisfaction and frustration with the public not fully realizing how hard they work at being a good doctor and often how much better the quality of medicine is at their practice than their formidable competitor's practice nearby. One common theme I've witnessed in these situations is that many of these practices have significant deficiencies in the customer service area which undermines the perception of the medical quality they are delivering. When customer service suffers there is no way to create a "wow" effect or a remarkable consumer experience. Interestingly fate often brings me the opportunity to visit other local competing practices, and despite some having arguable deficiencies in medical standards, sometimes these practices excel in customer service and achieve a ranking of good value in the minds of the clients. Clearly the subject practice has higher medical standards, which if consumers of veterinary services judged solely on medical quality, would be the clear choice but they can't adequately judge medical quality so they focus on what they are qualified to assess: customer service, overall value, and the consumer experience. It's not fair, but its reality.

Creating and Delivering Performance Appraisals

SAGE

An approachable team of specialists providing advanced, collaborative, and compassionate care.

John McClain, SHRM-SCP
Human Resources Director

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What is a performance appraisal?

- Examines and evaluates an employees' work behavior by comparing it to preset standards (**performance expectations**)
- **Documents** the results of the comparison
- Uses the results to provide feedback to the employee, highlighting successes as well as where improvements are needed, and why **(the impact!)**

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What is a performance appraisal?

- Job-related and standardized for all employees in like positons
- Free of bias, both in ratings used, and legally protected classes (e.g. age, disability, national origin, race, gender, religion)
- Written and conducted by someone who has adequate knowledge of the employees' performance

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Why are performance appraisals important?

- Fosters positive communication between managers and employees
- Reinforces good performance and encourages improved performance
- Allows employees to identify what skills may be lacking, and that need to be acquired or improved upon
- Holds employees accountable for their job performance
- Provides the opportunity to explain employer's goals and the ways to participate in their achievement

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Beyond evaluating performance, appraisals are also used to…

- Highlight strengths for advancement or the assumption of new and/or increased responsibilities (**promotion!**)
- Identify 'gaps' in performance (**expected vs. actual**)
  - Develop 'performance improvement' plan to close gaps
  - Recognize and agree upon specific training and development opportunities (one-time and ongoing)
- Provide feedback **to** employees, and hear reactions and feedback **from** employees (**collaborative**)
- Make staffing decisions (**alignment w/ business**)
- Make pay decisions (**rewards and recognition**)

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Types of appraisals

Behavior-based

- List of criteria (dimensions) that employees perform against
- Criteria is typically the same for all employees in the same job role, yet will differ by job roles (clinical vs. administrative)
- A favored method as the evaluation is done on the basis of individual employee performance, without comparisons to peers
- Involves goal setting up front so job expectations are clear and understood; **promotes** ongoing communication
- May be exclusively narrative or include a rating scale
  - Clearly defined definitions of rating scale
  - More and more employers are evaluating the use of ratings

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Types of appraisals

360-Degree Appraisal

Allows others (manager, peers, and direct reports) to contribute to the appraisal, detailing their specific experiences

- Feedback from peers and direct reports can be reviewed by the manager and made part of the appraisal. *Caution--consider the source, biased raters!*
- Can be "eye-opening" to see what others see
- May be narrative or include rating scale
- Can be challenging to maintain anonymity (enough raters)
- Tendency to focus too much on isolated pieces –*Why did he say that?!?*

Generally used as a developmental tool, and not as an appraisal of performance, particularly if tied to compensation

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Types of appraisals

A Hybrid

- Manager considers 2-3 thoughts and uses that as a 'discussion guide' for the appraisal conversation, such as:
  - "I would always want this person on my team"
  - "This person is at risk for low performance"
  - "This person is a long term fit and poised for growth within our organization"
- Manager provides a written summary of performance strengths, opportunities for growth and improvement
- **Requires** ongoing and effective performance feedback and direction, as 'performance drift' can occur more easily and without early warning

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Components of an appraisal

Whichever appraisal type is used, keep these elements in mind:

- **Employee Demographics;** name, title, performance period, signature of both manager and employee, and the date
  - Clarify that the signature denotes receipt and not agreement
- **What's being evaluated;** goals, performance dimensions, critical deliverables (e.g. projects)

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Components of an appraisal…(*continued*)

- **Rating scale and definitions;** if one is used
- **Performance summary;** both strengths and positive attributes, as well as opportunities for development and areas of needed improvement

Be cognizant that its also a document that can be used in a legal proceedings so consistency and appropriateness matter

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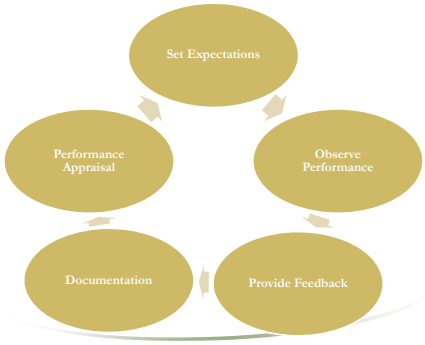
Performance Appraisal Cycle... Going from A to B…



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Why are setting expectations important?

Its one of the most difficult jobs for a manager.

With clear job expectations, employees can:

- Understand ***what*** is important; what they should be doing
- Understand ***why*** they are doing it
- Know ***how*** they are doing and when to ask for support
- Recognize where performance ***improvement can occur***

Without them, employees likely:

- Waste time with unnecessary work
- Waste effort due to a lack of priorities or direction
- Endure increased stress/anxiety because of uncertainty

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Why are setting expectations important?

Many sub-par performance issues revolve around this single question…

"Why isn't my employee doing what needs to do be done?"

And the answer from the employee is often…

"But, I thought I was doing a good job."

Poorly defined expectations creates frustrations for both managers and employees

- Managers, because their employees are not doing what needs to be done
- Employees, because they think they are doing what needs to be done, yet the manager is still not satisfied

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Employees Are Not Mind Readers

Why are clear expectations so important?

If managers **can effectively communicate** their expectations, they can **more reasonably expect** their employees to meet them.

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Setting Expectations…Make them S.M.A.R.T.

S-pecific
- Be specific; focus on 5 Ws (What, Why, Who, Where, Which)

M-easurable
- How will you know its been accomplished? Quantify or suggest an indicator of progress

A-chievable (or Attainable)
- How can it be achieved and is it realistic, based on constraints?

R-elevant (or Realistic, Reasonable)
- Is it relevant to the job, the individual, and the organization?

T-ime-bound
- Specify when the result will be achieved; establishes a sense of urgency

Observing performance is a critical role of a manager…sharing what you observe is equally important.

Consistent & Balanced

- Positive performance
- Areas of opportunity
- Performance improvement

1st Hand v. 2nd Hand

- See it…Acknowledge it!
- Hear it…Validate (investigate) it, then… Acknowledge it!

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Feedback--recognize good performance and identify where improvement is needed…

- Provide it as close to the observed behavior as possible, yet consider the situation (e.g. temperaments, privacy, patient care)
- Provide it on a regular basis, for both positive and constructive observations; helps in maintaining good performance and improving poor performance
- Be prepared, know what you are going to say, how you are going to say it, and your intended message/outcome
- Be specific and have examples
- Document; both positive and constructive feedback

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Documentation…its critical!

- Tracking both positive performance and the need for improvement helps during the appraisal process (writing and delivering), as well as if progressive discipline becomes necessary
- Include the **issue**, the **conversation**, and the **outcome**
- Can take the form of an "incident file", noting a conversation, or more formally as a "performance memo"
- While less formal, incident files should be equally specific, stick to the facts, and avoid opinions
- In the event of a termination, the documentation should support the decision, and any adverse post-termination action that may arise (e.g. EDD, DFEH, EEOC)

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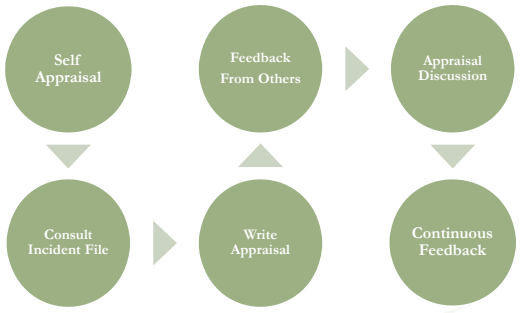
Appraisal Process—Ongoing and Collaborative



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For the employee, a self-appraisal should focus on…

- **Sharing their successes and accomplishments**
- **Sharing what they learned**
  - New skills and how they've applied them; challenges they've faced and how they overcame them, or the steps they took or will take in the year ahead to address them
- **Being honest and candid**
  - Avoid embellishing accomplishments, or dismissing their opportunities for growth or improvement
- **Taking time to do it well**
- **Ease of completion**

Often dreaded by employees as few enjoy rating themselves, particularly if they perceive that their pay is on the line.

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For the manager, the self-appraisal provides additional insight on…

- What employees are proud of and where they think they need to improve
- Discrepancies that may exist between their view and that of their employees' view of their own performance

Consider these question when reviewing employees' self appraisals:

- Has the employee captured all of their successes, and are they measurable and relevant?
- Have you observed learning moments?
- Have you observed any challenges?

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Why consult the Incident File?

- Helps managers to reflect back on the entire year of **performance**, **successes**, **challenges**, and **discussions**
- Reduces the "**halo**", "**horns**", and "**same-as-me**" effect

Halo – When only recent, **good** performance is the focus

Horns – When only recent, **poor** performance is the focus

Same-as-me – When only behaviors that are consistent with the manager's are the focus

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Writing the appraisal

- Incorporate the self-appraisal, incident file and feedback from others
- Content should not be a surprise!
  - If it is, likely miscommunicated (or didn't at all)
- Consider goals and objectives for the time frame of the appraisal; review the employees' job duties and compare to the job description
- Review attendance history
- Stay focused on the message
- If ratings are used (e.g. 5-point scale), they should align with the supporting comments

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Feedback from others

Why do we want feedback from others?

- Soliciting feedback from others is important, especially if employees predominately works on a shift or in an area their manager does not oversee directly
- Others (e.g. doctors) may observe strengths and development opportunities that the manager may not always observe first-hand

Ways to collect it

- Provide a draft of the appraisal for review and comment
- Ask specific questions related to the employees' performance
  - In-person or send the questions to be completed
  - Give a timeline for when feedback should be provided

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Incorporating feedback from others

When incorporating feedback, ask yourself:

- Is this a **trend** in performance or a one-off incident?
- Have **you observed** similar performance by the employee?
- Are there **examples to support** the feedback you've received?
- Is the feedback **significantly different** from what's already been written?

25

The performance appraisal discussion…

Employees should be encouraged to ask questions and propose ways they can continue to grow and develop, with feedback that is actionable; reinforcing positive performance, and deterring sub-par performance and behaviors.

Appraisal conversations should not be one-sided, yet its the time to provide **their assessment**, encompassing performance over an entire period. Managers set the stage for an effective discussion by:

1. Being prepared
2. Sending the document ahead of time
3. Making it a collaborative conversation
4. Appreciating the successes
5. Acknowledging the gaps
6. Looking ahead to the future, and
7. Saying Thank You!

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Be prepared

Writing an appraisal and giving one, are different. It takes time to think about what you want to say.

- Prepare, even practice, difficult statements ahead of time
- Describe behaviors and actions, not total impressions
- Make specific suggestions, not general ones

Employees usually hear and remember the negative statements, even if their overall feedback is excellent, so thoughtful preparation for the conversation will help avoid dissatisfaction.

27

Send the review ahead of time

- Performance appraisal discussions can be packed with emotion
- One way to diffuse the emotion is to provide a "sneak peak"
  - Send 24-48 hours in advance; this allows a chance to process and think about what's been written, and ultimately, a more meaningful dialogue

28

Make it a collaborative discussion

- Demonstrate respect for employees, without glossing over negative feedback or being vague
- Stop frequently and ask open-ended questions
- Pay attention to everyone's body language
- Be considerate of employees':
  - Feelings
  - Readiness and tolerance to hear
  - Level of trust and self-esteem
- Guide the conversation to be direct, honest and constructive

29

Appreciate the successes

The appraisal is the time to **acknowledge** employees' effort and successes.

Everyone appreciates some level of **recognition** on their effort and contributions, and how it fits into the organization's success.

30

Acknowledge any gaps

When there are disagreements between managers and employees, it's important to acknowledge and discuss them candidly and directly.

- The goal is not to prove managers are right and employee are wrong, but to accurately assess their performance through examples and explanations
- Ask questions of employees to understand their thinking
- Be open in how manager arrived at their thinking on their employees' performance – *provide examples*
- May not always agree, but at least both will better understand each others' thinking
- Managers should be willing to make adjustments if they uncover data that changes your view

31

Look ahead to future

- Continually carve out some time to talk about goals and development plans
- Focus on providing guidance and perspective of managers' expectations and where the organization is headed
- Where do the employees want to go from a career progression perspective, or what other skills they may want to pick-up to do their job effectively?
- Use ongoing 1:1 meetings to finalize the details (establishing goals and objectives for the next year)

32

Finally…say "thank you"

If it wasn't for your people – where would you be?

…Showing gratitude goes a long way!

33

Effective feedback…steps away!

What
Define the Issue

So What
Understand the need for change

Now What
Do something different

34

Effective feedback…done in a caring manner, helping to improve employees' knowledge, skills and abilities

- Discuss behavior observed compared to the desired expectation; be objective, have concrete examples, avoid vague statements
- Consider the impact, seriousness and frequency *(controlled drugs vs. kennel cleaning)*
- Identify implications to the role, to others, to the organization, and how it impacts job performance (*e.g. patient care, reliability)*
- Assess what should be the desired outcome, offering suggestions as to "how"
- Observe and review expectations going forward
- Document consistently

35

An effective feedback conversation…crucial to affect change in behavior

Be Positive & Focus on Improvement (not personalities)

- Being harsh, critical, or offensive is not productive

Be Timely (in-the-moment)

- Address the issue proximate to the observed behavior, avoiding the "element of surprise". The sooner it's done, the more the feedback will be expected and relatable. ***However…***

…if the situation is highly emotional, wait until things calm before engaging feedback. Don't risk getting worked up and saying something to regret later

36

An effective feedback conversation…crucial to affect a change in behavior…*continued*

Make It Regular – if something needs to be said, say it!

- Employees will know where they stand and there will be fewer surprises. With frequent, informal feedback, nothing said during a formal feedback session (e.g. appraisal) is unexpected, surprising or particularly difficult

Prepare Comments – don't read it, yet be clear

- Know what needs to be said; helps to stay on track and be focused, as well as prepares for any objections

Be Specific -- stick to the facts

- Describe what needs to be improved upon, which leaves less room for ambiguity

37

An effective feedback conversation…crucial to affect a change in behavior…*continued*

Praise in Public, Criticize in Private

- Safe, quiet place to talk; public recognition appreciated, public scrutiny is not

Use "I" Statements

- Give feedback from your perspective; avoid labeling employees
  - *"I noticed this…"* vs. *"You did this…"*

Watch Body Language

- Aware of physical demeanor (non-verbal gestures)

Limit Focus

- Limit the number of issues (1-2); avoids employees feeling attacked or demoralized. Acknowledge issues as new vs. recurring

Include Positives, Careful…heavy on the positives can become – "*Gee, I'm doing really well"* versus *"I'm good at 'this' but need to improve on 'that'*…yet be clear on the intended message

38

Reactions to *constructive* feedback…

- Resistance
- Defensiveness
- Anger
- Playing the victim
- Silence
- Crying

- Keep the outcome and working relationship in mind, even if employees are defensive or lash out
- Can't prepare for every possible thwarting mechanism, yet managers can control their own reactions

39

If an employee reacts negatively…

- ✓ **Resistance** -- *"I don't see a problem with my performance"*
  - Restate the situation and its result
  - Discuss additional consequences (e.g. progressive discipline)
- ✓ **Defensiveness** -- *"What about Jim? He's made many mistakes and does not do as much work as I do"*
  - Remind employee that the discussion is about his/her performance—not anyone else's
  - Reinforce that all employees receive feedback about their performance

40

If an employee reacts negatively…

- ✓ **Anger** -- *"I can't believe you're throwing that in my face."*
  - Remain calm and composed
  - Acknowledge the frustration without agreeing with or condoning it
  - Restate the issue and continue the discussion, or suggest another time to meet once the employee has had a chance to cool down
- ✓ **Playing the Victim** -- *"So, I never do anything right."*
  - Explore the extreme position. *"Are you saying you don't see value in the work you do?"*
  - Reinforce other positive performance, without losing sight of the need for development

41

If an employee reacts negatively…

- ✓ **Silence**
  - Pause and allow employee to digest the information provided
  - Eventually, restate the issue (e.g., the behavior and its impact), then ask for thoughts
  - If employee is being defiant by being silent, discuss consequences of non-involvement, and by not improving performance
- ✓ **Crying**
  - Manager's own silence may be the best first-reaction
  - Acknowledge that the discussion is difficult
  - Determine whether to meet at another time or restating the issues and continuing the discussion

42

The Manager's mindset…

Effective leaders will consistently exhibit:

- Self-awareness
- Vulnerability
- Helpfulness
- Clarity
- Involvement
- Accountability

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Effective leaders also solicit feedback about themselves

- Focus on the content, not the person
  - Are open and assume positive intent
  - Listen calmly and attentively
  - Are not defensive when hearing the message
- Clarify the feedback – ask questions, stay neutral and objective
- Acknowledge the feedback

44

When should performance appraisals be done?

Two cycles, based on the employer's needs and objectives

Anniversary Date – of the employee's hire date

- Spreads the process over 12-months, yet managers could be doing appraisals continuously (*or at least seem like it*)
- More challenging to tie compensation to performance, and ensure equal access to the budget for raises

Common Date – same time of year for all employees

- All at one time, yet depending on number of direct reports, can bog down managers' time *(short term pain)*
- Easier to align compensation with performance and allocate budget uniformly, and with full access

Some employers choose to do appraisals more frequently, especially for new employees; 30-60-90-180 days

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Recruiting Doctors: The Current Market and What Impact Does It Have on Negotiation and Retention

Julie D. Smith, DVM, DACVS, CCRT, MBA

Managing Partner/Medical Director

Audience Poll

- How many of you are actively looking for another doctor?
- Of those actively looking (or recently looking), what have been some of your struggles in finding the right person?
- Of those that have successfully hired recently, what do you think made that hire successful? Or was it successful?

Veterinary Market

- The "veterinary market" – this refers to actually 3 different markets that are interrelated and yet can appear to move separately in regards to how supply and demand can influence the outcomes of each market.
    - The market for veterinary students – currently there are more individuals that want to enter veterinary school than there are spaces available. We have for-profit veterinary schools opening in relation to this demand. This market does not appear to be influenced by actually how many veterinarians are needed – but rather the desire of the individuals to enter the profession based on intangible rewards.
        - 6700 students apply for 4200 seats (3200 in U.S.)
            - 87% women
            - 47% non-residents – so will be spending more on their education
            - Decision made at average age less than 10yrs
                - Vet school applicants have low concern over debt (increases over time in school)
                - Debt willing to pay: salaries in 2014 = 4:1!
                    - 20% taking on more debt than they were willing to at beginning of school
    - Debt to Income Ratios
        - Debt to Income Ratio overall 2:1
            - Highest in all professions (0.9 in medical doctors)
            - California
                - Average Debt of Western = $263K
                - Average Debt of UCDavis = $123K
            - 43% of all California vets surveyed had debt
                - Half of those with > $100K
            - Females higher ratio compared to males
        - 55% of California vets surveyed expect higher salaries
            - 84% of recent grads surveyed!
            - Impact on the practice profitability or ability to hire
    - Impact of Debt and the Future

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        - More seats at higher costs than individuals willing to take on debt
            - Drive down demand over time
            - Interest rates in the future will make debt less attractive
        - See individuals switch sectors within the market – higher salaries available in industry for veterinarians, low salaries expected in large animal medicine. Don't see too many individuals switch markets once they are in it. May change as debt becomes a larger factor in our industry.
        - Information age stimulate greater exploration of decision
            - Net Present Value (NPV) of a DVM degree – do you get back what you spent to get the degree?
                - Calculated against what an individual would make with a bachelor's alone
                - Since women have such lower salaries with a bachelors, they have positive NPV when compared to men
            - When the tangible might start to outweigh the intangible overtime as individuals do more economic modeling prior to making decisions about entering profession
- The market for veterinarians – demand should be based on the number of animals that need care. Because it is heavily influenced by how many individuals actually seek medical care for the animals and the willingness to spend money on that care, the supply and demand curve is based more on a "retail", elastic model vs. "medical", inelastic model.
    - Demand goes down as price goes up whether we like it or not! This makes the question about whether there are too many or too little vets a complex issue.
    - Excess Capacity = unused portion of total capacity
        - Takes into consideration price
            - Demand for services but not at current price
        - Compare need for veterinarians (FTE) for number of pets in households
            - Only 75% visit the vet at least yearly
                - Need 1600 FTE in SF Bay Area
            - If there was no influence of price and 100% visited vets
                - Need over 2000 FTE in the SF Bay Area
    - Increasing Demand for Veterinarians – rather than talk about whether we have "too many vets", we would all benefit if we focus on increasing demand for our services
        - Effect of GDP and increasing household income
            - Disposable income effects demand for vet service
        - Veterinary Service Pricing
            - 5.3% increase on average (2-3% in other markets)
                - Visits decreased with increases in prices that outstripped the inflation rate
            - Increase demand with client compliance and value
        - Pet Insurance has potential for HUGE impact – so why aren't we the biggest proponents of this to our clients?????

Recruiting for New Doctors

- The veterinarian market is your supply for your recruiting – so understanding the influences on this market and the individuals making up this market allows you to make decisions in how to recruit and how to negotiate.
    - Very low unemployment rates currently with 1.43 applicants for each job
    - Peak times for searchers for new jobs in the spring and summer
- Considerations
    - What are you looking for?
        - What are the practice needs?
            - How many hours and what hours?
            - New doctor or experienced?
            - New skill set?
            - Possible ownership in future desirable? 66% of associates surveyed did not want ownership in the future
        - Traits most often sought
            - Work ethic
            - Communication skills
            - Enthusiasm
        - How do you assess?
            - Resume review
                - Is it presentable? Attention to spelling and formatting?
                - Past experience at veterinary and other industry jobs
                - Training or CE courses
                - References listed
            - Initial phone interview
                - Have set questions prepared
                    - What do they enjoy about veterinary medicine?
                    - What are they looking for in a new position?
                    - Walk through the resume with them
                - Describe the opportunity
                    - Be honest and upfront regarding expectations
            - In-person interview
                - Have other doctors and staff members involved
                - Watch group interactions
                - Shadow client visits
                - More in-depth questions
                    - What are some strategies they use in client communication? Describe situations that went well and those that did not
                    - How do they involve staff as part of their team? Describe relationships that worked well and those that did not
                    - Describe previous work flow and what worked well for them and what did not
            - References
                - Have set questions prepared
                    - Clinical skill set
                        - Problem solving
                        - Procedures

        - Work flow efficiency
    - Interpersonal skill set
        - Interactions with clients, staff, and peers
        - Ability to receive and address feedback
- What are candidates looking for?
    - What is most important (in order):
        - Ethics of the owner/practice
        - Clinical quality of the practice
        - Work/life balance
        - Compensation
    - Why are they looking?
        - Better compensation
        - Better work environment
        - Not happy with management
        - Want different hours
- Effect of gender and age groups regarding hiring, retention and negotiations
    - Who are "they"?
        - New Graduate Data
            - 47.6% went for advanced degrees/internships in 2014
                - Collect more debt prior to coming into full time employment
                - 56.7% of Western students applied, 31.6% of UC Davis students applied
            - 66.5% entering companion animal
            - 65% of those passing NAVLE went to schools in the US – more individuals going to schools out of country that can also lead to increasing debt and more requirements to obtain licensure.
        - Gender
            - 87% of students entering veterinary medicine are women
            - Highest satisfaction for women is seen when they are working 42.4 hours/week (men 48.8)
            - Women tend not to negotiate – leading cause of wage gap across all industries
                - 2013 survey: male 95K mean, female 85K mean
        - The Millennials – characteristics have some potential influence on how they negotiate and what they are looking for in a position (although everyone is an individual!). Born between 1982 and 2004, largest living generation.
            - Technologically savvy
            - Tolerant and support equality
            - Globally aware
            - Socially responsible – charitable at work and values are important.
                - Will skip protocols if don't know why or how it will benefit the patient
                - Want transparency in the work place
            - Entitled – craving praise and expects to ascend the ladder quickly
            - Pragmatically ideal – wants to make the world a better place and understands that it takes working within and out of current institutions

- Great expectations and want attention ("everyone's a winner…..")
    - Constant engagement and feedback at work important
    - Wants to grow and learn
- Work to live
    - Wants to work together in a good environment
    - Collaborative problem solving – including during the negotiations
- Committed to health and fitness
- Short of cash – grew up in the recession, has higher debt, and delaying home purchase (so paying more in taxes).

Starting the Negotiation

- Preparing for negotiation
    - Millennials and women less likely to negotiate – but doesn't mean that they are happy with compensation
        - Less likely to ask for a raise, so could leave without the ask
        - Want to collaborate in negotiation and look for a win-win
    - Most are taught to "never accept the first offer" – consider the request and if reasonable or not
- What should you consider in developing your compensation strategy?
    - Compensation Models
        - Straight salary
            - Easy to budget for you and associate
            - Finances simplified – no additional calculations
            - Need to determine considerations for merit raise
            - Minimize competition between associates
        - Straight production
            - Can set based on desired profitability
            - Difficult for associate to budget
            - Promotes and rewards developing caseload
            - Can cause competition between associates
        - "ProSal" model
            - Blend of both with a base salary and production bonus
            - Allows for budgeting for associate and incentive for caseload
            - Most difficult to calculate and compare
                - How often to reward bonus?
                - Is there negative accrual?
                - What is included in production?
    - Market Rate
        - The "Usual" price in the market
            - What are mean and median incomes for veterinarians?
                - 2013 survey data
                - 90K median salary of associates surveyed in CA
                - 96K mean/87K median of associates surveyed in the Western Region
        - Associate income tends to plateau after 5 years
            - Income curve for women flatter mid-career
    - Other Offerings
        - Improve offer outside of compensation – may be more attractive than the compensation itself (look at Millennial characteristics)

        - Benefits – health, disability, life, retirement
            - Becoming more consistently offered
        - Liability and licensure
        - Vacation/PTO – make sure and have a cap on accrual or ends up being a large expense to the practice if someone leaves
        - CE and association dues – easy to budget for and encourages them to participate in the profession and learn and grow. A good return for the practice and helps with engagement
        - Signing bonus/moving expenses/first month rent – since many might be short of cash, allows to give something they might want and not a commitment to an ongoing amount
- Retention – after you make a successful hire, you are not complete!
    - More important than recruiting
        - Less costly to retain and less emotion involved regarding turnover within the practice
    - Know what is important to associates
        - Plan for meetings to discuss career path
        - Give feedback and ask for it
        - Listen if needs have changed
        - Keep them engaged in the practice

Conclusion

- Understanding the different markets in veterinary medicine important to understanding who you are recruiting
- Outside factors influence demand for veterinarians such as the economy and access to pet insurance
- Assess the candidates for the soft skills and make clear expectations for the position
- It is not all about compensation…..but it is

Management Roundtable: Your Questions Answered by the SAGE Management

SAGE Centers for Veterinary Specialty and Emergency Care

Objective: This will be a panel discussion for the participants to bring their largest challenges and have a varied group of managers give advice and guidance. Topics will be driven by the attendees, but will most likely cover:

- Value provision of service
    - Client satisfaction and the Net Promoter Score
    - How to discuss value vs cost
- Staff development
    - Skill based training
    - Leadership training
    - Communication training
- Talent management
    - How to motivate your star performers
    - Coaching to performance
    - Rewarding talent beyond the financial
- Finances
    - Revenue – are you driving growth and how do you know?
    - Are you profitable? Do you know where profit comes from?
    - Staff costs and managing your highest variable cost
- Marketing
    - SEO management
    - Social media – where do you need to be?
    - How to position yourself in the market – are you doing it unintentionally?

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Track 5

Bandaging 101

SAGE

An approachable team of specialists providing advanced, collaborative, and compassionate care.

William Scherrer
March 2016

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•Consider the Bandage: a walking billboard for your practice.

- Quiz 1:
- Which bandage says, "We love your pet like he/she is our own"?

- Quiz 2:
- Which bandage do you want an owner to share with friends on Facebook, Instagram, Twitter, Whatever-intheworld-thekidsaredoingtoday.com?

SAGE

Components

- Contact layer: therapeutic
  - If wound: adherent or non adherent
  - no wound: not needed
- Intermediate layer: bulk
  - roll cotton or cast padding (tight)
  - Follow with stretch gauze (not)
- Outer layer: protective
  - Vet wrap or Elasticon (not)

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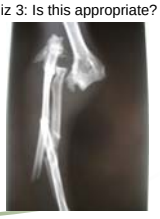
Decision Making

- Support fractures and soft tissues
- Protect tissue from trauma or desiccation
- Protect owner/environment from wound (MRSP)

SAGE

When to Bandage

- As soon as vitals are stable
- When it is safe for sedation
- When wounds are clean and defined
- WHEN APPROPRIATE
- Quiz 3: Is this appropriate?



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Type of Bandage

- First, think: Do I need a bandage?
- Next: What type of support is needed?
- Last, Is your patient a devil-bat-dog?

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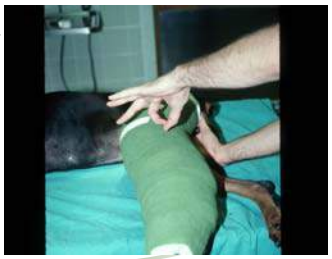
Types of Extremity Bandages

- Robert Jones
- Modified Robert Jones or Soft Padded Bandage
- Splint
  - Pre-made
  - aluminum rod
  - fiberglass casting material, Thermoplastic
  - Custom orthotics
- Cast: fiberglass

SAGE

True Robert Jones

- Big!!
- Roll cotton
- Long cylinder
- Ok fx distal to elbow or stifle (although, often not ideal.)



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Modified Robert Jones

- Most common
- Use cast padding for intermediate layer
- Beware
  - XS compression
  - Prominences
  - Slippage
- Quiz 4:
- How did this dog injure himself?
  - Perhaps, sitting in the street?

SAGE

Splints

- Pre fab
  - Mason Meta (spoon)
  - Quick splints
    - Limited sizes
  - Tongue depressor

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The most versatile splints

- Thermoplastic
- Fiberglass
  - Vet cast

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Custom Orthotic splints

- Therapaw
- Orthopets
- Local rehab specialist

Bootie Splint

Hock Splint

- Dogleggs
  - Useful for hygromas
  - Medial shoulder instability?

SAGE

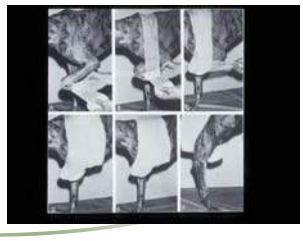
Special Extremity Bandages

- Ehmer: internally rotates and abducts femur
- "Ehmer for the Femur"

- Velpeau: non weight bearing on foreleg
- Spica: stabilize shoulder region
- Stent or Area bandage ("Tie Over")

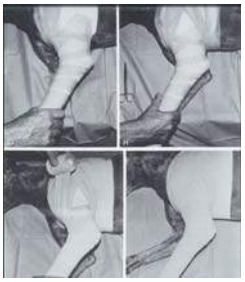
Velpeau

- Stabilize shoulder
- Non weight bearing bandage
- Watch for ischemia carpus
- Rarely used
- Indications?



Spica

- Weight bearing
- Shoulder immobilization
- Open humeral fractures
- Rarely used



Full Casts

- Not common
- Change q 2 weeks
- Cast cutters
- Clam Shell (bivalve)
  - Commonly used
  - Can re-use
  - Beware limb changes
    - Swelling at first
    - Atrophy later

SAGE

Rules: Extremity Bandage

- Must extend to pads
- Uniform tightness
- Address bony prominence
- Check, clean interdigital spaces when changed
- Fx: stabilize joint above and below
- Quiz 5: What happens if the bandage does not extend to the pads?

SAGE

Open Fractures

- Humerus
  - Stent bandage
  - Spica (+/-)
- Femur
  - Stent bandage

- These bandages are meant to be used for temporary protection; ultimate treatment would be external fixator or ORIF.

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Trunk

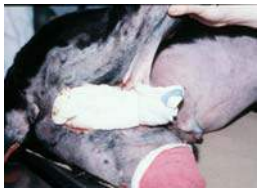
- Stent or Tie-Over bandage
- Belly bandage
- Harness-like bandage




SAGE

Stent or Tie-Over Bandage

- Can put ANYWHERE
- Loops in skin
  - Big bites
  - 2-0 monofilament
- Place lap pads
- Tie over with umbilical tape or large suture

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Wound Care

- Proper sedation and analgesics
- KY to wounds before clipping
- Clip with clean blade
- Remove debris: Scrub
- Excise dead tissue
- Cover with contact layer





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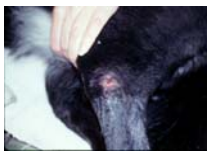
Chronic non-healing wound

- Don't forget to culture
- Era of drug resistant bacteria



Contact layer for wounds

- Wet to dry (adherent)
  - road dirt
  - contaminated wound with purulent debris

- Non-adherent
  - incision
  - clean wound
  - healthy granulation tissue

- Manuka honey, sugar, RediHeal, etc.

Examples of Non Adherent

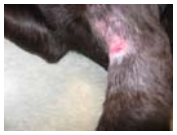
- Telfa
- Adaptic
  - Is petroleum impregnated gauze
- Release
  - More absorbent

Non adherent contact layer

- Semi-occlusive
  - Lets air in and has a moist environment to allow better epithelialization
- Only for clean wounds
- Not commonly used

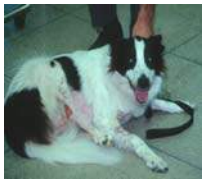
Complications

- Inadequate wound management and assessment
- Patient self trauma
- Wetness
- Motion inside of bandages
- Pressure related changes
- Quiz 6: Common or Uncommon?



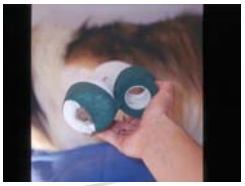
Bandage morbidity

- Result of
  - Crossing joints or bony prominences
  - Over padding OVER prominences
- What we see
  - Skin loss
  - Loss of underlying structures
  - Loss of limb function
  - Loss of limb



Swollen Toes

- Bandage must extend to pads
- Too tight
  - may even be in one small area
- Underlying surgery or trauma
- Treat by replacing
  - cutting bottom NOT OK
- Client education:
  - Toe swelling
  - Bandage moisture/licking
  - Tattered edges



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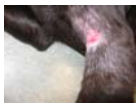
Pressure Sores

- High level of suspicion to prevent
- Pad around areas
  - calcaneus
  - lateral metatarsal (carpal) phalangeal joint
  - lateral and medial malleolus
- Slipping bandages
  - cranial hock



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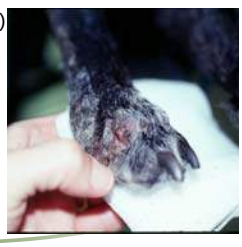
Interdigital Inflammation

- Examine with each change
- Clean with Betadine or Nolvasan solution
- Dry
- Consider powder (Neopredef)
- Pad with thin layer cotton



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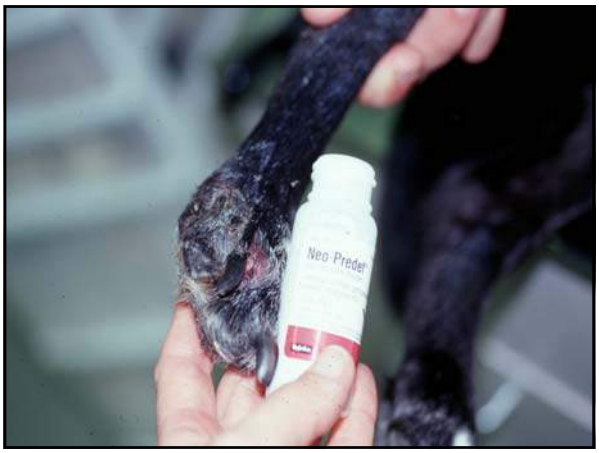

Slippage

- Apply with enough intermediate layer
- Make the limb look like a cylinder
- Uniform tightness
- Extend fore and rear leg allows higher application
- PROPER SEDATION
- Stirrups
- Quiz 7: What do you do if it slips?
  - A)try to pull it up like loose pants?
  - B)pull toes downward to fit better?
  - C)change bandage?

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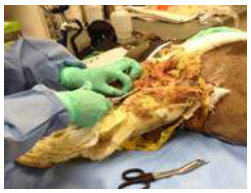
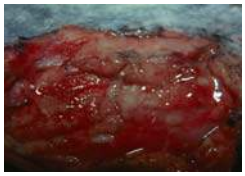
Wound Maceration

- Inappropriate contact layer
- Treat by cleaning and more frequent bandage changes (Culture?)





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Keeping bandage dry

- Plastic bag
- IV bag
- Medipaw Boot
  - Our favorite

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Chewing

- Always change bandage as there may be pressure sore or wound problems underneath
- Once treated, then add restraining device

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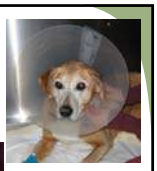
To Prevent chewing



- Elizabethan Collar
  - Quiz 8:
    - Who was her father?
      - Henry VIII
      - Henry Rollins
      - Henry Fonda
    - Who was her mother?
      - Anne Boleyn
      - Anne Hathaway
      - Angelina Jolie

*Bite Not collars usually not effective!

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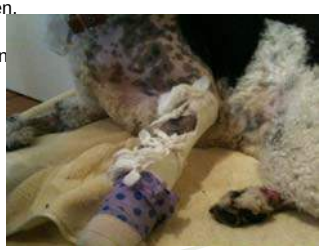
Half cast a la mode

- Quiz 9: Do I need to come in and have this replaced?
  - A)No, toes not swollen.
  - B)That's the way the bandage looked when she left the hospital.
  - C)Yes, ASAP.



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Spica du jour

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Ischemia

- Most dreaded complication
- Proximal construction when owners add
- Snug with compromised blood supply
- Muscle only: contracture
- All components: necrotic tissue, must amputate


Modified Robert Jones

- The most common
- Easy to add a splint to
- Step wise instruction

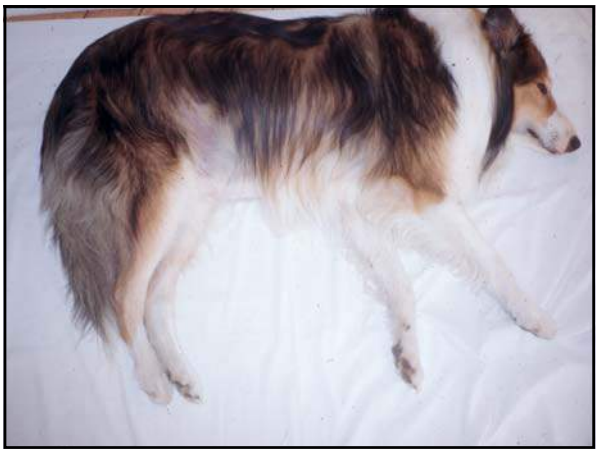
- This is what we will do in lab
- Not this---------->

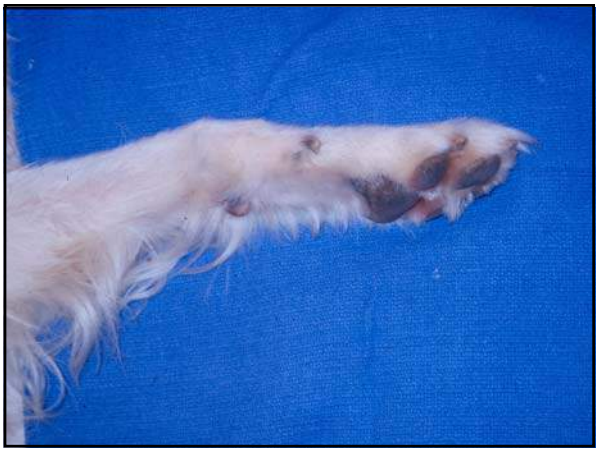
Bandage from toe up!

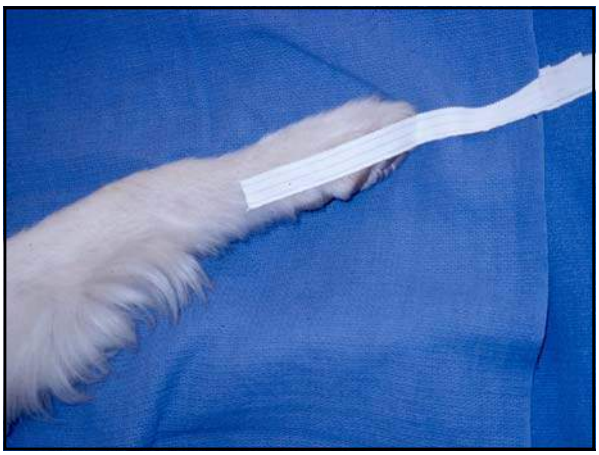
- If you do this:

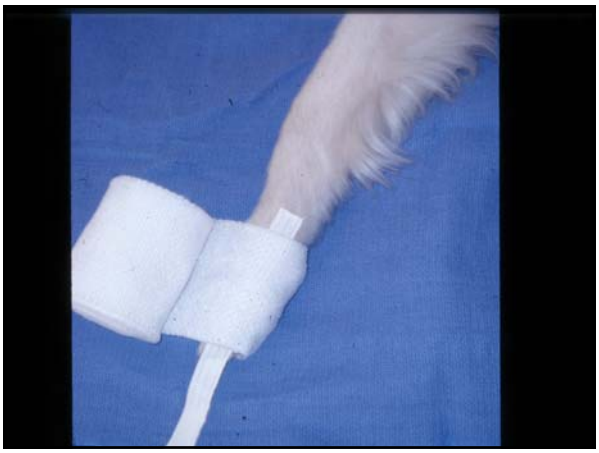
- You will get this:

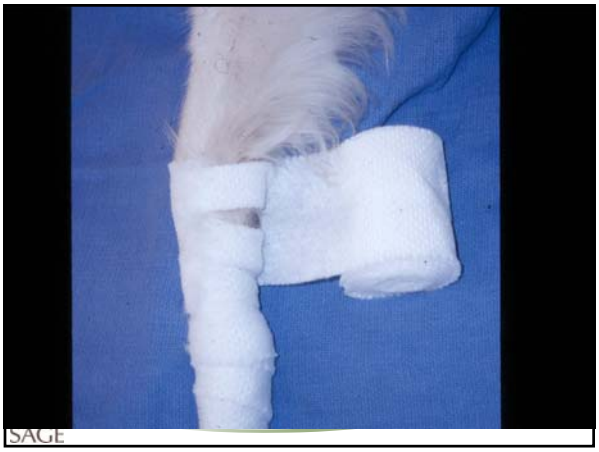
- Do ***not*** let your dog get a Big Foot







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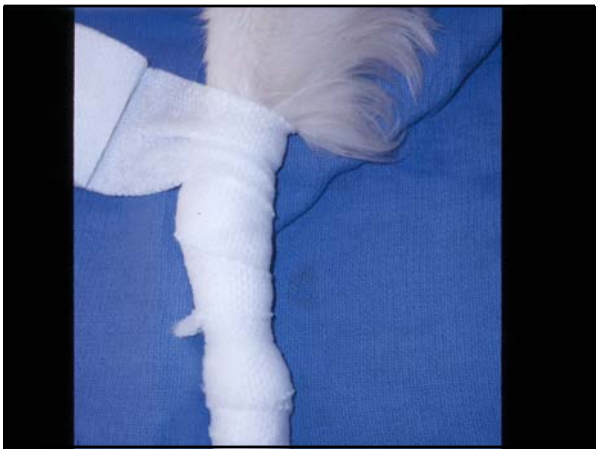

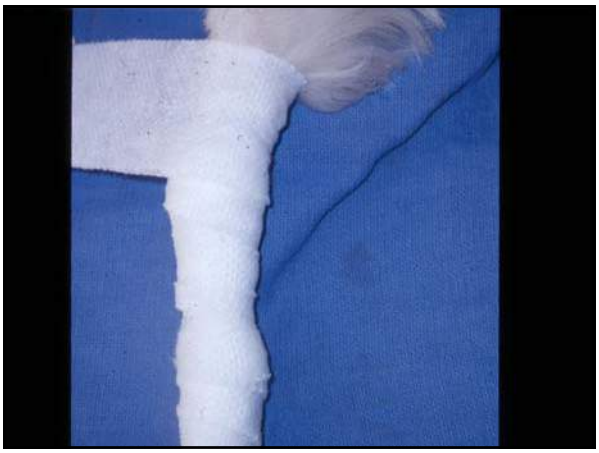

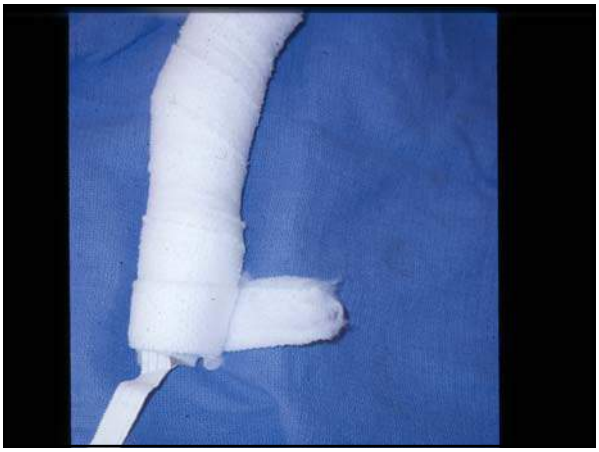

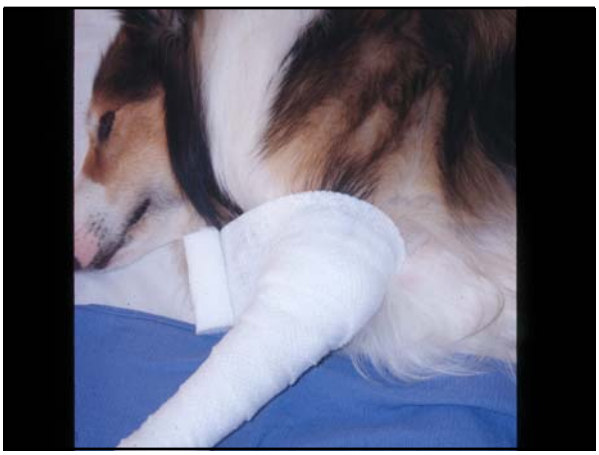

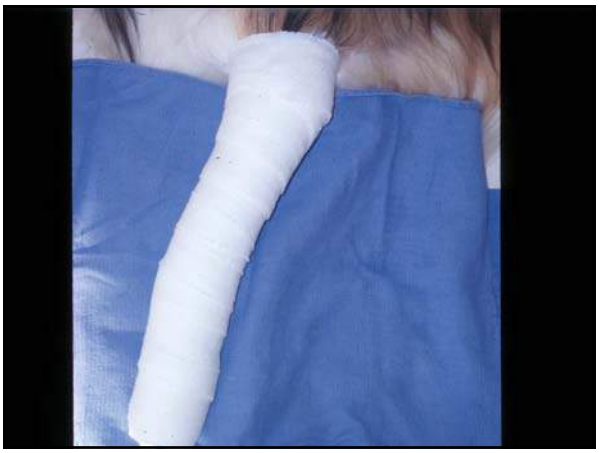

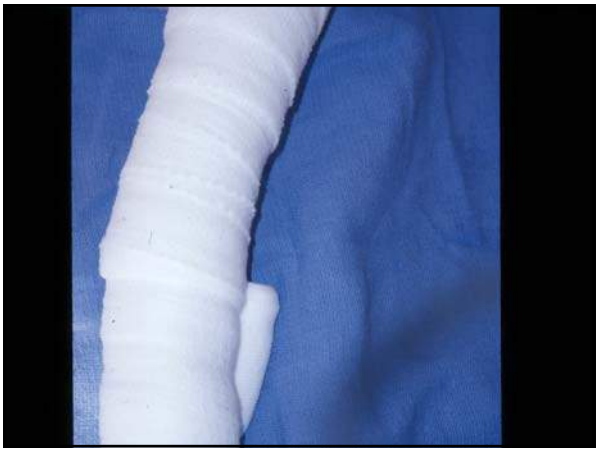

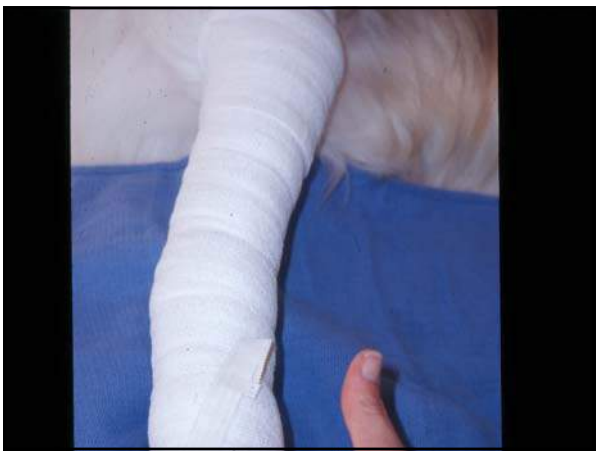

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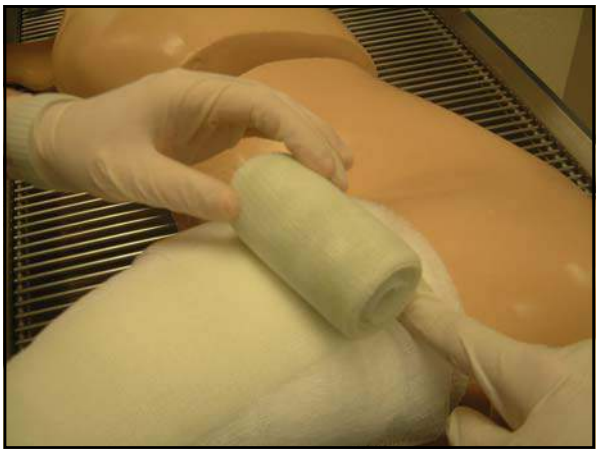

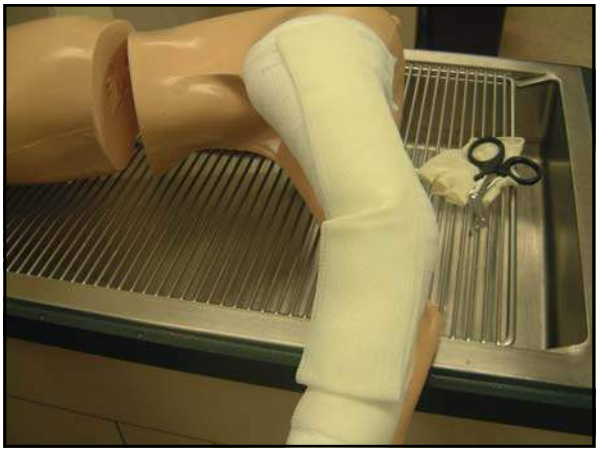

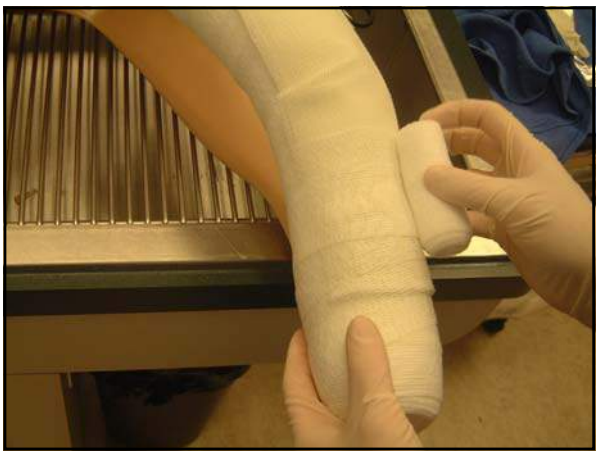

Notes on Fiberglass material

- Do not lay on table, it will transfer to surface
- Wipe scissors off right away after cutting
- Exothermic reaction: will get warm, not hot
- If wet with warm water, will harden faster
  - This is usually NOT a good thing, use cool water

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More notes on Fiberglass

- It will stick to cling gauze but can be removed and re used for the next bandage
- Remember:
  - Stirrup
  - Cast padding
  - Cling gauze
  - Plastic wrap?
  - Splint
  - Cling gauze
  - Vet wrap.

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To avoid casting material sticking

- Wrap bandage in plastic cling
- Apply splinting material
- Allow to harden
- ***Remove splint and plastic wrap***
- Replace splint
- Apply cling
- Apply outer wrap

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Tin Snips

- Can be used to trim cast material that is about 5 or less layers thick.
- Great to use to clean up edges or trim off some near the toes or the top

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Lab

- Groups
- Pair up
- Timing

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Lab

- First apply modified Robert Jones
- Applying a custom made lateral splint
  - Fibercast casting material
    - Use gloves
- Apply on each other from tips of fingers to mid radius
- Apply onto non-dominant arm
- WEAR IT A FEW HOURS…OR NOT

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Questions?

Thank you to our sponsors!!
Also, special thanks to Dr. Lissa Richardson

"It's a Bloat!:" The Dreaded GDV

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Los Angeles, CA

Introduction: GDV stands for gastric dilatation-volvulus. This is a life-threatening and represents one of the few true surgical emergencies. These patients need not only aggressive fluid resuscitation and pain management, but also require emergency surgery to de-rotate their stomach and restore abdominal blood flow. While primarily a syndrome of large breed dogs, GDV can occur in small dogs and cats. "Bloat" refers gastric distension and dilatation, often a result of overeating or ileus. Volvulus is added when the stomach rotates at the gastro-esophageal junction, and often can rotate at the level of the antrum as well. Gas and fluid that have accumulated in the stomach are retained, and burping, or eructation, is not possible. This combination of gas/fluid buildup and disability to relieve the pressure via eructation, dilates the stomach, creating increased intraabdominal pressure, decreased gastric perfusion, and multiorgan dysfunction.

Risk factors: Several anecdotal risk factors have circulated in the veterinary community. These include: post-prandial exercise, gluttony, and food types have been associated with GDV. However, studies have failed to elicit these as actual risk factors. The risk factors proven in the literature, surprisingly, cite increasing age, having a first-degree relative with GDV, and eating faster as risk factors. These, of course, are hard to identify on a brief phone call with an owner, unless they happen to mention the patient's sibling had "bloat" before!

Clinical signs: Clinical suspicion for bloat/GDV usually begins on the phone. That is why technicians answering the phone at an emergency facility should be on the lookout for key points presented by the owner:

-"He's trying to vomit"
-"He's been retching"
-"He just ate and now he keeps pacing…"

Vomiting, attempts to vomit, retching, and often abdominal discomfort can be indicative of GDV. It might be often associated with exercise or a meal, but it does not have to be. In large chested dogs, such as Weimaraners, Standard Poodles, and Setter breeds, owners may not notice a "distended" abdomen. These patients often won't present with abdominal distension as they "tuck up" their stomachs into their expansive chest cavity. However, any patient presenting with GDV will often have abdominal distension and tympany, ptyalism (drooling), and difficulty breathing. Patients may also present with signs of shock. Tachycardia, abnormal pulse rhythms (pulse deficits), injected or muddy MM's, and hypotension may occur.

Pathophysiology: As the syndrome begins, excessive gas and fluid develop within the stomach. The stomach will rotate around the gastro-esophageal junction, which prevents release of the digestive gases. The stomach may also rotate near the pyloric antrum, creating the "double bubble" effect, often seen on radiographs. The pylorus and duodenum will rotate towards the left, and the stomach is often moved ventrally as it fills with gas. This progression leads to respiratory difficulty, obstructive shock, decreased gastric perfusion, and decreased perfusion to the abdomen. Respiratory difficulty occurs due to gastric dilation and prevention of full expansion of the diaphragm. As the stomach swells, blood flow decreases as gastric arteries are compressed by the increasing pressure. Obstructive shock refers to decreased venous return and decreased perfusion from the result of physical obstruction to blood flow. In this case swelling of the stomach causes increased intra-abdominal pressure, increasing resistance to blood flow to the abdomen. In addition, the caudal vena cava is often compressed and blood flow to the heart is compromised, leaving less blood to be returned to the cranial portion of the body. These patients have a

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“relative” hypovolemia, meaning they have not usually suffered hemorrhage and subsequent blood loss, but still have decreased intravascular volume and hypoperfusion. The spleen is often affected and may be necrotic upon surgical exploration. In addition, these patients can suffer hemorrhage as the gastric distension and rotation cause rupture of the short gastric arteries. Other complications of decreased gastric perfusion include mucosal barrier erosion, ulceration, GI hemorrhage, and GI bacterial translocation. These patients are also at risk for aspiration pneumonia. Cardiac arrhythmias are reported to occur in 40% of GDV cases. These arrhythmias most often include ventricular arrhythmias, premature complexes, and tachycardia. In addition, GDV can cause significant metabolic acid-base abnormalities. Though blood pH might be normal, patients can be experiencing simultaneous acidotic and alkalotic states.

Initial interventions: The most important goal of treatment in GDV patients, initially, is correction of shock. Initial treatments include IV catheter placement, aggressive fluid resuscitation, and relief of gastric distension prior to surgery. Patients should be stabilized as best as possible prior to anesthesia to maximize positive outcome. Quite often large dogs require several liters of fluid, so placing 2 large bore cephalic catheters is ideal. Saphenous catheters are relatively contraindicated since there is no blood flow to the caudal portion of the body. Crystalloid and colloid fluids can be administered until perfusion parameters such as MM color, CRT, Heart rate, Mentation, and Lactate start to respond. Choices for fluid therapy include Lactated Ringer’s, Normosol-R, 0.9% Saline (although balanced solutions are ideal), and Hetastarch products. Initial bloodwork should consist of a minimum database: PCV/TS, Lactate, Venous blood gas, Electrolytes, BG, and Azostick/BUN. PCV/TS may be elevated consistent with dehydration or hypovolemia. TS may be low due to hemorrhage. If TS <5 g/dL, hemorrhage should be expected. PCV may or may not reflect hemorrhage due to the ability of the canine spleen to contract and release RBC’s into circulation. Lactate will often be high (>2 mmol/L) indicating hypoperfusion and anaerobic metabolism. Venous blood gas may reveal a normal pH, with a respiratory alkalosis ($PaCO_2$ <35 mmHg), and electrolyte abnormalities may include hyperchloremia, hypochloremia, hypokalemia, and low bicarbonate levels. Blood glucose levels may fall if the patient is presenting in decompensatory shock. Pain medication should be administered early as well. Options should be limited to pure mu opioid medications (Hydromorphone, Morphine, Fentanyl, Oxymorphone) as these are potent and highly reversible. Morphine is not given IV due to risk of histamine release. The other pure mu opioids may be superior in these types of patients. Trocharization may be necessary if perfusion to the abdomen is highly compromised, or respiratory distress is occurring. This is achieved using a 14ga catheter inserted into the left or right cranial, dorso-lateral abdomen. It is often the point of greatest distension. Gas and fluid may come out of the catheter and it often stinks! So be prepared! Flow-by oxygen therapy can be beneficial in providing additional oxygen for perfusion. It certainly doesn’t hurt! At this point ECG monitoring should also be instituted. If patients did not present with arrhythmias they often do after resuscitation begins.

Radiography: When the patient is stable, abdominal radiographs should be obtained to confirm the presence of gastric dilatation with or without volvulus. Right lateral abdomen radiographs are typically sufficient for diagnosis. Radiographic findings include: cranial and dorsal displacement of the pylorus, a “double bubble” appearance representing gas buildup in two portions of the stomach: from the gastoesophageal junction to the pyloric antrum, and from the pyloric antrum to the duodenum, splenomegaly, and pneumoperitoneum (if gastric rupture occurs).

Surgical interventions: Once the patient has been stabilized as best as possible, they need to be anesthetized in preparation for surgery. After anesthesia, the first step is often orogastric intubation. This involves placing a large bore tube into the stomach for decompression. The stomach may be lavaged prior to surgery as well. Anesthesia can be achieved with a variety of agents. Typical induction protocols

include: Pure mu opioid and a benzodiazepine (Midazolam, Diazepam), Ketamine/Diazepam, Propofol, and Etomidate. Opioid/Benzodiazepine combinations are the safest induction protocols. Ketamine is a sympathomimetic and will increase cardiac contractility and may increase risk of arrhythmias. Its use is controversial. Propofol is a negative inotrope and vasodilator. Excessive doses may exacerbate shock states. Etomidate is a cardiovascular-sparing anesthetic induction agent. Minimizing use of detrimental induction agents like Propofol and Ketamine can be achieved by maximizing doses of safe drugs like opioids and benzodiazepines. Lidocaine, in canine patients, can be used to help facilitate intubation. Drugs to avoid include: Phenothiazine medications (Acepromazine) and Alpha-2 agonists (Dexmedetomidine, Xylazine). Protocols for induction include:

Opioid/Benzodiazepine combinations:

Hydromorphone- 0.1 mg/kg and Diazepam/Midazolam 0.2-0.5mg/kg
Fentanyl- 5ug/kg and Diazepam/Midazolam 0.2-0.5mg/kg
****Fentanyl has a short half-life. CRI should be started intraoperatively.*

Propofol combinations:

Propofol should be used in lowest doses possible:
Add an opioid induction or pre-medication and a benzodiazepine to minimize propofol dose:
Hydromorphone 0.1 mg/kg and Diazepam/Midazolam 0.2-0.5mg/kg and Propofol 1-3mg/kg SLOW
Fentanyl 5ug/kg and Diazepam/Midazolam 0.2-0.5mg/kg and Propofol 1-3mg/kg SLOW

Ketamine/Diazepam combinations:

Add a pure mu opioid to cut down need for Ketamine
Hydromorphone 0.1mg/kg and Diazepam/Midazolam 0.2-0.5mg/kg and 1-2 mg/kg Ketamine
Fentanyl 5ug/kg and Diazepam/Midazolam 0.2-0.5mg/kg and 1-2 mg/kg Ketamine

Additional drugs:
Can add Lidocaine 1-2mg/kg IV as well
Etomidate 1mg/kg IV with opioid/benzodiazepine

Proposed protocol for critical patient:
Hydromorphone 0.1mg/kg IV given at presentation
Induction: Fentanyl 5ug/kg IV, Diazepam 0.2mg/kg IV, Lidocaine 2mg/kg IV, Propofol 1mg/kg (to effect) OR Ketamine 2mg/kg IV, OR Etomidate 1mg/kg IV.

Induction should be swift. An appropriate sized endotracheal tube should be used and the cuff inflated to limit aspiration and anesthetic gas leaks. Monitoring equipment should be immediately attached. While one technician is preparing for orogastric intubation, the other staff can be clipping the patient and preparing the surgical suite. The orograstric tube is lubricated and the patient is placed either sternal, or lateral recumbency. The tube is lubricated and measured from the nares to the point of the last rib. The tube is slid into the esophagus and gently advanced. Most often resistance will be felt when the tube approaches the lower esophageal sphincter. Gently advance and rotate the tube to try and guide it into the cardia. Sometimes blowing into the tube gently will facilitate passage into the stomach. Once the tube enters the stomach, gas, ingesta, and fluid may be released. Typically the tube is held over a bucket to collect these contents. Gastric lavage then occurs to clear the stomach of unnecessary contents. While

this is occurring the anesthetic technician should be checking vitals and assessing perfusion. The GDV patient should be on 10ml/kg/hr of IV fluids during anesthesia, and gas anesthetics kept as minimal as possible. Once the patient is lavaged, they should be quickly moved into surgery, prepped, and surgery should commence.

Surgical nursing: The surgeon will need to decompress the stomach, reposition it, assess viability of the stomach, spleen and liver, assess for any necrosis, assess for any hemorrhage, and perform a gastropexy, permanently adhering the stomach to the abdominal wall to prevent further voluvlus episodes. Gastric resection or splenectomy may be necessary and surgical equipment for these may be necessary. Hemorrhage should be promptly addressed and blood products administered if necessary. There are several types of gastropexy procedures described in the literature. These include the incisional, circumcostal, belt loop, and muscular flap gastropexies. When performed correctly, all of the above techniques have fairly good success rates: 92-97%.

Post-operative care: Get ready for an intense ride! These patients require very intensive nursing care post-operatively! After recovery and extubation, monitoring of vitals should occur fairly often for the first 8-12 hours. Constant ECG monitoring is recommended as some patients will develop arrhythmias after surgery. HR, RR, MM/CRT, BP, ECG, and blood gas/lactate/PCV/TS measurements are recommended. Urine output is another frequently overlooked determinant of perfusion. Food and water can be offered several hours after surgery. Pain assessment and management should be continued as needed. NSAID administration is relatively contraindicated initially due to decreased gastric perfusion and risk for perforation. GI protectant medications such as H2 blockers (Famotidine) and Sucralfate should be considered. Metoclopramide may be used to increase GI motility. Cardiac arrhythmias are typically ventricular in nature. Sometimes patients can have isolated VPC’s and it is not too much of a concern. Treatment of arrhythmias is usually instituted when any of the following criteria are met: Tachycardia sufficient to be affecting perfusion/blood pressure, R on T phenomenon occurs, Paroxysmal (runs) of V-tach are occurring, or multi-focal VPC’s are present. Treatment of ventricular arrhythmias is typically achieved initially using Lidocaine boluses of 2mg/kg. A CRI can then be started at 50-80 ug/kg/min. If Lidocaine is not successful in converting the rhythm to a sinus rhythm, Procainamide may be used at bolus doses of 2-6mg/kg and a CRI of 20-40ug/kg/min. Other options include oral anti-arrhythmics such as Sotalol and Mexilitine. Disseminated Intravascular Coagulation (DIC) and Multi-organ dysfunction syndrome (MODS) can occur with any patient in a shock state or with systemic inflammation. Coagulation times and platelet counts should be monitored to help ward off this drastic state. MODS can manifest as acute renal failure, acute liver failure, cerebral dysfunction, respiratory distress, or devastating thromboembolic events. Another reason to monitor a minimum database and perfusion parameters often! In addition, ischemia/reperfusion injury has been a hotly debated and talked about issue in GDV. Simply put, when blood flow is blocked to an area of the body, such as in GDV, mesenteric torsion, or thromboembolism, cells fail to receive adequate oxygen. Their metabolism is able to continue for a short amount of time, but often fails quickly. Chemicals are released in chemical reactions to help keep the cell alive. When perfusion is restored, oxygen reacts with these byproducts and incites the inflammatory cascade, coagulation cascade, and causes further cell irritation and death due to toxic byproducts (reactive oxygen species). These molecules travel around the body and create havoc, causing cell death in various susceptible organs including the kidneys and heart. Lidocaine is an effective free radical scavenger. It will bind and remove detrimental radical oxygen species. However, studies are conflicting. Some had positive results and prevented injury, and another recent study (2003) failed to show a reduction in mortality from use of Lidocaine.

Conclusion: GDV is a disastrous syndrome requiring top-notch critical care from beginning to end! Patients require aggressive fluid therapy, oxygen therapy, and pain management initially. A diagnosis

must be made rapidly and gastric decompression must occur quickly. Surgery is curative to relieve distension, replace gastric position, deal with organ ischemia, and prevent recurrence. The critical post-operative period remains a source of mortality in these patients and veterinary technicians must be ready to provide intensive nursing care to these patients!

References available upon request.

All Sugar and no salt: DKA

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INTRODUCTION

Diabetes Ketoacidosis (DKA) represents a smaller subset of patients who suffer from Diabetes mellitus (DM). This form of DM is quite severe and represents a true endocrine emergency. DKA patients should not be casually treated; more invasive care might be needed to ward off a plethora of disastrous sequelae. The initial "hit" often comes in the form of some severe physiologic stress, exacerbating diabetogenic hormones and resulting in a ketonemic and acidemic patient. Due to this initial "hit" these patients need diagnostic work as well as medical interventions to treat their severely compromised state.

PATHOPHYSIOLOGY

Diabetes mellitus in canine patients results mainly from primary pancreatic failure; beta cells in the Islets of Langerhans stop producing insulin. This can result from idiopathic or immune-mediated processes. As the insulin level drops, blood glucose levels rise. In contrast, diabetes mellitus in felines is most often resultant of Type II diabetes, insulin resistance and glucose toxicity. Several conditions can cause peripheral insulin resistance and interfere with insulin's ability to affect transfer of glucose intracellularly. The resuling hyperglycemia is toxic to beta cells in the pancreas and this process eventually results in a destruction of beta-Islet cells leading to primary pancreatic failure.

In diabetic patients, cellular metabolism is disrupted by the absence of intracellular glucose. Typically, glucose is converted into pyruvate (via glycolysis) and then enters the citric acid cycle, with acetyl-CoA, to create ATP. However, in diabetic patients glycolysis does not occur. Thus, a buildup of acetyl-CoA ensues. Acetyl-CoA is produced normally by the mitochondria as a byproduct of fatty acid (FA) metabolism. However, as the excess acetyl-CoA cannot be utilized in the citric acid cycle, due to the lack of pyruvate substrates, excess builds. Acetyl-CoA is metabolized into ketone bodies when it is unable to be fully utilized.

This shift from ATP production to ketone body production is thought to occur in the presence of hypoinsulinism and the excess of diabetogenic hormones (catecholamines, glucagon, growth hormone). Insulin is an anabolic hormone, causing fat storage in adipose tissue, production of glycogen (storage form of glucose) and decreased utilization of amino acids, in favor of protein production. Glucagon, by contrast, is a catabolic hormone stimulating lipolysis, glycogenolysis and proteolysis. The condition of hypoinsulinism and glucagonemia effectively shifts the paradigm from the storage of nutrients to the breakdown of sugar, fat, and protein, into glucose, fatty acids, and amino acids. The lack of insulin renders the circulating glucose useless, and fatty acid metabolism prevails. As more and more acetyl Co-A is made ketone bodies are produced from exhaustion of cellular processes made to deal with them.

The three major ketones produced in the body include: acetoacetate, β-hydroxybutyrate, and acetone (which is derived from the breakdown of acetoacetate). Ketones can be used for energy

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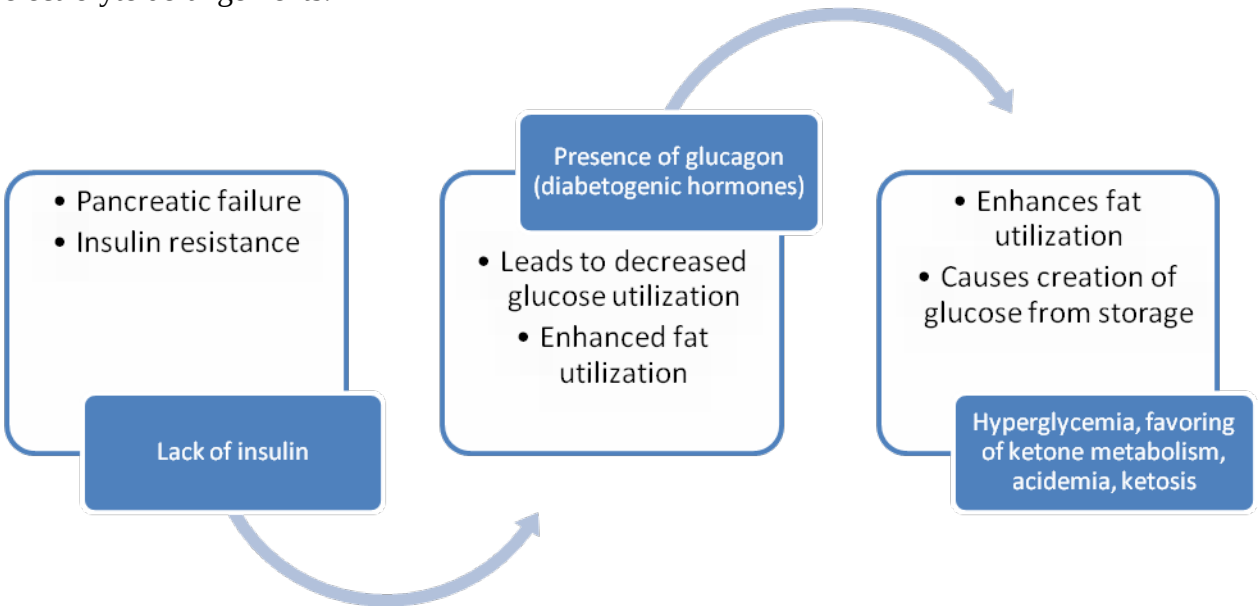
in times of starvation. However, they are quite acidic and contribute to an acidemia (presence of excess Hydrogen in the bloodstream). The dipsticks used to assess biochemical parameters in urine are effective in measuring only two types of ketones: acetone and acetoacetate. This might seem like an effective tool, but the ketone that is typically in higher concentration systemically in DKA patients is β-hydroxybutyrate. Anecdotally, placing one drop of hydrogen peroxide on the urine strip can enhance a reaction to convert acetoacetate to β-hydroxybutyrate and can potentially reveal underlying ketosis. In addition, studies have confirmed the accuracy of using serum, from a PCV tube, to assess ketosis using the urine dipstick method.

As ketosis and hyperglycemia ensue, glucose and ketones cause an osmotic diuresis leading to polyuria. Excess water and sodium losses leads to dehydration and potential hypovolemia. Anorexia and vomiting also contribute to severe fluid losses and metabolic/electrolyte derangements. Acidosis is compounded by hypovolemia, decreased tissue perfusion and subsequent hyperlactatemia. Osmotic diuresis can lead to total body depletion of sodium, chloride, and potassium. Death can ensue from hypovolemia, severe acidosis, and untreated electrolyte derangements.



RISK FACTORS

DKA is typically a disease of middle-older age animals. The median age reported is 8 years. The consistent risk factor is the presence of a concomitant illness or condition. 70-90% of dogs and cats affected with DKA have the presence of detectable clinical illness, which may be related or unrelated to the underlying DM. Various conditions associated with DKA include: acute pancreatitis, urinary tract infection (UTI), Cushing's disease, chronic renal failure (CRF), hepatic lipidosis (feline), neoplasia, infection. Treating the DKA may not result in an improvement in overall clinical signs in the long term if the occult precipitating factor is not identified and treated.

CLINICAL SIGNS

Clinical signs can vary from mild to severe. Very rarely DKA patients can present who are still eating. But typically history includes polyuria, polydipsia, anorexia, vomiting, lethargy, mental dullness, and weight loss. Physical exam can reveal: tachycardia, tachypnea or Kussmaul respirations (deep and labored breathing pattern), 8-10% dehydration, lateral recumbency, obtundation, and potentially other clinical signs such as harsh lung sounds, coughing, abdominal pain, cataracts, and icterus.

INITIAL INTERVENTIONS

DKA patients should ideally receive fluid resuscitation if they are in shock. This involves placement of a large-bore peripheral catheter and administration of IV fluids. Blood should be drawn for acid-base analysis, lactate, chemistry and electrolyte panel and complete blood count. Further tests can be performed once the patient is stable including testing for Cushing's, among other underlying endocrinopathies. If there is the presence of urine a sample should be obtained for urinalysis, urine culture, ketones, and a urine specific gravity. A balanced isotonic crystalloid can be administered in incremental boluses until improvement in resuscitation end-points is achieved (improved HR, improved blood pressure, reduction in lactate, etc).

LABORATORY ANALYSIS

There are many common abnormalities associated with the DKA patient. These are summarized in the table below:

Abnormality	**Pathophysiology**	**Clinical Signs/Consequences**	**Treatment**
Non-regenerative anemia	Anemia of chronic disease	Decreased PCV and HCT	Transfusion-potentially
Hyperglycemia	Low insulin levels and increased conversion of glycogen to glucose	Polyuria, osmotic dehydration, glucosuria	Insulin therapy
Elevated liver enzymes: ALT, Alk Phos	Decreased perfusion to liver, underlying condition	Icterus, Hepatitis, Hepatic lipidosis	Hydration, diagnosis underlying cause
Azotemia: Increased BUN, Creatinine	Pre-Renal or Renal azotemia	Vomiting (uremia),	Fluids, treat CRF if present
Hyponatremia/Hypochloremia	Renal, GI loss-hyperglycemia contribution	Seizures	Fluid replacement
Hypokalemia	Renal, GI loss, eventual insulin administration causes	Hypoventilation	Fluid replacement

	extracellular shift of potassium intracellularly		
Hypophosphatemia	Decreased body stores, eventual insulin administration causes extracellular shift of phosphorus intracellularly	Cardiac arrhythmias, hemolytic anemia	Fluid replacement
Hypomagnesemia	Renal loss	Inability to correct serum potassium concentrations,	Fluid replacement
Metabolic acidosis	Presence of ketoacids, uremic acids (potentially), lactic acid	Worsening acidosis, cellular disruption	Fluids, Insulin therapy to lower ketonemia
Hyperlactatemia	Decreased tissue perfusion	Acidosis	Same as above

CONTINUED THERAPY

Once the patient has been diagnosed with DKA, aggressive fluid therapy should be focused on correct hypovolemia (life-threatening) and then addressing hydration deficits over a 12-24 hour period. Electrolyte replacements include: potassium, phosphorus, magnesium.

Hypokalemia can often be severe and potassium can be replaced using a sliding scale chart (as below). Potassium administration should not exceed 0.5-1meq/kg/hr.

Phosphorus may need supplementing, and this can be provided parenterally in the form of Potassium Phosphate injection. Phosphorus can be supplemented using rates of 0.03-0.12 mM/kg/hr (Silverstein & Hopper, 2009). Sample calculations are found below. Amount of raw potassium found in potassium phosphate injection can be calculated and the remaining amount of potassium supplementation needed can be added in the form of potassium chloride. Potassium phosphate contains 3 millimoles (mM) per mL of phosphate and 4.4 meq/mL of potassium ions.

Magnesium can be supplemented in the form of magnesium sulfate injection can be administered at a rate of 0.5-1 meq/kg/day.

Hyperglycemia is treated with insulin administration. There are two different methods of replacing insulin in the DKA patient: the IM method, and the CRI method. The IM method involves giving 0.2U/kg of regular insulin IM, followed by a blood glucose (BG) an hour later, and then an additional dose of insulin (ranging from 0.05-2U/kg) depending on the resultant BG concentration. The goal is to lower the BG about 50-100 mg/dL every hour. IV fluids should be administered for a few hours prior to insulin administration.

The CRI protocol involves delivering insulin as a constant rate infusion and is prepared as follows: Add 2.2 U/kg insulin (dogs) and 1.1 U/kg insulin (cats) to a 250 bag of 0.9% NaCl. Flush 50mL out of the line to prevent insulin binding to the IV line. Then initially start the rate at 10mL/hr. BG's are measured every 2 hours. Using the sliding scale chart found below, adjust the insulin CRI as needed to lower the BG.

Central lines are ideal for patients that are in DKA crisis, as they will be receiving multiple fluid therapies and will need multiple repeat venipunctures for blood chemistry analysis. Catheters can be through-the-needle or Seldinger (mutli-lumen) types and can be placed in the jugular or saphenous veins. Proper catheter care should be followed to ensure patency and minimize catheter-related infections or phlebitis.

Persistent acidosis can potentially be treated with intravenous Sodium bicarbonate therapy. However, this is a controversial subject and most recommend stabilizing the patient with sufficient IV fluids and insulin therapy first. Bicarbonate has some nasty side effects including: exacerbation of hypokalemia, hypocalcemia, hypernatremia, and paradoxical CNS acidosis. Bicarbonate replacement can be achieved by using the following formula: (0.3 x BW (kg) x BE) = Bicarbonate amount (in meq) desired. Administer ½-1/3 of this amount slowly over 20-30 minutes. Monitor pH every hour. (Recommendations from: Silverstein and Hopper, 2009).

OUTCOME

DKA patients have a favorable prognosis. However, once the DKA crisis is solved the patient is typically either insulin-dependent or maybe able to convert to a non-insulin dependent state; both of which require intensive owner compliance and finances. If the owner is willing, DKA is treatable and patients can lead long, healthy lives as healthy diabetics.

Potassium supplementation chart

Potassium Concentration	Amount to add to 1 L of fluids
<2 meq/L	80 meq
2.1-2.5 meq/L	60 meq
2.6-3.0 meq/L	40 meq
3.1-3.5 meq/L	30 meq
3.6-5 meq/L	20 meq

*****DO NOT EXCEED 0.5-1meq/kg/hr rate*****

Example calculation of Phosphorus, Magnesium and Potassium Chloride concentrations:

10 kg patient
1 L of fluids at 25 ml/hr = 40 hours
Patient's potassium is 2.2 meq/L (reference range: 3.6-5 meq/L)
Patient's phosphorus is: 1.0 meq/L (reference range: 2.5-6 meq/L)
Patient's magnesium level is: 0.7 meq/L (reference range: 1.5-2.5 meq/L)

Potassium Chloride is: 2meq K^+ /mL
Potassium Phosphate is: 4.4 K^+ /mL AND 3mM PO_4^{3-} /mL
Magnesium sulfate is: 4 meq/mL Mg^{2+}

Step 1: Calculate Phosphorus requirements first:
0.03mM/kg/hr = 0.03 x 10 = 0.3mM/hr x 40 = 12 mM to add to 1 L. 12/3 = 4cc KPO4/L

Step 2: Figure out the amount of K+ in the phosphorus supplementation:
4cc of KPO4 x 4.4 = 17.6 meq of K^+

Step 3: Use the sliding scale chart to figure out amount of total K+ supplementation needed:
Patient's potassium is 2.2 meq/L. According to the chart we need to supplement 60 meq/L.

Step 4: Subtract total amount of K to add from amount coming from phosphorus supplementation:
60 – 17.6 meq = 42.4 meq/L to add from KCL.
KCL is 2meq/mL SO 42.4 /2 = 21.2 mL of K+ to add to the IV fluids.

Step 5: Double check you are not exceeding the 0.5meq/kg/hr rule.
10 kg patient x 0.5 meq/kg/hr = 5 meq/hr
60 meq KCl / 40 hours = 1.5 meq/hr

Insulin CRI Chart

Blood Glucose (mg/dL)	CRI Rate (0.9% Saline + Insulin)	Dextrose to add to regular cystalloids
> 250	10mL/hr	NONE
200-250	7mL/hr	2.5% Dextrose
150-200	5mL/hr	2.5% Dextrose
100-150	5mL/hr	5% Dextrose
<100	STOP	5% Dextrose

The Blocked cat

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Introduction: FLUTD, FUS, Blocked, urethral obstruction. What does it all mean!? Thankfully most, if not all, ER techs are familiar with the "blocked cat." These patients are suffering from a urethral obstruction, most often show lower urinary tract disease signs: stranguria, dysuria, hematuria, pollakiuria, and most often have urolithiasis. Cats tend to get oxalate and struvite stones evenly, thus technicians should be familiar with urinalysis and the appearance of these crystals. These patients are most often stable when presenting, although around 12% can have life-threatening electrolyte and acid-base abnormalities that if aren't treated immediately may result in death. However, this is a relatively exciting and rewarding emergency!

History and Clinical signs: Owners often report a myriad of clinical signs with their male cats. Often an owner will notice urine spotting, potentially with hematuria, frequency of visits to the litter box, excessive perineal licking, yowling, vocalizing, lethargy, anorexia, constipation, or actual visualization of straining to urinate. These patients should have their bladder palpated immediately upon arrival, in addition to a TPR and primary survey. Clinical signs of these patients include a large firm bladder, abdominal pain, tachycardia, bradycardia, hypothermia, hyperthermia, tachypnea, lateral recumbency, obtundation, and vocalization. We all know that blocked cat yowl!

Pathophysiology: Most cats obstruct because of a mucous plug or small urolith (grit) that lodges in their urethra. As urinary output decreases the bladder fills. Inability to urinate causes an increase in uremic toxins, such as BUN and creatinine, and a decreased excretion of potassium and phosphorus ions. Because these patients stop taking in water, they become dehydrated and hypovolemic. The severe metabolic derangements can contribute to a metabolic acidosis, caused by inability to excrete hydrogen ions and accumulation of lactate, and this worsens the process. Hyperkalemia can cause a large amount of detrimental cardiovascular arrhythmias which can result in death. As the bladder continues to fill, wall necrosis occurs, placing the animal at risk for bladder rupture and subsequent uroperitoneum. In addition to mucous plugs, neoplastic causes of urethral obstruction are seen. These are typically associated with transitional and squamous cell carcinomas. Urethral strictures can also cause obstruction, albeit rare.

Initial interventions: When a critical blocked cat presents to the emergency room there are several treatment goals: 1- Identify and treat any underlying metabolic/acid-base abnormalities, 2- Relieve the urethral obstruction, 3- Restore urethral patency. The first step in treating a blocked cat involves placing an IV catheter, attaching an ECG, and running a minimum database. The minimum database often reveals: low pH, low bicarbonate, low calcium, high potassium, high BUN, and high lactate. The high lactate, low bicarbonate and low pH levels indicate a metabolic acidosis. The high BUN is a result of post-renal, pre-renal, and potentially renal azotemia. The high potassium is a result of decreased ability to excrete potassium. Initial IV fluid therapy is achieved with a balanced crystalloid solution. It was once recommended to use 0.9% NaCl fluids, because they have no potassium. However, recent studies indicate the small amount of potassium in balanced solutions like Normosol-R, Lactated Ringer's, or Plasma-lyte, rarely contribute to worsening hyperkalemia. In addition, these solutions have buffers to

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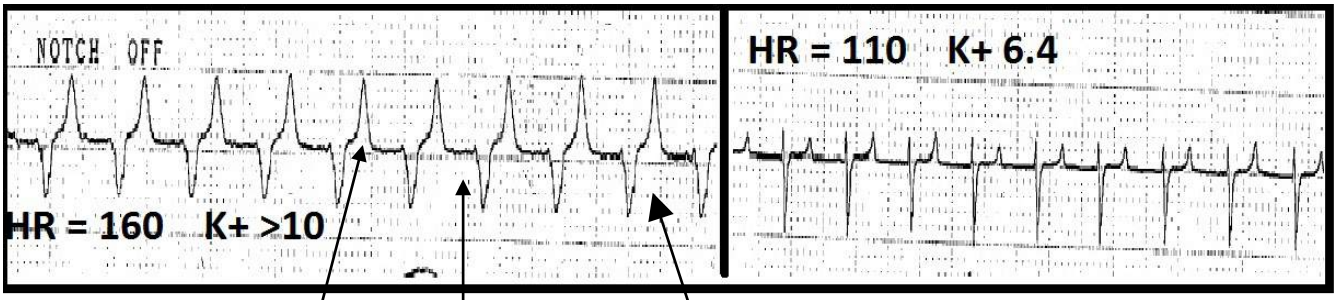
help restore normal blood pH; in contrast, 0.9% NaCL is acidic. Sometimes an initial fluid bolus will be enough to lower the serum potassium to normal, or lower than critical range. In addition, fluids may help restore perfusion and return lactate levels to normal. Common ECG findings with hyperkalemia include: depressed or absent P-waves (atrial standstill), wide QRS complexes, tall T-waves, ventricular tachycardia, or sinus tachycardia.



Metabolic and [illegible] be a[illegible] fore the patient is anesthetized/se[illegible] hral deobstruction pr[illegible] nce IV fluids have been given, if the patient is still severely hyperkalemic other tre[illegible] ld be instituted to address that. The following chart summarizes the additional treatments used in hyperkalemia:

Drug	**Dose**	**Mechanism of action**
Calcium gluconate	50-100mg/kg IV slow	Cardioprotective, reduces excitability of myocytes. Does not lower serum potassium
Regular insulin	0.1-0.25U/kg IV	Mobilizes serum potassium ions intracellularly
50% Dextrose	0.5g/kg (1mL/kg) diluted 1:3	Counteracts hypoglycemia from regular insulin administration. ALSO: incites insulin release from pancreas to lower K+ levels.
Sodium bicarbonate	0.3 x Base Deficit x BW (kg). Administer ½-1/3 dose IV SLOW (20-30 mins). BD is typically calculated on a blood gas machine.	Not often needed. Fluids plus additional treatments above often necessary. Do not use unless other treatments not working. Will cause worsening of hypocalcemia. Also will cause metabolic alkalosis.

Once the blocked cat has been stabilized, serum potassium has been lowered, they are ready for deobstruction. A variety of anesthesia protocols have been studied in these patients:

-Opioid/benzodiazepine combinations (termed neuroleptanalgesia) may provide enough sedation for urethral catheterization. Protocols include:

-Hydromorphone 0.05mg/kg IV and Diazepam/Midazolam 0.2-0.4mg/kg

-Oxymorphone

Cats do not tend to do well with large doses of opioids. Oxymorphone, anecdotally, seems to be a decent choice for a pure mu opioid in the cat.

Butorphanol (0.2-0.4mg/kg) is an excellent sedative, but provides not much analgesia. It can be combined with a benzodiazepine for excellent sedation.

-Ketamine/Diazepam combinations are controversial. Ketamine is excreted in the kidney in the cat, and urethral obstruction can cause accumulation of the drug. Yet if the goal is to quickly deobstruct these patients some authors think it is not much of a concern. It is also contraindicated in heart disease, so make sure the cat has been ausculted first!

Ketamine 2-5mg/kg and Diazepam 0.2-0.4mg/kg IV

-Propofol can be used but it is a negative inotrope and vasodilator. Thus, it is recommended to use as little Propofol as possible. (1-2mg/kg IV slow)

-Once anesthesia is achieved, these patients can be maintained on gas anesthesia and titrated as needed.

For any critical patient, they should receive adequate analgesia before the procedure. Also, all equipment should be ready before induction of anesthesia, to minimize anesthesia time. In addition, full monitoring equipment should be available including Spo2, BP, ECG, Temp, and ETCO2 if possible. These patients should be kept on surgical rate of fluids during the procedure, and kept warm if possible. All general principles for anesthesia of critical patients (MAP reduction, pre-oxygenation, monitoring/treatment of hypotension, anticipation of problems) should occur with these patients as well.

Deobstruction procedure: Once the patient is ready for catheterization, the penis can be extruded and should be examined for mucous or grit. Sometimes this can be teased out and the obstruction relieved. If urohydropropulsion is to be used, an open end tomcat catheter, or sterilized olive tip catheter can be used. The catheter is lubricated and gently advanced into the urethra until resistance or grit is felt. Saline is gently pulsed into the catheter to relieve the obstruction. The process can be long and tedious, but once the obstruction is popped back into the bladder, the bladder should be emptied. Then longer term urinary catheterization is performed using a 3.5Fr or 5Fr Red rubber catheter. This catheter should be pre-measured, and an x-ray post placement should be taken to avoid excessive lengths of catheter in the bladder. This can be sutured in using a variety of methods including placing stay sutures, tape or Elastikon, staples, or suturing directly to the prepuce. An E-collar should be placed on the patient to avoid removal. The bladder should be flushed with copious amounts of saline. Anecdotally, it is recommended to

flush until the urine is somewhat clear, especially if gross hematuria is present. Once this is done, the urinary catheter should be connected to a sterilized closed system. This is often done using a re-sterilized IV bag, and new IV line tubing. A closed system is recommended to prevent development of a UTI. The majority of cases of urethral obstruction are caused by sterile cystitis, and some are caused by idiopathic cystitis unlrelated to urolithiasis. Leaving a catheter open puts the patjent at risk for an iatrogenic resistant UTI. Antibiotics are also listed as relatively contraindicated when a urinary catheter is in place. Patients can be stabilized, discharged, and seen a few days later for a cystocentesis and urine culture submission. Rarely, patients cannot be catheterized and cystocentesis is a viable option for urine removal. However, there is certainly a risk of bladder rupture. Urohydropropulsion can cause bladder rupture as well, causing uroperitoneum and requiring surgical repair of the bladder wall.

Nursing care: Post-obstruction patients require intensive nursing care. Firstly, they need their analgesic needs met as best as possible. Most often intravenous, and later transmucosal, buprenorphine is sufficient. Patients may be started on anti-spasmodic medications such as phenoxybenzamine or Prazosin. Phenoxybenzamine takes 72 hours for full effect, while Prazosin has a much shorter onset of action. However, these drugs are vasodilators and hypotension can occur. It is recommended to monitor the cat's cardiovascular status when starting these drugs. Secondly, these patients have an invasive device in place; the urinary catheter. Proper care and maintenance is required to prevent iatrogenic UTI development. Catheters should be inspected for patency q4-6h, and cleaned with a dilute chlorhexidine solution. Urinary output should be monitored q4hours by using a needle and syringe, and not disconnecting the IV bag from the soluset. The bag and line should be changed q24hours as needed. Urine output should be monitored closely in these patients. They often suffer from post-obstructive diuresis and the kidneys go into overdrive. Urine output can exceed 3-5ml/kg/hr! In this case, the patient needs their IV fluids increased, not decreased. Lowering the fluids in evidence of excessive urinary output could be detrimental. These patients may be on 2-4x maintenance fluid rates! Once patients are more stable, 12 hours after deobstruction, their nursing care can be limited to pain assessment/scoring, urinary catheter care, and TLC (food and water should be offered). Most patients go home 2-3 days after obstruction relief!

Sample nursing orders:

Pain assessment/scoring q4hours
Swab U-cath with dilute chlorhexidine q4h
Empty/quantify urine q4h (Alert if <1ml/kg/hr OR >2ml/kg/hr)
Body weight BID
Change Urinary catheter bag/line q 24hours
E-collar on at all times
Recumbent care/TLC as needed (q4-8hours)

Medical management: This is considered very important in feline patients to prevent recurrence of FLUTD. If the cause is not a urolith, it is often feline idiopathic sterile cystitis. Treatments include: stress reduction, moist food, increased water intake, glycosaminoglycans (glucosamine and chondroitin), and potentially feline pheromones (Feliway®). If uroliths are the cause, feeding a canned urinary diet, with increased access to water (fountains, etc) helps dilute urine. In

addition, glycosaminoglycans can also be used. Urine should be submitted for culture and sensitivity and antimicrobial medications used if indicated.

References available upon reqest

STICKING TOGETHER: IMHA

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Introduction

Immune-mediated hemolytic anemia (IMHA) is a very common disease process seen in small animal internal medicine or emergency practice. A typical presentation is a small breed dog (often white), who presents pale and yellow. Understanding the pathophysiology and treatment of this condition is vital for the veterinary technician to take quick action.

Pathophysiology

The hallmark to this disease is red blood cell destruction at the behest of the immune system. Simply put, red blood cells become targeted to be destroyed by antibodies and complement. This mechanism is typically reserved for foreign pathogens and serves as a defense mechanism to the host. But somehow, the immune system begins to recognize its own red blood cells as foreign and initiates the campaign of destruction. When RBC's are perceived as foreign, antibodies will attach to them. Antibodies will label the red blood cells for monocytes and macrophages to engulf and lyse. The Fc receptor on the macrophage corresponds to the Fc region on the antibody and initiates destruction. This system, known as the mononuclear phagocyte system, is usually a very important system in the body; removing pathogens and old blood cells. This system is essentially fluid and can occur all throughout the body, but particularly in the spleen and liver. Lysis of red blood cells outside of the vasculature is called extravascular hemolysis. Intravascular hemolysis can also occur, and this is often caused by the complement system, another attack system of the immune system which punches holes in a cell membrane allowing influx of water, cellular swelling, and eventually lysis.

Agglutination of red blood cells occurs when patients have an excessive amount of anti-RBC antibodies on the red blood cell surface. These are "sticky" with other antibodies and RBC's will clump together. Additionally, these patients typically have impaired ability of these clumps to allow movement of blood cells into smaller capillaries, and thus they are more readily targeted by the MPS.

Typically, antibodies are only directed towards mature red blood cells. But red blood cell precursors, in the bone marrow, can also be targeted for destruction. When this occurs, the typical

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highly regenerative pattern of anemia seen in IMHA is absent, and severe non-regenerative anemia is present.

IMHA is typically categorized as primary or secondary. The primary form is typically idiopathic, with no underlying cause found. Secondary IMHA is the result of some incitement of the immune system through either infection, drug therapy, or neoplasia.

Table 1- Causes of secondary IMHA

Infectious	**Drug therapy**	**Immunological**
FeLV	Antibiotics (Sulfa, Penicillins, Cepha's)	Lupus
Mycoplasma infection	Methimazole	Transfusion reactions
Ehrlichiosis	NSAIDS	Other immune-mediated disease
Babesiosis	Chlorpromazine	
Heartworm disease	Quinidine	

Categories of IMHA

There are 5 categories of IMHA categorized by either warm antibody, cold antibody, agglutination or hemolysis. These will be briefly described here.

Warm antibody type, agglutination: This type presents with high levels of anti-RBC antibodies, severe extravascular hemolysis and agglutination.

Warm antibody type, intravascular hemolysis: This type presents with hemoglobinemia and hemoglobinuria, representing intravascular hemolysis. Complement fixation is mainly responsible for cell death intravascularly.

Warm antibody type, incomplete antibody: This type can be the chronic IMHA, that's PCV waxes and wanes. These patients have few anti-RBC antibodies, no agglutination, and no intravascular hemolysis.

Cold antibody type, agglutination: This type of IMHA represents antibodies that are not physiologically active at body temperature, but rather become active at colder temperatures. These patients will develop agglutination.

Cold antibody type, non-agglutinating hemolysis: Similarly to above, these antibodies do not react at warm temperatures. But during cold temperatures can cause hemolysis.

Clinical Signs

Clinical signs of IMHA can be variable. Many patients will exhibit signs consistent with lack of oxygen-carrying capacity of the blood such as collapse, tachypnea, exercise intolerance. Presenting complaints can range from anorexia, to vomiting, to the "ADR" category. It is not uncommon that an owner brings in a cocker spaniel who hasn't been eating, and the triage nurse lifts the gums to find pallor and icterus. Occasionally patients can present with fever or lymphadenopathy. Jaundice/icterus is very commonly observed, representing hyperbilirubinemia from excessive extravascular hemolysis. Rarely, either in hospital, or at home, patients can suddenly die; mostly attributable to their hypercoagulability and potentially a pulmonary thromboembolism.

Diagnosis and Clinical Signs explained

First, let's discuss the common clinical signs and lab findings and how they relate to this disease:

Collapse, tachypnea, tachycardia, exercise intolerance: Anemia causes a decrease in the ability of blood to deliver oxygen to tissues (as there are less red cells to carry oxygen). This results in early exhaustion and compensatory mechanisms (tachycardia, tachypnea) to try to maximize oxygen delivery.

Pallor: Pale mucous membranes represent vasconconstriction and anemia.

Icterus: Extravascular hemolysis results in free hemoglobin release. Hemoglobin is metabolized to bilirubin in the blood stream, becomes conjugated (combined) with albumin and is presented to the hepatocyte for further metabolism. When extravascular hemolysis exceeds the liver's ability to deal with the bilirubin, hyperbilirubinemia, bilirubinuria, and icteric colored mucous membranes occur.

Fever/lymphadenopathy: Severe inflammatory response (infectious or non-infectious) can result in fever, and lymphadenopathy.

Diagnosis of IMHA includes the presence of several factors: anemia (typically regenerative), spherocytosis, and agglutination. The anemia is typically regenerative, with an elevated reticulocyte count; although if RBC precursors are targeted it may not be regenerative. Agglutination is a common finding, and often starts in the blood tubes that are drawn for analysis. It can also be seen in microhematocrit tubes. A saline agglutination test should be run to confirm or identify occult

agglutination. Spherocytes, seen on blood smear, are fairly pathognomonic for an immune mediated process targeted at RBCs. Spherocytes are small, circular red blood cells, that lack the central pallor. These are cells that have had sections of their phospholipid cell wall and antibodies that normally coat the surface "shaved" off by attempted clearance by the MPS. The monocytes took chunks out of the RBC exterior, which eventually resulted in a circular shape.

Initial diagnostics should include: complete blood count, blood smear, PCV, total solids, saline agglutination test, and platelet count. About 10% of IMHA patients will develop an immune-mediated thrombocytopenia (IMT), called Evan's syndrome when IMHA and IMT occur simultaneously. Further diagnostics include infectious disease testing, removal of offending drug regimens, and investigation of possible underlying immune-mediated disease or neoplasia. Typically, tick borne disease titers/PCR, full body radiographs, blood chemistry panel and abdominal ultrasound are indicated. Blood typing an blood for cross-matching will also become quickly necessary as most of these patients require a blood transfusion.

Treatment

Treatment of IMHA includes immunosuppressive therapy, thromboprophylaxis, and blood transfusions. It also includes investigation into the underlying cause and initiating treatment. Immunosuppressive therapy is the hallmark of the treatment of this disease. There are a variety of immunosuppressive drugs that can be used. Typically, glucocorticoid therapy is started initially. Prednisone is administered at immunosuppressive doses to suppress the immune response targeted against red blood cells. These drugs have significant side effects at these doses so gastric ulcer prevention strategies, (gastroprotectants) should be used, and the prednisone will often be tapered to the lowest maximal dose. Other immunosuppressive drugs include: cyclosporine, azathioprine, mycophenolate, and cyclophosphamide.

Blood transfusions will be required to restore effective oxygen carrying capacity. Unfortunately, the patient may accelerate hemolysis by targeting the freshly transfused RBC's with antibodies. It often takes several transfusions, and several days of immunosuppressive therapy to get a handle on the hemolysis.

IMHA patients are hypercoagulable, meaning they have an imbalance in their hemostatic system leaning more towards making clots. These clots, or thrombi, are potentially disastrous as they can lodge in various organ systems (kidneys, heart, brain, lungs) and occlude blood flow causing organ failure. Thromboprophylaxis, or the administration of anti-clotting drugs such as heparins or aspirin, have been indicated in IMHA. Several studies have identified a high prevalence (80% or greater) of

hypercoagulability in IMHA patients. Anticoagulants used in these patients include: aspirin, clopidogrel, unfractionated heparin, and low-molecular weight heparin.

Table 2- Anticoagulant drugs

Drug	Mechanism of action	Administration
Aspirin	As an NSAID it inhibits the formation of Thromboxane-A2- which is responsible for platelet aggregation	PO at low-doses
Clopidogrel (Plavix®)	Inhibits function of the ADP receptor on platelets, inhibiting their binding ability	PO
Unfractionated heparin	Binds to antithrombin and enhances its activity against clotting factors. Can cause bleeding side-effects	SQ, IV- aPTT monitoring must be available. Should administer until aPTT is 1.5-2.5 normal
Low-molecular weight heparin	Similar effects to heparin, but targeted against factor X (which directly converts prothrombin to thrombin). Less side effects than heparin	SQ

Another immunosuppressive drug sometimes discussed with IMHA is the use of human intravenous immunoglobulin (IVIG). This is a transfusion of human IgG immunoglobulins and can be used in a variety of immune-mediated disease. The mechanism of action is not completely understood, but it is thought that the IgG binds to the Fc receptor on macrophages/monocytes preventing them from binding to the receptor on red blood cell antibodies and thereby limiting the destruction of RBCs. There is no definitive evidence to support or restrict the use of IVIG in veterinary medicine; however, IVIG is very expensive and typically reserved for severe refractory cases.

Patients hospitalized for IMHA should receive excellent nursing care including: TLC, husbandry, attempts at nutrition, and avoidance of excessive venipuncture. Vital signs should be monitored regularly, and development of tachypnea should cause the veterinary team to have a high index of suspicion for development of PTE.

When the PCV stabilizes, and these patients have improved mentation and are eating, they may be discharged. Owners may be sent home with a variety of medications, including immunosuppressants, anticoagulants, and maybe disease-specific treatments (antibiotics). The veterinary technician needs to be ready to counsel owners through all of these medications, side effects, and continued monitoring (repeat PCV/CBC's etc).

Conclusion

IMHA can be a devastating disease with a poor outcome. Many patients relapse and have to be re-hospitalized or suffer from thromboembolic complications. However, many patients can survive with long-term immunosuppressive therapy. The veterinary technician needs to be ready to deal with these patients and handle the complications.

References available upon request