2025

OVMA

CONFERENCE & TRADE SHOW

CANADA'S PREMIER VETERINARY CONFERENCE

CONFERENCE PROCEEDINGS

JAN 30 TO FEB 1 2025

The Westin Harbour Castle

TORONTO, ON

ONTARIO VETERINARY MEDICAL ASSOCIATION

#OVMA2025

FOREWORD

Thank you for attending the OVMA Conference and Trade Show! We hope you enjoy our continuing education program and learned new ideas and perspectives on trending topics in the veterinary profession.

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OVMA and its Conference Committee extend a warm thank you to our speakers and sponsors, who have supported the veterinary profession by participating in this year's event. Our sponsors' continued commitment has enabled us to offer you top calibre education at our annual conference.

Thank you again for supporting the OVMA Conference and Trade Show, and the continuing education of veterinary teams.

Heather Fretz, DVM
2025 Conference Chair
Ontario Veterinary Medical Association

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Owner, Philibert Wilson Veterinary Professional Organization

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Amy Thomson, DVM, DAVDC
Head of Dentistry & Oral Surgery, Thomson Veterinary Dentistry - at Lakeshore Animal Health Partners

DERMATOLOGY

Anthony Yu, BSc, DVM, MS, DACVD
Veterinary Allergy Dermatology & Ear Referral (VADER) Clinic

FELINE

Margie Scherk, DVM, DABVP
Director, catsINK

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Michael Lappin, DVM, PhD, DACVIM

The Kenneth W. Smith Professor, Colorado State University

NEUROLOGY

Chris Mariani, DVM, PhD, DACVIM

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David Maggs, BVSc (hons), DACVO, MANZCVS

Professor Emeritus, University of California, Davis

PRACTICE PEARLS

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Professor of Small Animal Surgery, Colorado State University

FOCUS ON INFECTION PROGRAM

Samantha Evans, DVM, PhD, DACVP
Assistant Professor, Colorado State University

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SPORTS MEDICINE AND REHABILITATION

Tena Ursini, DVM, PhD, DACVSMR, CERP
Assistant Professor, University of Tennessee

AILMENTS OF THE YOUNG HORSE

Nathalie Cote, DMV, DVSc, ACVS diplomate
Equine Surgeon, King Animal Hospital/ University of Guelph

PRACTICE MANAGEMENT PROGRAM

SUCCESION

Greg Toner, CPA, CA, TEP, CLU
Owner, Vet CPA Professional Corporation

Darren Osborne, MA

STAFF MANAGEMENT

Andrea Crabtree, BS, CVPM, PHR, SPHR, PHRca, CCFP, FFCP
Veterinary Business Consultant, FurPaws Consulting

CLIENT EXPERIENCE

Alison Lambert, BVSc CMRS
Founder, Onswitch Limited

PLENARY SESSIONS *(PROCEEDINGS ARE OPTIONAL FOR PLENARY SESSIONS)*

Alison Lambert, BVSc CMRS
Founder, Onswitch Limited

Maureen E.C. Anderson, DVM, DVSc, PhD, Dip. ACVIM
J. Scott Weese, DVM, DVSc, Dip. ACVIM

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PSYCHOPHARMACOLOGY: DRUGS, NATURAL SUPPLEMENTS & PHEROMONES

BEHAVIOUR

Colleen Wilson, BSc., DVM

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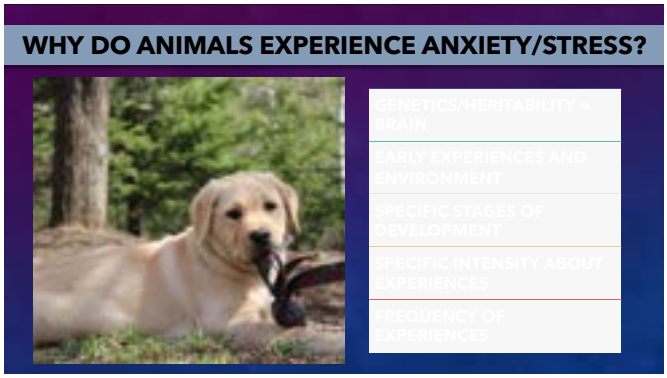

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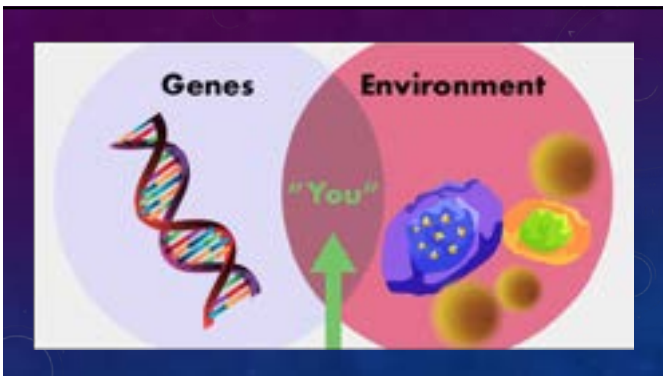

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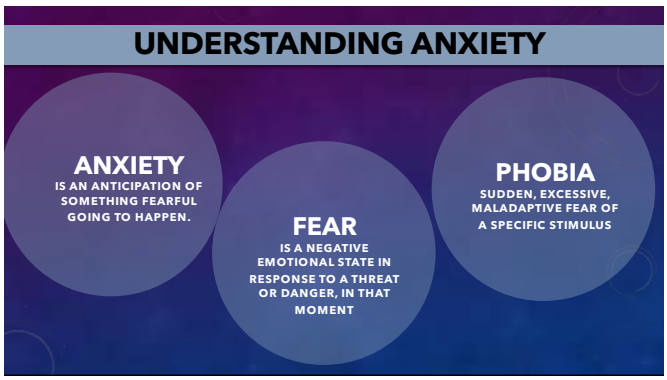

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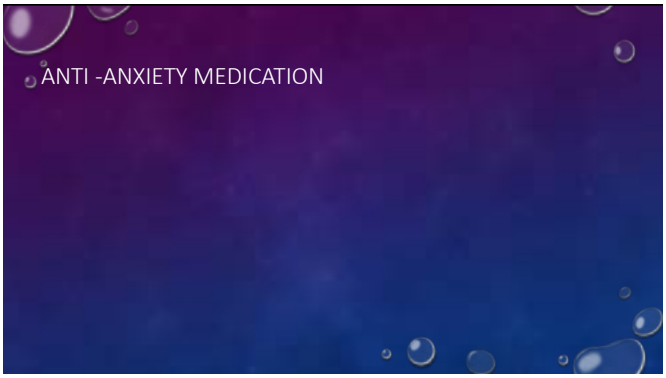

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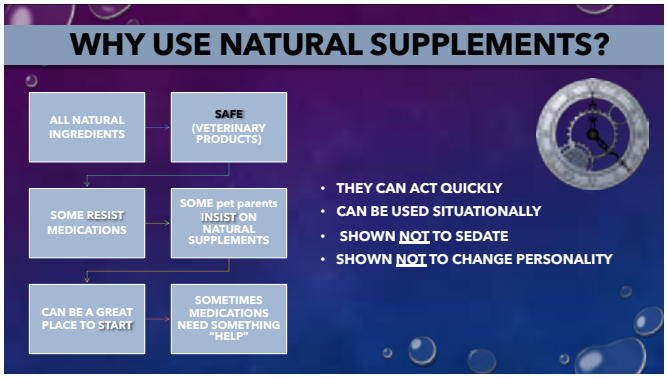

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VALIDATING NUTRACEUTICALS

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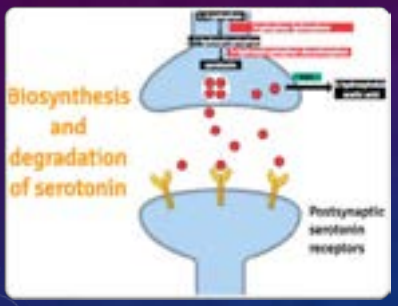

L-TRYPTOPHAN

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L-THEAINE	Tryptophan	MILK proteins
May increase GABA Major inhibitory neurotransmitter May increase serotonin and dopamine	anxiolytic effects Precursor to melatonin and serotonin (5HT) Regulation of mood, aggression, stress	Similar in structure to gamma amino butyric acid (GABA) -when digested affects- the brain's inhibitory neurotransmitter GABA

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NATURAL PRODUCTS

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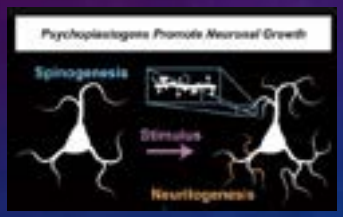
MAKING A CHANGE CAN BE HARD.....



- Anxiety
- Depression
- Compulsive Disorder
- Addictions

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VALIDATING NUTRACEUTICALS

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HUMAN STUDIES

Effects of L-theanine or caffeine intake on changes in blood pressure under physical and psychological stresses

Protective Effects of Milk Casein on the Brain Function and Behavior in a Mouse Model of Chronic Stress

Effects of L-theanine Administration on Stress–Related Symptoms and Cognitive Functions in Healthy Adults: A Randomized Controlled Trial

L-Tryptophan: Basic Metabolic Functions, Behavioral Research and Therapeutic Indications

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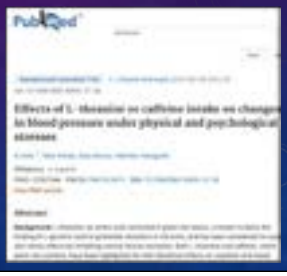
L-THEANINE

Effects of L-theanine or caffeine intake on changes in blood pressure under physical and psychological stresses



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L-THEANINE

Effects of L-theanine Administration on Stress–Related Symptoms and Cognitive Functions in Healthy Adults:
A Randomized Controlled Trial

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MILK CASEIN

Protective Effects of Milk Casein on the Brain Function and Behavior in a Mouse Model of Chronic Stress

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L-TRYPTOPHAN

L-Tryptophan: Basic Metabolic Functions, Behavioral Research and Therapeutic Indications

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MORE NATURAL PRODUCTS

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ANXITANE: L-THEANINE

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VALIDATING NUTRACEUTICALS

STUDY	ACTIVE INGREDIENTS	DESIGN	STUDY TIMEFRAME	RESULTS
Anxitane tablets reduce fear of human beings in laboratory model of anxiety-related behaviour	L-Theanine	Dogs characterized as anxious (N = 10) showed reduced interaction with an unknown human being as compared with normal controls (N = 7). Objective behavioral measures of anxiety were obtained using an open-field test, a human interaction test, and an actiwatch protocol that allowed monitoring of activity over 24-hours.	57-days	The ANXITANE® tablets group had significantly higher levels of human approach.
An open-label prospective study of the use of l-theanine (Anxitane) in storm-sensitive client-owned dogs	L-Theanine	Owners completed a questionnaire for the initial storm. Then, after starting the test article, owners completed the same questionnaire for each of 5 subsequent storms. owners evaluated time to return to the patient's normal baseline behavioral state. Eighteen dogs completed the trial and were available for statistical analysis.	Minimum of 4 weeks, maximum of 8 weeks (subjects experienced >5 storms)	The use of L-theanine decreases the severity of the dog's global anxiety during storms, reduces the time it takes for the dog to return to baseline normal behavior after the storm ends, and diminishes the most commonly reported manifestations of hiding, following people, pacing, panting, and trembling.

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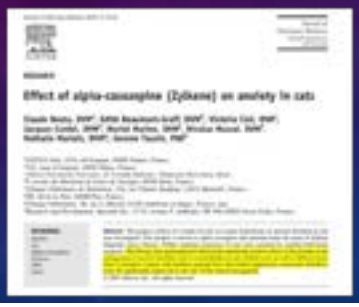
ZYLKENE: ALPHA-CASOZEPINE


Effect of alpha-casozepine (Zylkene) on anxiety in cats

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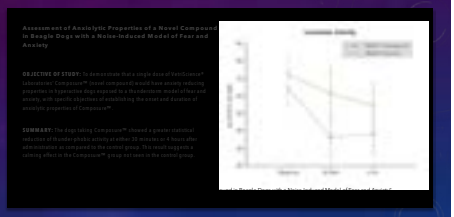
L-THEANINE

TRYPTOPHAN

COLOSTRUM CALMING COMPLEX

THIAMINE



COMPOSURE™ PRO is clinically proven to work **within 30 minutes** and last up to 4 hours

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VALIDATING NUTRACEUTICALS

STUDY	ACTIVE INGREDIENTS	DESIGN	STUDY TIMEFRAME	RESULTS
Effect of alpha-casozepine (Zylkene) on anxiety in cats	tryptic bovine as l-casein hydrolysate	Thirty-four (34) cats were enrolled in and completed the trial. Accordingly, random assignment of cats resulted in 17 cats in each treatment group (placebo and alpha-casozepine)	56-days	This study provides evidence for the efficacy of alpha-casozepine in the management of cats exhibiting anxiety in socially stressful conditions. No differences were seen in aggressive behaviors, but the study was relatively small.
Assessment of Anxiolytic Properties of a Novel Compound in Beagle Dogs with a Noise-Induced Model of Fear and Anxiety	Colostrum Calming Complex® Biopeptide Blend, Thiamine (Vitamin B1) and L-Theanine	Each test dog was placed in the test room. Using software that measures the distance a dog travels around the room, the distance each dog traveled was measured prethunder, and then again for two separate administrations of the thunder track.	30 minutes, up to 4 hours	The group taking Composure™ showed more thunder-phobic activity at baseline than at either 30 minutes or 4 hours after administration.

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CALMING CARE

- PRO BIOTIC
- BL999
- *Bifidobacterium longum*

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ACTIVE INGREDIENTS

COMPOSURE™ PRO	ANXITANE	ZYLKENE	CALMING CARE	SOLLIQUIN
Colostrum Calming Complex™ Suntheanine® L-theanine L-Tryptophan Thiamine (Vitamin B1)	L-Theanine	Alpha S1 tryptic casein	Bifidobacterium longum BL999	L-theanine Magnolia/phellodendron Whey protein
No loading dose	Loading period of **60 days** for dogs and **30 days** for cats	Loading period of **1-2 days** prior to an event, some may need **5-7 days**	May require **6-8 weeks** to see results (clinical trial results based on 6-week administration)	Loading period of **30-45 days**

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PHEROMONES


Effects of dog-appeasing pheromones on anxiety and fear in puppies during training and on long-term socialization

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WHEN REACH FOR A NATURAL SUPPLEMENT?

- EARLIER BETTER
- SITUATIONS THAT ARE NEW
- DAILY IF YOU NEED
- OWNER PREFERENCE
- WITH MEDICATION
- PAIN OR STRESS
- SENIOR PETS

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WHERE?

- VETERINARY VISITS
- CAR RIDES
- NEW PEOPLE AND DOGS IN THE HOME OUT ON WALKS
- UNPREDICTABLE NOISE (THUNDERSTORMS, FIREWORKS)
- BOARDING/OBEDIENCE CLASS
- BEING ALONE

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PSYCHOPHARMACOLOGY: WHEN TO CHOOSE DRUGS?

Getting fearful, stressed and aggressive dogs to you… safely

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PVP's

Trazodone 5-5-10mg/kg

Clonidine 0.01-0.05mg/kg

Gabapentin 10-20 mg/kg

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FEARFUL DOGS

Dexmedetomidine 10-20 ug/kg
Zenalpha (drug chart)

+/- Butorphanol
0.2 – 0.4mg/kg IM

+/- Ketamine
2-5 mg/kg IM

+/- PANIC?

Midazolam 0.3 mg/kg

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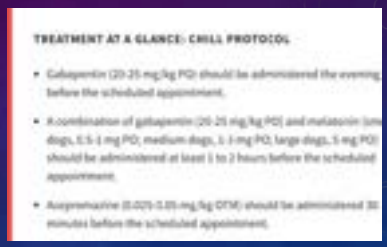
CHILL PROTOCOL



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THE BEHAVIOUR VET

DR Colleen WILSON

BSc., DVM, Dipl. ACVB

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FELINE ENRICHMENT: TREATING FELINE INAPPROPRIATE ELIMINATION AND INTERACT AGGRESSION

BEHAVIOUR

Colleen Wilson, BSc., DVM

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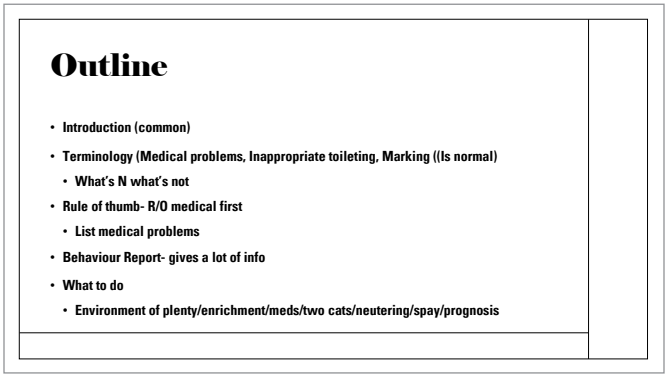

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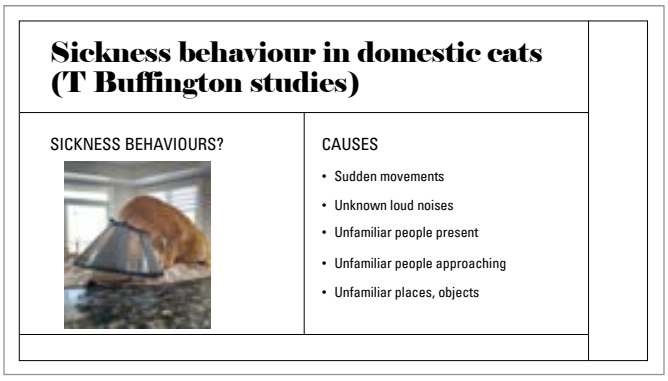

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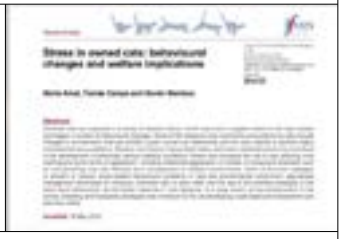
STRESS



7

Common:

1. Patroneck GL, Glickman LT, Beck AM, et al. Risk factors for relinquishment of cats to an animal shelter. J Am Vet Med Assoc 1996;209(3):582–8.
2. Kogan L, New JG Jr, Kass PH, et al. Reasons for relinquishment of dogs and cats to 12 shelters. J Appl Anim Welf Sci 2000;3(2):93–106.
3. Scarlett JM, Salman MD, New JG Jr, et al. Behavioral reasons for relinquishment of companion animals in U.S. Animal Shelters: selected health and personal is- sues. J Appl Anim Welf Sci 1999;2(1):41–57.
4. Clifton M. Counts finds 5 million a year – AHA says 12 million. Animal People 1993;1:8.

8

Clinical evaluation of multimodal environmental modification (MEMO) in the management of cats with idiopathic cystitis

- T. Buffington, J. Fel Med and Surg., 2006
- Client-reported recurrence of LUTS
  - Hematuria, dysuria, pollakiuria and urinating inappropriately in the owners home
  - Diagnosed with LUTS based on absence of uroliths, bacterial infection
- Added to usual care: MEMO
- After 10 months clients reported**" significant reduction in LUTS, fearfulness, nervousness, signs referable to lower intestinal tract (P<0.05), and a trend toward lower aggression (P<0.01)**

9

Conclusion: "MEMO resulted in significant improvement of LUTS in cats with Idiopathic cystitis, as well as improvement in other organ systems"

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MARKING

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BEHAVIOUR CARDINAL RULE:

RULE OUT MEDICAL FIRST

- A comprehensive **physical exam**
- Any medical problems such as urinary tract disorders or diseases causing PU/PD

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Typical PRIMARY workup

YOUNG CATS

- **PHYSICAL EXAM**
- **URINE TEST**
- **IMAGING (RADS)**
  - **Cystitis**
  - **Urolithiasis**
  - **crystalluria**

GERIATRIC CATS

- **PHYSICAL EXAM (arthritis)**
- **URINE TEST**
- **IMAGING**
- **Concerns?**
  - **Add CBC/chem/thyroid**
  - **Organ failure?**
  - **Neoplasia**
  - **Metabolic disease**

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DISEASE?

- Can contribute to not using the litter and then an **AVERSION** develops
- LOWER URINARY TRACT DISEASE
  - FLUTD, CYSTITIS, FIC
- ANY **PU/PD**
  - DIABETES, RENAL DISEASE, OTHER
- ANY **PAIN**?
  - ARTHRITIS, CONSTIPATION

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Environmental NEEDS

Safe Place

- Importance of Cat sense of Smell
- Positive Predictable human interaction

Resources of Plenty

- Food
- Litter pans
- Scratching
- Play
- Resting/sleeping areas

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What is environmental enrichment ?

- **Quality and quantity of space**
- **Free roaming cats, compare**
- **Cats should have at least 2 rooms to access**
- **Vertical dimensions**
- **Interactive toys**

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Dr. Wilson et. al.

Cat Scratching research

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A WORD... or two... on Punishment

- "Pets trained with **punishment** based techniques, especially if they are inconsistent and poorly timed, **are ineffective and result in the pet exhibiting higher levels of anxiety, fear and stress"** (Schilder 2004, Schalke 2005)
- It may worsen the problem
- It decreases the human animal bond
- **SOLUTION:** predictable routines and interactions to reduce stress.

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THE ULTIMATE LITTER BOX

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LOCATION

Box outside the core area	Away from physical challenges	Away from noisy locations	Away from dark areas
Multiple cats = multiple boxes= **multiple locations**	Multiple levels? One on each level	Avoid dead ends/corners where a trap	Multiple **LOCATIONS**

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COMFORT

- What is available today is too small
  - Recc: **1 ½ length of the cat...tip to tip!**
- Consider older cats/overweight cats/arthritis
- Needs
  - To move around
  - squat
- Covered Boxes
  - Trap odours
  - Trap the cat (other cats/dogs/kids)
  - Hard to move around

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LITTER BOX MANAGEMENT...what can we do?

- LOTS:
- "Causes of urine marking in cats and effects of **environmental management** on frequency of marking"
  - Pryor et al, JAVMA, 2001
  - Results: Male cats and cats from multicat households more likely to mark
  - Also: Attention to environmental and litter box **hygiene can reduce marking frequency in cats regardless of sex or household status**.

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OTHER SITES....what can we do?

Avoid conflict
- Prevent, confinement
- Counter condition to resolve conflicts (this is another lecture!)

Avoid areas
- Block access/Booby traps?..they may just move their target
- Change the function of the area (feeding, scratching, sleeping, etc)
- Would you allow a site or two for spraying?

Clean
- THE ULTIMATE LITTER BOX....

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Anti-anxiety medications

Fluoxetine	Paroxetine
• **Selective serotonin reuptake inhibitor**	• SSRI
• Dose: 0.5mg/kg SID	• Sister drug to fluoxetine
• Side effects: Anorexia, lethargy	• Less serotonergic
• Double blinded placebo trial, Pryor et al	• Tablets!
• Findings: **90% reduction** in spraying after 8 weeks of 1.0 mg/kg (2 cats had 1.5mg/kg)	• 0.5mg/kg SID in the morning

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Fluoxetine vs. Clomipramine

Study by Hart et. al., "**Control of urine marking by use of long-term treatment with fluoxetine or clomipramine in cats**", JAVMA, 2005
(22 neutered cats)

Fluoxetine 1mg/kg Q 24hrs

Clomipramine 0.5mg/kg Q 24 hrs

Results: Equally effective, longer treatment had increased efficacy, returned to clinical sings with abrupt withdrawal, second course just as effective

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Clomipramine:

- Tricyclic anti-depressant
- **Inhibit serotonin and norepinephrine reuptake**
- **Dose: 0.25-0.5mg/kg SID**
- Side effects: sedation
- Study by Landsberg Wilson JAAHA, 2005
- 20/25 cats had 75% decrease by 4 weeks, 90% decrease in marking or resolved in total of 17 of the cats

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OTHERS

- **BUSPIRONE**
  - **also increases serotonin, pre and post blocking**
  - Hart et al, JAVMA 1993
  - 55% reduction in spraying, 33% resolved, 50% recur
- **DIAZEPAM- ataxia**
  - **Facilitate GABA**
  - Marder, Vet Clinics of North America, 1991
  - Up to 75% reduction, 43% resolved, 75% recur

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Others

- **Cyproheptadine**
  - Study Kroll et al,Congress Vet. Beh., 2001
  - Clomipramine found to be better
- **Progestins**
  - Have been shown to improve marking 30%
  - SIDE EFFECT: immunosuppression , breast tumours high
- **Feliway**
  - Study Frank J Appl. An Beh Sc., 1999 and Study Ogata J. Vet. Med, 2001
  - **Feliway diffusers have proven effective in reducing marking in 47%-97% of cats**

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I.D. the Culprit

- ISOLATE
- Urination
  - Give cats 10 mg of fluorescein dye orally and for 24 hours you can identify a bright yellow green color with fluorescent black light
  - May stain
- Defecation
  - Moist food containing colored non-toxic crayons
  - A different color for each cat s

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PROGNOSIS

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Inter cat aggression

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- Most commonly due to:
- Social stress
  - Vital resources (competition#), retreats (hiding is coping), new cat
- Fear and anxiety
  - Redirected
  - Core territory
- Frustration….somebody new!!
- Inappropriate play
  - Kittens 3-12 weeks, 14 weeks aggressive play

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Cornerstone of treatment:

- Safety, avoidance
- Separation of the aggressive cat and the "victim"
- Establishing outlets for normal behaviour
- Coping mechanisms (flight/hide)
- Behaviour modification
- Environmental enrichment
- Medication and/or pheromone therapy

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treatment:

- Social stress
  - TX: core territory, number of cats, litter, food , water, perches, 3-d needs
- Fear and anxiety
- Frustration
- Inappropriate play
  - Age of cat, normal predatory outlets/misdirected, specific rod type toys, reward appropriate play, food puzzle toys

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Predatory Play

- Simple often best
- Un-predictable, rapid, high pitched sound ideal

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Perches and places to hide

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Counter conditioning and Desensitization

To fear inducing stimuli

Establish an incompatible reaction

Controlled exposure

One sense at a time

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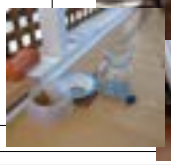
Social stress/frustration treatment:

Number of cats

- Litter
- Food
- Water
- Perches
- Hiding places



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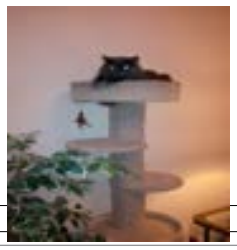
Perches and places to hide



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Normal cat play

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CASE example:

MOXIE AND DUSTER

-both 4.5 yrs old

-adopted together at 10 weeks

-no issues until 3 yrs

-after viewing strange cats outside, Moxie attacked Duster no and then over last year

-getting worse: Each time they saw one another, Duster loosing bladder control

-Duster described as "shy"

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Moggie and Duster con't:

- R/O
- Re-directed aggression
  - Most likely
- Fear aggression
  - A contributing factor still
- Social conflict aggression
  - Plenty of resources
- Play aggression
  - Claws not retracted
- Learned aggression from underlying medical conditions
  - CBC/CHEM12/T4/FELV/FIV test WNL

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Period of avoidance:

- Started **both** cats on 0.5mg/kg fluoxetine once daily
- Restricted access
- Food, water, perch, toys, litter pan, FELIWAY diffuser, daily interaction
- Daily interaction, teach "come"
- 100% monitoring..then they could begin gradual reintroduction process

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Systematic Desensitization and counter conditioning

-Controlled and gradual interactions with food

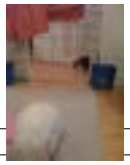
-monitoring for signs of arousal

-dilated pupils, twitching/ fluffed tail, ears back, stiff body posture, vocalization

-allogrooming

44

Controlled and gradual interactions with FOOD



45

THE END

What a lie!

46

Moxie and Duster: end

THE END!

-people go too fast

-signs of arousal

-wean meds or remove Feliway first

47

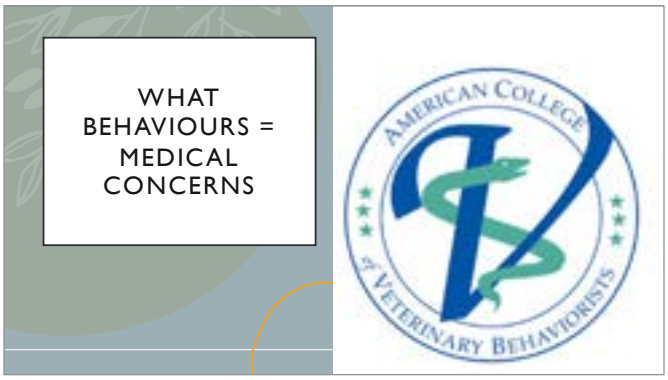
1003

5 COMMON BEHAVIOUR COMPLAINTS MASQUERADING AS MEDICAL ISSUES

BEHAVIOUR

Colleen Wilson, BSc., DVM

1



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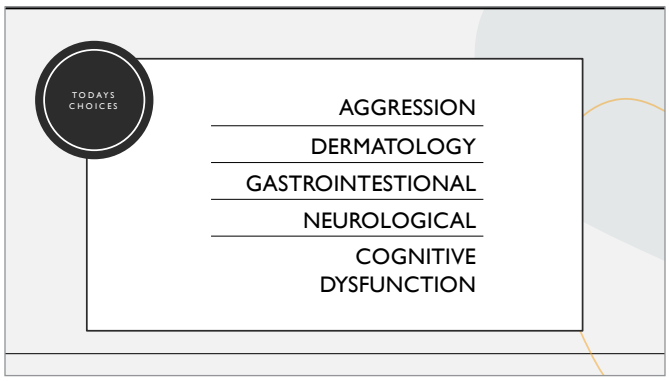

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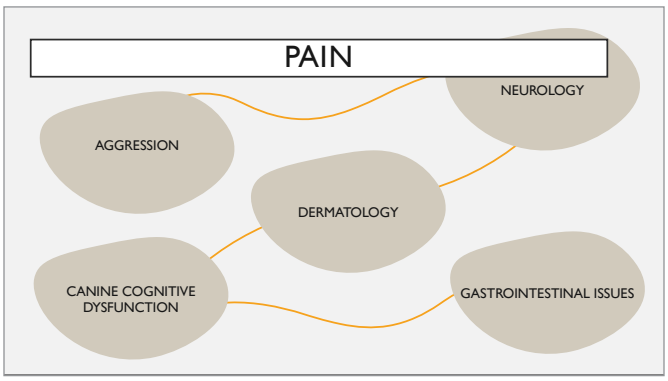

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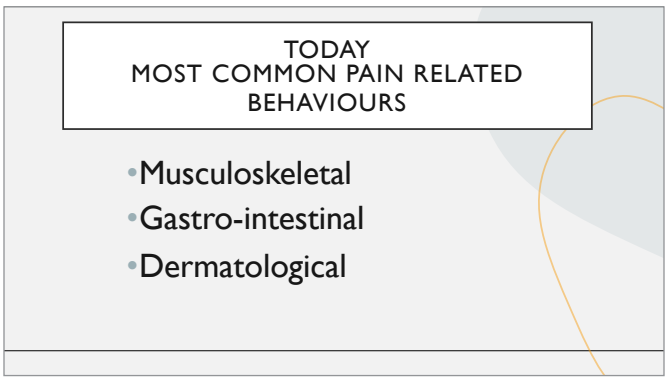

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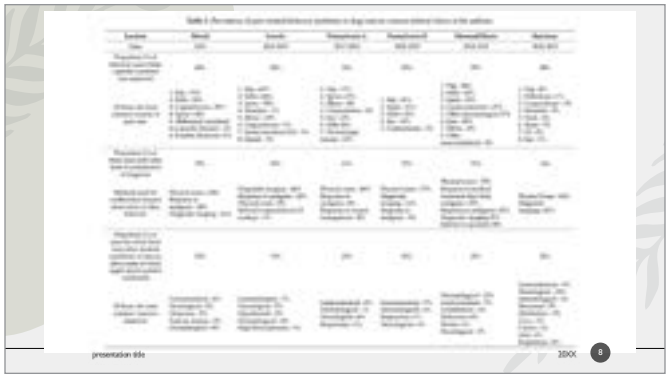

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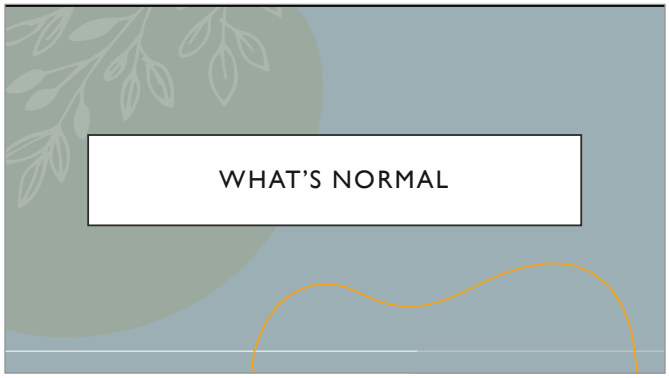

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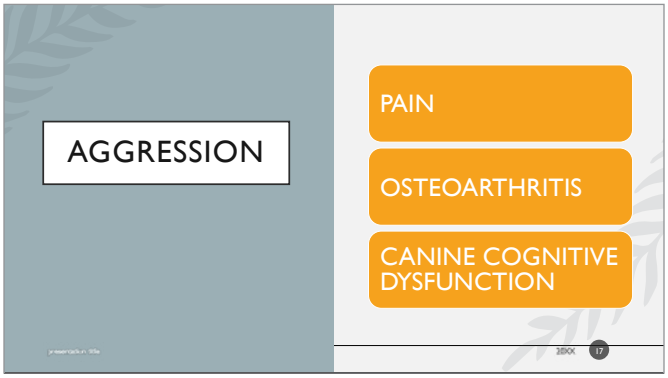

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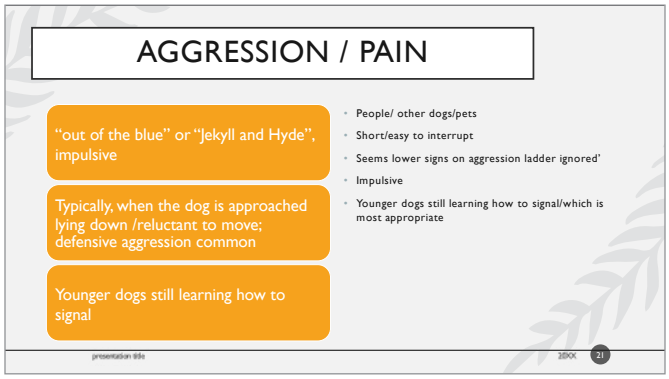

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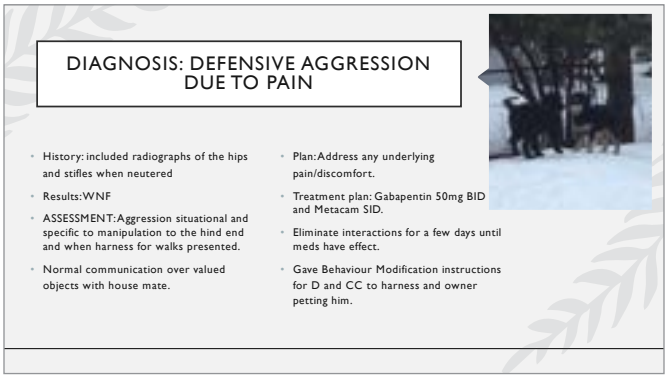


14

PRECAUTIONARY PRINCIPLE

15

TWO WEEK FOLLOW UP PHONE CALL:

Owner reports: Oakley initiates interactions with owners and the other dog now,

Plays with toys and doesn't growl when they are near picking up the toys

Reverts to some "grumpy" behaviour in the evening after playing all day.

- ONGOING PLAN:
  - Disc exercise needs and limits
  - IF this doesn't change the behaviour add more pain control.
  - Disc referral recc to Neurologist for further assessment.

16

FELINE DEFENSIVE/OFFENSIVE/ANXIETY

17

SENIOR PET PROBLEMS/ PAIN

- Dogs
- Aggression
- Separation Anxiety
- Inappropriate Elimination
- Excessive vocalization
- Phobias and anxiety
- Night waking
- Compulsive Disorders

- Cats
- House soiling
- Aggression
- Vocalization/restlessness/night waking
- Excessive grooming

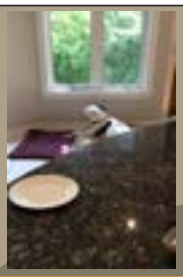
presentation title 36

18

PRECAUTIONARY PRINCIPLE

19

SENIOR PET PROBLEMS



presentation title/sub 38

20

DISHA

Interaction

Sleep wake cycle

21

DISHA

H= House soiling

22

A= ACTIVITY

23

ACTIVITY

NIGHT RESTLESSNESS

R/O PAIN

24

GASTROINTESTINAL DISEASE SYSTEM

25

GASTROINTESTINAL DISEASE

26

NOT NORMAL BEHAVIOUR.....

19 dogs with ELS, 10 controls.

GPE, neuro, videos. Monitored 90 days.

GI abnorm in 14/19; improvement in 10/17

27

NORMAL

28

NORMAL VS NOT

- INTENSITY

29

"FINDLEY"

-4 year old, M/N, Airedale Terrier

-Aggression mainly directed towards one owner

-Multiple bites and holding on

-Licks the couch

-eats hypo diet and on Atopica

-Owners report "bunny hop" and runs with both hind legs together

-frequent burping/licking and smacking lips and licking of inanimate objects frequently

30

CLINICAL OBSERVATIONS AND DIAGNOSIS:

- **Impressions:**
  - Findley is able and prefers to settle on his own in the house, does not want to interact, displays frequent lip licking, lip smacking and burping
  - NOT NORMAL
- **DX:**
  - Active Defensive Aggression, primarily directed towards one owner generalizing to others
  - Nausea
  - Discomfort /pain in the hind end

31

START FROM THE BOTTOM

32

PRECAUTIONARY PRINCIPLE

33

TREATMENT RECC:

- Address underlying medical issues
- PE and radiographs of hind end under sedation
- Blood work: CBC, CHEM panel including T4
- Fecal tests (giardia antigen test and complete…ect ect….

34

INGESTING FOREIGN OBJECTS = NOT NORMAL

-rocks
-Sticks
-toys/toy stuffing
-plastic toys, balls
-clothes (socks, underwear,…..)

35

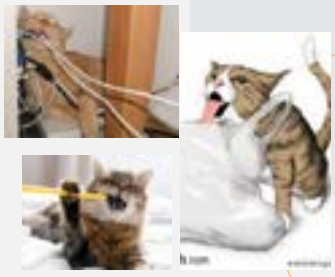
36

- Normal behaviours for cats do not include ingesting, chewing and sucking on inanimate objects, frequently, routinely or excessively.

37

PICA: CATS EATING, SWALLOWING AND CHEWING NON-FOOD ITEMS

- "Pica does not seem to be due to suboptimal environment or early weaning"
- "Frequently vomiting"



38

PRECAUTIONARY PRINCIPLE

39

GI MERGING WITH NEURO?

Article

Prospective medical evaluation of 7 dogs presented with fly biting

presentation title

60

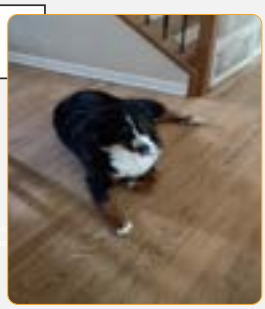
40

"MAISY" HAYLEY

- "Maisy"
- 4 year old, F/S Bernese Mountain Dog
- Primary complaint: Fly Snapping
- Hx includes: increased intensity of fly snapping events, use of punitive/balanced trainer/uses ultrasonic training.
- Referral to Neurologist: Neuro referral

41

- Star gazing
- Fly snapping
- Compulsive chewing
- OUTCOME: Disc with Neuro about phenobarbital, progress



42

TREATMENT FOR MAISY:

- Limited ingredient diet
- Gabapentin three times daily
- If owner misses a dose fly biting Maisy starts fly biting approx. 45 min later
- Owners report 90% improvement with gabapentin treatment
- Didn't like the 3 times a day routine
- Added behaviour modification - eliminate punishment, teach her behaviours you prefer

43

GASTROINTESTINAL DISEASE AND BEHAVIOUR IN PEOPLE

- Higher prevalence of psychiatric disorders (anxiety and depression): Crohn's disease and Ulcerative Colitis.
- **Stress (acute or chronic):**
  - Enhances intestinal permeability
  - Weakens tight junctions
  - Increases microbial load in the colonic tissue.

J Pyschosom Res 2016 Aug; 87:70-80. doi: 10.1016/j.jpsychores.2016.06.001

Curr Neuropharmacol 2016;14(8):892-900. doi: 10.2174/1570159x14666160404124127.

CW

44

DERMATOLOGY

45

PRECAUTIONARY PRINCIPLE

46

SX REPAIR OF TAIL/NOT TAIL:

47

SEGWAY…..

ABNORMAL ORAL BEHAVIOURS

GASTROINTESTINAL DISEASES

NEUROLOGICAL DISEASES

48

BEHAVIOURS THAT INDICATE NEUROLOGICAL ISSUES

And perhaps GI issues…..

49

NEUROLOGICAL

- Head scratching

50

JAJA: REALLY NOT A LOT NORMAL…..

51

JAJA'S TREATMENT INCLUDES…(BUT IS NEVER ENDING AND SEEMINGLY GOES ON FOREVER…..)

- Hydrolyzed protein Diet
- 20 mg Omeprazole BID
- Sx repair/removal of "cork screw tail"
- Antibacterial facial wipes
- Sx repair of entropion
- Daily Optimmune
- Daily Face cleaning
- Plan: needs dental next
- Trial of NSAIDS

52

SUMMARY

53

DISCLAIMER/ (DISCLOSURE?)

- DON'T GET A BULL DOG

54

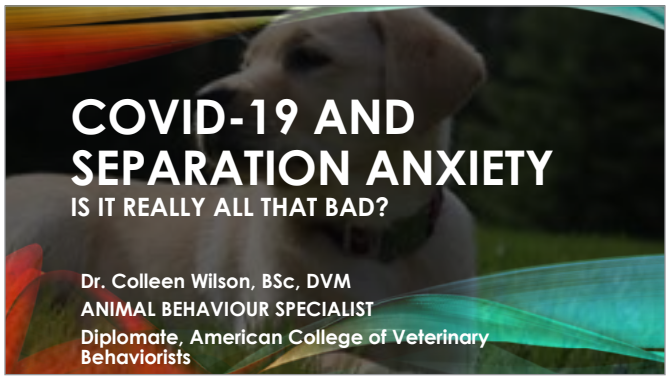
55

1004

DISTRESS, STRESS, UNTIDINESS – HELP!

BEHAVIOUR

Colleen Wilson, BSc., DVM



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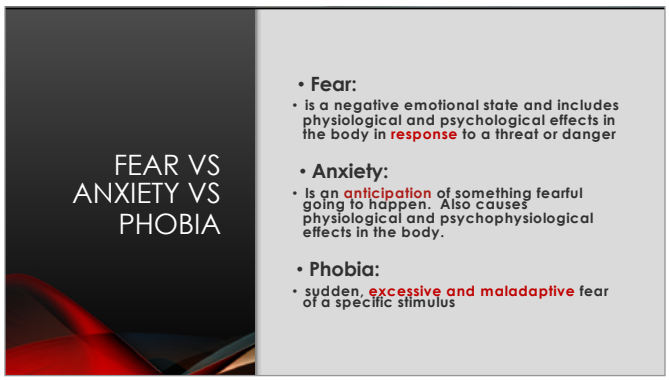

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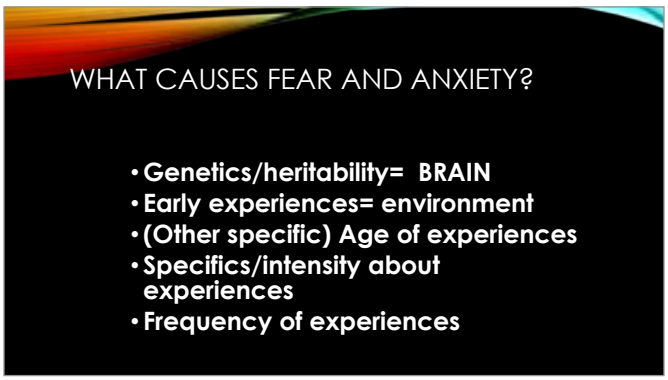

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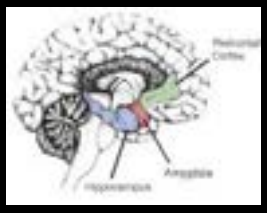
WHY IS THIS IMPORTANT?



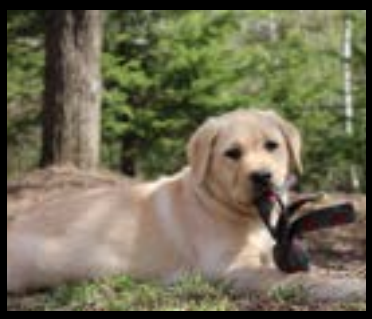
- Cortex
  - **Thinking brain**
- Amygdala
  - **Emotional brain**

7

DEVELOPMENT IS CRITICAL

- **Socialization: 4- 14 weeks**
- Fear responses strong up to socialization
- Little contact before socialization leads to a markedly disrupted brain- not normal

  Isolation, even partial, for several weeks for the first year reduces sociability and increases fearfulness



8

NATURE AND NUTURE

- We can't eliminate everything
- about a dog

9

I think we are forgetting how medical issues happen to each individual

10

FACTS

BEFORE COVID-19

- Any canine could be genetically available for anxiety
- Trauma/excessive stress can ignite anxiety genes
- Owners often get a dog and need to leave ….next day/a few days later/maybe a week or two later= trauma

During COVID-19

- Any canine could be genetically available for anxiety
- Trauma/excessive stress can ignite anxiety genes
- Owners are home much of the time= bonding with the pet/pup=better coping skills=increased resilience= able to teach independence

11

MORE FACTS

Before COVID-19

- Socialize! Socialize! Socialize!!!

During Covid-19

- Predictable controlled exposure to novel things/people (maybe not places but….*)

12

SOCIALIZATION VS SENSITIZATION

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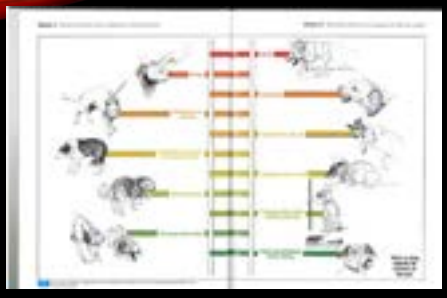

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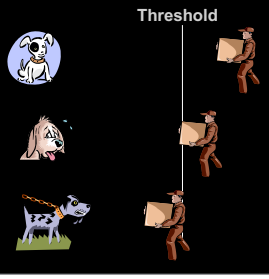
Learning Principles – The Threshold



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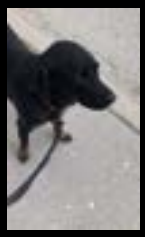
SOCIALIZE…SOCIALIZE…SOCIALIZE



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MORE FACTS

Before COVID-19

- Socialize! Socialize! Socialize!!!
- Typically, little time to teach independence in a tangible way
- Lower perception of dogs suffering SA
- Can participate in puppy class/training classes
- Not every dog suffers SA

During Covid-19

- Predictable controlled exposure to novel things/people (maybe not places but….*)
- Opportunity to teach independence and be successful
- Increase in owners noticing SA (home more/able to notice, more dogs)
- Cannot participate in puppy class/training classes
- Not every dog suffers SA

17

FACTS DURING COVID FOR DOGS…AND PUPPY'S

- What owners perceive will happen:
- What is happening is more fluid with small and/or fewer changes less often
- Professions/types of work….
- Many working 100% from home, yes!

18

THIS ISN"T ALWAYS HAPPENING…..BUT WHAT DO WE DO WHEN IT DOES?

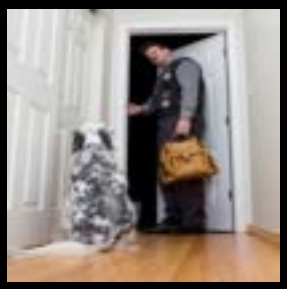
19

SEPARATION ANXIETY

= Distress when separated from THE OWNER and can't relax when home alone

= Inability to COPE without their person(s)

=because of Anxiety/Fear



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WHAT WE KNOW:

- WE DON'T KNOW WHERE SA COMES FROM
- WHAT WE DO KNOW: CANINES ARE SOCIAL ANIMALS, SEPERATION FROM THEIR FAMILES CAN CAUSE DISTRESS
- LIKE PEOPLE/CHILDREN- DOGS ARE BORN WITH GENETIC TENDENCIES TO BE ANXIOUS OR TO HAVE MORE RESOLVE
- DEPENDS ON WHERE YOU CAME FROM –HEREDITORY FACTORS AND WHAT YOU EXPERIENCED, MOST IMPORTANTLY IN SPECIFIC STAGES OF DEVELOPMENT

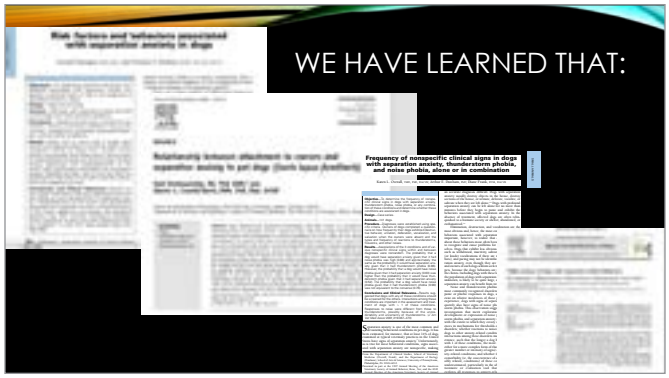
21

WHO?

- **BREED?**
- **Any dog breed**
- **GENDER?**
- **Any sex (maybe more males)**
- **AGE?**
- **Any age: Usually before 3 yrs, also geriatric pets**
- Ref: SA: Canine from Blackwell's Five Min Consult, Canine and Feline Behavior, 2nd Ed.)

22

WE HAVE LEARNED THAT:



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ASSOCIATIONS WE KNOW:

- EXCESSIVE VOCALIZATION
- HYPERSALIVATION
- INAPPROPRIATE ELIMINATION
- DESTRUCTIVE BEHAVIOURS/ESCAPE
- REPETITIVE BEHAVIOUR (PACING)
- ANOREXIA
- EXCESSIVE FOLLOWING
- WITHDRAWL/INACTIVITY
- SHAKING TREMBLING
- AVOIDING THE OWNER
- AGGRESSION
- EXCESSIVE GREETING BEHAVIOURS
- DOGS FROM SHELTERS/RESCUES/VET CLINICS/FOUND
- SINGLE OWNERS
- NOISE SENSITIVITY

24

THINGS TO WATCH FOR:

- Is your dog ALWAYS with you or someone when home
- Destruction: doorways, windows, exit sites
- Exaggerated greetings?
- Age:4-5 years old
- Aggressive at the door? Prevent you from leaving?
- Fear of noises
- Adopted from a rescue organization/history of multiple homes

25

MYTHS…WHAT'S NOT HAPPENING:

- Vengeance
  - This is about distress
- Spite
  - Dog brains don't have this ability
- "my dog is mad at me"/angry
  - this is about distress
- My dog is punishing me
  - New boyfriend/girlfriend/schedule
- He loves me too much
  - Love is not pathological
- I spoil my dog
- He needs a companion
  - Hyper attachment to the person
- He's bored
  - Signs occur within 10-20 minutes of departure

26

GUILTY LOOK:

- " he knows he shouldn't poop on the floor
- "He looks guilty"
- " after he comes to me to say sorry"

27

THE "GUILTY LOOK"

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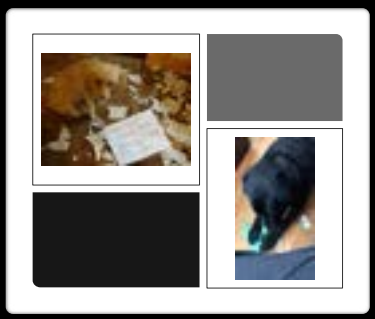

NOTICE THE DIFFERENCE IN BEHAVIOUR

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"PARKER" GOTH, MIXED BREED LARGE DOG, 1 YR M/N

- **August 27, 2019**
- Hx: Anxiety in crate, when left alone: excessive salivation, chipped teeth, blood found in the crate, breaking out of the crate, previous training with punitive trainer
- Clinical Impression: In GGH, mildly reactive initially, able to settle on his own
- R/O separation anxiety barrier frustration, inadequate mental enrichment

31

DIFFERENTIAL DIAGNOSIS:

- Generalized anxiety only
- Noise phobia (thunderstorm, fireworks, other)
- Destructive behaviour (adolescence)
- House soiling
- Territorial aggression
- Compulsive Disorder
- Cognitive Dysfunction

32

TREATMENT PLAN:

Is also: PREVENTION PLANNING

33

TREATMENT PLAN:

- Treatment Recc:
- 1. confirm intensity of anxiety via video/audio analysis
- 2. Until departure medications established, best not to be alone
- Eliminate crate confinement
- Discontinue recc given by trainer who recc punishment-based training
- Everyone that interacts in the same way- day care, home, other

34

BEST CASE SCENARIO FOR INITIAL TX:

Never leave the dog alone until some independence is learned

- Take your dog with you to work
- Kennels
- Doggy day cares
- Relative, friend or neighbor
- Cars= you're coming back= constant

35

TREATMENT PLAN

Independence training

- -food puzzle toy
- -Special bone/treat
- 15-20 minutes is ideal
- Establish this focused behaviour first
- Calming "Q"
- Then: systematic desensitization training and counter conditioning

36

USUALLY HOW IT GOES:

- 1-3 MINUTES RELAXATION IS THE START OF THE TRAINING
- 15-20 MINUTES AND /OR NEAR DOORWAYS HARD PART
- ONE YOU GET PAST 20 – 30 MINUTES IT IS MORE SRAIGHT FORWARD TO ADD LARGER TIMES ALONE (5-10 MIN).
- ONCE IT GETS TO 1-2 HOURS- TYPICALLY OK FOR A WORKDAY DEPARTURE

by Unknown Author is licensed under

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34

ADDITIONAL HELP:

Medication

- Anti-anxiety medication
- Not sedatives
- Not gravol if your dog IS anxious

Natural products

- Adaptil pheromones
- Zylkene
- Anxitane
- Calm diet

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PREVENTION PLAN

- Address physical and social needs – age dependent
- Teach independence
- When home also ignore them sometimes (nap/shower/laundry/cooking)
- Constant attention is what makes it harder for them when you leave
- Schedule alone time- age dependent
- Every single time you leave –offer a food puzzle toy/lasting treat
- Newly adopted, puppies consider ADAPTIL collar, diffuser
- Arrivals and departures calm- uneventful
- Fearfreepets.com :Debbie Martin LVT, VTS Behavior Dr. Ken Martin DACVB

40

EXCELLENT RESOURCES:

Blackwell's Five-Minute Veterinary Consult Canine and Feline Behavior, 2nd Ed.

41

THANK YOU!

Dr. Colleen Wilson, Bsc., DVM, ANIMAL BEHAVIOUR SPECIALIST
DIPLOMATE OF THE AMERICAN COLLEGE OF VETERINARY BEHAVIOR

42

43

- Does this happen to your dog?
- If you don't help the dog, he gets better and better and this undesirable behaviour
- Teach him what you want him to know
- Not so easy

44

1005

WHAT NEVER TO DO WHEN DEALING WITH AN AGGRESSIVE DOG

BEHAVIOUR

Colleen Wilson, BSc., DVM

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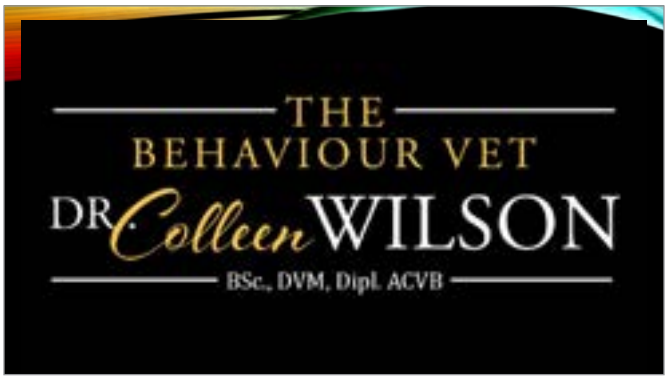

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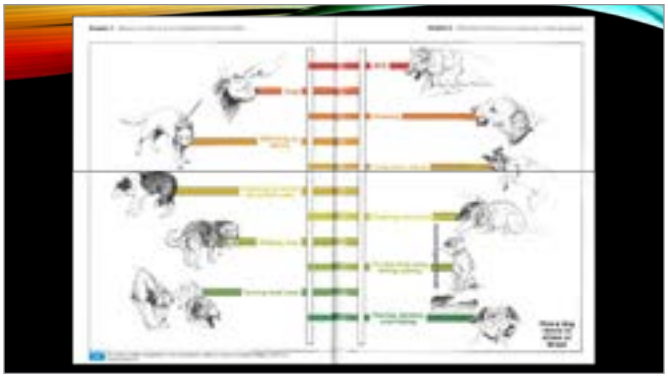

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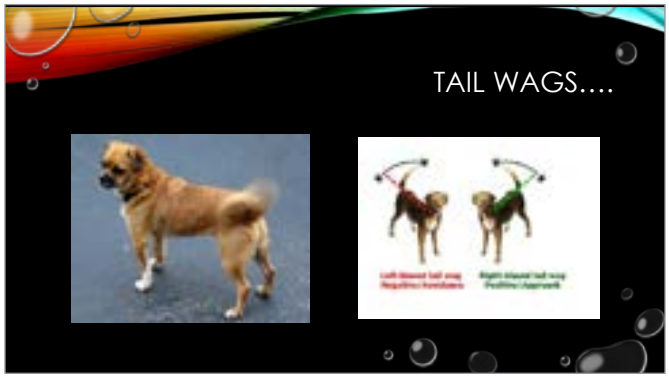

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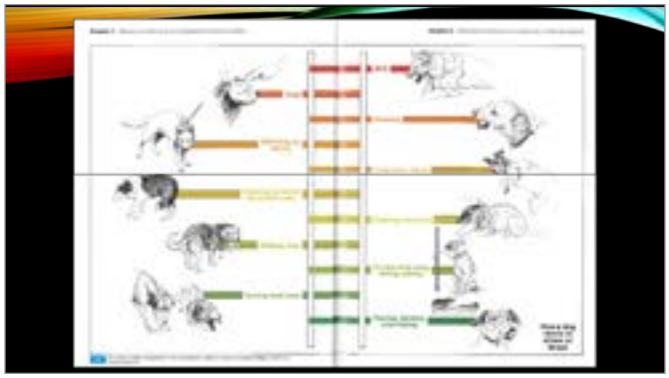

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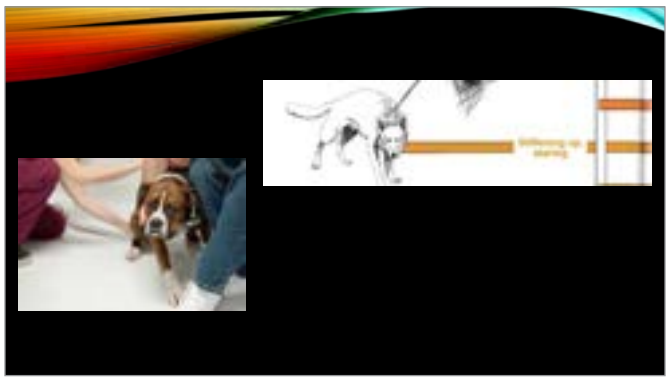

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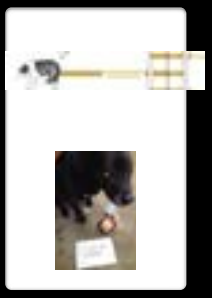
BITE.....

23

THE GUILTY LOOK

- FEAR



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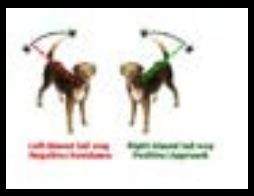
DISTANCE INCREASING BEHAVIOUR

25

THIS……AND THIS

26

TAIL WAGS....



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WHO IS AT MOST RISK MOST OFTEN?

28

29

BREED RESPONSIBILITY?

•NOPE

30

HOW DOES IT HAPPEN?

In the first place……

31

WHAT CAUSES FEAR AND ANXIETY?

- **Genetics/heritability= BRAIN**
- **Early experiences**
- **(Other specific) Age of experiences**
- **Specifics about experiences**
- **Frequency of experiences**

32

THIS IS WHY DEVELOPMENT IS CRITICAL

- Sensitive stages:
  - Natal/ in utero
  - Neonatal period (1-2 weeks old)
  - Transitional period (3 weeks old)
  - Socialization period (4- 14/16 weeks old)
  - Juvenile period (to sexual maturity)

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THIS IS WHY DEVELOPMENT IS CRITICAL

- **Socialization:**
  - 8 weeks play begins, avoidance and fear reactions seen
  - Fear responses strong up to socialization
    - Isolation, even partial, for several weeks for the first year reduces sociability and increases fearfulness

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TRAINING METHODS

How the pet Is treated

37

38

WHAT IS CONSIDERED PUNISHMENT?

Positive punishment/aversive applied

- Prong collars
- Choke collars
- Shock collars
- Harsh verbal reprimands
- Smacking
- Muzzle grasps
- Collar grabs
- pinning

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WHEN WE APPLY POSITIVE PUNISHMENT...

- Increase in dog's **fear**
- Increase in dog's **anxiety**
- Increase in dog's **stress**
- Increase in **aggressive behaviour**
- Increases **bites...**
- **Decreases human animal bond**
- **Special note:** in the wrong pet, at the wrong time, it's detrimental

40

FIRST THINGS TO DO:

- Proximal **A**ntecedents (eg, people within 6 feet of the dog and/or reaching toward her)
- Problem **B**ehaviour (eg, growling and lunging at people)
- **C**onsequence that reinforces the behaviour (eg, the person withdraws, owner pets dog)

41

DESENSITIZE AND COUNTER-CONDITION

42

MEDICATION

- **Selective Serotonin Reuptake Inhibitors (SSRI's)**
- :
- Fluoxetine 0.5-2.0mg/kg SID
- Paroxetine 0.5-2.0mg/kg SID

- **Adjunctive medications:**
  - Trazodone 3-5mg/kg Q 8-12 hrs
  - Clonidine 0.01-0.05mg/kg Q12hrs

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NATURAL SUPPLEMENTS

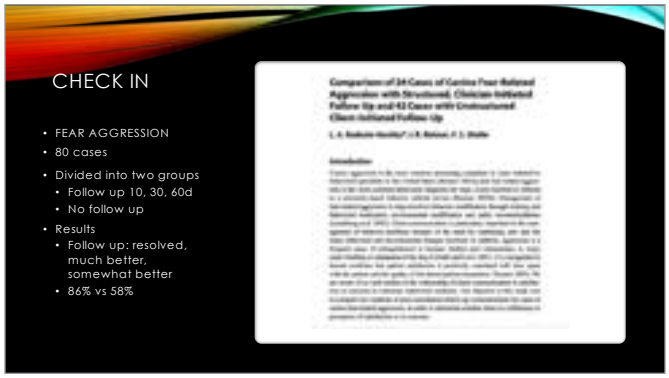
- Anxitane:
  Active ingredient L-theanine
- Zylkene:
  - Alpha casozepine
- Composure Pro
  - added tryptophan
    - Colostrum calming complex

45

PROGNOSIS

- Impulsive
- Unpredictable
- Chronicity of problem
- Punishment /corrective training history
  - Especially when inconsistent
- Inconsistent interactions with people

46


CHECK IN
• FEAR AGGRESSION
• 80 cases
• Divided into two groups
• Follow up 10, 30, 60d
• No follow up
• Results
• Follow up: resolved, much better, somewhat better
• 86% vs 58%

47


TREATMENT SUMMARY:
• no punitive punishment
• Prevent
• Medication / Natural supplements
• Teach what you want them to know
• Re-checks

48


AMERICAN COLLEGE
of VETERINARY BEHAVIORISTS

49

2001

PERIDONTAL DISEASE: DIAGNOSIS & TREATMENT

DENTISTRY

Amy Thomson, DVM, DAVDC

Periodontal Disease is one of the most common diseases in our companion animals. In addition to being highly prevalent, this disease is progressive and can impact not only oral health and comfort, but also systemic health. For this reason, finding and diagnosing this disease is imperative as well as making the correct recommendation for treatment to owners.

WHAT IS PERIODONTAL DISEASE?

Periodontitis: inflammation of the periodontium

- **Periodontium is the anchoring structure for teeth**

  Gingiva
  - Mucosal lining of maxilla and mandible
    Marginal/free, attached and interdental gingiva

  Alveolar bone
  - Thickened ridge of bone within maxilla and mandible containing teeth – alveolus or 'socket'
  - Extending to the cementoenamel junction (CEJ)

  Periodontal Ligament
  - Group of specialized connective tissue fibers anchoring tooth (cementum) to the alveolar bone

  Cementum
  - Mineralized connective tissue covering the root surface
    Serves as anchor of gingiva & PDL fibers

- **Inciting cause of inflammation/periodontitis = PLAQUE**
  Plaque is a biofilm of NON-plankton bacteria that binds to pellicle
  - Pellicle = glycoprotein of the saliva

- **Benefit of antibiotics is MINIMAL to none against plaque**

  There is an innate antimicrobial resistance
  - Plankton: 1000 – 1500x more resistant

- **Calculus: calcified/mineralized plaque; these minerals come from the SALIVA**

  Propagating cycle: plaque > calculus > rough surface > plaque

Periodontitis: this inflammation if left untreated, leads to destruction of periodontium

With this destruction, loss of anchorage or attachment
- The HOST mediates this destruction
  MMPs, Elastase, Cytokines, Prostaglandins

Other contributing factors: genetics, calculus, restorations, malocclusions, orthodontia, exodontia, radiation therapy, gingiva; enlargement, xerostomia, lifestyle, systemic health (ex. Diabetes Mellitus)

PERIODONTAL DISEASE & CONDITIONS

Gingival Disease; Chronic Periodontitis; Aggressive Periodontitis; Periodontitis as manifestation of systemic disease, necrotizing periodontal disease, periodontal abscess, periodontitis associated with endodontic lesions,

*** will focus on first three (3), as most common ***

Gingival Disease

- Gingivitis: inflammation of the gingiva
- Gingival enlargement: overgrowth of gingiva

(hyperplasia vs fibroma vs POF vs neoplasia)

- Gingival recession: receding gingiva OR loss of free and/or attached gingiva

Chronic Periodontitis

- VERY common disease, seen in adults in which the severity of plaque is equivalent to the level of destruction that is slow to moderate in progression and can be both localized or generalized

Aggressive Periodontitis

- MUCH less common disease, seen in young/juvenile patients with rapid attachment loss as severe destruction with only mild plaque that very rapidly progresses and can be both localized or generalized; often familial and sometimes immune dysfunction

Staging of Periodontal Disease

- Stage 0: no gingivitis
- Stage 1: gingivitis alone, no attachment loss
- Stage 2: gingivitis, with <25% attachment loss
- Stage 3: gingivitis, with 25-50% attachment loss
- Stage 4: gingivitis, with >50% attachment loss

Tools for staging of Periodontal Disease

- Imperative to treatment and prognosis
- Oral exam: to determine IF attachment loss
- Intra Oral Radiographs: to QUANTIFY percentage (%) & character of attachment loss

Oral exam: Plaque Index, Calculus Index, Gingival Index, Furcation exposure, Mobility

- Measure 4-8 sites around each tooth in a "walking motion"
  - Gentle pressure (~20 mmHg) as to not want to create a pocket
  - Normal periodontal probing: DOG: <3 mm* and CAT:<0.5 mm

    Keep in mind the size of the patient and size of tooth

    Vast difference in size in canine patients

  - Important to differentiate pocket depth vs attachment loss

    Attachment loss= probing depth + recession – enlargement

Intra Oral Radiographs

- Important to evaluate:
  - Alveolar bone height

    Bone loss ~ attachment loss, both defined as a percentage of root length (root apex to CEJ)

    Horizontal bone loss – loss spanning >1 tooth and parallel to occlusal plane

    Vertical bone loss – angular loss, more localized

  - Pulp chamber
  - Periodontal ligament (PDL)
  - Periapical space

ANESTHESIA FREE DENTAL "CLEANINGS"

The mainstay of appropriate dental care cannot be performed in awake patients: radiographs cannot be taken nor can thorough oral exam/charting – which are the MAINSTAY of diagnosing and staging the disease. Additionally, SUB-gingival debridement is not possible, and can be quite dangerous, and polishing is not possible as water/flushing and suction needed to ensure no ingestion or incidental aspiration.

- AAHA, AVMA and AVDC have statements on dental scaling without anesthesia.

PERIODONTAL TREATMENT

- The foundations is MECHANICAL DEBRIDEMENT; removal of supra- and sub-gingival plaque and calculus
- Determination of stage of disease for EACH tooth is imperative to guiding treatment
  - Thorough oral exam/charting
  - Full mouth intra oral radiographs
  - Treatment/therapy options depend on:

    Stage of disease
    Tooth affected
    Owner commitment

- Equipment is very important!!
  - Visualization is paramount: treating very small "patients" thus need to be able to see them and see them well

Appropriate light: overhead and loupes
Magnification: loupes
Dental mirrors
Suction

- Ultrasonic scaling: larger calculus, can be both supra- and/or subgingival
- Hand Scaling: less calculus, both supra- & sub-gingival; TACTILE feel

  Scalers and curettes
- Polish: prophy paste or pumice: to remove micro etches and smooth enamel

  Prophy paste can NOT be used IF restorations are to be placed (due to fluoride)

- Techniques for periodontal treatment
  - Root Planing: removal of plaque & calculus from exposed root surface

    Insert subgingival curette into sulcus at 0°, advanced towards base of pocket, angulate 45°-90°, then pull stroke
    Closed (non-surgical): <5 mm, no flap
    Open (surgical): >5 mm, flap is required for treatment
  - Gingival Curettage: removal of inflamed soft tissue along lateral aspect of pocket

    GOAL (if combined with root planing)= re-attachment
    This re-attachment is NOT normal/initial attachment tissue, but is a long junctional epithelium
  - Locally Delivered Antimicrobials

    NEVER without mechanical debridement
    Can help improve reattachment with root planing
    Examples: Doxirobe & Clindoral
  - Guided Tissue Regeneration: Bone Graft + Membrane

    Used with significant bone loss, and thus re-attachment with long junctional epithelium is NOT enough
    Types of bone grafts: autogenous, allografts, xenografts, alloplasts
    Types of membranes: thin cortical bone vs locally delivered antimicrobials
    This technique GUIDES which tissue grow/heal into the defect
    - *Gingiva wants to heal and down grow into defect first, therefore this needs to be prevented long enough to allow bony healing*

DON'T FORGET HOME CARE!!!

You can do a lot of amazing work, BUT if owner is not committed to home care and maintaining oral hygiene your treatment will NOT be successful!!

- TOOTHBRUSHING is still the GOLD standard
  - Take the time to demonstrate this, this little step will go a long way!

2002

ORAL TUMOURS: ALWAYS BIOPSY... THE RIGHT WAY

DENTISTRY

Amy Thomson, DVM, DAVDC

Any mass in the mouth has the potential to be a problem for your patient. Now some will only be a problem in the way a large benign mass leads to pseudo-pocketing around a tooth or teeth and lead to advanced periodontal disease.

However, when there is a chance the mass could be malignant or even locally aggressive knowing that information early can improve outcomes.

While it can be tempting to hypothesize what an oral or gingival mass could be – especially when the owner wants you to tell then WITHOUT the diagnostics, it is always best to get the definitive answer.

RIGHT WAY #1: BIOPSY EARLY.

There is not benefit to waiting to see if something grows.

RIGHT WAY #2: NEVER CALL IT AN EPULIS.

This term is used incorrectly in veterinary medicine and has led to serious harm in some cases.

Epulis = (Greek; pleural epulides) is ANY tumour-like enlargement (ie. lump) situated on gingiva or alveolar mucosa

This word literally means "growth on the gingiva" and described ONLY the location of the mass and has NO further implications on the nature of the lesion.

HOW can THIS lead to harm?

- A diagnosis of "epulis" is synonymous with "benign" and can lead to treatment not being pursued OR not being pursued in an appropriate timeline
- The "TOP HITS" on Google for epulis is: "a benign or non-cancerous mass"

This term had been previously used as a diagnosis:

- "Acanthomatous Epulis" = Canine Acanthomatous Ameloblastoma
- "Fibromatoys Epulis" = Peripheral Odontogenic Fibroma OR Hi/Lo Fibrosarcoma
- "Ossifying Epulis" = Peripheral Odontogenic Fibroma, Ossifying type

RIGHT WAY #3: TAKE THE DENTAL RADIOGRAPH.

While this cannot replace this histopathology, it can help with make the biopsy plan.

No changes to the underlying bone can favour benign OR minimal progression of a malignant or locally aggressive mass.

A cystic lesions would favour an Odontogenic (non-malignant/locally aggressive) mass.

"Floating bone" would favour an Ossifying Peripheral Odontogenic Fibroma

RIGHT WAY #4: STAY AWAY FROM THE MARGINS.

Unlike in dermatopathology where comparing the lesion and the surround tissue can be helpful with diagnosis, with oral masses this will lead to a larger definitive surgery.

2003

DENTOALVEOLAR TRAUMA: HOW NOT TO MISS THESE

DENTISTRY

Amy Thomson, DVM, DAVDC

Dentoalveolar trauma (DAT) is very common in our companion animals and can lead to many sequalae if not diagnosed and treated.

DAT most often causes ENDODONTIC disease, which is the opposite of periodontal (often incorrectly called "dental") disease.

Periodontics: Perio = around & ODONTO = tooth; the study of the structures around the teeth

- Periodontium = gingiva, alveolar bone, cementum & periodontal ligament

Endodontics: ENDO = inside & ODONTO = tooth; the specialty of study and treatment of dental pulp

The root canal system is made up of the pulp chamber (crown) and root canal (root)

Anatomy of the Pulp-Dentin Complex

- The outer most layer of the pulp is made of odontoblasts.
  - cells of neural crest origin and their function is production of dentin (= dentogenesis)
- The cell body of the odontoblast sit on the outer surface of the pulp, while the cytoplasmic processes of these cells extend into the dentin, within the dentin tubules.
- This allows communication between the pulp tissue and dentin

Pulpal Response to Insult

- Initial/early response is recruitment of inflammatory cells, leading to pulpitis
  - This will either be reversible OR irreversible and lead to pulp necrosis

Response to Exposed Dentin

- Tertiary dentin is formed when there is damage/loss of enamel and exposure of dentin
  - Reactionary Dentin = existing odontoblast lay down the dentin
    - be due to mild stimulus and is well organized
  - Reparative Dentin = reserve mesenchymal cells differentiate to odontoblast
    - due to strong stimulus and is poorly organized

NON-TRAUMATIC DENTOALVEOLAR INJURIES

Caries (cavities)

- Dental hard tissue decay
- UNCOMMON in dogs (~5% of population)

Abrasion

- **Dental wear** – loss of dental hard tissue due to contact with external objects (ex. bones, antlers, mental, hard plastics, hair/alopecia)
- SPEED of wear:
  - slow = pulp retreats & tertiary dentin,
  - fast often get pulp exposure (leading to pulpitis)

Attrition

- **Dental wear** – loss of dental hard tissue due to ABNORMAL contact between teeth (malocclusion or physiologic)
- SPEED of wear:
  - slow = pulp retreats & tertiary dentin,
  - fast often get pulp exposure (leading to pulpitis)

Treatment of Abrasion & Attrition

- **FIRST THING needed: RADIOGRAPHS – to check for vitality**
  - Evidence of non-vitality: wider pulp chamber and/or periapical lucency
    - IF NON-vital: RCT or extraction
    - IF VITAL: needs to continue to be monitored **RADIOGRAPHICALLY**

TRAUMATIC DENTOALVEOLAR INJURIES

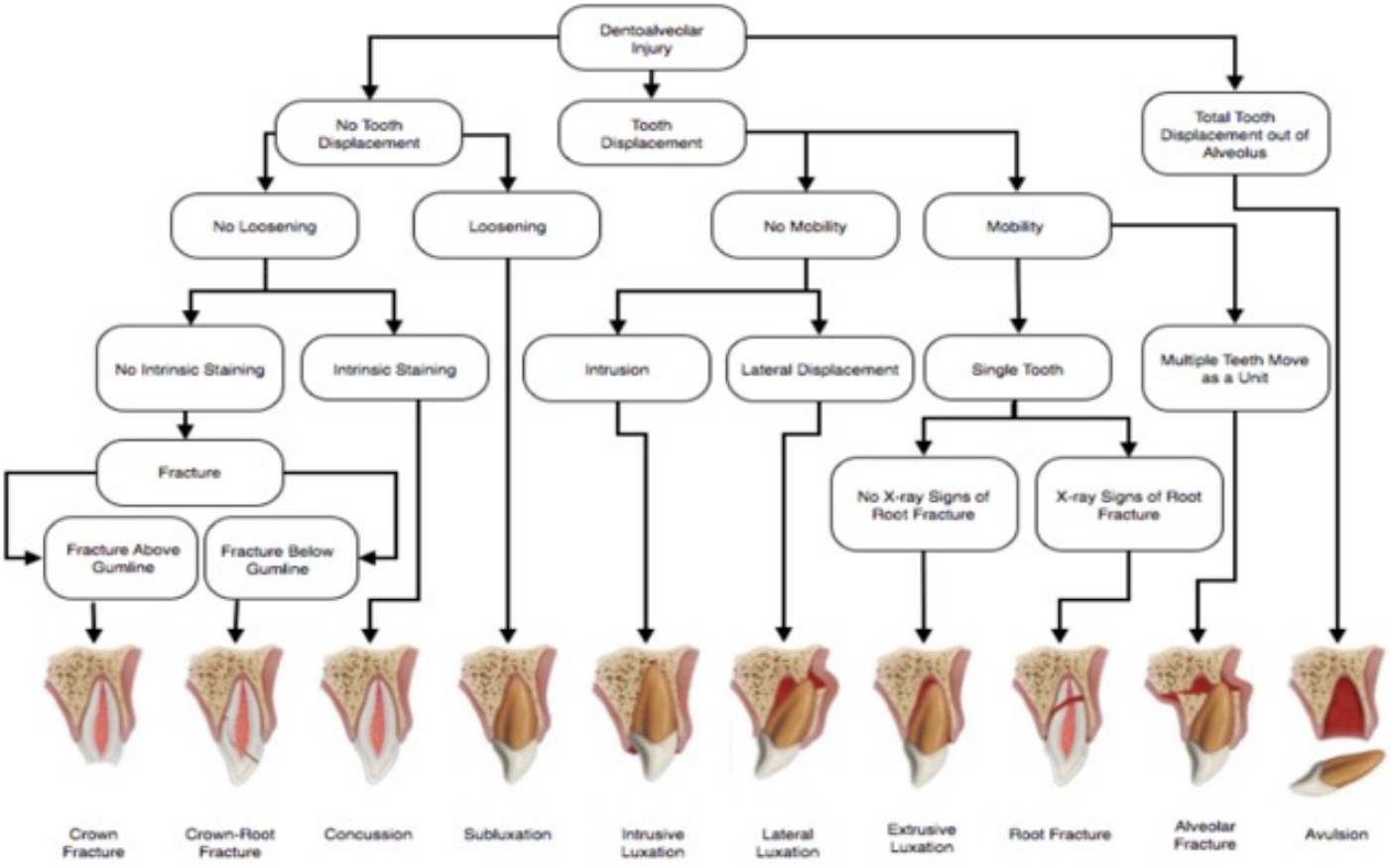


Figure Courtesy of Dr. Jason Soukup, DVM, DAVDC (permission from Dental Traumatology)

Enamel Infarction

- Crack/craze lines in enamel (and enamel ONLY); no treatment required (document)

Enamel Fracture

- Loss of enamel ONLY, no dentin exposure
- VERY Uncommon in cats and dogs:
    - Enamel is very thin, only 0.1 – 0.6 mm thick
- Treatment: smooth rough (plaque retentive) edges; Monitor RADIOGRAPHICALLY

Enamel-Dentin ("Uncomplicated") Fractures

- Can be crown only OR crown-root
- BUT are these injuries Uncomplicated?
    - Exposed dentin – sensitivity and pain
    - Risk of pulpitis/infection
- RADIOGRAPHS are needed initially – need to check/assess vitality
    - IF ACUTE – analgesics
- Treatment: "close" dentinal tubules
    - This may already be done – by tertiary dentin, OR can place restoration/sealant
    - IF ROOT involvement, must make a flap to explore and treat
    - The CONTINUE with radiographic monitoring

Enamel-Dentin-Pulp (Complicated) Fractures

- With pulp exposure treatment is ALWAYS REQUIRED
    - There will be pulpitis and eventual pulp necrosis
    - Most states require by law, and is ethical obligation, to discuss all treatment options
    - Treatment options: Vital Pulp Therapy*, Root Canal Therapy or extraction

Vital Pulp Therapy vs Root Canal Therapy

Depends on two main things:

- DURATION of pulp exposure
    - With increased time: increased contamination & increased depth of pulpitis
    - >48 hours significant decrease in success of procedure
- AGE of patient
    - Immature tooth = open apex and THIN dental walls
    - Want the tooth to remain vital to allow further dentinogenesis/development of dentin walls

Vital Pulp Therapy = pulpotomy = removal of contaminated pulp, followed by hemostasis and then placement of MTA (mineral trioxide aggregate), glass ionomer and composite

Root Canal Therapy = pulpectomy = removal of entire pulp; involves the Endodontic Triad

- Preparation (instrumentation): shaping the canal
- Sterilization: removing and/or killing organic material
- Obturation: complete filling & sealing or canal

Crown-Root Fractures

- Almost ALWAYS require exploratory surgery: to determine extent of fracture
- Need to consider the relationship of PERIODONTAL health and extent of restoration
    - Need to consider biological width

ENDODONTIC TREATMENT DECISION MAKING

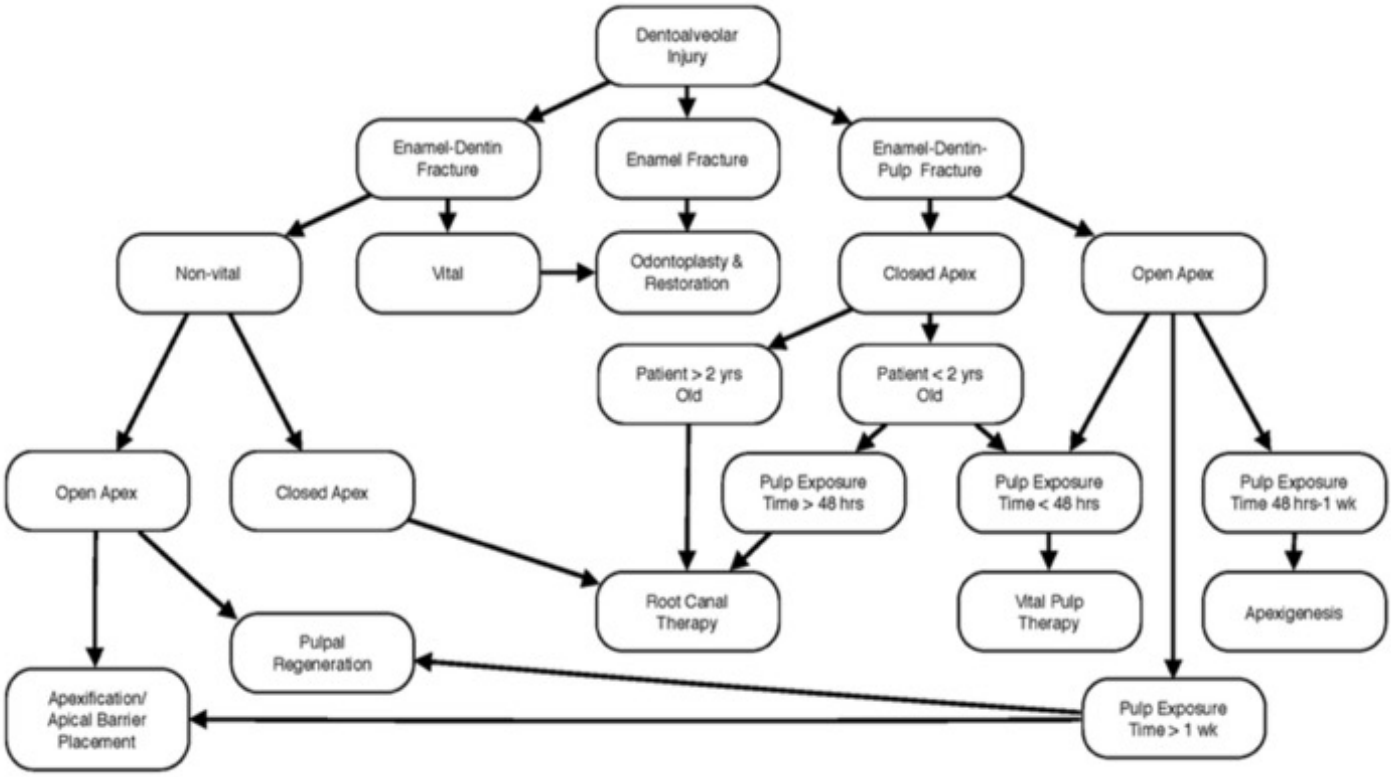


**Figure courtesy of Jason W. Soukup, DVM, DAVDC*

Root Fractures

- Apical third: little-no mobility (alveolus acts as splint); great prognosis
- Middle third: increased mobility; fair to good prognosis
- Coronal third: significant mobility; grave prognosis
- Pulpal healing in tooth fractures
    - With MINIMAL displacement = pulp INTACT: hard tissue union
    - With increased displacement: pulp STRETCHED: connective tissue growth
    - OR necrosis of coronal segment: pulp severed: granulation tissue between segments

Luxation Injuries

Concussive Injury

- Most concussion injuries show NO signs of injury
- Typical no long-term consequences, however with severe injury can get hemorrhage, thus intrinsic staining/discolouration
- Literature: ~90% of discoloured teeth are NON-vitality
- RADIOGRAPHS ONLY show non-vital is ~50% of these NON-vital teeth
    - Need to discuss with owners risk-benefit of treatment with normal radiographs
        - With non-vitality on radiographs = 100% dead
        - With NO signs of non-vitality still 80% chance NON-vital

Subluxation Injury

- Injury that leads to "loosening" of PDL;
  - NO fracture, Periodontal dz
  - Contusion of PDL +/- concussion
- Treatment: IF mobile: splint, IF NOT mobile no treatment (alveolus as splint)
  - MONITOR RADIOGRAPHICALLY for evidence of non-vitality

Luxation Injuries

- Intrusion
  - NTO alveolus (intruded), very rare
  - Typically maxillary canine INTO nasal cavity
- Extrusion
  - Partially OUT of alveolus (extruded), VERY Uncommon
- Lateral
  - In any AXIAL direction (accompanied by alveolar fracture)
  - Part of tooth OUT of alveolus, partial PDL severed
  - Often apical blood supply compromised or severed
  - Typically canine teeth (maxillary OR mandibular)
  - Treatment: soft tissue healing (PDL) AND bony healing (alveolus)
  - URGENT IF going to save the tooth
    - Time sensitive: <4 hours; need to preserve PDL cells
    - Need to splint in place, and then RCT later
- Avulsion
  - ENTIRE tooth OUT of the alveolus, entire PDL severed
  - Apical blood supply is SEVERED
  - Treatment: soft tissue healing (PDL) *rarely bony/alveolus involvement
- Extremely time sensitive: <1 hour
  - Need to prevent desiccation of PDL fibroblast
    - Milk, egg albumin, Hank's balanced salt solution, saliva

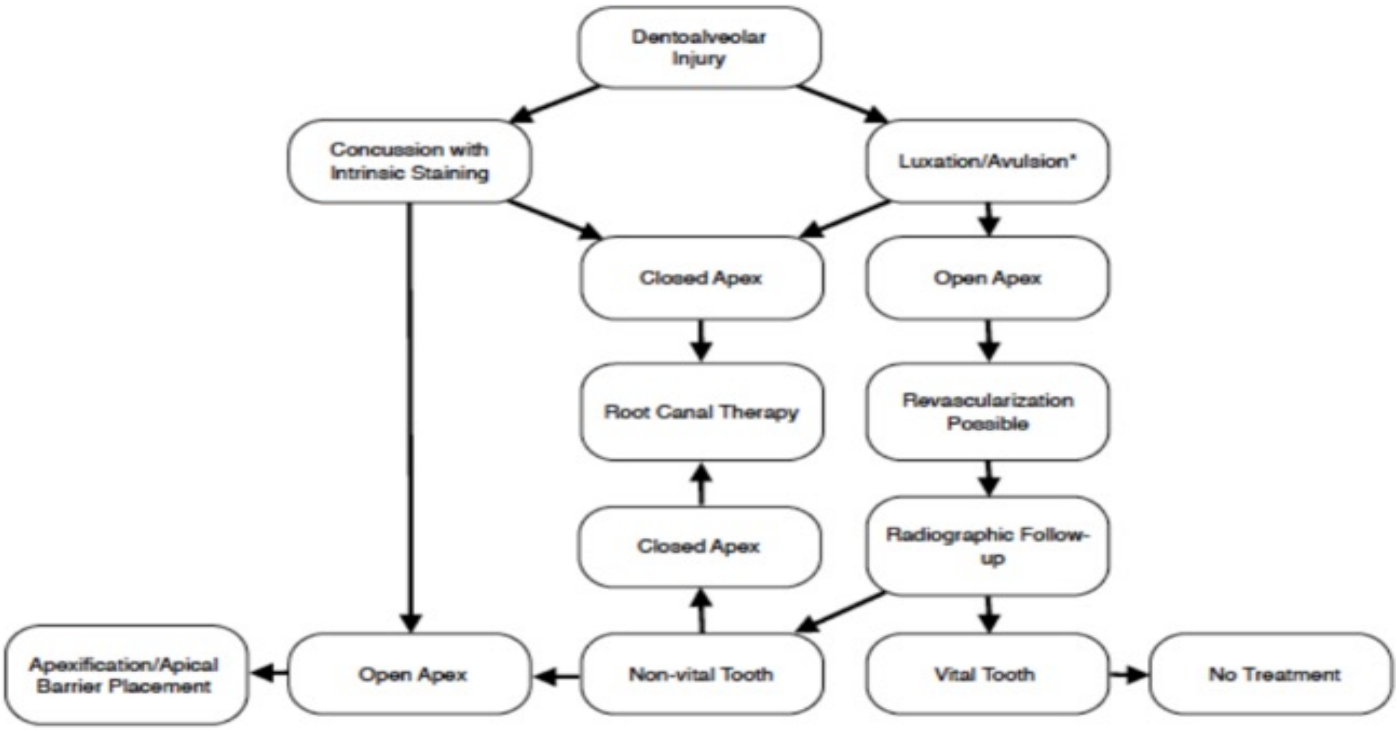


Figure courtesy of Jason W. Soukup, DVM, DAVDC

2004

DENTAL RADIOLOGY: THE GOOD, THE BAD AND THE INCONCLUSIVE

DENTISTRY

Amy Thomson, DVM, DAVDC

This lecture has been designed to help you improve you dental radiographs.

First reviewing everyone's least favourite technique: Bisecting Angle.

With my "cheater codes" for this technique you might even like taking radiographs.

Then we will review my process for evaluating all "bad" radiographs to make sure the next one you take is good!

The Intraoral Parallel Technique is the same technique used to acquire diagnostic images for the rest of the body. The plate or sensor used to capture the image is positioned parallel to the target anatomy; the generator, also called the tube head, is positioned perpendicular to both the sensor and the patient.

This technique is preferred as it allows for the most accurate image of the target anatomy with minimal distortion. However, due to the absence of a vaulted palate and the presence of a mandibular symphysis, the sensor can only be placed parallel to the entire tooth for the mandibular molars and in some patients the most caudal mandibular premolars.

The anatomic differences between humans and veterinary patients means that the majority of our patient's teeth will require the Bisecting Angle Technique to obtain radiographs.

One way to consider this technique is a way to create an intraoral parallel technique when the sensor cannot be placed parallel to the entire tooth. Remember, with parallel the tube head directed perpendicular to BOTH the tooth and the sensor, which is not possible for most of our patient's teeth. By bisecting the angle between the sensor and the tooth, this creates the angle to which the tube head can be placed perpendicularly.

The problem is often the angel between the tooth and the sensor is odd, inconsistent and hard to visually bisect.

Therefore, my "cheater code" is not CHOOSE the angel between the tooth and sensor: place it perpendicular to the tooth. This small change in sensor positioning allows for a 90-degree angle between the tooth and sensor which is visually much simpler to bisect: 45 degrees.

To ensure the sensor is perpendicular to the long axis of the maxillary teeth, the sensor should be placed parallel to the hard palate. For the mandibular teeth: using the patient's tongue to fill the intermandibular space will give your sensor a flat surface to lay against that is perpendicular to the tooth roots.

NOW about those "bad" radiographs.

Often a non-diagnostic radiograph, missing part of the tooth or inappropriate angle, is called "bad". The radiograph is only "bad" if you do not then use it to help you get the diagnostic radiographs you want.

When you take a radiograph and it is non-diagnostic, STOP and make sure you ask yourself what you DO like about it before deciding what you do not.

If part of the tooth or teeth are missing, move the sensor to ensure they are 'on the plate' and do not touch the tubehead.

If on the other hand you have the tooth, but it is too short or too long, do not move the sensor or plate but adjust the tubehead.

And while this seems simple, it is very easy to dislike a radiograph and want to change everything. However, in my experience it is bets to change only one thing at a time.

This lecture will go through many visual representation of "bad" radiographs leading to great ones.

2005

TOOTH EXTRACTION 101: HOW TO IMPROVE YOUR TECHNIQUE

DENTISTRY

Amy Thomson, DVM, DAVDC

Understating the anatomy of the tissue involved in the periodontium as well as PHYSICS is absolutely necessary for "taking the insanity OUT" of dental extractions.

GINGIVAL IS STRONG:

There are 6 different fibers within the gingival tissue, and while you do not need to know all of these fibers it is important to know this tissue is DESIGNED to neutralize the coronal-to-apical forces of mastication. Therefore, using your periosteal elevator in this SAME direction is going to result in your fatigue, longer time to make a flap and ultimately injury to the flap itself.

- INSTEAD, use your periosteal elevator PERPENDICUAR to the flap, OR is you must place your instrument in a coronal to apical direction, make sure the FORCE you use is in a mesial-to-distal (NOT coronal-to-apical) direction

"ROUND PEG AND A SQUARE HOLE":

The contours and width of tooth roots are not always regular, nor is the apex (tip) of the root always narrower that the coronal aspect. This is important to remember when removing teeth.

This is look for on radiographs:

- Dilacerated roots: wider coronal opening to alveolus and more "rocking" than twisting
- Bulbous apex: the alveolus will need to be widened to allow the root to be fully avulses
- Root developmental grooves (MN 1st molars): coronal-to-apical over rotational stretching of PDL

Remember: ELEVATORS STRETCH and LUXATORS CUT the Periodontal Ligament. Both instruments and techniques can be very useful. It is best to use these together based on the patient and teeth being extracted, as it is not a "one size fits all".

The periodontal ligament (PDL) fibers are designed to hole the teeth within the alveolar bone during mastication and therefore are able to withstand quick and intense forces.

- There fibers are weakest to sustained pressure
  - You MUST hold pressure for 20-30 seconds!!!!

The PDL fibers are also most concentrated at the apex of the root, therefore ensure you work apically.

VERTICAL ELEVATION

- You need the perfect sized instrument to MATCH the contour and width of the root you are removing
  - The instrument is meant to fit INTO the PDL space to that the ENTIRE working end of the elevator is engaged with the tooth
  - IF the elevator is too SMALL it will chip away at the tooth
  - IF it is too LARGE it will not fit into the space
  - IF the entire "working edge" is not engaged with the tooth, force is NOT being appropriately generated

- the overall FORCE is apical, therefore HIGER risk a root ending up in the nasal cavity OR mandibular canal with this technique, especially when:
    - Insufficient buccal bone removal,
    - Surrounding bone is diseased and friable
- The only PDL fibers being stretched with vertical elevation is those at the same level OR coronal to the instrument; therefore you need to ensure you continue to move the instrument more and more apical

THE WAY TO AVOID THIS COMPLICATION IS THROUGH HORIZONTAL ELEVATION:

- The size of instrument increases as you loosen the tooth
- The winged portion/working end is placed BETWEEN crown-root segments, perpendicular to the long axis of the tooth (vs. parallel as in vertical elevation)
- The overall FORCE here is CORONAL, or up and OUT of the alveolus
- This technique allows the PDL attachment stretch along TWO roots at the same time = EFFICIENT

Luxators are NOT meant to be used to generate FORCE to stretch the PDL as are elevators.

These instruments are thin and sharp and used to CUT the PDL fibers instead of stretching them and are very useful in certainly situations where elevators are either inefficient or risk damage:

- Thin and/or calcified PDL attachment
- Curved roots – IF have the curved luxators CAN cut along the entire root
- Severely thin or disease alveolar bone – torque is not created on this bone

"You can always TAKE MORE bone; BUT you cannot put it back"

- It is OK to start with little bone removal, however IF you are not making any progress AND you are using your instrument correctly – Take. More. Bone.

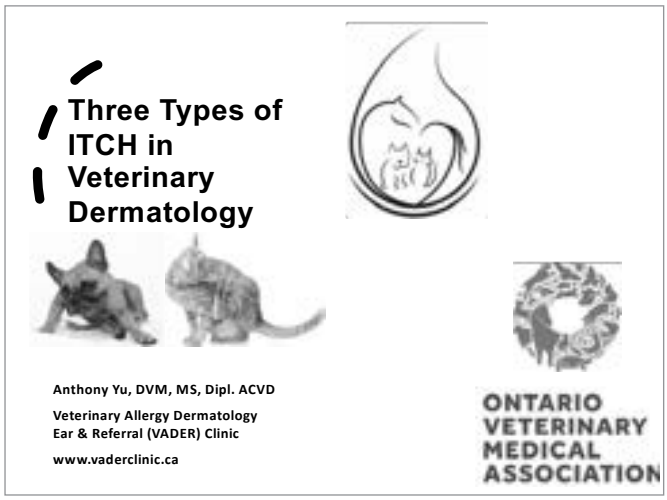
There is NOT a 'magic' amount of bone you should or should not remove; the "right" amount is the amount it takes to get the tooth out quickly, without trauma to collateral tissue OR compromise to future function.

3001

THREE TYPES OF ITCH IN VETERINARY DERMATOLOGY

DERMATOLOGY

Anthony Yu, BSc, DVM, MS, DACVD



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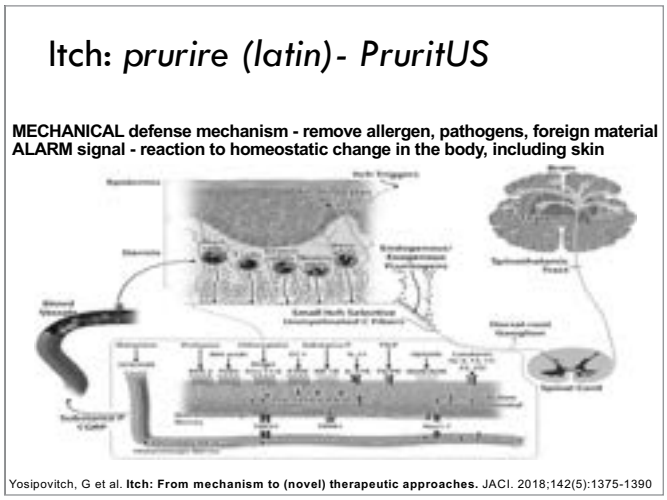

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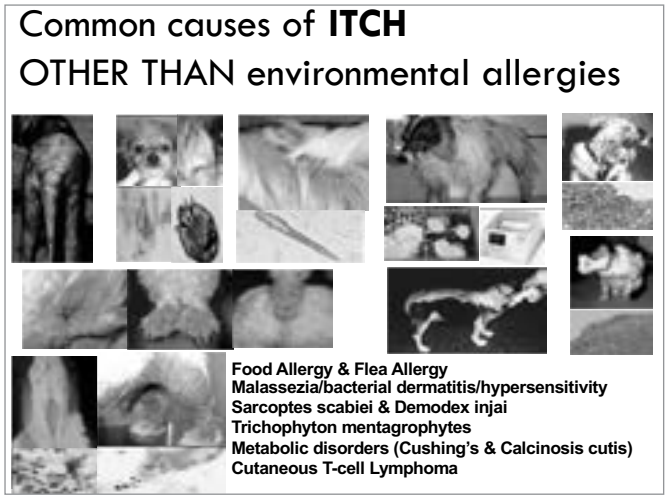
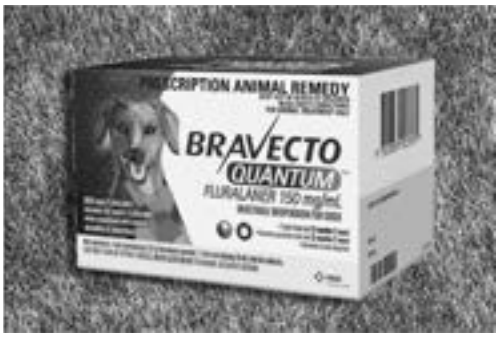


3

4

One injection lasts 12 months (AUS/NZ)



5

How would I approach this moderate/severely allergic dog?



6

Immunotherapy is still our best long-term approach.

Mueller, R. S. A systematic review of allergen immunotherapy, a successful therapy for canine atopic dermatitis and feline atopic skin syndrome. *JAVMA. 2023;261*(S1):S30-S35.

ONLY treatment that MODULATES pathomechanism of atopic dermatitis

All other treatments = symptomatic


Routes of administration
- Subcutaneous (SCIT; RUSH; Intralymphatic)
- Oral immunotherapy (SLIT; OIT; trypanophobia)

Benefits of Immunotherapy
1) Potential to eventuate a cure/remission
2) Weight-independent dosing - cost efficacious
3) Fewest side effects
4) Improved Quality of Life

Kotnik T. Quality of Life of Allergic Dogs Treated with ASIT — A Retrospective Study. *Vet Sci.* 2023;10(2):72.

- Follow-up and client education paramount to success
- My personal response rates: **70-100%**

Future of Immunotherapy

- **Recombinant allergens - key allergenic proteins only - faster response**

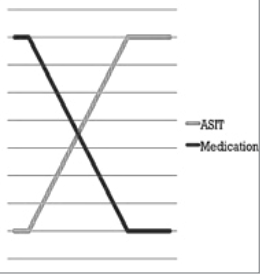
Martini F, et al. Open trial of recombinant Der f 2 pullulan-conjugated immunotherapy in cats. Vet Dermatol. 2024 Apr;35(2):175-183.

7

+Immunotherapy

- NOT "light switch"
- SLIT SID-BID
- SCIT q48hrs 30d, then 0.5cc Q 7 days
- Ancillary meds during induction
  - Glucocorticoids
  - Apoquel, Zenrelia
  - Cytopoint
  - Atopica® - complimentary

(Brandt et al, Allergy 2009)
(Nahm DH, Yonsei Med J 2012)



8

Three types of itch

Psychogenic	Inflammatory	Neurogenic (anti-IL31)
DAP®/Feliway® Stress Reducing Pheromone	**Atopica** Calcineurin Inhibitor Inhibits T-cell activation → Decreased IL-2,4,10,15,18, IFN-γ and TNF-a Decreased mast cell production and degranulation Decreased eosinophil survival and function	**Apoquel®** JAK 1,3 inhibitor **Zenrelia™** JAK 1,2 & TYK 2 inhibitor
Fluoxetine Selective Serotonin Re-Uptake inhibitor (SSRI)		Reduction in serum IL-31 Binds capsaicin receptor TrpV1 (Vanilloid)
Clomipramine **Amitriptyline** Inhibit serotonin (5-HT) and norepinephrine reuptake	Antihistamine Blocks H1	Neuropathic pain
	Steroids GC/GCR binds to NF-κB, GResponseElements, and Activator Protein-1 → altering inflammation	**Cytopoint®** Monoclonal antibody directed against IL-31

Anthony Yu©

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Medical Management of Anxiety

Tricyclic Antidepressants (TCAs)
- Inhibit NE/Serotonin re-uptake, antihistaminic, antinociceptive
- Amitriptyline (Elavil®) **1-4 mg/kg PO q12h**
- Clomipramine (Clomicalm®) **1-3 mg/kg PO q24h**

Selective Serotonin Reuptake Inhibitors (SSRIs)
- Fluoxetine (Reconcile®, Prozac®) **1 mg/kg PO q24h**

NB: Minimum of 3-4 weeks tapering to lowest effective dose
SE: Sedation, anticholinergic effects.
CE: Contraindicated in patients with cardiac disease or seizures
: Interactions w/MAOIs, selegiline, L-tryptophan, thyroid meds

10

Behavioural Management of Anxiety

ENVIRONMENTAL MODIFICATION

Address/remove sources of stress

Increase activity (eg, more walks)

Companion for your dog

COUNTER-CONDITIONING

Associate something good with a stimulus that previously predicted something bad

DESENSITIZATION

Present offending stimulus at gradually increasing levels until habituation develops.

11

VADER Atopica Drawer
Replenished twice weekly

12

+ Atopica™ - SAFE & reliable treatment option

Nuttall T, et al. Life-long diseases need life-long treatment: longterm safety of ciclosporin in canine atopic dermatitis. Vet Record 2014;174(suppl 2):3-12

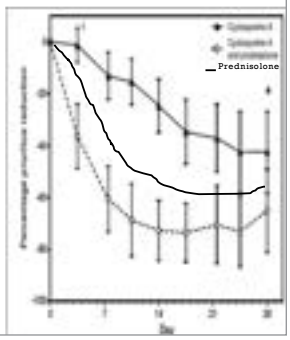
- **Atopica ORAL cyclosporine CAPSULES**
  - 10mg, 25mg, 50mg, 100mg
  - 5mg/kg daily for 4-8 weeks then taper
  - FREEZE capsules
  - **Stability and pharmacokinetics of ciclosporin capsules stored at -20C.**
    J. Bachtel et al. Vet Derm, 2015:26:133–159.
- **Atopica ORAL cyclosporine LIQUID**
  - 100mg/ml
  - Extra-label use in dogs - calibrate/change syringe
  - 7mg/kg daily for 4-8 weeks then taper (cats)
  - 5mg/kg daily for 4-8 weeks then taper (dogs)
  - REFRIGERATE liquid
- **Sandimmune INJECTABLE cyclosporine**
  - 50mg/ml ampules X box of 10 ampules
  - Extra-label use in dogs and cats
  - 2.5-5mg/kg SQ q48-72h (cats, dogs)
  - **Koch, SN, et al. Vet Derm 2018;29:107**

13

Short-Term Strategies
Speeding up the response of Atopica

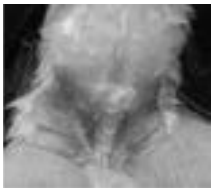
- QUICK anti-inflammatory effect with short-term steroids
- **How to institute**
  - 5mg/kg/day for 30-**60** days
  - With steroids to start 14d±28d
- *Concurrent short-term use of prednisolone with cyclosporine A accelerates pruritus reduction and improvement in clinical scoring in dogs with atopic dermatitis*

Dip et al. BMC Vet Res 2013, 9:173



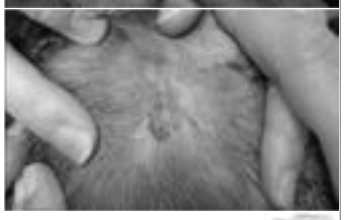
14

Westies and Frenchies
BEST Atopica® responders



Scruffie
Hoch
8 weeks
Atopica®

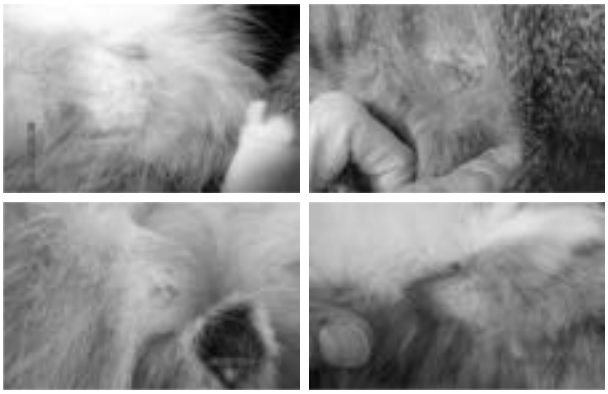
15



'Squeek" Indoor 4yo FS DSH
Numerous diet trials w/ no effect

16

<1 week Update on Squeek



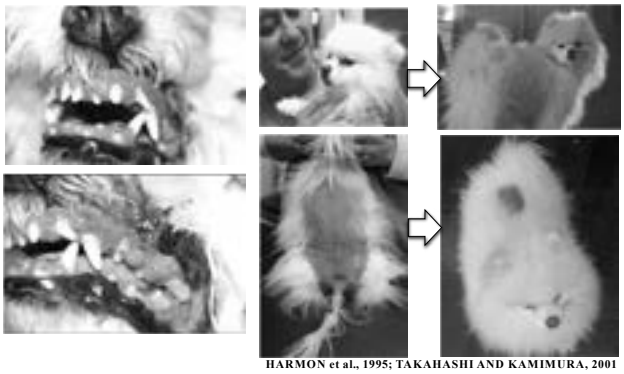
17

Squeek Loy at 4 weeks recheck

1) DECREASE **ATOPICA** TO EVERY OTHER DAY
2) START DIET CHALLENGE IN 2WKS
3) DECREASE **ATOPICA** TO EVERY 3RD DAY IN 4WKS

18

+ GINGIVAL HYPERPLASIA PROMOTES HAIR GROWTH



HARMON et al., 1995; TAKAHASHI AND KAMIMURA, 2001

19

+ Steroids Are Good BUT...

- Infections
  - bladder infections, skin infections,
  - septicemia, respiratory infections
  - generalized demodecosis
- Alopecia, thin skin, skin fragility (cats)
- Calcinosis cutis, atrophic remodeling of scars,
- Milia-like comedones, follicular cysts,
- Musculoskeletal atrophy, ruptured cranial cruciate
- Hyperlipidemia, steroid hepatopathy, colitis,
- Adrenal suppression/atrophy,
- DM, ESS, increased PTH levels,
- Behavioral changes, PU/PD, polyphagia, panting

20

Address Concurrent Factors

Maximum allowable steroid dose per year

- Prednisone/olone dose: 0.5-1.0mg/kg/d 7d, taper EOD
  - **Max Pred dose/yr = 30mg X BW in kg (doubled in cats)**
- Dexamethasone dose: 0.05-0.1mg/kg/d 7d, q2d 14d, q3d
  - **Max Dex dose/yr = 4.5mg X BW in kg (doubled in cats)**

Sousa C. Glucocorticoids in veterinary dermatology. CVT XIV 2009; 400-405.

Calcinosis Cutis due to Steroids
Courtesy of Dr. John MacDonald

Iatrogenically steroid-induced feline skin fragility syndrome
(Also watch HCM and DM)

21

Interleukin-31 blockers

Apoquel (oclacitinib) now available as chewable tablet with pork liver powder

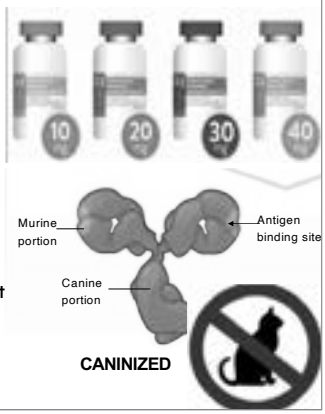
Cytopoint (lokivetmab)

This combination does not address allergic inflammation
It helps itch and stop traumatically-induced skin irritation only

22

Cytopoint® to Control Neurogenic Itch

- Lokivetmab
- "Caninized" IL-31mAb
- Weight dependent dosing
  - 2 mg/kg SQ q4-8weeks
  - 50-70% respond in 1-3 days
  - Test dose of 0.25-0.5mg/kg especially in larger dogs $$$
- **Antipruritic/Anti-neurogenic**
  - Indirect anti-inflammatory effect
  - OK to use in cancer patients
  - NO age restrictions listed
- 3-4% may develop autoAb



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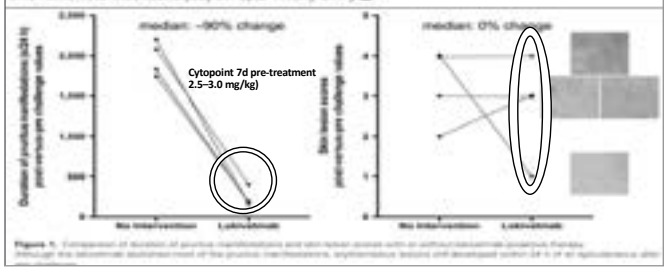
Veterinary Dermatology

Scientific Paper **2019;30(2):98-e26**

Proactive maintenance therapy of canine atopic dermatitis with the anti-IL-31 lokivetmab. Can a monoclonal antibody blocking a single cytokine prevent allergy flares?

Chie Tamamoto-Mochizuki, Judy S. Paps, Thierry Olivry



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Veterinary Dermatology

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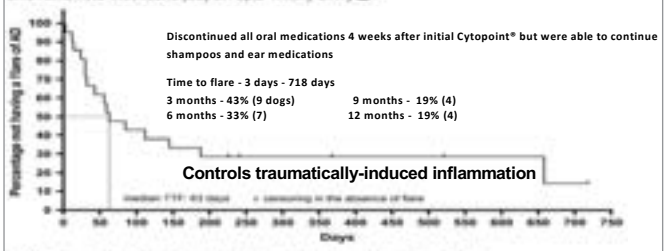

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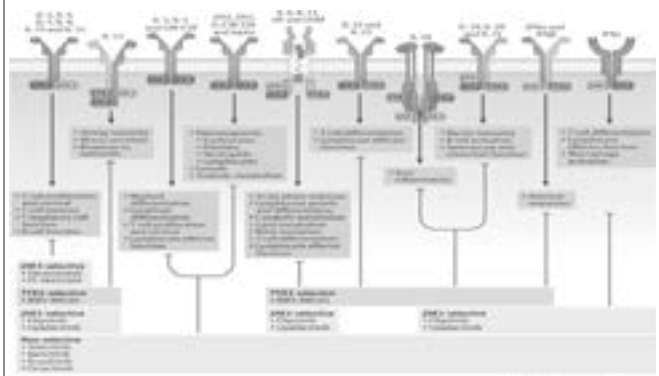
JAK Inhibitors in humans



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Janus Kinase Inhibitor

apoquel (oclacitinib tablet)

- QUICKLY BLOCKS JAK 1,3-Signal Transducer and Activator of Transcription (STAT) signaling pathway preventing **IL-31** effect on neurons stopping NEUROGENIC itch
  - **0.4-0.6mg/kg BID 14d, then once daily**
  - **Onset of action within 1-4 hours**

Veterinary Dermatology

Vet Dermatol 2015; 26: 171–e35

Long-term compassionate use of oclacitinib in dogs with atopic and allergic skin disease: safety, efficacy and quality of life

- **SIGNIFICANT** improvement in Quality of Life
- SAFE for longterm use
- q24h dose, mainly anti-ITCH, minimal anti-INFLAMMATORY
- **Does decrease traumatically-induced inflammation**

27

Apoquel® in Cats [Extra label]

- Frank RK et al. **Use of Oclacitinib (Apoquel® ®, Zoetis) for treatment of cutaneous mastocytosis in a cat.** Vet Derm 2014;25:153. **1 mg/kg BID 31 days**
- Whitehouse W et al. **Clinical use of 10 emerging therapies.** Update In Feline Therapeutics 2015;17:220–234 **0.5-1 mg/kg BID 28d**
  - eosinophilic airway inflammation and pruritus
- Loft KE. **Feline idiopathic ulcerative dermatosis treated successfully with oclacitinib.** Vet Dermatol (2015) 26:134–5 **1 to 1.5 mg/kg SID 6 weeks**
- Veterinary Dermatology
  2015;26(4):235–e52
  Oclacitinib in feline nonflea-, nonfood-induced hypersensitivity dermatitis: results of a small prospective pilot study of client-owned cats
  - 12 cats **0.47 mg/kg BID** 5/12 good improvement

28

Veterinary Dermatology

Scientific Paper **2019;30(2):110-e30**

A double-blinded, randomized, methylprednisolone-controlled study on the efficacy of oclacitinib in the management of pruritus in cats with nonflea nonfood-induced hypersensitivity dermatitis

N=20 - Apoquel - 1mg/kg BID 28d
N=20 - Medrol - 0.5-1mg/kg BID 28d
Both equally efficacious
4/14 renal enzyme elevations w/Apoquel
Possible anemia

Chiara Noli, Irina Matricoti, Carlo Schievano

sciendo

Apoquel 1mg/kg BID until resolved (`3 months) then discontinued
Lesion free 5 months at time of writing the paper

Case Report

DIFFUSE CUTANEOUS MASTOCYTOSIS (PIGMENTED MACULOPAPULAR CUTANEOUS MASTOCYTOSIS) IN A CAT

Marcin Szczepanik[1], Piotr Wilkołek[1], Anna Śmiech[1], Iwona Tasiłkun[1]

29

JAVMA | AVMA

Oclacitinib 10 years later: lessons learned and directions for the future

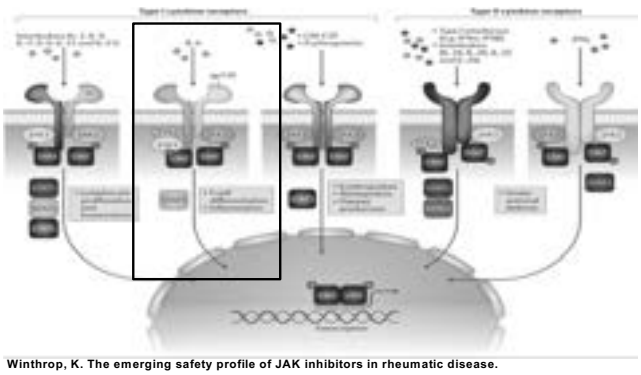
- Not labeled for use in dogs less than 12 months of age
- Immunosuppressive effects occur at higher doses
  - ischemic dermatopathy, subepidermal blistering dermatosis, ulcerative ear tip dermatosis, Cutaneous lupus erythemaosus, hyperkeratotic EM, PF, perianal fistulae

□ **Adverse Effects:**

- **Dogs:** V/D, weight gain, demodicosis, pyoderma, otitis
  : reversible behaviour changes
  : mild neutropenia,
  : no statistical difference on incidence malignant and non-malignant skin masses in treated vs control
- **Cats:** anemia, vomiting, increased: ALT, creatinine, BUN
- : one FIV+ cat with fatal toxoplasmosis

30

Zenrelia™ - new JAKinib on the block



Winthrop, K. The emerging safety profile of JAK inhibitors in rheumatic disease. *Nat Rev Rheumatol* 2017;13:234–243

31

Zenrelia™ - new JAKinib on the block

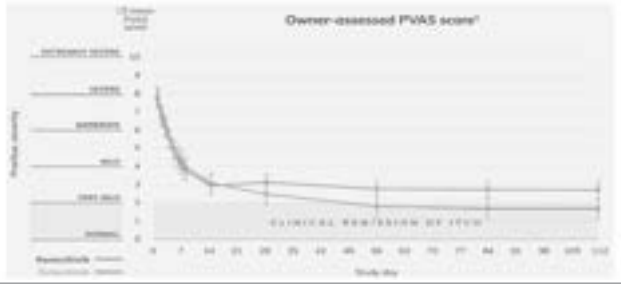
- Ilunocitinib
- JAK-1, 2, TYK-2 inhibitor
- Dogs >12 months
- 0.6-0.8mg/kg PO
- ONCE DAILY dosing
  - No rebound effect
- Tablet cost
  - Inverse log
- SE: Same as Apoquel®

32

Zenrelia™ - new JAKinib on the block

In a head to head clinical trial in 338 client owned dogs, Zenrelia showed similar efficacy as Apoquel (at primary endpoint Day 28)

Results at additional endpoints show promising potential for Zenrelia



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Veterinary Dermatology

Immunomodulatory *in vitro* effects of oclacitinib on canine T-cell proliferation and cytokine production

- **Oclacitinib (Apoquel®) at 337 ng/ml (prescribed dose) NO effect on T-cell proliferation/inflammation**
- **Cyclosporine (Atopica) at 200 ng/ml (prescribed) significantly inhibited T-cell proliferation; ↓ IL-2, IL-10, IL-15, IL-18, IFN;TNF-a**
- **CONSIDER COMBINATION THERAPY**
- **ANTI-INFLAMMATORY + ANTINEUROGENIC**

French bulldog on Apoquel alone

34

Can I use **Apoquel** with **Steroids**?

Veterinary Dermatology

Scientific Paper **Vet Dermatol 2015;26:171-E35**

Long-term compassionate use of oclacitinib in dogs with atopic and allergic skin disease: safety, efficacy and quality of life

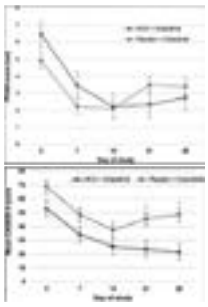
Drug Class	Prescription (n=247)
	n (%)
Antihistamines	85 (34.4)
Systemic Glucocorticoids	37 (15.0)
Topical Glucocorticoids	51 (20.6)
Psycholeptics	46 (18.6)
Omega 3 Fatty Acids	42 (17.0)
Analgesics	41 (16.6)
Non-steroidal anti-inflammatory products	38 (15.4)

35

Veterinary Dermatology

Efficacy and safety of 0.0584% hydrocortisone aceponate topical spray and systemic oclacitinib combination therapy in dogs with atopic dermatitis: a randomized, double-blinded, placebo-controlled trial

- Apoquel + placebo
- Apoquel + Cortavance



- CADESI-4/PVAS
- sig reduced D7&D14 in both groups ($P < 0.05$)
- increased in placebo ($P<0.005$) D14-21
- reduced in HCA-treated compared to placebo
  - D21 (59.9% versus 27.6%, $P = 0.0216$)
  - D28 (56.0% versus 30.5%, $P = 0.0109$)
- Cortavance useful for preventing exacerbation of pruritus and clinical lesions when tapering oclacitinib therapy in dogs with AD.

36

Veterinary Dermatology

A randomised controlled trial testing the rebound-preventing benefit of four days of prednisolone during the induction of oclacitinib therapy in dogs with atopic dermatitis

- oclacitinib at 0.4–0.6 mg/kg twice daily for 14 days then once daily alone
- or with prednisolone at 0.5 mg/kg, orally, twice daily for the first 4 days
- D21➔sig fewer rebounds w/prednisolone (3/20, 15%) vs (9/20, 45%)
- Sig lower:
- PVAS10 (D4&D28)
- CADESI4 + 2D-IGA (D28)
- OGATE (D21&D28)

- Take Home:
  - Oclacitinib ➔ pruritic inflammation
  - Prednisone ➔ allergic inflammation

37

Can I use **Apoquel** with **Atopica** ?

Veterinary Dermatology

Repeated oral dose tolerance in dogs treated concomitantly with ciclosporin and oclacitinib for three weeks

- **N=8 dogs (4F:4M/group) – Apoquel® or Apoquel®+Atopica**
- **Dosed daily at labelled doses for 3 weeks**

Conclusions and clinical importance

- No increase number of adverse events or laboratory abnormalities beyond those associated with oclacitinib given alone.

- **Consider combination therapy**
  - Start **Apoquel®** BID for 14d, then SID for 14d OR SID 28d then as needed
  - Start **Atopica** +/- ketoconazole every OTHER day for 6 weeks, then taper
  - **Day 1 - Atopica/KCZ; Day 2 - Apoquel; repeat**
  - **Day 1 - Atopica/kCZ; Day 2- Apoquel; Day 3 - Apoquel; repeat**
  - **Day 1 - Atopica/KCZ; Day 2 - Apoquel; Day 3 - dexamethasone; repeat**

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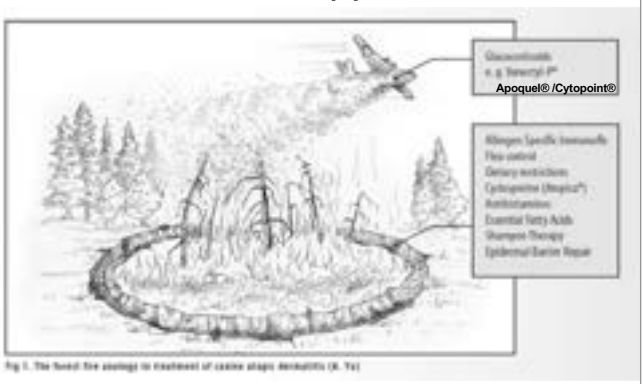
Multi-Modal Therapy



Olivry T, et al. Treatment of canine atopic dermatitis: 2015 updated guidelines from the International Committee on Allergic Diseases of Animals (ICADA). *BMC Veterinary Research* 2015, **11**:210

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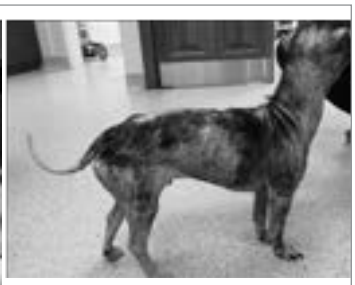


Atopica /Keto daily
Convenia q2weeks for 6weeks
Dexamethasone
Apoquel
ProHex:Micoderm
Hypo HP food.

40

D/C antibiotics and Keto
Atopica (adjust dose)
Apoquel
Added in amitriptyline for anxiety.

41

Atopica every third day, Apoquel PRN, amitriptyline bid.
Started dietary challenges

42

3002

HERE'S TO THE EARS

DERMATOLOGY

Anthony Yu, BSc, DVM, MS, DACVD

Here's to the Ears
Dx & Tx of pinnal dermatitis and otitis externa

Anthony Yu, DVM, MS, Dipl. ACVD
Veterinary Allergy Dermatology
Ear & Referral (VADER) Clinic
www.vaderclinic.ca

ONTARIO VETERINARY MEDICAL ASSOCIATION

1

Top 10 Most Common Medical Conditions

Top Dog Conditions

1. Skin Allergies
2. **Ear Infection**
3. Non-cancerous Skin Mass
4. Skin Infection
5. Arthritis
6. Upset Stomach/Vomiting
7. Periodontitis/Dental Disease
8. Diarrhea/ Intestinal Upset
9. Bladder/Urinary Tract Disease
10. Soft Tissue Trauma
    (Bruise or Contusion)

2

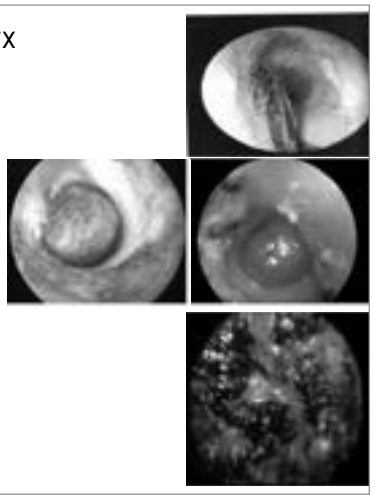
Minimize client frustration

- ACVD client survey
  - clients give general practitioners three (3) attempts at controlling/ resolving skin issues before seeking another opinion from another general practitioner or a specialist.
- 10 Steps to successful treatment of pinnal dermatitis and otitis externa

3

1. Address easiest to ID/TX underlying etiologies:

- **Foreign bodies**
- **Trauma**
- **Abnormal anatomy**
- **Ear canal tumours**
- **Parasites**
- Excessive moisture
- Atopic dermatitis
- Adverse reaction to food
- Hypothyroidism



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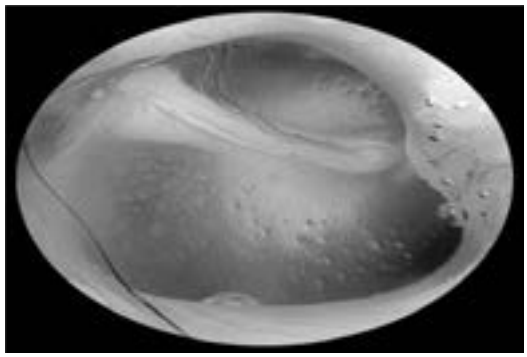
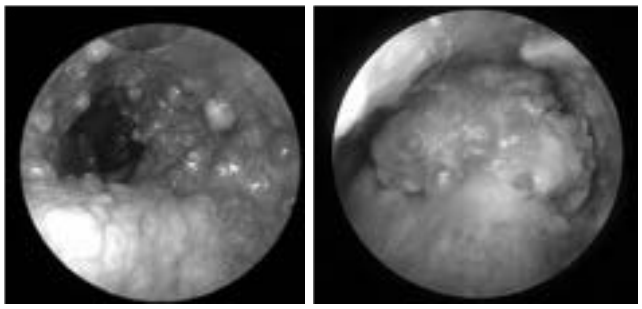
5

Video-otoendoscopy



6

Inflammatory Polyps vs Ceruminous gland adenoma



7

Isoxazolines address ALL ectoparasites including Otodectes cynotis

8

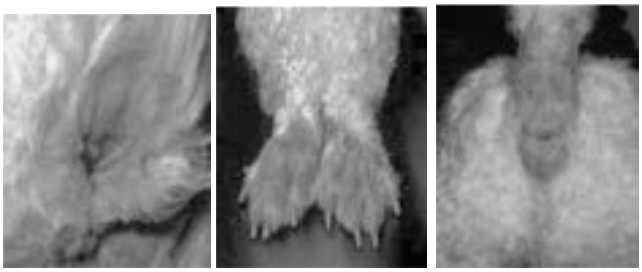
2. Address next top 3 underlying etiologies for chronic recurrent otitis externa

- Foreign bodies
- Trauma
- Abnormal anatomy
- Ear canal tumours
- Parasites
- Excessive moisture
- **Atopic dermatitis**
- **Adverse reaction to food**
- **Hypothyroidism**

9

10

Adverse Food Reaction



24% of food allergic patients present with otitis externa as the ONLY presenting clinical sign

11

Proliferative Otitis Externa On Apoquel and Cytopoint

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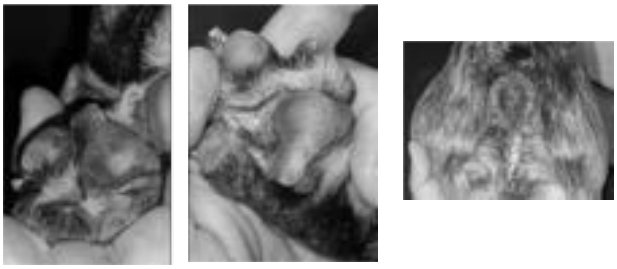
Proliferative Otitis Externa



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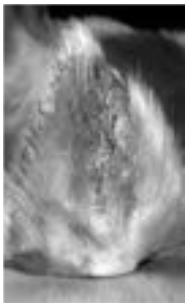


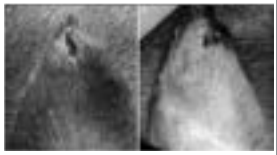
My dog is bleeding from his ear tips
It's like a crime scene in my house

14

Ear tip ulcerative dermatitis treated with oclacitinib in 25 dogs: a retrospective case series. Vet Dermatol, 2021;32:363-e100.

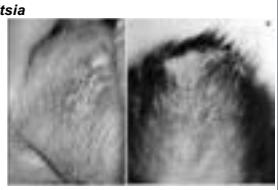
Pinnal vasculitis may be the only clinical sign
Erythema, erosions, ulcerations, crusting tips of pinnae
Typical wedge-shape lesion pointing towards the ear canal
Bilateral >>> unilateral
Intermittent bleeding ("CSI crime scene")
+/- Pruritus/pain

Bx: proliferative thrombovascular necrosis



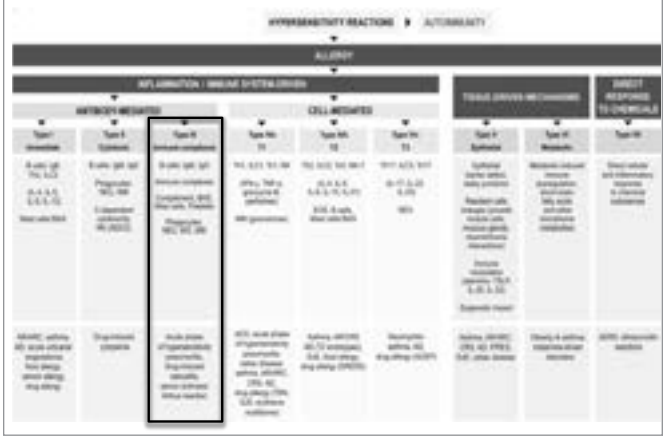
Differentials

Leishmania, *Bartonella henselae, Rickettsia rickettsia*
Frostbite, cryoglobulinemia, cryofibrinogenemia
Breed-specific (familial) vasculitides
- Jack Russell terrier, Dachshunds, Rhodesian ridgebacks
Idiopathic vascular disease
Type III hypersensitivity
- **food, environmental, drug or vaccine reaction**
- **Otoscopic/cytologic examination to address otitis**



15

Nomenclature of allergic diseases and hypersensitivity reactions: Adapted to modern needs: An EAACI position paper Allergy. 2023;78:2851–2874



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Ear tip ulcerative dermatitis treated with oclacitinib in 25 dogs: a retrospective case series. Vet Dermatol, 2021;32:363-e100.

Treatment Options
Topical steroids (6) or tacrolimus (1)
Pentoxifylline (11), vitamin E
Doxycycline (1)/tetracycline + niacinamide
Sulfonamides
Oral steroids (6) and/or cyclosporine
Photobiomodulation - (Phovia)
Surgery (conchectomy)

Oclacitinib (Apoquel)
T-cell proliferation; IL-2,10,15,17,18,21 + IFN-γ

Italy, multiple breeds, 1-13yrs, 14M/3MN/4F/4FS
13 pendulous, 5 folded, 3 erect, 4 unknown mix
N= 22/25 resolved within 1-3 months
@0.4-0.6mg/kg BID 14d, then SID
N=2/22 required BID
N=3/25; 1=PTX+tacro; 1=CSA; 1=Apoquel BID
N=4 relapsed within one month of d/c Apoquel
1=Apoquel SID; 1=conchectomy; 2=???

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PINNAL VASCULITIS

Baxter – 10y, MN, American Bulldog.
Pinnal vasculitis - initially responded to BID oclacitinib & PTX before relapsing.
Topical steroid cream was added to the treatment regimen with incomplete response
Once weekly back-to-back Phovia sessions performed for total of 8 weeks.
Consider underlying food allergy ➔ Type III hypersensitivity


Week 1


Week 6: Vasculitis improved, one area of crusting present on concave surface


Owner update 3 months post Phovia: Baxter's ear responded well to Phovia treatment. Scaring present but progression of vasculitis has not occurred

Credit photo: Dr. Yu

18

Unusual signs of Hypothyroidism

- Muscle atrophy
- Facial nerve paralysis
- Acral lick granuloma
- **Ceruminous otitis externa**
- ACL rupture
- Laryngeal paralysis/voice
- Megaesophagus
- Corneal lipid deposits
- Cardiomyopathy
- Peripinnal hyperkeratosis

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Hypothyroid - Peripinnal hyperkeratosis

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3. Cytology is the pillar to successful treatment of otitis externa

- Ear cytology best diagnostic tool to ID microbes


Giant's Causeway, near Belfast, Ireland

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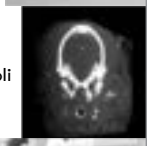
Diagnostic Otology



- Cytology ★★★★★
  - Heat fix and DiffQuik®
  - Provides information about the inflammatory response
  - Helps direct appropriate therapy
  - **Cocci:** generally Staphylococcus or Streptococcus
  - **Rods:** generally Pseudomonas>>Coryne, Proteus>>E. coli
  - **Yeast** is typically Malassezia, but can be Candida.
  - **Drug Reaction** w/neutrophils and no bugs
- Radiography
  - CT scan (e.g. VetCat®, Xoran) is best option
- Biopsy
  - To diagnose a tumour or confirm proliferative tissue

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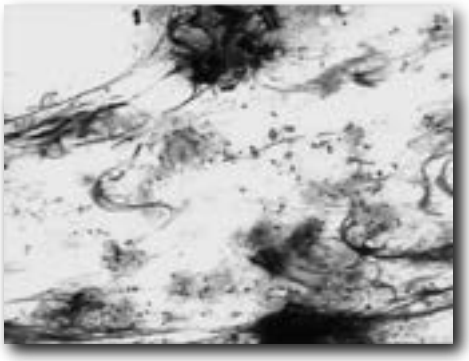
Cytology - cocci, neutrophils, Cytology - cocci, neutrophils Tyeast



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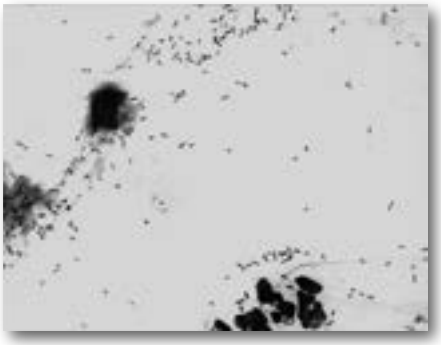
Cytology – rods, neutrophils



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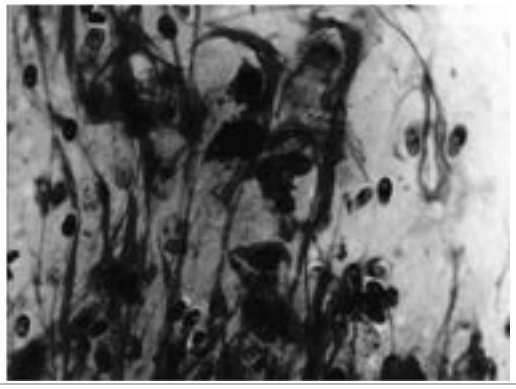
Cytology - Malassezia

25

Cytology - Yeast engulfed in neutrophils... likely Candida albicans



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4. Don't fall into the C&S trap

Stonehenge, England

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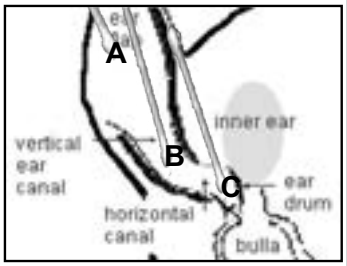
The Culture Debate

- Sampling issues
  - Location; location; location
  - transport/sample processing
- Laboratory issues
  - Culture technique
  - Heteroresistance
  - Lack of repeatability
  - CLSI in humans vs pets



- Interpretation issues
  - Cytological + culture consistency → clinical relevance
  - Request drugs you are using
  - Sensitivity = serum drug level NOT topical meds

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Ear product antibiotic concentrations

Product Name	Antibiotic	Concentration of Antibiotic (µg/mL)
Canaural	Diethanolamine fusidate	5000
Baytril Otic	Enrofloxacin	5000
Mometamax	Gentamicin	3000
Posatex	Orbifloxacin	8500
Tresaderm	Neomycin	3200
Panalog	Neomycin	2500
Surolan/**Otizole**	Polymixin B	529.3

Minimum of 2X the MIC (Surolan/Otizole)
and up to <1000X MIC (neomycin, gentamicin, fusidic acid & Orbi/enro/marbofloxacin

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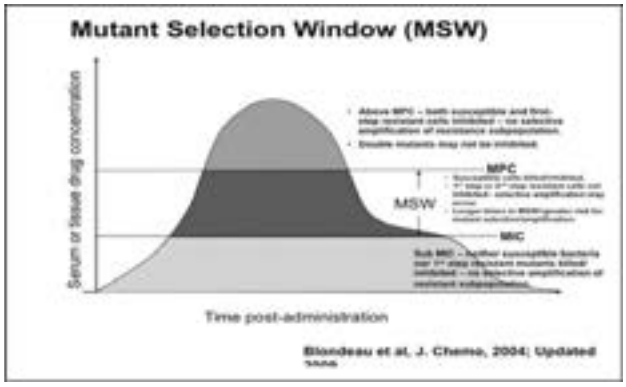
Topical otic therapy achieve antibiotic concentrations that well EXCEED **Mutant Prevention Concentrations**



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Topical Otic Product Sensitivity Testing

- TOPS study using E-Test at VADER
  - Testing of "resistant" Pseudomonas and MRSP otitis patients
  - E-Testing various antibiotic concentrations from:
    - Aminoglycosides (Gentamicin, Mometamax®; neomycin, Tresaderm®)
    - Fluoroquinolones (Marbofloxacin, Aurizon®; enrofloxacin, Baytril Otic®)
    - Fusidic acid (Canaural®), polymyxins (Surolan®, Otizole®), Triz-EDTA®, others
  - **TOPS panels** instead of Kirby-Bauer/MIC to select topicals

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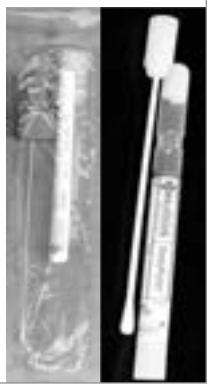
Bacterial Culture Recommendations Until TOPS Testing is Commercially Available

- Chronic/recurring infections
- Severe infections
- Rods on cytology
- Inflammatory cells on cytology
- Topical treatment failure
- Systemic treatment

- Send to the lab with a microbiologist
  - Animal Health Lab (Durda Slavic)
  - None at IDEXX and Antech Labs



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5: Calm the microenvironment

- Steroids and/or Atopica – NOT Apoquel/Cytopoint
- Deliver appropriate volumes
- Claro or Osurnia first line - helps improve compliance
- Vit E - 20 IU/kg TID - minimizes transient hearing loss

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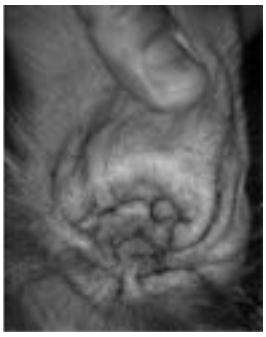
Ears on Apoquel



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Calming the microenvironment

35

Calming the microenvironment

Systemic:

- Dexamethasone – 0.05-0.1mg/kg/d
- Prednisolone – 1-2 mg/kg/d
- Cyclosporine – 5 mg/kg/d

Topical corticosteroid safety ranking

1. Hydrocortisone, Mometasone
2. Prednisolone
3. Betamethasone
4. Fluocinonide, Triamcinolone
5. Dexamethasone

SAFEST

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6. Breakdown Virulence factors

Blue Grotto - Capri, Italy

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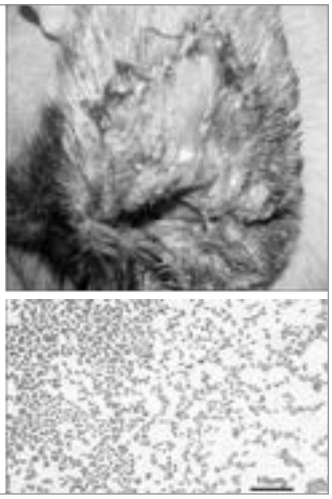
Pseudomonas

- Gram negative rod
- Saprophytic
- Ubiquitous
  - Stagnant water, taps, drains, ear cleaners, solutions
  - Individual pipette cleansers
- Opportunistic
  - Immunocompromised patients
  - Allergies
  - Endocrinopathies
  - ChemoRx Medications



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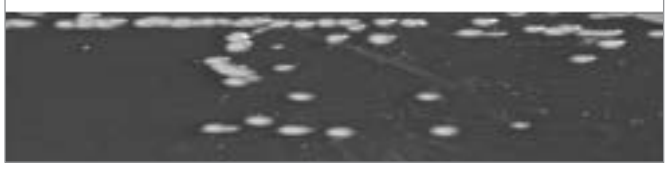
Pseudomonas resistance

- Virulence factors:
  - Low cell wall permeability; B-lactamases; efflux pumps
  - Plasmid, transposon and bacteriophage transfer mutations
- Improper use of antibiotics selects for resistance
  - Failure to reach **effective concentrations** at tissue level
  - **Premature discontinuation** of antibiotics
  - Lack of **owner compliance/adherence** is important



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Tris-EDTA to breakdown biofilm

- Licensed for use in dogs AND cats
- Active ingredient
  - Salicyclic acid 0.2% w/w
- Inactive ingredients
  - Diethylhexyl sodium sulfosuccinate
  - **Trometamol edetate disodium**
  - Chloroxylenol
  - Propylene glycol
  - Hydrochloric acid
  - Pleasant fragrance

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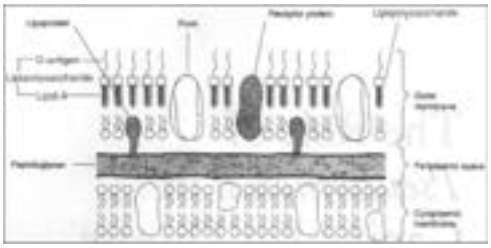
Tris-EDTA mechanism of action

- Tris = Surfactant product ➔ peptidoglycan breakdown
- EDTA = chelates Ca++ ➔ porin channels open
- Instill 15 minutes prior to medicating



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PHOVIA POST-CO2 LASER Surgery

Dean – 4y, MN, mixed breed canine

- Small mass on ventral thorax
- Chronic otitis and stenotic ear canals

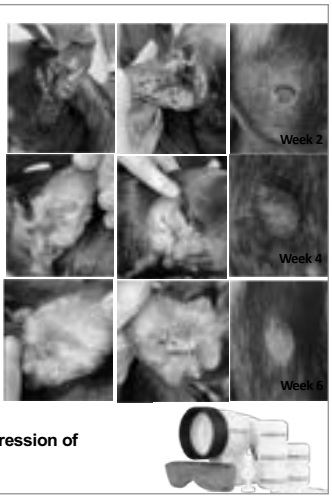
Veterinary Dermatology

2021;32(3):262-e72

Carbon dioxide laser surgery for chronic proliferative and obstructive otitis externa in 26 dogs

- 4 weekly B2B Phovia post CO2 LASER
- Anticipate residual activity

- FLE renders bacteria **hypersusceptible**
  1) **Cell wall damage** in microbes ➔ enhance the penetration of antibiotics
  2) Induced **disruption of biofilm**
  3) Oxidative stress-induced **under-expression of drug resistance genes**



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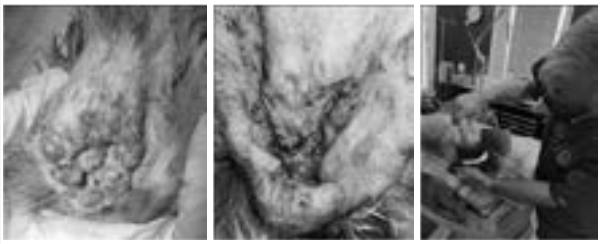
Fluorescent Light Energy post-CO2 LASER Surgery for Chronic Proliferative Otitis Externa



Credit Photo: Dr. Yu

PHOVIA

43

4 weeks post LASER Surgery with Phovia®: 2 cycles once weekly

Credit Photo: Dr. Yu

PHOVIA

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7. Knowledge of Ingredients & VOLUME is Key

Drop versus cc equivalent

Small dog or cat	e.g., Yorkshire terrier	0.25 ml	10 drops
Medium breed dog	e.g., Cocker spaniel	0.5 ml	20 drops
Large breed dog	e.g., Golden retriever	0.75 ml	30 drops
Giant breed dog	e.g., Newfoundland	1.0 ml	40 drops

- Opposite active ingredients
  - Effective against Pseudomonas
  - Switch withOUT duplication
  - Switch if adverse reaction
- Bottle/Syringe delivery
  - Volume coats canal
  - Dispense exact volume
  - Owner can see product
  - ALL meds given SID

45

8. Use of MONTH-long topical otics

- The more you give a client to do,
  The less compliance you will achieve
  The more resistance we will see

- Take ear treatment out of owners' hands
  - Residual otics as FIRST LINE treatment for otitis externa
  - Allergy Tx is complicated, minimize client confusion

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Claro® (Elanco)

- FIRST LINE TREATMENT
  - Florfenicol **16.6mg/ml** (vs 10mg/ml)
  - Terbinafine **16.6mg/ml** (vs 10mg/ml)
  - **Mometasone furoate 2.2mg/ml**
- Liquid formulation - ideal for stenotic ears
- ONE and DONE

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Administration

1. Clean and dry the ear canal
2. Shake the dropperette before use
3. Remove the cap while holding the dropperette upright
4. Turn the cap over and use it to break the seal
5. Screw the applicator onto the dropperette
6. Insert tip of dropperette into affected external ear canal
   OR DRIP INTO EAR CANAL
7. Use **0.5-**1.0 mL depending on size of pet's ears
8. Massage base of ear to allow distribution

****Dr. Yu's adjustments**

CLARO

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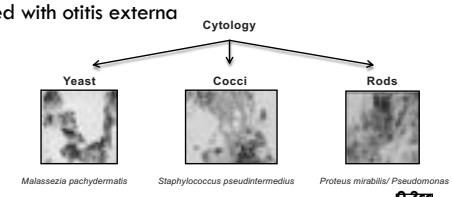
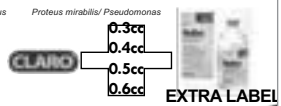
Case Selection

Claro® is indicated for the bacteria and yeast most commonly associated with otitis externa





CLARO

49

Osurnia® - Dechra

- Florfenicol 10mg/ml
- Terbinafine 10mg/ml
- Betamethasone 1mg/ml
- 0.5-1ml per ear
  - Applied Day 0 and ??7??
  - Recheck 28 days after Day 7
- Staphylococcus
- Malassezia
- Pseudomonas only if…

50

Osurnia®, Dechra

- 235 dogs with otitis externa
  - Double-masked field
  - N=159 OSURNIA; N=76 Vehicle
  - 64.78% vs 43.42% (p=0.0094) @ day 45
- Transient loss of hearing
  - Consider Vitamin E ~20 IU/kg TID PO esp 8y+
  - **Oishi** N et al. Ototoxicity in dogs and cats.VCNA. 2012;42(6):1259-1271
- **EXTRA LABEL** use in cats
  - ½ tube per ear

51

Bercovitz GR, et al Long-lasting otic medications may be a rare cause of neurogenic keratoconjunctivitis sicca in dogs. JAVMA 2023; *261*(1): 97-103

- **Retrospective study**
  - N = 29 patients - 76% small breed dogs
  - Onset neurogenic KCS within 1 day after application
  - Documentation of low STT (< 15 mm/min)
- **Findings:**
  - Return of clinically normal tear production = 24/29
  - Median time 86 days (19 to 482 days)
  - Corneal ulcer was diagnosed in 68% (20/29)
    - associated with a longer time to STT ≥ 15 mm/min.
- **CLINICAL RELEVANCE**
  - Good prognosis for return of normal tear production within 1 year.
  - Possibly 1.0ml too much for small breed dogs - vesiculation of TM??

52

9. Three pronged approach to Yeast Otitis Externa

1) Calm the microenvironment
- Oral steroid or cyclosporine
- Potent topical steroid
- Not absorbed systemically

2) Address any infection
- Appropriate antimicrobial
- Malassezia vs Candida

3) Address underlying etiology
- HypoT4, Atopy, Food allergy

53

Antifungal products

- MALASSEZIA spp.
  - Clotrimazole (Mometamax®, Aurizon®)
  - Enilconazole (Imaverol®)
  - Miconazole (Surolan®)
  - Silver Sulfadiazene (Baytril Otic®)
  - Posaconazole (Posatex® - USA)
  - Terbinafine (Osurnia®)
  - Systemic: ketoconazole, itraconazole, fluconazole, terbinafine
- CANDIDA spp
  - Nystatin (Canaural®)

54

10. Maintenance Otic Therapy

- Pending cytology findings
  - If cytology has abnormal levels of yeast or bacteria
    - Monthly Claro
    - Monthly Osurnia
  - If cytology is normal and ear canals are inflammed
    - Twice weekly Cortavance
    - Daily to every other day Burow's HC
    - +/- Monthly Claro or Osurnia

55

What about Burow's HC solution

- Karl August Burow (1809-1874)
- Surgeon and anatomist
- Burow's Solution, Compounded
  - 1% Hydrocortisone
  - 2% Burow's solution (aluminum acetate)
  - Propylene glycol (NOT acetic acid)
- Good for maintenance
- Anti-inflammatory
- Some antimicrobial

56

Can topical hydrocortisone aceponate effectively control allergic otitis externa and reduce the risk of recurrence? A double-blinded, placebo-controlled, prospective study

BERGVALL KE et al, ESVD 2017 page 251

- 0.5 ml HCA daily for 7 to 14 days then...
- HCA or placebo - 2 days/wk for 16 weeks
  - 41 dogs (100%) in treatment improved
  - 38 dogs went onto maintenance
    - 82% of HCA - free of clinical signs
    - No adverse events of long term HCA

57

FUTURE ONCE MONTHLY TOPICALS

- Consider using Claro or Osurnia monthly
- Future direction for Dechra and Elanco:
  - Claro-M - with mometasone only
  - Claro-MT - with mometasone and terbinafine
  - Osurnia-B - with betamethasone
  - DuOtic with betamethasone and terbinafine

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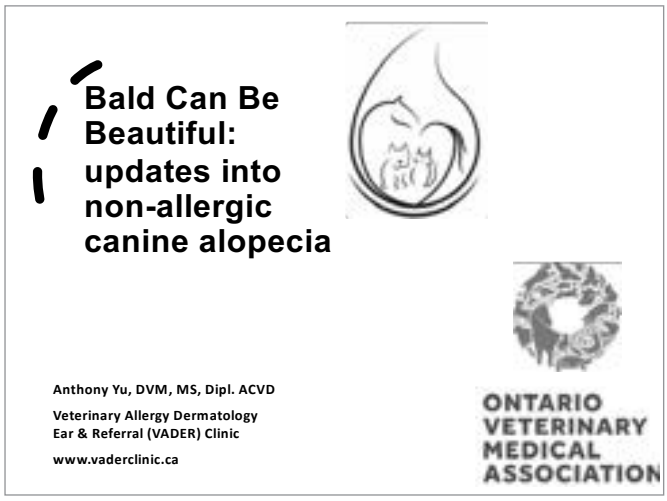
3003

BALD CAN BE BEAUTIFUL

DERMATOLOGY

Anthony Yu, BSc, DVM, MS, DACVD



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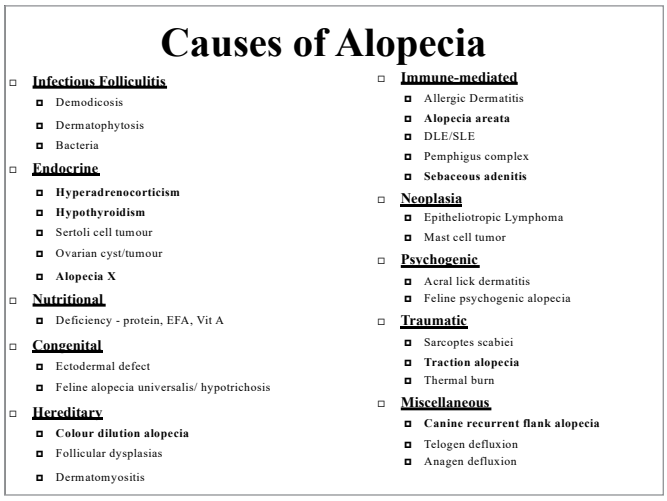

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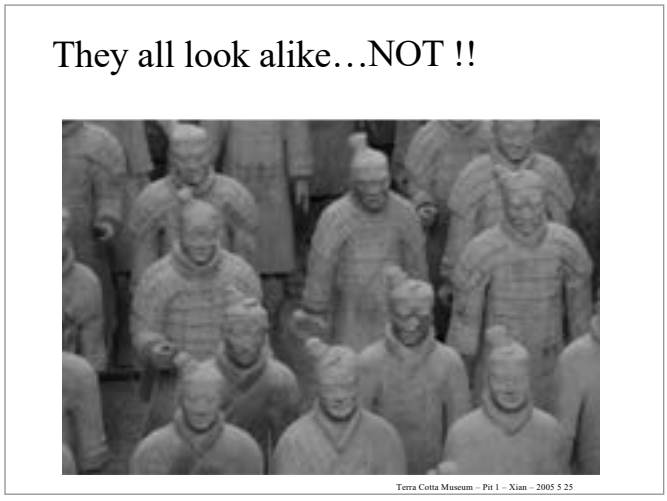

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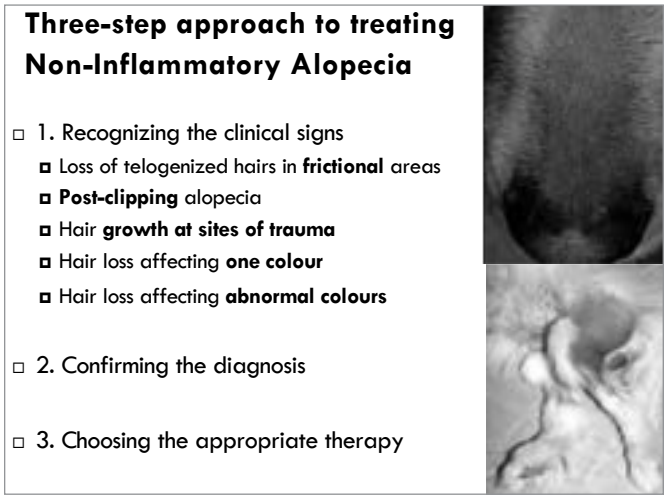


4

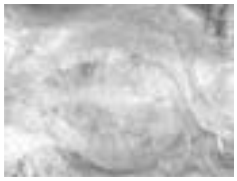
Treatment of Alopecia ….Questions??

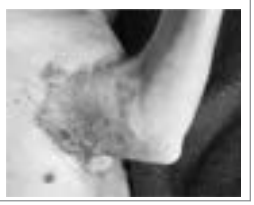
Knit a Sweater !!!

5

Cushing's: the unusual clinical findings

- **Loss of muscle mass**
- **Split ends**
- Coat color change
- Calcinosis cutis
- **Collagen breakdown**
  - **Spay incision**
  - **Poor healing wounds**
- **Anterior cruciate rupture**
- Reproduction failure
  - ↓FSH – failure to cycle
  - ↓LH – testicular atrophy
- **Secondary hypothyroidism**
  - **↓TSH – reversible hypothyroidism**





6

Cushing's - Diagnosis

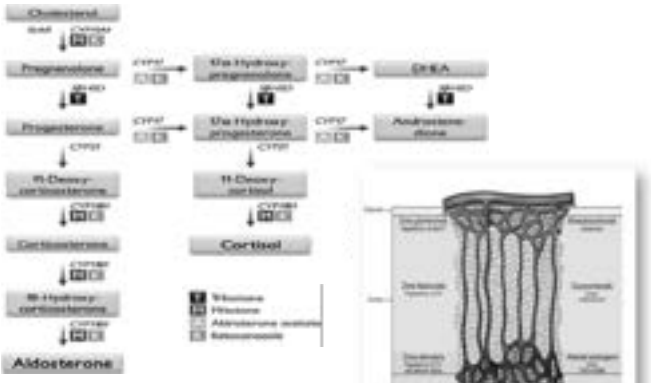
- Urine Cortisol:Creatinine ratio
  - Very few false negatives
  - Collection of 3 sample times at home within 24h
- ACTH stimulation test (5ug/kg IV Cortrosyn)
  - Required as baseline prior to Tx anyway
  - 0 and1 (+/- 2hr)
- LDDST (0.01mg/kg IV)
  - Screening and Differentiating test
  - 0, 4, and 8hr cortisol samples
- Abdominal Ultrasound/CT/MRI

7

Cushing's Syndrome - Treatment

- Pituitary Tumor/*Food-induced Cushings
  - Lysodren® 25-50mg/kg 1-3X/week
  - *Trilostane 1-2.5 mg/kg SID or **divided BID**
  - Ketoconazole, Retinoids
  - Sx:Hypophysectomy or adrenalectomy
  - Radiotherapy
  - Melanocortin 2 receptor antagonists
  - Abiraterone acetate ➔ cP450C17
- Adrenal Tumor
  - Hi Dose Lysodren®, Trilostane
  - Sx: Adrenalectomy
- Adjust thyroid dose

8



Sanders K et al. Treating canine Cushing's syndrome: Current options and future prospects. Vet Journal 2018;241:42-51

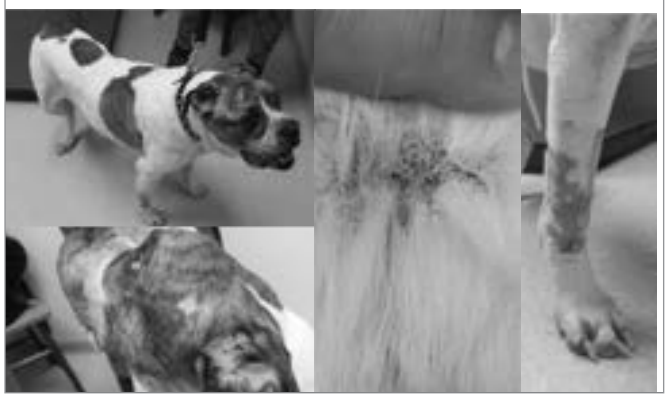
9

Treatment & monitoring

- Trilostane
  - competitive inhibitor of 3β HSD
  - competitive inhibitor of 11β HSD??
  - only treatment for food-induced Cushing's
  - 2-5mg/kg q24h OR 1-2.5 mg/kg BID
  - ACTH 4-6h post-Trilostane 10d, 4wks, 3mos, 6-12 mos
  - goal post-ACTH = 50-150 nmol/L **4-6h post Trilostane**
  - post-ACTH>250 nmol/L double the dose
  - post-ACTH<20 nmol/L, d/c 2d, next lower dose
- SE: cost, lethargy, anorexia, V/D, adrenocortical necrosis or hyperplasia, hypoadrenocorticism

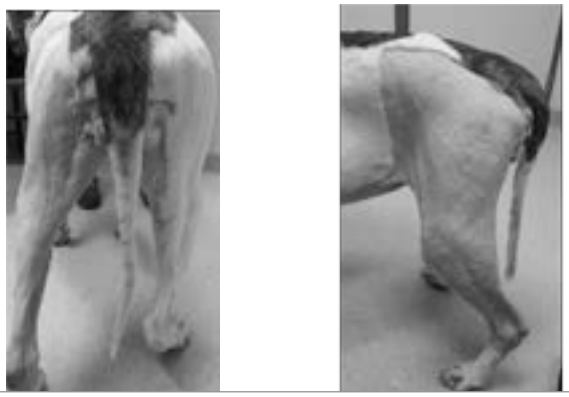
10

Grr – more than post-clipping alopecia



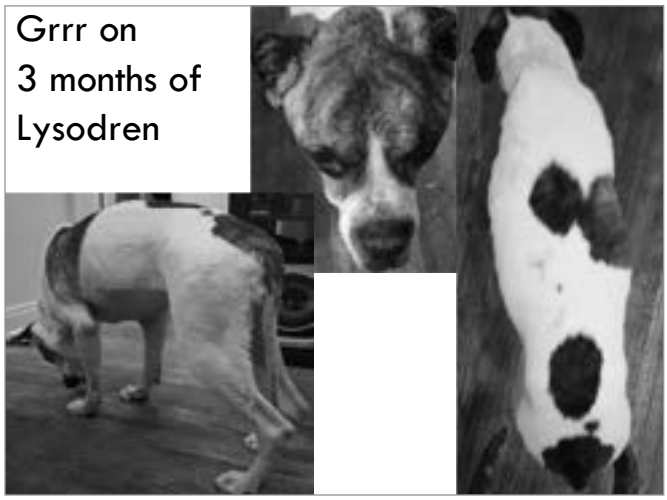
11

Post-Clipping Alopecia …1 year later



12

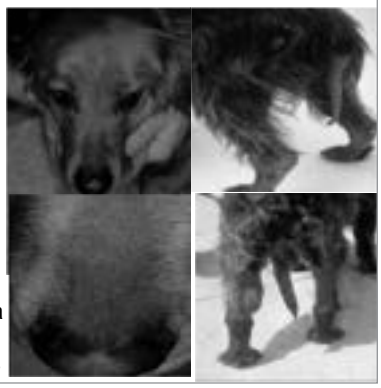
Grrr on 3 months of Lysodren



13

Unusual signs of Hypothyroidism

- Muscle atrophy
- Facial nerve paralysis
- ACL rupture
- Laryngeal paralysis
- Megaesophagus
- Corneal lipid deposits
- Cardiomyopathy

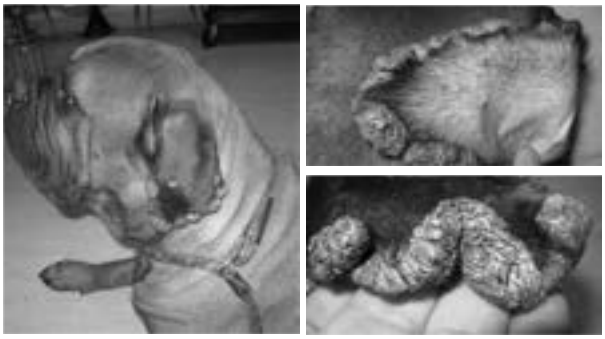


CUTANEOUS SIGNS

- Frictional alopecia
  - nose, tail
- Ceruminous otitis externa
- Acral lick granuloma
- Peripinnal hyperkeratosis

14

Peripinnal hyperkeratosis - Girlfriend 5yo FS Mastiff



Presented for ear margin seborrhea treatment options
Dorsal diffuse hypotrichosis extending onto tail
Was NOT overweight, lethargic or bald

15

Hypothyroidism - Dx & Tx

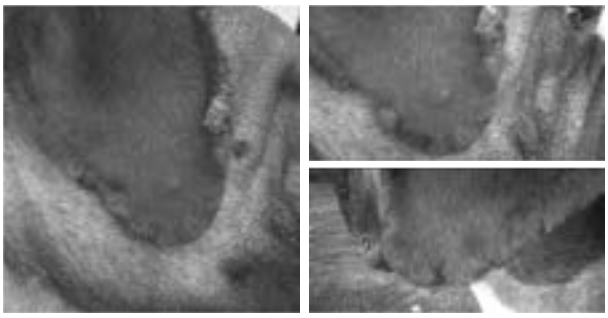
CBC, BIO CHEM, T4, Free T4, TSH
T4, Free T4, TSH
T4, Free T4
T4 - Especially useful on annual Wellness profiles
Look for a downward trend

Response to therapy
3 months – NO long-term effects on HPT axis

- Levothyroxine 0.02 mg/kg **BID**
  - Start at half dose BID for 14d
  - S.E. = anxiety, panting, PU/PD
- Adjust dose if concurrent illness or cardiac compromise
- Monitoring
- **6-hour** post-pill Total T4
- Expected responses
  - **7 days – behavior**
  - **4-6 wks – hair/scale**
  - **3 months - normal**

16

Girlfriend – 3 months post-thyroid



17

"Alopecia X"...fka...

- Adrenal sex hormone imbalance
- Adrenal hyperplasia-like syndrome
- Growth hormone-responsive alopecia
- Castration-responsive alopecia
- Alopecia of plush-coated breeds
  - Pomeranian, Chow
  - Poodle, sled breeds
  - M/F; neutered or intact
- Malamute or Husky "Coat Funk"
- Biopsy-responsive alopecia
- Pseudo-Cushing's Syndrome

18

"Alopecia X": The presentation

- Gradual loss of hair of ventral neck, caudal thighs, trunk
- Hyperpigmentation

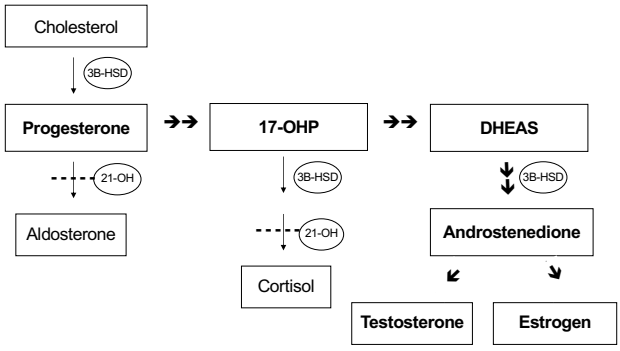
19

Alopecia X - Diagnosis

- Rule-out other endocrinopathies
  - Hypothyroid, Canine Cushing's Syndrome
- Skin biopsy to dermatohistopathologist
- Catagen arrest/Flame Follicles
- +/- U of Tennessee adrenal sex hormone imbalance
  - Pre- and post-ACTH
  - Sent to U of Tennessee on dry-ice
  - Sex hormones, cortisol, 17-OH-progesterone
  - Can be abnormal in haired individuals
  - Atypical Cushing's Syndrome

20

Adrenal Steroidogenesis



21

To Treat or not to Treat?

Atypical Cushing's; Anemia dt hyperestrogenism; Solar damage; Methyltestosterone hepatotoxicity; Suspected animal neglect

- First, spay / neuter if intact

If no response in 3-6 months, then try

- Melatonin (30-40% success)
  - 1 mg/kg/day divided TID
- +/- Knit sweaters

If no response in 3-6 months, then try

- Cyclosporine
- +/- Phovia or LLLT
- +/- Microneedling

If no response in 3-6 months, then try

- Lysodren OR Trilostane

If no response in 3-6 months, then try

- Lupron OR Growth Hormone
- Methyltestosterone or medroxyprogesterone

22

LOW DOSE Lysodren

15-25mg/kg daily 7-14 days, then weekly

- Watch Coumpounding RX

- One month after starting Lysodren

23

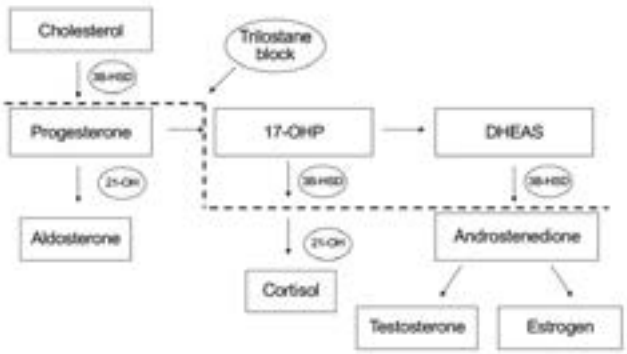
Trilostane

- Orally active steroid analogue
- Competitive inhibitor of the 3 B- hydroxysteroid dehydrogenase enzyme system
- Blocks production of several adrenal steroids

- Possible side effects
  - Hypoadrenocorticism – Addisonian crisis:
    - Electrolyte abnormalities (hyperkalemia and/or hyponatremia); Shivering; Weakness; Reduced appetite/anorexia; Vomiting; Diarrhea
  - Adrenal necrosis

24

Adrenal Steroidogenesis



25

Trilostane

- Cerundolo *et al*, 2004
- 16 Poms and 8 Min Poodles with Alopecia X
- Hair re-growth: 14/16 (85%) Poms; 100% Min Poodles
- Dose: 12 mg/kg and 9 mg/kg, respectively
- Re-growth occurred most often within 4-8 weeks
- In dogs that did not have hair re-growth at that time, the dose was doubled and re-growth occurred

- Cost: Pomeranian (3 kg) $80/mo; Malamute (50 kg) $563/mo

26

6 Months After Trilostane

27

Cyclosporine for Alopecia X?

- Cyclosporin prolongs the anagen phase of the hair cycle

- Cyclosporin inhibits the expression of Protein kinase C
  - → stimulating hair follicle growth and hair fibre production

HARMON et al., 1995
TAKAHASHI AND KAMIMURA, 2001

28

Rocky 8 weeks post-Atopica®

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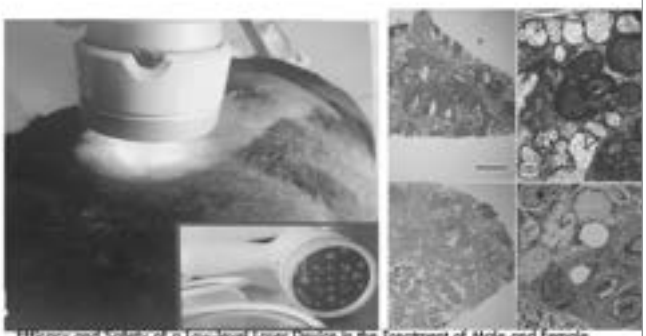
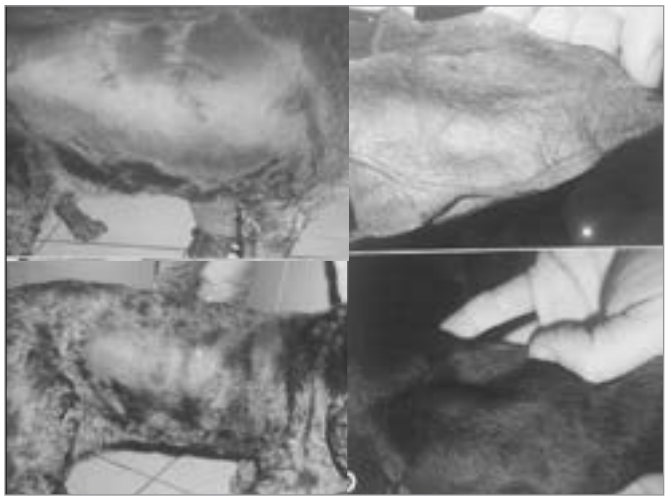
Phovia OR Low Level Laser therapy



Efficacy and Safety of a Low-level Laser Device in the Treatment of Male and Female Pattern Hair Loss: A Multicenter, Randomized, Sham Device-controlled, Double-blind Study
Joaquin JJ et al in Am J of Clinical Dermatology (2014)

30



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Veterinary Dermatology

Microneedling as a successful treatment for alopecia X in two Pomeranian siblings

Steve Stoll*, Christian Dietlin† and Claudia S. Natt-Mettler‡

- Successful in 2 FS 4yr Pomeranian siblings
- NO response to melatonin, deslorelin, topical minoxidil
- Microneedling technique:
  - General anesthesia + Dermaroller MC925
  - Vertically, horizontally, diagonally until microbleeding
- 5 weeks ➔hair re-growth + dec hyperpigmentation
- 12 weeks ➔ 90% regrowth + dec hyperpigmentation
- 12 months follow-up ➔ good coat quality

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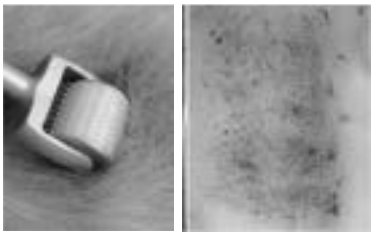
Dermaroller MC 925

2.5mm



Kang et al. **Optimal microneedle length for hair regrowth in hair cycle arrest (alopecia x) in 6 dogs.** Vet Dermatol. 2023;00:1-10

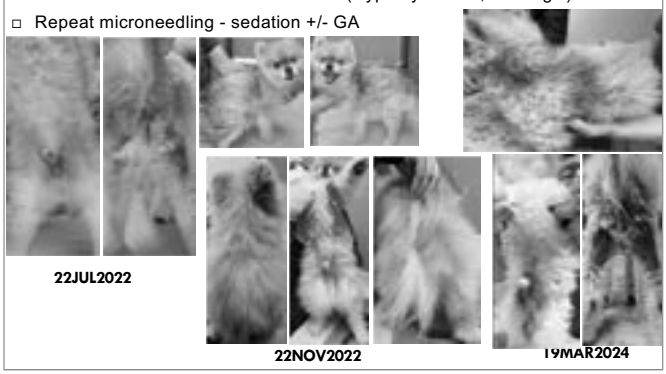
33

Before and 12-weeks after micro-needling

34

If initial response, then relapse...

- Bloodwork to r/o metabolic conditions (Hypothyroidism,Cushing's)
- Repeat microneedling - sedation +/- GA

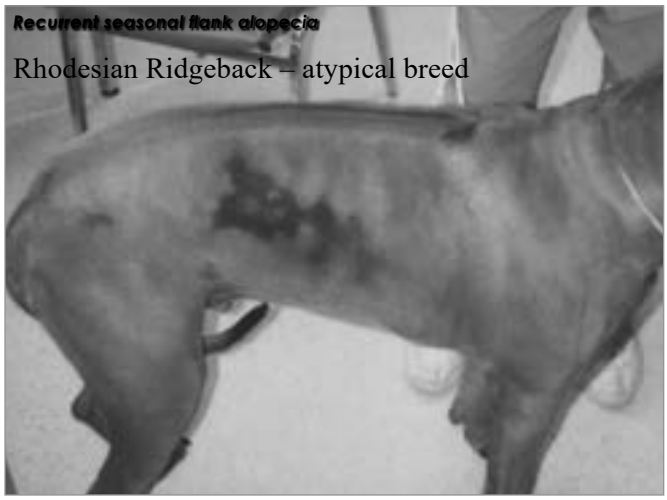


35

Alopecia is not always a bad condition

The Buddhist Monks of Xian

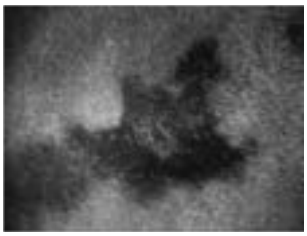
36



37

Recurrent seasonal flank alopecia

- Seasonal, recurrent
- Breed predisposition
  - Airedale, Bulldog, Boxer
- Bilaterally symmetrical
  - flanks
  - hyperpigmentation
- Spontaneous regrowth
  3-6 months later
- Can become permanent

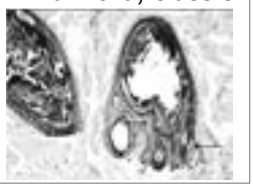


38

Seasonal Flank Alopecia

- Diagnosis:
  - R/O endocrinopathies
  - Trichogram to evaluate for follicular dysplasia
  - Clinical impression

In a word, classic



- +/- Dermatohistopathology
  - distorted hair follicles filled with keratin
  - "witches feet"

39

Seasonal Flank Alopecia

- Recurrent annually
- Fall and Spring
- +/- skip a year

- **Melatonin imbalance**
  - **Fall – insufficient production**
  - **Spring – too much of a drop**

40

Seasonal Flank Alopecia with lichenoid plaques
Melatonin 0.33mg/kg TID
Tetracycline/Niacinamide 500mg TID
Vitamin E 20 IU/kg TID

41

Melatonin

More Than a Jet Lag Pill

- Pineal gland ➔ L-tryptophan ➔ serotonin ➔ melatonin
- Promotes ↑IL-12 ➔Th-1 response
- Enhance IL-2 antitumor/cytotoxic lymphocytes
- ↑ pulsatile Hypothalamic LHRH
- Controls photoperiod-dependent molting/coat color
  - via direct effects on hair follicles
  - via pars tuberalis altered MSH and/or prolactin secretion

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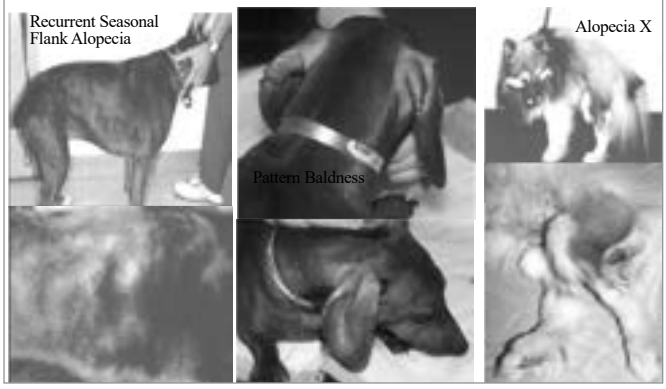
Melatonin Uses



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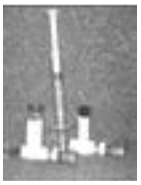
Melatonin

More Than a Jet Lag Pill

- CRFA (50-75%),
- Canine Pattern Alopecia (50%+)
- Alopecia-X of the Nordic breeds (30-40%)
- Oral melatonin – 1mg/kg divided TID
  - **withOUT XYLITOL**
- Injectable melatonin 20mg q2wk X 3
- Implantable melatonin – adverse FB reaction
- Peach fuzz in 4-6wks
- Maximal growth in 3-4 months
- +/- Lethargy, aggressiveness,
- Insulin resistance (not in DM)
- NOT w/neuropathies or breeding dogs



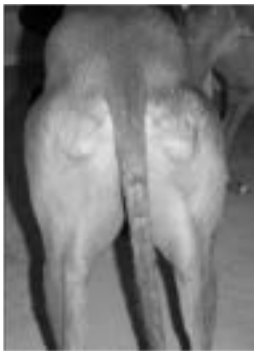
44

BBBTS Greyhounds

- Nonpruritic, noninflammatory alopecia
- Affects the caudal aspect of the pelvic limbs
- Can be progressive and involve the abdomen.

45

BBBTS Greyhounds



46

Non-specific hair growth promoters

- Soft, warm bedding
- Melatonin
  - 1mg/kg divided TID
- Thyroxine
  - 0.1mg/10lbs BID
- Cyclosporine
- Pentoxifylline
  - 10-30mg/kg TID

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Ischemic folliculopathies

Pentoxifylline candidates 10-30mg/kg TID

- bald thigh/bald belly syndrome in greyhounds/whippets
- post-clipping
- traction alopecia
- elbow/hock calluses
- deep scarring pyoderma/pododermatitis

48

CDA: Clinical Signs

- Normal at birth
- Gradual onset of dry, dull coat

- Alopecia on dorsum of trunk
  - Progresses to partial/complete alopecia
  - Hair regrowth is poor
  - Poor quality of remaining hairs

- Usually most severe on trunk/colour diluted areas
  - Often spares head, tail, limbs

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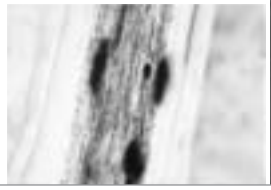
CDA: Clinical Signs

- Secondary superficial pyoderma
- Comedones, scaly
- Usually not pruritic
  - Only if secondary infection
- Trichogram ➔ melanin clumping



50

CDA: Treatment Options

- No known specific treatment
- Hair growth promotion
  - Melatonin – 1mg/kg divided TID
  - Cyclopsorine 5mg/kg PO q24h
  - Pentoxifylline 10-30mg/kg TID
- Help with epidermal barrier
  - Vitamin A/synthetic retinoids
  - Antiseborrheic/antibacterial shampoos
  - Epidermal Barrier Repair Products
    - DouxoSeb, ProHex4, Microsilver+, DermRestore
- Systemic antibiotics to treat secondary pyoderma

51

Canine Sebaceous Adenitis

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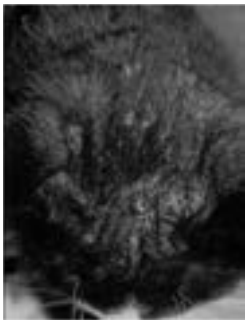
Feline Sebaceous Adenitis



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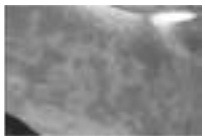
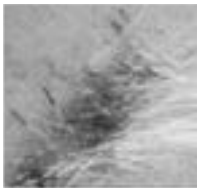
Sebaceous Adenitis

- Immune-mediated attack of sebaceous glands
- Autosomal recessive
- **Follicular casts**
- Breed variation
  - Poodle - hyperkeratosis, alopecia, flat coat
  - Vizsla - annular scaling / alopecia
  - Akita - generalized erythema / seborrhea
  - Samoyed - follicular cast, truncal alopecia/scale
  - Cats - start on head / pinnae, scaling, crusting
- Secondary infections
  - Loss of sebaceous secretions
  - Hairloss resulting in open follicular ostia





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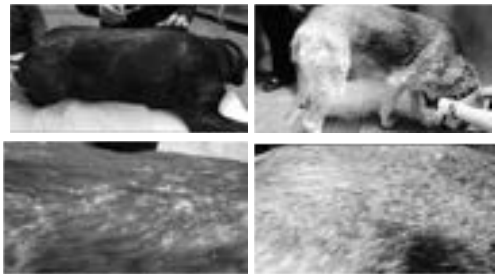
Differentials for Sebaceous adenitis

Infectious folliculitis - Demodicosis, Dermatophytosis, Bacteria
Immune mediated - Pemphigus foliaceus
Endocrinopathy - Hypothyroidism



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Sebaceous Adenitis -diagnosis & treatment

- Dermatohistopathology
  - granulomatous inflammation
  - destroying sebaceous glands
  - +/- absence of sebaceous glands
- Address secondary bacterial infections
- Topical therapy
  - Ophytrium/phytosphingosine shampoo, mousse, pipettes
  - Propylene glycol 50-75% spray

Vet Dermatol. 2024; 35: 238–241

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Sebaceous Adenitis - Systemic therapy

- Omega-3 & 6 Faty Acids,
- High dose vitamin A 1000 IU/kg/d
- Synthetic retinoids
  - Acitretin (Soriatane) 1-3mg/kg
  - Isotretinoin (Accutane) 1-8 mg/kg
- Cyclosporine 5mg/kg
- Oclacitinib 0.6mg/kg q24h + prednisone 0.5mg/kg
- IL-2,-15, IFN-α,-γ,
  - Successful treatment of sebaceous adenitis with oclacitinib and low-dose prednisolone in a dog. *Vet Derm* 2024;35:238
- Consider dietary trial to eliminate trigger

Vet Dermatol. 2024; 35: 238–241

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Sebaceous adenitis

Before & After Atopica and diet trial

58

Remind owners that hair growth takes time

- **Maximum 0.7-2.6 mm/day**
- **AFTER underlying etiology identified and addressed, it may take 10-12 weeks to see significant response**

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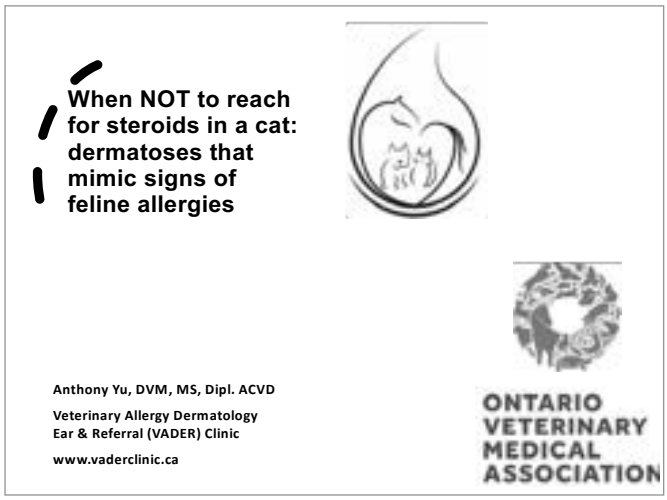
3004

WHEN NOT TO REACH FOR STERIODS IN A CAT

DERMATOLOGY

Anthony Yu, BSc, DVM, MS, DACVD



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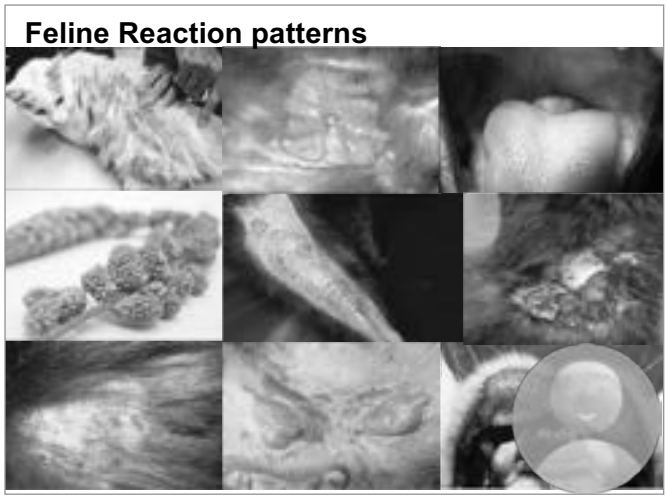

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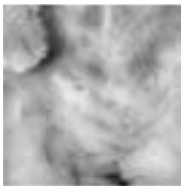
4

Top Feline Dermatoses Differentials

- Parasite:
  - *Notoedres, Cheyletiella, Otodectes*
  - fleas, lice, chiggers
  - *Demodex gatoi or cati*
- Allergy:
  - Atopy, food, contact
  - Mosquito bite hypersensitivity
- Infection/Immune-mediated/Idiopathic:
  - Bacteria, Bartonella, *Malassezia*, dermatophytes
  - Feline herpesvirus/calicivirus/pox/FeLV/FIV dermatitis
  - Pemphigus, Lupus,
  - Drug reaction/erythema multiforme
  - Idiopathic ulcerative and facial dermatoses
- Neoplasia:
  - SCC/SCC in situ (Bowens)
  - Cutaneous T-cell lymphoma
  - Paraneoplastic Syndromes



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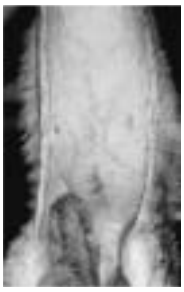
Steroids Are Good BUT…

- Infections
  - bladder infections, skin infections,
  - septicemia, respiratory infections
  - localized/generalized demodecosis
- Alopecia, thin skin, skin fragility (cats)
- Milia-like comedones, follicular cysts
- Calcinosis cutis, atrophic remodeling of scars

- Decompensating hypertrophic cardiomyopathy
- Behavioral changes, PU/PD, polyphagia, panting
- Diabetes mellitus, ESS, increased PTH levels
- Hyperlipidemia, steroid hepatopathy
- Musculoskeletal atrophy
- Adrenal suppression/atrophy

6



Demodex cati - d.t. Steroid Puffer

7

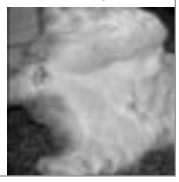
Feline Demodex spp.

Demodex cati

- Miliary dermatitis
- Non-pruritic, non-contagious
- Head and neck involvement
- Hair follicle dweller
- Global
- Dx: deep skin scrapes
- Check for immunosuppression
  - FeLV, FIV, DM, HAC, steroids

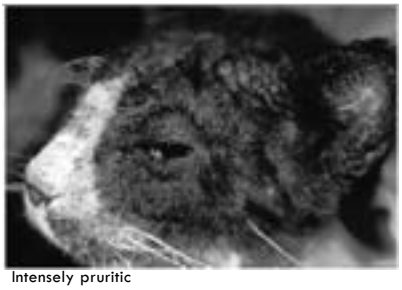
Demodex gatoi

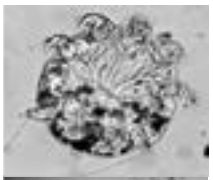
- Symmetric alopecia
- Pruritic, contagious (multiple cats)
- Truncal involvement
- Superficial dweller
- Coastal regions
- Dx: tape, superficial skin scrape, fecal
- Tx if suspected

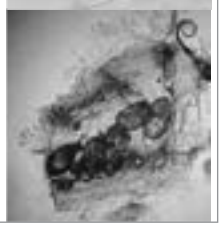


8

Notoedres cati (feline scabies)



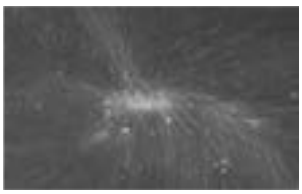


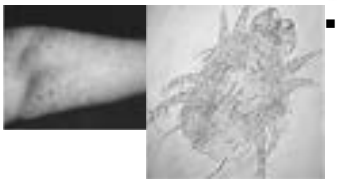


Intensely pruritic
Miliary dermatitis; peripinnal hyperkeratosis
+/- Generalized distribution
Multiple cats affected; human lesions
Skin scrapings - EASY to find

9

Cheyletiella blakei





- Diffuse erythema/crusts, scale
- Dorso-truncal distribution
- **"Walking Dandruff"**
- **Winter time (no flea control)**
- **Owners affected – zoonotic**
- **Treat all in-contact pets**
- Skin scapings
  - **Hooked mouth parts**

10

Otodectes cynotis

- Dark black otic exudate
- Severe erosive, crusting dermatitis
- Head tilt
- Can move from one ear to the other
- **Can involve entire body**
- Hypersensitivity reaction
- Direct visualization
- Ear swabs in mineral oil
- **Treat all in-contact animals**

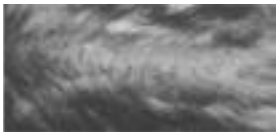
11

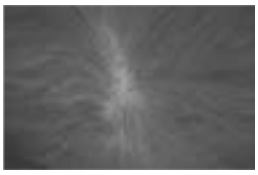
Pediculosis (Lice)

- Felicola subrostrata
- **Nits** attached to the hair
- White spots are scale, lice, nits
- Winter months
- Pruritic
- Multiple cats affected
- **Treat all in-contact animals**

12

Flea allergy dermatitis





- **Rump and neck**
- **Flea dirt**
- Seasonal to non-seasonal
- Steroid responsive
  ONLY IF used concurrently with ectoparasiticide

13

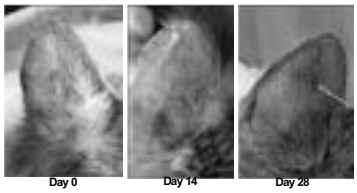
Isoxazolines and combo products treat ALL ectoparasites

14

15

Microsporum canis (Dermatophytosis)

'Cooper' 10m M Siberian show-cat
Asymmetric areas L pinnal alopecia
Folliculitis, furunculosis
PCR and DTM - positive
Rx: Itraconazole 5mg/kg 7d on:off
: Topical terbinafine
: Cattery screening/quarantine



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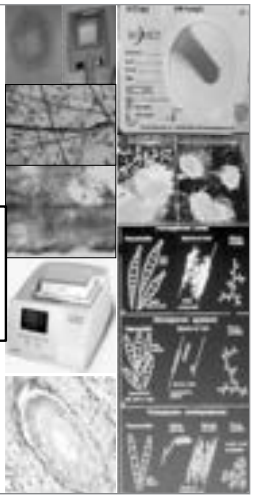
Dermatophytes

Diagnostic options

- In vivo testing: human media (zoonotic)
- Wood's Lamp - tryptophan - apple green
- Trichogram - microconidia
- Skin biopsy - folliculitis, microconidia, hyphae

- Fungal PCR (IDEXX) - DX NOT monitoring
- Fungal culture - Dx + Monitoring Rx response
- Derm Duet (Bacti-Lab) - RSM/DTM
  - InTray DM (BioMed) - enriched DTM
  - Send to central laboratory with mycologist

- Identification is KEY
  - In-house vs mycology lab
  - *T mentagrophytes* ➔ rodent
  - *Nannizia gypsea (fka M. gypseum)* ➔ soil
  - *M canis* ➔ zoophilic/carriers



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Dermatophytosis - topical treatment

Shampoo, Spray, Mousse

- Chlorhexidine + Keto/Miconazole
- Accelerated Hydrogen Peroxide
- Microsilver Plus
- Lime Sulfur

Courtesy of Karin Beale

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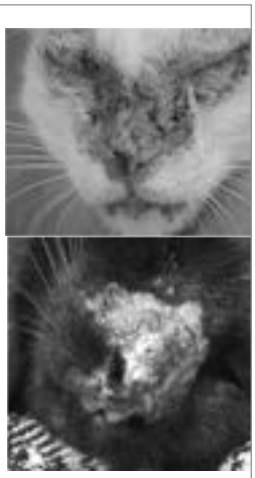
Dermatophytosis - systemic treatment

- IMIDAZOLES/TRIAZOLES
  - inhibits lanosterol 14α-demethylase, inhibiting the conversion of lanosterol to ergosterol in the fungal cell membrane.
  - **Ketoconazole** 10mg/kg q24h or 5mg/kg q12h (large dogs)
    - Caution: Cytochrome P450-3A4 effect
  - **Itraconazole** 5mg/kg q24h (cats, small dogs)
    - Pulse dosing 1 week on:1 week off
  - **Fluconazole** – 5mg/kg q24h
    - Least effect on CP450-3A4; Metabolized by kidney
- ALLYLAMINE
  - inhibits squalene epoxidase in synthesis of ergosterol
  - **Terbinafine** – 30mg/kg q24h
- TWO (2) negative fungal CULTURES 2-4 weeks apart

19

Feline Viral Dermatoses

- **Feline herpesvirus 1 (FHV1)**
- **Feline calicivirus (FCV)**
- HX: respiratory/oropharyngeal infections
- HX: waxing/waning recurrent infections
  - Vesicles, crusts, erosions or ulcerations
  - Necrotizing facial dermatitis
    - Nasal planum, bridge of nose, periocular skin
  - +/- Conjunctivitis/keratitis/corneal ulcers
  - +/- Gingivitis/stomatitis or oral ulcers
- Multiple cats affected
- Immunocompromised
  - Concurrent disease (e.g. Diabetes)
  - Immunosuppressive medications (corticosteroids, cyclosporine, chemotherapy)
- Latency (FHV1 trigeminal sensory ganglia)



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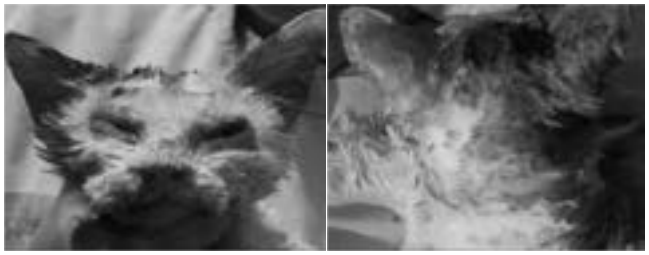
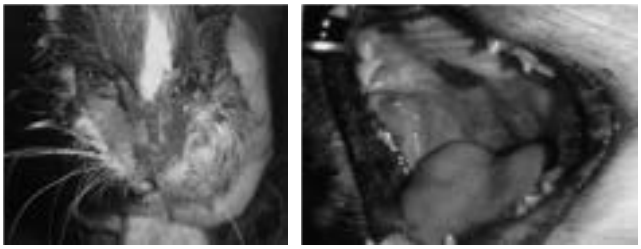
Feline Viral Dermatoses (FHV1)



DDX: Severe allergies, Eosinophilic granuloma, Pemphigus, Lupus, Erythema multiforme lesions

21

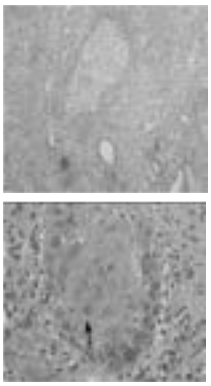
Feline Herpesvirus 1 (FHV1)



22

Feline Herpesvirus 1 (FHV1) - DIAGNOSIS

- Histopathology
  - Neutrophilic **necrolytic** ulcerative dermatitis
  - Intranuclear inclusion bodies
  - Multinucleated giant cells
  - Interstitial inflammatory dermatitis w/eosinophils
  - +/- Immunohistochemistry
- PCR of conjunctival smear (FHV1 Ag)
  - sensitivity 100%, specificity 95%
- Serology



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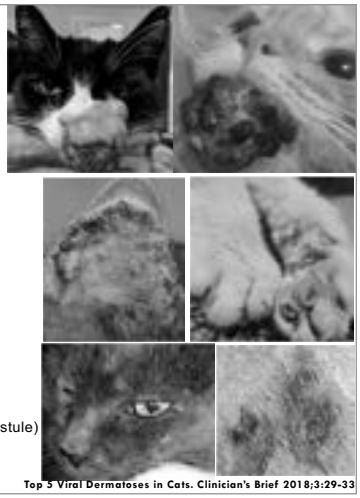
Feline Viral Dermatoses

- **Papillomaviruses (PVs)**
  - Bowens disease (SCC in situ)
  - Sarcoids
  - Basal cell carcinoma
- **Feline Leukemia Virus (FeLV)**
  - oncogenic retrovirus
  - cutaneous horns (paws)
  - giant cell dermatosis
  - cutaneous lymphomas
- **Feline poxvirus**
  - Cowpox virus, wild rodents
  - Europe, Western Asia
  - Pock lesions (umbilicated papulopustule)
  - Head, ears, neck, legs (bite areas)
  - ZOONOTIC



Top 5 Viral Dermatoses in Cats. Clinician's Brief 2018;3:29-33

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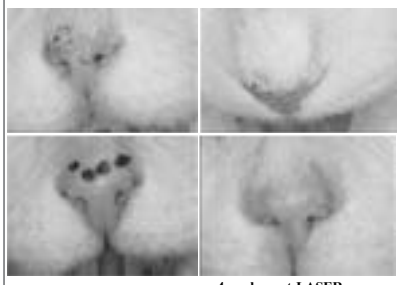
Feline Viral Dermatoses - TREATMENT

- **NO STEROIDS** - May worsen condition (awaken latency FHV1)
- Immunocompetent patient - self-limiting disease
- Lysine - competes with arginine for DNA synthesis (FHV1)
  - Dose: 250 mg PO q 12 hours
- Oral Famciclovir (Famvir)
  - 60-90 mg/kg q12 hours until resolved
- Imiquimod 5% (Aldara) - topical SID-BID
  - Stimulates Toll-like receptors → Local immune
- Feline Interferon Omega (Virbagen; Virbac)
  - Cytokine to boost local immune defense (antiviral)
  - 50,000 IU PO q 24h (dilute vs concentrate)
  - 1.0-1.5 X 10^6 IU/m^2 3X/week subQ
  - Available from the UK and Ireland via EDR

25

Feline Viral Dermatoses

CO2 LASER Surgery - Bowens Disease - SCC in situ due to FeLV



4 weeks post-LASER

8 weeks post-LASER

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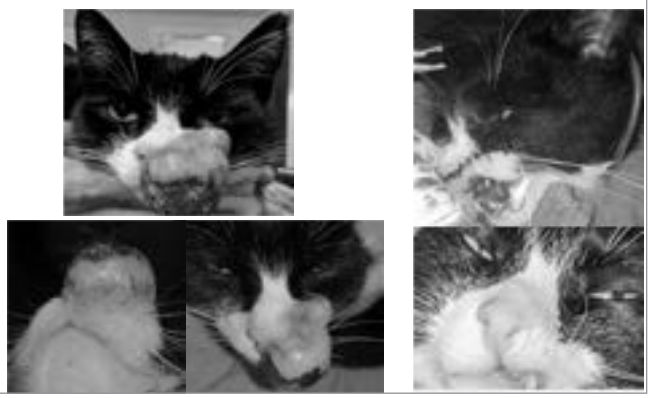
Feline Viral Dermatoses - TREATMENT

CO2 LASER Surgery - Feline Sarcoids



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Psychogenic alopecia - *Pippin. 2 yr MN DSH*

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Psychogenic alopecia – does it exist?

Waisglass, S, Landsberg G. **Underlying medical conditions in cats with presumptive psychogenic alopecia.** JAVMA. 2006;228(11) 1705-1709

- Study: 21 cats w/"psychogenic" alopecia
  - 16 cats: associated medical condition
    - Adverse food reaction, atopy, flea allergy dermatitis, parasitic dermatosis, bacterial dermatitis, hyperthyroidism, hypersensitivity of unknown cause
  - 3 Cats - Medical condition + compulsive component
  - 2 Cats - Psychogenic Alopecia

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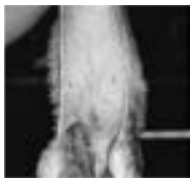
Psychogenic Alopecia

- Behaviour modification
  - Remove stressor if possible;enrich environment
  - E collar
- Behaviour Modifying medications
  - Feliway®
  - Tricyclic Antidepressants
    - Amitriptyline - 5-10 mg/cat q12-24h
    - Clomipramine -1.25-2.5 mg/cat q24h
  - Selective Serotonin Reuptake inhibitors
    - Fluoxetine - 0.5-1 mg/kg q24h
    - Paroxetine - 0.5-1 mg/kg q24h
    - Sertroline - 0.5-1 mg/kg q24h
  - Minimum 6 weeks, then taper

30

Hyperadrenocorticism

- Adrenal tumour, ACTH from pituitary
  - Iatrogenic
  - Rare
- Thin, fragile skin, poor wound healing
  - Hyperpigmentation
  - Flanks and trunk
  - Curling of ear tips
  - Non-pruritic
- PU/PD, polyphagia, hepatomegaly
- Insulin resistant diabetes
- Dx: CBC/Chem/UCCR/HDDST+ACTH



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Hyperadrenocorticism - Treatment

- Taper off glucocorticoids if iatrogenic
- Surgery if obvious tumour
- Trilostane
  - Competitive inhibitor of 3β-hydroxysteroid dehydrogenase
  - Inhibits aldosterone and cortisol synthesis
  - 15-30 mg per cat q 12-24 hrs
- Metyrapone
  - Inhibits cortisol synthesis via steroid 11β-hydroxylase
  - 65 mg/kg, PO q 12 hrs
- Monitor ACTH stim test q1-3 months, then 2X/year
- Diabetes often resolves - adjust insulin

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Paraneoplastic Alopecia

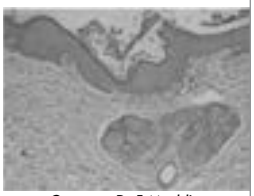
- Older cats
- Systemic disease
- Pancreatic adenocarcinoma
- Biliary carcinoma

- Progressive alopecia
  - Hair epilates easily
- Thin, shiny skin
  - Stratum corneum slough
- Footpad involvement

33

Paraneoplastic Alopecia

- Biopsy
  - Atrophy of hair follicles
  - Absence of stratum corneum
  - Perivascular inflammatory infiltrate
- Diagnostic imaging
  - Abdominal ultrasound
  - Thoracic radiographs
- Treatment
  - Removal of tumour
  - Symptomatic therapy?



Courtesy: Dr E Mauldin

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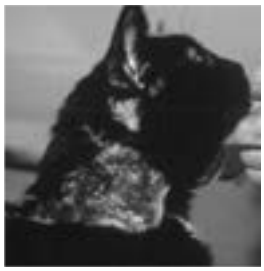
Feline idiopathic ulcerative dermatitis

- Unknown etiology
  - ?injection reaction?, vasculopathy
  - check FIV/FeLV/FHV1 status
- Necrotic ulcer on back of neck
- Exacerbated by self-trauma
- Diagnosis
  - Dermatohistopathology
  - R/O Vx-induced Fibrosarcoma
- Therapy:
  - Address secondary bacterial infections
  - Physical barrier (duct tape)
  - Neurogenic pain modulators
    - Amitriptyline 1-2 mg/kg BID
    - Apoquel 1-1.5mg/kg BID 14-28d, then q24h

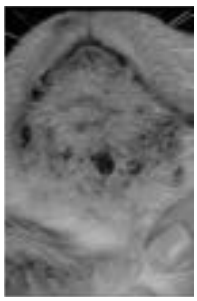


35

Feline Idiopathic Acne

- **Follicular keratinisation disorder**
- Common in cats
- Comedones (blackheads)
- Chin, lower and upper lip
- Papules and pustules if 2ry infected

- Diagnosis
  - Rule out other etiologies
  - Skin Scrapes, DTM, Cytology
  - Dermatohistopathology



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Differentials for Feline Acne

Eosinophilic granuloma complex

Dermatophytosis

Squamous Cell Carcinoma

37

Feline Chin Acne

- **P**arasite:
  - *Demodex cati*
- **A**llergy:
  - ****Atopy, food, contact**
- **I**nfection/**I**mmune-mediated/**I**diopathic:
  - Bacteria, Bartonella
  - ****Malassezia,** dermatophytes
  - Drug reaction/erythema multiforme
  - ****Idiopathic feline chin acne**
- **N**eoplasia:
  - SCC/SCC in situ (Bowens)
  - Cutaneous T-cell lymphoma

** Steroids may be indicated

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Feline Acne ~ Treatments

- **Address underlying etiologies**
- Benign neglect – overTx may worsen lesion
- Clip hair, warm water compresses
- Benzoyl peroxide shampoo
  - q 1-2 d until lesions resolve, then as needed
- **Topical options**
  - **Mupirocin or Silver Sulfadiazine ointment/cream**
  - **2.5% benzoyl peroxide gel**
  - **0.01-0.025% tretinoin gel**
  - **0.75% metronidazole gel**
  - **Topical clindamycin, erythromycin or tetracycline**
- Systemic
  - Antibiotics, antifungal
  - Vitamin A, retinoids (Accutane®) if needed

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In Summary:
Before you reach for steroids in the PRURITIC cat

1) Flea comb
2) Fecal exam
   - Ingested parasites (e.g. Demodex gatoi), Hair
3) Ectoparasiticidal therapy
   - Isoxazolines
4) R/O dermatophytosis
5) R/O Viral etiology especially if ulcerative

THEN....

6) Steroid trial
   - Catopy (100% response)
   - FAD (100% response if w/parasiticidals)
   - CAFR (50% response)
   - Psychogenic (0% response)

40

One Last Word About Cats…

- Cats only eat what THEY want.
- Cats are in touch with the inner barbarian.
- Cats don't like pills.
- Cats don't like elixirs.
- Cats don't like water.
- Cats remember!
- Are steroids bad….

Maybe not so much in some cats

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Question?

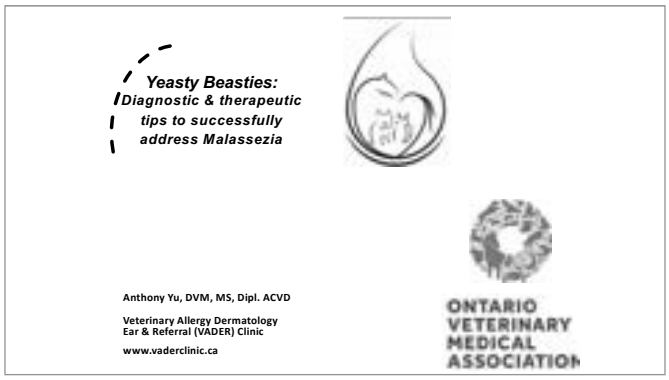
42

3005

YEASTY BEASTIES

DERMATOLOGY

Anthony Yu, BSc, DVM, MS, DACVD



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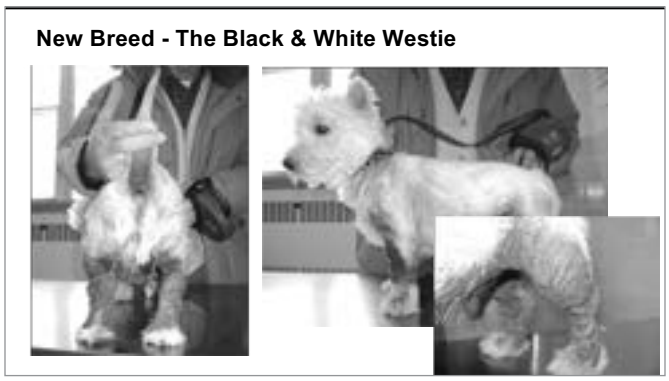

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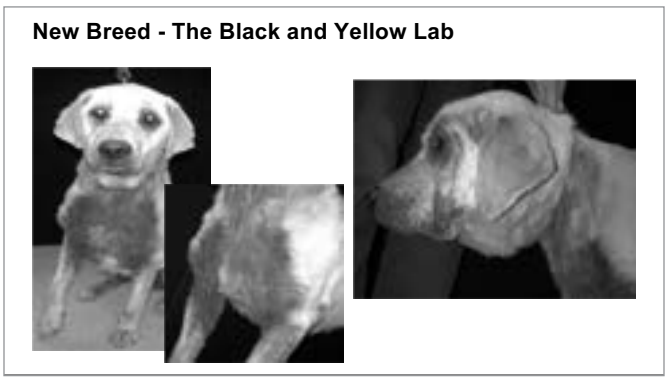

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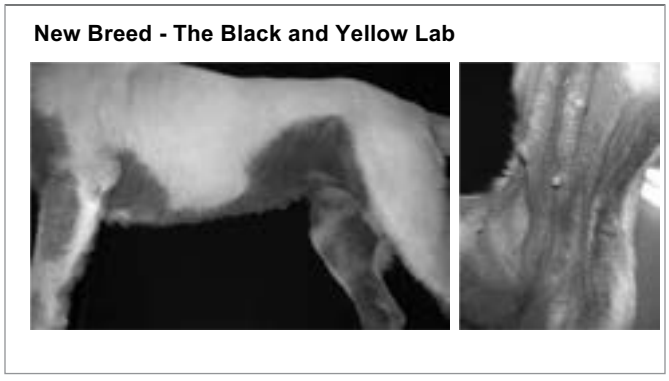

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6

Malassezia History

1889 – *M furfur* in humans
1925 - Indian rhinoceros with an exfoliative dermatitis
1950 - otic pathogen of dogs
1989 - cause of canine otitis Advances in Vet Derm. 1989;Vol 1:412-416
1990's - common cause of canine dermatitis

7

Malassezia - currently 18 species and counting

M. furfur	Humans, Cats
M. pachydermatis	Dogs, Cats +/- Humans
M. sympodialis	Humans, Animals
M. globosa	Humans, Animals
M. obtusa	Humans, Cats
M. slooffiae	Humans, Pigs, Cats (claws)
M. restricta	Humans, Cats
M. dermatis	Humans, Cats
M. japonica	Humans, Cats

M. nana	Cats, Horses, Cattle
M. yamatoensis	Humans, Cats
M. caprae	Goats
M. equina	Horses
M. cuniculi	Rabbits
M. arunalokei	Humans
M. brasiliensis	Parrots
M. psittaci	Parrots
M. vespertilionis	Bats

8

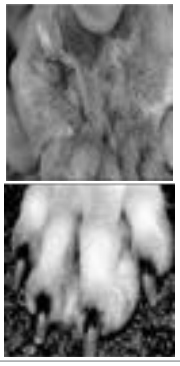
Malassezia Dermatitis

Intensely pruritic
- Zymogen → activates complement → IL-31
- Incomplete response to steroids, Apoquel, Cytopoint, Atopica

Erythema of chin / perioral skin - lower lip fold
- Frenzied facial pruritus in dogs
- Misdiagnosed as neurological disease

Malassezia pododermatitis
- Interdigital erythema and hyperplasia
- Brown waxy interdigital discharge
- "Brown line" on nails

Secondary to underlying etiology
- Allergic dermatitis (food, environmental)
- Endocrinopathy
- Primary seborrhea/keratinization disorder
- Fold dermatitis/Intertrigo
  - lower lip, perivulvar, neck, interdigital, stenotic/hyperplastic ears
- Epidermal Dysplasia Syndrome of WHW??



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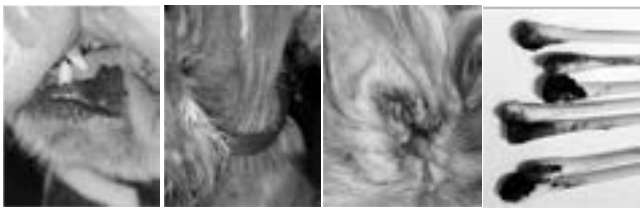

Malassezia in dogs

- PRURITUS !!!
- Erythema, alopecia
- Greasy exudation and scaling
- Secondary excoriation, lichenification, hyperpigmentation
- Generalized cases can be associated with a rancid odor
- Otitis externa

10

Malassezia in Cats

- Devon rex and Sphynx cats
- Pruritus, erythema, self-excoriation
- Interdigital/nailbed brown discharge
- Feline chin acne
- Seborrheic brown ventral abdomen
- Less commonly lichenification

- Allergies & Otitis Externa
- Idiopathic facial dermatitis
  - Persian/Himalayan
- Paraneoplastic alopecias
- Pancreatic adenocarcinoma
- Superficial necrolytic dermatitis
- Thymoma exfoliative dermatitis

11

Dog with yeast on its back.

Not all of them Are this obvious!

Don't forget to do cytology

12

Malassezia are KERATINOPHILIC...

Need sticky tape that will pull your own eyebrows out

Adhesive Tape Impression Tips

3M™ ScotchPad™ Packaging 3750P Tape Pad
- Purchase online, supply store (e.g. U-Line)
- Stickier than regular scotch tape
- Tends not to curl as readily when staining
- Can also use as pharmacy label cover

- Place tape on skin and strip 5X

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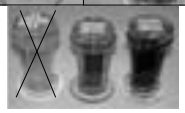
Staining Acetate Tape Prep



AVOID METHANOL DIP
AS IT WILL REMOVE GLUE
AND ANY ATTACHED ORGANISMS

14

Stained Acetate Strip Test with *Malassezia*

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Cytology Tips Summary for *Malassezia*

- Impression smear - edge of slide to scrape skin
- Scalpel blade - to remove brown wax from nails
- Q-Tip - rub vigorously - *Malassezia* are keratinophilic
  - Ears - sample lesion site - not always H-V junction
  - Roll samples across short length of slide
  - Include more sites; focus your search
  - Spend less time staining
  - Save the environment
- Heat fix - *Malassezia* are lipophilic
  - w/BBQ lighter (X-Lite WP)
  - Blue flame
  - Condensate evaporates
- Stain with Diff Quik
- Scan 3D w/fine-tuning knob for *Malassezia*

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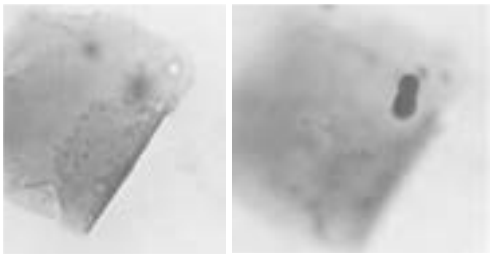
Heat Fixing a Slide for Staining & Cytology (video)

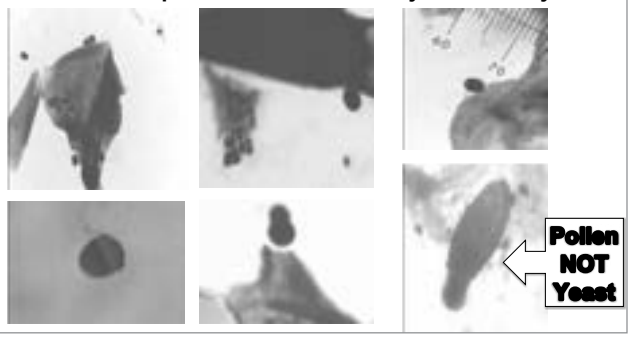
17

Malassezia are KERATINOPHILIC...
Use Your Microscope Fine-Tuning Knob



18

The different shapes of *Malassezia...they don't always bud*



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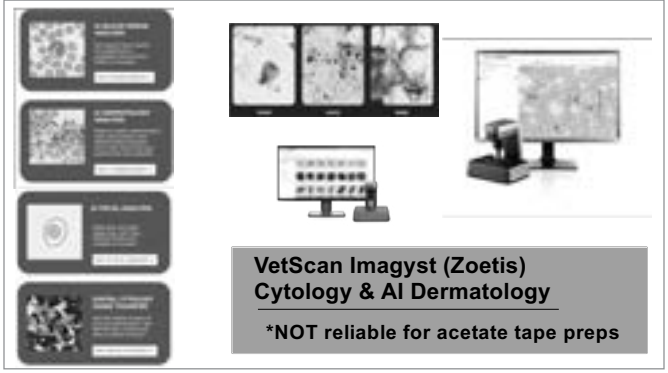
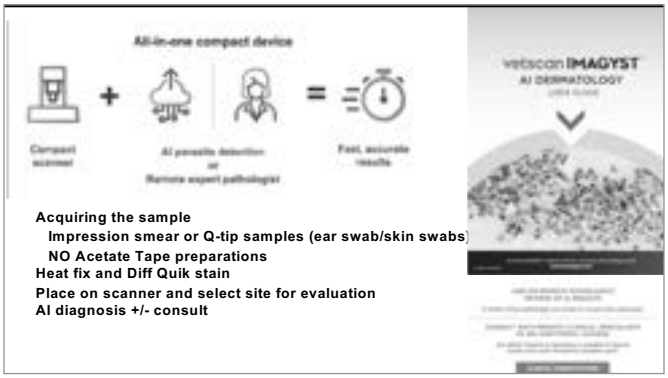


VetScan Imagyst (Zoetis)
Cytology & AI Dermatology

***NOT reliable for acetate tape preps**

20



Acquiring the sample
- **Impression smear or Q-tip samples (ear swab/skin swabs)**
- **NO Acetate Tape preparations**

Heat fix and Diff Quik stain
Place on scanner and select site for evaluation
AI diagnosis +/- consult

21

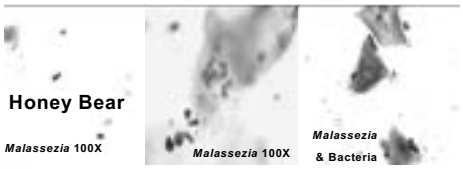
VetScan Imagyst; Zoetis

22

Skin Cytology Interpretation

What number is significant?

Which cytology belongs to Honey Bear?



Location, location, location – normally ears >>>>skin
Clinical relevance : 1 = relevant if pruritic or lesional
: 75 = irrelevant if non-pruritic or normal

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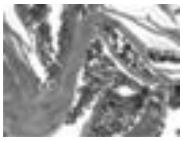
Diagnosis - Culture, Molecular & Histopath

Culture - Modified Dixon's agar
- Slow-growing and lipid-dependent
- Susceptibility - $$/drug (Texas)

Molecular diagnostics
- Next Generation Sequencing
- Quantitative rt-PCR

MiDOG

Histopathology
- Only evident in large #'s
- Lost in fixation
- Acanthosis and spongiosis
- Lymphocytic exocytosis
- Lichenoid pattern with basal cell vacuolation (cats)



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Malassezia Hypersensitivity

****Need to break the vicious cycle**

25

PAX testing for *Malassezia*

Measures circulating IgE

Crude extract & component
- Mala p, Mala s 1, 5, 6, 9, 11

USA 20,000+ PAX submissions
- 1.3-3.4% of submissions
- Correlation with response to ketoconazole???

Immunotherapy
- Crude extract
- Component - not yet

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Immunotherapy is still our best long-term approach.

Mueller, R. S. A systematic review of allergen immunotherapy, a successful therapy for canine atopic dermatitis and feline atopic skin syndrome. *JAVMA. 2023;261*(S1):S30-S35.

ONLY treatment that MODULATES pathomechanism of atopic dermatitis
All other treatments = symptomatic

- **Potential to eventuate a cure/remission**
- **Subcutaneous (SCIT; RUSH; Intralymphatic)**
- **Oral immunotherapy (SLIT; OIT; trypanophobia)**
- **Weight-independent dosing - cost efficacious**
- **Fewest side effects**
- **Improved Quality of Life**

Kotnik T. Quality of Life of Allergic Dogs Treated with Allergen-Specific Immunotherapy — A Retrospective Study. *Vet Sci.* 2023;10(2):72.

- **Follow-up and client education paramount to success**
- **My personal response rates: 70-100%**

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Calming the microenvironment

29

Bathing Pets with Malassezia Dermatitis
- Use COOL/COLD H2O
- 1-3 times weekly
- CHX + miconazole or ketoconazole combination shampoo
- 4% Chlorhexidine +/- climbazole shampoo/spray/mousse
- Microsilver PLUS +/- 4% Chlorhexidine
- Accelerated Hydrogen Peroxide
- Enilconazole dips
- Lime sulfur (3%)

Benefits:
1) Wash off superficial allergens
2) Rehydrate the skin
3) Control yeast overgrowth

30

Using Ear Medications on the skin

Apply and massage a thin film into the affected area(s)
- **interdigital skin, lower lip margins, perivulvar region, ventral aspect of the base of the tail**

Once daily for 14 days, then decrease to TWO CONSECUTIVE DAYS WEEKLY

Allow 15 minutes of contact time to allow for absorption of active ingredients

31

Treating Atopic & *Malassezia* Dermatitis

½ Atopica® dose (2.5mg/kg vs 5mg/kg) when combining with 2.5mg/kg ketoconazole

If discontinuing KCZ, increase Atopica® dose
If decreasing frequency of Atopica/KCZ ➔ same day @ same time

32

The effect of ketoconazole on whole blood and skin concentrations of cyclosporine
Gray L, et al. Vet Derm 2012:23 suppl:13

Treatment Groups
- **T1 = 5mg/kg Atopica**
- **T2 = 2.5mg/kg CSA**
- **T3 = 2.5mg/kg CSA + 5mg/kg KCZ**
- **T4 = 2.5 mg/kg CSA + 2.5 mg/kg KCZ**

Results
- **Blood: T3 (644ng/ml) >> T4 (417ng/ml), T1 (307ng/ml) > T2 (169 ng/ml)**
- **Skin: T3 (1.2 ng/mg) >> T4 (0.7ng/mg), T1 (0.6ng/mg) > T2 (0.26ng/mg)**

Adjusting the Dose of Atopica
- **e.g. 30kg dog with yeast**
- **Normal Atopica dose @ 5mg/kg = 150mg**
- **2.5mg/kg KCZ (75mg) + 2.5mg/kg Atopica (75mg)**
- **Consider instead: 5mg/kg KCZ (150mg ➔200mg) + 1.67mg/kg Atopica (50mg; 67% less Atopica) = Cost savings and fewer side effects**

33

Antifungal Resistance Regarding *Malassezia pachydermatis*: Where Are We Now?

Potential mechanisms of resistance
1) **reduced affinity of the target for the azole (lanosterol demethylase)**
2) **energy-dependent efflux mechanism ➔decreased intracellular accumulation of azoles**
3) **up-regulation of target enzyme ➔antifungal agent being overwhelmed**
4) **development of bypass pathways ➔ergosterol replaced by precursor 14α-methylfecosterol**

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Seattle, WA | USA
May 9 to 12, 2023

In vitro susceptibility of *Malassezia pachydermatis* to low-level light therapy

DR. NATALIA GIL ; DR. DOMENICO SANTORO , DR. ROSANNA MARSELLA

University of Florida

In vitro study - Malassezia pachydermatis grown on culture plates
Culture plates exposed to Blue LED from Phovia Lamp (2 min + 30 sec break between exposures)
31.2% - 59.2% decline in colony counts with 4 - 8 min of exposure versus placebo
Effect using chromophore gel in vivo likely better and no resistance concerns

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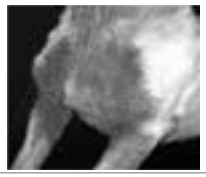

Malassezia Dermatitis Treatment Summary

- **1) Address underlying etiologies**
  - Environmental and/or Food Allergies
- **2) Calm the microenvironment**
  - Steroids, cyclosporine
- **3) Address the yeast**
  - Topical Shampoo and Mousse
    - **Chlorhexidine + Miconazole/ketoconazole**
    - **Microsilver Plus**
    - **Topicals only - 2-week deadline for improvement**
  - Systemic
    - **Ketoconazole 5-10mg/kg SID-BID**
    - **Itra-/Fluconazole – 5mg/kg q24h**
    - A single-blind randomised study comparing the efficacy of fluconazole and itraconazole for the treatment of Malassezia dermatitis in client-owned dogs. Vet Dermatol. 2024; 35: 284-295
    - **Terbinafine – 30mg/kg q24h**
    - **Pulse Rx for hypersensitivity**
  - Fluorescent Light Energy
  - Malassezia Immunotherapy
    - **10,000-20,000 PNU weekly SQ (SCIT)**
    - **1500-3000 PNU daily PO (SLIT)**

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Malassezia Dermatitis Honey Bear 8 wks after CSA/KCZ

37

38

4001

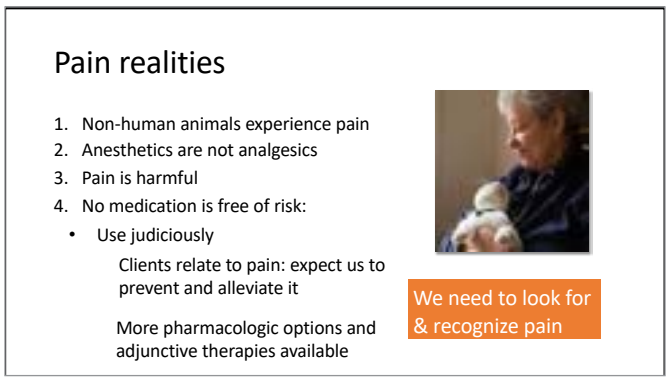
IT HURTS – MAKE IT STOP

FELINE

Margie Scherk, DVM, DABVP

1

2



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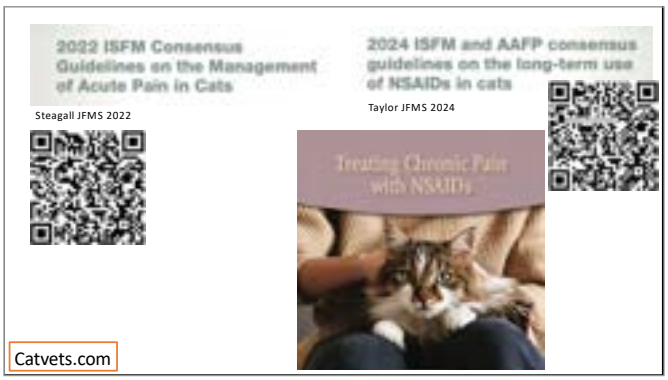


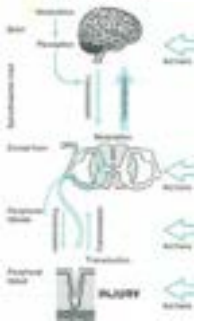
6

Understanding pain

7

Initially protective, becomes pathological

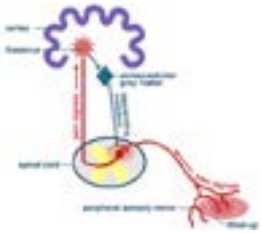
Stimulation of nociceptors
- Crush/tear
- Thermal
- Chemical



8

NMDA receptors

- Dorsal horn of spinal cord
- Also in brain
- Activated by chronic, painful stimuli => allodynia, hyperalgesia, neuropathic pain
- Responsible for opioid tolerance



brainchemist.wordpress.com

9

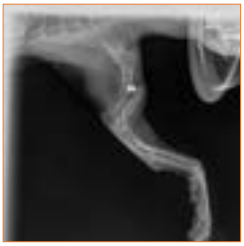
Stress response to pain

Increases survival (short-term)
- ➡ Sympathetic tone:
  - ⬆ Vasoconstriction
  - ⬆ HR and cardiac output
  - ⬆ Respiratory drive
  - ⬆ Muscular tone
  - ⬇ GI tone
- ➡ Fatigue and ⬇ end tissue O2 levels

10

Acute vs. chronic pain

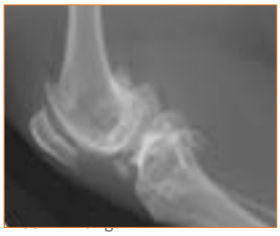
- **Acute**: associated w tissue damage and serves to change behaviour in order to minimize /avoid damage
- Optimizes conditions in which healing can take place
- Stops once healing is complete
- Self limiting!



www.herald.ie

11

Acute vs. **chronic** pain



- **Chronic**: persists beyond the expected healing process
- Serves no biological purpose
- Does not have a clear end-point
- Can have significant effect on physical wellbeing and psychology of the sufferer

Radiograph: John Graham

12

Pain => Catabolic state

- Chronic pathologic pain ➡ *exhaustion* and *delayed wound healing*
- Hyperglycemia
- Protein catabolism
- Lipolysis
- Retention of water & Na^{++}
- Increased K^{+} excretion
- Decreased GFR
- Sleep deprivation

- *Especially* critical in critical patients!
- Pain hurts
- Prevents healing
- Pain KILLS

blog.ferplast.com

13

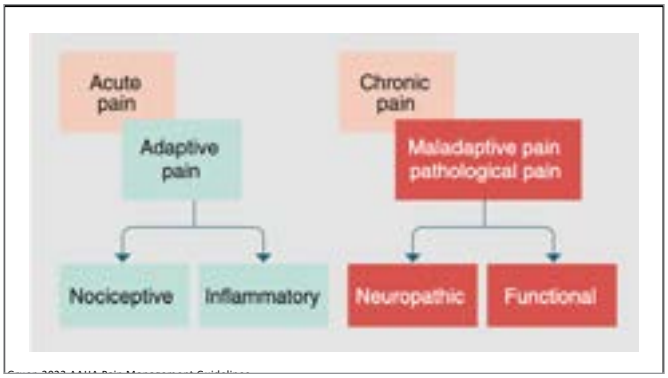
Chronic pain

- **Maladaptive** – dysfunctional
  - Doesn't support healing
  - Persists
  - Reflects nervous system damage &/or remodeling

Chronic pain may be considered to be *a disease state*

Honestpaws.com

14

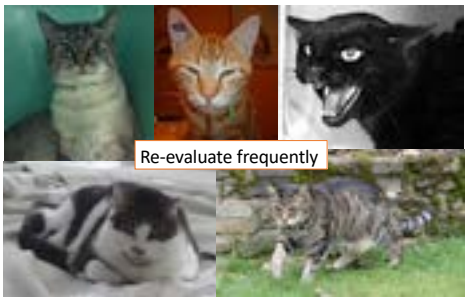


15

Inflammation: -itis!

16

Pain is different for each individual



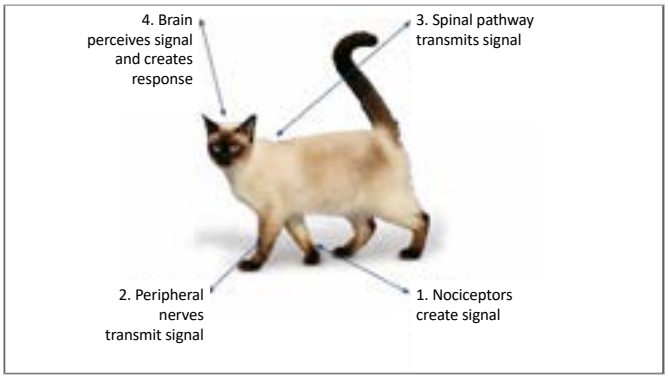
17

Pain: more than chemistry

- Pain is influenced by complex interactions between numerous internal and external factors;
- These may result in increased or decreased pain perception

Monteiro Vet Clin N Am 2020

18



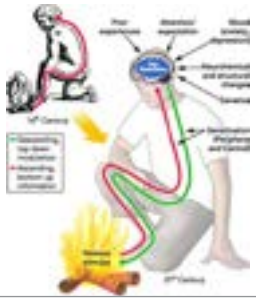
19

Q: How many places can be addressed with multimodal analgesia?

a. Two
b. Three
c. Four ← At least!
d. Five

20

Uncontrolled pain => neuropathic pain



- "Wind-up"
- Hyperalgesia
- Allodynia

21

Neuropathic pain

- Initiated by a primary lesion, injury or dysfunction in the peripheral or central nervous system
- Changes occur in the peripheral nervous system, spinal cord, brainstem and brain
- Damaged nerves fire spontaneously becoming hyper-responsive to both inflammatory and normally innocuous stimuli.
- ANY CHRONIC PAIN condition can develop a neuropathic component

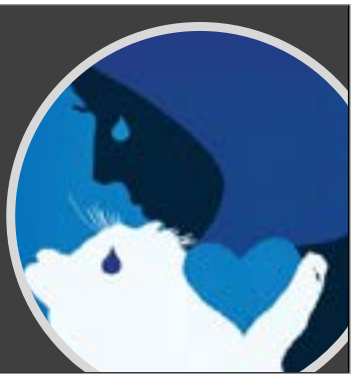
22

Prevent through adequate and multimodal analgesia

Pre-emptive and therapeutic, for a long enough duration

23

Consequences of inadequately treating pain



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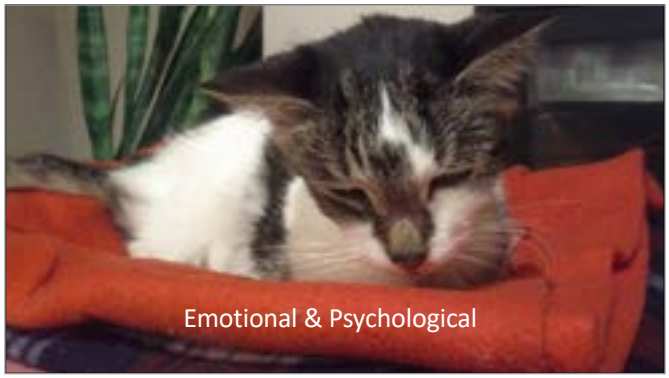


25

26

Catvets.com

FREE!

2022 AAHA Pain Management Guidelines for Dogs and Cats*

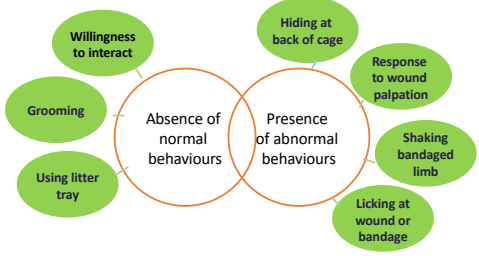
27

Pain Management Standards (AAHA/AAFP)

- Assess in every patient
- Record in the medical record for every patient evaluation *regardless of the presenting complaint*
  - Pain: Yes or No
  - Grade 1-10/10

28

Behavioural indicators of pain in cats



Rely on practice staff observing normal behaviours prior to surgery

29

How do we detect pain?

- Normalization of behaviours after analgesics given = pain was present
  - Re-evaluate often!

30

31

Behaviours indicating acute pain in cats (1)

- Change in body posture or body position
- Decreased activity and/or playfulness
- Decreased interest in the environment
- Decreased willingness to interact
- Decreased appetite
- Abnormal gait or shifting of weight
- Sitting or lying in abnormal positions (reflecting discomfort and protection of an injured area)
- Quiet, hiding
- Hissing, growling or fear-related aggressiveness

Monteiro WSAVA JSAP 2022

32

Behaviours indicating acute pain in cats (2)

- Attention towards a specific area of the body (usually involving surgical wounds)
- Guarding behaviour
- Stop grooming (or increased grooming in a specific location)
- Tail flicking
- Hunched position and/or a tense abdomen
- Difficulties grasping food and increased head shaking during feeding
- Depression and immobility; appears tense and distant from the environment

33

34

35

Altered facial expression

36

Mouse facial pain scale

37

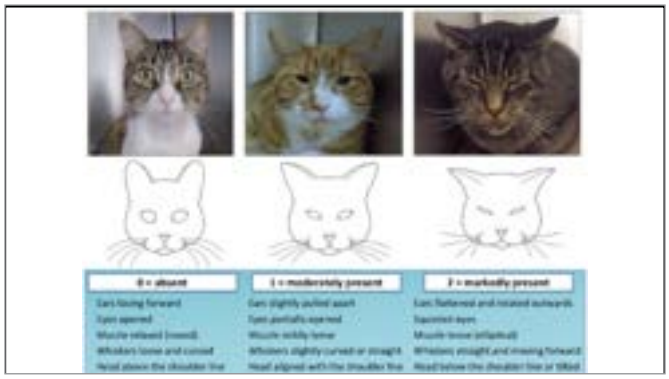
ABSTRACT PRESENTED AT THE AVA SPRING MEETING GRENADA (MARCH 12 & 13th, 2018)

Facial expressions of pain in cats: development of the Feline Grimace Scale

Authors: [redacted], R Watanabe', E O'Toole', DSJ Pang', PV Steagall'

Grimace Scale

38



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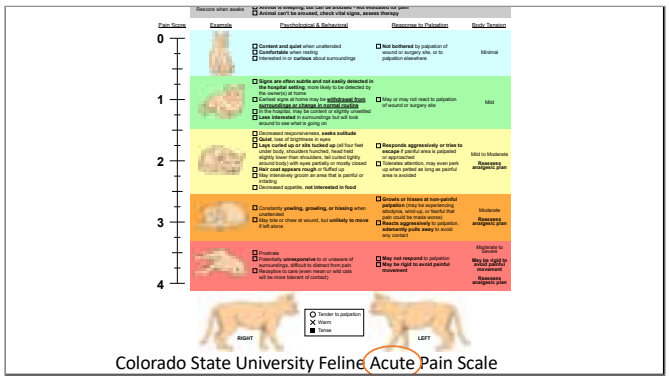


42

Not always straight forward even ACUTE pain

- Factors that can confound pain scores in cats

1. The cat's personality and demeanour
2. Fear and stress
3. Upper airway disease
4. Anaesthetic drugs
5. The observer
6. A full bladder

43

It's harder to detect pain in:

- Adults and older cats: more stoic
  - Procedural pain
- Severely ill, obtunded patients: not able to show behavioural signs of pain

44

Fear and stress

Fear and stress

Fear and stress or pain? – post-op Palpation required

45

Upper respiratory signs challenging

- Half-closed eyes with tight muzzle and whiskers pulled back
- Upper respiratory vs. acute pain

Image from Robertson In Practice 2018

46

47

Acute pain

1. If in doubt: assume that
   a) procedure will be painful - prevent pain,
   b) pain will be present – treat it
2. Pain is different for each individual
3. Know medical status to choose appropriate analgesics

- Re-evaluate frequently!

48

General approach to treating pain

- Start early
- Be thorough
- Harder to combat pain once it is established
- Preempt pain where possible
- MULTIMODAL
- Long enough
- Recognize that *chronic* disease isn't static

49

Q: What are the top three rules in pain management?

Choose three

a. Start early
b. Be thorough
c. Harder to combat pain once it is established
d. Preempt pain where possible
e. MULTIMODAL
f. Long enough
g. Recognize that *chronic* disease isn't static

50

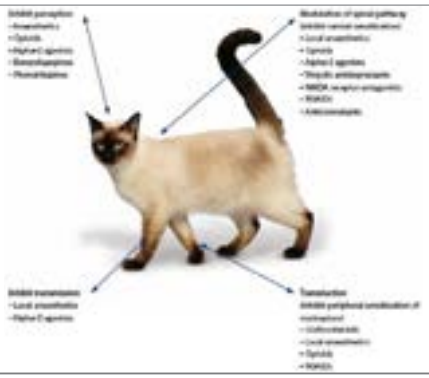
Treating acute/peri-operative pain

- ALL surgery is painful therefore should be pre-empted and treated with frequent reassessment
- Duration of analgesic requirement should be tailored to individual patient
- **Gold standard is *response to treatment***
- Facial, postural and behavioural components
- Reassess frequently
- Be prepared to change protocol
- Starting point = "predictable" pain
- Pain doesn't just STOP after 24 hours!

51

Safe and effective analgesia

Multimodal



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Prioritizing Pain Management Therapies

Gruen 2022 AAHA Pain Management Guidelines

53

Gruen 2022 AAHA Pain Management Guidelines

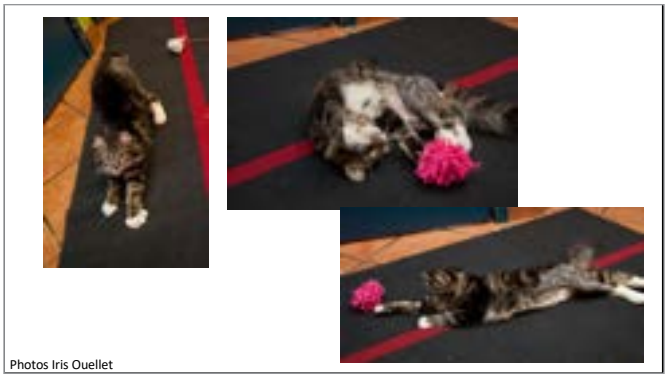
54

NSAIDs

NSAIDs are indicated for:

- Acute & chronic pain
- Mild to severe pain
- Effective musculo-skeletal pain control
- Strong anti-inflammatory action

55



Photos Iris Ouellet

56

57

58

Q: Do you use local anaesthesia? (more than one choice)

a. Dental blocks
b. Other nerve blocks
c. Inside cavities/through drains
d. No

59

Chronic and Neuropathic Pain in Cats: More than Musculoskeletal

60

61

CHRONIC PAIN IN CATS
Recent advances in clinical assessment

CHRONIC

- Feline Musculoskeletal Pain Index
- Client Specific Outcome Measures (CSOM)
  - www.cvm.ncsu.edu/research/labs/clinical-sciences/comparative-pain-research/clinical-metrology-instruments
  - Undergoing validation
- Montreal Instrument for Cat Arthritis Testing
  - One for client, one for veterinarian
  - Undergoing validation

Monteiro JFMS 2019

62

Gradual changes of chro ic pain

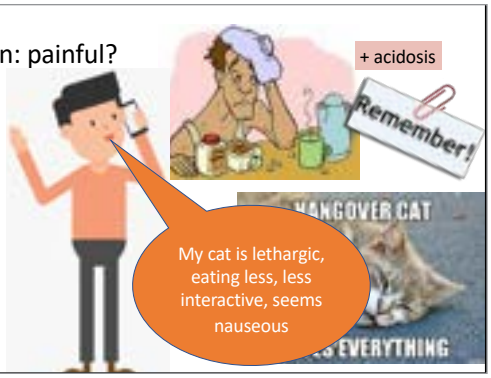
- Masters o ce
  - Feign well
- "Just gettin
- "Just slo
- Less energy, sle
- Chronic pain: l ss pre able n acute ain

Dehydration
Metabolic acidosis
Pain
Hypokalemia
Inadequate dietary protein
Muscle wasting
Anemia

63

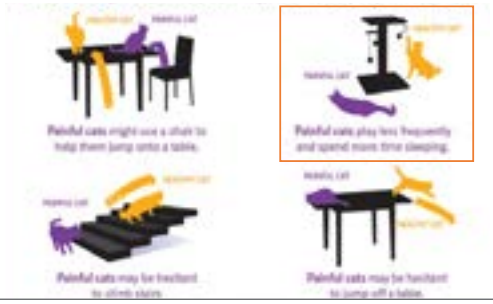
Dehydration: painful?

- Headache?
- Inappetence?
- Nausea?
- Lethargy?
- Grumpy
- Constipation



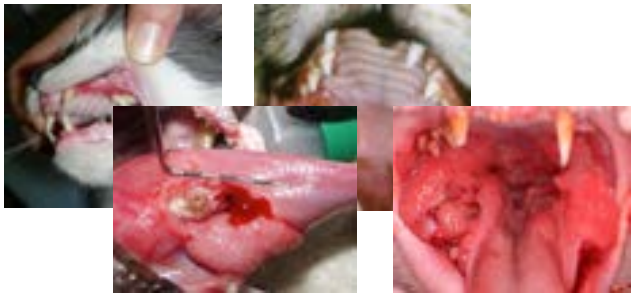
64

Normal Aging? PAIN



65

Chronic pain: oral disease



66

Pain: common in older cats

- Oral diseases
- Urinary tract
- Neo
- erative joint disease
- Procedural pain
- Sterile/idiopathic cystitis and IBD (not age related)

Any -itis can result in transient or neuropathic pain

Image from Facebook

67

Recognizing and assessing pain

- General **mobility** (e.g. ease of movement, fluidity of movement)
- Performing **activities** (e.g. playing, hunting, jumping, using a litter-box)
- **Eating, drinking**
- **Grooming** (& scratching)
- Resting, observing, **relaxing**
- **Social** activities involving people and pets
- **Temperament**

68

69

Treatment of chronic pain

- Depends on:
  - Underlying cause
  - Duration
  - How well managed in the past
- Cornerstones = NSAIDs + adjunct drug therapies, physical and other approaches
- Correct dehydration
- Client education observation and reporting
- Regular reassessment

70

Concerns regarding analgesics

- Hydration
- Renal function
- Hepatic function
- Intestinal health

Balance effects: uncontrolled pain vs. risks of drug

71

Degenerative Skeletal Disease: -itis

- ***Lameness may not be present***
- Insidious signs
- Inappropriate elimination (near box)
- Decreased grooming
- Dislike being combed
- Reluctance to jump
- Moving less
- "Grumpy"
- Seeking seclusion

At what age does osteoarthritis start in cats?

72

Cross-Sectional Study of the Prevalence of Radiographic Degenerative Joint Disease in Domesticated Cats

DJD pain started at 1 year of age!!!

Objective: To determine the prevalence of radiographic signs of degenerative joint disease (DJD) in a randomly selected sample of domestic cats.
Study Design: Prospective observational study.
Animals: Client-owned cats.
Methods: Cats (n = 100) from a single practice and equally distributed across 4 age groups (0–5; 5–10; 10–15, and 15–20 years old) were randomly selected (regardless of health status) and sedated for orthogonal radiographic projections of all joints and the spine. Quasi-Poisson regression analysis was used to investigate the relationship between patient demographics, blood biochemistry, hematologic and urine analysis variables, and DJD severity.

Lascelles Vet Surg 2010

73

All-age incidence of DJD radiographic changes

Axial (55%)
- Thoracic 7-10
- Lumbosacral
- Lumbar
- Cervical

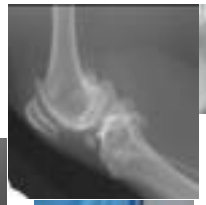
Appendicular (91%)
- Hip
- Stifle
- Hock
- Elbow

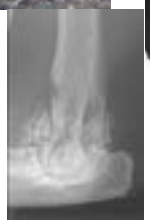
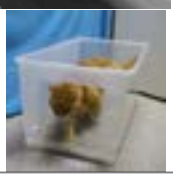
Lascelles Vet Surg 2010

74

Video courtesy Susan Little

75


Radiographs: John Graham

76

Meloxicam Safety & Efficacy: Evidence

Long-term safety, efficacy and palatability of oral meloxicam at 0.01–0.03 mg/kg for treatment of osteoarthritic pain in cats

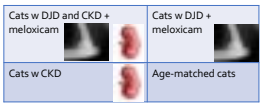
- Mean treatment duration of 5.8 months
- No deleterious effect on renal function was detected in cats studied
- Gastrointestinal upset in 4% of cats was the only adverse effect noted

Gunew JFMS 2008

77

Meloxicam Safety & Efficacy: Evidence

- There was no difference in sequential serum Cr or USG measurements b/n the 'non-renal group' treated with meloxicam vs. control cats not treated with meloxicam.
- There was less progression of renal disease in the 'renal group' treated with meloxicam compared to the age- and IRIS-matched cats with CKD not given meloxicam.



Gowan JFMS 2011, 2012

78

Clinical safety of robenacoxib in feline osteoarthritis: results of a randomized, blinded, placebo-controlled clinical trial

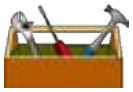
- 193 cats, include 40 w CKD (IRIS 1-4)
- SID robenacoxib for osteoarthritis for 1 month
- Blinded, placebo controlled prospective
- Well tolerated
- No renal, hepatic or GIT damage

- Since 2018, licenced in the EU for treatment of pain and inflammation associated with musculoskeletal disorders for both short and long-term

King JFMS 2015

79

Meloxicam or robenacoxib use?



- Use lowest effective dose
  - Titrate: But *not above* label dose
  - Re-evaluate your patient!
- Use cautiously (~~or not at all~~) in cats with moderate-severe (IRIS stages 3, 4) renal disease
  - Ensure/optimize hydration
- Avoid in patients with known bleeding disorders or pre-existing GI bleeding
- ***Multimodal therapy***

80

Do not give NSAIDs with

- Corticosteroids
- Aspirin
- Be careful with ACE-inhibitors, diuretics, warfarin, phenobarb, chemo agents

81

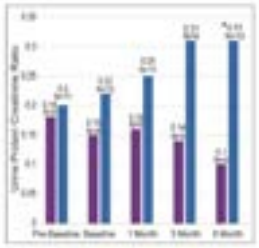
Effects of low-dose meloxicam in cats with chronic kidney disease

- 21 cats stable IRIS stage 2 or 3
- 1 group placebo, 1 group meloxicam 0.02 mg/kg/d X 6 months
- No statistical difference on BP, BUN, Cr, SDMA, GFR, urine transforming growth factor-ß:creatinine ratio, urine clusterin, urine cystatin B or serum inosine
- 4 cats adverse GI effects
- And…

KuKanich JFMS 2020

82

- Mean UPC was greater in the meloxicam group (0.33) than the placebo group (0.1) at 6 months (*P*=0.006)
- Weigh the *risk of potential increased proteinuria* against quality of life benefits when considering meloxicam for analgesia in CKD cats



Placebo; Meloxicam

KuKanich JFMS 2020

83

2024 ISFM and AAFP consensus guidelines on the long-term use of NSAIDs in cats

Taylor JFMS 2024

Catvets.com

84

Opioids

- Cornerstone of managing moderate to severe pain
- Highly efficacious and remarkably safe
- Perioperative setting, preemptively and as part of multimodal protocols
- Useful to reduce inhalant anaesthetic dose
- Not very useful for arthritis (other than possibly breakthrough pain)
- Vary widely in potency , efficacy, receptor specificity and duration

85

Tramadol in Cats

- Opioid derivative with effects on serotonin and norepinephrine reuptake
- Poor bioavailability in dogs (65+/-38%) but much better in cats (93+/-7%)
  - Unpredictable production of active metabolite
- Adjust doses in patients with liver or renal impairment
- Side effects: sedation (esp. if in combo with amantadine or gabapentin), constipation, seizures (rare)
- 1-4 mg/kg BID-TID (Plumb's)

86

Other analgesic agents

- Local anaesthetics
  - Numerous delivery modes
- Alpha 2 adenoreceptors agents
- Ketamine
- Amantadine
- Gabapentin
- Imipramine and amitriptyline

87

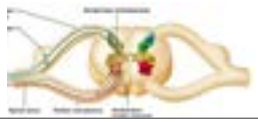
Alpha 2 adrenoreceptor agonists

- Analgesic adjuvants
  - Supplement analgesia and reduce stress
- Reversible (sedation, but analgesia is also reversed)
- Xylazine, romifidine, dexmedetomadine
- Hepatic metabolism, renal excretion
- Only use in HEALTHY animals due to significant hemodynamic changes
- Contraindications: cardiopulmonary disease, hypo/hypertension, diabetes, hepatic/renal failure

88

NMDA receptor antagonists: Ketamine

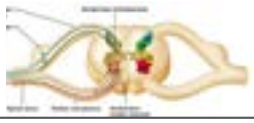
- Modulates central sensitization
- Anti-hyperalgesia effects
- Opioid effects
- Use for major surgery, trauma and to desensitize chronic pain
- For surgery: bolus with 0.5 mg/kg IV, then CRI 0.3-1.2 mg/kg/h
- Discharge, switch to amantadine



89

NMDA receptor antagonist: Amantadine

- NMDA receptor antagonist
- 3-5 mg/kg PO q24h
- High doses can => seizures



90

Cancer pain

- Some but not all
  - Oral cavity, bone, genitourinary system, eyes, nose, nerve roots and gastrointestinal tract cause considerable pain
  - Bone
  - Neuropathic pain
- Pain is likely to be present and more severe as cancer progresses
  - Pain can be associated with the cancer itself, diagnostic procedures, or treatments, or it can be unrelated to cancer
- We are afraid of cancer pain

Monteiro WSAVA JSAP 2022

91

Treating cancer pain

- Pain can be associated with the cancer itself, diagnostic procedures, or treatments, or it can be unrelated to cancer
- NSAIDs +
- Opioids, gabapentin
- Bisphosphates, chemotherapy, radiation (as warranted)

92

Gabapentin

- Mode of action not fully understood, many pathways
- Few studies in cats
- Suggested for chronic pain esp. if neuropathic
  - Amputation, diabetic neuropathy, pelvic trauma, intervertebral disc disease
- Start with 5 mg/kg PO q12h, then increase/decrease dose depending on response
  - Treat several weeks
  - Gradual withdrawal is recommended
  - May be sedating

93

Novel mediators of pain

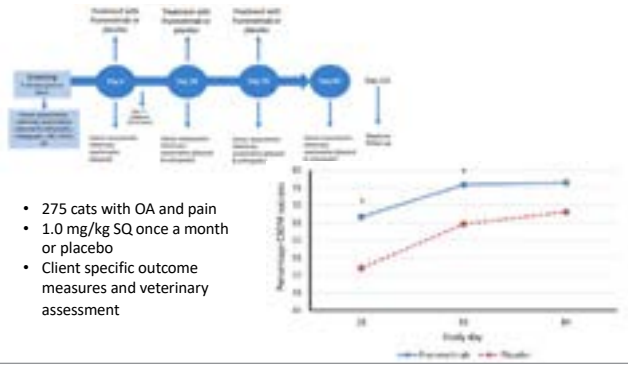
Nerve growth factor

- Mediates inflammatory and neuropathic pain
- Inhibition of NGF alleviates allodynia

- Monoclonal antibodies
- Felinized anti-NGF Ab NV-01 (Frunevetmab)

- For CHRONIC pain

94



- 275 cats with OA and pain
- 1.0 mg/kg SQ once a month or placebo
- Client specific outcome measures and veterinary assessment

95

Other modalities for pain management

- Physical rehabilitation
  - Therapeutic exercise
  - Physical modalities (heat, cold, laser, electrical stim, etc.)
  - Manual techniques (joint mobilization, trigger point, massage)
- Optimize body condition
- Dietary supplements
  - Omega-3 fa (EPA, DHA)
  - Glucosamine, chondroitin
  - Green-lipped mussels
- Caution
  - Variable quality, potency
  - Conflicting study results

96

Other modalities for pain management

- Nursing
- Acupuncture
- Medical massage
- Surgical salvage procedures

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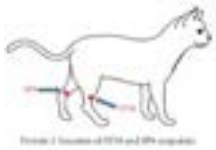
97

Laser Acupuncture for Postoperative Pain Management in Cats

Conclusion: Laser acupuncture reduced postoperative analgesic requirements in cats undergoing ovariohysterectomy



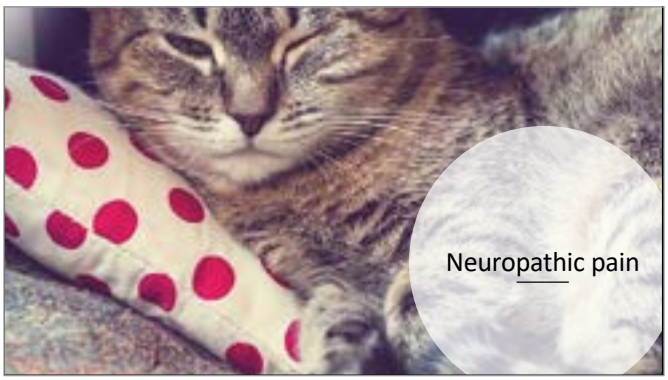
Marques Evidence-Based Complementary and Alternative Medicine 2015

98

Treating DJD

- Frunevetmab (Solensia) +/- NSAIDs
- Environmental modifications
- Weight management
- Adjunctive analgesics (tramadol, gabapentin, amantadine, amitriptyline)
- Nutritional supplements, diet change
- Polysulfated glycosaminoglycan s, glucosamine, chondroitin sulfate
- Rehabilitation (including laser)
- Acupuncture

99



100

Neuropathic pain

Examples

- May wax and wane or be ongoing

- Idiopathic cystitis
- Diabetic neuropathy
- Onychectomy
- Feline orofacial pain syndrome

101

Peripheral and central sensitization

Increased sensitivity around the lesion site

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Hyperalgesia

- An exaggerated and prolonged response to a normally painful stimulus
  - Also may occur when normal touch applied to normal tissue (secondary site)

103

Allodynia

- A pain response to a low-intensity, normally innocuous stimulus such as light touch to the skin or gentle pressure

104

Treating Neuropathic Pain

- **Gabapentin** to block acute sensitization peri-operatively or once already established.
- **NMDA receptor antagonists**
  - Ketamine, then amantadine long-term

PLUS

- **NSAID** (due to i… component of peripheral and central nervous system respon…)
- **Opioids** (never alone)
- +/- local anaesthetics, alpha-2 agonists, acupuncture, massage, amitriptyline
- Look for aggravating cause (e.g., tooth or bone fragment)

And environmental modification

105

Clinical signs suggestive of neuropathic pain

- Repeated chewing, biting, scratching at the same site
- Spontaneous crying
- Adverse reaction to touch without visible pathology
- Poor response to standard therapy (NSAID, opioid)
- Requires treatment of underlying cause as well as the neuropathic pain

106

Neuropathic pain: idiopathic cystitis

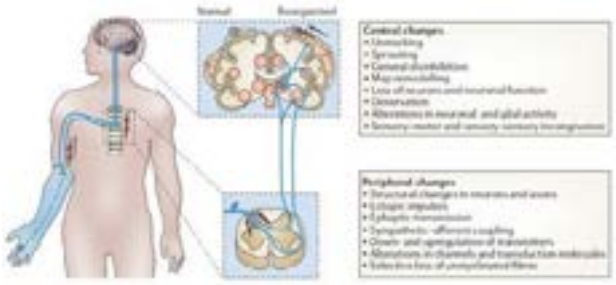
- Waxes and wanes

107

Neuropathic pain: diabetic neuropathy

108

Phantom limb pain



109

Neuropathic pain: onychectomy

- 21- day course of:
  - Buprenorphine +
  - Amantadine +
  - Meloxicam

Neuropathic Pain Syndrome
OF FELINE ONYCHECTOMY

cliniciansbrief.com

Gaynor, Clinician's Brief, 2005

Littlebigcat.com

110

Feline Orofacial Pain Syndrome: FOPS

111

Clare Rusbridge

www.veterinary-neurologist.co.uk/FOPS/

NSAIDS + gabapentin + opioids

112

Neuropathic Pain: First Line Treatment in Humans

Dworkin H et al. Arch Neurol. 2003

113

Treating Neuropathic Pain

- **Gabapentin** to block acute sensitization peri-operatively or once already established.
- **NMDA receptor antagonists**
  - Ketamine, then amantadine long-term

PLUS

- **NSAID** (due to i… component of peripheral and central nervous system respon…)
- **Opioids** (never alone)

And environmental modification

- +/- local anaesthetics, alpha-2 agonists, acupuncture, massage, amitriptyline
- Look for aggravating cause (e.g., tooth or bone fragment)

114

Assessing response to treatment of pain

- One dose is not enough!
- Follow-up is critical!
- Adopt a rigorous protocol for assessing pain
  - Undertake baseline assessment
  - Involve client in ongoing assessment
    - Keep asking!!!
  - Perform repeat assessments
- Changes will be subtle - longterm…months
  - Compare to 3-6-12 months ago

115

Predicting & preventing pain

If it might hurt you… *assume it will hurt the patient*

116

Anticipate pain (acute or chronic)

- Oral diseases
- Inflammation (-itis)
  - Bowel inflammation
  - Biliary tree disease
- Neoplasia (stretch/compression)
- Idiopathic LUTD
- Tissue trauma
  - Blood collection, catheter placement
- Restraint
  - Frail, thin, dehydrated
  - Arthritic

117

Mild-to-moderate

- Abscesses and their management
- Castration
- Chest drains
- Dental disease
- Cystitis
- Otitis
- Superficial lacerations

Moderate

Examples

- Cystitis
- Dental disease
- Arthroscopy and laparoscopy
- Osteoarthritis (it can be severe if neuropathic pain is involved; end-life stages)
- Ovariohysterectomy
- Some soft tissue injuries
- Urethral obstruction

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Moderate-to-severe (varies with degree of illness or injury)

- Capsular pain due to organomegaly
- Corneal abrasion/ulceration
- Corrective orthopaedic surgery (osteotomies; cruciate surgery; open arthrotomies)
- Dystocia
- Early or resolving stages of soft tissue injuries/inflammation/disease
- Extensive resection and reconstruction for mass removal
- Frostbite
- Glaucoma
- Hollow organ distension
- Intervertebral disc disease
- Mastectomy
- Mastitis
- Mesenteric, gastric, testicular or other torsions
- Mucositis (including radiation therapy associated mucositis)
- Oral cancer
- Panosteitis
- Peritonitis/Pleuritis
- Stomatitis
- Trauma (*i.e.* orthopaedic, extensive soft tissue, head)
- Ureteral urethral/biliary obstruction
- Uveitis

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Severe to excruciating pain

- Aortic saddle thrombosis
- Articular or pathological fractures
- Bone cancer
- Burn injury
- Central nervous system infarction/tumours
- Ear canal ablation
- Fracture repair where extensive soft tissue injury exists
- Hypertrophic osteodystrophy
- Inflammation (extensive, *e.g.* peritonitis, fasciitis)
- Limb amputation
- Meningitis
- Necrotizing pancreatitis or cholecystitis
- Neuropathic pain (nerve entrapment/inflammation, intervertebral disc herniation, etc.)
- Spinal surgery
- Thrombosis/ischemia

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120

WSAVA Pain management protocols

121

Multimodal for acute pain

- NSAID + opioid +
  - NMDA receptor antagonist
  - Topical and local anaesthetics
  - Short acting corticosteroids?
  - Sedation ≠ analgesia

122

Multimodal for chronic pain & neuropathic pain

- NSAID plus
- Opioids?
  - Better for break-through pain or palliative care
- NMDA receptor antagonist (ketamine or amantadine)
- Sedative
- Tricyclic antidepressant (amitriptyline): idiopathic cystitis
- Gabapentin (anticonvulsant), amantadine (NMDA): neuropathic pain
- Disease modifying agents: polysulfated glycosaminoglycan, glucosamine and chondroitin sulfate: DJD

123

Q: Have I made my point about multimodal analgesia?

a. Yes, I get it!
b. No
c. Yawn

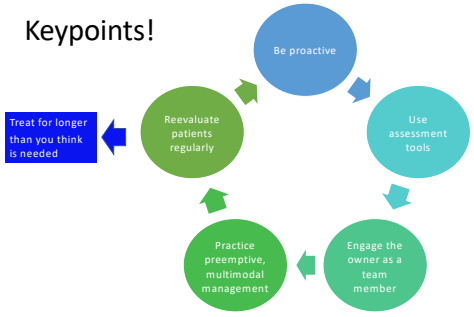
124

Multimodal ALWAYS

- ≠ multi-choice
- Tool box
- Menu: opioid + NSAID + local + NMDA receptor antagonist
  - Diet
  - Physiotherapy
  - Laser therapy?
  - Stem cell therapy?
- Assess efficacy
- Purrsonalized medicine

125

Keypoints!



Gruen 2022 AAHA Pain Management Guidelines

126

127

WSAVA

WSAVA.org

2022 WSAVA guidelines for the recognition, assessment and treatment of pain

FREE!

2022 AAHA Pain Management Guidelines for Dogs and Cats*

Catvets.com

128

WSAVA Pain management protocols

- Cesarean patients (EN / EE / PT)
- Cancer related pain (EN / EE / PT)
- Castration and ovariohysterectomy/ovariectomy in Cats (EN / EE / PT)
- Castration and ovariohysterectomy/ovariectomy in Dogs (EN / EE / PT)
- Degenerative joint disease (EN / EE / PT)
- Emergency and critical care (EN / EE / PT)
- Medical pain (EN / EE)
- Neonatal and pediatric patients (EN / EE / PT)
- Neuropathic pain (EN / EE / PT)
- Orthopedic surgery (EN / EE / PT)
- Pregnant or lactating patients (EN / EE / PT)
- Soft tissue surgery (EN / EE / PT)
- Perceived level of pain associated with various conditions (EE / PT)

WSAVA

129

130

2022 ISFM Consensus Guidelines on the Management of Acute Pain in Cats

Steagall JFMS 2022

2024 ISFM and AAFP consensus guidelines on the long-term use of NSAIDs in cats

Taylor JFMS 2024

Catvets.com

FREE!

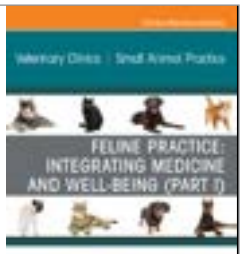
131

Analgesia
What Makes Cats Different/Challenging and What is Critical for Cats?

July 2020

Feline Chronic Pain and Osteoarthritis

Feline Neuropathic Pain



Neurobehavioral Disorders
The Corticolimbic System in Health and Disease

September 2020

132

Ask your Vet about ways to treat pain in your pet.

IVAPM

International Veterinary Academy of Pain Management:
www.ivapm.org

133

Goal: Freedom from pain & normalize activity

www.ivapm.org

hypurr@aol.com

134

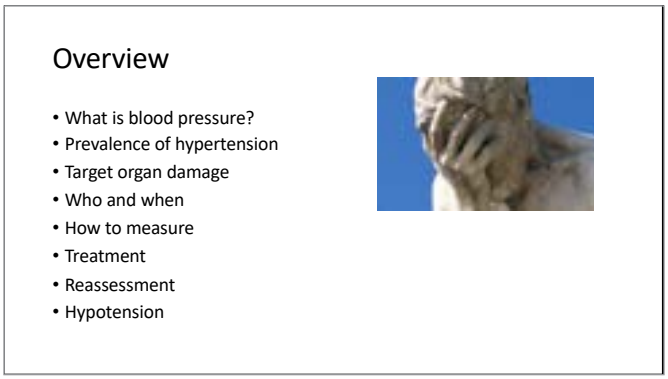
4002

BLOOD PRESSURE: A CRITICAL FACTOR

FELINE

Margie Scherk, DVM, DABVP

1



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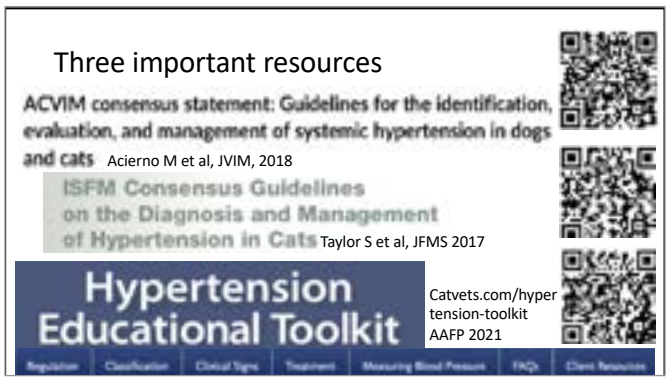

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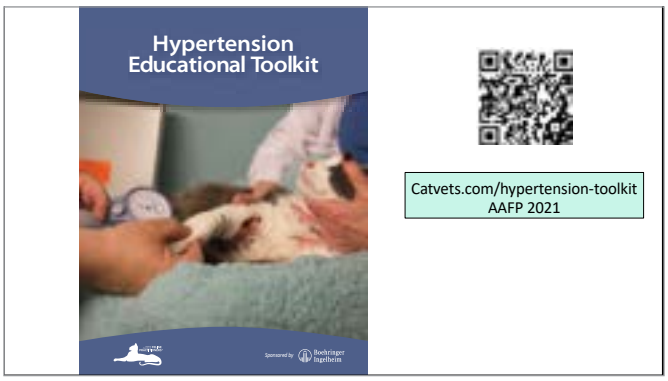

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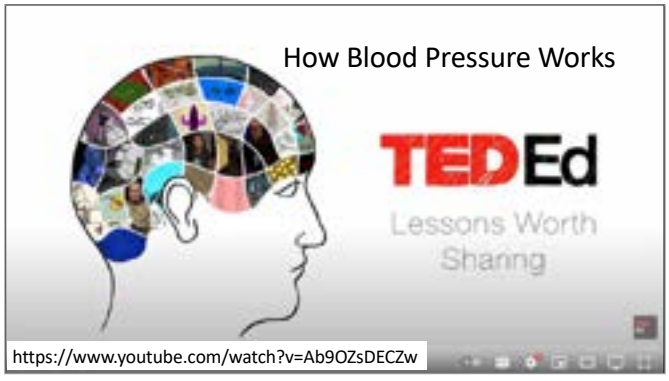


5

6

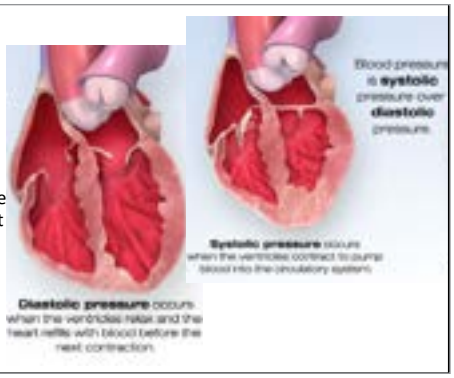
Key points

1. 

7

Key points

2. Systolic pressure = aortic valve is open and heart is ejecting blood (120 mmHg)
3. Diastolic = aortic valve is closed, heart at rest (80 mm Hg)



8

Key points

4. Mean = closer to diastolic, more time at rest (90 mm Hg)
5. Mean arterial pressure > 60 mm Hg needed for perfusion of brain, heart and kidneys
6. Only systolic measurements should be used for clinical assessment
   - Diastolic and mean are less accurate

9

Key points

7. Excitation, stress can *transiently* raise values
   - Autoregulatory mechanisms
   - Consistent values > 160 mm Hg reflect hypertension
   - "White coat syndrome"
   - Acclimate 10 minutes
   - Perform BP before other evaluations

10

What's normal?

Blood Pressure Measurements in 780 Apparently Healthy Cats

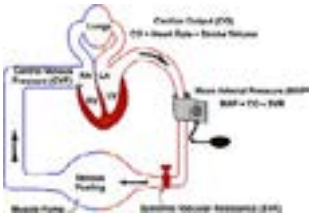
J.R. Payne, D.C. Brodbelt, and V. Luis Fuentes

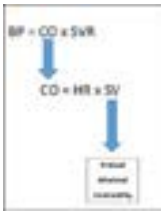
- 780 apparently healthy cats in shelter screening program
  - Excluded if hyperthyroid, CKD, hypertensive, systemic disease, pregnant, nursing
- Median BP 120.6 mmHg (110.4-132.4)
- "Factors significantly associated with higher systolic blood pressure in a general linear model were increased age, increased nervousness, male sex, neutering, or history of being a stray."

Payne, JVIM 2017

11

Blood pressure - mechanisms

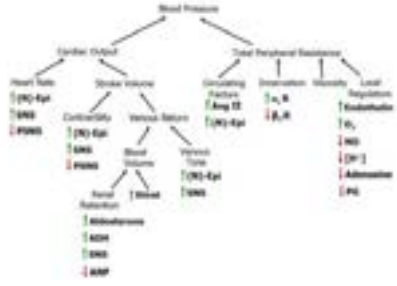




BP = cardiac output X systemic vascular resistance

12

What regulates BP?



- Heart
- Brain
- Blood vessels
- Kidneys

13

Who is at risk for hypertension?

- Older cats
- Have a disease associated with hypertension (CKD, hyperthyroidism, adrenal mass)
- Evidence of target organ damage
- Diagnosis requires measurement of blood pressure in conscious cats using validated methods.

14

Poll 1: Do you measure blood pressure in cats?

a. Yes
b. No

15

Prevalence of feline hypertension

16

Poll 2: Do you measure BP in conscious cats?

a. Yes
b. No

17

Poll 3: What type of equipment do you use?

a. Parks Doppler
b. CAT Doppler
c. Other Doppler
d. HDO oscillometric
e. PetMap oscillometric
f. Other oscillometric

18

Poll 4: What do you see as the biggest obstacle in measuring BP in conscious cats?

a. Cat stress
b. Noise
c. Time
d. Cost
e. Doctors not interested
f. Don't know how

19

Cross-sectional survey of non-invasive indirect blood pressure measurement practices in cats by veterinarians

- VIN survey: 733 respondents
- 96% measured BP with 85.3% being performed by nurses
  - Only 4.1% of respondents didn't measure

1. How did they measure BP?
2. How did they reduce situational hypertension?
3. What obstacles are there to measuring BP?

Navarro JFMS 2022

20

Cross-sectional survey of non-invasive indirect blood pressure measurement practices in cats by veterinarians

VIN survey: 96% of the 733 respondents

1. How did they measure BP?
   - 69% used Doppler flow, and
   - 91% of them believed more trustworthy than oscillometric

2. How did they (90%) reduce situational hypertension?
   - Quiet location, minimizing restraint, measure before other procedures, avoid other animals, allow time to acclimate

21

Cross-sectional survey of non-invasive indirect blood pressure measurement practices in cats by veterinarians

Of the 4.1% of the 733 respondents

3. What obstacles are there to measuring BP?
   - Difficulty interpreting results with the occurrence of fear, anxiety and stress in cats (20/30; 66.7%);
   - Difficulty performing measurements in cats (17/30; 56.7%); and
   - Technical staff being uncomfortable performing measurements (12/30; 40.0%).

Navarro JFMS 2022

22

Prevalence of feline hypertension

- BP increases with age in healthy cats
- Primary hypertension
  - 13-20% of hypertensive cats
- Secondary hypertension
  - 65% of cats w CKD
  - 10-23% of hyperthyroid cats
    - Check during treatment
  - 46% of cats with hyperaldosteronism
    - Pheochromocytoma
    - Diabetes?

23

Consequences of hypertension

Target organs = brain, eye, kidneys, heart

Drawing from Semintra HT promo material

24

Reliability of detecting fundus abnormalities associated with systemic hypertension in cats assessed by veterinarians with and without ophthalmology specialty training

news.vet.tufts.edu

- Determine the reliability of indirect fundoscopy in cats at-risk for hypertension performed by a new graduate veterinarian without ophthalmology specialty training
- Reliability of detecting fundus abnormalities by new grad was 72% for visual cats and 100% for avisual cats when hypertensive

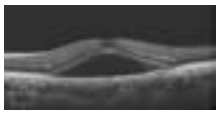
JUST DO IT.

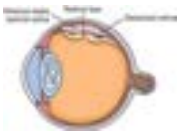
Moretto JFMS 2021

25

Hypertensive chorioretinopathy

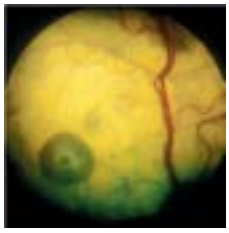
- Changes begin with BP persistently > 160 mmHg
- Optical coherence tomography
- Changes occur before detachment apparent
- Occurs in 50% of hypertensive cats

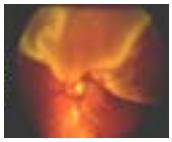




26

Hypertensive chorioretinopathy





27

Hypertensive chorioretinopathy

- Over 57% of blind cats improve with treatment
  - At least developing a menace response
  - Even in some cats blind for 2 months
  - May need prolonged period of ophthalmic treatment (+ BP)
    - 20% took > 60 days

28

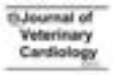
Encephalopathy and myelopathy

- 15-46% of hypertensive cats have neurological signs
  - Lethargy
  - Disorientation
  - Night-time yowling
  - Vestibular signs
  - Seizures

- Rapid increase in BP
- Severe hypertension
  - Cerebral edema, arteriosclerosis
  - MRI
  - Clinical signs resolve with treatment
- Hypertension may be implicated in some cases of ischemic myelopathy

29

Cardiovascular injury



- Cardiac murmurs and/or gallop sounds are common
- Left ventricular hypertrophy is most common change
  - But LVH common in healthy cats as well (15% of 780 cats)
  - Prevalence increases w age

Cardiomyopathy prevalence in 780 apparently healthy cats in rehoming centres (the CatScan study)

2015

- Rarely, heart failure or aortic dissection have been reported

30

Cardiovascular injury

- Complications may occur if hypertension superimposed on pre-existing heart disease or other risk factors
  - Anemia
  - Corticosteroids
  - Fluids

- Effective antihypertensive therapy may result in cardiac remodeling and regression of LVH

Elliott JSAP 2001, Acierno JVIM 2018

31

Kidney damage?

- Increased glomerulosclerosis and renal arteriosclerosis in hypertensive cats
- Unclear which came first

32

Kidney damage?

- Increased BP => increased proteinuria
- Proteinuria linked to decreased survival in cats with CKD or with hypertension

33

Target organ damage- summary

Table from Reusch: *Endocrine Hypertension in Small Animals* Vet Clin Small Anim 2010

34

35

ACVIM consensus statement: Guidelines for the identification, evaluation, and management of systemic hypertension in dogs and cats

Mark J. Acierno | Scott Brown | Amanda E. Coleman | Rosanne E. Jepson | Mark Papich | Rebecca L. Stepien | Harriet M. Syme

Journal of Veterinary Internal Medicine ACVIM

ISFM Consensus Guidelines on the Diagnosis and Management of Hypertension in Cats

Acierno JVIM 2018 Taylor JFMS 2017

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Management of CKD: systolic BP

- ACVIM Panel on Hypertension (Consensus statement 2018)
- Believe increases > 160 mm Hg

Category	SBP (mmHg)	Risk of future TOD
Normotensive	<140	Minimal
Pre-hypertensive	140 to 159	Mild
Hypertension	160 to 179	Moderate
Severe hypertension	>180	Severe

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- Not all pre-hypertensive cats will become hypertensive
- Require more frequent monitoring
- Changes may occur rapidly

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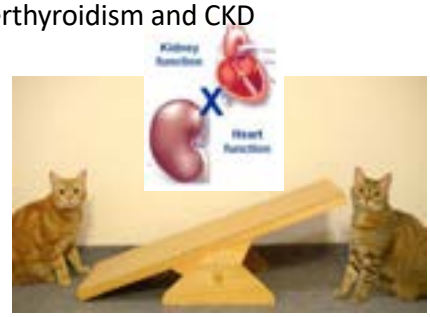
Hyperthyroidism and CKD



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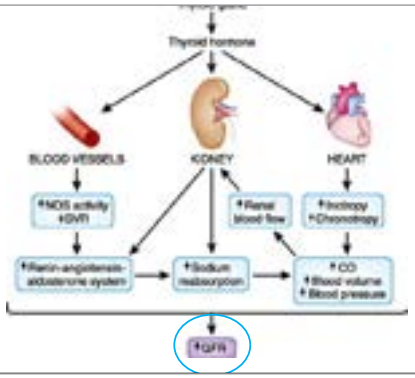

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Renal: thyroid relationship

1. By reducing cardiac output by achieving euthyroidism, GFR is reduced (as much as 50%)
   a) Revealing CKD
   b) Worsening previously recognized CKD
2. Should see these changes by 4 weeks of euthyroidism
3. Cannot predict
4. Initial treatment of choice is methimazole

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Balancing act

- If renal insufficiency becomes apparent:
  - L-thyroxine or
  - Reduce methimazole dose
  - Balance thyroid and renal function
- But pay attention to blood pressure as well!

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Who will benefit from measurement?

Who & when?

- Cats > 10 years
  - Predisposed to CKD, hyperthyroidism, hyperaldosteronism
  - BP increases 1-2 mmHg/year after age 9

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ISFM Consensus Guidelines on the Diagnosis and Management of Hypertension in Cats

Who & when?

Frequency for healthy cats:

- Adults (3–6 years of age): consider every 12 months
- Seniors (7–10 years of age): at least every 12 months
- Geriatrics (⩾11 years of age): at least every 6–12 months

45

ISFM Consensus Guidelines on the Diagnosis and Management of Hypertension in Cats

Who & when?

Frequency for other cats:

With recognized risk factors including: CKD, treated and untreated hyperthyroidism, primary hyperaldosteronism	Measure immediately and reassess every 3-6 months

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Regarding young cats

Who & why?

- Purpose: to obtain baseline values
- Caution regarding interpretation
- To acclimate to procedure using positive reinforcement
- Improves skill of operator

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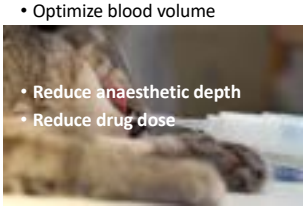
Also in hypotensive cats

Who else?

- Hypotensive cats
  - Ill, fragile, moribund
- At risk for hypotension
  - Under anaesthesia
  - Antihypertensive agents
  - Vasodilatory agents
- Optimize blood volume



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Equipment for measuring BP

How?

- Implanted radiotelemetric devices: 117-132 mmHg systolic in 4 studies on healthy, conscious cats
- Doppler: 118-162 mmHg in healthy, conscious cats in 6 studies
- Traditional oscillometric devices underestimate BP in conscious cats
- High definition oscillometry (HDO) is much more accurate (when compared to implanted devices)

49

Doppler

Oscillometric

50

Use standardized procedures!

- Quiet environment
- Let cat acclimate
- Less is more (personnel)
- Less is more (restraint)
- Use the right cuff

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Correlation Between Radial & Coccygeal Artery Indirect Doppler Blood Pressure Measurements in Cats

- Measuring arterial BP on tail is better tolerated than forelimb, but discordant results are common
- Goal:
  - Assess the impact of age, BCS, & MCS on radial and coccygeal BP measurements
- 66 pet cats
  - BCS & MCS evaluated by 2 trained observers
  - Order of site used was randomized
  - For FL in lateral recumbency; tail in sternal

Mawby, JVIM 2015

54

Radial & Coccygeal Artery Indirect Doppler Blood Pressure

- Results:
  - High inter-rater reliability for BCS & MCS
  - No effect of order of pressure collection site
  - Association b/n radial artery pressure & MCS due to confounding influence of age
  - Tail measurements not affected by age, BCS, MCS
- Conclusion:
  - Use tail to reduce the impact of age & sarcopenia on interpretation of results in cats

Mawby, JVIM 2015

55

Doppler Learning Curve

- Calm, patient acclimated
- Reduce fear (environment unthreatening, slow cuff pressure changes, comfortable positioning, minimal noise)
- Use stereo headset
- Operator relaxed
- Shaved vs. not

56

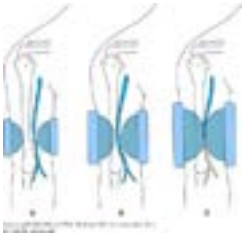
Learning curve: cuff size

- Should have sizes 2-5 in small animal practice
- Cats usually need a #1, #2, #2.5 or #3 cuff
- Reuse disposable cuffs

57

Learning curve: cuff size

- Use correct size
- Don't tape closed



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How to measure for cuff

- Cuff size is critical!
  - 40% of circumference of limb
  - Cuff too small – false ↑ BP
  - Cuff too big – false ↓ BP
  - Measure and use chart
  - Record in medical record

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Consequences of Common Errors and Artifacts

Error or Artifact	*Consequence*
Cuff too wide	Falsely low reading
Cuff too small or narrow	Falsely high reading
Cuff too loose	Falsely elevated reading
Cuff over a joint	Less likely to compress artery
Hole in cuff	Pressure leaks too fast to reliably record
Cardiac arrhythmias	Erratic readings
Heavy respiration	Can be mistaken for flow with Doppler
Motion	Interferes with obtaining a Doppler signal and oscillometer devices

vetfolio.com

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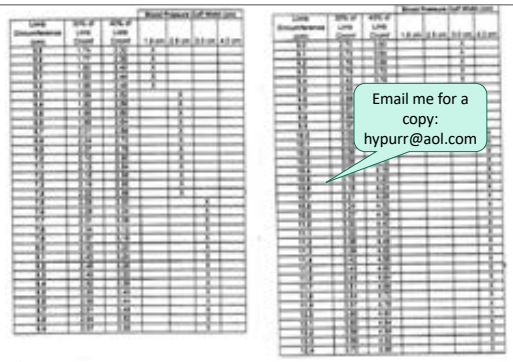


Email me for a copy: hypurr@aol.com

Table by Dr. Jane Brunt

61

- Median artery
- Metatarsal artery
- Median coxygeal artery

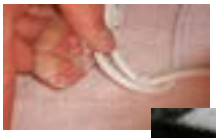
- Cuff should be level with heart

62

How to place cuff

- Avoid vertical distance between cuff and heart

63



Lessons learned

Photos: Susan Little

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Step-by-step technique

- Inflate cuff until all sound of blood-flow stops
- Slowly release pressure, watching the gauge
- Record pressure at which blood-flow first resumes.
  - Once BP is not dropping further, take 5-7 measurements with < 20% variability and use the average value

66

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HDO: high definition oscilliometry

68

Comparison of Doppler ultrasonic and oscillometric devices (with or without proprietary optimisations) for non-invasive blood pressure measurement in conscious cats

- Compared Doppler and oscillometric (PetMAP+) devices (with or without proprietary optimisations) in conscious cats
- Doppler and oscillometric devices cannot be used interchangeably
- Methodology should always be taken into account and reference intervals (RIs) need to be defined for the different methodologies
- Makes sub-staging for IRIS challenging

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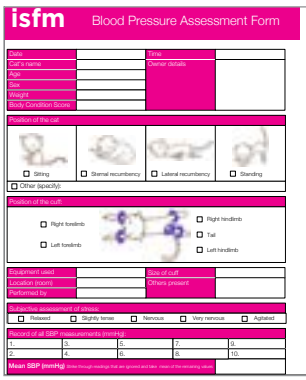

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Treatment of hypertension

- Goal = get BP < 160 initially, then to 140 mmHg
1. Amlodipine (Norvasc)
   - Calcium channel blocker that affects systemic vascular resistance
   - 0.625 mg q24h (0.2 mg/kg PO q24h)
     - If starting BP ≥ 200, 1.25 mg po q24h
     - Titrate to effect
- Minimal cardiac effects
- May be beneficial in proteinuria??

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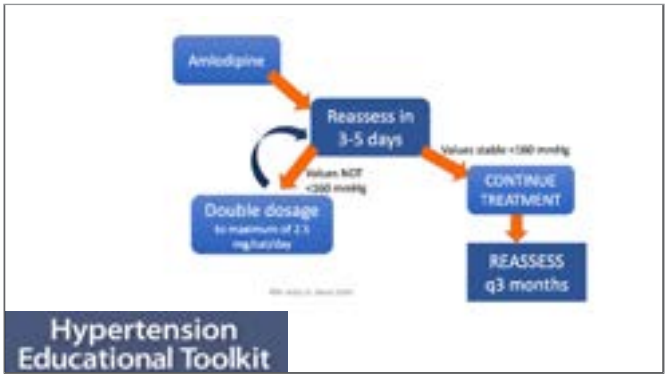

73

Treatment of hypertension

2. Telmisartan (Semintra HT)
   - Angiotensin receptor blocker
     - Affects the AT-1 receptor subtype, that mediates the adverse effects of Ang II on the cardiovascular and renal systems
   - 2 mg/kg PO q24h
     - (1.5 mg/kg PO BID X 14 days, then 2 mg/kg PO q24h)

Partially or uncontrollable hypertension with amlodipine

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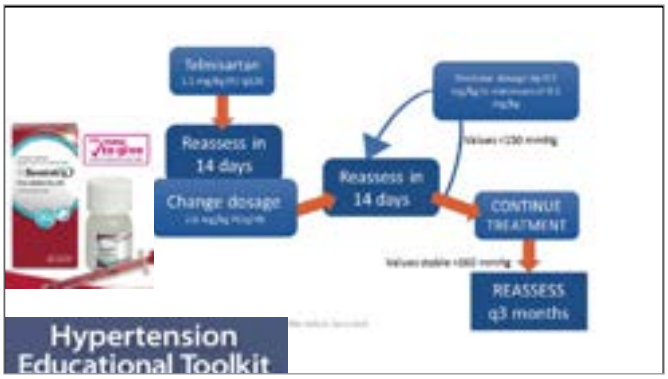


75

Evaluation of orally administered telmisartan for the reduction of indirect systolic arterial blood pressure in awake, clinically normal cats

- Angiotensin receptor blocker
- Comparing once a day to BID dosing of telmisartan or placebo on BP in awake, clinically normal cats
- Systolic BP values were significantly lower in cats treated with TEL at all tested dosages by the second week of treatment compared to placebo
- SBP remained significantly lower than in PLA-treated animals for 2 days following last dose
- Telmisartan daily dose of 1–3 mg/kg

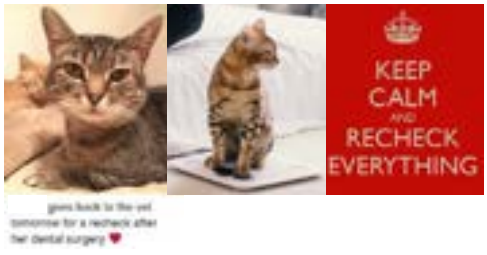
Coleman JFMS 2019

76

Treatment – ineffective on their own

- ACE inhibitors
- Beta-blockers

77



REASSESS!!!

78

Reassess BP

- Ensure dose is adequate
- Ensure not hypotensive (BP < 110 mmHg)
- 7-10 days after new dose
- If initial BP ≥ 200 mmHg or TOD is evident, repeat q4h within initial 24-72h period
- Once BP at desired level, recheck q 3 months

79

Does gabapentin affect BP?

Gabapentin Reduces Blood Pressure and Heart Rate through the Nucleus Tractus Solitarii

- Oral and intravenous gabapentin can markedly attenuate blood pressure (BP) in hypertensive **rats**

Effects of a single preappointment dose of gabapentin on signs of stress in cats during transportation and veterinary examination

- 20 healthy pet cats with a history of fractious behavior or signs of stress during veterinary examination
- No significant differences were identified between treatments in SAP and MAP values, and only a mild difference in heart rate was found in the present study

Chen Acta Cardiol Sin 2019 van Haaften JAVMA 2017

80

Does gabapentin affect BP?

Can I use a sedative prior to measuring BP?

Many sedatives will impact blood pressure, depending on their impact on the cardiovascular system.

Does the use of gabapentin prevent accurate measurements of blood pressure?

Using reasonable doses of gabapentin pre-appointment should not be avoided if it eases the cat's anxiety and improves the patient experience. Document the dose given and the time the gabapentin was administered. Any evidence of TOD must be taken into consideration when interpreting readings. If hypertension is suspected, reevaluation on a lower dose of gabapentin can be helpful in making a determination. The impact of gabapentin on BP is not clear at this time.

81

Hypotension

- Due to rapid, abrupt reductions in BP
- May result ischemic injury to :
  - Kidneys => AKI
  - Heart => heart failure
  - Brain => coma
- Hospitalize to titrate in *hyper*tensive emergencies to prevent over treatment
  - Goal = normalize (160 mmHg) by 24h
  - May need other agents

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Other causes of hypotension

- *False* because cuff is too large
- Anaesthesia
- Pain
- Hypoperfusion
  - Shock
  - Cardiac arrhythmias
  - Heart failure
  - Blood loss
  - Sepsis
  - Disseminated Intravascular Coagulation (DIC)

83

Treatment of hypotention

- Treat the underlying cause (i.e., reduce the amount of inhalant anaesthetic flow and support hypovolemia with adequate fluids)
  - Oxygen, colloids
  - Use dopamine or dobutamine as needed
  - Administer analgesic agents

84

'The Mercury Challenge': feline systolic blood pressure in primary care practice – a European survey

- International European-based multicentre study
- Data from 8884 cats 7-26 years old having systolic BP measured in 811 clinics, 16 countries

- Doppler ~ 50%, oscillometry ~ 50% of clinics
- Demeanor: Calm 45.7%; anxious 41.9%; nervous 8.9%
- Duration of assessment: < 5 min 50.4%; 5-10 min 41.7%; > 10 min 7.99%

Sparkes JFMS 2022

85

'The Mercury Challenge': feline systolic blood pressure in primary care practice – a European survey

- Concurrent illness: CKD: 21.8%; hyperthyroidism 12%; both: 3.1%
- Median SBP 150 mmHg (80-310)
- Classification:
  - Hypertensive 18.6% (160-179 mmHg)
  - Severely hypertensive 21.1% (≥180 mm Hg)

- *Conclusions and relevance* SBP can be measured in most cats within a short period of time using either Doppler or oscillometric equipment.
- The presence of CKD or hyperthyroidism was associated with significantly higher SBP values, and
- Anxious or nervous cats had higher SBP values and took longer to obtain SBP assessments.

Sparkes JFMS 2022

86

No more:

87

Measure in every cat!

88

Three important resources

FREE

ACVIM consensus statement: Guidelines for the identification, evaluation, and management of systemic hypertension in dogs and cats

Acierno M et al, JVIM, 2018
onlinelibrary.wiley.com/doi/10.1111/jvim.15331

ISFM Consensus Guidelines on the Diagnosis and Management of Hypertension in Cats

Taylor S et al, JFMS 2017
Catvets.com/guidelines

Hypertension Educational Toolkit

Catvets.com/hypertension-toolkit
AAFP 2021

89

Thank you for participating.

Do you have any questions?

hypurr@aol.com

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4003

RECENT THOUGHTS ON IBD & SMALL CELL LYMPHOMA

FELINE

Margie Scherk, DVM, DABVP

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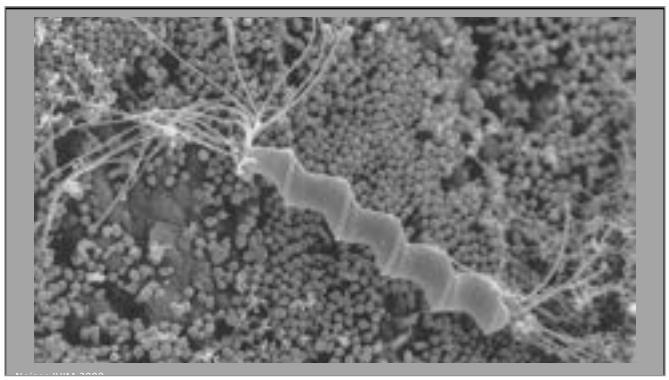

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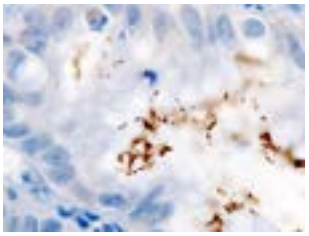
Helicobacter & gastrointestinal disease

- *H. pylori* is serious in humans
  - Gastritis, peptic ulcers and gastric cancer
  - Concern re zoonotic risk from cats, dogs
- Stray cats – Helicobacter spp. in oral secretions but unrelated to
  - +/- colonization of gastric mucosa
  - Species of *Helicobacter*

4

Helicobacter & gastrointestinal disease in cats

- Many cats (and dogs) are infected and have gastric pathology, yet unclear what role bacteria play
- Most infected patients are healthy
  - Some with chronic vomiting , inappetence, diarrhea, and weight loss respond to therapy



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Hypotheses re *Helicobacter heilmannii* in cats

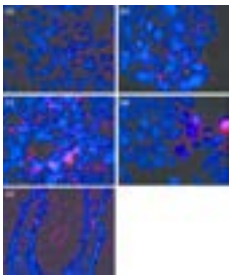
Associated with
- 2008: lymphoblastic lymphoma
- 2016: large intestinal mucinous adenocarcinoma

Suggestive evidence (2017)
- Cats with large cell lymphoma more likely to have a variety of invasive bacteria, (intravascular and serosal) than small cell lymphoma or lymphocytic-plasmacytic enteritis
  - Higher rate of sepsis in large cell lymphoma

6

Diagnosing *Helicobacter heilmannii*

- Breath urease testing (in humans +/- cats)
- Histopathology
- Cytology
- Fluorescent in situ hybridization (FISH)
  - Superior to silver staining



Lertpiriyapong J Med Microbiol 2014

7

Treating *Helicobacter heilmannii* in cats

Only treat if:
- Clinical signs
- Histopathologic confirmation
- Other disease ruled out

Generally will not result in long-term eradication of *Helicobacter* spp

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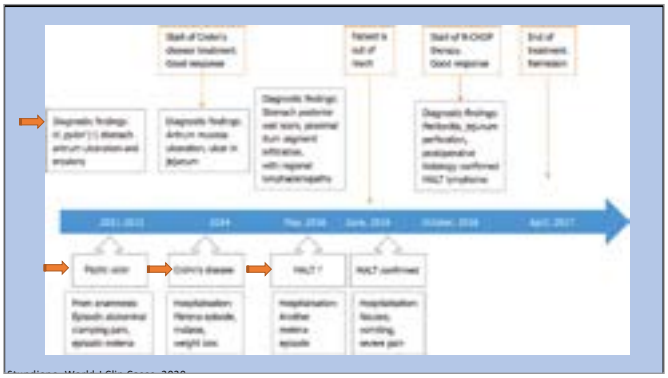

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IBD vs. chronic enteropathy

10

Is it IBD or Chronic Enteropathy?

- IBD is a histopathologic diagnosis (biopsy required for confirmation)
- CE is a broader term for a syndrome of diarrhea, vomiting, and weight loss of > 3 weeks duration
- More accurate to refer to "CE" pending a diagnosis.

www.catvets.com/public/Powerpoints/Update-on-IBD-CE-Management-NOTES.pdf

Datz: Update on management of CE (IBD) in cats Nov 2020

11

Summary from Dr. Datz's presentation: 1.

- Cats with intermittent or continuous GI signs > 3 weeks have chronic enteropathy (CE)
- Some cats with CE have IBD, others have lymphoma or less common causes
- CE/IBD can be food-responsive, antibiotic- responsive, steroid-responsive, or non- responsive (idiopathic)

www.catvets.com/public/Powerpoints/Update-on-IBD-CE-Management-NOTES.pdf

Datz: Update on management of CE (IBD) in cats Nov 2020

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Chronic enteropathies history & differentials

- Chronic, nonresponsive
- Weight loss may be dramatic
- Stool character varies widely
- +/- voracious appetite
- +/- not use litter box
- Otherwise unremarkable history

- Hyperthyroidism
- Lymphoma
- Exocrine pancreatic insufficiency
- Cholangitis
- Bacterial overgrowth?
- Endoparasitism (giardiasis)
- Adenocarcinoma
- Histoplasmosis
- FIP/FeLV/FIV

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Chronic enteropathy in cats

ACVIM consensus statement guidelines on diagnosing and distinguishing low-grade neoplastic from inflammatory lymphocytic chronic enteropathies in cats

- GI signs lasting > 3 weeks
  - After ruling out infectious, metabolic, and extraintestinal causes
  1. Food responsive enteropathy,
  2. Inflammatory BD/steroid responsive enteropathy,
     - Most commonly lymphoplasmacytic enteritis (LPE)
  3. Small cell/low grade intestinal T-cell lymphoma (LGITL)
- Common in older cats
  - Prevalence of diagnosing alimentary lymphoma has increased

Marsilio JVIM 2023, Louwerens JVIM 2005

14

Challenges

- Differentiating LPE from LGITL
  - Overlap of clinical signs, laboratory data, imaging, and histopathologic findings
  - Small cell/low grade lymphomas characterized by monoclonal or oligoclonal rearrangements of the lymphocyte receptors, clonality is not equivalent with malignancy
  - Not all reactive lesions are polyclonal
  - Yet histopathology remains key

15

When is it IBD vs. small cell lymphoma?

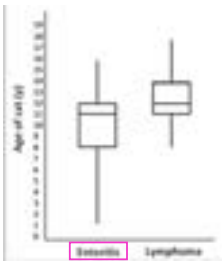
Diagnosis of chronic small bowel disease in cats: 100 cases (2008–2012)

Norsworthy, JAVMA 2013

16

50: 50?

- 100 cats with small bowel disease signs & ultrasonographically thickened small bowel
- 49 had enteritis (l/p >> eos)
- 46 had lymphoma



Norsworthy, JAVMA 2013

17

Lymphoma in cats

- Most common feline tumour
  - 42-52% of lymphomas are alimentary
- Risk factor: second-hand smoke exposure?
  - Relative risk of lymphoma for cats with any household ETS exposure was 2.4
  - 5 years exposed to smoke → increased risk by 3.2 X
  - Type of lymphomas not reported

Bertone Am J Epidemiol 2002, Marsilio JVIM 2023

18

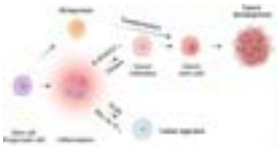
Pathogenesis

- Infectious:
  - ?? Regressive FeLV in small cell lymphoma
  - No studies of regressive FeLV and LPE
- Mucosa-invading bacteria more common in cats with large cell lymphoma (than LGITL)
  - Intravascular bacteria only in large cell lymphoma
- Dysbiosis? Promotes inflammation and malignant transformation in people and non-feline animal models
  - No difference in bacterial populations between cats with LPE and LGITL

Marsilio JVIM 2023, Hoehne Vet Path 2017, Marsilio Sci Rep 2019

19

Pathogenesis



- Chronic inflammation
  - Promotes oncogenesis
  - Frequent coexistence of LPE and LGITL
  - Continuum?
  - Duration of clinical signs in cats with LGITL is longer than LPE
- Environmental tobacco smoke exposure?
- Disruptions in crosstalk between immune system, intestinal environment, microbiome in a genetically predisposed individual

Marsilio JVIM 2023

Image from Wang Adv Biomarker Sci Tech 2024

20

In whom and how? Signalment and clinical presentation

- Age?
  - LGITL older but overlap
- Breed?
  - DSH, Siamese
- Sex?
  - NM

21

Clinical presentation

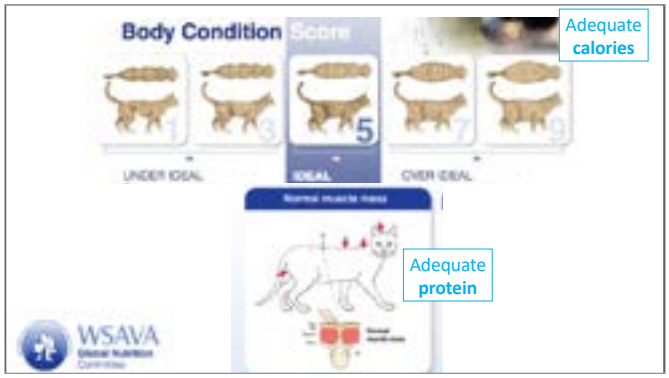
- Weight loss (80%–90% of cats with FCE)
- Vomiting (70%–80%)
- Anorexia (60%–70%)
- Diarrhea (50%–65%)

- Sarcopenia and low body condition score
- Muscle loss predominates

Marsilio VCNA 2021, Marsilio JVIM 2023

Image from Resident Cats on Facebook

22



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High BCS with low MCS

Sarcopenic obesity

Image courtesy of Mark Peterson

24

Muscle Condition Score

25

Clinical presentation: +/-

- +/- Segmental or diffusely thickened intestinal loops
- +/- Abdominal discomfort/pain (often cranial abdomen, possibly ⇔concurrent pancreatitis and/or cholangitis)

- More acute onset and rapidly progressive clinical signs with intermediate to large cell lymphoma
- Abdominal masses, intussusception, obstruction, perforation

Marsilio VCNA 2021, Marsilio JVIM 2023

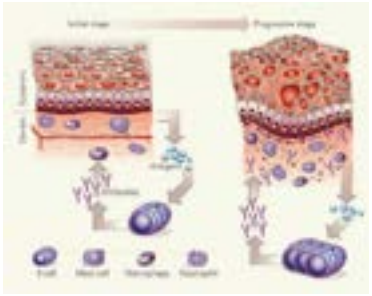
Image from wagwalking.com

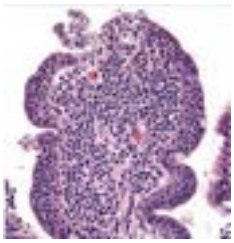
26

Progression of disease: from IBD to lymphoma

- Chronic insult
- Unknown whether severe lymphoplasmacytic IBD is pre-cancerous
- Mechanism unknown

27





28

Where is chronic enteropathy located?

- Anywhere
- LGITL: jejunum > ileum > duodenum (> stomach > colon)
  - If stomach, more likely large cell lymphoma ☹
  - If colon, more likely LPE ☺

29

Location matters

- Lymphoma: muscularis +/- serosa, more severe infiltration, severe architectural changes
- IBD: 50% mild, 30% moderate, 20% marked plasma cell infiltration of mucosa with architectural changes

- Most common sites for IBD or small cell lymphoma (T): ileum & jejunum (70-90%)
- Stomach: IBD (29%) > small cell lymphoma (7%)
- 41% cases small cell lymphoma had ***concurrent IBD***
- Large (B) cell lymphoma stomach

Briscoe 2011

30

Does lab work help?

Statement

Laboratory tests cannot differentiate between LPE and LGITL and currently there are no specific cancer markers for LGITL in cats. Low serum cobalamin concentrations are more frequent in cats with LGITL.

Evidence level

II

Panel recommendation

6 out of 6 members strongly agreed

- CBC, serum biochemistry panel, urine and fecal analyses, total T4
- Retroviruses
- Needed to rule out metabolic, endocrine, and infectious diseases

Marsilio JVIM 2023

31

Does lab work help differentiate? No

- +/- Protein changes:
  - Mild hypoalbuminemia?
  - Hyperglobulinemia?
  - +/- ⇧ total protein due to concurrent hyperglobulinemia (part of the Feline Chronic Enteropathy Activity Index)
  - Panhypoproteinemia?
- +/- ALT changes
- +/- fPLI changes
  - fPL⇧ in healthy older cats
- Exocrine pancreatic insufficiency is a significant differential – check fTLI
- ⇩ Folate (proximal small intestine) +/- ⇩ cobalamin (ileum)
  - Normal cobalamin doesn't rule out CE

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32

What about imaging?

Statement

Abdominal ultrasonography is an important diagnostic tool in the diagnostic evaluation of cats with CE. It allows for cross-sectional evaluation, anatomical localization, characterization of bowel wall moral architecture, and mesenteric lymph nodes as well as evaluation of other abdominal organs. The sonographic abnormalities of CE have been well described, however, substantial crossover between the LGITL and LPE exists and clinically relevant pathology can be present in the bowel with a normal ultrasound appearance. Thus, currently no imaging technology reliably differentiates LPE from LGITL, and intestinal histopathology is required for establishing the diagnosis of CE.

Evidence level

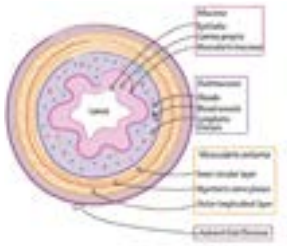
II/III

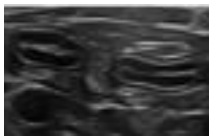
Panel recommendation

5 out of 6 members strongly agreed, 1 member agreed

- Radiographs: no benefit over ultrasound
- Abdominal ultrasound to assess other organs and identify affected abnormal segments but not to differentiate between LPE and LGITL

33





Healthy jejunal wall

Arrow = thickened muscularis in cat with CE

drawing from ditki.com

Images from Marsilio JVIM 2021

34

Ultrasound

- Diffuse thickening: muscularis propria, submucosa, or mucosa layer in the small bowel are common
- Ultrasonographic abnormalities in the mucosa were highly predictive of mucosal histologic lesions, the presence of thickened submucosa or muscularis layer did not correlate with histopathologic lesions
- But ultrasonographic and histopathologic changes occur even in clinically healthy cats
- Muscularis-to-submucosa ratio >1 was indicative of an abnormal bowel segment, but no difference was found between LPE and LGITL

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Ultrasound summary

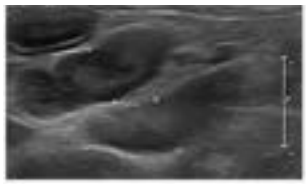

FIGURE 2 Ultrasonographic aspect of the jejunal lymph nodes in a LGITL case. The lymph nodes appears rounded and hypoechoic.

Abdominal lymphadenopathy

- One study showed differences in jejunal nodes: LGITL had rounder, thicker and more hypoechoic than LPE
- Same study showed greater chance of mild abdominal effusion with LGITL than LPE
- But overall: lymph nodes larger than in healthy cats but don't differentiate

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Freiche JVIM 2021

36

Is cytology useful?

Statement

Although cytology is helpful to exclude important differential diagnoses in cats with CE, cytology cannot be used to differentiate LPE from LGITL.

Evidence level

III

Panel recommendation

5 out of 6 members strongly agreed, 1 member agreed

- Fine needle aspirates help to rule out high-grade lymphomas, other round-cell neoplasia, fungal disease
- Can't diagnose CE or differentiate between LPE and LGITL
  - Lack of architecture
  - Inflammation present regardless

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37

Good grief. How about biopsies?

Statement

The collection of intestinal tissue biopsy specimens is the current gold standard for the diagnosis of and differentiation between LPE vs LGITL in cats. No clearly demonstrated superiority in quality exists for biopsy specimens obtained by laparotomy (full thickness) vs endoscopic biopsy specimens, because poor technique can affect sample quality and hamper diagnostic evaluation for both methods.

It has been shown that all inflammatory and neoplastic lesions are present in the lamina propria and hence, if mucosal samples of sufficient quality are procured endoscopically, a diagnosis is possible without obtaining full-thickness biopsy specimens. However, because of limited access to the jejunum by endoscopy, jejunal lesions cannot be reliably sampled although this small intestinal segment is frequently abnormal.

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38

Endoscopy

Maximizing the diagnostic utility of endoscopic biopsy in dogs and cats with gastrointestinal disease

- Flexible endoscopy is a valuable tool but the techniques must be performed carefully so that the results are meaningful.
- Recent studies have defined endoscopic biopsy guidelines for the optimal number and quality of diagnostic specimens from different regions of the gut.
- They also have shown the value of ileal biopsy in the diagnosis of canine and feline chronic enteropathies, and have demonstrated the utility of endoscopic biopsy specimens beyond routine H & E histopathological analysis, including their use in immunohistochemical, microbiological, and molecular studies.

39

Optimizing endoscopy

- Preliminary upper GI evaluation
- Intra-operative evaluation of jejunum and ileum
  - Mucosal surface
  - Place "stay sutures"
  - Full thickness biopsies

40

Laparoscopy

Comparison of laparoscopic-assisted technique and open laparotomy for gastrointestinal biopsy in cats

- Laparoscopic-assisted gastrointestinal biopsy technique provided diagnostic specimens and decreased postoperative pain compared to open surgical techniques.
- No difference was detected in surgical duration, complications, or duration of hospitalization.

Kovak McClaran Vet Surg. 2017

41

GI Diagnostics

- *Can clinical signs, clinicopathological findings and abdominal ultrasonography predict the site of histopathological abnormalities of the alimentary tract in cats?*
- Clinical signs, and clinicopathological and ultrasonographic abnormalities lack precision for hepatic and pancreatic histopathological lesions in cats with alimentary tract signs and cannot reliably predict from which organs biopsies should be collected.
- Exploratory coeliotomy is necessary to determine the site of histopathological abnormalities in feline alimentary tract disorders

Freiche JFMS 2016

42

How do you differentiate between LPE and low grade intestinal T-cell lymphoma?

1. Severity of clinical presentation
2. Serum biochemistries
3. Response to dietary trial
4. Ultrasound findings
5. Endoscopic biopsies
6. Full-thickness biopsies

43

Full-thickness biopsies preferable? Controversy

- Both IBD and lymphoma mostly in jejunum and ileum supporting full-thickness, rather than endoscopic biopsies
- Lymphoma and IBD may occur concurrently in some cats
- Good quality endoscopic biopsies less risk to patient
  - Samples must have sufficient, undamaged mucosal surface

Evidence level

II

Panel recommendation

4 out of 6 members strongly agreed, 2 members agreed

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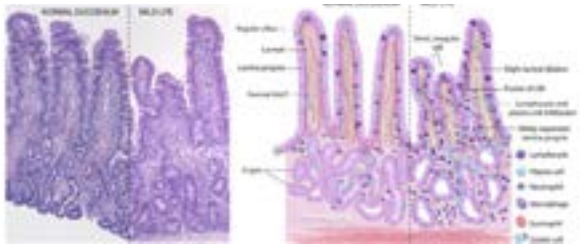
Architectural changes



45

Answer

- How do you differentiate between IBD and intestinal lymphoma?
  1. Severity of clinical presentation
  2. Serum biochemistries
  3. Response to dietary trial
  4. Ultrasound findings
  5. *Endoscopic biopsies*
  6. *Full-thickness biopsies*

46

Histopathology & immunohistochemistry?

- Gold standard for diagnosis of CE
- Alignment of samples for processing is critical
- Attach to cucumber slices/synthetic sponges

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47

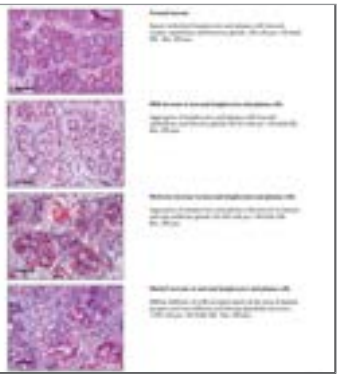
Pathologists may disagree

- Controversy as to what is "normal"

Day J Comp Path 2008

48

- But interobserver variability remains an issue despite attempts to simplify the grading scheme
- And did not encompass the differentiation of LGITL from LPE in cats, and the standardization was designed and validated only for IBD

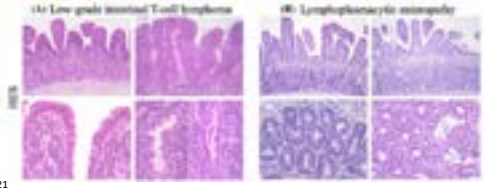


Day J Comp Path 2008

49

Histopathologic, phenotypic, and molecular criteria to discriminate low-grade intestinal T-cell lymphoma in cats from lymphoplasmacytic enteritis

- Differentiating LPE from LGITL can be improved if epithelium and lamina propria are assessed separately and in a structured fashion
- *"New histologic, immunohistochemical, and clonality criteria to distinguish LGITL from LPE"*



Freiche JVIM 2021

50

But still….many exceptions. So, more…

- Special stains
- Immunohistochemistry
- Immunophenotyping
- Clonality tests

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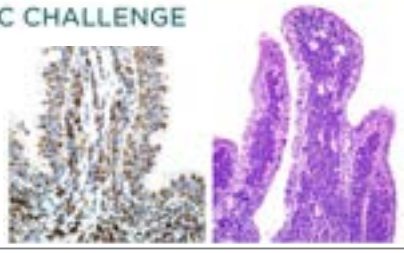
51

DIFFERENTIATING FELINE INTESTINAL LYMPHOMA FROM INFLAMMATORY BOWEL DISEASE (IBD)

THE DIAGNOSTIC CHALLENGE

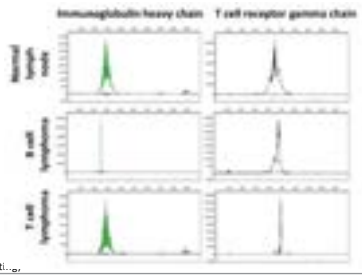


cvm.msu.edu

52

PCR for Antigen Receptor Rearrangement (PARR)

- Single ⇔ neoplasia; multiple ⇔ reactive, inflammation
- Blood, stained non-fixed and non-cover-slipped slides okay, formalin fixed samples okay
- 91% of confirmed feline lymphoma detected w PARR
- A negative result does not rule out lymphoma

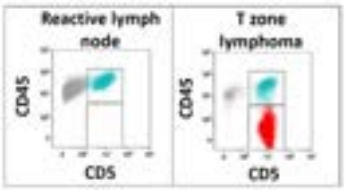


vetmedbiosci.colostate.edu/chl/principles-of-testi...

53

Immunophenotype by flow cytometry

- Stains live cells w fluorescently labeled Ab that bind to proteins on cell surface
- Different proteins expressed by different cell lines
- Counts #s and types of cells
- Homogeneous cell lineage (more consistent with neoplasia) vs. heterogeneous (more consistent with a reactive process)



vetmedbiosci.colostate.edu/chl/principles-of-testing/

54

Which test should I choose?

GUIDE TO TESTING FOR HEMATOPOIETIC MALIGNANCY

csu-cvmbs.colostate.edu/academics/mip/ci-lab/Pages/Lymphoid-Neoplasia-Testing.aspx

55

Still not there

- These tests complement each other and aren't definitive on their own
  - Histopathology
  - Immunohistochemistry
  - PARR
  - Flow cytometry

56

Don't overinterpret

JSAP BSAVA

Differentiating feline inflammatory bowel disease from alimentary lymphoma in duodenal endoscopic biopsies

- Implied that clonality was associated with shorter survival times
- A subset of cats with clonal rearrangements showed long- term survival of >500 days
- But…cats with LGITL tend to be older than cats with LPE so shorter survival times are expected
- Shorter survival times could be ⇔ longer standing or more severe intestinal inflammation => benign clonal expansion rather than malignancy
- In human medicine, only 5% to 15% of cases are considered to benefit from additional molecular clonality diagnostic testing so using molecular clonality as the single determining factor in the decision on malignancy vs. begin lesions is unjustified

57

Does it matter?

58

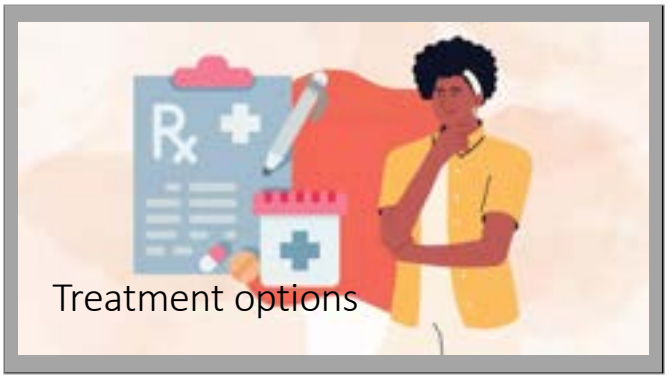
Differentiating Inflammatory Bowel Disease from Alimentary Lymphoma in Cats: Does It Matter?

- Will a comprehensive diagnostic evaluation of cats with chronic enteropathy change prognosis or treatment?
- Prognosis and treatment strategy may change based on the underlying diagnosis w further research
- May not be appropriate for every individual, but should be available
- Without scientific curiosity and research advancing the field, the profession won't advance

Marsilio VCNA 2021

59

Treatment options



60

Summary from Dr. Datz's presentation: 2.

- After diagnostic workup and ruling out causes such as parasites, start a dietary trial
- Up to 3 weeks of exclusive diet (no treats, human foods, etc.)
  - Gastrointestinal
  - Novel protein, limited ingredient
  - Hydrolyzed protein
  - Home-cooked recipe (ACVN diplomate, BalanceIT.com)

www.catvets.com/public/Powerpoints/Update-on-IBD-CE-Management-NOTES.pdf

Datz: Update on management of CE (IBD) in cats Nov 2020

61

Summary from Dr. Datz's presentation: 3.

- Don't give up if first trial doesn't work. May need 2, 3, or 4 different diets sequentially.
  - If all dietary trials fail, offer biopsies
- If biopsies not available, start a steroid or antibiotic trial
  - Steroid trial first
  - Wait on antibiotic trial until other treatments have failed

www.catvets.com/public/Powerpoints/Update-on-IBD-CE-Management-NOTES.pdf

Datz: Update on management of CE (IBD) in cats Nov 2020

62

Treating LPE: Diet plus…

- Prednisolone, at 2 mg/kg/d either SID or divided BID
- Taper by 25% q 3 weeks to 4 weeks to lowest effective dose
- Budesonide (3 mg/m2 every 24 hours) is an alternative for diabetic cats
- If malabsorption present, start with dexamethasone SQ
- For refractory LPE add chlorambucil

63

Chlorambucil

- Metronomic: 2 mg PO/cat q2 or 3 days
  - "low doses of chemotherapy drugs continuously over a long period of time, modulating the tumour microenvironment due to the cytotoxic, antiangiogenic and immunomodulatory effects"
- Pulse: 20 mg/m^2 PO every 2 weeks or 15 mg/m^2 for 4 days q 3 weeks

64

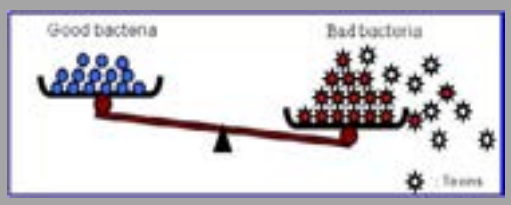
Treatment low grade intestinal T-cell lymphoma

Three groups:

- Oral prednisolone and high-dose pulse chlorambucil
- Modified Madison-Wisconsin multi-agent protocol
- Combination of both protocols
- Good to excellent response in 76% (13/17)
- 1999 Fondacaro: Disease free interval = 20.5 months (5.8 - 49 months) - Chlorambucil 2 mg PO/cat q2 or 3 days
- Stein 2010: overall clinical response rate 96%, with median clinical remission of 786 days
  - Rescue: 100% w cyclophosphamide + glucocorticoid

Lingard J Feline Med Surg 2009

65



Is there a role for probiotics?

66

Dysbiosis - imbalance

Caused by:

- Bacterial or viral infection, parasites
- Diet or diet change
- Drug therapy
- Altered gut secretions

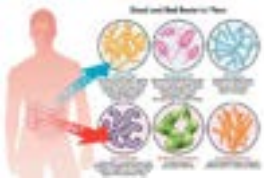
Can result in:

- Maldigestion and malabsorption
- Dietary intolerance or hypersensitivity
- Inflammation

67

Probiotics in human diarrhea

- Help in the prevention of diarrhea, as well as reduce the duration and frequency of diarrhea in humans
- But variable results
  - Specific diarrhea
  - Strain of organisms
  - # of organisms



68

Probiotics, prebiotics and synbiotics

- Probiotics are live organisms of the physiologic bacterial ecosystem that provide a health benefit to the host when provided in adequate quantities
  - Mechanism: directly inhibit colonization of pathogens &/or enhance immune effects on gut-associated lymphoid tissue
- Prebiotics are supplements or foods that stimulate the growth or activity of endogenous intestinal microorganisms
- Products that contains a prebiotic plus a probiotic is a synbiotic

69

What's important in a probiotic?

Stable

- Until consumed
- Manufacturing, shipping, storage
- Bacteria numbers must be ensured until end of shelf life
- Survive in GIT

Safe: *does not*

- Acquire antibiotic resistance
- Transmit antibiotic resistance
- Produce pathogenic factors
- Promotes a normal, balanced microbiome

70

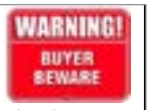
Assessment of commercial probiotic bacterial contents and label accuracy

Probiotics are widely available for use in animals but quality control of veterinary probiotics has been shown to be poor. The objective of this study was to evaluate the labels and bacterial contents of commercial probiotics marketed for use in animals. Twenty-five probiotics were purchased, labels were scrutinized, and bacterial contents were enumerated. Twenty-one (84%) products listed specific microorganisms. Expected bacterial numbers were listed for 15 (60%) products. One or more organisms were misspelled on the labels of 3/12 (32%) of products that listed specific organisms. Viable growth ranged from 6 to 2×10^{9} colony forming units (CFU)/g. Only 4/15 (27%) products that had specific claims of viable organisms met or exceeded their label claim. Only 2 of these also had an acceptable label, which properly described the contents. Deficiencies in veterinary probiotic quality remain. Veterinarians and owners should scrutinize commercial probiotics and demand evidence of quality control and efficacy.

Weese Can Vet J 2011

71

What to look for



- *"Currently, selection of bacterial strains for most commercial probiotic products is mostly based on their ability to survive the passage through the stomach and small intestine, their ability to adhere to mucus, and in vitro immunomodulation."*
- Consistently meet label claim
- Published clinical data for the species

Suchodolski Clinical Small Animal Internal Medicine 2020

72

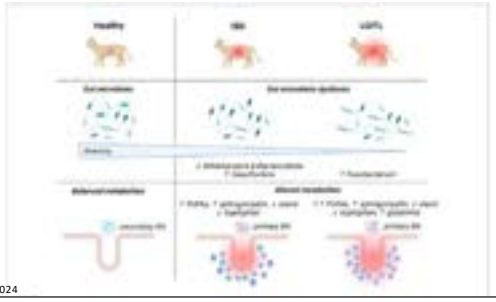
Resource

frontiers in Microbiology

Past, Present, and Future of Gastrointestinal Microbiota Research in Cats

73

Are there changes in GI microbiome with CE?



Drut Frontiers 2024

74

What about the fecal microbiome?

Characterization of the fecal microbiome in cats with inflammatory bowel disease or alimentary small cell lymphoma

- Lower fecal microbial diversity
- Patterns of dysbiosis like people with IBD
- Decreased numbers of obligate anaerobes (Firmicutes, Bacteroidetes, and Actinobacteria)
- Increased numbers of facultative anaerobes (*Enterobacteriaceae* and *Streptococcaceae)*

75

Prognosis

- LPE
- Diet + prednisolone +/- chlorambucil
- ???

- LGITL
- Prednisolone + chlorambucil
- Diet?
- Median survival time 1.5 - 3 years

Marsilio VCNA 2021

76

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GI perforation

Spontaneous gastrointestinal perforation in cats: a retrospective study of 13 cases

- Lymphoma may be a cause of spontaneous perforation in cats. Therefore, histological examination of ulceration is essential in all cases.
- The direct and sole implication of anti-inflammatory administration in a gastrointestinal perforation is not clearly established in this study.

Bernardin JFMS 2015

78

Feline large-cell lymphoma following previous treatment for small-cell gastrointestinal lymphoma: incidence, clinical signs, clinicopathologic data, treatment of a secondary malignancy, response and survival

- 748 cats with lymphoma
- 12 cats treated for small-cell GI lymphoma (12/121) were diagnosed w any anatomic form of large cell lymphoma subsequently
- Include large-cell lymphoma as a differential in cats diagnosed with small-cell GI lymphoma that develop weight loss, anemia, hypoalbuminemia or hypoproteinemia.

Wright JFMS 2018

79

Thank you for your attention!

hypurr@aol.com

80

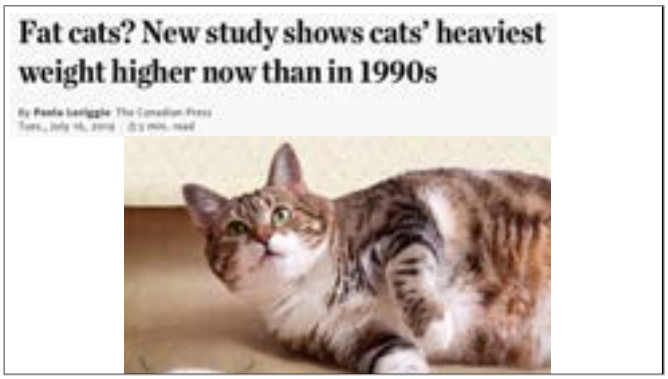
4004

OBESITY: WINNING THE BATTLE OF THE BULGE TAKES MORE THAN A BAG OF FOOD

FELINE

Margie Scherk, DVM, DABVP

1



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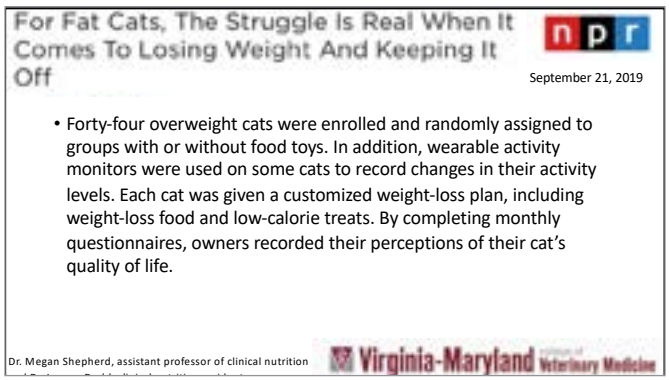

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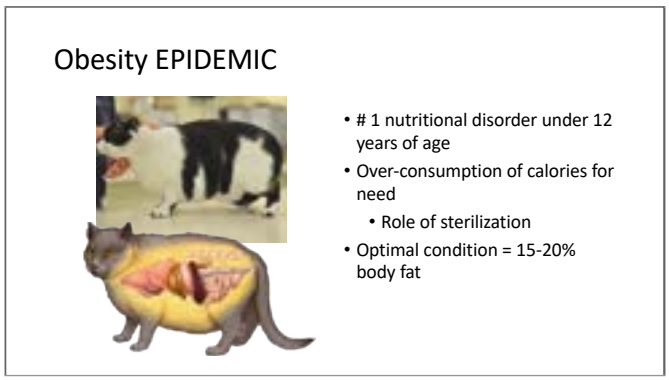

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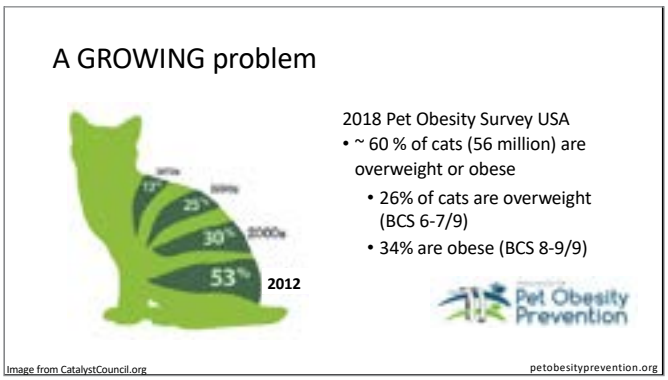
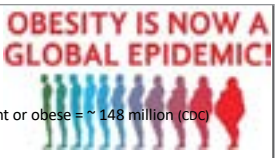


6

What about humans?



- In 2010, 68% of adult Americans were overweight or obese = ~ 148 million (CDC)

2016 WHO fact sheet:

- Worldwide obesity has nearly tripled since 1975.
- Most of the world's population live in countries where overweight and obesity kills more people than underweight.
- > 1.9 billion (39%) adults, > 18 years, were overweight. Of these over 650 million (13%) were obese.
- 41 million children < 5 years were overweight or obese
- > 340 million children and adolescents aged 5-19 were overweight or obese

www.who.int/news-room/fact-sheets/detail/obesity-and-overweight

World Health Organization

7

8

These 14 animals are the 'Biggest Losers' of the pet world

9

Overview: problem & solutions

- Consequences
- Causes
- Need to address
  - Cat (Diet, Activity)
  - Client (Diet, Activity)
  - Environment
- Solutions: immediate weight loss protocol , long-term plan bigger picture

10

Consequences…

- Obesity is a risk factor for:
  - Diabetes mellitus
  - Skin disease
  - Hepatic lipidosis
  - Lameness
  - Neoplasia
  - Oral cavity disease
  - Urinary tract disease
- Risk is proportional to duration
- Middle-aged, obese cats have a 2.7 times greater risk of mortality than cats at optimal body weight.

Scarlett 1998, Lund 2005

11

Consequences: other species (and cats)

- Hyperlipidemia
- Feline lower urinary tract disease
- Insulin resistance and glucose intolerance
- Anaesthetic complications
- Dyspnea, Pickwickian syndrome, exercise intolerance, heat intolerance
- Impaired immune function,
- Exacerbation of degenerative joint disorders

ottawacatgrooming.com

12

13

14

15

16

Intrinsic risks

- Male
- Mixed breed
  - Genetics?
  - Awareness, husbandry?
- Middle age
- Gonadectomized

nytimes.com

17

Body composition

Assessment of body weight for age determination in kittens

- Kittens: ~ 1lb/month until 10 weeks of age
- Reach adult weight at 12 months of age
- General rule:
  - Increases until 2 years
  - Plateaus
  - Decreases after 12 years
- Among neonatal kittens, predicted age based on the 1 lb (0.45 kg) of body weight gain per month of age guideline corresponded to within 1 week of actual age for 98.8% at 2 weeks, 95.1% at 4 weeks, 82.5% at 6 weeks and 77.6% at 8 weeks of age

DiGangi JFMS 2019

18

Investigation of relationships between body weight and age among domestic cats stratified by breed and sex

- Records from 19 million cats (1981-2016)
- Mean BW of non-pedigree cats peaked at 8 years
- Mean BW for cats of the 4 most common recognized breeds (Siamese, Persian, Himalayan, and Maine Coon) peaked between 6-10 years of age and then declined
- Weight subjectively higher for neutered than for sexually intact cats
- Mean BW of neutered 8-year-old non-pedigreed cats increased between 1995-2005 then levelled off between 2005-2015

Campigotto JAVMA 2019

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Effect of surgical sterilization

- Gonadectomy reduces metabolic energy needs by 7-33% (20-25%)
  - From 60-80 kcal/kg/day to 40-50 kcal/kg/day
- Counsel clients at time of neutering

20

Effect of surgical sterilization

- Increased leptin levels
- => reduced insulin sensitivity
- + Glucose intolerance
- *Potential* for diabetes

21

Extrinsic risks

- Single or 2 cat households
- Living indoors
- Inactivity
- Boredom
  - No challenge
- Limitless food
  - Calorically dense, palatable
- Caregiver
  - Food = love
  - Stress

Care2.com

22

Meeting the a cat's needs

Feeding routine
- Hunt and eat alone
- Hunting challenge
- Success and failure
- Multiple SMALL meals/day
- Control and choice

zooplus.co.uk

23

24

Feeding is love

"Pets don't open the fridge by themselves," so stressed owners may stress-feed their pets, Dr. Linder says. "The concept of food and love are tightly interconnected, and we need to address it."

One of the best ways to get a cat to love you is through its stomach!

Our Fat Pets, Susan Jenks, New York Times, Aug 2, 2018

25

Caregiver lack of awareness

- Myth: Cats only eat as much as they need"

- A 4 kg cat consuming a mere 20 kcal/day in excess of its daily energy needs (the equivalent of about 10 kibbles) will gain 12% of its body weight in just 1 year.

26

Define "need"

- More than metabolic/physiologic
- Stress/distress
  - Anxiety
  - Boredom

27

28

Which has more calories?

29

=

30

Meeting a cat's needs

- Anxiety and depression
- Failure to recognize satiety

pets4homes.co.uk

31

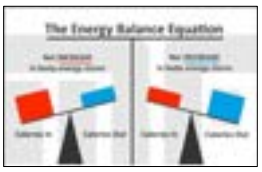
Obesity management

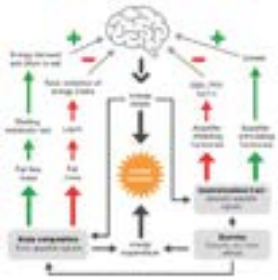
Dailymail.co.uk

32

Lots of weight loss programs

- Decrease caloric intake





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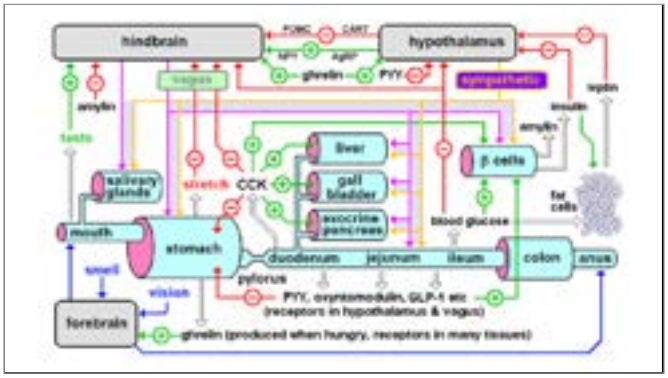

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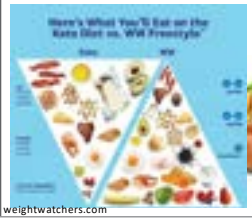
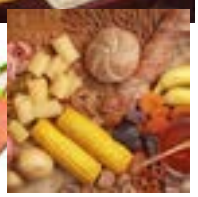
Lots of weight loss programs

- Decrease caloric intake
  - Micronutrient balance



Type of food



weightwatchers.com

35

Felineculture.com

36

37

Purpose designed weight loss diets

- Calorie restricted
- Meet nutritional requirements
  - Macronutrients
  - Micronutrients
- Sufficiently palatable

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Effects of Protein: Carbohydrate Ratios on Weight Loss in Overweight Cats

- Goal:
  - To compare effects of two diets w comparable levels of dietary fat but differing in protein-carbohydrate ratios of 4.44 (HPC) & 1.03 (LPC) on weight loss, fat loss, & body composition in overweight cats
- 30 overweight cats divided into 2 groups
  - Groups matched for MER, weight, % body fat
  - Random assigned to diet

ACVIM

39

Protein: Carbohydrate Ratios & Weight Loss

- Fed 75% of their MER for 6 months
- Monitored daily food intake, weekly body weight, and monthly quantitative magnetic resonance for body composition
- Results:
  - No signif difference in weight loss
  - HPC cats lost twice as much body fat
  - Mean % body fat in LPC did not change over the study

Pan Y ACVIM 2015

40

Success or failure: reality check

41

Rate of weight loss

Research setting

- 0.5-2%/week of starting weight
  - All cats are healthy
  - ~ 20% overweight
  - 50-75% MER
  - Lose weight in 3-6 months

Hoelmkjaer Vet Med Res Reports 2014

nc3rs.org.uk

42

Rate of weight loss

Real life

- 0.5-1%/week
  - Concurrent illness common
  - May be very obese
  - Rate of weight loss slows over time
- Caregiver – cat relationship
- Hidden calories

USAtoday.com

German JFMS 2010, German Acta Vet Scand 2016, Flanagan Plos 2018

43

An international multi-centre cohort study of weight loss in overweight cats: Differences in outcome in different geographical locations

PLOS ONE

- 3-month weight loss program
- 188 practices, 22 countries, 730 cats
- Median BCS 8 (7-9)
- 413 completed program
- Fed commercially available wet or dry weight loss diets
  - Median energy intake was 53 kcal/kg $BW^{0.711}$/day
- 402/413 (97%) lost weight
  - 0.3-1.3%BW/week
- Quality of Life questionnaire
  - Activity, QoL improved
  - Begging decreased

Flanagan Plos 2018

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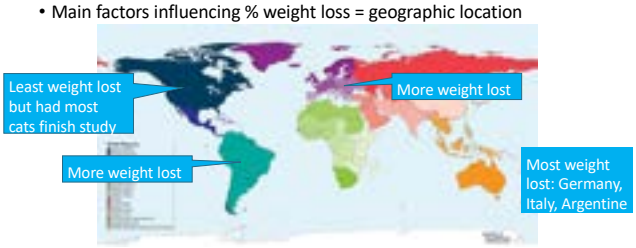
An international multi-centre cohort study of weight loss in overweight cats: Differences in outcome in different geographical locations

PLOS ONE

- Main factors influencing % weight loss = geographic location



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Success and failure

- Royal Canin Weight Management Clinic, 62 client-owned cats
- Rate of weight loss steadily decreased throughout the weight loss period
  - Energy intake required to maintain weight loss also progressively decreased
- By day 84, good compliance, but only 5% had reached target weight
  - > 80% continued to lose weight, losing 6% BW
- Conclusion: First few months not representative of entire weight loss process

Deagle BSAVA 2015

46

Evaluation of a Weight Management Food Designed to Increase Basal Metabolism in a Home Setting

- Test diet designed to increase basal metabolic rate
- Daily ration of the test food to achieve ideal body weight (IBW) was estimated using morphometric measurement method and online tool
- 60 days, 81% of cats lost weight
  - 52% of cat owners perceived weight loss
  - Found it "easy" and cats were "full and satisfied"

Towell, IJARVM 2015

47

48

Clarify expectations

- Weight loss of 6-10% can improve health and QoL significantly
  - Concurrent illness may improve
  - Improved mobility (dogs)
- Initial weight loss more rapid than later
  - 0.8%/week for 28 days then decreases to < 0.3% after 12 months even with decreasing energy intake
- Redefining success
  - Improves compliance

German Acta Vet Scand 2016, Daegle JVIM 2015, Deagle BSAVA 2015 bettergraph.com

49

Clarify expectations

Effect of feeding a weight loss food beyond a caloric restriction period on body composition and resistance to weight gain in cats

- REAL WORLD: With a lot of weight loss, may lose a lot of muscle

Deagle BSAVA 2015

50

Set realistic goals

- Lifelong process
- Select interested and engaged clients

Don't waste your breath

51

Set realistic goals

- Lifelong process
- Select interested and engaged clients

- Clients keen for first 12 weeks but by 48 weeks only 32% of cats remained in program
- Client screening is key

German 2016

52

Set realistic goals

53

Caring follow-up ⇔ Compliance

- Compliance/Adherence
  - Active understanding
  - Being engaged
  - Being able to perform
- EDUCATION & *caring follow-up*
  - Progress reports
  - Good investment showing caring & dedication
- Remember, weight loss will slow

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Tailor weight management program

something WeCanDo

custom MADE

Richmondfamilymagazine.com

55

Tailor weight management program

something WeCanDo

custom MADE

- Determine "ideal" weight (IBW)
- Determine realistic weight – aim for 10% weight loss
- 80% of clients are able to achieve this goal
- Discuss new targets or how to maintain this weight
- May be better with concurrent illness

German 2016

56

Changing behaviour - beyond the bag

- What motivates the client?
- Must fit their lifestyle
  - Consider their routines and needs
  - Other people living with them
- Are there other forms of interaction with the cat?

57

What motivates the caregiver?

"Thirty percent of owners of overweight cats compared with 12% of owners of normal cats stated that they did not feel very happy prior to acquiring a cat, and the cat was intended to console and encourage them. These results suggest:

- A closer relationship between overweight cats and their owners than between normal cats and their owners,
- More over-humanization of overweight cats than of normal cats,
- A potential role of overweight cats as a substitute for human companions."

Kienzle J Nutr 2006

58

What motivates the caregiver?

- People with overweight/obese cats
  - Underestimate BCS
  - Talk about different things with their cats than do people with cats of normal weight
  - Get positive feelings from watching their cats eat
  - Give same amount of food but give more treats and scraps
  - Feed less canned food
- More interested in their own health
- Less likely to take preventative care of their cat

Kienzle J Nutr 2006

59

Understanding human behaviour

Churchill, Compendium 2010

60

Positive strokes

- Emphasize the positive
  - Other forms of interaction they enjoy
  - Pride in weight loss achieved

61

Set a realistic target

Aim for 10% weight loss

REASSESS

- Determine the "ideal weight" based on wha[illegible] cat [illegible] at about 12-15 months of age, when they last had a BCS of 5/9, or using a morphometric table to determine body fat index (BFI)

62

Hidden calories

A 5 kg cat needs 250 kcal/day. 1 cubic inch of cheddar ≈ 28% of daily requirements

This is equivalent to a double hamburger (540 kcal) for someone on a 2000 calorie/day diet

Treat/dental product	Kcal
Typical commercial cat treats	2-4/piece
Dental treats for cats	1-2/piece
"Cat milk"	2/tsp
2% milk	9/Tbsp
Cheddar cheese	113/28g
Chicken breast (skinless roasted)	51/28g

63

Teach clients

- Accurately body condition score
- MEASURE food
- Communicate exact quantities

64

- "Treasure hunt"
  - Thermic effect of food
  - Small meals, frequently
    - Mentally stimulating, seek
    - Exercise
    - Not gorging

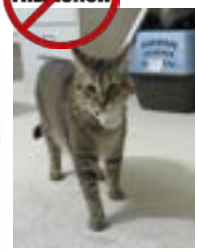
65

FOOD PUZZLES FOR CATS
Feeding for physical and emotional wellbeing

Dantas JFMS 2016

66

Put the hunt back in meal time



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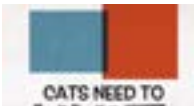

Indoor Hunting Cat Feeder

68

Drive to hunt is always turned on

Small frequent meals
Many hunting failures
Mental stimulation/challenges

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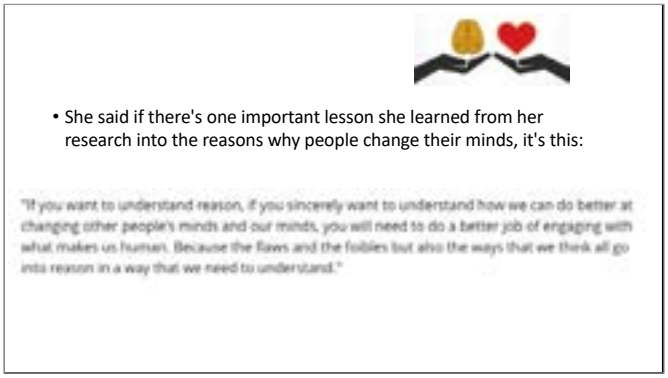

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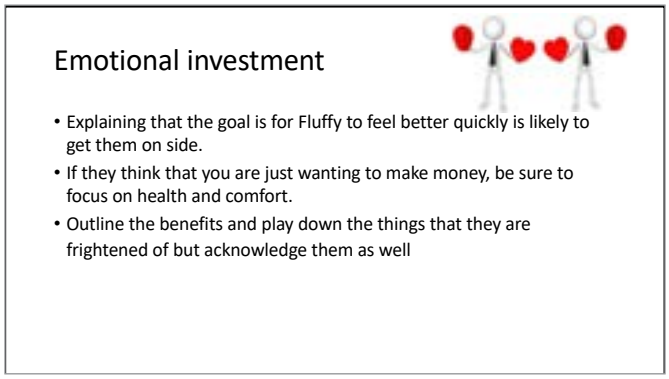

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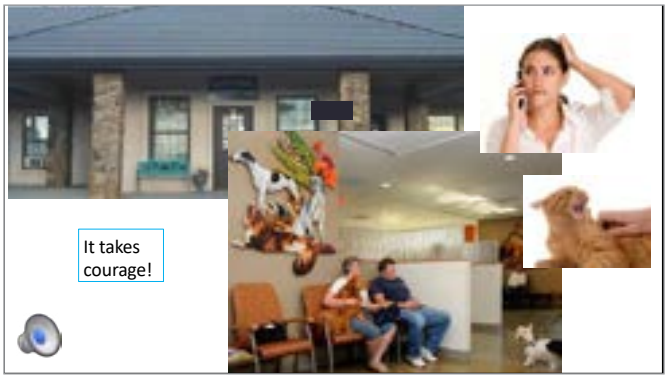

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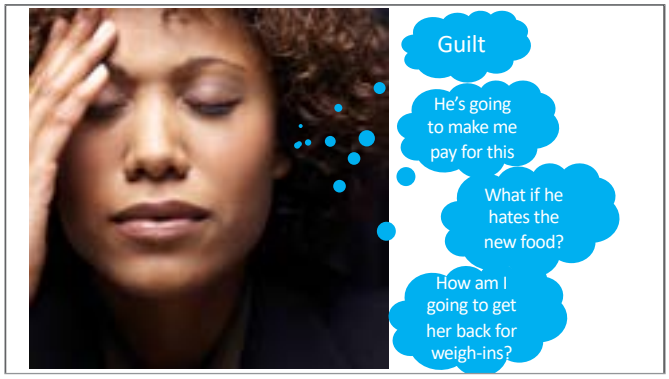

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Make it easy

- Multiple small, painless, incremental changes
- Provide positive reinforcement
- Check up on progress and show commitment to their success

79

Be supportive!

- Emphasize benefits rather than risks

80

81

Investigation of relationships between body weight and age among domestic cats stratified by breed and sex

- 9.9 million (52%) of cats had only 1 BW on record

Campigotto JAVMA 2019

82

83

First year of life…shaping lifelong habits

- Teach about how cats hunt and eat
  - Many SMALL meals (8-10/d)
  - Problem solving
  - Puzzle feeding
  - **Measure amount**
  - Expose to variety
- Weigh and calculate % weight change every visit
- Remind to decrease quantity after gonadectomy
- 9 month nutrition consultation as part of program
  - Reassess what, how much and how kitty is being fed
  - Teach how to body and muscle condition score
  - Determine if kitty is over-conditioned

84

Small changes are big news

85

Physical Exam

1) Attitude/Appearance ☐N ☐A ☐NE BCS ____ / Muscle Condition ____	**2) Oral Cavity/Teeth** ☐N ☐A ☐NE Breath odour ____	**3) Mucous Membranes** ☐N ☐A ☐NE Colour ____ CRT ____ SEC Moisture ____	**4) Integumentary** ☐N ☐A ☐NE Skin tent: Present ☐ Absent ☐
5) Eyes ☐N ☐A ☐NE	**6) Ears** ☐N ☐A ☐NE	**7) Cardiovascular** ☐N ☐A ☐NE	**8) Respiratory** ☐N ☐A ☐NE
9) Gastrointestinal ☐N ☐A ☐NE	**10) Musculoskeletal** ☐N ☐A ☐NE	**11) Lymph Nodes** ☐N ☐A ☐NE	**12) Urogenital** ☐N ☐A ☐NE
13) Nervous System ☐N ☐A ☐NE	**14) Pain** ☐Y ____/10 ☐N	**BP** ____ Cuff Size ____ Limb ____	
T ____ PR ____ HR ____ RR ____ Wt ____ % wt ____			
Frequency ☐AM ☐PM ☐Free choice # ____ Meals Amount ____ Diet ____ ☐Treats ☐Supplements			
☐Indoors ☐Outdoors ☐Contact with others ____			**N** – Normal **A** – Abnormal **NE** – Not Examined
Other observations/findings ____			

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Assessing body composition

- Body condition score: 1-5, or 1-9
  - At every visit
- Emaciated, thin, ideal, heavy or grossly obese
  - Ideally: pelvis, ribs readily palpated but not seen or felt above skin surfaces
  - Readily able to palpate abdominal organs

Assesses body calorie balance

At EVERY visit

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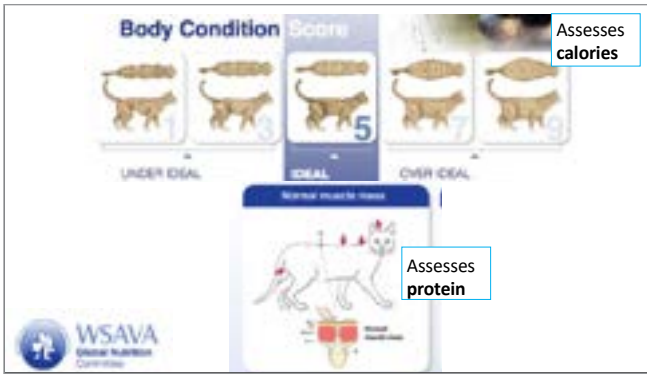

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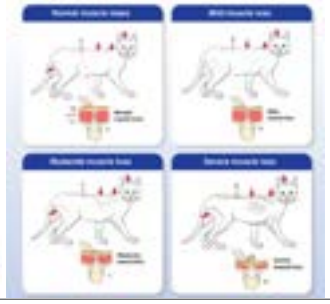
Muscle Condition Score



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Physical Exam

1) Attitude/Appearance ☐N ☐A ☐NE BCS ____ / Muscle Condition ____	2) Oral Cavity/Teeth ☐N ☐A ☐NE Breath odour ____	3) Mucous Membranes ☐N ☐A ☐NE Colour ____ CRT ____ SEC Moisture ____	4) Integumentary ☐N ☐A ☐NE Skin tent: Present ☐ Absent ☐
5) Eyes ☐N ☐A ☐NE	6) Ears ☐N ☐A ☐NE	7) Cardiovascular ☐N ☐A ☐NE	8) Respiratory ☐N ☐A ☐NE
9) Gastrointestinal ☐N ☐A ☐NE	10) Musculoskeletal ☐N ☐A ☐NE	11) Lymph Nodes ☐N ☐A ☐NE	12) Urogenital ☐N ☐A ☐NE
13) Nervous System ☐N ☐A ☐NE	14) Pain ☐Y ____/10 ☐N	BP ____ Cuff Size ____ Limb ____	
T ____ PR ____ HR ____ RR ____		Wt ____ % wt Δ ____	
Frequency ☐ AM ☐ PM ☐ Free choice # ____ Meals Amount ____ Diet ____ ☐ Treats ☐ Supplements			
☐ Indoors ☐ Outdoors ☐ Contact with others ____			N – Normal A – Abnormal NE – Not Examined
Other observations/findings ____			

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Body condition/nutritional assessment

- Key to determining appropriate diet
- Individual!
- At every visit:
  - Body weight
  - Body composition:
    - Body condition score
    - Muscle condition
  - % weight change (subtle changes, trends)
  - Dietary history

$$\frac{\text{Previous wt} - \text{current weight}}{\text{previous weight}}$$

Example:

- $\frac{5.0\text{ kg} - 5.5\text{ kg}}{5.0\text{ kg}} = 10\%$ gain

That's 6.3 kg (14.0 lb) on me!

91

Cat's weight (kg)	Percentage overweight	Equivalent weight for a 5'4" adult woman (kg)	Equivalent kg over-weight for adult woman	Equivalent weight for a 5'9" adult man (kg)	Equivalent kg over-weight for adult man
4.5	0%	65.8	0	76.7	0
4.9	10%	72.6	6.8	84.4	7.7
5.9	30%	85.7	20.0	99.8	23.1
6.8	50%	98.9	33.1	115.2	38.6
9.0	100%	131.5	65.8	153.3	76.7
9.5	110%	138.3	72.6	161.0	84.4
10.4	130%	151.5	85.7	176.4	99.8
11.3	150%	164.7	98.9	191.9	115.2

Adapted from the Association for Pet Obesity Prevention

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Other body composition assessment options

- Radiographs: falciform, paralumbar, perirenal fat
- Ultrasound
- Dual energy X-ray absorptiometry (DEXA)
  - Bone density, muscle mass, fat
- Morphometric Measurements - University of Tennessee
- Body Fat Index

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DEXA: Lean body mass

Dual-energy x-ray absorptiometry
Image courtesy of Royal Canin Weight Management Clinic

- With increasing age, LBM decreases
- LBM = skeletal muscle, organs, bone, skin
- LBM drives metabolism
- Therefore, as muscle decreases, metabolic energy requirements decrease

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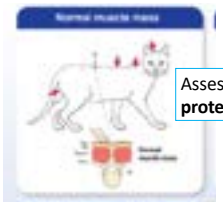
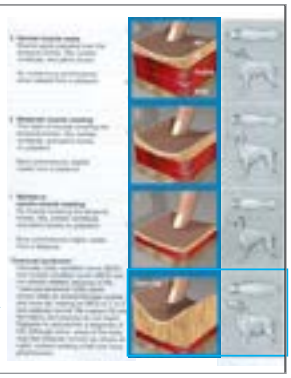
Don't miss sarcopenic obesity



Assesses **protein**



Overcoat syndrome,
Sarcopenic obesity

95

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Body fat estimates

5 point BCS	% Body fat	9 point BCS	% Body fat
3	16-25	5	11-27
4	25-35	6	28-32
5	36-45	7	33-38
		8	38-44
		9	45-47

16-25% body fat is healthy

98

99

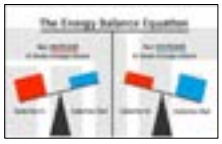
What to feed for weight loss

- Satiety ⇔ protein needs & energy needs
- Protein protects against loss of lean muscle mass
- Diet with 45% calories from protein vs. diet with 35% calories from protein:
  - Less lean and more fat lost with first strategy
  - Similar total weight lost and rate of loss
- Benefits of higher protein variable results
  1. Eating more calories from protein didn't result in weight gain
  2. Eating more protein → satiety
  3. More fat lost on isocaloric higher protein diet; lean body mass (LBM) protected – three studies
  4. LBM not protected in 4th study

LaFlamme Intl J Appl Res Vet Med 2005

100

Feeding strategies



1. ~~Smaller amounts normal diet?~~
2. High protein?
   - Stimulates cellular metabolism, satiety, protective
3. High moisture?
   - Works for some cats, may take time to transition
4. High fiber?
   - Works for some cats (decrease calories, induce satiety)
5. Low fat?
   - Decrease calories

101

Diet hack

- Fortiflora probiotic product was useful in getting cats to eat vegetables as a low-calorie snack
- The research team's hypothesis—that cats in the food-toy group would be perceived to have a better quality of life—was not proven. Interestingly, quality of life also was not tied to the amount of weight each cat lost or the cat's "success" in the program.

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Any weight loss program

Three components:
- DIET
- EXERCISE
- FOLLOW-UP

- Client commitment
- Clinic support
- Team work

BEHAVIOUR MODIFICATION

103

Weight loss program

- Initial extended consultation (40 min)
- Evaluate feeding diary
- Clarify current feeding habits and routines
- Comprehensive physical examination
  - Baseline bloodwork? (age and condition)
- *Detailed* history

Successthroughreferrals.com

104

History

- Amounts, type of food (including treats, supplements)
- Milk? Broth?
- People food?
- Hunting?
- Who feeds the cat?
- Frequency of feeding?
- How is food measured?
- Medications? Are meds given in food or with a treat?
- Does cat nibble or gorge?
- What other pets are there in the home?
- Do they have access to this food?
- Does this cat have access to their food?
- Where is cat fed?
- Is there any known stress?
- Activity level?

105

Feeding diary

ideally

- One-two weeks
- All members of household
- Amount and brand
- Food and treats

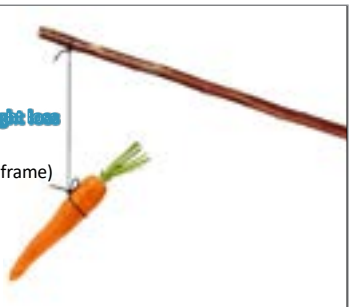
- Provides the info for determining caloric intake that kitty has been receiving

106

Week one: discuss

- Risks of obesity/Benefits of weight loss
- Why cats become overweight
- Realistic goals (weight and time frame)
- Behaviour modification

- Tools:
  - Send home weight loss pack
  - Catnip, cat dancer, toys, comb



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Calculations for weight loss

BCS	5/9	6/9	7/9	8/9	9/9	9+/9
% lean body mass	80	75	70	65	60	< 60
% body fat	20	25	30	35	40	> 40

- To estimate Ideal Body Weight (IBW) you need
- BCS & current weight

- Example – 4 y old NM cat
- BCS 7/9 & he weighs 15lb/6.8 kg
- LBM = 6.8 X 70% = 4.8 kg lean
- 4.8 kg lean / 80% (which is the % LBM at IBW of 5/9) = 6 kg is IBW

From Laura Gaylord webinar Sept 24, 2024

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Calculations for weight loss

- Example – 4 y old NM cat
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- 4.8 kg lean / 80% (which is the % LBM at IBW of 5/9) = 6 kg is IBW

RER = (6 kg IBW) $^{0.75}$ X 70 = 268 kcal/day for ideal weight

DERs for cats:

Intact adult maintenance: RER X 1.4

SF/NM adult: RER X 1.2

Obese prone: RER X 0.8-1.0

For weight loss: RER X 0.8

- What about for weight loss?
- DER = RER (268) X 0.8 (for weight loss) = 215 kcal/kg/day

- Feed 90% of calories complete and balanced = 195 kcal
- 10% reserved for treats = 20 kcal

From Laura Gaylord webinar Sept 24, 2024

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Hugo

BCS	5/9	6/9	7/9	8/9	9/9	9+/9
% lean body mass	80	75	70	65	60	< 60
% body fat	20	25	30	35	40	> 40

Currently: 18.6 lb (8.4 kg) BCS 9/9 & is eating 375 kcal/day

Ideal BW for BCS 5/9: LBM current = 60% X 8.4 kg = 5.1 kg lean

5.1 / 80% ideal lean = 6.4 kg ideal

$6.4^{0.75}$ X 70 = 281 kcal/day (RER)

DER = RER X 0.8 = 281 X 0.8 = 224 kcal/day

224 X 90% = 202 kcal complete and balanced

224 X 10% = 22 kcal treats

110

How long will this take?

Weight loss goal: 0.5-1% current weight/week
(0.1- 0.25 kg/month or 0.24-0.5 lb/month)

- 6-10% is a healthy goal

For Hugo: Weight loss of 3.3 kg (7.3 lb) will take ~ 12 months
But 10% =0.8 kg (1.9 lb) ~ 5-6 months

111

Diet options for cats

	Royal Canin Satiety Support	Hill's Prescription Diet w/d	Purina ProPlan Veterinary Diet OM	Virbac Veterinary HPM Adult Spay/Neuter Cat
Protein (g/100 kcal)	11.5	11.8	15.88	12.3
Fat (g/100 kcal)	3.64	2.5	2.74	4
Fibre (g/100 kcal)	10.77	3.3	4.63	4
Kcal/kg	2887	3163	3237	3410
Kcal/cup	214	278	286	324

From Laura Gaylord webinar Sept 24, 2024

112

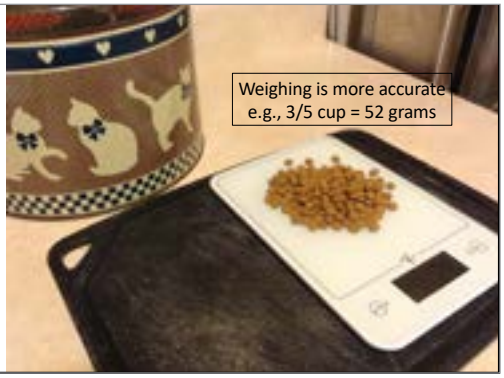
Volumetric measurement is inaccurate

80% over estimate portion size

18% underestimate

German J Anim Physiol Anim Nutr 2001

113



114

Team work!

- Be supportive!
  - Acknowledge and praise
- The client is the one doing the work!
- Discuss benefits of weight loss
- Goal weight may be > ideal weight
- **Discuss length of time this may take**

115

Exercise

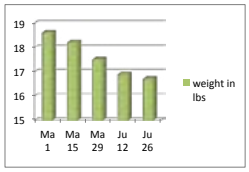
Three 5-minute intense play periods/day

Appropriate toys: dangle, flies, scurries, squeaks?

116

Follow-up!!! The key to a successful program

- Week 1: support phone call (assigned Buddy)
- Week 2: 15 minute visit with DVM supervisor
  - Weigh in
  - Discuss problems, high-lights
  - Update graph
- Weigh in q 2 weeks
  - Update graph
- After 4 months, 15 minute visit with DVM
  - Plateau, new calculations
- Unlimited buddy support (phone)



117

There's more to it

"The simplicity of the energy balance equation has led to an inappropriate focus on obesity as being either a problem of food intake control or of energy expenditure; in practice, a rather more holistic and integrated approach is required"

Trayhurn The Biology of Obesity Proc Nutr Soc 2005

118

Viewpoints

Meeting needs

"Actual components"
- Cat
- Environment
- Caregiver/client

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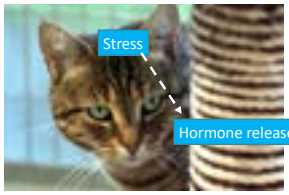


Mental and physical effects linked to stress:
- Activation of immune system
- Release of proinflammatory cytokines
- Changes in mood, depression and pathologic pain

Non-specific clinical and behavioural signs "sickness behaviours", include:
- Vomiting
- Diarrhea
- Hypo-/anorexia
- Fever
- Decreased activity
- Increased sleeping
- Decreased or increased grooming
- Lethargy, and
- Decreased interactivity

120

Five pillars for a healthy environment

Provision of:
1. A safe space
2. Multiple and separated resource stations (food, water, toileting areas, scratching areas, play areas, resting and sleeping areas)
3. Opportunity for play and expression of predatory behaviours
4. Positive, consistent and predictable interactions with humans
5. An environment that respects the importance of a cat's sense of smell

Ellis JFMS 2013

121

Effect of environment

- Diet and environmental history
- Groups
  - 1 = Usual care (diet advice)
  - 2 = Usual care + EE
- Duration – 10 weeks
- Measures
  - Weight
  - Activity
  - Client satisfaction

HAPPY CAT
FELINE ESSENTIALS

Trippany Effects of Environmental Enrichments on Weight Loss in Cats JVIM 2003

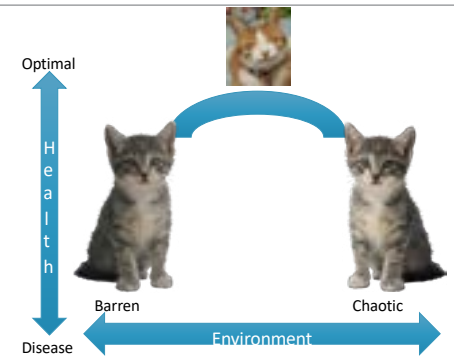
happycatvancouver.com

122

Freedom to express normal feline behaviours

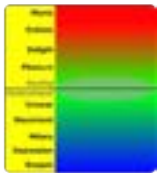
- Play
- Investigation
- Observation
- Hunting
- Feeding
- Drinking
- Grooming
- Scratching
- Travelling
- Scent marking
- Eliminating
- Resting
- Sleeping
- Crepuscular activity

123



124

Lack of control over environment ⇔ stress!



Hedonic budget

125

Ongoing monitoring

darmalanas.wordpress.com

126

Effect of feeding a weight loss food beyond a caloric restriction period on body composition and resistance to weight gain in cats

- 50 overweight cats, prospective clinical study, research setting
- Diet containing coconut oil and supplemental L-carnitine, lysine, leucine, and fiber
- Weight and fat lost but retained lean body mass during the weight loss phase and continued to lose body weight and fat mass but gained lean body mass during the weight maintenance phase
- Metabolomic data suggested that fat metabolism was improved from baseline for cats fed the test food

Floerchinger JAVMA 2015

127

Reasons for failure

Factors associated with overweight cats successfully completing a diet-based weight loss programme: an observational study

BMC

- 62 overweight client-owned cats
- 45% reached target weight
- Reasons for failure related to lack of client support

Effects of weight loss with a moderate-protein, high-fiber diet on body composition, voluntary physical activity, and fecal microbiota of obese cats

- Energy requirements for neutered cats may be overestimated and should be reconsidered

O'Connell BMC Vet Res 2018, Pallotto AJVR 2018

128

Maintaining weight loss

- To prevent rebound weight gain, food measurement MUST be maintained
- Previously overweight cats < 7 years of age at greatest risk

129

something WeCanDo

1. Counsel clients at time of neutering
2. Communicate quantity
   - Caloric density higher in dry
   - **Weigh and measure**
3. Teach re working for food – "hunting"
4. Weigh every visit
5. Assess BCS and MCS
6. Teach BCS to client

130

Multimodal approach most effective

131

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133

Congratulations, Felines and Friends!

Thank you for being part of our weight-loss study.

Virginia-Maryland College of Veterinary Medicine

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4005

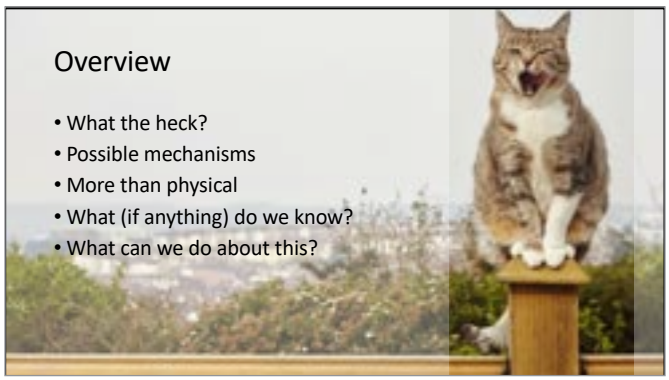
WHY ARE COMORBIDITIES THE NEW NORM FOR CATS?

FELINE

Margie Scherk, DVM, DABVP

1

2



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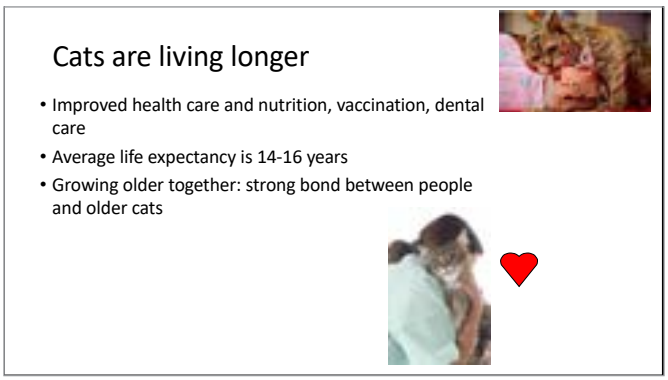


4

5

6

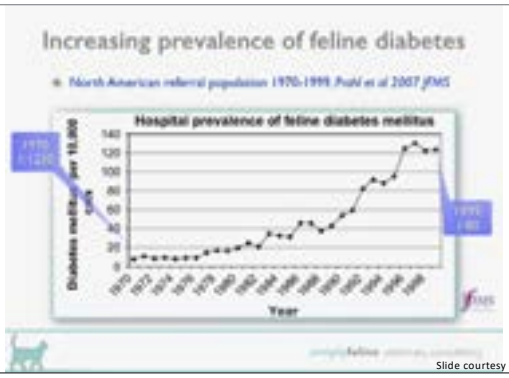
Is it real?

- Cats are living longer
- We are screening

Petersen 2012, Prahl 2007, Lefebvre 2011, Lulich 1992, Reynolds 2013, Ross 2006

depositphotos.com

7



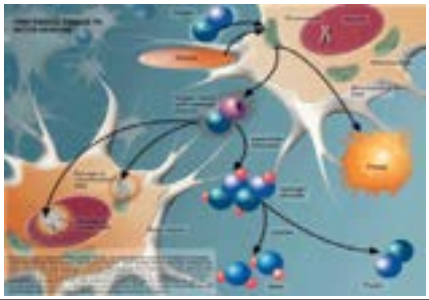
Slide courtesy of Andy Sparke

8

Possible mechanisms for comorbidities

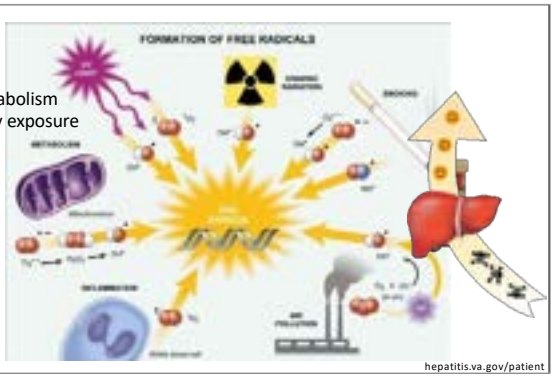
9

Oxidative stress?



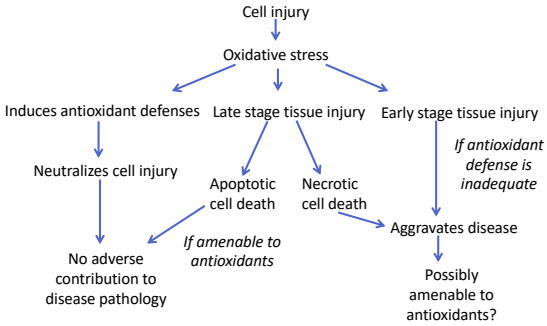
10

Normal metabolism
Enhanced by exposure



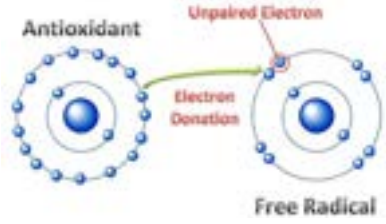
hepatitis.va.gov/patient

11



12

Electron donors



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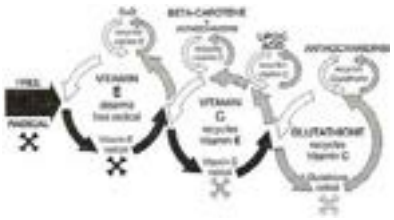
Free radical chain reaction



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Inflammation?

- Prone to inflammation?
  - Lymphocytic plasmacytic response

Inflammation and Cancer: A Comparative View

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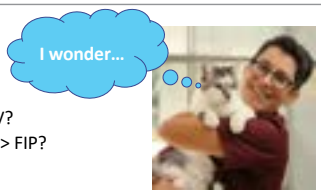
Infectious?

16

Infectious?



- Recrudescence of regressive FeLV?
- Loss of cell mediated immunity => FIP?
- Gammaherpesvirus?
- Morbillivirus?
- Hepatitis B virus?

NOVEL FELINE VIRUSES
Emerging significance of gammaherpesvirus and morbillivirus infections

Beatty JFMS 2019

17

Finding lots of new viruses

- Sequencing technologies + computer-based algorithms
- But what is the clinical relevance?
- Novel viruses *may* contribute to morbidity and mortality…or not
- Potential for treatment and prevention
- EXCITING
- And confusing

Beatty JFMS 2019

18

Gammaherpesviruses – current understanding and pathogenic potential

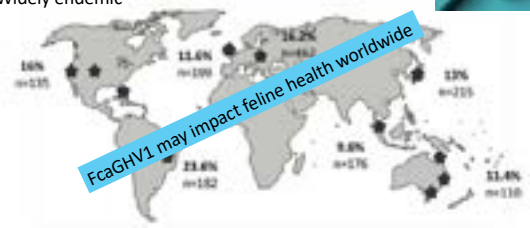
- FHV-1 is an alphaherpesvirus
- *Feline catus* gammaherpesvirus (FcaGHV)
- Human GHVs cause lymphomas arising in immunodeficient patients are causally linked to one or both of the GHVs that infect humans, namely Epstein- Barr virus (EBV) and Kaposi's sarcoma-associated herpesvirus
- FIV-infected cats are at increased risk of lymphoma
- FIV infection on its own isn't lymphomagenic

Beatty JFMS 2019

19

FcaGHV

- Widely endemic



Beatty JFMS 2019

20

Transmission and pathogenesis of FcaGHV1

- Horizontal transmission during territorial aggression is suggested
- Also possibly via oronasal secretions based on swabs and tissues from shelter-housed and client-owned cats (Tse)
- Pathogenicity?
- If FcaGHV1 infection is pathogenic, it is likely that disease would be only a rare outcome of chronic infection, with most infected animals remaining asymptomatic

21

Morbilliviruses – current understanding and pathogenic potential

- FeMV first reported in domestic cats in Hong Kong and China in 2012
  - Since then, Japan, Europe and the Americas
- Original paper suggested link to CKD (Woo)
- The link between FeMV infection and tubulointerstitial nephritis requires definitive pathogenesis studies to assess causation
- Naturally acquired FeMV may be an asymptomatic, self-limiting infection
  Or
- It may cause disease previously attributed to other causes or considered to be idiopathic

Beatty JFMS 2019, Woo Proc Natl Acad Sci USA 2012

22

Immunocompromise and immunosenescence

E XAM INE YOUR ASSUMPTIONS

THE IMMUNE RESPONSE TO VACCINATION

A brief review

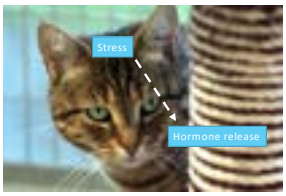
- Nutrition
- Physiology
- Pathophysiology

Scherk JFMS 2013

23

Immunological?

- Mental and physical effects linked to stress:
  - Activation of immune system
  - Release of proinflammatory cytokines
  - Changes in mood, depression and pathologic pain



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What mechanisms may cause comorbidities?

- Direct underlying mechanisms?
  - Ischemia => CKD?
  - Crandall Rees feline kidney cell antibodies => CKD?

New Perspectives
The aging feline kidney: a model mortality antagonist?

Brown Vet Path 2016, Lappin JFMS 2006, Whittemore JVIM 2010, Finch JVIM 2016, Conroy Vet Rec 2019, Lawler JFMS 2006

25

Evolutionary mechanisms do not favor complex and prolonged energy investment

New Perspectives
The aging feline kidney: a model mortality antagonist?

- "… suggests that at least some diseases of late life represent only the point of failure in essentially survival-driven adaptive processes. In the feline renal model, individuals that succumbed to failure most frequently displayed progressive tubular deletion and peritubular interstitial fibrosis but had longer mean life span than cats that died from other causes.

Lawler JFMS 2006

26

New Perspectives
The aging feline kidney: a model mortality antagonist?

- Additionally, among cats that died from non-renal causes, those that had degrees of renal tubular deletion and peritubular interstitial fibrosis also had longer mean life span than those cats with no changes, even though causes of death differed minimally between these latter two groups.
- The data indicate that selective tubular deletion very frequently begins early in adult life, without a clear initiating phase or event."

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'The truth is rarely pure and never simple.'

– Oscar Wilde –

28

29

Speculated comorbid combinations

- Chronic kidney disease (CKD) + hyperthyroidism
- CKD + degenerative joint disease (DJD)
- CKD + heart failure
- CKD + periodontal disease (PD)
- Hyperthyroidism + diabetes mellitus (DM)
- DM + obesity
- DM + CKD
- DM + lower urinary tract disorders
- DM + urinary tract infections + hyperthyroidism + CKD
- Obesity + DJD/DM/cardiac disease/respiratory illness
- Triaditis
- Hypertension + hyperthyroidism
- Hypertension + CKD
- Hypertension + hyperaldosteronism
- Knees and teeth syndrome
- CKD + thin body condition/ PD/cystitis
- Underweight + DJD/DM/CKD/hyperthyroidism/neoplasia

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Diabetes +

- Hyperthyroidism:
  - Glucose intolerance, insulin resistance
- Any chronic inflammatory condition
  - Not just pancreatitis
  - Cell receptor defects
  - Obesity

32

Degenerative joint disease + CKD

Prevalence and classification of chronic kidney disease in cats randomly selected from four age groups and in cats recruited for degenerative joint disease studies

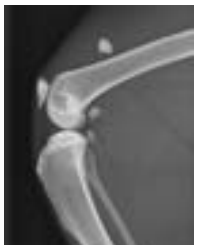
Inflammation from arthritis can take a toll on your kidneys. Here's what you need to know to avoid kidney disease.

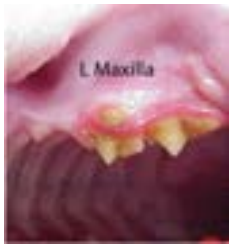
- High co-prevalence could be due to:
  - Perceived changes in activity level and interaction (CKD)
  - Shared etiology of CKD and DJD (inflammatory and immune-mediated)

What Inflammatory Arthritis Does to Your Kidneys

33

"Knees and teeth" syndrome





Langley-Hobbs Vet Rec 2013

34

From having access to information on over 150 patellar fractures some interesting findings have been discovered about this unusual condition. Transverse patellar fractures (Fig 1) most commonly occur in young cats (one to three years old) with over 50 per cent of cats subsequently fracturing the contralateral patella, usually within three months of the first fracture. Up to 75 per cent of these affected cats have deciduous teeth (cheek and/or canine) (Fig 2) persisting beyond six months of age. This syndrome is suspected to be a variation of osteogenesis imperfecta. Other concurrent pathologic traumatic bony lesions include pelvic, humeral, femoral, tibial and fibular fractures, as well as coxofemoral luxations (Langley-Hobbs and others 2009). While surgical correction would seem appropriate for these patellar fractures, failure after pin and tension band was reported in 86 per cent of patients in a retrospective report (Langley-Hobbs and others 2009).

Langley-Hobbs Vet Rec 2013

35

Skull pathology in 10 cats with patellar fracture and dental anomaly syndrome

- Case series describing clinical and radiographic features of mandibular and maxillary abnormalities in cats diagnosed with patellar fractures
- *Patellar Fracture and Dental Anomaly Syndrome (PADS)*
  - Multiple persistent deciduous teeth
  - Gingivitis
  - Jaw swelling
  - Marked bony and periosteal proliferation, hypodontia, root resorption, root malformation and unerupted permanent teeth
  - Osteomyelitis, osteopetrosis

Howes JFMS 2019

36

Skull pathology in 10 cats with patellar fracture and dental anomaly syndrome

- Relevance: radiographically can look aggressive and mimic neoplasia
- Medical therapy:
  - Short-term: antibiotics and anti-inflammatories
  - Long-term: extraction of retained deciduous teeth and unerupted permanent teeth
    - Debridement of proliferative and necrotic bone
- Genetic cause suspected and being evaluated

Howes JFMS 2019

37

Periodontal disease and CKD

Survival analysis to evaluate associations between periodontal disease and the risk of development of chronic azotemic kidney disease in cats evaluated at primary care veterinary hospitals

- 169,242 cats !!!
- PD was associated with increased risk of CKD; risk was highest for cats with stage 3 or 4 PD

Trevejo JAVMA 2018 Allpetsdental.com

38

Obesity

- DJD
- Diabetes
- Cardiac disease
- Respiratory disease
- Neoplasia
- Urinary tract diseases
- Oral cavity disease

- Records from 19 million cats (1981-2016)
- 9.9 million (52%) of cats had only 1 BW on record

Investigation of relationships between body weight and age among domestic cats stratified by breed and sex

39

40

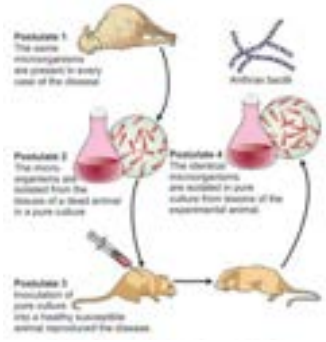
Role of indoor housing?

- Asthma
- Idiopathic inflammatory bowel disease (IBD)
- Pancreatitis
- Cholangitis
- Cholycystitis
- Hyperthyroidism
- Idiopathic cystitis
- Dermatological conditions

Buffington JAVMA 2002

41

42



43

44

Body systems – nice for teaching, but…

- *"The disparity between physical and psychological stressors is an illusion. Host defense mechanisms respond in adaptive and meaningful ways to both."*
- Interconnected

45

Concept: "Sickness behaviours"

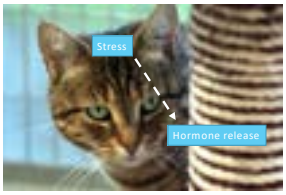
- Vomiting
- Diarrhea
- Hypo-/anorexia
- Fever
- Lethargy
- Increased sleeping
- Decreased or increased grooming
- Decreased interactivity

Buffington JFMS 2014

www.rd.com

46

Psychoneuroimmunology

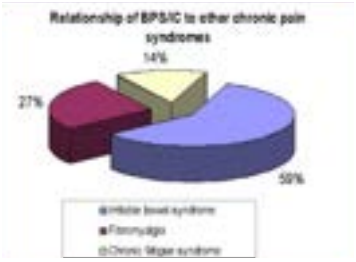


Mental and physical effects linked to stress:

- Activation of immune system
- Release of proinflammatory cytokines
- Changes in mood, depression and pathologic pain

47

Interstitial Cystitis/Painful Bladder Syndrome and Associated Medical Conditions With an Emphasis on Irritable Bowel Syndrome, Fibromyalgia and Chronic Fatigue Syndrome

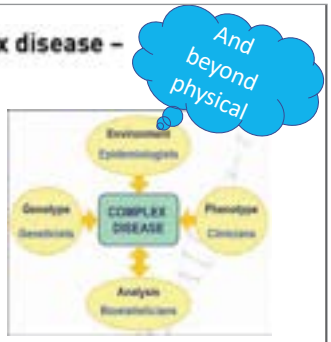


Nickel, et al. J Uro 2010

48

Heavy burdens and complex disease – an integrated perspective

- *"On the basis of a physiological understanding of organ-specific disease and an immunological understanding of infectious and inflammatory disorders, we (contest) that a biomedical understanding is too limited to embrace complex disease and breakdown of functions."*



Kirkengen Tidsskr Nor Laegeforen 2007

49

50

The Dutch famine and its long-term consequences for adult health

Roseboom, Early Human Development, 2006

Transgenerational effects of prenatal exposure to the 1944–45 Dutch famine

Veenendaal, BJOG, 2013

Long-Term Effects of Early-Life Development
Evidence from the 1959 to 1961 China Famine

Almond, National Bureau of Economic Research, 2010

51

Parental Posttraumatic Stress Disorder as a Vulnerability Factor for Low Cortisol Trait in Offspring of Holocaust Survivors

Yehuda, JAMA, 2007

Acute maternal stress in pregnancy and schizophrenia in offspring: A cohort prospective study

Malaspina, BMC Psychiatry, 2008

Association of the World War II Finnish Evacuation of Children With Psychiatric Hospitalization in the Next Generation

Santavirta, JAMA, 2017

52

An abnormal stress response

STRESS

Exaggerated SNS response and catecholamine release

Blunted endocrine and cortisol responses

Slide courtesy of Liz O'Brien

53

FROM FUS TO PANDORA SYNDROME
Where are we, how did we get here, and where to now?

greekgoddesses.fandom.com/wiki/Pandora

Buffington, JFMS 2014

Image from youtube

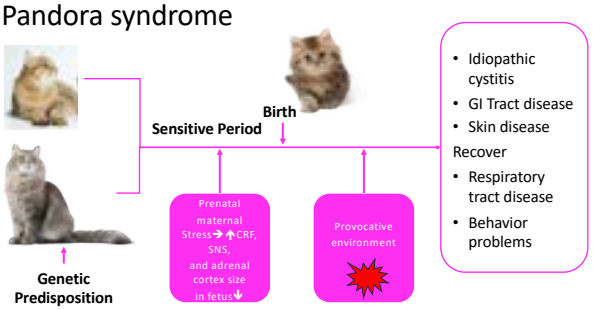
54

Pandora syndrome

- The bladder is not always the perpetrator of LUTS, and suggests that the ***bladder*** can also be one victim of a ***systemic process*** associated with a ***sensitized central stress response system***
- Vulnerability <=**a susceptible individual in a provocative environment**
- May explain impact of stress and waxing-waning nature of signs
- Majority of cats with LUTS have *idiopathic* disease

55

Pandora syndrome



56

What are the causes in cats?

57

Epidemiological study of feline idiopathic cystitis in Seoul, South Korea

- Objective: to investigate potential risk factors for the diagnosis FIC in indoor cats
- n= 58; Questionnaire based, case controlled
- Risk factors:
  - **No vantage point**
  - **Living with other cats**
  - Using non-clumping litter
  - Apartment vs. house
  - Male

Kim JFMS 2018 www.animalwised.com

58

Age and etiology: idiopathic cystitis

- 2-7 years
- Indoor
- Overweight
- Multiple cat homes
- Susceptible to stress

59

60

Science is a way of thinking and approaching a problem

Scientists do not seek to impose their needs and wants on Nature, but instead humbly interrogate Nature and take seriously what they find. We understand human imperfection. We insist on independent, and to the extent possible, quantitative verification of proposed tenets of belief. We are constantly prodding, challenging, seeking contradictions or small persistent residual errors, proposing alternative explanations, encouraging heresy. – Carl Sagan

61

A Comparison of Biochemical and Histopathologic Staging in Cats With Chronic Kidney Disease

- Tubular degeneration, interstitial inflammation, fibrosis, glomerulosclerosis more severe in later stages
- Proteinuria ⇔ increased severity
- Vascular lesions not associated with hypertension
- Therapeutic interventions should target early stages

McLeland Vet Path 2014

Histologic Assessment of the Aging Feline Kidney in Cats Without Kidney Disease

- Similar to humans, renal aging in cats without CKD is characterized by increasing glomerulosclerosis, tubular atrophy, interstitial inflammation, fibrosis and frequency of fibrointimal hyperplasia.

McLeland JVIM Abstract 2019

62

63

64

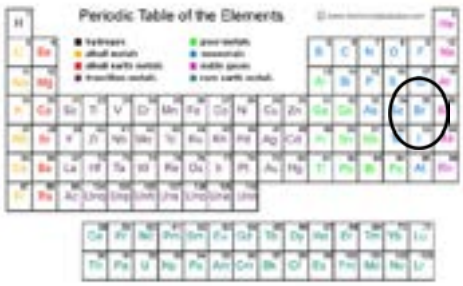
Hyperthyroidism: one environmental etiology

Elevated PBDE Levels in Pet Cats: Sentinels for Humans?

- PBDE flame retardants are believed to be carcinogens
- Elevated PBDE levels in cats
- Link to hyperthyroidism?
- Flame retardants are endocrine –disrupting compounds
- PBDEs phased out 2004-2010

Dye Environ Sci Technol 2007

65



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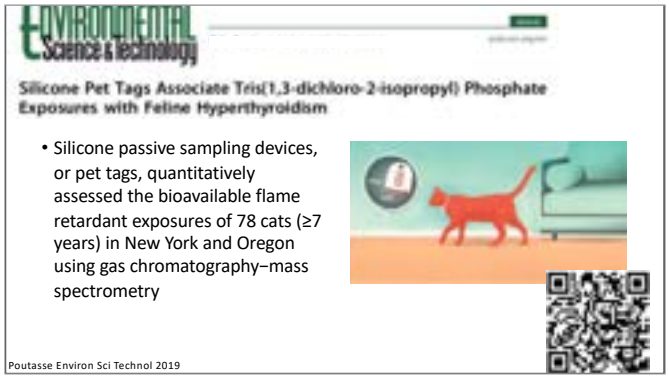

- Silicone passive sampling devices, or pet tags, quantitatively assessed the bioavailable flame retardant exposures of 78 cats (≥7 years) in New York and Oregon using gas chromatography–mass spectrometry

Poutasse Environ Sci Technol 2019

67

- Pet tags were analyzed for 36 polybrominated diphenyl ethers, six organophosphate esters (OPEs), and two alternative brominated FRs
- Exposure of 39 euthyroid cats compared to that of 39 hyperthyroid cats
- Measured total T4, free T4, T3, TSH
- Tris(1,3-dichloro-2-isopropyl) phosphate levels higher in hyperthyroid pet tags
- Higher TDCIPP associated with higher fT4 and TT4

Poutasse Environ Sci Technol 2019

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Summary pathogenesis hyperthyroidism

- Numerous nutritional and environmental factors
- Multifactorial
- Continuous, lifelong exposure to environmental thyroid disruptor chemicals or goitrogens in food or water, acting together in an additive or synergistic manner to affect multiple sites of thyroid hormone metabolism or action, appears first to lead to euthyroid goiter and then to autonomous adenomatous hyperplasia and adenoma, in some cats, eventually progressing to carcinoma

69

- Cats with outdoor access have a lower prevalence of hyperthyroidism

Buffington JAVMA 2002

70

Role of indoor housing?

- Asthma
- Idiopathic inflammatory bowel disease (IBD)
- Pancreatitis
- Cholangitis
- Cholycystitis
- Hyperthyroidism
- Idiopathic cystitis
- Dermatological conditions

Buffington JAVMA 2002

71

72

Meeting unmet needs

Ellis JFMS 2013 www.alamy.com

73

SPECIAL ARTICLE

AAFP and ISFM Feline Environmental Needs Guidelines

1. A safe space
2. Multiple and separated resource stations
3. Providing an opportunity for play and expression of predatory behaviours
4. Providing positive, consistent and predictable interactions with humans
5. An environment that respects the importance of a cat's sense of smell

Ellis JFMS 2013

74

75

"Sickness behaviours"

Effects of stressors on the behavior and physiology of domestic cats

- *"Short term stressors led to a significant increase in SB in both healthy cats and cats with FIC. "*
- *"Daily monitoring of cats for SB may be a noninvasive and reliable way to assess stress responses and overall welfare of cats."*

Stella Appl Anim Behav Sci 2013, Buffington JFMS 2014 Veterinarynews.dvm360.com

76

Worth reconsidering?

Increased risk from going outside

- Infectious diseases (FeLV, FIV, rabies, parasites)
- Vehicular trauma
- Altercative trauma (other animals)
- Other trauma (falls)
- Getting lost
- Theft
- Poison exposure (antifreeze, rodenticide)
- Eating unapproved food
- Pregnancy

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Increased risk from living strictly indoors…

WOW!

- Lower urinary tract diseases (FIC, urolithiasis)
- Hyperthyroidism
- Boredom
- Inactivity, decreased fitness
- Obesity
- Diabetes
- Dental resorptive lesions
- Household hazards (swallowing small objects, burns)
- Poison exposure (cleaning chemicals, plants, medications)
- Secondhand smoke
- Trauma (falls, blind cords, electrocution)
- Problem behaviours (spraying, scratching, aggression)
- Behaviour problems (obsessive behaviours)
- Dermatologic problems (atopic dermatitis, acral lick dermatitis)

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What proof do we have?

- The risk factors for developing diabetes are indoor confinement and physical inactivity rather than the proportion of dry food.
- *Two studies showed increased risk for hyperthyroidism in cats living indoors only.*
- Six studies report that LUT signs are also seen more often in multicat households than in those with single cats, in cats that have recently experienced moving to a new house, and during rainy seasons. Even outdoor access to prey was associated with a lower risk for LUT signs
- *Merely by making environmental modifications in a shelter, the prevalence of upper respiratory tract infections decreased, cats were more relaxed and adoption rates increased*

Slingerland 2009, Buffington 2002, Kim 2018, Gourkow 2006

79

Animal Welfare: Five Freedoms

1. Freedom from hunger and thirst
   - *Ready access to fresh water and a diet to maintain full health and vigour*
2. Freedom from discomfort
   - *Provision of an appropriate environment including shelter and a comfortable resting area*
3. Freedom from pain, injury and disease
   - *Prevention or rapid diagnosis and treatment*
4. Freedom to express normal behaviours
   - *Provision of sufficient space, proper facilities and company of conspecifics*
5. Freedom from fear and distress
   - *Ensuring conditions and treatment which avoid mental suffering*

80

Normal feline behaviours

- Play
- Investigation
- Observation
- Hunting
- Feeding
- Drinking
- Grooming
- Scratching
- Travelling
- Scent marking
- Eliminating
- Resting
- Sleeping
- Crepuscular activity

81

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What can we do? Trends

- Screen healthy cats for insipient disease.
- Look for trends (e.g., weight loss, changes in body condition score [BCS], muscle condition score [MCS], blood pressure, creatinine, etc.) before the values fall outside the normal reference interval.
- **But** be selective. Consider the prevalence not only within the region but also age of the patient. Similar to vaccination, where the goal is to perform risk assessment of the individual, not every patient needs a comprehensive "minimum" database.
- Be aware of the limitations of every test.

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What can we do? Nutrition

- Weigh, BCS, MCS cats at every visit. Serial changes (e.g., percentage weight change) are much more meaningful than absolute numbers or scores. Blood pressure should be measured in all cats over 3 years of age as elevations suggest conditions that should be further assessed.
- Consider nutritional intervention
  - Prevent obesity
  - Free radical damage
  - Slowing/correcting muscle loss

84

What can we do? Avoid sarcopenia

- Digestive changes
  - Ability to digest fat decreases
  - 20% of cats > 14 years have reduced protein digestion
- 15% of cats > 12 years have low body condition
- > 14 years of age, cats have a 15X greater risk for being under condition

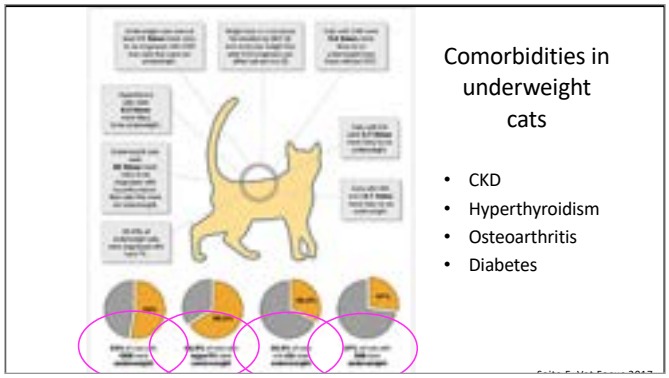
85

Decreasing Lean Body Mass

- Related to increased morbidity & mortality
- Some degree of sarcopenia occurs with normal aging independent of disease

Perez-Camargo G. Compend Contin Educ Pract Vet Suppl 2004

86



Comorbidities in underweight cats

- CKD
- Hyperthyroidism
- Osteoarthritis
- Diabetes

87

What can we do?

- Promote overall good health, *including dental health* regardless of age.
- Optimize nutrition.
- Become informed about the physical and social home environment. Teach the signs that may indicate stress in their cat.
- Optimize hydration. Ensure that litter boxes are pleasant.
- Provide analgesia. Be suspicious of hidden pain or discomfort. Look for, and counsel the client on, the subtle changes that may indicate that pain is present.

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Summary

- Why and mechanisms are unknown
- Consider an integrated view rather than biomechanics

89

Why are Comorbidities the (New?) Norm for Cats?

Thank you!

hypurr@aol.com

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5001

UPDATE ON MANAGEMENT OF COMMON INFECTIOUS CAUSES OF DIARRHEA

INTERNAL MEDICINE

Michael Lappin, DVM, PhD, DACVIM

Clinical problem and differentials. Vomiting is the forceful ejection of stomach and proximal duodenal contents through the mouth. Vomiting can be induced by vestibular, vagal, chemoreceptor trigger zone, or direct input to the emetic center. Diarrhea is a characterized by increased frequency of defecation, increased fluid content of the stool, or increased volume of stool. Markedly increased frequency of defecation, small volume stools, tenesmus, urgency, hematochezia, and mucus are consistent with large bowel diarrhea. Slight increase in frequency of defecation, large volume, melena, steatorrhea, and polysystemic clinical signs are more consistent with small bowel diarrhea. Mixed bowel diarrhea is a combination of characteristics or clinical signs.

Gastrointestinal (GI) signs can be the result of primary diseases of the GI system or secondary GI diseases. The secondary GI diseases are generally those of the kidneys, liver, pancreas (pancreatitis or exocrine pancreatic insufficiency [mainly dogs]), endocrine system (hyoadrenocorticism; diabetic ketoacidosis; hyperthyroidism [mainly cats]), or central nervous system.

Differential diagnoses for primary GI diseases are often grouped into obstruction (masses, foreign body, and intussusception), dietary intolerance, drugs/toxins (garbage gut), inflammatory gastric and bowel diseases, neoplasia, infectious diseases, and parasites. The primary bacteria associated with gastrointestinal tract disease in dogs and cats include *Salmonella* spp., *Campylobacter* spp., *Clostridium perfringens*, *Helicobacter* spp., bacterial overgrowth syndrome, bacterial peritonitis, and bacterial cholangiohepatitis. The primary viral agents include parvoviruses, coronaviruses, canine distemper virus, feline leukemia virus, and feline immunodeficiency virus.

The primary nematodes are *Ancylostoma/Uncinaria, Trichuris vulpis* (dogs), *Strongyloides*, *Dirofilaria immitis* (vomiting in cats), *Toxocara* spp., *Toxascaris leonina*, *Ollulanus tricuspis* (cats), and *Physaloptera* spp. (Table 1). Common enteric protozoans include *Giardia* spp., *Cystoisospora* spp., *Cryptosporidium* spp., and *Tritrichomonas foetus*. The cestodes *Taenia*, *Dipylidium*, and *Echinococcus* generally cause subclinical infection.

Diagnostic plan. Occasionally, otherwise healthy dogs or cats with acute vomiting and normal physical examination findings can be handled conservatively by withholding food for 24 hours followed by introduction of a bland food for several days. For all animals with diarrhea with no apparent cause on physical examination, I will perform a fecal flotation, fecal wet mount examination, complete blood cell count (CBC), and rectal cytology if diarrhea is present. While the CBC generally does not lead to a specific diagnosis, the presence of eosinophilia makes inflammatory bowel diseases and parasitism more likely.

I perform acid-fast staining of a fecal smear or immunofluorescence antibody staining (Merifluor *Giardia/Cryptosporidium*, Meridian Diagnostics) on all animals with diarrhea to assess for the presence of *Cryptosporidium* spp. oocysts. A wet mount examination may aid in identifying trophozoites of *Tritrichomonas* and *Giardia*. If neutrophils or spirochetes are evident on rectal cytology I recommend fecal culture (or PCR) for *Salmonella* spp. and *Campylobacter* spp.. If spore-forming rods consistent with *Clostridium perfringens* are present in large numbers, fecal enterotoxin assay can be performed to help confirm the diagnosis. However, this test can be positive in healthy animals as well and so has less than 100% predictive value

A biochemical profile, urinalysis, FeLV antigen assay (cats), and FIV antibody assay (cats) are indicated if secondary GI diseases are on the differential list or if dehydration is present. I generally perform a total T4 on all cats with vomiting or small bowel diarrhea that are greater than 5 years of age. While amylase and lipase are poor predictors of pancreatitis in cats, a pancreatic lipase immunoreactivity assay has now been validated. It can be used to diagnose pancreatitis (increased) in dogs and cats. The positive predictive value is better for acute pancreatitis than chronic pancreatitis. The negative predictive value (negative test correlates well with a lack of pancreatitis) appears to be high. If an animal with suspected pancreatitis has abdominal effusion, assay lipase concentrations in the serum and effusion; if pancreatitis is occurring the effusion lipase is usually greater than serum.

Fecal fat assessment with Sudan IV stain can help confirm malabsorption/maldigestion but is not specific for a single disease. If the MCV is low, chronic iron deficiency should be suspected; this occurs almost exclusively with gastrointestinal diseases. A serum iron panel can be used to confirm iron deficiency. Panhypoproteinemia is often associated with gastrointestinal tract disease. Measurement of B12, folate, and trypsin like immunoreactivity (TLI) are also used to screen animals for small intestinal bacterial over growth syndrome, inflammatory bowel disease, and exocrine pancreatic insufficiency (TLI).

Most commonly used imaging techniques include radiographs, contrast radiographs, and ultrasound. I commonly perform abdominal radiographs in dogs or cats to support my palpation findings. I use contrast radiographs occasionally; often I perform endoscopy or exploratory laparotomy based on physical examination and abdominal radiographic findings. Ultrasound of the intestinal tract can be hard to interpret and is operator dependent.

Diagnosis of gastric foreign bodies and diffuse inflammatory diseases can be made by endoscopy. Endoscopically obtained biopsies are small; I generally take at least 8-10 biopsies from stomach, duodenum, colon, and ileum if possible. Even if a lesion is present, endoscopically obtained biopsies can be falsely negative requiring full thickness biopsies.

Gastric biopsies should be placed on urea slants to assess for urease which is found in the cell wall of *Helicobacter* spp.. Full thickness biopsies can be made by using laparoscopy to pull appropriate loops of bowel to the abdominal wall and performing a key hole incision for biopsy procurement.

DIAGNOSTIC PROCEDURES FOR INFECTIOUS DISEASES

Infectious diseases are common causes of vomiting and diarrhea. The following is a brief discussion of the commonly used procedures.

Direct smear. Liquid feces or feces that contains large quantities of mucus should be microscopically examined immediately for the presence of protozoal trophozoites, including those of *Giardia* spp., *T. foetus* and *Pentatrichomonas hominus*. A direct saline smear can be made to potentiate observation of these motile organisms. The amount of feces required to cover the head of a match is mixed thoroughly with one drop of 0.9% NaCl. Following application of a coverslip, the smear is evaluated for motile organisms by examining it under 100X magnification. The sample should be fresh. The material for evaluation should be collected from the surface of the fecal material, preferably mucous if present. Alternately, a rectal scraping can be used. The organisms are approximately the size of a neutrophil. *Giardia* has motility that has been described as "falling leaf" and *T. foetus* generally darts forward quickly.

Stained smear. A thin smear of feces should be made from all animals with large or small bowel diarrhea. Material should be collected by rectal swab if possible to increase chances of finding white blood cells. A cotton swab is gently introduced 3-4 cm through the anus into the terminal rectum, directed to the wall of the rectum, and gently rotated several times. Placing a drop of 0.9% NaCl on the cotton swab will facilitate passage through the anus, but not adversely affect cell morphology. The cotton swab is rolled on a microscope slide gently multiple times to give areas with varying smear thickness. Following air drying, the slide can be stained.

White blood cells and bacteria morphologically consistent with *Campylobacter* spp. (spirochetes) or *Clostridium perfringens* (spore-forming rods) can be observed after

staining with Diff-Quick or Wright's-Giemsa stains. *Histoplasma capsulatum* or *Prototheca* may be observed in the cytoplasm of mononuclear cells. Methylene blue in acetate buffer (pH 3.6) stains trophozoites of the enteric protozoans. Iodine stains and acid methyl green are also used for the demonstration of protozoans. Acid-fast or monoclonal antibody staining of a fecal smear should be performed in cats with diarrhea to aid in the diagnosis of cryptosporidiosis. *Cryptosporidium* spp. are the only enteric organisms of approximately 4 to 6 µ in diameter that will stain pink to red with acid-fast stain.

Presence of neutrophils on rectal cytology can suggest inflammation induced by *Salmonella* spp., *Campylobacter* spp., or *Clostridium perfringens*; fecal culture is indicated in these cases. Fecal enterotoxin measurement or enterotoxin gene PCR assays can be considered for animals with spore-forming rods morphologically consistent with *C. perfringens*. However, approximately 10% of healthy animals have enterotoxin in stools and so the predictive value of a positive test is not 100% in animals with diarrhea. The presence of the enterotoxin gene and enterotoxin may have the best positive predictive value.

Fecal flotation. Cysts, oocysts, and eggs in feces can be concentrated to increase sensitivity of detection. Most eggs, oocysts, and cysts are easily identified after zinc sulfate or sugar centrifugal flotation (www.capcvet.org). This procedure is considered by many to be optimal for the demonstration of protozoan cysts, in particular, *Giardia* spp. and so is a good choice for a routine flotation technique in practice. Sugar centrifugation can be used for routine parasite evaluation and may be superior to many techniques for the demonstration of oocysts of *Toxoplasma gondii* and *Cryptosporidium* spp.. *Giardia* cysts are distorted by sugar centrifugation but can still be easily identified. Fecal sedimentation will recover most cysts and ova, but will also contain debris. This technique may be superior to flotation procedures for the documentation of *Eurytrema procyonis*, the pancreatic fluke. *Strongyloides* larva may be easier to identify after concentration using the Baerman funnel technique.

Culture. Culture of feces for *Salmonella* spp. and *Campylobacter* spp. is occasionally indicated in small animal practice. Approximately 2-3 grams of fresh feces should be submitted to the laboratory immediately for optimal results, however, *Salmonella* and *Campylobacter* are often viable in refrigerated fecal specimens for 3-7 days. Appropriate transport media should be available through your laboratory. The laboratory should be notified of the suspected pathogen so appropriate culture media can be used. More than 1 culture may be needed to prove infection. *Tritrichomonas foetus* can be cultured from feces of cats in general practice using a commercially available kit (InpouchTM, Biomed Diagnostics). Some *Giardia* spp. isolated from cats will grow on culture media, but this technique is not generally performed in small animal practice.

Immunologic techniques. Parvovirus, *Cryptosporidium parvum*, and *Giardia* spp. antigen detection procedures are available for use with feces. Canine parvovirus antigen assays appear to detect feline parvovirus antigen. While these assays also can detect vaccine strains of the virus, most animals do not shed enough of the antigens to be positive in the assays. An fluorescent antibody (FA; Meridian Diagnostics) assay for concurrent detection of *C. parvum* oocysts and *Giardia* cysts has been validated for use with dog and cat feces; this assay is commonly available at commercial laboratories. While most human *Giardia* spp. antigen assays (including the SNAP®Giardia) appear to detect dog and cat genotypes, human *Cryptosporidium* antigen assays do not consistently detect *C. felis* or *C. canis*. The *Giardia* assays can be used to increase sensitivity in dogs or cats with diarrhea but should be interpreted in conjunction with results from fecal examination techniques which can be used to detect many other organisms.

Serum antibodies against *D. immitis* and *D. immitis* antigen can be measured in cat serum but positive test results do not prove current infection or disease induced by *D. immitis*. FeLV can cause lymphoma and induces the panleukopenia-like syndrome. FIV has been associated with lymphoma and can cause enteritis. Cats that are immune suppressed by either organism can have secondary causes of diarrhea. Detection of FIV antibodies or FeLV antigen in serum documents exposure, but does not prove that clinical disease is due to the virus. The only way to document that gastrointestinal signs are due to FeLV or FIV is to exclude other known causes.

Electron microscopy. Electron microscopy can be used to detect viral particles in feces of dogs and cats with gastrointestinal signs of disease. Approximately 1-3

grams of feces without fixative should be transported to the laboratory offering this service by overnight mail on cold packs.

Endoscopy or exploratory laparotomy. *Ollulanus* and *Physaloptera* rarely pass ova in feces and so frequently are diagnosed only by endoscopy. Diagnosis of diffuse inflammatory diseases can be made by evaluation of endoscopy or surgically obtained tissue samples.

Endoscopically obtained biopsies are small; I generally take at least 8-10 biopsies from stomach, duodenum, colon, and ileum if possible. Even if a lesion is present, endoscopically obtained biopsies can be falsely negative requiring full thickness biopsies. In these cases, full thickness biopsies can be made by exploratory laparotomy or by laparoscopy and a key hole biopsy.

Gastric biopsies should be placed on urea slants to assess for urease which is found in the cell wall of *Helicobacter* spp.. The combination of inflammation, exclusion of other causes of inflammation, presence of gastric spiral bacteria, and positive urease testing can be used as a presumptive diagnosis of gastric helicobacteriosis. There is no benefit to performing duodenal aspirates for quantitative bacterial cultures or *Giardia* trophozoite evaluations in cats; the normal bacterial count range is very broad in cats and *Giardia* is found in the distal small intestine.

Regional enteritis due to feline infectious peritonitis can be confirmed by documenting the organism in tissue after immunohistochemical staining.

POLYMERASE CHAIN REACTION

Many laboratories now offer pathogen or parasite PCR panels. As discussed, the diagnosis of *Giardia* spp. infection is generally made with the combination of fecal flotation techniques, wet mount examination with or without fecal antigen tests or FA. Fecal PCR assays for *Giardia* are often falsely negative because of PCR inhibitors in stool and so PCR should not be used as a screening procedure for this agent. However, *Giardia* spp. PCR can be used to determine whether the infective species is a zoonotic assemblage which is the primary indication for this technique. However, it now appears that assemblage determination should be performed on more than one gene for most accurate results. Genotyping is available at Antech Diagnostics.

While *Cryptosporidium* spp. infection is common, it is unusual to find *C. felis* or *C. canis* oocysts after fecal flotation. Acid-fast staining of a thin fecal smear is cumbersome and insensitive. Antigen assays titrated for use with human feces are inaccurate when used with cat or dog feces. Thus, PCR may be aid in the diagnosis of cryptosporidiosis in dogs and cats and has been shown to be more sensitive than FA in cats. I personally recommend FA as the screening procedure. *Cryptosporidium* spp. PCR assays are indicated in FA negative cats or dogs with unexplained small bowel diarrhea and when the genotype of *Cryptosporidium* is to be determined (http://dlab.colostate.edu/). However, *C. felis* and *C. canis* infections are common and so positive tests results do not always prove that the agent is the cause of the clinical disease. No drug is known to eliminate *Cryptosporidium* spp. infections and small animal strains are not considered significant zoonotic agents so PCR is never indicated in healthy animals.

PCR assays are also available for amplification of DNA of *Tritrichomonas foetus, Salmonella* spp., *Campylobacter* spp., *Clostridium* spp., parvoviruses, and *T. gondii* and a RT-PCR assay is available for coronaviruses. Trophozoites of *T. foetus* can often be detected on wet mount examination of fresh feces which can be completed as an in clinic test. DNA of *T. foetus* can be amplified from stool of healthy carrier cats and so positive results do not always prove illness from the organism. Cases with suspected salmonellosis or campylobacteriosis should be cultured rather than assessed by PCR to determine the anti-microbial susceptibility patterns. In dogs, the PPV of *Clostridium* spp. PCR assays on feces is low and if used, should be combined with enterotoxin assays. Information in cats is currently lacking.

There is no current evidence that parvovirus PCR on feces is superior to currently available antigen assays. In one recent study in our laboratory, approximately 40% of healthy cats vaccinated with a modified live FVRCP vaccine were PCR positive for panleukopenia virus DNA in feces one week after vaccination. Thus, the currently used assays cannot differentiate vaccine strains from natural infections which should be considered when making case management decisions. *Toxoplasma gondii* is only shed for about 7-10 days and millions of oocysts are generally shed during this time making the organism very easy to identify. Thus, PCR assays are usually not needed to diagnosis this infection.

Because virus isolation is not practical clinically, RT-PCR is used most frequently to detect coronaviruses RNA in feces.

However, positive test results do not differentiate FIP inducing strains from enteric coronaviruses and in one study in our laboratory, there was no association between diarrhea and positive test results for coronavirus in cat feces.

Treatment of select infectious GI diseases. There are multiple drugs used in the treatment of gastrointestinal parasitic infections. For all puppies and kittens, the strategic deworming recommendations for the control of hookworm and roundworm infections from the Centers for Disease Control and the American Association of Veterinary Parasitologists should be followed by veterinarians (www.cdc.gov/ncidod/dpd/parasites/ascaris/prevention.htm; www.capcvet.org). If owners are interested in more in depth information a good website is available (www.petsandparasites.com).

Heavily infected kittens should be administered an anthelmintic at 3, 5, 7, and 9 weeks of age and then periodically monitored or treated. Puppies can be started at 2 weeks of age. If the kitten is not presented to the clinic until 6-8 weeks of age, administer the anthelmintic at least 2-3 times, 2-3 weeks apart. Pyrantel pamoate and fenbendazole are usually effective drugs for use in strategic deworming programs and for the treatment of nematodes causing gastrointestinal tract disease. Albendazole is more likely to cause hematologic side-effects than fenbendazole and so should not be used. Even if anthelmintics for hookworms and roundworms are administered, a fecal flotation should be performed to evaluate for other parasites. Some monthly *D. immitis* preventatives can help control or eliminate some nematode infections as well as prevent heartworm infection. Use of these products is the easiest way to perform strategic deworming after completion of the vaccine and deworming series. *Dipylidium* and *T. taeniaformis* infestations usually are eliminated by praziquantel or espiprantel; fenbendazole is effective for *Taenia taeniaformis*. Since *Echinococcus multilocularis* can be a significant zoonosis transmitted to cats by carnivorism, hunting cats in endemic areas should be treated up to monthly. Administration of a pyrantel/praziquantel combination may be effective in these cats since praziquantel is approved for the treatment of *Echinococcus* and roundworms are also transmitted by carnivorism.

Giardia infections often respond clinically to the administration of metronidazole but infection is usually not eliminated. Administration of metronidazole benzoate at 25 mg/kg, q12hr, PO, for 7 days was effective in suppressing cyst shedding to below detectable limits in 26 cats. This is the maximum dose of metronidazole that should be used; CNS toxicity can be induced by overdosing or as a cumulative neurotoxin. Until recently, fenbendazole has not been studied for the treatment of giardiasis in cats. In eight cats with *Cryptosporidium* spp. and *Giardia* spp. coninfection, only 50% eliminated the *Giardia* infection when treated with fenbendazole. I personally use fenbendazole at 50 mg/kg, PO, daily for at least 5 days in dogs or cats with giardiasis. Since metronidazole can induce significant microbiome changes, started Giardia treatment with fenbendazole or febantel, which do not affect the microbiome is logical (Fukuhia). If I do need to prescribe metronidazole, I concurrently prescribe a probiotic like *Enterococcus faecium* strain SF68 (Purina PetCare; Lappin et al,).

Metronidazole and fenbendazole can be combined in resistant cases but is usually not needed. Febantel containing products have been used successfully in dogs and cats and this drug is approved for the treatment of giardiasis in some countries. The empirical dog dose is the deworming dose, daily for at least 3 days.

There are other drugs that have be used for the treatment of resistant giardiasis (paromomycin, ronidazole, nitazoxanide, others). However, in my experience, dogs or cats with *Giardia* that fails to respond to metronidazole and fenbendazole (or febantel) have another underlying problem. Tinidazole at 30 mg/kg, PO, daily for 7 - 14 days may be effective in some dogs and cats. Secnidazole at 30 mg/kg, PO, once was reported for treatment of cats with *Giardia* in Brazil. Additional information is needed before this protocol can be widely recommended.

Multiple drugs have been evaluated for the treatment of cats with *T. foetus* infections; until recently no drug eliminated infection and diarrhea rarely resolves during the treatment period. Recently ronidazole at 30 mg/kg, PO, q24hr, for 14 days eliminated clinical signs of disease and trophozoites from cats infected with one strain of the organism. In another one small study, administration of metronidazole and enrofloxacin lessened diarrhea in kittens but it is unknown if the organisms infecting those

cats was *T. foetus*. Tinidazole may control the diarrhea but was less likely to eliminate the infection compared to ronidazole. It is possible that some cats with *T. foetus* have other enteric coinfections and so antihelmintics or drugs with activity against *Giardia* spp., *Cryptosporidium* spp., and enteric bacteria like *Campylobacter* spp. are often prescribed. Paromomycin should be avoided cats with bloody stools because of the potential for being absorbed and inducing renal disease or deafness. Some puppies have recently been shown to be infected by *T. foetus*. *Pentatrichomonas hominis* DNA is also sometimes amplified from some dogs or cats with diarrhea; whether disease develops or the optimal treatments are unknown.

Sequential administration of clindamycin followed by tylosin blocked oocyst shedding and resolved diarrhea in one cat with chronic, clinical cryptosporidiosis. Tylosin (10-15 mg/kg, PO, twice daily) has been apparently successful in lessening diarrhea and oocyst shedding in multiple other cats and dogs with diarrhea that were *Cryptosporidium* positive. However, infection is not eliminated. Unfortunately, tylosin is very bitter and usually has to be given to cats in capsules.

Treatment duration may need to be weeks. In cats with naturally occurring cryptosporidiosis, response to azithromycin has been variable (Lappin MR, unpublished data, 2005). If tried, use 10 mg/kg, PO, weekly for at least 10 days. If responding, continue treatment for at least 1 week past clinical resolution. Paromomycin can be effective for lessening diarrhea and oocyst shedding associated with cryptosporidiosis in cats and also is an alternate anti-*Giardia* drug.

However, this orally administered aminoglycoside may cross the diseased intestinal wall to induce renal insufficiency and should never be used in cats with bloody diarrhea. Nitazoxanide is a new drug being studied for the treatment of *Cryptosporidium* and *Giardia*. Little information is available concerning dosages, but we have used 10-25 mg/kg, PO, q12 hours in some of our research studies. The drug Alinia® is available and is labeled for both organisms in humans. The primary side-effect to date has been vomiting and so it should be given with food if used.

The *Toxoplasma gondii* oocyst shedding period can be shortened by administration of clindamycin or sulfadimethoxine. *Cystoisospora* spp. generally respond to the administration of sulfadimethoxine, other sulfa-containing drugs, macrolides, or ponazuril. Ponazuril is superior to other drugs and should be administered at 50 mg/kg, daily for 3 days. If there are multiple puppies or kittens with diarrhea, treatment of all in contact animals should be considered.

Since many of the gastrointestinal parasites that infect dogs and cats are transmitted by carnivorism, they should not be allowed to hunt or be fed raw meats. Additionally, infection of by many parasites results from ingestion of contaminated water. Clinical disease in some parasitized animals can be lessened by eliminating stress and providing a quality diet and clean environment.

Unless signs of bacteremia are present or signs are persistent, most bacterial enteritis cases are now treated by diet change and probiotics. If antibiotics are needed, *Clostridium perfringens* and bacterial overgrowth generally respond to treatment with tylosin, metronidazole, ampicillin, amoxicillin, or tetracyclines. The drug of choice for campylobacteriosis is erythromycin; however, oral administration of tylosin or quinolones is often less likely to potentiate vomiting. Salmonellosis should only be treated parenterally due to rapid resistance that occurs following oral administration of antibiotics. Tylosin can be administered at either 5 mg/kg or 15 mg/kg per dose; 25 mg/kg was shown to not be needed in one study. Boxer colitis is due to *E. coli* and should be treated with enrofloxacin at 5 mg/kg, PO, daily for 6 – 8 weeks. Appropriate antibiotics for the empirical treatment of salmonellosis while awaiting susceptibility testing results include ampicillin or trimethoprim-sulfa; quinolones are also effective. Some animals with infectious diarrhea will respond to the administration of a probiotic. We recently showed administration of *Enterococcus faecium* SF-68 (FortiFlora, Nestle Purina PetCare) lessened diarrhea in shelter cats and to improve metronidazole responses in non-specific diarrhea in shelter dogs. The probiotic is not resistant to metronidazole and so both can be administered simultaneously.

Helicobacter spp. infections are usually treated with the combination of metronidazole, amoxicillin, and bismuth subsalicylate in dogs. Clarithromycin or azithromycin may be logical choices in cats since the species is often difficult to treat with multiple drugs. Whether to concurrently

administer an antacid like famotidine is controversial but seems to lessen vomiting in some cats.

Dogs or cats with apparent bacteremia due to enteric bacteria should be treated with parenteral antibiotics with a spectrum against anaerobic and gram negative organisms. The combination of enrofloxacin with a penicillin or first generation cephalosporin is generally effective. Intravenous metronidazole can also be used. Second generation cephalosporins or imipenem are also appropriate choices. Dogs or cats that have hepatic infections and signs of bacteremia should be treated with antibiotics that kill gram positive, gram negative and anaerobic bacteria as discussed before. In cats, pradofloxacin (Veraflox, Elanco) can be used and it is a 4-quadrant antibiotic. Non septic hepatic infections generally respond to amoxicillin, first-generation cephalosporins, or chloramphenicol. Decreasing numbers of enteric flora by oral administration of penicillins, metronidazole, or neomycin can lessen the clinical signs of hepatic encephalopathy.

Panleukopenia virus, feline leukemia virus, feline immunodeficiency virus, and coronaviruses are the most common viral causes of gastrointestinal tract disease in cats. Viral diseases are managed by supportive treatment. Make sure to maintain hydration, correct hypoglycemia, and maintain normal potassium concentrations. Use of jugular catheters is superior to leg veins since blood samples can be drawn and CVP can be measured. Based on results in dogs with parvovirus infection, administration of a commercially available monoclonal antibody (Elanco Animal Health) or if unavailable, plasma or serum (1 ml/kg) from your hyperimmune blood donor cat may lessen morbidity in cats with panleukopenia or dogs with parvovirus due to passive transfer of immunity. This is effective because parvoviruses induce a viremic state; virus particles are complexed by the antibodies transferred passively. The use of oseltamivir (Tamiflu) in dogs with suspected parvovirus is infections did not have clear benefit in one study. Antibiotics effective against gram negative and anaerobic bacteria are commonly indicated. Vaccines are available for the prevention of parvovirus, coronaviruses, and feline leukemia virus infection.

Histoplasma capsulatum infection is the most common fungal infection of the gastrointestinal tract of dogs and cats in some regions of the United States. Treatment with itraconazole or fluconazole can be effective.

Zoonotic considerations. Infection of people by animal associated enteric agents is usually from contact with feces in the environment, by ingestion of contaminated food or water, or by ingestion of undercooked meat (*T. gondii*). Contact with infected dogs and cats is an unlikely way for humans to acquire infection. The following guidelines may lessen the risk of transfer of enteric zoonotic agents to people.

- Perform a thorough physical examination and zoonoses risk assessment on all new pets.
- Perform a physical examination and fecal examination at least once or twice yearly.
- Take all pets with vomiting or diarrhea to a veterinarian for evaluation.
- Fecal material produced in the home environment should be removed daily, preferably by someone other than an immunocompromised individual.
- Use litterbox liners and periodically lean the litterbox with scalding water and detergent.
- Do not allow pets to drink from the toilet.
- Follow the CDC strategic deworming guidelines.
- Wear gloves when gardening and wash hands thoroughly when finished.
- Filter or boil water from sources in the environment.
- Wash your hands after handling pets.
- Maintain pets within the home environment to lessen exposure to other animals.
- Feed pets only commercially processed food.
- Do not share food utensils with pets.
- Avoid being licked by pets.
- Control potential transport hosts like flies, rodents, and cockroaches.
- Cook meat for human consumption to 80 C for 15 minutes minimum (medium-well).
- Wear gloves when handling meat and wash hands thoroughly with soap and water when finished.

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2. *Leutenegger CM, Lozoya CE, Tereski J, Andrews J, Mitchell KD, Meeks C, Willcox JL, Freeman G, Richmond HL, Savard C, Evason MD. Comparative study of a broad qPCR panel and centrifugal flotation for detection of gastrointestinal parasites in fecal samples from dogs and cats in the United States. Parasit Vectors. 2023 Aug 16;16(1):288. doi: 10.1186/s13071-023-05904-z. PMID: 37587483; PMCID: PMC10433665*
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5002

UPDATE OF SELECT FLEA & TICK-BORNE DISEASES COMMON TO CANADA

INTERNAL MEDICINE

Michael Lappin, DVM, PhD, DACVIM

There are multiple vector borne diseases in dogs; those transmitted by ticks (multiple agents), fleas (multiple agents), mosquitoes (*Dirofilaria immitis*) or sandflies (*Leishmania* spp.) are among the most common. The Companion Animal Parasite Council website (www.capcvet.org) and European guidelines ((https://www.esccap.org/guidelines/) are excellent sources of information about vector borne diseases.

The organisms of the Order Rickettsiales, in the families *Rickettsiaceae* and *Anaplasmataceae*, were reclassified in 2001 following phylogenetic analyses of the 16S rRNA and groESL gene sequences (Dumler and colleagues, 2001). Some *Ehrlichia* spp. were transferred to the *Neorickettsia* genus (including *E. risticii*) and some *Ehrlichia* spp., including *E. phagocytophila* (also called *E. equi* and human granulocytic *Ehrlichia*) and *E. platys)* were placed into the genus *Anaplasma*. The genera *Ehrlichia* and *Neorickettsia* were transferred to the family *Anaplasmataceae;* the genera of *Rickettsia* and *Orientia* remained in the *Rickettsiaceae*. The organisms in the *Ehrlichia*, *Anaplasma*, and *Neorickettsia* genera are classified genetically and by cell tropism (Monocytotropic, granulocytotropic, or thrombocytotropic).

Babesia spp., *Borrelia burgdorferi, Fransicella tularensis, Hepatozoon* spp., *Mycoplasma haemocanis*, and *Rickettsia rickettsii* are also vectored by ticks, relatively common within geographical ranges, and are associated with illness in dogs. It is also possible that some *Bartonella* spp. of dogs, which are usually flea-borne, are tick transmitted. The purpose of this proceedings is to provide attendees an update on the management of a select group of tick borne disease agents that infect dogs and cats in Canada.

CANINE AND FELINE BORRELIOSIS

In some parts of Canada, *Ixodes* spp. ticks capable of transmitting *Borrelia burgdorferi* and *Anaplasma phagocytophilum* occur. For this part of the lecture, the following 2 open access papers will be used as the proceedings.

Hoyt K, Chandrashekar R, Beall M, Leutenegger C, Lappin MR. Evidence for Clinical Anaplasmosis and Borreliosis in Cats in Maine. Top Companion Anim Med. 2018 Jun;33(2):40-44. doi: 10.1053/j.tcam.2018.05.002. Epub 2018 Jun 20. PMID: 30223986.

Littman MP, Gerber B, Goldstein RE, Labato MA, Lappin MR, Moore GE. ACVIM consensus update on Lyme borreliosis in dogs and cats. J Vet Intern Med. 2018 May;32(3):887-903. doi: 10.1111/jvim.15085. Epub 2018 Mar 22. PMID: 29566442; PMCID: PMC5980284.

GRANULOCYTOTROPIC ANAPLASMOSIS

Etiology and epidemiology. *Anaplasma phagocytophilum* (previously *E. equi*, *E. phagocytophila*, canine granulocytic *Ehrlichia*, and human granulocytic ehrlichiosis agent) is known to infect a variety of animals, including small mammals, mountain lions, coyotes, sheep, cattle, deer, dogs, cats, horses, and people. Small mammals and deer are natural reservoirs. The distribution of *A. phagocytophilum* is defined by the range of *Ixodes* ticks and so is most common in California, Wisconsin, Minnesota, and the northeastern states and other areas of the world with this tick genus including Europe, Asia, and Africa. Birds may play a role in spreading infected ticks and may also serve as a reservoir. *Borrelia burgdorferi* is

also transmitted by *Ixodes* ticks and so co-infections can occur. The vector needs to be attached for approximately 24-48 hours in order to transmit the agent. Clinical signs usually develop approximately 1-2 weeks after infection. Neutrophils (and rarely, other leukocytes) phagocytize the organism, and once intracellular, *A. phagocytophilum* prevents phagolysosome fusion. This mechanism allows for multiplication within the phagosome, which gives the appearance of morulae in neutrophils under light microscopy. The exact pathogenesis of disease is still undetermined and it is unclear why some dogs but not others develop clinical signs of disease.

Clinical features. *Anaplasma phagocytophilum* infection appears to be primarily an acute disease in dogs and cats. It has been associated most commonly with non-specific signs of fever, lethargy and inappetence. Stiffness and lameness consistent with musculoskeletal pain are also common and *A. phagocytophilum* has been associated with polyarthritis. Vomiting, diarrhea, difficult breathing, cough, lymphadenopathy, hepatosplenomegaly, and central nervous system signs (seizures and ataxia) have also been reported. Dogs can be chronic, subclinical carriers and so exacerbation of disease could occur in some dogs. However, chronic disease syndromes like those associated with *E. canis* infection have not been documented. In one study of valvular endocarditis, all dogs with *Bartonella* spp. associated disease were also seropositive for *A. phagocytophilum* (MacDonald and colleagues, 2004). Whether the coinfection potentiated the *Bartonella* associated disease is unknown.

Diagnosis. Morulae of *A. phagocytophilum* are commonly detected in neutrophils of most clinically affected dogs and so infection is usually confirmed during performance of a complete blood cell count. While thrombocytopenia and lymphopenia are common, neutrophil counts are usually normal. Reported biochemical panel and urinalysis abnormalities are mild and nonspecific. The morulae cannot be distinguished from those of *E. ewingii*, but the geographical range of the infections varies between the organisms and so the travel history can aid in ranking the differentials. Serologic test results (IFA and ELISA) can be used if morulae are not identified. A point of care assay that detects antibodies against *A. phagocytophilum* is available (SNAP®4Dx, IDEXX Laboratories, Portland, ME) and several commercial laboratories have antibody assays (Antech Diagnostics, Lake Success, NY). Antibody assay results can be falsely negative in acute cases and so a convalescent test 2-3 weeks later may be required to confirm exposure. As *A. phagocytophilum* infections are limited geographically, this antibody test result is not needed in the majority of the United States and Canada. Polymerase chain reaction assays performed on blood collected in EDTA can be used to confirm infection and differentiate *A. phagocytophilum* infection from other infections, but microbial DNA can also be amplified from healthy dogs. Most dogs infected by *A. phagocytophilum* have subclinical infections, most infected dogs only have an acute phase, exposure rates in endemic areas are high, and the disease syndromes associated with infection have multiple other causes. Thus, antibody test results and PCR assay results alone cannot be used to prove clinical disease associated with *A. phagocytophilum* infection.

Treatment. Several antibiotics are effective against *A. phagocytophilum* in vitro. Doxycycline administered at 5-10 mg/kg, PO, q12-24 hr for at least 10 days is recommended by most clinicians. Whether a 28 day course of doxycycline therapy as recommended for *E. canis* is needed is unknown. If tetracyclines are used, 22 mg/kg, PO, q8hr for 2-3 weeks is recommended. Chloramphenicol administered at 15-25 mg/kg, PO, q 8hr for 14-21 days may be effective in puppies and should be used to avoid dental discoloration. Most dogs or cats respond to therapy within hours to days of initiating therapy.

Zoonotic aspects and prevention. *Anaplasma phagocytophilum* infects people as well as dogs and so the organism is zoonotic. Human infections most likely acquired by direct tick transmission, however, handling infected blood and carcasses can also lead to infection. Care should also be taken when handling ticks. There is currently no vaccine for *A. phagocytophilum* infection. Infection can be avoided by controlling ticks or prophylactic use of tetracyclines when visiting endemic areas. In one study, application of imidacloprid-permethrin prevented transmission of *A. phagocytophilum* from naturally infected *Ixodes scapularis* ticks to dogs (Blagburn and colleagues, 2004). In another study, application of permethrin 54.5% and fipronil 6.1% (Effitix; Virbac) was effective for *Ixodes ricinus* and so the product could also lessen likelihood of transmission of *A. phagocytophilum* and *Borrelia burgdorferi* (Bonneau et al, 2015). Dogs appear to be susceptible to reinfection and so tick control should be maintained at all times in endemic areas. Dogs

used for blood donors that reside in endemic areas should be screened for *A. phagocytophilum* infections by serology or PCR (Wardrop et al, 2016).

MONOCYTOTROPIC EHRLICHIOSIS

Etiology and epidemiology. Organisms that are associated with monocytotropic ehrlichiosis in naturally-infected dogs and sometimes cats, include *Ehrlichia canis, E. chaffeensis,* and *Neorickettsia risticii* var *atypicalis*. An individual dog can be infected by more than one ehrlichial agent and coinfections with other tick borne pathogens are common (Kordick and colleagues, 1999).

Ehrlichia canis is the most common of these agents and causes the most severe clinical disease; it is maintained in the environment from passage from ticks to dogs or cats. *Rhipicephalus sanguineus* and *Dermacentor variabilis* are the known vectors. The organism is not passed transovarially in the tick, so unexposed ticks must feed on a rickettsemic dog in the acute phase to become infected and perpetuate the disease. Male *R. sanguineus* can take multiple feedings and can both acquire and transmit *E. canis* in the absence of female ticks. Dogs seropositive for *E. canis* have been identified in many regions of the world and most of the United States, but the majority of cases occur in areas with high concentrations of *R. sanguineus* such as the Southwest and Gulf Coast.

Ehrlichia chaffeensis is a cause of human mononuclear ehrlichiosis. White tailed deer, voles, coyotes, and opossums are reservoirs and *Amblyomma americanum*, *D. variabilis*, and some *Ixodes* ticks are vectors. Infections by *E. chaffeensis* are detected primarily in the southeastern United States. Clinical manifestations in dogs are currently being detailed and appear to be rare.

Ehrlichia canis infection causes acute, subclinical, and chronic phases of disease. Infected mononuclear cells marginate in small vessels or migrate into endothelial tissues, inducing vasculitis during the acute phase. The acute phase begins 1 to 3 weeks after infection, and lasts 2 to 4 weeks; most immunocompetent dogs survive. The subclinical phase lasts months to years in naturally infected dogs. Although some dogs clear the organism during the subclinical phase, the organism persists intracellularly in some, leading to the chronic phase of infection. Many of the clinical and clinicopathologic abnormalities that develop during the chronic phase are due to immune reactions against the intracellular organism. The variable duration of the subclinical phase of disease explains why *E. canis* infection does not have a distinct seasonal incidence like Rocky Mountain spotted fever (RMSF). However, acute phase disease is recognized most frequently in the spring and summer when the tick vectors are most active.

Clinical features. Clinical disease from ehrlichial infection can occur in any dog, but its severity varies depending on the organism, host factors, and presence of coinfections. Virulence is thought to vary with different field strains of *E. canis.* Dogs with depressed cell-mediated immunity develop severe disease.

Clinical findings in dogs with *E. canis* infections vary with the timing of infection. The clinical manifestations of acute phase disease are very similar to those of RMSF, owing to the development of vasculitis. Ticks are most commonly found on dogs during the acute phase of infection. Fever can occur in both clinical phases of infection but is more common in dogs with acute ehrlichiosis. Petechiae or other evidence of bleeding noted during the acute phase are generally caused by a combination of mild thrombocytopenia (consumption or immune-mediated destruction) and vasculitis; thrombocytopenia (consumption, immune-mediated destruction, sequestration, decreased production), vasculitis, and platelet function abnormalities occur in the chronic phase. The thrombocytopenia in the acute phase is generally not severe enough to result in spontaneous bleeding and so bleeding may be primarily from vasculitis and decreased platelet function.

Pale mucous membranes usually only occur in the chronic phase during the development of pancytopenia. Hepatomegaly, splenomegaly, and lymphadenopathy are from chronic immune stimulation (i.e. lymphoreticular hyperplasia) and are detected most frequently in dogs in the chronic phase. Interstitial or alveolar edema secondary to vasculitis or to inflammation, pulmonary parenchymal hemorrhage secondary to vasculitis or thrombocytopenia, or secondary infections from neutropenia are mechanisms resulting in dyspnea or cough in some dogs with ehrlichiosis. Polyuria, polydipsia, and proteinuria are reported in some dogs that develop renal insufficiency.

Stiffness, exercise intolerance, and swollen painful joints occur in some dogs with suppurative polyarthritis. Most dogs with polyarthritis from which the organism has

been demonstrated have been infected with *E. ewingii* or *A. phagocytophilum.* Ophthalmic manifestations of disease are common; tortuous retinal vessels, perivascular retinal infiltrates, retinal hemorrhage, anterior uveitis, and exudative retinal detachment occur. CNS signs can include depression, pain, ataxia, paresis, nystagmus, and seizures.

Diagnosis. Neutropenia is common during acute phase vasculitis and after bone marrow suppression in the chronic phase. Chronic immune stimulation causes monocytosis and lymphocytosis; lymphocytes often have cytoplasmic azurophilic granules (i.e., large granular lymphocytes). Regenerative anemia is from blood loss (acute and chronic phases); normocytic, normochromic nonregenerative anemia is from bone marrow suppression or anemia of chronic disease (chronic phase). Thrombocytopenia can occur with either acute or chronic ehrlichiosis, but is generally more severe with chronic phase disease. Thrombocytopathies from hyperglobulinemia potentiate bleeding in some dogs with chronic ehrlichiosis. Chronic ehrlichiosis is classically associated with pancytopenia, but any combination of neutropenia, thrombocytopenia, and anemia can occur. Changes in bone marrow cell lines associated with ehrlichiosis vary from hypercellular (acute phase) to hypocellular (chronic phase). Bone marrow plasmacytosis is common in dogs with subclinical and chronic ehrlichiosis, and the disease can be confused with multiple myeloma, particularly in those dogs with monoclonal gammopathies. Dogs with ehrlichiosis are usually not hypercalcemic and do not have lytic bone lesions.

Hypoalbuminemia in the acute phase is probably caused by third spacing of albumin in tissues because of vasculitis, whereas in chronic phase disease it is due to glomerular loss from immune complex deposition or chronic immunostimulation (i.e., monoclonal or polyclonal gammopathy). Prerenal azotemia can occur with acute or chronic disease; renal azotemia develops in some dogs with severe glomerulonephritis from chronic ehrlichiosis. The combination of hyperglobulinemia and hypoalbuminemia is consistent with subclinical or chronic ehrlichiosis. Polyclonal gammopathies are most common, but monoclonal (e.g., IgG) gammopathies can also occur.

Aspirates of enlarged lymph nodes and spleen reveal reactive lymphoreticular and plasma cell hyperplasia. Nondegenerate neutrophils are the primary cells in synovial fluid from dogs with polyarthritis caused by any *Ehrlichia* spp.; *E. ewingii* and *A. phagocytophilum* morulae can be identified in synovial neutrophils from some dogs. Bone marrow aspirates in dogs with chronic ehrlichiosis typically reveal myeloid, erythroid, and megakaryocytic hypoplasia in association with lymphoid and plasma cell hyperplasia. Morulae from *E. canis* are rarely detected in the cytoplasm of mononuclear cells. Ehrlichiosis generally causes mononuclear pleocytosis and increased protein concentrations in CSF. Antiplatelet antibodies, antinuclear antibodies (ANA), antierythrocyte antibodies (by direct Coombs' test), and rheumatoid factors are detected in some dogs with ehrlichiosis, leading to an inappropriate diagnosis of primary immune-mediated disease.

No pathognomonic radiographic signs appear in dogs with ehrlichiosis. The polyarthritis is nonerosive, and dogs with respiratory signs most commonly have increased pulmonary interstitial markings, but alveolar patterns can occur. Identification of morulae in cells documents *Ehrlichia* infection, but it is uncommon with monocytotropic strains. Examination of buffy coat smears or blood smears made from blood collected from an ear margin vessel may increase the chances of finding morulae. Some *Ehrlichia* spp. can be cultured, but the procedure is low-yield and expensive and so is not clinically useful.

Most commercial laboratories (using IFAs or ELISA) and one point-of-care diagnostic test (SNAP®4Dx, IDEXX Laboratories, Portland, ME) use reagents that detect antibodies against *E. canis* in serum. These tests are generally used as the first screening procedures in dogs suspected to have ehrlichiosis. The American College of Veterinary Internal Medicine (ACVIM) Infectious Disease Study Group suggests that *E. canis* IFA antibody titers between 1:10 and 1:80 be rechecked in 2 to 3 weeks because of the potential for false-positive results at these titer levels.

If serum antibodies against *E. canis* are detected in a dog with clinical signs consistent with ehrlichiosis, a presumptive diagnosis of canine ehrlichiosis infection should be made and appropriate treatment begun. However, detection of antibodies alone is not diagnostic of ehrlichiosis because some dogs are subclinically infected. Additionally, negative test results do not totally exclude ehrlichiosis from the list of differential diagnoses, because

clinical disease can be detected before seroconversion and not all *Ehrlichia* spp. induce antibodies that consistently detected in *E. canis* assays (Moroff et al, 2014).

PCR assays are now available commercially and can be used to detect organism-specific DNA in peripheral blood. It can be performed on joint fluid, aqueous humor, CSF, and tissues. Blood PCR results can be positive before seroconversion in some experimentally inoculated dogs, and positive results document infection, whereas positive serologic tests only document exposure (Moroff et al, 2014). However, as for serology, no standardization between laboratories currently exists, and insufficient quality control can lead to both false-positive and false-negative results. Until more information is available, the ACVIM Infectious Disease Study Group suggests using PCR with serology, not in lieu of it. Because antibiotic treatment rapidly induces negative blood PCR results, the clinician should draw the blood sample for testing and place it in an EDTA tube before treatment. In one recent study, tissues (lymph nodes, spleen, liver, bone marrow, and blood) from naturally infected dogs were assayed by PCR. Blood and lymph nodes were the most likely to be positive, but were falsely negative in approximately 30% of the samples.

Treatment. Supportive care should be provided as indicated. Several different tetracycline, doxycycline, chloramphenicol, and imidocarb diproprionate protocols have been used. The ACVIM Infectious Disease Study Group currently recommends doxycycline (5 mg/kg, PO, q12hr or 10 mg/kg PO q24h for at least 28 days). In one study of experimentally infected dogs, ticks still could acquire *E. canis* from feeding on dogs previously treated with doxycycline for 14 days (Schaefer and colleagues, 2007). Clinical signs and thrombocytopenia should rapidly resolve. If clinical abnormalities are not resolving within 7 days, other differential diagnoses should be considered. Results of studies using imidocarb diproprionate (5 to 7 mg/kg IM or SQ repeated in 14 days) to treat canine ehrlichiosis have been variable. In one study, thrombocytopenia persisted and infection was not cleared in experimentally inoculated dogs (Eddlestone and colleagues, 2006). Some patients develop pain at the injection site, salivation, oculonasal discharge, diarrhea, tremors, and dyspnea after administration of this drug. Quinolones are not effective for the treatment of *E. canis* infections in dogs.

Positive antibody titers have been detected for up to 31 months after therapy in some naturally infected dogs. Dogs with low (< 1:1024) antibody titers generally revert to negative within 1 year after therapy. Dogs with antibody titers greater than 1:1024 often maintain positive antibody titers after therapy. It is undetermined whether these dogs are persistent carriers of the organism. Based on these findings, antibody titers are considered to be ineffective for monitoring response to therapy. The ACVIM Infectious Disease Study Group recommends monitoring resolution of thrombocytopenia and of hyperglobulinemia as markers of therapeutic elimination of the organism.

It is currently unknown whether ehrlichial infections are cleared by treatment. If PCR is to be used to monitor treatment, the ACVIM Infectious Disease Study Group recommends the following steps be taken: The PCR test should be repeated 2 weeks after stopping treatment. If still positive, treatment should be reinstituted for 4 weeks and retesting performed. If PCR results are still positive after 2 treatment cycles, an alternate anti-*Ehrlichia* drug should be used. If PCR results are negative, the test should be repeated in 8 weeks, and if still negative it can be assumed therapeutic elimination is likely. In one study, PCR assay performed on splenic aspirates was superior to blood PCR to document elimination of infection (Harrus and colleagues, 2004).

Whether to treat seropositive, healthy dogs is controversial. Arguments for and against testing or treating healthy dogs were reviewed by the ACVIM Infectious Disease Study Group. The primary reason to treat a seropositive, healthy dog is to try to eliminate infection before development of chronic phase disease. However, treatment of healthy dogs is controversial for at least six reasons: (1) it is unknown whether treatment halts progression to the chronic phase; (2) not all seropositive dogs are infected; (3) not all seropositive dogs progress to the chronic phase; (4) it is unknown whether treatment eliminates infection; (5) even if infection is eliminated, reinfection can occur; and (6) treatment of healthy carriers may result in antimicrobial resistance. Because further data are needed to make definitive recommendations, owners should be given the pros and cons and asked to make treatment decisions.

The prognosis is good for dogs with acute ehrlichiosis, and it is variable to guarded for those with chronic ehrlichiosis. Fever, petechiation, vomiting, diarrhea,

epistaxis, and thrombocytopenia often resolve within days after initiation of therapy in acute cases. Bone marrow suppression from chronic phase ehrlichiosis may not respond for weeks to months, if at all. Anabolic steroids and other bone marrow stimulants can be administered but are unlikely to be effective because precursor cells are often lacking. Immune-mediated events resulting in the destruction of red blood cells or platelets are likely to occur with ehrlichiosis, leading to the recommendation to administer anti-inflammatory or immunosuppressive doses of glucocorticoids to acutely affected animals. Prednisone (2.2 mg/kg PO divided q12h during the first 3 to 4 days after diagnosis) may be beneficial in some cases.

Zoonotic aspects and prevention. Dogs and people are both infected by *Ehrlichia canis*, *E. ewingii*, and *E. chaffeensis*. Although people cannot acquire ehrlichiosis from handling an infected dog, dogs may be reservoirs for these agents and may play a role in the human disease by bringing vectors into the human environment. Ticks should be removed and handled with care.

Tick control should be maintained at all times as reinfection can occur. Products that repel ticks are likely to be the best products for prevention of *E. canis* infection as the transmission times after tick attachment may be as short as 3 hours. In one study comparing topically applied permethrin/imidacloprid to 2 orally administered acaracides, the topical product was superior for blocking *E. canis* transmission (Jongejan et al, 2016). Use of collars (examples Seresto; Bayer Animal Health; Preventic; Virbac) that also repel ticks can be beneficial for blocking transmission of vector borne agents with short transmission times and can increase compliance.

Because *Ehrlichia canis* is not passed transovarially in the tick, it can be eliminated in the environment by tick control or by treating all dogs through a generation of ticks. *Rhipicephalus* can only transmit *E. canis* for approximately 155 days; if tick control is not feasible, tetracycline can be administered (6.6 mg/kg PO daily for 200 days). During this time, infected dogs will not infect new ticks and previously infected ticks will lose the ability to transmit the organism. Doxycycline given at 100 mg/dog per day was also used successfully as a chemopreventative (Davoust and colleagues, 2005). Dogs used as blood donors should be screened serologically yearly and seropositive dogs should not be used.

Summary. Tick control is warranted for cats as well as dogs. Products with efficacy against fleas should also be used as fleas can be vectors for several *Bartonella* spp., potentially the hemoplasmas, potentially *Coxiella burnetii*, (Cypress), *R. felis* and *Yersinia pestis*.

SELECT FLEA ASSOCIATED DISEASES

Ctenocephalides felis is the most common flea infecting cats and dogs; Pulex and C. canis are occasionally found. Fleas are important in veterinary practice because of being general pests as well as ingesting large volumes of blood as well as being vectors of several important zoonotic diseases. The most risk to people is from *Bartonella* spp., *Rickettsia felis*, and *Yersinia pestis*. *Coxiella burnetii* DNA and FeLV have also been detected in fleas collected from cats but these agents are unlikely to be vectored by fleas. Feline haemotropic *Mycoplasma* spp. (*Mycoplasma haemofelis* and others) were previously thought to be vectored by fleas, the recent information suggests that this route of transmission is unlikely.

The purpose of this part of the lecture is to provide an update on the diagnosis and management of clinical abnormalities associated with Bartonella spp. of cats and dogs. How to recognize bartonellosis in staff members will be emphasized as well as data proving that use of flea control can lessen transmission of flea associated zoonoses to people. Fleas on dogs and cats around the world are potential sources of zoonotic agents. When working with pets with heavy flea infestations, veterinarians should wear gloves or wash their hands carefully.

To supplement the lecture and these proceedings, please download the following open access paper.

Lappin MR, Tasker S, Roura X. Role of vector-borne pathogens in the development of fever in cats: 1. Flea-associated diseases. J Feline Med Surg. 2020 Jan;22(1):31-39. doi: 10.1177/1098612X19895941. PMID: 31916873.

CANINE BARTONELLOSIS

Etiology and epidemiology. *Bartonella vinsonii* subsp. *berkhoffii* was initially isolated from a dog with endocarditis in North Carolina (Breitschwerdt and colleagues, 1995). Since that time, dogs in multiple areas of the world have been shown to seroreact with *B. vinsonii*

(*berkhoffii*) antigens. *Bartonella vinsonii* (*berkhoffii*) is thought to be tick-borne. Serum of some infected dogs also seroreacts with *B. henselae* and *B. clarridgeiae* antigens; these *Bartonella* species are transmitted by fleas. *Bartonella* species that have been isolated from dogs or from which DNA has been amplified from blood or tissues include *B. vinsonii* (*berkhoffii*), *B. henselae*, *B. clarridgeiae*, *B. washoensis*, *B. quintana*, and *B. elizabethae*. Each of these organisms potentially can induce illness in dogs. Dogs infected with a *Bartonella* species are commonly coinfected with other agents like *Anaplasma* spp. or *Ehrlichia* spp. which may play a role in the pathogenesis of disease.

Clinical features. Clinical findings or syndromes most frequently attributed to *Bartonella* spp. infections of dogs include endocarditis, fever, arrhythmias, hepatitis, granulomatous lymphadenitis, cutaneous vasculitis, rhinitis, polyarthritis, meningoencephalitis, thrombocytopenia, eosinophilia, monocytosis, immune-mediated hemolytic anemia, epistaxis, and uveitis. *Bartonella vinsonii* (*berkhoffii*) and *B. henselae* seem to be the most likely species to be associated with clinical disease. In one study of valvular endocarditis, all dogs with *Bartonella* spp. associated disease were also seropositive for *A. phagocytophilum* (MacDonald and colleagues, 2004). Whether the coinfection potentiated the *Bartonella* associated disease is unknown. Both *B. vinsonii* and *B. henselae* have been associated with endocarditis in dogs in Colorado and Wyoming (Fenimore et al, 2011) suggesting transmission from contact with fleas infesting coyotes and possibly fox.

Diagnosis. Serum antibodies can be detected in both healthy and clinically ill dogs, and so the presence of antibodies does not always correlate to illness. Some *Bartonella* species, in particular *Bartonella vinsonii* (*berkhoffii*), can be difficult to culture and so amplification of DNA by PCR assay with or without culture is often needed to confirm infection (Duncan et al, 2007). If positive test results are detected in a clinically ill dog and there is no other obvious explanation for the illness, treatment is indicated.

Treatment. As many cases of bartonellosis in dogs have been apparently resistant to administration of doxycycline, some clinicians believe that azithromycin is the treatment of choice. Fluoroquinolones, alone or in combination with amoxicillin, were apparently effective for the treatment of some dogs with suspected clinical bartonellosis. Rifampin may be required for resistant cases. No matter which drug is used, a minimum of 4-6 weeks of treatment is usually needed.

Zoonotic aspects and prevention. *Bartonella vinsonii* (*berkhoffii*) and *B. henselae* have been detected in both dogs and humans and cat scratch disease has been documented in a humans after exposure to dogs and by blood contaminated needles. Care should be taken to avoid bites or scratches while handling or treating infected dogs. Flea control is known lessen transmission of *B. henselae* amongst cats (Bradbury and Lappin, 2010). Flea and tick control is likely to lessen transmission of *Bartonella* species between dogs and perhaps from dogs to people.

REFERENCES

1. *Additional references available on request to mlappin@colostate.edu*

5003

MANAGEMENT OF ACUTE RESPIRATORY DISEASE IN CATS

INTERNAL MEDICINE

Michael Lappin, DVM, PhD, DACVIM

Abstract. There are many causes of bacterial, viral, and fungal causes of upper respiratory infections (URI) in cats. The primary purpose of this presentation is to update attendees on management of cats with acute disease that is likely induced by bacterial or viral causes.

KEY POINTS

1. The most common primary bacterial infections are due to *Bordetella bronchiseptica*, *Mycoplasma* spp., and *Chlamydia felis*.
2. The most common viral infections are feline herpesvirus 1 and feline calicivirus.
3. Amoxicillin or doxycycline are the best antibiotics to try for acute bacterial infections.
4. It can be difficult to interpret results of PCR assays on discharges in cats with upper respiratory infectious because of vaccines and subclinical carriers.
5. Lessening stress can lessen recurrent upper respiratory tract infections in cats.
6. In acute upper respiratory infections in cats, treatment may not be needed.
7. If signs of infection last more than 10 days, a complete diagnostic workup should be completed.
8. Making the home less stressful can lessen recurrent signs of upper respiratory disease due to viral causes.

Key words. Feline, *Bordetella*, *Mycoplasma*, herpesvirus, calicivirus, PCR, famciclovir

Please see the ISCAID respiratory treatment guidelines for further information on this very important topic.

Lappin MR, Blondeau J, Boothe D, Breitschwerdt EB, Guardabassi L, Lloyd DH, Papich MG, Rankin SC, Sykes JE, Turnidge J, Weese JS. Antimicrobial use Guidelines for Treatment of Respiratory Tract Disease in Dogs and Cats: Antimicrobial Guidelines Working Group of the International Society for Companion Animal Infectious Diseases. J Vet Intern Med. 2017;31:279-294.

Bacterial causes. Almost all cats with chronic mucopurulent or purulent nasal discharge have a bacterial component to their disease. Diagnosis and treatment was reviewed by the International Society for Companion Animal Infectious Diseases. Primary bacterial disease is rare but may be associated with *Bordetella bronchiseptica*, *Mycoplasma* spp., *Chlamydia felis*, and some *Pasteurella* spp.. Recently it was shown that *Bartonella* spp. are not a causes of chronic rhinitis in cats.

Most cases of chronic or recurrent bacterial rhinitis are secondary to other diseases including trauma, neoplasia, inflammation induced by viral infection, foreign bodies, inflammatory polyps, and tooth root abscess. Thus, if routine antibiotic therapy fails with doxycycline or amoxicillin, a diagnostic workup should be performed (Lappin et al, 2017). If the diagnostic workup fails to find a primary disease and neutrophilic or mixed inflammation is noted, other antibiotics could be considered. Pradofloxacin has been evaluated as a treatment of feline rhinitis and conjunctivitis in several studies and can be considered as a rescue drug for cats with suspected bacterial disease. This fluoroquinolone is known to be safe for the use in cats.

In a placebo-controlled, double-blind clinical trial, 39 cats with signs of bacterial upper respiratory infections or conjunctivitis were entered. The cats were randomly entered into 1 of 2 treatment groups: treated orally with either 5 mg/kg pradofloxacin q24hr or 5 mg/kg doxycycline q12hr for 42 consecutive days. Changes in health status and clinical scores were evaluated. The presence of *C. felis* and *Mycoplasma* spp. DNA was determined by quantitative polymerase chain reaction (PCR) and nested PCR of conjunctival swabs, respectively. Prior to treatment, DNA of *C. felis* and *Mycoplasma* spp. was amplified from samples from 23 and 20 cats, respectively. Clinical signs improved markedly within the first week for cats of both groups. Complete elimination of *Mycoplasma* spp. DNA was achieved in both groups. During treatment with either drug, *C. felis* DNA copy number declined quickly, all cats administered doxycycline became *C. felis* DNA negative and 4 cats treated with pradofloxacin remained *C. felis* DNA positive. In this study, it was concluded that both pradofloxacin and doxycycline have good efficacy against *C. felis* and *Mycoplasma* spp., resulting in a marked improvement of clinical signs. The study showed evidence that the pradofloxacin protocol studied may eliminate *Mycoplasma* spp. infections. However, since *C. felis* DNA was still amplified from samples from some cats after treatment with pradofloxacin, infection might not always be eliminated using this protocol.

Since bacterial rhinitis leads to chondritis and osteomyelitis, antibiotic therapy may need to be continued for weeks in cats with chronic disease. Drugs with an anaerobic spectrum that also penetrate bone and cartilage well are often effective. Clindamycin or amoxicillin-clavulanate are frequently used. Amoxicillin-clavulanate has the advantage of killing most *B. bronchiseptica* isolates. Clindamycin has the advantage of effective against *Mycoplasma* spp. and is effective against many anaerobes. After being administered twice daily on the first day, azithromycin can be administered every third day. Cefovecin can be used in cats that are difficult to treat orally, but since it is a beta-lactam, there is no effect against *Mycoplasma* spp.. Topical administration of antibiotics by drops or nebulization may be beneficial for some cats but controlled studies are generally lacking. Lessening stress and immune stimulants as discussed for viral disease may be of benefit.

Viral diseases. Herpesvirus 1 (rhinotracheitis; FHV-1) and calicivirus (FCV) are the most common viral causes of sneezing and nasal discharge in the cat. Other viruses may be involved in some cases including the current H3N1 avian influenza, SARS-CoV-2 (from the owners), and H3N2 (from the dog). If oral ulcers are present, calicivirus is most likely. If corneal ulcers are present, herpesvirus 1 is most likely. FHV-1 has now also been associated with chronic stomatitis, facial dermatitis, and endogenous uveitis. Viral rhinitis with or without secondary bacterial infection can be recurrent. FHV-1 can be documented by direct fluorescent staining of conjunctival scrapings, virus isolation, or polymerase chain reaction. Since FHV-1 DNA can be amplified in conjunctival cells of approximately 25% of healthy cats, the positive predictive value of these tests in diseased cats is low. Quantitative PCR may ultimately prove to correlate to the presence or absence of disease but some cats with chronic FHV-1 infections do not have high values. Currently used PCR assays also detect vaccine strains of FHV-1. RT-PCR assays can be used to amplify the RNA of FCV. However, these assays have the same problems with predictive value as those to detect DNA of FHV-1. In one study, the PCR assay result for FHV-1 or Mycoplasma spp. did not predict the treatment response.

Zirofsky D, Rekers W, Powell C, Hawley J, Veir J, Lappin M. Feline Herpesvirus 1 and Mycoplasma spp. Conventional PCR Assay Results From Conjunctival Samples From Cats in Shelters With Suspected Acute Ocular Infections. Top Companion Anim Med. 2018;33:45-48.

Feline viral rhinitis with or without secondary bacterial infection can be recurrent. There are no consistently effective primary therapies. For FHV-1, lysine at 250-500 mg, PO, once or twice may be helpful in some cats lessening recurrent disease and has been shown to be safe but should be given as a dose, not fed with food and is not a treatment for active disease. Lysine has been shown to be ineffective for prevention of upper respiratory tract infections in shelter studies and so should not be used for this purpose.

Administration of human alpha 2b interferon at 50 U, PO, daily may help some cats with suspected chronic calicivirus or FHV-1 infection. However, may be difficult to prescribe now in the USA. In Europe, feline interferon may been beneficial in the management of some cats. Intranasal administration of modified live, intranasal FHV-1

and FCV vaccines may lessen disease in some chronically infected cats. If there is a positive response to intranasal vaccination in a cat with chronic disease, I will use this form of immunotherapy up to 3 times per year.

Fenimore A, Carter K, Fankhauser J, Hawley JR, Lappin MR. Evaluation of intranasal vaccine administration and high-dose interferon- α2b therapy for treatment of chronic upper respiratory tract infections in shelter cats. J Feline Med Surg. 2016;18:603-611.

Famciclovir is currently the orally administered drug of choice for management of acute (and possibly chronic) FHV-1 infections in cats. The drug has been prescribed mostly at 40 or 90 mg/kg and is safe at up to 90 mg/kg, PO, q8hrs and so the dose should be increased if lower doses were used and the initial response is suboptimal and FHV-1 is still suspected. Administration of one dose of famciclovir (125 or 500 mg) on admission to an animal shelter was ineffective in lessening clinical signs of disease.

Topical cidofovir (product for humans) can be used for the treatment of FHV-1 conjunctivitis twice daily and was effective in a controlled research project. The drug is easier to administer (twice daily) than idoxuridine or other anti-FHV-1 ocular therapies and does not cause as much irritation. This drug is available in some compounding pharmacies (www.rxfixer.com). In a recent research study, raltegravir was effective for the management of FHV-1 associated clinical signs in a model.

Immune modulation with a the probiotic *Enterococcus faecium* strain SF68 (FortiFlora®, Purina Pet Care) was effective in lessening stress reactivated FHV-1 signs in a model. Field studies with this probiotic are in progress.

Lappin MR, Veir JK, Satyaraj E, Czarnecki-Maulden G. Pilot study to evaluate the effect of oral supplementation of *Enterococcus faecium* SF68 on cats with latent feline herpesvirus 1. J Feline Med Surg. 2009;11:650-654.

Recently, the use of an intranasal product containing 2 Toll-like receptor agonists was beneficial in lessening signs and shedding of FHV-1 in a model. The compound is also effective for treating ocular FHV-1. Field studies with this compound are in progress.

Lappin M, Wotman K, Chow L, Williams M, Hawley J, Dow S. Nanoparticle ocular immunotherapy for herpesvirus surface eye infections evaluated in cat infection model. PLoS One. 2023;18:e0279462. doi: 10.1371/journal.pone.0279462. PMID: 36607992; PMCID: PMC9821494.

Stress relief. Many of the cats with chronic recurrent signs of upper respiratory disease are likely to be infected by FHV-1 or FCV. Stress reactivation of feline viral infections is thought to be common, in particular for FHV-1. All the principles of stress relief for management of feline interstitial cystitis also apply to cats with recurrent signs of URI. In a recent study, use of a facial pheromone diffusor could lessen recurrent signs of FHV-1 in a mild stress model in experimentally inoculated cats. Use of a stress relieving probiotic (Calming Care for Cats; Purina) was also effective.

Fungal diseases. *Cryptococcus neoformans*, *C. gattii*, and *Aspergillus* spp. are the most common causes of fungal infection in cats. Aspergillosis in cats carries a grave prognosis. *Sporothrix* spp. are also occasionally found in cats with chronic URI and are zoonotic.

Cryptococcosis is the most common systemic fungal infection of cats and should be considered a differential diagnosis for cats with respiratory tract disease, subcutaneous nodules, lymphadenopathy, intraocular inflammation, fever, and CNS disease. Infected cats range from 6 months to 16 years of age, and male cats are over represented in most studies. Infection of the nasal cavity is reported most frequently (56.3 to 83.0% of cases) and commonly results in sneezing and nasal discharge. The nasal discharge can be unilateral or bilateral, ranges from serous to mucopurulent, and often contains blood. Granulomatous lesions extruding from the external nares, facial deformity over the bridge of the nose, and ulcerative lesions on the nasal planum are common. Submandibular lymphadenopathy is detected in most cats with rhinitis. Definitive diagnosis of cryptococcosis is based on antigen testing or cytologic, histopathologic, or culture demonstration of the organism. Cats with cryptococcosis have been treated with amphotericin B, ketoconazole, itraconazole, fluconazole, and 5-flucytosine alone and in varying combinations. Good to excellent treatment responses in cats were seen with fluconazole (96.6%), itraconazole (57.1%), and ketoconazole (34.6%). Because of toxicity, I no longer use ketoconazole. I generally use fluconazole at 50 mg/cat per day because it has the least side-effects and or the azoles, has the best penetration across the blood-brain and blood-ocular barriers. If life-

threatening infection is occurring or the cat is failing to respond to the azole, drugs liposomal amphotericin B should be used. Care should be taken if voriconazole is used as it has been associated with neurotoxicity in cats. Nasal and cutaneous cryptococcosis generally resolve with treatment; CNS and ocular disease are less likely to respond to treatment. Treatment should be continued for at least 1 to 2 months past resolution of clinical disease. People and animals can have the same environmental exposure to *Cryptococcus spp.* but zoonotic transfer from contact with infected animals is unlikely.

Parasitic diseases. While nasal mites (*Pneumonyssoides*) and a nasal worm (*Eucoleus*) occur in dogs in the United States, there are no significant nasal parasites in cats of the USA.

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6001

THE GAIT EXAM: UNDERUTILIZED BUT CRITICALLY IMPORTANT

NEUROLOGY

Christopher L. Mariani, DVM, PhD, DACVIM

Gait examination is arguably the most important part of the neurologic examination but is often not performed by veterinary practitioners. This may be due to limited appointment times, restricted exam room space or a variety of other factors. However, with observation of an animal walking (or attempting to walk), an astute practitioner can frequently localize a neurologic lesion (or get very close) based on this information alone.

PERFORMANCE OF THE GAIT EXAMINATION

The animal can be initially observed within the exam room as historical information is gathered from the client. In fact, for cats and some dogs, this constitutes the entire exam. Choosing a room without areas to escape or hide is particularly important for cats. For most dogs, it is important to have the owner or an assistant walk them on a lead for more detailed observation.

The patient should be evaluated while walking towards and away from the examiner (best way to identify ataxia) and should also be observed from the side (best way to assess paresis and stride length). If the animal is unable to stand or bear weight, adequate support of the limbs in question should be provided while assessing the ability of the animal to voluntarily advance its limbs, bear weight, and move in a coordinated manner.

Some abnormalities may be accentuated by having the animal climb or descend stairs or a hill if available, although the author does not employ this routinely. Occasionally it is useful to have cats jump on or off an elevated service such as a chair or stool, which can also accentuate subtle abnormalities. Finally, it is useful to invest some time in watching many clinically normal dogs & cats ambulate, which helps one to appreciate subtle abnormal gaits.

ABNORMALITIES

Several abnormalities may be detected with the gait examination. These include:

Ataxia: incoordination characterized by a failure to walk or move the limbs in a straight line, crossing of the limbs over the body midline, and possibly stumbling and falling. Ataxia always indicates neurologic dysfunction and may be caused by involvement of several areas of the nervous system.

Sensory (proprioceptive) ataxia: Lesions of the peripheral sensory nerve, spinal cord or brainstem commonly cause incoordination. With spinal cord and brainstem lesions, ataxia is typically accompanied by paresis (see below). Peripheral sensory nerve lesions are rare in veterinary patients.

Cerebellar ataxia: Cerebellar lesions can cause a profound ataxia characterized by dysmetria (hypermetria and hypometria), and intention tremors. Animals with pure cerebellar lesions maintain good strength without obvious paresis.

Vestibular ataxia: Characteristic incoordination typified by leaning, drifting, stumbling, falling, and occasionally rolling to one side. Usually accompanied by a head tilt, nystagmus, and possibly positional ventral strabismus. Bilateral involvement of the vestibular system can lead to ataxia and bizarre, wide head excursions but without an obvious head tilt or ataxia. Loss of a normal physiologic nystagmus can often be appreciated in these cases.

Paresis: muscular weakness or incomplete voluntary movement. On the gait exam, this is characterized by scuffing of the nails, dragging of one or more limbs, a

short-strided gait, or rapid tiring with activity/exercise. Paresis denotes dysfunction of the nervous (motor) or muscular systems.

Lameness: Inability or reluctance to bear weight on one or more limbs. Lameness often indicates a lesion in the long bones, joints, tendons, or musculature (i.e., orthopedic diseases), although entrapment or compression of a nerve or nerve root can also lead to lameness (known as a "root signature").

Short-strided Gait: This gait may occur secondary to paresis as described above. A short-strided gait in all four limbs (particularly in the absence of ataxia) is suggestive of a neuromuscular condition (i.e., disease affecting the peripheral nerves, muscles or neuromuscular junctions). However, such gaits may also occur from pain secondary to orthopaedic conditions affecting multiple limbs (e.g., polyarthritis).

Disconnected Gait: Also known as a "two-engine" gait, this describes an animal with different stride lengths between the thoracic and pelvic limbs. Most commonly, the thoracic limbs have the shorter stride although the opposite can also be seen.

Compulsive Pacing and Circling: This is typical of a forebrain lesion and such animals typically also show alterations of mentation and consciousness.

Dysmetria: Dysmetria refers to both hypermetria and hypometria and is typically seen with cerebellar ataxia as mentioned above. In rare cases, dysmetria may be seen in one or more limbs without ataxia.

Postural Abnormalities: These may be observed during ambulation or when the animal comes to a stop and include keeping the head and neck low or ventroflexed, kyphosis, standing with a plantigrade or palmigrade stance, maintaining a wide-based stance and holding the pelvic limbs in a rostral position (i.e, flexed at the hip).

EXAMPLES OF GAIT ABNORMALITIES AND THEIR CAUSES

Compulsive pacing and/or circling: forebrain lesion

Ataxia in all 4, paresis in all 4 limbs: cervical spinal cord or brainstem lesion. Best differentiated by cranial nerve examination or identifying a vestibular quality to the ataxia

Ataxia in all 4 limbs with a tendency to drift, stumble fall or roll to one side: vestibular lesion

Ataxia (usually profound) in all 4 limbs without paresis: cerebellar lesion (usually accompanied by intention tremors & hypermetria)

Disconnected (two-engine) gait characterized by short-strided thoracic limbs and ataxia with paresis in the pelvic limbs: C6-T2 myelopathy (particularly cervical spondylomyelopathy or "Wobbler's syndrome")

Ataxia and paresis in the pelvic limbs: T3-L3 myelopathy; L4-S2 myelopathy is also possible but less likely

Paresis in the pelvic limbs characterized by a short-strided gait: L4-S2 myelopathy or neuromuscular lesion

Paresis in all 4 limbs characterized by a short-strided gait; often worsens with activity or exertion; notable lack of ataxia: neuromuscular lesion; polyarthritis also possible

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6002

IS IT ORTHOPAEDIC OR NEUROLOGIC? SORTING OUT LAMENESS, PARESIS AND DOGS THAT WON'T GET UP

NEUROLOGY

Christopher L. Mariani, DVM, PhD, DACVIM

Lameness, difficulty walking, and reluctance or inability to rise are common presentations for patients presented to small animal practitioners. Disorders of the central and peripheral nervous systems, spine, long bones, joints, tendons, or musculature can all result in these clinical signs. Identifying the organ system and anatomic structures responsible are critical to recommending subsequent diagnostic testing, therapy, and possibly referral to the appropriate specialist. The most critical steps in this evaluation are careful observation of the animal's gait, and thorough neurologic and orthopedic examinations.

CLINICAL EVALUATION

Gait Examination

See accompanying proceedings for a detailed description of the gait exam.

Orthopedic Examination

Orthopedic examination should include careful examination of the affected and normal limbs, evaluating the long bones, joints, tendons and musculature. Joints are palpated for evidence of effusion and pain, and flexed and extended for evidence of instability, excessive or restricted motion, and pain. Long bones and tendons are palpated for evidence of swelling or pain. Muscles are palpated to assess tone, atrophy or hypertrophy, and for evidence of pain.

Neurologic Examination

A thorough neurologic examination is an important part of the evaluation of animals with gait dysfunction. Particular attention should be paid to gait examination (see above), assessment of postural reactions, segmental spinal reflexes, and muscular and spinal palpation.

Postural reactions: These include tests such as proprioceptive placing ("conscious proprioception"), hopping, hemistanding, hemiwalking, wheelbarrowing, and the extensor postural thrust. A convincing postural reaction deficit definitively indicates a lesion within the nervous system. However, the sensory nerves, spinal cord, brainstem, thalamus, cerebral cortex, and descending motor systems are all involved in these responses, and more information is needed to localize the lesion further.

Segmental spinal reflexes: These include limb reflexes as well as the perineal reflex, and require sensory neurons, associated spinal cord segments, motor neurons, neuromuscular junction, and muscles to function correctly. The patellar and withdrawal reflexes are the most reliable and should probably be performed in every animal with a gait abnormality. Convincing reflex deficits indicate dysfunction of the peripheral nerves, associated spinal cord segments, or neuromuscular junction.

Muscular Palpation: Animals that are not bearing full weight or using a limb may develop disuse atrophy of the muscles of this limb. However, neurogenic atrophy is typically more severe and happens more quickly than

disuse atrophy. Animals with lower motor neuron lesions (motor nerves, neuromuscular junction, or associated spinal cord segments [C6-T2 or L4-S3]) may have reduced muscle tone and neurogenic atrophy.

Spinal Palpation: Palpation of the cervical, thoracic and lumbar spine for evidence of pain or discomfort is an important part of the neurologic examination. Pain may be a sign of a lesion affecting the spine itself (e.g., diskospondylitis, vertebral neoplasia) or referred from nervous system structures (intervertebral disk disease causing nerve root compression, meningomyelitis, CNS neoplasia). Evaluation of the lumbosacral space should also include elevation of the tail and rectal palpation of the ventral surface of the disk space.

CLINICAL PRESENTATIONS

Acute Lameness

Acute lameness is most often associated with orthopedic disease. Evidence of joint or soft tissue swelling or effusion, long bone fracture, or joint instability on orthopedic examination increases the index of suspicion or confirms the diagnosis. However, a neurologic etiology is possible and is seen most often as a "root signature" secondary to nerve root compression by a herniated intervertebral disk. Neck or back pain, ataxia and paresis suggest nervous system involvement.

Orthopedic

- Ligament tear or rupture (e.g. cranial cruciate)
- Muscle strain
- Long bone fracture
- Panosteitis
- Hypertrophic osteodystrophy

Neurologic

- Intervertebral disk disease
- Lumbosacral stenosis

Chronic Lameness

Chronic lameness is also most often orthopedic in nature, and the etiology is confirmed by similar means. However, neurologic etiologies can certainly lead to similar gait dysfunction. A chronic lameness (particularly involving a thoracic limb) that is unresponsive to nonsteroidal anti-inflammatory drugs (NSAIDs) should raise concern for a peripheral nerve sheath tumor.

Orthopedic

- Osteoarthritis
- Ligament injury
- Bone neoplasia

Neurologic

- Peripheral nerve sheath tumor
- Intervertebral disk disease
- Lumbosacral disease

Stiff, Short-Strided Gait

A stiff, short-strided gait may occur in single or multiple limbs due to either pain or paresis. A number of etiologies can potentially cause this presentation, and careful orthopedic and neurologic examinations are required to sort these out.

Orthopedic

- Polyarthritis
- Panosteitis
- Hypertrophic osteodystrophy

Neurologic/Neuromuscular

- Myasthenia gravis (pelvic limbs or all four limbs)
- Polymyopathy
- Polyneuropathy
- Lumbosacral disease (pelvic limbs only)
- C6-T2 lesion (IVDD, cervical spondylomyelopathy; thoracic limbs only)

DIAGNOSTIC TESTING

Diagnostic tests chosen depend on the results of the gait, orthopedic, and neurologic examinations, index of suspicion of disease, severity of presentation, and financial constraints of the owner.

ORTHOPEDIC DISEASE

Radiographic examination of the appendicular skeleton is a useful and relatively low-cost diagnostic test to identify a number of disease processes. In some cases, advanced diagnostic imaging such as radionucleotide imaging (bone scan), computed tomography (CT), or magnetic resonance

imaging (MRI) may provide additional information, and are more sensitive than survey radiography. Collection of a sample of joint fluid is usually diagnostic for polyarthritis.

NEUROLOGIC OR NEUROMUSCULAR DISEASE

Survey radiographs of the spine are useful to detect diskospondylitis, intervertebral disk disease, and lytic vertebral tumors. CT and MRI are frequently used to identify spinal cord compression or inflammation secondary to intervertebral disk disease, meningomyelitis, CNS or PNS neoplasia, and vertebral tumors. Cerebrospinal fluid (CSF) analysis is used to document inflammation within the CNS. Electrodiagnostic testing (e.g., electromyography [EMG], nerve conduction velocity, repetitive nerve stimulation, spinal evoked potentials) is particularly useful in documenting evidence of peripheral nerve or neuromuscular disease, and occasionally myopathic diseases. Muscle biopsy with or without nerve biopsy is effective in confirming neuropathic or myopathic disease and in further defining potential etiologies.

THERAPEUTIC INTERVENTION

A discussion of treatment of all of the conditions outlined in this talk is outside the scope of these proceedings. However, a few relevant points of therapy can be highlighted. Directly addressing the underlying disease process is the ideal goal of any therapeutic strategy, and may involve surgical correction (e.g., cranial cruciate ligament repair, hemilaminectomy for intervertebral disk removal) or medical therapy (e.g., immunosuppression in animals with myasthenia gravis or polyarthritis). Where correction of the underlying condition is not possible, palliation of the signs associated with the disease (often pain) is warranted.

Opiods and NSAIDS are often effective against the acute pain associated with trauma or operative interventions. NSAIDS are frequently used with chronic orthopedic conditions, and although they can provide some relief in animals with neuropathic pain, this drug class is often not the most effective in this scenario. Muscle relaxants (e.g., diazepam or methocarbamol) are useful in animals with intervertebral disk disease or neoplasia causing nerve root irritation, as much of the pain in these conditions is associated with muscle spasm. Gabapentin and pregabalin are some of the most effective drugs for neuropathic pain, and act through inhibition of voltage-gated calcium channels. Finally, the NMDA receptor antagonists ketamine and amantadine can be useful in acute and chronic scenarios respectively for the control of the "windup" phenomenon, which can lead to sustained pain or aberrant responses to typically nonpainful stimuli (allodynia). A multimodal therapeutic strategy using several drugs and other interventions often works best for animals with significant pain as part of their clinical presentation.

6003

ETIOLOGIES AND DEVELOPING A DIAGNOSTIC PLAN FOR SEIZURES

NEUROLOGY

Christopher L. Mariani, DVM, PhD, DACVIM

DEFINITIONS

Seizures are an important clinical problem in both dogs and cats and account for a substantial proportion of admissions to both general and referral veterinary hospitals. Although most veterinarians are very familiar with seizures, it helps to define this term explicitly as follows:

Seizure: the clinical manifestation of excessive and/or hypersynchronous neuronal discharge within the brain; may be manifested as episodic impairment or loss of consciousness, abnormal motor phenomena, psychic or sensory disturbances, or autonomic signs such as salivation, vomiting, urination or defecation. Synonyms – convulsion, ictus, fit.

Seizures have been classified in the past according to the areas of the brain that they affect. Thus, they have been termed generalized or focal (partial) seizures, defined as follows:

Generalized: abnormal electrical discharges affecting the cerebral hemispheres bilaterally and typically causing symmetric signs. Several manifestations are possible, including generalized tonic-clonic ("Grand mal"), or exclusively tonic, clonic or atonic activity.

Focal (Partial): abnormal electrical discharges of neurons in a focal area of the brain; may result in asymmetric motor or sensory signs such as twitching of the eyelids, lips, ears or one limb. Focal seizures may secondarily generalize to involve both hemispheres; that is, the electrical activity spreads across the cerebral cortex. This often gives rise to focal motor activity, which then evolves; this can often be recognized by owners.

Epilepsy: a chronic neurologic condition characterized by recurrent seizures, with an intracranial origin.

SEIZURES VERSUS SEIZURE IMPOSTORS

A number of other clinical conditions can mimic seizures, including syncope, acute vestibular dysfunction, tremors, narcolepsy/cataplexy, rapid eye movement (REM) behavior disorder, movement disorders (e.g., distemper myoclonus), and behavioral disorders. The majority of patients presenting for seizures are normal in the veterinary hospital, and the clinician's first task is to decide whether the events in question are true seizures, or seizure "mimics". Some guidelines for this determination follow:

- Seizures frequently involve alterations in consciousness and autonomic signs
- Most seizures occur when animals are at rest or sleeping; syncope and narcolepsy/cataplexy occur predominantly with exercise or excitement
- Animals with syncope and narcolepsy/cataplexy have brief episodes with loss of consciousness but are usually normal on recovery. Animals with seizures and loss of consciousness usually have post-ictal alterations of mentation
- REM behavior disorder occurs during sleep, but the animal is normal when awakened/roused
- Vestibular dysfunction usually has characteristic signs including head tilt and nystagmus
- Animals with tremors, movement disorders, and behavioral disorders will have a normal mentation and level of consciousness

DIFFERENTIAL DIAGNOSIS FOR SEIZURES

Once the clinician is reasonably certain that the events are true seizures, differential diagnoses may be considered. These can be broadly categorized into extracranial

and intracranial etiologies. A seizure disorder indicates dysfunction of the forebrain (cerebrum or diencephalon). Extracranial causes of seizures originate from outside the central nervous system, causing dysfunction in an otherwise normal brain, and include metabolic, nutritional and toxic causes. Intracranial etiologies cause structural alterations of the forebrain itself, and include degenerative, anomalous, neoplastic, inflammatory, infectious, idiopathic, and traumatic causes.

EXTRACRANIAL CAUSES OF SEIZURES

Hypoglycemia: main causes are juvenile hypoglycemia, hyperinsulinemia (secondary to insulin overdose or an insulin-secreting tumor), and sepsis.

Hypocalcemia: causes include eclampsia, hypoparathyroidism and ethylene glycol intoxication.

Hyponatremia: results from loss of sodium rich fluids (e.g. vomiting, diarrhea, diuretics), hypoadrenocorticism or increased water intake (psychogenic polydipsia).

Hypernatremia: results from the loss of free water or sodium poor fluids (diabetes insipidus, excessive panting, high temperatures), decreased water intake (primary adipsia or limited access) or rarely through ingestion of high levels of salt.

Hepatic encephalopathy: results from altered filtration of gastrointestinal portal blood by the liver. Main causes are the presence of an anomalous portosystemic shunting vessel or severe parenchymal hepatic disease with secondary shunting.

Hypertriglyceridemia: primarily a disease of miniature Schnauzers, which have a congenital enzyme deficiency, allowing triglyceride accumulation in the blood.

Nutritional disease: rare cause of seizures; historically thiamine deficiency has been implicated.

Intoxication: a wide variety of toxins may lead to nervous system dysfunction and seizures. Examples include ethylene glycol, lead, heavy metals, metaldehyde, strychnine, and organophosphates.

INTRACRANIAL CAUSES OF SEIZURES

Degenerative disease: very rare causes of seizures; include lysosomal storage disorders and neuronal abiotrophies

Anomalous conditions: hydrocephalus is the most common and occurs predominantly in toy and brachycephalic breeds. Less common conditions include epidermoid, dermoid, and arachnoid cysts, and lissencephaly.

Neoplasia: common cause of seizures in older animals. Brain tumors may be primary (arise from brain itself) or secondary (metastatic or arise from adjacent structures [e.g., skull]). It is common to see seizures as the sole clinical sign of a brain tumor.

Inflammatory disease: common cause of seizures in animals of any age. Encephalitis may be infectious (viral, fungal, protozoal, bacterial, rickettsial, or parasitic) or more frequently, non-infectious. Common non-infectious causes in dogs include granulomatous meningoencephalitis (GME) and necrotizing meningoencephalitis.

Idiopathic disease: common cause of seizures – see below.

Traumatic disease: seizures can occur immediately at the time of head trauma or as a late sequela after recovery.

Idiopathic (Genetic) Epilepsy: Idiopathic epilepsy denotes "a syndrome unto itself"; that is, a well-recognized clinical condition of recurrent seizures without other neurologic signs and without any identifiable underlying cause. It is considered to be an inherited or familial condition, although the details of the genetic transmission are unknown in veterinary medicine except for a few families of dogs. Although somewhat controversial, inherited epilepsy appears to be very rare in cats.

Seizures have generally been considered to be of the generalized tonic-clonic variety, although this is not always be the case. The seizures almost always begin between 1 and 5 years of age, although a few dogs may have an onset between 6 and 12 months or up to 7 years of age. One of the hallmarks of idiopathic epilepsy is its insidious onset; seizures initially occur weeks to months apart and gradually become more frequent. Many dogs progress to develop cluster seizures or status epilepticus. In rare cases, this may be the first known seizure activity. Although certain breeds of dogs are predisposed to the development of genetic epilepsy, this condition can probably occur in any dog.

In the absence of a genetic test, diagnosis is based upon an appropriate signalment and description of seizure onset and character, and by ruling out other potential etiologic diagnoses with appropriate tests (typically cerebrospinal fluid [CSF] evaluation and brain imaging). Outside of the

ictal and immediate post-ictal periods, the neurologic examination should be normal.

Unknown Epilepsy: This condition is synonymous with acquired, cryptogenic, and probably symptomatic epilepsy. It implies that an acquired structural lesion within the brain is suspected to be causing the seizures but cannot be detected with the available diagnostic tests (e.g., patients with a previous intracranial disease such as head trauma or meningoencephalitis that have since recovered but are left with recurrent seizures because of assumed scarring or fibrosis within the brain). Seizures may be focal, focal with secondary generalization, or generalized, and may occur at any age. All breeds of dog and cat can be affected. Although occasional focal neurologic deficits (e.g., reduced conscious proprioception on one side) are noted, the majority of these patients have a normal neurologic examination. The diagnosis is based on signalment, neurologic examination, and by ruling out other causes of seizures with appropriate diagnostic testing. Unless concerned about the potential for breeding a patient and passing on the trait, it is not that critical to differentiate unknown epilepsy from genetic/idiopathic epilepsy, as the treatment for both is identical.

SIGNALMENT AND COMMONLY ASSOCIATED ETIOLOGIES

Less than 1 year of age: Head trauma, intoxication, hypoglycemia, meningoencephalitis, hepatic encephalopathy, hydrocephalus, lysosomal storage disease, other congenital disorders

From 1 to 5 years of age: Genetic epilepsy, unknown epilepsy, head trauma, intoxication, meningoencephalitis, hydrocephalus, hepatic encephalopathy, cerebrovascular disease, cerebral neoplasia

Greater than 5 years of age: Cerebral neoplasia, unknown epilepsy, meningoencephalitis, cerebrovascular disease, hypoglycemia

DIAGNOSTIC TESTING AND PLAN

Diagnostic testing for seizures can be divided into three steps:

Step 1: CBC, serum biochemistry, urinalysis

Step 2: Thoracic and abdominal imaging, serum bile acids, toxin assays (e.g., lead, ethylene glycol, cholinesterase), infectious disease titers, skull radiographs

Step 3: CSF evaluation, computed tomography (CT) or magnetic resonance imaging (MRI), electroencephalography (EEG)

Step 1 should probably be performed on any patient with seizures. Step 2 consists of tests that are readily available to most veterinary practitioners and may be chosen in certain cases depending on the index of suspicion for a certain disease. Step 3 consists of specialized tests that specifically evaluate the nervous system and are usually only available at specialty referral hospitals. The signalment, history and physical and neurologic examinations are critical when deciding which diagnostic tests are appropriate.

Important History Questions to Ask: Character, onset, frequency and progression of seizures; evidence of asymmetry; evidence of prodrome or post-ictal period, neurologic status between seizures, vaccination status and travel history, previous illness and medications

Important Physical Examination Findings: Evidence of cardiovascular disease, evidence of hepatic disease, retinal (fundic) examination

Important Neurologic Examination Findings: Altered mentation, focal cranial nerve deficits, circling or turning in one direction, focal postural reaction or conscious proprioceptive deficits, visual deficits, cervical hyperesthesia

When to choose Step 3?: An important decision point is when to refer a patient with seizures to a specialty hospital to receive the tests listed above in Step 3. In the author's opinion, this may be considered in any animal with seizures where extracranial etiologies have been ruled out, if the owners are so inclined. However, other patient circumstances may increase the suspicion of an active intracranial process, and thus the urgency for referral. These include focal seizure activity, focal neurologic deficits, dogs less than 1 year or greater than 5 years of age, and all cats.

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6004

MANAGEMENT OF ROUTINE AND DIFFICULT-TO-CONTROL SMALL ANIMAL EPILEPTICS

NEUROLOGY

Christopher L. Mariani, DVM, PhD, DACVIM

INTRODUCTION

Deciding on a treatment plan for an animal with seizures depends on a number of factors, including the suspected etiologic cause of the seizures, the frequency and severity of the observed seizures, and the financial constraints or intentions of the owner.

ADDRESS THE UNDERLYING CAUSE

If an underlying cause of the seizures is known or suspected, it should be appropriately addressed, if possible. Thus, animals with hypoglycemia or electrolyte abnormalities may require no therapy other than correction of these deficiencies (or excesses). Likewise, patients with hepatic encephalopathy, hypertriglyceridemia or various intoxications may not require long-term anticonvulsant therapy if the primary disease is appropriately addressed, although they may benefit from shorter-term treatment with these drugs. In some cases, damage to the brain may lead to acquired (probably symptomatic) epilepsy, requiring long-term treatment.

Animals with intracranial diseases also benefit from addressing the underlying condition, although these patients are more likely to require maintenance anticonvulsant therapy. Thus, placement of a ventriculoperitoneal shunt for hydrocephalus, anti-inflammatory and/or antimicrobial medications for meningoencephalitis, surgery or radiation therapy for brain tumors, and other specific therapies address the underlying disease process and may reduce or eliminate the need for anticonvulsant therapy.

MAINTENANCE ANTICONVULSANT THERAPY

Maintenance anticonvulsant therapy is used as an adjunct in symptomatic epilepsy and is the cornerstone of therapy for patients with idiopathic or probably symptomatic (acquired, cryptogenic) epilepsy. The first question to address is: **When to start anticonvulsant therapy?** There are no hard and fast rules on this issue, and each patient much be approached individually. However, some guidelines apply. In general, maintenance therapy should be considered if:

- Seizures are more frequent than once every 6-8 weeks
- Seizures are obviously increasing in frequency
- Status epilepticus or cluster seizures occur
- Seizures last longer than 5 minutes
- Seizures are very severe or involve aggression towards the owner

The second question to address is: **Which anticonvulsant should I choose?** Historically in dogs, the two main initial options for therapy have been phenobarbital and (potassium) bromide. These medications are chosen because of their long history of use, apparent efficacy, ease of dosing, favorable pharmacokinetics and inexpensiveness. There is limited evidence to suggest that phenobarbital may be slightly more efficacious as a first line agent in the dog. Diazepam is not effective as a maintenance anticonvulsant in the dog due to a very short elimination half-life, and the development of tolerance within several weeks.[1] Some of the newer anticonvulsant medications can be effective

as initial therapy, and the author uses zonisamide and levetiracetam frequently as first-line agents in dogs. Zonisamide is particularly attractive in this setting due to its low incidence of side effects and its relatively long half-life, allowing twice daily administration.[2,3] A generic extended release formulation of levetiracetam is now available, and pharmacokinetic studies suggest that twice daily administration may be effective in canine patients. However, published reports of efficacy in this setting are lacking in veterinary patients. Use of these newer drugs has been limited in the past by their expense when compared with traditional anticonvulsants, although generic versions of most of these newer generation drugs are now available at reduced costs. In the cat, phenobarbital (preferred) and diazepam are the historical maintenance drugs of choice. Bromide is an effective anticonvulsant in the cat, but is associated with a very high incidence of inflammatory lung disease, and is not recommended.[4,5] Diazepam should be used with extreme caution in cats and is generally not recommended, as it has been associated with idiosyncratic hepatic necrosis after oral administration.[6] Levetiracetam may be a reasonable choice in the cat if phenobarbital is not an option, although the drug must be administered three times daily.[7] A recent pharmacokinetic study suggests that extended release levetiracetam may be safe to use once daily in cats.[8] There is limited information available on zonisamide in cats,[9] although side effects seem to occur more frequently in this species.[10]

Initial Maintenance Therapy for Epileptic Animals

- Phenobarbital – 2.5-3 mg/kg q 12 hours (Dogs or cats)
- Potassium bromide – 40-50 mg/kg/day q 24 hours or divided (q 12 hours) (Dogs)
- Zonisamide – 3-5 mg/kg q 12 hours (Dogs and cats)
- Levetiracetam – 20 mg/kg q 8 hours (Dogs and cats)
- Levetiracetam (extended release) – 30 mg/kg q 12 hours (Dogs); 500 mg total q 24 hours (Cats)
- Diazepam – 0.2-1.0 mg/kg q 12 hours (Cats, use with caution and not recommended)

Phenobarbital is available in generic tablets (15, 30, 60, 90, 100 mg) or suspension (3 and 4 mg/ml) formulations, as is diazepam (2, 5, 10 mg tabs; 1 and 5 mg/ml suspension). Zonisamide is available as 25, 50 and 100 mg capsules. Levetiracetam is available as 250, 500, 750 and 1000 mg tablets and a 100 mg/ml suspension. Extended release levetiracetam is available as 500 and 750 mg tablets; these should not be broken, as it interferes with the extended-release mechanism. Bromide is typically compounded from the chemical grade salt and complexed with potassium (KBr) or less frequently with sodium (NaBr). It should be noted that due to molecular weight differences between the cation, equal amounts of KBr and NaBr do not contain the same amounts of bromide, and therefore have different anticonvulsant potencies (250 mg KBr = 211 mg NaBr). KBr is available from a number of compounding pharmacies. Liquid formulations are preferred over capsules, as they facilitate dosage adjustments, and KBr is best administered with food to reduce gastrointestinal irritation. Dietary salt affects serum levels of bromide, and a constant salt level should be maintained in the diet.

MONITORING MAINTENANCE THERAPY

A complete blood count (CBC), serum biochemical evaluation and urinalysis should be performed before starting maintenance anticonvulsant therapy, both as part of the diagnostic evaluation (see previous talk) and as a baseline before starting therapy. In addition, the metabolism of these drugs varies between patients. Blood levels are essential to guide therapy for phenobarbital and bromide and may be indicated for some of the newer drugs, depending on the response to therapy. Steady state of a drug after regular oral dosing depends on its half-life in the body and varies between medications and species. Monitoring times and desired blood levels are shown below for dogs.

Table 1. Monitoring Anticonvulsant Therapy in Dogs

Medication	Time to Steady State	Desired Blood Levels
Phenobarbital	10-14 days	15-35 mg/ml (65-101 mmol/l)
Bromide	3-4 months	1000-3000 mg/ml (or 1-3 mg/ml or 100-300 mg/dl)
Diazepam	5-10 days	Monitoring not typically performed
Zonisamide	3-5 days	10-40 mg/ml
Levetiracetam	1-2 days	5-45 mg/ml

These desired blood levels are a guide only and must be interpreted in light of the resulting seizure frequency and clinical condition of the patient. When measuring therapeutic blood levels, as with any medications, a serum separator tube ("tiger top") should be avoided, as the separator device may bind the drug and artificially decrease the serum levels. After the establishment of acceptable therapeutic levels of the medication, it is generally recommended that blood levels along with a CBC, serum chemistry and urinalysis be monitored every 6-12 months or in the event of an acute change in seizure frequency or new onset of sedation, weakness or ataxia. Animals receiving phenobarbital may also benefit from pre-and post-prandial serum bile acid evaluation at these times to detect changes in hepatic function. Therapeutic monitoring is very important for phenobarbital and bromide but has been utilized less frequently for the newer generation of drugs. As these newer medications are quite safe, they have generally been used to effect, and until fairly recently, routine therapeutic monitoring for these drugs was not available.

POTENTIAL ADVERSE EFFECTS OF MAINTENANCE THERAPY

Adverse effects of phenobarbital include sedation, polyphagia, polyuria, polydipsia, weight gain, pelvic limb ataxia, weakness, and in rare cases hepatic failure and blood dyscrasias. Overt hepatic damage is an unusual sequela with this medication, particularly if the serum levels are maintained below 35 μg/ml. It should be noted that increases in liver enzyme levels are common with this medication, and this does not indicate hepatic failure. If this is a concern, serum bile acids should be evaluated. Side effects of bromide are similar for the most part, including sedation, polyphagia, polyuria, polydipsia, and weight gain but in rare cases also includes pancreatitis. Vomiting related to the salt content can be minimized by administration with food. Diazepam may lead to sedation and polyphagia, and rare idiosyncratic reactions causing acute hepatic necrosis have been described in the cat.[6] Zonisamide and levetiracetam may both cause sedation, while the former may also cause vomiting, diarrhea and inappetence. A reversible idiosyncratic hepatic failure has been reported in dogs that received zonisamide, but this appears to be a very rare sequela.

DEALING WITH THE REFRACTORY EPILEPTIC

Monotherapy with one of the medications above controls an estimated 60-80% of epileptic dogs and the majority of cats. However, a number of animals will have their condition remain unchanged or worsen in the face of this therapy. In this situation, a number of additional steps may be considered:

- Ensure owner is administering drug correctly
- Ask about dietary changes, other medications or herbal preparations, and topical anti-parasite medications that may interfere with seizure control

- Reconsider diagnosis, pursue additional diagnostic testing
- Ensure optimal blood levels of maintenance drug
- Increase dosing frequency (Phenobarbital - from q12 h to q 8 h) if seizures occur at times corresponding to "trough" blood levels (base on therapeutic monitoring)
- Ensure female dogs have been spayed
- Add a second anticonvulsant drug

Regarding these points, many animals require blood levels of phenobarbital above 25 μg/ml for seizure control, although levels exceeding 35 μg/ml should be avoided. Although unusual, some animals receiving phenobarbital metabolize the drug very rapidly, and may benefit from dosing every 8 hours. Having the owner maintain a seizure diary is useful to document these cases, as seizures may occur during the expected "trough" period of drug metabolism and peak and trough serum levels may be beneficial in guiding therapy. Serum levels of bromide above 3000 μg/ml are tolerated in some dogs, especially when used as a monotherapy.

ADDING A SECOND ANTICONVULSANT DRUG

In the cat, diazepam may be added successfully to phenobarbital to control seizures (although as mentioned above, this drug should be used with extreme caution in this species). In the dog, a combination of phenobarbital and KBr (starting dose 20-30 mg/kg daily) is effective in controlling the majority of patients refractory to monotherapy with either drug alone. However, side effects are common with this protocol and may be unacceptable to the owner. These include sedation, pelvic limb weakness and ataxia, polyphagia, polyuria and polydipsia. It should be noted that side effects may subside approximately 1-2 weeks after initiating the new drug, and so patience can pay off. Generally, a balance must be achieved between an acceptable seizure frequency and these side effects, although this may be impossible in some dogs. Generally, the best success is achieved by aiming for a serum bromide level between 2000-3000 mg/l and maintaining a lower phenobarbital level (e.g. 10-20 μg/ml).

If seizure control cannot be obtained with this combination of drugs, then other options exist. Many refractory dogs experience cluster seizures at varying time intervals, with relatively good control between cluster episodes. In this case, administration of rectal or nasal diazepam or other benzodiazepines (see "Cluster Seizures" lecture) may help to control the cluster events and avoid an emergency visit to the hospital. A third anticonvulsant medication may be added, and includes the following choices:

- Zonisamide (Zonegran) – 6-10 mg/kg q 12 hours (dogs only [dose doubled when administered with phenobarbital])
- Levetiracetam (Keppra) – 20 mg/kg q 8 hours
- Felbamate (Felbatol) – 15-20 mg/kg q 8 hours (dogs only)
- Gabapentin (Neurontin) – 10-30 mg/kg q 8 hours (dog) or q 8-12 hours (cat)
- Pregabalin (Lyrica) – 2 mg/kg q 8-12 hours, increasing 1 mg/kg/dose each week to a total of 3-4 mg/kg (dogs only)

These drugs have a variety of mechanisms of action, which appear to be different from phenobarbital and bromide, and patients may receive additional benefit from a multimodal antiseizure effect. Another advantage of the newer drugs is their improved side effect profile, as side effects are essentially limited to sedation (which tends to be less severe than that seen with either phenobarbital or bromide) and gastrointestinal side effects (vomiting, diarrhea) for some drugs. Felbamate is an exception, as there is concern with hepatic dysfunction, particularly when used in combination with phenobarbital. Elimination half-lives are relatively short, and drug steady state levels are reached relatively quickly with administration of a regular oral dose. The main disadvantages of these newer drugs are their expense (although most are now available generically, and costs are decreasing) and requirement for administration every 8-12 hours.

Of these newer generation drugs, the author generally prefers to use zonisamide or levetiracetam. Assays to measure blood levels of these drugs are available at a few select laboratories, but these drugs are often administered to effect. In some cases, success with the addition of an anticonvulsant drug may allow the eventual withdrawal of the initial medication, although this must be accomplished gradually and with caution. The author

frequently uses these newer anticonvulsant medications (particularly zonisamide and levetiracetam) as the second drug choice (typically instead of bromide). Other potential interventions to consider in select situations include acupuncture and the administration of a hypoallergenic diet.

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6005

EMERGENT MANAGEMENT – TREATMENT OF CLUSTER SEIZURES AND STATUS EPILEPTICUS

NEUROLOGY

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DEFINITIONS

Cluster seizures – two or more seizures occurring within a 24-hour period.

Status epilepticus – continuous seizure activity lasting longer than 5 minutes, or the occurrence of multiple seizures without recovery of baseline neurologic function between episodes.[1,2] Status epilepticus can be generalized or focal in nature, or in rare cases, can be nonconvulsive.[3]

PATHOPHYSIOLOGY AND GOALS OF TREATMENT

The vast majority of seizures are self-limiting events, with eventual spontaneous return to resting or baseline neurologic function. However, during status epilepticus, a variety of changes occur within cells and networks of cells that result in a situation where the seizure activity becomes self-sustaining. These changes may be independent of the initiating cause of the seizure and involves a variety of molecular mechanisms. Repetitive seizures cause inhibitory $GABA_A$ receptors to move from the synaptic membrane to the cell interior, while excitatory N-methyl-D-aspartate (NMDA) receptors may be recruited to the cell surface.[1] Stores of inhibitory neurotransmitters may become depleted and increased expression of drug efflux transporters such as P-glycoprotein may occur.[4] After a period of time, these cellular alterations may lead to pharmacoresistance to first-line agents that would normally be effective in seizure termination at earlier phases, such as the benzodiazepines.

Generalized status epilepticus can cause profound acidosis, hyperthermia, cardiac arrhythmias, hypoxia, neurogenic pulmonary edema, rhabdomyolysis, myoglobinuria, renal failure, cerebral edema, elevated intracranial pressure and neuronal necrosis, and therefore constitutes a medical emergency.[5] The goals of treatment are to stop the seizures, support systemic organ functions, and protect brain function. Finally, ongoing seizure activity/seizure control should be closely monitored. These goals are described in greater detail below.

1) Stop the Seizures

The most critical and pressing goal of therapy is to stop the seizures, by any means necessary. The initial drug chosen is usually a benzodiazepine (diazepam or midazolam) but depends on the suspected underlying cause.

- If hypoglycemia is suspected (juvenile toy breed dog, hunting dog or insulin overdose), administer 1-2 ml/kg of 50% dextrose intravenously (IV) diluted 1:1 in saline. Oral dextrose may be used in animals able to swallow when intravenous access is not readily achieved.
- In small or toy breed dogs that have recently whelped and are nursing puppies, the administration of calcium gluconate may be considered to address potential hypocalcemia.

Animals with known idiopathic, symptomatic or probably symptomatic (acquired/cryptogenic) epilepsy and those with unknown etiologies typically receive a benzodiazepine as the first-line drug.

- Diazepam (0.5 mg/kg) can be administered IV, intranasally or rectally to control seizures. The dose can be repeated twice, if necessary. Anticonvulsant

action only lasts about 15-30 minutes, and therefore some form of longer acting therapy is required if the seizures stop. Midazolam can be substituted for diazepam in this scenario, and lorazepam (0.2 mg/kg) may also be considered. These drugs may also be administered IV or intranasally but are not likely to be effective with rectal administration. In addition, unlike the others, midazolam can be successfully administered intramuscularly (IM).

- If the animal responds to a benzodiazepine bolus, phenobarbital may be considered for longer-term control. Naïve animals not previously receiving anticonvulsants can be loaded with 16-20 mg/kg divided into 4 doses and administered every 30-120 minutes (i.e., 4-5 mg/kg q 30-120 minutes). Epileptics already receiving phenobarbital may benefit from an additional "mini-loading dose" (5-10 mg/kg) depending on their serum levels of the drug. Phenobarbital should be continued at regular maintenance intervals (2-3 mg/kg IV, IM or PO q 12 hours or at the animal's regular dose) after this.
- Animals with severe cluster seizures or status epilepticus with some inter-ictal time (i.e., non-continuous) usually respond to a constant rate infusion (CRI) of diazepam (0.1-2.0 mg/kg/hour IV). The CRI can be started at the low end of the range (0.1-0.25 mg/kg) and gradually increased as necessary to control seizure activity. Once controlled, a seizure-free state is maintained for 12 hours, after which the infusion is gradually tapered (usually reduce dose by half every 4-6 hours) and stopped. The CRI can be administered with a syringe pump, if available, or by mixing with 0.9% saline in a small IV bag or Buretrol system. Diazepam is degraded by light and binds to plastic, and the syringe and tubing should be covered with brown plastic or aluminum foil, if possible. Midazolam can again be substituted in this scenario and is less likely to cause thrombophlebitis.
- Animals with continuous, prolonged seizure activity or those refractory to benzodiazepines may receive pentobarbital (3-15 mg/kg IV to effect), if available. This drug induces general anesthesia and is extremely effective in stopping the outward manifestation of the seizure. However, respiratory and cardiovascular function may be depressed, and these systems must be monitored very closely. Although intermittent bolus doses can be used, a CRI (2 mg/kg/hr adjusted to effect) may be more effective. Similar to benzodiazepine CRIs, animals may be kept seizure free for approximately 12 hours, and then weaned from the drug. It can be difficult to distinguish recovery from pentobarbital anesthesia from overt seizure activity. However, paddling movements of the limbs typically indicate the former, while seizure activity is usually characterized by overt tonic or clonic muscle contractions. Electroencephalography, if available, can help to differentiate these two scenarios. This medication also reduces the metabolic requirements of the brain and is considered to have neuroprotective effects.
- Propofol may be used as a substitute for pentobarbital if general anesthesia is required to control seizure activity. Due to its short duration of action, this drug must be given as a CRI (6 mg/kg initial bolus followed by 0.1-0.6 mg/kg/min). Substantial respiratory depression is common with this medication, and anesthesia must be closely monitored. In addition, propofol can have pro-convulsant effects in some patients. Some consider this to be the treatment of choice for patients in status epilepticus secondary to hepatic encephalopathy (typically after surgical repair of a portosystemic shunting vessel).
- If pentobarbital and propofol are not available, the use of an inhalant anesthetic (e.g., isoflurane or sevoflurane) to maintain general anesthesia should be considered as a last resort. Both require close monitoring of respiratory and cardiovascular parameters.
- A parenteral formulation of levetiracetam is also available. Although its use in animals with status epilepticus or cluster seizures has been limited to date, it may prove useful in this role, based on reports in humans and preliminary experience in canine patients.[1,6] Pharmacokinetic studies in dogs suggest that a dose of 20-60 mg/kg IV results in blood concentrations within the range considered to be effective in humans (5-45 μg/ml) for greater than 8 hours.[7,8] Levetiracetam is approximately 100% bioavailable after IM

administration and results in similar blood levels, although peak concentrations are not reached until about 40 minutes after the drug is given.[8]

- Reports of other medications for refractory status epilepticus are infrequent in veterinary medicine. There is a report of a dog with granulomatous meningoencephalitis and status epilepticus responding to intravenous ketamine infusion after failure to respond to diazepam and propofol.[9] This report follows several human case reports reporting similar efficacy for ketamine in the scenario of refractory status epilepticus, the rationale being blocking of NMDA receptors which may be responsible for the self-sustaining nature of this condition.[1,6,10] Ketamine infusions are gaining in favor for animals with refractory status epilepticus and dexmedetomidine has also been advocated in this scenario. Fosphenytoin is another newer medication that has been investigated for emergent seizure control in dogs. Additional therapies reported in refractory human cases include valproic acid, lidocaine, and topiramate.[6,11,12]
- A recent American College of Veterinary Internal Medicine consensus statement has developed a 3-tiered treatment system with documented levels of supporting evidence (Charalambous et al., 2023).

2) Support and Monitor Systemic Functions

As described above, status epilepticus can have profound effects on many body systems, and systemic functions must be closely monitored. These include:

- Mental status and level of consciousness
- Respiration, oxygen saturation and blood gases (if available)
- Cardiac rate and rhythm, blood pressure
- Body temperature
- Serum electrolytes, glucose, BUN and creatinine
- Fluid status and hydration
- Muscle damage and evidence of myoglobinuria (which may cause renal failure)

Intravenous fluid therapy is often indicated in order to maintain hydration and may help prevent renal damage if myoglobinuria is a concern. As severe seizure activity may lead to non-cardiogenic pulmonary edema, thoracic radiographs, pulse oximetry, and blood gas analysis should be considered in animals with compromised respiration. Aspiration pneumonia is also a concern, particularly in large recumbent dogs. Oxygen therapy may be administered in some of these patients. Active cooling should be considered in animals that are severely hyperthermic. Basic supportive nursing care must be performed in recumbent and stuporous animals, including applying artificial tears/lubrication to the eyes, providing adequate bedding/padding, periodically changing body position, turning from side to side, and passive range of motion of the limbs.

3) Protect Brain Function

Prolonged, severe seizure activity can lead to cerebral edema, increases in intracranial pressure and neuronal necrosis. Select cases may benefit from oxygen therapy, mannitol (0.25-1.0 g/kg IV over 10-20 minutes) or hypertonic saline (4-5 mg/kg of 4% or 7.2% solution IV) in order to address these effects. Compression of the jugular veins, coughing and sneezing all increase intracranial pressure, and should be avoided in animals where this is suspected to be increased. Therefore, jugular catheters, collection of blood from the jugular vein, neck bandages, nasogastric tubes, and nasal oxygen catheters should all be avoided. Intravenous lidocaine should be considered to reduce the coughing reflex if intubation is required. Elevation of the head approximately 30 degrees from the horizontal is a simple way to promote venous return from the brain and potentially reduce intracranial pressure. Pentobarbital administration, in addition to stopping seizure activity, also has the advantage of reducing cerebral metabolism, which can have neuroprotective effects in patients with status epilepticus.

4) Monitor ongoing seizure activity

Patients should be closely monitored to ensure the cessation of seizures and for the recurrence of seizure activity after initial therapy. This is typically done by visual observation and examination of animals for motor activity consistent with seizures. Whenever possible, cessation of seizure activity should be confirmed electrophysiologically with the aid of electroencephalography (EEG). This may detect animals whose outward motor manifestations of the seizure activity have stopped, but who continue to have

abnormal electrical brain activity, known as nonconvulsive status epilepticus (NSE). Although NSE has rarely been reported in veterinary patients,[9] this is likely a reflection of the infrequency with which veterinary clinicians perform EEG in this setting. The author has documented a number of canine patients with apparent NSE after prolonged convulsive status epilepticus or presenting with a primary complaint of altered mentation (Mariani, unpublished observations). An EEG is part of the routine diagnostic evaluation of human patients presenting with stupor or coma, and in the author's opinion, the same should be offered to veterinary patients wherever possible.

AT-HOME THERAPY FOR CLUSTER SEIZURES

Some owners can be taught to administer benzodiazepines in the home environment in order to reduce the number of seizures in dogs (or cats) prone to cluster seizure events. The goal is usually to prevent further seizures, reduce the number and severity of subsequent seizures, and avoid an emergency visit to the veterinary hospital. Diazepam has been used most often via the rectal route; a standard dose (0.5 mg/kg) can be administered, although in some animals on chronic phenobarbital therapy, a higher dose (1-2 mg/kg) may be required due to increased metabolism of the drug.[13,14] Drugs administered rectally in the dog undergo a substantial first-pass effect and hepatic metabolism as the majority of absorbed drug enters the portal circulation.[15] As a result, the bioavailability of diazepam after rectal administration is only about 2.7-7.4% at doses of 0.5 and 2.0 mg/kg respectively in dogs not receiving phenobarbital.[16] However, some anticonvulsant effect is achieved as the main metabolites of diazepam (desmethyldiazepam and oxazepam) possess 20-50% of the activity of the parent drug.[17-19] Lorazepam is unsuitable for rectal administration, as its primary metabolite (lorazepam glucuronide) does not have anticonvulsant activity.[15] Midazolam does have a metabolite with reported pharmacologic activity (1-hydroxymidazolam), although the contribution of this reported activity is controversial.[20]

Intranasal (IN) administration of benzodiazepines avoids several of the shortcomings of the rectal route. The IN route avoids substantial first pass metabolism, and drug is directly absorbed into the systemic circulation thorough the dense vascular plexus present in the nasal passages.[21] In addition, there is evidence for direct movement of drug through the cribriform plate and into the central nervous system.[22-24] Several studies suggest that the bioavailability of diazepam is much higher after IN administration than after rectal.[25,26] and this route has been used successfully by the author in several emergent clinical cases (0.5 mg/kg). Preliminary experience suggests that intranasal lorazepam (0.2 mg/kg) may also be useful as an alternative to rectal diazepam for at-home use by owners.[27] Intranasal or IM midazolam (0.5 mg/kg) is another option available for these scenarios.

Lastly, pulse dosing of a novel anticonvulsant (i.e., one that the animal is not taking as a maintenance drug) can be effective in controlling cluster seizures and avoiding a visit to the hospital. The author typically recommends a loading dose, followed by administration of the pulse therapy for 48-72 hours. Options for such therapy include levetiracetam (60 mg/kg once then 20 mg/kg q 8 hrs) and pregabalin (4 mg/kg once then 2-3 mg/kg q 8-12 hrs).

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7001

PEARLS OF THE OPHTHALMIC EXAM (TOP 10 TIPS FOR A COMPLETE EXAM)

NEUROLOGY

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SUMMARY

It is undoubtedly important to know how to treat disease but without a diagnosis, treatment is often ineffective or worse. Fortunately, reaching an ophthalmic diagnosis relies almost completely on performing a thorough ophthalmic examination, which can be done with the simplest of instrumentation. Indeed, never were the famous words "more is missed through not seeing than not knowing" more apt. Here we present the top 10 tips for the eye exam using equipment that is almost certainly already in your clinic.

THE TOP 10 TIPS FOR AN EXCELLENT EYE EXAM

1. Avoid sedation
2. Get at eye level with your patient
3. Dim the ambient light
4. Use a bright light source and magnification
5. Take an orderly approach
6. Tilt the nose down so that the eye rolls up
7. "Always"
    - Retroilluminate
    - Check for aqueous flare
    - Measure intraocular pressure (IOP)
    - Use fluorescein last
8. "Always" dilate the pupil to examine the
    - Lens
    - Vitreous
    - Fundus
9. Always make an etiologic diagnosis (For example, there is no treatment for conjunctivitis, there are various treatments for herpetic conjunctivitis, foreign body conjunctivitis, allergic conjunctivitis, keratoconjunctivitis sicca, etc.)
10. Look, look, and look again. (More is missed through not looking than not knowing).

FOUR BASIC REQUIREMENTS FOR AN EXCELLENT EYE EXAM

There are 4 essential requirements for a thorough ophthalmic examination:

1. The patient and doctor must be at eye level with each other
2. The exam must be performed in dim ambient light
3. A bright, focal light source and a means of magnification are essential
4. Always perform an orderly and complete examination

Detection of minute but very significant changes necessitates use of magnification and a focal, intense light source. This combination can be provided using an Optivisor® head loupe and Finoff® transilluminator. A readily available alternative is the otoscope used without the plastic cone. This provides a focal light source and approximately 2-3x magnification. The slit lamp biomicroscope maximizes the combination of magnification and focal light source. By focusing the light to a narrow slit, an optical section provides microscopic detail in transparent ocular media such as the tear film, cornea, aqueous, lens, and vitreous. Small, less expensive hand-held slit lamps are also available.

The ophthalmic examination should be carried out in a repeatable and sequential manner to ensure that nothing

is overlooked. Examining the unaffected eye prior to the affected eye in animals with unilateral disease ensures that it is not forgotten and provides information on the individual patient's normal ocular appearance. A prepared exam sheet reminds the practitioner to perform all necessary tests in the correct sequence.

FOUR BASIC TECHNIQUES FOR A COMPLETE EYE EXAM

Mastering just 4 procedures will provide all of the essential information from the anterior segment:

1. Retroillumination
2. Focal illumination or transillumination
3. Tonometry (measurement of intraocular pressure)
4. Assessment of aqueous flare

Retroillumination is a simple but extremely useful technique for assessment of pupils and all parts of the transparent ocular media (tear film, cornea, aqueous, lens, and vitreous). A focal light source held close to the examiner's eye and directed over the bridge of the patient's nose from at least arm's length is used to elicit the fundic reflection or reflex. This is usually gold or green in tapetal animals or red in atapetal individuals. Each eye is illuminated equally and the fundic reflex is used to assess and compare pupil size, shape, and equality. Additionally, opacities in the ocular media will obstruct the fundic reflection and are noted for more detailed subsequent examination using transillumination or retroillumination again after pupil dilation. Both of these subsequent techniques can be augmented by magnification. Retroillumination is particularly useful for differentiating nuclear sclerosis from cataract.

The anterior segment includes all structures in front of and including the lens. These are best-examined using focal illumination and subsequently magnification. To maximize the benefits of **focal illumination**, the light source should be directed from an angle that differs from the observer's viewing angle. Varying the viewing and lighting angles relative to each other permits the examiner to utilize parallax, reflections, perspective, and shadows to gain valuable information regarding lesion depth. This technique is particularly useful for examining the anterior chamber since changes within the anterior chamber can be more easily differentiated from corneal, iridal, or lenticular changes when viewed transversely. In cats and horses, the corneal curvature and anterior chamber depth are so great that limited visualization of the iridocorneal angle is also possible.

The importance of a sequential anterior segment examination cannot be overstated. Following retroillumination and assessment of menace response and pupillary and palpebral reflexes, an obvious method is to begin at the front and progress to the back of the eye. This ensures that the lids (including skin, margin, and cilia), conjunctiva (nasolacrimal puncta, third eyelid, bulbar, and palpebral conjunctival surfaces), sclera, cornea (including tear film and particularly the limbus), anterior chamber, iris, and lens are examined completely. Anterior segment examination should be initiated prior to dilation so that the iris face is easily examined; however complete examination of the lens requires full dilation.

Assessment of intraocular pressure (tonometry) is essential for differentiation of the two major, vision-threatening conditions in which red-eye is the hallmark feature – uveitis and glaucoma. The availability of easily used and reasonably priced tonometers such as the Tonopen® or TonoVet® make measurement of intraocular pressure (IOP) easier in all species, particularly cats. Unlike the Schiotz tonometer, the Tonopen and TonoVet measure IOP directly and do not require any conversion. They can also be held horizontally and therefore allows measurements to be performed with the patient's head held in a normal, relaxed position. Finally, their small probes/footplates permit easy measurement of IOP in even the smallest feline and pediatric canine eyes.

The TonoPen comes with an excellent instructional video and manual, however the following tips may assist you to get the most from your Tonopen. A drop of topical anesthetic is applied to the cornea. A disposable cover is placed over the Tonopen tip and the pen is turned on with firm, somewhat protracted pressure on the large black button about one third way down the shaft. The equipment should be periodically calibrated according to the manufacturer's directions. Correct patient restraint is essential. The patient should be lightly restrained so as to not artificially raise IOP. In particular,

direct pressure on the jugular veins and on the globe itself via the eyelids should be avoided. I prefer to have an assistant (and not the owner!) restrain the patient's head using the angle of the mandible. I then hold the Tonopen in my dominant hand, and gently part the patient's eyelids using my non-dominant hand but such that pressure is applied to the underlying orbital rim; not globe. I then rest the hand holding the Tonopen onto the hand holding the eyelids or onto the patient's head itself and gently touch the central cornea with the Tonopen tip. Minor movements away from the cornea and very gentle "blotting" of the cornea with the tip will enhance the reliability and reproducibility of the readings while reducing the number of readings necessary. Particular attention should be paid to the "approach angle" of the Tonopen tip to the cornea. The tip's flat surface should be exactly parallel to the corneal surface. This is best achieved by viewing the interface between the cornea and the tip from the side. The approach angle of the Tonopen itself should be exactly perpendicular to the corneal surface. However, note that due to corneal curvature, this means the approach angle must be changed dramatically if any area other than the central cornea is used.

Each time the cornea is appropriately "blotted" with the probe, an electronic tone will advise the operator that a reading has been obtained. When a suitable number of readings has been obtained, a tone of a different pitch will sound and no further readings can be obtained without restarting the Tonopen using the large black button again. The number of readings required to achieve an average varies depending on how disparate the readings are from each other and from the normal physiologic range. A small digital screen at the end distant from the tip displays the IOP in mmHg and provides an estimate of the "reliability" (coefficient of variance) of the result. This appears as a small bar above one of 4 percentage readings. This bar should be above the 5% mark or tonometry should be repeated on that eye. Across large populations, normal canine and feline IOP is reported as approximately 10-25 mmHg. However, significant variation is noted between individuals, technique, and time of day. Comparison of IOP between right and left eyes is therefore critical to interpretation of results. A good rule of thumb is that IOP should not vary between eyes of the same patient by more than 20%.

The obvious application for tonometry is the diagnosis of glaucoma (where IOP is generally elevated). However tonometry is also used to diagnose uveitis; in which IOP is lowered due to loss of function of the inflamed ciliary body. Perhaps the most important role for tonometry is the monitoring of progress of these diseases and the adjustment of medications based on these data.

Aqueous flare is a pathognomonic sign of uveitis and is due to breakdown of the blood-ocular barrier with subsequent leakage of proteins into the anterior chamber. Aqueous flare is best detected using a very focal, intense light source in a totally darkened room. The passage taken by the beam of light is viewed from an angle. In the normal eye, a focal reflection is seen where the light strikes the cornea. The beam is then invisible as it traverses the almost protein- and cell-free aqueous humor in the anterior chamber. The light beam is visible again as a focal reflection on the anterior lens capsule and then as a diffuse beam through the body of the normal lens due to presence of lens proteins. If uveitis has allowed leakage of serum proteins into the anterior chamber then these will cause a scattering of the light as it passes through the aqueous. Aqueous flare is therefore detected when a beam of light joining the focal reflections on the corneal surface and the anterior lens capsule is visible traversing the anterior chamber. A slit lamp provides ideal conditions for detecting flare; however the beam produced by the smallest circular aperture on the direct ophthalmoscope held as closely as possible to the cornea in a completely darkened room and viewed transversely will also provide excellent results. The slit beam on the direct ophthalmoscope is not as intense and does not provide as many "edges" of light where flare can be appreciated most easily. Assessment of flare may be easier after complete pupil dilation due to the apparent dark space created by the pupil. Combined assessment of IOP and aqueous flare should be performed whenever glaucoma or uveitis is suspected because of the frequency with which these conditions co-exist.

7002

DOING A GREAT RETINAL EXAM – AS EASY AS "FUNDIC MATHEMATICS"

NEUROLOGY

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INTRODUCTORY PHILOSOPHY

Fundic examination is probably the greatest challenge in the ophthalmic exam. Fortunately, anterior segment abnormalities tend to outnumber fundic abnormalities in general practice. Funduscopy is however critical in the assessment of animals presenting with visual disturbance, pupil abnormalities, or systemic disease.

WHICH OPHTHALMOSCOPE?

Traditionally, there have been two methods for viewing the fundus; indirect and direct ophthalmoscopy. Recently, Welch Allyn (The Panoptic®) and Keeler (The Wide Angle Twin Mag®) have both introduced new ophthalmoscopes that combine some of the best features of both methods.

THE DIRECT OPHTHALMOSCOPE

This instrument hangs on the walls of most veterinary exam rooms the world over and yet is not the best method for examining the fundus. It is used by turning the lens power to "0", selecting the largest circle of light that it emits, turning the light to almost full brightness, turning the room lights off or to a dim setting, and resting the brow rest of the ophthalmoscope against the operator's brow. Ideally the operator's right eye should be used for examining the patient's right eye and vice versa, although some people have trouble using their non-dominant eye. The examiner should hold the animals eyes open while an assistant holds the head steady. Begin viewing at arm's length from the patient and move around until a bright tapetal reflection is obtained (as for retroillumination). The examiner should then slowly approach the animal, while always aiming at the tapetal reflection of the eye to be examined. Good focus in a normal patient should be reached within just a few centimeters of the eye. It is therefore sometimes useful to extend the index finger of the hand holding the ophthalmoscope so as to rest against the patient's cheek.

The direct ophthalmoscope presents a highly magnified, upright image of a very small region of the patient's fundus. In compliant (human) patients this instrument can then be used to slowly and sequentially examine the whole fundus in minute detail. In our patients, this small field of view frequently means that areas of the retina, particularly peripherally, are never examined and that one area of the retina cannot readily be compared to another region.

THE INDIRECT OPHTHALMOSCOPE

Although more technically difficult when first learned, indirect ophthalmoscopy is the preferred method for examining the veterinary fundus. The reasons for this are the larger field of view that is permitted by this technique. This permits the examiner to compare regions of the fundus one against the other such that focal areas of subtle pathology may be detected by comparison with neighboring normal areas. It also makes a complete exam of the whole fundus more likely and easier than when performed with direct ophthalmoscopy. The perceived downfalls are that the image is les magnified, however this can be countered by moving closer to the patient while performing indirect ophthalmoscopy or by a subsequent (more magnified) examination of any "suspect" areas using the direct ophthalmoscope. Another difficulty for beginning ophthalmoscopists is that indirect ophthalmoscopy produces an inverted view. This makes navigation around the fundus and correct geographic localization of lesions a little more difficult at first but can

be readily overcome with practice. I prefer to use a Volk 20D or 2.2 Pan Retinal indirect lens.

The Welch Allyn Panoptic® or Keeler Wide Angle Twin Mag® Ophthalmoscopes. Welch Allyn have recently released a new ophthalmoscope that produces a view with many of the best features of those produced using direct and indirect ophthalmoscopy. The image produced is upright, moderately magnified and includes more of the fundus than is possible with the direct ophthalmoscope but less than that provided by the indirect ophthalmoscope. Perhaps best of all, it is extremely user friendly and encourages fundic exams – and that is a good thing! The scope may be purchased alone and fits directly onto the standard Welch Allyn handset already in your clinic.

AND SO, WHICH OPHTHALMOSCOPE?

For the specialist ophthalmologist, indirect ophthalmoscopy for the initial fundic examination followed, when necessary, by direct ophthalmoscopy to view areas of interest detected during indirect ophthalmoscopy remains the preferred method of performing a complete fundic examination. In general practice, the Panoptic provides a very useful compromise between these two techniques and in my opinion provides a view that is definitely superior to that provided by the standard direct ophthalmoscope. No matter which ophthalmoscope you use, the golden rules seem to be:

- Always dilate the patient's pupils (if safe)
- Allow yourself the pleasure of doing this "special diagnostic examination" away from the owner and when you have time to do it properly
- Do this exam regularly enough that you become proficient at it.

SO WHAT AM I SEEING BACK THERE?

The ocular fundus represents a group of tissues that is sometimes challenging to examine and whose lesions are often difficult to interpret. In this session we will demonstrate, 2 potentially novel means for examining the fundus of animals. The first approach acknowledges that the fundus is a compound structure that can be best understood by "constructing" it and that the fundic exam findings might best be interpreted by "deconstructing" them and considering them in light of those basic elements from which the fundus is composed. Secondly, we will approach the fundic exam by asking 5 questions designed to systematically interpret what you are seeing "back there" in a clinically applied and relevant manner.

THE "BUILD A FUNDUS" APPROACH TO INTERPRETING FUNDIC EXAM FINDINGS.

The fundus is a collective term describing all structures in the posterior portion of the globe that can be viewed with the ophthalmoscope (sclera, choroid, tapetum (in most dogs and cats), retinal pigment epithelium (RPE), neural retina, optic nerve head, and retinal vasculature). Normal appearance of each of these structures varies widely and is further altered by the appearance of each overlying structure. Therefore, knowing the order of these layers and how they hide or alter the appearance of those layers behind them is critical.

The sclera is tan-white and usually not visible due to overlying layers of the fundus. However, if other layers are thinned or absent, then sclera can be seen. An example would be a subalbinotic animal with little melanin. In such animals, sclera is seen between the larger choroidal vessels.

The choroid (posterior uvea) is composed of large blood vessels with variable amounts of melanin typically arranged in a somewhat linear fashion between blood vessels. The choroidal vessels need to be distinguished from the retinal vessels that overly them. Choroidal vessels are generally larger, more orange, and branch less overtly than retinal vessels.

The tapetum is technically part of the choroid but, because it is variably present and differs in appearance so much from the choroid proper, is considered separately here. The tapetum occupies a variable percentage of the dorsal fundus where it overlies and obstructs view of the choroid and sclera. Tapetal size can vary greatly but tends to be larger in cats and smallest in small dog breeds. Primary tapetal pathology is rare, however (like all other fundic layers) changes in funduscopic appearance of the tapetum are observed very frequently. These result from changes in the subjacent choroid and/or overlying retina.

The retinal pigment epithelium (RPE) is variably pigmented but uniformly present in the normal fundus. It is the outermost layer of the retina but is dealt with separately here because it has a distinctly different appearance from the neurosensory retina. It lies between the neurosensory retina and the tapetum (if present) or choroid (if no tapetum is present. It is predictably non-pigmented where it overlies

the tapetum and is usually a relatively homogenous liver to dark brown/black color in the ventral (inferior) non-tapetum. The most frequent pathologic changes involving the RPE are alterations in the degree of melanosis; i.e., depigmentation of the non-tapetal fundus seen most dramatically in uveodermatological or VKH-like syndrome in dogs, or hyperpigmentation secondary to any chronic, inflammatory process.

The neurosensory retina is composed of the other 9 layers of the retina and lies upon the RPE. It is slightly translucent and therefore reduces the intensity of light reflected from the tapetum to the observer. Therefore, "tapetal hyper-reflectivity" is actually not a tapetal finding but represents retinal thinning which allows more light to be reflected from a normal tapetum. Conversely, thickening of the retina due to edema, cellular infiltration, hemorrhage, or pigmentation will reduce or even obstruct the tapetal reflection.

The retinal vasculature, which lies within the neurosensory retina, varies between individuals and species. Both arterioles and venules can be seen in dogs and cats. In both species, blood vessels should extend to the retinal periphery, bifurcating frequently but without excessive tortuosity. Changes visible funduscopically are usually due to systemic diseases (vasculitis, hypertension, anemia, hyperlipidemia, hyperviscosity), or local retinal vascular changes such as attenuation as seen with retinal degenerations.

The optic nerve head (ONH or optic papilla or optic disc) in the cat is a small, relatively dark gray/red, non-myelinated circle within the tapetal region. Variable degrees of ONH myelination in dogs dictate that normal ONH appearance may range from small, flat and circular through larger, raised, and irregular triangular-shaped. ONH position in dogs relative to the tapetal/non-tapetal border is extremely variable and dependent on tapetal size. Frequently observed ONH pathology includes inflammatory changes (hyperemia, edema, hemorrhage, or cellular infiltration), "cupping", or atrophy.

THE FIVE FUNDIC QUESTIONS AND "FUNDIC MATHEMATICS"

When I am training veterinarians and veterinary students to interpret their observations from the fundic examination, I have found "The 5 Fundic Questions" and the principle of "Fundic Mathematics to be really useful. These 2 approaches utilize principles we have introduced in the last section on "building a fundus". The 5 fundic questions are:

1. **Can I get all parts of the fundus in focus at once?** If not, then the defocused region is almost certainly protruding in front of the normally positioned tissues (e.g., retinal detachment, ONH swelling, subretinal granulomas/masses, and orbital masses impressing the globe). Other alternate mechanisms that may explain this are fortunately seen very rarely but might include that the defocus is due to regions of the fundus being further behind the normal plane (e.g., scleral ectasia and out-pouching of the rest of the fundic structures), or due to vitreous debris between you and a normal fundus. Of all of these, retinal detachment is "far and away" the most common reason for difficulty focusing on all parts of the fundus at once.

2. **What is the general tapetal "sheen" over the whole fundus?** Don't forget to wobble or "shimmy" the indirect lens back and forth and assess the fundus from different directions before making this judgment. If the tapetum is hypo-reflective, then there is likely addition of material in front of the tapetum - in the subretinal space, RPE, or retina. If the tapetum is hyper-reflective, then there is likely subtraction of material from in front of the tapetum. This is almost always retinal thinning due to retinal degeneration. This principle of addition or subtraction introduces the concept of fundic mathematic – "every fundic change can be explained by addition of something that is not supposed to be there (and reduction in your view of a normal structure) or subtraction of a layer that is supposed to be there (with exposure of another layer that you usually do not see at all or so clearly).

3. **Are there any focal areas of unusual coloration within the fundus?** If so, what color are they and do they represent an "addition" of a "new" cell or structure in the fundus or exposure of a normally hidden layer or structure within the fundus due to "subtraction" of a normal constituent? (Another example of fundic mathematics).

4. **How is the retinal vasculature?**
    - Normal
    - Prominent (hypertension, hyperviscosity, congenital abnormalities)
    - Reduced in size or intensity (anemia, hypovolemia, retinal degeneration)

5. **How is the optic nerve head?** Inflamed (optic neuritis/papilledema) or small (hypoplasia or atrophy)?

7003

WHAT'S NEW IN OCULAR PHARMACOLOGY (ONE DROP OR TWO)

NEUROLOGY

David Maggs, BVSc (hons), DACVO, MANZCVS

ONE DROP OR TWO?

Always administer one drop. The conjunctival fornix of the cat and dog can "hold" about 16 µL and a drop is about 50 µL. Therefore, one drop is already ~3-fold too much. Giving a second drop is a 6-fold excess. But it is not just wasteful; each drop causes reflex tearing, so 2 drops still only delivers 16 µL but that 16 µL is washed out by increased reflex tearing.

Ointments or drops?

This is one of the topics that you will find lots of opinions on and rarely does it make much difference. I am often guided by client preference or apparent patient preference.

However, the following guidelines may help:

- Solutions are not practical in large animals unless a subpalpebral lavage system is placed.
- Ointments blur vision more
- Multiple ointments can be applied simultaneously but drops should be separated by > 5 mins.
- Ointments increase contact time (and may permit decreased dose frequency)
- Ointments provide lubrication (and are therefore good for patients with entropion or recent eyelid surgery)
- Ointments protect against desiccation (and are therefore good for patients with KCS)
- Solutions, not ointments should be used when corneal rupture is present or likely
- Solutions, not ointments should be used prior to ocular surgery
- Both ointments and drops retard wound healing and should be used at the minimum effective frequency and stopped ASAP.

When I give a systemic drug, does that get to the eye?

When deciding via what route to medicate the eye, there is a tendency to group all "eye diseases" together. In fact, the eye is composed of a variety of tissues that vary greatly:

- Drugs penetrate through the cornea and sclera very poorly
- Some ocular tissues are vascular and some are avascular
- Some ocular tissues are "behind" the blood-ocular barrier.

Systemically administered drugs typically reach only vascular tissues (eyelids, conjunctiva, parts of the iris, ciliary body, optic nerve, and retina, the choroid, and orbital structures. There are 2 exceptions to this rule: a) inflammation causes a (temporary) breakdown of the blood ocular barriers and b) a few drugs cross the intact blood-ocular barrier and are released into the aqueous or vitreous humors (e.g., chloramphenicol, fluconazole) or reach meaningful concentrations in the tears (e.g., famciclovir, doxycycline).

As a rule, orbital, adnexal, conjunctival, and intraocular disease can be treated via the systemic route but corneal disease cannot.

Which eye drops/ointments penetrate inside the eye?

The cornea is a trilaminar sandwich composed of a hydrophobic epithelium, hydrophilic stroma, and hydrophobic endothelium. Very few topically applied drugs penetrate well through this barrier. Even those that are specially formulated to breach that anatomic barrier become rapidly diluted by tears (especially in painful eyes with epiphora), absorbed by conjunctival capillaries and taken away from the eye, or are lost down the nasolacrimal duct or onto the face over the eyelid margin. Therefore, even for drugs which do cross the cornea only a small percentage of the topically applied drug enters the eye, and that is rapidly diluted in the aqueous humor before being "washed" out of the eye through the iridocorneal angle. Even those drugs that penetrate very well do not reach clinically meaningful concentrations in tissues behind the lens. Therefore, diseases of the posterior segment (or orbit) should always be treated with systemically administered drugs. Corneal penetration of topically applied drugs is increased in the presence of ulceration.

The following drugs are categorized according to how well they penetrate the eye across an intact cornea following topical application:

Class	Penetrates well	Penetrates poorly
Antiviral	Trifluridine	All others
Antibiotic	Fluoroquinolones Chloramphenicol	All others
Corticosteroids	Prednisolone Dexamethasone	Hydrocortisone
NSAIDs	All	None
Glaucoma medications	All	None

Some golden rules of ocular pharmacology

These preceding topics lead to some golden rules of ophthalmic pharmacology:

1. Always administer one drop
2. Always leave 5 minutes between drops of a different type
3. Always work "up" in viscosity when applying two or more different drops or ointments to the same eye
4. Drugs required in high concentration in the cornea or conjunctiva usually are best administered by frequent topical application (but no more frequently than 5 minutes)
5. Drops are quickly diluted and eliminated from the eye by tears. This can be overcome by increased frequency of application (but no more frequently than 5 minutes)
6. The cornea is a trilaminar (lipid-water-lipid) sandwich across which many topically applied drugs do not cross
7. Even those topically applied drops that cross the cornea do not penetrate to tissues caudal to the anterior uvea; posterior segment disease must be treated via the systemic route
8. Increased "dose" of topically applied drugs may be achieved by increasing drug concentration in the topical preparation (within physiologically acceptable limits); increasing the frequency of application; slowing absorption (increasing contact time)
9. Systemic absorption of drugs from the conjunctival sac following topical application is rapid and may result in notable blood concentrations.

What ophthalmic drugs should I stock in my pharmacy?

Obviously, no answer to this question is correct for every practice but here are some basics I could not manage without for canine and feline practice. The list is compiled without consideration of availability and price which vary so frequently and widely nowadays.

Antibiotics

- Triple antibiotic ointment and solution
- Ofloxacin solution
- Compounded cefazolin solution (see below)
- Doxycycline tablets/oral suspension

Anti-inflammatory/Immunomodulatory agents

- Neopolydex ointment and solution
- Prednisolone acetate1% suspension
- Diclofenac solution
- Cyclosporine ointment and suspension
- Tacrolimus suspension
- Oral prednisone/prednisolone
- Oral NSAIDs

Glaucoma

- Dorzolamide solution
- Dorzolamide/timolol combo solution
- Latanoprost solution
- Demecarium bromide solution

Antivirals

- Idoxuridine solution
- Cidofovir (compounded) solution
- Famciclovir tablets

Tear substitutes

- Hyaluronate solution
- Petrolatum ointment

Diagnostics

- Fluorescein strips
- Proparacaine
- Tropicamide

Other

- Atropine ointment and solution
- Serum

Compounded cefazolin

1. Remove 2 ml from a 15-ml bottle of artificial tear solution and discard.
2. Reconstitute a 500-mg vial of cefazolin with 2 ml of sterile water.
3. Add entire 500 mg of the reconstituted cefazolin (2.4 ml) to the bottle of artificial tear solution.

Final concentration = 33 mg/ml (3.3% solution).
Shelf life: 28 days.
Keep refrigerated.

REFERENCES

References available from the author on request.

7004

MY APPROACH TO NON-HEALING CORNEAL ULCERS IN DOGS & CATS

NEUROLOGY

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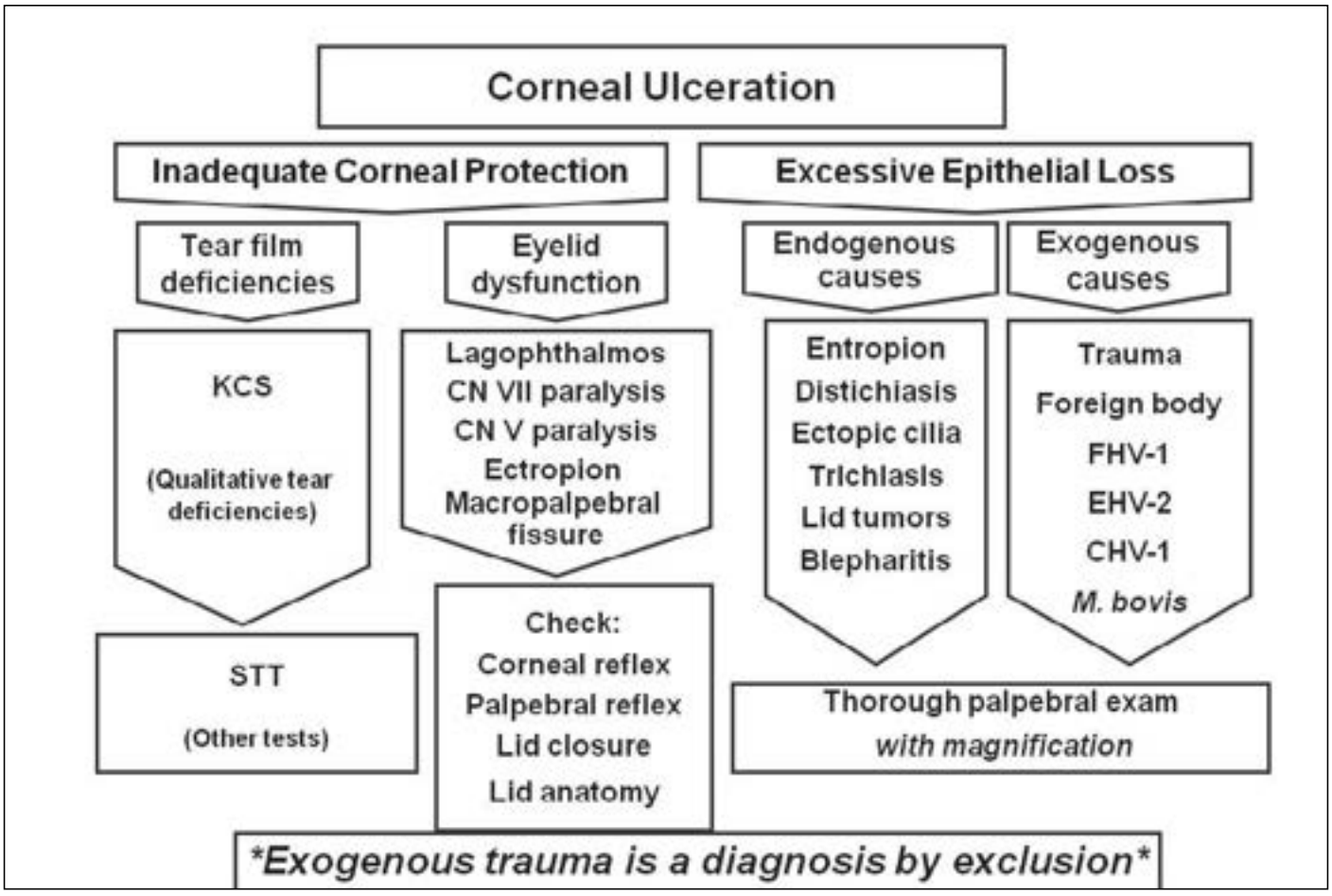
INTRODUCTION

When an ulcer hasn't healed at the recheck, a few golden rules can help us see that this ulcer has identified itself as a "complicated ulcer" with only one of only 2-3 broad causes possible.



THE GOLDEN RULES OF ULCER MANAGEMENT

1. Always find and remove the primary cause

All patients with corneal ulcers should undergo a very thorough examination aimed at determining and removing, correcting, or treating the cause. Because corneal epithelium is constantly being abraded and desiccated but is protected from excess abrasion by the eyelids and tears, ulcers may be thought of (in a mechanistic sense) as arising when this situation becomes unbalanced i.e., there is decreased corneal epithelial protection due to tear film or eyelid dysfunction, or increased corneal epithelial loss due to exogenous or endogenous causes (Figure 1).

2. Although trauma is common, it can only be diagnosed by ruling out all other causes.

This requires Schirmer tear testing, assessment of palpebral reflexes, thorough examination of eyelid and conjunctival anatomy and function (including the posterior or bulbar face of the third eyelid), and consideration of infectious causes. Failure to do this (and attributing the ulcer to "a scratch") makes it more likely you will break Rule #1 and that the ulcer will become chronic or progressive.

3. Other than herpesviruses (in "all" species) infection does not cause ulceration

Although secondary bacterial infection is the most feared complication in an ulcer, no bacteria initiate corneal ulcers in small animals. In dogs and cats, the only organisms known to initiate ulcers are feline and canine herpesviruses. Failure to recognize these ulcers leads to failure to use an antiviral drug and again leads to breaking of Rule # 1.

4. Know the speed of normal corneal wound healing

Epithelial healing occurs within hours to days and should leave no long-term scar. Stromal healing requires activation of resting keratocytes into active fibroblasts and/or fibrovascular ingrowth. This may take weeks to months for a deep ulcer and grey wispy scars are expected. Endothelial cells are post-mitotic in adult animals and regeneration is extremely limited to non-existent, with persistent focal corneal oedema expected following deep ulcers. These healing rates have led to one of the definitions of a complicated ulcer, i.e., one that fails to heal within 7 days.

5. Prior to therapy, always characterize ulcers as "simple" or "complicated"

"Simple" (or "uncomplicated" ulcers, by definition, are superficial and present less than 7 days. If the ulcer fails to fit either of these criteria (i.e., it has been present more than 7 days or is deep) then it is defined as "complicated". Based upon their clinical appearance including their fluorescein staining pattern, complicated ulcers should be further defined as indolent ulcers (also known as Boxer ulcers or superficial chronic cornea epithelial defects – SCCEDs – and seen in dogs only), infected ulcers (in cats or dogs), or ulcers in which the primary cause is still present (in cats or dogs).

Fortunately, each of these ulcer complications has a characteristic appearance:

- Indolent ulcers - are seen in dogs only. They are superficial, uninfected, chronic (or will become chronic), and have a lip of redundant non-healing corneal epithelium that is easily debrided with a cotton-tipped applicator (CTA). This lip often produces a characteristic "halo" fluorescein staining-pattern due to leakage of stain under the non-adherent lip. They arise from a failure of replicating and migrating epithelium to adhere to the underlying stroma. Diagnosis is reliant on characteristic signalment, chronicity, appearance and staining-pattern of the ulcer, as well as the ease with which the epithelium is manually debrided with a CTA.
- Ulcers in which the primary cause is still present typically appear like simple ulcers but remain chronic. That is, they don't necessarily worsen; they just don't heal.
- Bacterially infected ulcers have one or more of 3 features in any combination - stromal loss (i.e., the ulcer is deep), corneal malacia (or "melting"), and/or infiltration of the stroma with white blood cells (which turns the stroma yellow-green).

Treatment of indolent corneal ulcers

The first step is removal of all redundant, non-adherent epithelium by debridement with multiple dry cotton-tipped applicators (CTA) following application of topical anaesthetic +/- sedation. If this fails, a second CTA debridement can be done, but many move to anterior stromal irritation using a diamond burr (DBD) or a needle passed in either a grid or multifocal punctate pattern. Grid keratotomy (GK) is performed by making linear striations in the cornea in a "cross-hatch" or grid pattern using the tip of a 25-gauge needle. Topically applied antibiotics are also indicated since loss of epithelium predisposes the corneal stroma to infection. To control reflex uveitis, minimal applications of atropine (just sufficient to dilate the pupil). Excess atropine dries tears essential for ulcer healing. Hyperosmotic (5%) sodium chloride ointment (Muro 128® and others) is recommended if corneal edema is marked as this may further decrease already impaired epithelial adhesion. DBD/GK may be combined with application of a soft contact lens sometimes held in place with a partial (lateral) temporary tarsorrhaphy. This provides increased protection for the healing cornea and seem to increase comfort. I prefer ophthalmic solutions over ointments in this disease.

A success rate of approximately 80% can be expected using GK alone. Treatment failures tend to arise when patients are "under-treated" by inadequate debridement, or too few, too superficial, and/or too short score marks in the cornea. Indolent ulcers that have not healed 10-14 days after an initial GK may need the procedure repeated. GK is contraindicated in all other ulcer types in dogs and in all ulcers in cats. In cats, GK is a very "reliable" means of inducing a corneal sequestrum. Indolent ulcers in dogs should be pre-treated for a few days with a topical broad spectrum, bactericidal ophthalmic antibiotic to sterilize the corneal surface before the GK is performed.

Treatment of deep (infected) corneal ulcers

1. Corneal culture and sensitivity
2. Frequent (as often as once hourly) therapy with an antibiotic with appropriate spectrum such as tobramycin, fortified/compounded amikacin or gentamicin, or a fluoroquinolone such as ofloxacin/ciprofloxacin. Consider a "loading dose" technique of 1 drop every 5 mins for the first 30-60 minutes.
3. Frequent (as often as once hourly) topical therapy with serum (the patient's or another animal's serum).
4. Topical atropine to effect for reflex uveitis
5. I prefer solutions over ointments for deep ulcers at risk of perforation
6. Surgery for ulcers that are rapidly progressive, have obvious areas of melting, or are deeper than half corneal thickness:
    a. Conjunctival grafts are preferred
    b. Temporary partial (lateral) tarsorrhaphy are a reasonable alternative for owners unwilling to have conjunctival grafts applied.
    c. I do not recommend third eyelid flaps. They do provide a "bandage" which reduces desiccation and frictional irritation of the cornea by eyelids, but they completely prohibit penetration of medications to the cornea or observation of the disease.
7. Frequent observation via hospitalization or recheck (sometimes as early as the following day).

7005

UVEITIS – IT'S JUST INTRAOCULAR LYMPHADENOPATHY

NEUROLOGY

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SUMMARY

The uvea contains familiar tissues and cell types (lymphocytes, smooth muscle, and blood vessels, for example), is inflamed by familiar antigens (infectious agents, neoplasia, auto-antigens) and reacts with the 5 cardinal signs of inflammation seen elsewhere (heat, pain, swelling, etc.). And yet it can be a very confusing disease. This lecture aims to provide aids to diagnosis and therapy of uveitis by likening it to lymphadenopathy (because it is more similar than it is different) while highlighting differences (because these are helpful). For further information, please refer to a review article from the Journal of Feline Medicine and Surgery upon which these notes are based (2009 Mar;11(3):167-82).

CLINICAL SIGNS

Active (acute) uveitis

Uveitis has few pathognomonic signs and these are notably subtler in cats than they are in dogs. Therefore, uveitis in cats often goes undetected by owners and untreated by veterinarians until potentially blinding sequelae such as glaucoma, cataracts, and retinal detachment or degeneration occur. For these reasons, clinicians must maintain a high index of suspicion regarding uveitis in all cats with ocular disease and even those with nonspecific signs such as lethargy, "hiding", anorexia, or fever.

Uveitis, like inflammation elsewhere, is evident as one or a combination of the 5 cardinal signs of inflammation: heat, pain, swelling, redness, and loss of function:

- Intraocular pain: blepharospasm or epiphora; however, cats seem more likely to show subtle and less localizing signs such as lethargy or anorexia
- Iridal swelling requires that the eye is examined using a source of magnification (such as the Optivisor®) in association with a bright and focal light source (such as the Finoff transilluminator®) directed very obliquely across the globe. Look for a loss of the normal "texture".
- Redness (scleral injection) can be particularly subtle in many cats.
- Dysfunction: Loss of function manifests as breakdown of the blood aqueous barrier (BAB), miosis, corneal edema, and hypotony. Of these, BAB breakdown is pathognomonic. In particular look for hypopyon (white blood cells), hyphema (red blood cells), and fibrin, aqueous flare (albumin and other small proteins) and keratic precipitates (white blood cells and inflammatory proteins clumped against the corneal endothelial surface).

Chronic uveitis and its sequelae are evident as glaucoma (due to scarring or clogging of the iridocorneal angle), anterior or posterior synechia (adhesions between the iris and corneal endothelium or lens, respectively), phthisis (globe contracture) or retinal detachment (due to contraction of vitreous fibrin). Altered aqueous humor composition and circulation also causes a relative malnutrition of the lens (evident as cataract) and inner cornea (evident as corneal edema, vascularization, fibrosis etc.). Lens luxation may occur due to enzymatic lysis or phagocytosis of the lens zonules, secondary to cataract development, or as a sequela to buphthalmos due to secondary glaucoma. Neovascularization of the face of the iris (rubeosis iridis) is a pathognomonic sign of subacute or chronic active uveitis. It is one of the signs that is seen more easily in cats than in dogs due to the typically lighter iris color of cats.

DIAGNOSIS

What is a reasonable diagnostic approach to a patient with uveitis?

Having a high degree of clinical suspicion and performing a targeted clinical examination with appropriate ocular diagnostic testing (especially checking for aqueous flare, testing intraocular pressure (IOP), and assessing for miosis) will ensure that uveitis is diagnosed when present. However, detecting uveitis is the beginning of the diagnostic process; not the end. Confirming or eliminating all suspected etiological diagnoses is the essential next step. By conducting an excellent general physical and ophthalmic exam as well as gathering a focused history, the initial goal is to establish whether further diagnostic testing is strongly supported. I do this by categorizing the uveitis as present in a well or systemically ill patient, unilateral or bilateral; exogenous or endogenous; acute or chronic; and as involving the anterior uvea, choroid, or both. An etiologic diagnosis should then be pursued through a diagnostic workup identical to that employed for a cat with lymphadenopathy. Consider CBC, Biochemistry, urinalysis, serology, chest and abdominal imaging, etc. as appropriate for the following agents.

ETIOLOGY

The known causes of endogenous anterior uveitis in cats are expanding but still too few to explain the majority (~ 70%) of cases.

INFECTIOUS AGENTS AS A CAUSE OF UVEITIS

Viral	Bacterial	Parasitic	Fungal/Algal	Protozoal
• FIP • FeLV* • FIV • FHV • CDV • CAV	• Bartonella spp. • Mycobacterium spp. • Ehrlichia spp.¥ • Borrelia burgdorferi¥ • Brucella • Leptospira	• Cuterebra • Larval migrans	• Cryptococcus neoformans† • Histoplasma capsulatum† • Blastomyces dermatitidis† • Candida albicans • Coccidioides immitis† • Aspergillus spp.	• Toxoplasma gondii • Leishmania spp.

** Via immunosuppression or oncogenesis*

† Chorioretinitis predominates

¥ Seroprevalence data only; no clinical evidence

TREATMENT

Treatment of anterior uveitis must be tailored to the individual case based on proven or suspected cause, severity, anatomical location, chronicity, and presence of systemic or other intraocular disease. Regardless, some general therapeutic guidelines are possible. Optimal treatment relies upon identification and removal or reduction of the causative antigen; however, this is rarely possible. Additionally, all patients with uveitis need their intraocular inflammation controlled rapidly and completely, since it is painful and produces vision-threatening sequelae. Thus, immunomodulating drugs form the mainstay of therapy for uveitis. The major decisions are therefore which immunomodulating drugs should be given, via what route and, at what dose.

IMMUNOMODULATORY THERAPY

Corticosteroids are highly potent, available in topical or systemic forms, relatively inexpensive, generally well tolerated by cats, and can be administered at anti-inflammatory or immunosuppressive dosages. For these reasons, they are commonly used for uveitis. Their systemic use should be reserved until a definitive cause responsive to corticosteroids has been found or, failing this, until causes known to be worsened by glucocorticoids have been adequately eliminated. In particular, the systemic mycoses must be adequately eliminated as potential causes. Likewise, patients in which lymphoma is possible and which would benefit from a multidrug chemotherapeutic regimen should not be treated with systemic corticosteroids alone. By contrast, topical administration of corticosteroids may be employed safely even when an infectious or neoplastic cause might prevent systemic administration of the same drugs. This is possible because systemic effects are insignificant with short-term topical application. It is possible that topically administered corticosteroids may alter cytologic findings and so, if safe, their use should perhaps be delayed until after ocular centesis is performed. Topical corticosteroids should never be used in the face of corneal ulceration because they can be associated with rapid worsening of the ulcer due to superinfection, collagenolysis, local immunosuppression, and delayed wound healing. Prednisolone acetate (1% or 0.125%) and dexamethasone (0.1%) will penetrate intact corneal epithelium and reach the anterior uveal tract. Hydrocortisone (as found in many combined antibiotic–corticosteroid ophthalmic preparations) does not penetrate intraocularly and should not be used. The frequency of application should be tailored to the severity of the uveitis; starting as frequently as q 2 hours and tapering as a clinical response is noted. When safe, corticosteroids should be administered systemically for posterior uveitis and when more significant immunomodulation is necessary, or when corneal ulceration prohibits their topical use. Typical doses of prednisolone range from 1 mg/kg q 12 hours when notable inflammation is present to 0.5 mg/kg once daily when a more moderate anti-inflammatory effect is desired. As with topical corticosteroids, dose and dose frequency of systemically administered glucocorticoids should be carefully reduced based entirely upon clinical evidence of waning disease. In cats with acute or subacute uveitis, this can be fairly rapid. Animals with chronic idiopathic (immune-mediated) uveitis require slow tapering (perhaps a halving of dose or dose frequency every 2-3 weeks), with the expectation that inflammation may return below a critical dose. In these patients, returning to the previously effective dose will be necessary. Some cats will suffer herpetic recrudescence when receiving corticosteroids, regardless of route.

Compared with corticosteroids, non-steroidal anti-inflammatory drugs (NSAIDs) are not immunosuppressive, and may be more expensive, sold in smaller volumes (as topical ophthalmic solutions), and sometimes unavailable in ointment form. These limitations must be borne in mind for cats with uveitis; however, they may be preferred over corticosteroids in patients with diabetes or other endocrinopathies in which corticosteroid use may not be wise. They can also be administered systemically instead of corticosteroids when systemic infectious disease is suspected or proven, or until lymphosarcoma is eliminated as a differential consideration. And they may be given in conjunction with a topical steroid. As such, they may make an excellent choice for initial control of inflammation while likely causes are being ruled in or out. The same general comments regarding dose frequency and route made for corticosteroids apply equally to NSAIDs.

IRIDOCYCLOPLEGIC AGENTS

Parasympatholytic drugs such as atropine have multiple favorable actions in eyes with uveitis and form a critical component of treatment. These drugs paralyze the parasympathetically innervated iris sphincter and ciliary body muscles causing mydriasis and cycloplegia,

respectively. The effects of pupil dilation are numerous and important. Pupil dilation reduces leakage of vascular elements into the aqueous humor by causing radial blood vessels within the iris stroma to "concertina" upon themselves (thus providing a physiological tamponade); decreasing iris surface area (from which inflammatory mediators and vascular components originate); reducing uveal vascular endothelial permeability; and by reducing chances and consequences of posterior synechiation. However, "bunching" of the iris in the periphery does increase the chance of anterior synechia and potentially obstruction of the iridocorneal angle. Cycloplegia reduces ocular pain but also increases resistance to aqueous outflow. Therefore, pupil dilation and cycloplegia are desirable in all cases of uveitis except those where secondary glaucoma is present or likely. The effect of mydriasis upon IOP can be tested by a single application of the short acting drug tropicamide followed by tonometry when the pupil is fully dilated. If IOP is increased by tropicamide, atropine should not be administered. If atropine is initiated, IOP should be rechecked regularly and application discontinued if IOP increases above normal. In cats, atropine should be applied as an ophthalmic ointment rather than a solution because it is bitter and passage down the nasolacrimal duct can cause violent salivation and frothing that is harmless but disturbing to the cat and its owner. Atropine should be applied to effect. Since cycloplegia cannot be observed, the pupil is used for monitoring dose. Depending on the severity of uveitis, once to twice daily application for the first day or two may be needed to open the pupil. Subsequently, once to twice weekly application will often keep the pupil mydriatic. Posterior synechia will not be resolved with atropine and will prohibit use of pupil size as an indicator of drug efficacy. However, atropine should still be administered to patients with synechia since the analgesia resulting from cycloplegia should not be affected and is still desirable.

MONITORING AND SEQUELAE

Prompt specific treatment of uveitis with tapering of therapy based upon reduction of clinical signs may result in some sequelae but these are usually mild and should be static. If they are not, this suggests chronic or recurrent uveitis and further investigations and treatment are necessary. Classic sequelae include corneal fibrosis, cataract, or posterior synechia. None of these changes should result in pain or, unless severe, vision disturbance. By contrast, more severe, unrecognized, persistent, or recurrent uveitis frequently results in a blind and sometimes painful globe. The most common sequelae (and their prevalence in cats) include cataracts in 20-36% of eyes, lens luxation in 11-18%, glaucoma in 16-46% and enucleation in 29%. Many patients experience more than one of these sequelae. For these reasons, frequent and careful monitoring of a patient with uveitis is essential. This should be performed as for patients with immune-mediated disease elsewhere with gradual tapering of medications and re-examination at doubling intervals presuming there is improvement; more often if there is not. Re-examination and tapering of medications should be continued until there is complete resolution of every clinical sign of active uveitis. I believe that tonometry is the most sensitive test with which to monitor uveitis during treatment because subtle hypotony (sometimes only relative to the contralateral eye) can continue long after other more overt signs have normalized. Continued treatment of these patients may prevent or delay development of sight-threatening ocular complications.

REFERENCES

References available from the author on request.

8001

DOWN DOGS: NSAIDS, STEROIDS, SURGERY?

PRACTICE PEARLS

Chris Mariani, DVM, PhD, DACVIM

INTRODUCTION

The first consideration when dealing with a dog (or cat!) that cannot walk or is having substantial difficulties ambulating is to sort out which body system is responsible. The main considerations are the nervous and musculoskeletal systems although other etiologies are possible (e.g., certain metabolic conditions such as Addison's disease or hypoglycemia, hemoabdomen). Thus, a full physical examination followed potentially by neurological and orthopaedic examinations is indicated. Key parts of the exam to consider are gait evaluation, postural reactions, segmental spinal reflex evaluation, spinal palpation, and bone and joint palpation. For more details on these considerations, please see the accompanying proceedings entitled "Gait Exam: Underutilized but Critically Important" and "Is it Orthopaedic or Neurologic? Sorting out Lameness, Paresis and Dogs that Won't Get Up".

DEFINITIONS

Paresis is strictly defined as incomplete voluntary movement; it presents as weakness in the limbs due to a neurologic cause and can be quite variable in severity (ambulatory to non-ambulatory). Acute paraplegia is a complete loss of voluntary movement; that is, a sudden onset of paralysis in the pelvic limbs and is a common problem in small animal patients. Ataxia is incoordination and typically presents as crossing of limbs, drifting, stumbling or falling during attempts to ambulate. Lameness can have several definitions but the most useful is incomplete bearing of weight on one or more limbs.

ETIOLOGIES AND DIFFERENTIAL DIAGNOSIS

Intervertebral disk disease (IVDD) is responsible for the majority of cases presenting to the small animal practitioner with acute paraplegia or nonambulatory tetraparesis or paraparesis. However, it is important to bear in mind that other etiologies are possible, and an appropriate differential diagnosis list should be developed. Other disease mechanisms to consider are trauma, meningomyelitis, neoplastic disease, vascular myelopathies and acute neuromuscular disorders.

Trauma is usually obvious based on the history and physical examination findings and potential trauma patients should be handled very carefully and preferably restrained until radiography can be used to assess the extent of their injuries. Meningomyelitis often presents with multifocal signs affecting the thoracic limbs and brain as well as the pelvic limbs, but focal thoracolumbar myelopathies can also be seen. Neoplasia is typically more chronic and insidious in onset, but can present acutely, sometimes in association with sudden hemorrhage or pathologic fractures. Vascular diseases, such as fibrocartilaginous embolism (FCE), are a common differential for IVDD in many cases, as they have an acute onset and are often asymmetrical. However, in contrast to IVDD, these conditions are non-painful. Diskospondylitis typically presents as severe spinal pain, although some animals may present with paresis. Some neuromuscular conditions (e.g., acute polyradiculoneuritis, tick paralysis, botulism) may have an acute presentation. These disorders usually progress to involve all four limbs and are characterized by flaccidity in association with reduced or absent segmental spinal reflexes. Myasthenia gravis is another neuromuscular condition that can present acutely although it is easily differentiated from myelopathies with a careful neurological examination.

Finally, a number of non-neurological conditions may cause an animal to be presented for inability to ambulate. These include long bone fractures, severe osteoarthritis, bilateral cranial cruciate ligament rupture, and polyarthritis or other acute orthopaedic injuries. These conditions

are differentiated from spinal cord disease by thorough physical, neurologic and orthopaedic examinations.

NEUROLOGIC EXAMINATION

The hallmarks of spinal cord disease are ataxia, paresis/plegia and postural reaction deficits (hopping, proprioceptive placing). Patients with orthopedic or other non-neurologic conditions should not display these abnormalities. Animals with spinal cord lesions caudal to the T2 level can almost always pull themselves around with their front legs. If the animal is laterally recumbent or cannot use its thoracic limbs in this manner, a lesion in the C1-T2 region should be considered. This gait assessment, together with evaluation of segmental spinal reflexes should allow the practitioner to put the lesion at one of four spinal cord levels: C1-5, C6-T2, T3-L3 or L4-S2. This is important when considering diagnostic testing and potential etiologies. As mentioned above, animals with acute neuromuscular conditions typically show reduced or absent reflexes in all four limbs along with flaccid paralysis (except for myasthenia gravis).

Deep pain perception, assessed by squeezing on the bone of one of the digits of an affected limb with hemostats or between the handle of a boxed instrument, is a crucial part of the examination in animals that are plegic, as it impacts the prognosis and potential time to recovery of the patient. The appropriate response is cerebral recognition of the pain, characterized by crying out, turning to look at the limb or trying to bite the examiner. Withdrawal of the limb is a reflex only and does not indicate pain perception.

DIAGNOSTIC PLAN

Diagnostic testing typically revolves around imaging tests. Survey spinal radiographs are indicated to investigate traumatic processes (fractures, luxations), diskospondylitis or neoplastic conditions affecting the vertebrae. If the lesion is localized to the T3-L3 spinal segment, it is important to image the entire thoracic and lumbar spine to avoid missing potential lesions outside of the expected location for IVDD (i.e. T11-L4). Although calcified disks in situ are often seen on survey radiographs, and a narrowing of the intervertebral disk space and increased density within the intervertebral foramen are suggestive of IVDD, additional diagnostic imaging is typically required before definitive therapy.

Specialized diagnostic imaging of the spinal cord may involve myelography, computed tomography (CT), magnetic resonance imaging (MRI) or combinations of these modalities. Cerebrospinal fluid (CSF) analysis is a useful test to investigate meningomyelitis. Electrodiagnostic testing (e.g., electromyography [EMG], motor nerve conduction velocity) is useful in documenting and defining neuromuscular lesions.

TREATMENT PLAN

The main decision point when considering therapeutic options is whether to pursue referral to a specialty center, which is typically required to pursue advanced imaging, CSF analysis and potentially spinal surgery (if indicated). The driving factors contributing to this decision are usually the severity of the neurological dysfunction and the financial means and motivations of the owner. Radiographs of the spine should almost always be performed, if possible, for the reasons mentioned above. If lytic lesions of the vertebrae (diskospondylitis, neoplasia) or traumatic lesions are found, then additional diagnostics or appropriate therapy for these conditions can be initiated. However, often survey radiographs are unremarkable or show changes suggestive of IVDD and the clinician has to decide to treat an animal with a more conservative medical approach versus initiating a referral for additional diagnostics and potentially surgical intervention.

Animals with spinal pain but without neurological deficits can usually be successfully managed conservatively. This therapy is undertaken with the assumption that you are managing a patient with IVDD. Such therapy includes strict cage or crate confinement for 4 weeks, analgesics, muscle relaxants and potentially sedatives. Controlled physiotherapy exercises can be considered but unrestricted activity either outside or in the house often leads to therapeutic failure. Potential analgesics include NSAIDs (see below), oral opioids (generally not preferred) and gabapentin (or pregabalin). Muscle relaxants contribute to analgesia and include diazepam or methocarbamol. Trazadone is usually the sedative of choice, if needed.

Acutely paraplegic (or rarely tetraplegic) animals should be considered an emergency situation and urgent referral for advanced imaging and potentially surgical intervention to decompress the spinal cord is ideal. This is

particularly the case for animals with a loss of nociception (deep pain). The space between these two presentations (just spinal pain vs. paraplegia) is a bit fuzzier, although a good rule of thumb is that nonambulatory animals are better off being referred, while ambulatory tetraparetic or paraparetic dogs can often be successfully managed with a conservative medical strategy.

NSAID OR STEROID?

A common dilemma is whether to initiate therapy with an NSAID or a corticosteroid. In the author's opinion, NSAIDs are usually the better option in this scenario for several reasons. Extruded nucleus pulposus disk material creates an inflammatory reaction in the epidural space, irritating meninges and nerve roots and causing pain and muscle spasm. Both classes of medication provide anti-inflammatory effects, which can mitigate these sequelae. However, NSAIDs also provide additional analgesic effects. Corticosteroids frequently lead to polydipsia and resultant polyuria, which can complicate management in animals unable to urinate (typically lost when the ability to voluntarily move the legs is lost). These drugs can also cause immune suppression and predispose animals with urine retention to urinary tract infections. Importantly for animals that might be referred either immediately or later if the disease progresses, corticosteroids can complicate specialized diagnostic testing, particularly CSF analysis but also enhancement patterns after contrast administration when performing advanced imaging.

Some clinicians have concerns that if corticosteroids become indicated in the future (e.g., in a dog with meningomyelitis), switching from an NSAID to a corticosteroid will require a washout period. However, a washout period is not based in any sort of objective evidence and these drugs can be exchanged without serious concerns for adverse reactions as long as both are not administered concurrently. As a final note, older recommendations of high-dose intravenous corticosteroid therapy (e.g., methylprednisolone sodium succinate, prednisolone sodium succinate or dexamethasone) are no longer recommended for acute disk herniations or traumatic myelopathies.

8002

DOGS ON THE MOVE: A NEED-TO-KNOW ON CANINE IMPORTATION

PRACTICE PEARLS

Maureen Anderson, DVM, DVSc, PhD, DACVIM

RECENT CHANGES TO FEDERAL DOG IMPORT REGULATIONS REGARDING RABIES

- **Phase 1 (May 2021):** The Canadian Food Inspection Agency (CFIA) implemented additional requirements for importation of commercial dogs less than 8 months of age (the only class of dogs for which an import permit was and continues to be required).
    - Commercial dogs are those intended to be transferred/given to another person (including rescue dogs) or intended for commercial purposes such as breeding or sale of offspring, showing/exhibition, scientific use/research or special training status (regardless of whether a profit is made or a transfer of funds occurs).
- **Phase 2 (September 2022):** A temporary prohibition (until further notice) on importation of commercial dogs (of any age) from countries considered high-risk for canine rabies at all international airports in Canada.
    - This is similar to the ban that was implemented by the US in July 2021, but the Canadian ban does not currently apply to personal dogs (including service animals)
- **Phase 3 (TBD):** The CFIA has indicated that they are continuing to explore options for long-term changes to dog importation requirements, including those for personal dogs and assistance dogs from countries at high-risk for canine rabies, in order to protect Canadians and their pets and to reduce the risk of dog rabies entering Canada.

DOGS TRAVELLING TO CANADA

Requirements are the same for personal dogs coming to Canada from any country that is not considered free of non-bat variant rabies, regardless of whether it is high-risk or low-risk for canine variant rabies:

- Valid rabies vaccination certificate
    - OR a rabies country-freedom certificate if the dog has only been in a rabies-free country for the last 6 months or since birth
    - OR proof of dog's age (if less than 3 months)
- Appears healthy and meets humane transportation requirements

If NOT accompanied by the owner, personal dogs less than 8 months of age also require:

- Valid veterinary certificate of health

Commercial dogs over 8 months of age from low-risk rabies countries must fulfill the same requirements as personal dogs.

Commercial dogs less than 8 months of age from low-risk rabies countries must fulfill the updated requirements put in place in May 2021, including:

- Import permit, which requires:
    - Kennel of origin certification
    - Rabies vaccination no earlier than 3 months of age and at least 28 days prior to import (so all dogs must be at least 16 weeks of age at time of import, with limited exceptions for recognized breeders in Canada)

- Vaccination for distemper, hepatitis, parvo virus and parainfluenza no earlier than 6 weeks of age
- Treatment for internal and external parasites
- ISO approved microchip
- A scheduled inspection prior to the shipment leaving the country of origin
- For dogs transported by air, availability of a CFIA pre-approved post-import quarantine facility

- Valid veterinary certificate of health

Commercial dogs of any age from countries considered high-risk for canine variant rabies are currently prohibited from entering Canada (by air) entirely.

ONTARIO RABIES REGULATIONS

Remember that Ontario is the only jurisdiction in Canada with a legal requirement for rabies vaccination for domestic animals (including all dogs, cats and ferrets over 3 months of age). Based on the most recent updates to HPPA Regulation 567 (July 2023):

- Rabies vaccination may be carried out by veterinarians practicing in either Canada or the USA, or by the lawfully authorized delegate of such a veterinarian (e.g. an RVT)
- Rabies vaccines approved for use in the jurisdiction where it's given (in either Canada or the USA) are accepted, but the vaccination certificate must include all of the required information listed in Regulation 567

Rabies antibody titres (RAT) are not accepted as proof of adequate immunization in Ontario. Remember that there is in fact no antibody level that has ever been demonstrated to be "protective" against rabies, because humoral immunity is only one component of the protective immunity provided by vaccination. The internationally accepted RAT of 0.5 IU/mL is considered demonstrative of a response to vaccination for the purposes of international travel. Only vaccination according to the intervals specified on the rabies vaccine product label have been shown by the manufacturer to protect the majority of animals from disease challenge.

- If there is a valid medical contraindication to vaccinating an animal for rabies in Ontario, the reason must be clearly documented in the medical record, and a certificate of exemption issued by the veterinarian, containing the same basic animal / owner information as a vaccination certificate, and specifying the duration of the exemption.

DOGS TRAVELLING TO THE US FROM CANADA (AND BACK)

It has been a rollercoaster ride over the summer of 2024 trying to determine what the new rules will be for dogs travelling to the US from different countries, including Canada. The first version of the rules was announced in May, followed by a number of changes in June and July ahead of the August 1 effective date. These included some detailed record requirements aimed at proving where the dog had been living for the 6 months prior to entering the US. Canadian authorities promptly shared numerous concerns about the new requirements with their US counterparts. Then on July 22, the US CDC announced they were dramatically reducing the requirements for dogs coming from low-risk countries for canine rabies, like Canada. Here's what stuck.

All dogs travelling from Canada to the US must:

- Be at least 6 months old
- Have a microchip (readable by a universal scanner)
- Appear healthy on arrival
- Have a CDC Dog Import Form receipt (which is good for multiple entries for 6 months, as long as the dog does not travel outside the US and Canada)

Dogs must also not have visited a high-risk country for canine rabies in the 6 months prior to travel to the US, but currently there is no documentation required for this, only the attestation of the dog's importer. The requirement for a current rabies vaccination certificate for dogs coming from Canada has been dropped – for now. But the US CDC still strongly recommends vaccination against rabies for dogs visiting the US, and remember that dogs returning to Canada will still require proof of current rabies vaccination, even if they have only visited the US. Individual US states may have separate requirements for rabies vaccination; visit www.rabiesaware.org for state-specific rabies vaccination laws/regulations.

Canada's Minister of Health (Hon. Mark Holland) confirmed in July that the US has agreed to a nine-month grace period (until May 2025) while Canada and the US find a workable, permanent solution that will minimize disruption to Americans and Canadians crossing the border with their dogs. The CVMA has advocated for the involvement of both a CVMA and AVMA representative in future negotiations.

DOGS TRAVELLING TO THE US FROM A HIGH-RISK COUNTRY VIA CANADA

Dogs from high-risk countries cannot transit through Canada to the US – they must arrive directly from their country of origin and fulfill all the updated CDC requirements for such dogs. If dogs arrive in Canada from these countries, they must remain in Canada for at least 6 months prior to crossing the US border.

Rules for dogs travelling to the US directly from high-risk countries depend on whether the dog was vaccinated in the US prior to travel, or if the dog was vaccinated abroad.

A QUICK NOTE ABOUT CATS

Cats travelling to the US are subject to inspection and can be denied entry if there is evidence of infection with a disease of public health concern. If a cat appears ill, examination by a licensed veterinarian at the owner's expense might be required before entry is permitted. The US CDC does not require cats to have proof of rabies vaccination for importation, but does recommend vaccination. Many states and territories have their own rabies vaccination requirements for cats.

Cats over 3 months of age being imported into Canada require a valid rabies vaccination certificate (or a veterinary certificate if arriving from a rabies-free country), and must appear healthy at the time of entry.

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Canadian Food Inspection Agency:

- *Rabies cases in Canada (by month, species and province/territory): https://inspection.canada.ca/animal-health/terrestrial- animals/diseases/reportable/rabies/in-canada/eng/1356156989919/1356157139999*
- *Ask questions before you buy or adopt a dog: https://inspection.canada.ca/en/importing-food-plants-animals/pets/ask-questions-you- get-dog*
- *Bringing animals to Canada: Importing and travelling with pet https://inspection.canada.ca/en/importing-food-plants-animals/pets*
- *Dogs travelling to the United States: https://inspection.canada.ca/en/animal-health/terrestrial-animals/exports/pets/dogs-usa*

Ontario Health Protection and Promotion Act (R.R.O 1990) regulations:

- *Rabies immunization (Reg. 567) https://www.ontario.ca/laws/regulation/900567*

US Centers for Disease Control

- *Entry Requirements for Dogs from Dog-Rabies Free or Low-Risk Countries https://www.cdc.gov/importation/dogs/rabies-free-low-risk-countries.html*
- *Entry Requirements for US-Vaccinated Dogs from High-Risk Countries https://www.cdc.gov/importation/dogs/us-vaccinated-high-risk-countries.html*
- *Entry Requirements for Foreign-Vaccinated Dogs from High-Risk Countries https://www.cdc.gov/importation/dogs/foreign-vaccinated-high-risk-countries.html*

United States Department of Agriculture – Animal and Plant Health Inspection Service

- *Bring a Pet From Another Country into the United States (Import) https://www.aphis.usda.gov/pet-travel/another-country-to-us-import*

Ontario Animal Health Network:

- *Need-2-Know: Rabies in Pets (whiteboard video for pet owners) https://www.oahn.ca/resources/video-need-to-know-rabies-in-pets/*
- *Rabies titres in imported dogs (companion animal network research project) https://www.oahn.ca/resources/oahn-companion-animal-research-project-rabies-titres-in-imported-dogs/*
- *Rabies resource page for veterinarians (OAHN login required) https://www.oahn.ca/resources/rabies-resource-page-for-veterinarians/*

Rabies – Ontario.ca website

- *Information for veterinarians: https://www.ontario.ca/page/rabies-information-veterinarians*
- *Wildlife rabies outbreaks and control operations (including interactive case map): https://www.ontario.ca/page/wildlife-rabies-outbreaks-and-control-operations*
- *Rabies cases (including annual case maps and positive cases by region, including bats): https://www.ontario.ca/page/rabies-cases*

8003

CENTRAL VS. PERIPHERAL VESTIBULAR DISEASE

PRACTICE PEARLS

Andrew Barker, DVM, DACVIM

BACKGROUND

Vestibular conditions are relatively common in veterinary medicine with a prevalence of ~0.1% of cases in general practice (Radulescu et al. 2020) and is the basis for ~ 1/3 MRI cases in referral practice (Boudreau et al 2018)

VESTIBULAR DISEASE LESION LOCALISATION

1. Central Vestibular Disease (CVD)– Involving the brainstem and/or cerebellum
2. Peripheral Vestibular Disease (PVD)– Involving structures external to the brain (vestibulocochlear nerve, middle and/or inner ear) (Rossmeisl 2010)

CLINICAL SIGNS THAT INDICATE CENTRAL DISEASE – AS TAUGHT IN SCHOOL

1. Mentation Changes
2. Placing Deficits
3. Involvement of cranial nerves Other than VII and Horner syndrome
4. Presence of Vertical Nystagmus

IS THIS TRUE?

1. Mentation Changes – Yes but can be very difficult to differentiate between changes due to involvement of the Ascending Reticular Activating System (ARAS) and signs of general illness
2. Placing Deficits – Yes but can be difficult to test in a "rolling dog"
3. Involvement of Cranial Nerves Other than VII and/or Horner syndrome - Yes
4. Presence of Vertical Nystagmus – No (Troxel et al. 2005).

HOW ACCURATE IS THE NEUROLOGICAL EXAMINATION FOR DIFFERENTIATING? – PRETTY GOOD ACTUALLY

1. Central Vestibular Disease ~ 98% accuracy
2. Peripheral Vestibular Disease ~ 77% accuracy (Bongartz et al. 2020)

WHY DIFFERENTIATION MAY BE USEFUL

Differentials

- Central – Neoplasia
  - Meningoencephalitis
  - Cerebrovascular Event (Harrison et al. 2021)
- Peripheral – Otitis Media/ Interna
  - Idiopathic/ Geriatric Vestibular Disease (Harrison et al. 2021)

Diagnostics

- Otoscope
- MRI vs CT

Therapy

- Antibiotics – and which ones
- Corticosteroids
- None

Recommendations from the Presenter

Differentiating CVD and PVD can be challenging in the clinical setting but is beneficial in regards to forming a proper differential list and diagnostic/ therapeutic plan.

Placing deficits and the presence of cranial nerve deficits other than the facial nerve and Horner syndrome are the most reliable.

Approximately 25% of all suspected peripheral vestibular conditions based on a neurologic examination alone are actually central so it is important to keep an open mind as the case progresses.

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8004

LABWORK ABNORMALITIES – BREED DOES MATTER

PRACTICE PEARLS

Jinelle Webb, DVM, MSc, DVSc, Diplomate ACVIM (SAIM)
Stipe Vicente Jelovcic, BSc, DVM, PGC

INTRODUCTION

The interpretation of lab work results can guide clinicians in diagnosing a disease process, supporting additional diagnostic steps, and/or aiding in treatment strategies. However, the assessment of lab work can be complicated by breed specific alterations to these parameters. Healthy cats and dogs within certain breeds have reference intervals of specific parameters that differ from those published for all breeds. The ability to accurately interpret lab work for these breeds requires knowledge of the breed specific anomalies. Without knowledge of these breed specific changes, additional diagnostic testing may be recommended that may not be indicated. However, it is also important to consider the patient in these scenarios, as these breed specific alterations should not result in clinical signs. Therefore, additional diagnostic testing may be indicated in patients with clinical signs.

ALTERATIONS IN COMPLETE BLOOD CELL COUNT PARAMETERS

Parameter	Below reference range	Above reference range
Red blood cell count	Maine Coone cat	Sighthound, Miniature Dachshund
Mean corpuscular volume	Shiba Inu, Akita, Hokkaido	Sighthound, Miniature and Toy Poodle
White blood cell and neutrophil counts	Sighthound	
Platelet count	Cavalier King Charles Spaniel, Cairn Terrier, Norfolk Terrier, Jack Russell Terrier, Bichon Frise, Miniature Schnauzer, Boxer, Havanese, Shih Tzu	

ABNORMALITIES IN WHITE BLOOD CELL APPEARANCE

Parameter	Abnormality	Breed
Granulated white blood cells	Pelger-Huët anomaly (hypo-segmentation of neutrophils, eosinophils and basophils)	Multiple breeds, although most common in the Australian Shepherd
Grey eosinophils	Eosinophils that contain greyish cytoplasm and vacuolation (can appear as toxic neutrophils)	Greyhound
Reddish granulation of neutrophils	Neutrophils that contain red granules (can appear as toxic)	Birman cat

ALTERATIONS IN SERUM BIOCHEMISTRY PARAMETERS

Parameter	Above reference range
ALT	Greyhound, Italian Greyhound, Whippet, Ariégeois, Bleu de Gascogne, Bracco Italiano, Segugio Italiano
AST	Dogue de Bordeaux, Greyhound, North American Scottish Deerhound, Bleu de Gascogne, Segugio Italiano
ALP	Bernese Mountain Dog, North American Scottish Deerhound, Maine Coon Cat, Norwegian Forest Cat
GGT	Bernese Mountain Dog, Maine Coon Cat
Urea	Yorkshire Terrier, Maine Coon Cat, Birman cat
Creatinine	Greyhound, Birman cat
Thyroid hormones	Greyhound, North American Scottish Deerhound, Dogue de Bordeaux, Saluki, Sloughi

ALTERATIONS IN SERUM BIOCHEMISTRY PARAMETERS

Parameter	Below reference range	Above reference range
Cholesterol	Miniature Dachshund, Shiba Inu, Birman cat	Bernese Mountain Dog, Dogue de Bordeaux, North American Scottish Deerhound, Newfoundland Dog, Shetland Sheepdog, Miniature Schnauzer
Glucose	German Shepherd puppies less than 8 weeks old, adult North American Scottish Deerhound, Ragdoll Cat	Shetland Sheepdog, Birman cat
Serum protein	Greyhound, other Sighthounds	Miniature Schnauzer, Maine Coon Cat, Birman cat, Abyssinian cat, Siberian cat
Sodium	Pug	Greyhound
Chloride	Pug, North American Scottish Deerhound	Greyhound
Potassium		Akita, Shiba Inu, Chinese-Shar Pei, other East Asian dog breeds
Calcium		Greyhound, North American Scottish deerhound, other Sighthounds, Norwegian Forest Cat
Phosphorus	Whippet, Borzoi, Italian Greyhound, Greyhound	Norwegian Forest Cat
Magnesium		Brachycephalic breeds
Creatine Kinase	Ragdoll Cat	Greyhound, other Sighthounds

FELINE COAGULATION

An important coagulation parameter alteration is a markedly prolonged aPTT and ACT in some cats. This is due to a factor XII deficiency (Hageman factor) in certain cats, such as Himalayan, Siamese, DLH and DSH Cats. However, factor XII deficiency is not associated with increased bleeding risk. This is an important normal variation to be aware of in cats, as it could lead to further diagnostic testing and/or unnecessarily postponing or cancelling procedures with the perceived potential for hemorrhage.

CONCLUSION

When interpreting lab work in a healthy feline or canine patient, the patient's breed can be a potential factor contributing to abnormal values, and must be considered, especially before initiating invasive and/or advanced diagnostic testing. Along with the importance of breed-specific variations in reference range, sample handling, sample type (i.e., plasma vs. serum), sample timing with respect to feeding/exercise, and the patient's muscle condition score when analyzing lab work can be influencing factors. As more data becomes available regarding breed specific variations in reference range for individual parameters, it becomes increasingly important for this information to be disseminated, and ideally included in reported lab work. The ability to integrate this information into lab work interpretation relies on both the pet owner and clinician accurately reporting a patient's breed.

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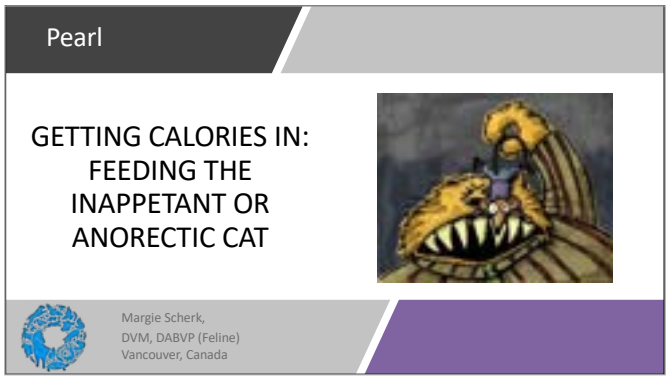
References provided upon request.

8005

GETTING CALORIES IN: FEEDING THE INAPPETANT OR ANORECTIC CAT

PRACTICE PEARLS

Margie Scherk, DVM, DABVP



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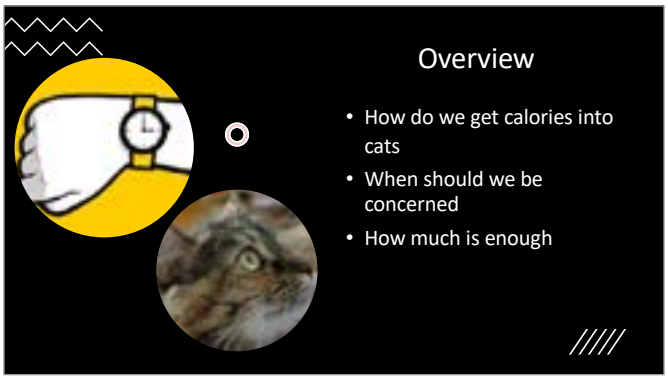

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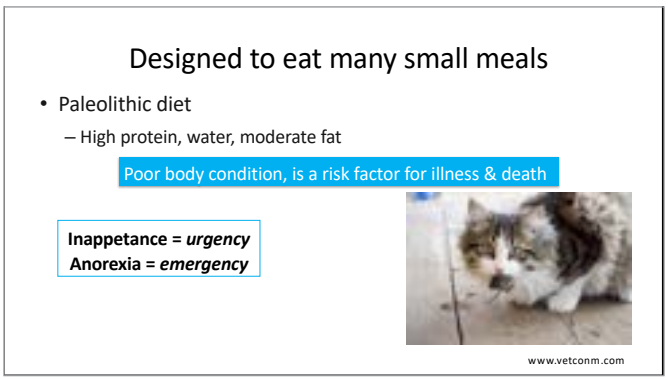

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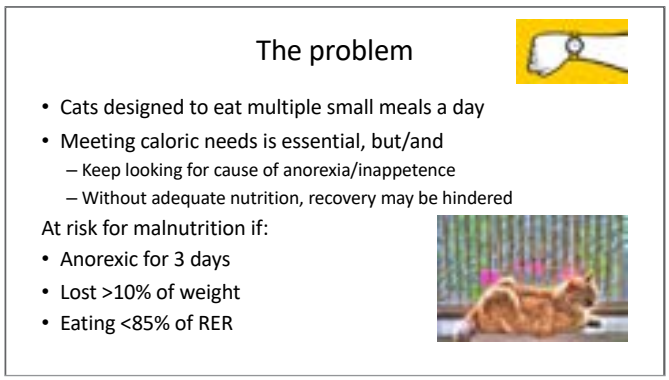

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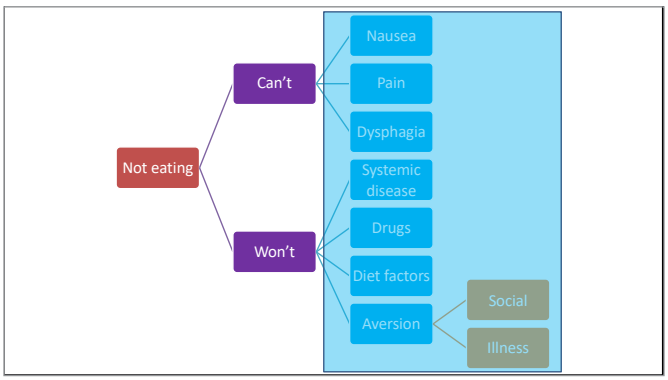

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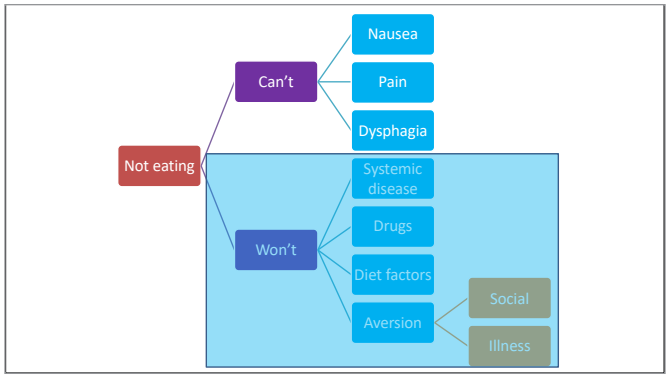

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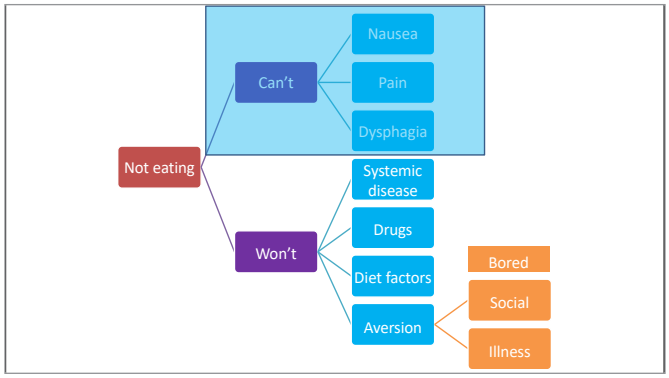

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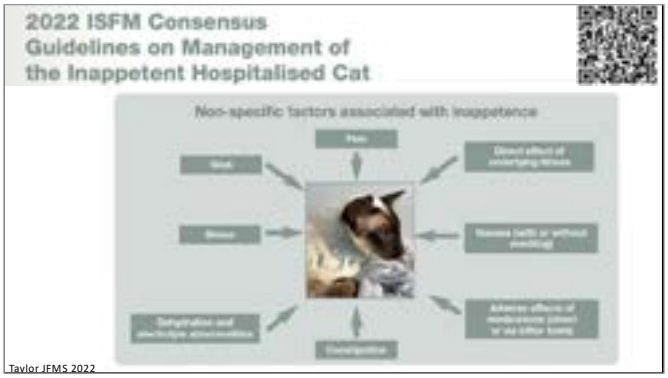

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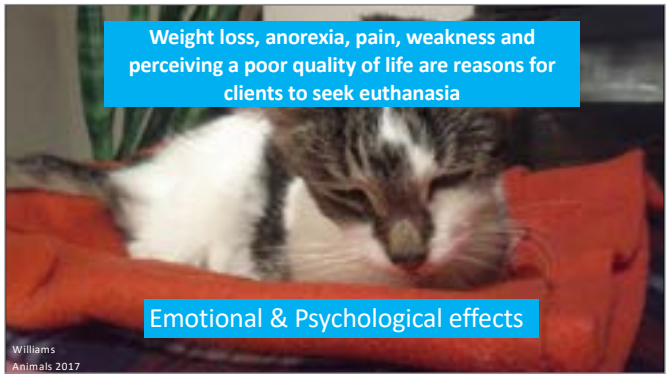

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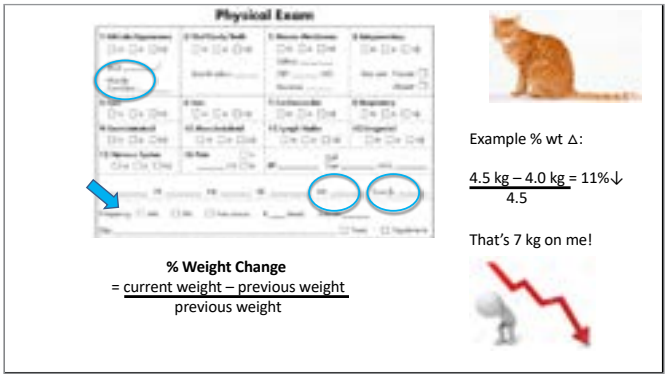

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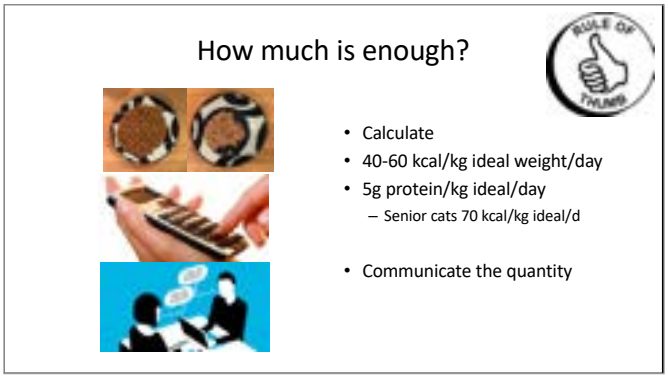

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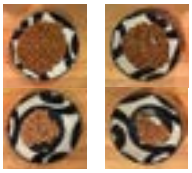
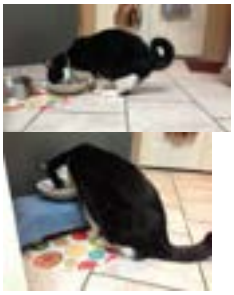
Why is she not eating enough



- Illness?
- Palatability?
- Nausea?
- Vomiting?
- Pain?
- Anxiety?
- Difficulty accessing food

15



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How is dehydration like a hangover?

- Hangover = dehydration + acidosis + oxidative stress + inflammation
- Many older cats?

- **Inappetence/anorexia**
- **Nausea/vomiting**
- Lethargic
- Painful
- Withdrawn/grumpy
- Constipation

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Feeding tips

- **Space** between water and food and litter tray
- **Easy to access**
- **Avoid aversion**
  - Don't change in clinic
- **Safety**
  - Wider bowls
  - Noises
  - Strangers
  - Compatriots
  - Feline facial pheromone
  - Warm to body temperature

catster.com

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Cats need to feel safe

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Nausea

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Nausea, vomiting

- Optimize hydration
- H2 antagonists or proton pump inhibitors
- Antiemetics

Generic Name	Product™	Dose
Maropitant	**Cerenia**	**0.5-1.0 mg/kg SC q24h**
Mirtazapine	**Remeron**	**2 mg/cat PO q48h**
Ondansentron	**Zofran**	**0.1-0.15 mg/kg slow IV q6-12h**
Dolasetron	**Anzemet**	**0.6 mg/kg IV, SC, PO q 24h**
Metoclopramide	Reglan	1-2 mg/kg CRI IV over 24 h

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Inappetance: tempting food

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Appetite stimulation

- Cyproheptadine: 1 mg/cat PO q12h
- Mirtazapine: 2 mg/cat PO q24-48h
- Capromorelin: 2 mg/kg PO q24h

FDA Approves Elura (capromorelin oral solution) for Managing Weight Loss in Cats with Chronic Kidney Disease

www.alamy.com

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Appetite stimulation

- **Be sure to *calculate* and communicate requirements and monitor amount eaten**

www.alamy.com

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Recognizing, describing, and managing reduced food intake in dogs and cats

Proactive intervention should be strongly considered for animals that are anorexic, hyporexic, or dysrexic, rather than waiting until malnutrition develops.

ACVIM consensus statement on pancreatitis in cats

If appetite stimulation fails to induce adequate appetite within 48 hours, place a feeding tube! Cats presenting with a prolonged history of anorexia should have a feeding tube placed promptly.

Johnson, JAVMA 2017 Forman, JVIM 2021

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2022 ISFM Consensus Guidelines on Management of the Inappetent Hospitalised Cat

"… as a species they are susceptible to stress (especially) in the hospital environment, which may result in reduced food intake;

- previous negative experiences may compound the problem.
- In particular, an inappropriate clinic environment and/or inappropriate handling may cause or exacerbate inappetence in hospitalised patients, with negative impacts on recovery.

"Prescription diets should generally not be introduced while the cat is hospitalised"

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Start early

"A patient with two or more high-risk factors present should receive nutritional support immediately once stabilised. Patients with fewer than two risk factors should be closely monitored and reassessed daily."

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Method of feeding?

- Oral assisted vs. tubes
- Nasoesophageal
- Esophageal
- Advantages and disadvantages
  - +/- anaesthetic, when start using, types of foods, patient acceptance

31

Oral assisted

Small oral capacity!

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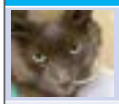
TUBE TYPE	DURATION	ADVANTAGES	DISADVANTAGES
	Less than 5 days	• Inexpensive • 3-5 French • Easy to place • No anaesthesia	• Liquid diets only (1 kcal/mL) • May not eat with tube in place
	Long term	• Inexpensive • 10-16 Fr • Easy to place • Use calorically dense diets • Can eat with tube in place	• Short anaesthesia • Cellulitis
	Long term	• Use calorically dense diets • 10-16 French • Can eat with tube in place	• Anaesthesia • Surgery or endoscopy • Tube displacement • Expensive

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Slide courtesy of Susan Little

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CliniCare Canine/Feline
CliniCare Feline Renal

Liquid diets for cats

Royal Canin Recovery Liquid
Royal Canin Renal Liquid

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Esophagostomy Tubes

- Placement requires brief anaesthesia
- Size 14-16 Fr. red rubber feeding tube
- Long curved forceps
- Injection port/prn adapter
- Bandage neck/Kitty Kollar™

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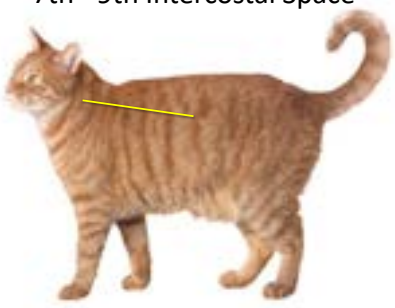
7th - 9th Intercostal Space



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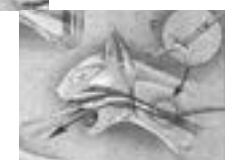

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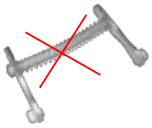
Mouth gags: a bad idea

Maxillary artery compression
- Cerebral ischemia
- Blindness



Stiles J et al, Vet J, 2012

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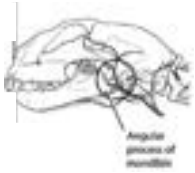
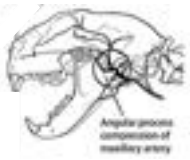
Maxillary artery compression causes cerebral ischemia

Mouth closed	Mouth open
	

Stiles J et al, Vet J, 2012; Barton-Lamb JL et al, Vet J, 2013

susan little dvm, dabvp

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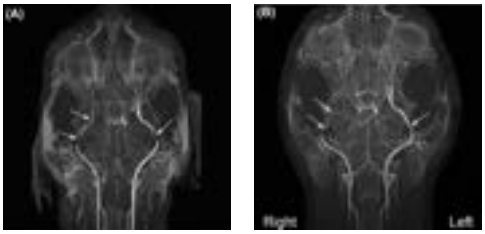

Barton-Lamb AL et al, Vet J, 2013

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E-tube placement

Movie Susan Little

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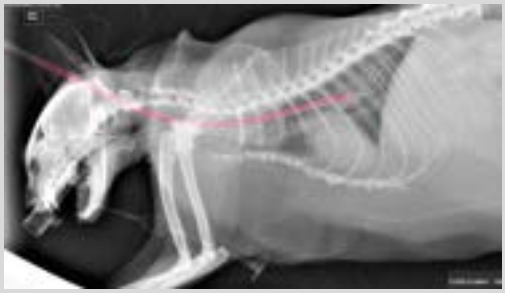

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www.youtube.com/watch?v=qF14Jfajkhw

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- www.Kittykollar.com
- Less scary than bandage
- Cats can eat with tubes in place
- Website has information about:
  - Living with an E-Tube
  - How to change collar

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Tube care

- Post-operatively e-tube npo 1-2h
- Start with 6-10 cc of warm water only through the tube
- First feeding start with 6 cc of slurry and flush tube with 6 cc water
- After each feeding, flush
- If clogged, instill 6 cc cola for 10 minutes
  - Pancreatic enzymes, meat tenderizer
- Clean stoma site BID for 2-3 days
- Cats can eat with tubes in place
  - But avoid offering food for a few days to limit aversion development

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Using an e-tube is easy – video for staff and clients

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Caloric densities

- Clinicare RF: 1 kcal/ml
- Royal Canin Recovery Liquid: 1 kcal/ml
- Royal Canin Recovery: 1.04 kcal/ml
- Hill's a/d: 1.17 kcal/ml
- Purina PPVD CN: 1.33 kcal/ml

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How much to feed? Example

Old cat 4.0 kg sick BCS 3/9, healthy weight 4.5 kg BCS 5/9
Feeding for 4.5 kg = 315 kcal/day (@ 70kcal/kg ideal weight/day)

315 kcal = 302 ml of Royal Canin Recovery (1.04 kcal/ml);
269 ml of Hill's a/d (1.17 kcal/ml);
236 ml of Purina PPVD CN (1.33 kcal/ml)

Example, if using PPVD CN, most calorically dense:

- Day 1 feed 80 ml => Day 2 feed 160 ml => Day 3 feed 236 ml

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Caloric densities – using renal diets

- Clinicare RF: 1 kcal/ml
- Royal Canin Recovery: 1.04 kcal/ml
  - Royal Canin Renal Support E: 1.09 kcal/ml before slurrying
- Hill's a/d: 1.17 kcal/ml
  - Hill's k/d: 1.13 kcal/ml before slurrying
- Purina PPVD CN: 1.33 kcal/ml
  - Purina PPVD NF: 1.3 kcal/ml before slurrying

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Caloric densities in slurried renal diets

	Caloric density Kcal/ml	Kcal/ml mixed w equal volume water	Kcal/ml mixed w equal volume Clinicare RF or RC Renal Liquid
Clinicare RF or RC Renal Liquid	1 kcal/ml		
Purina NF	1.3	0.65	1.15
Hill's k/d chicken	1.13	0.57	1.06
Royal Canin Renal Support E	1.09	0.55	1.04

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How often to feed?

- Based on caloric requirements & volume tolerance
  - Example: Start with 6 ml and increase by 6 ml increments to ~ 36-48 for most cats
  - Tolerance is very variable: be patient, don't get disheartened

Communicate quantities and frequencies clearly

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How long to assist feed?

- How long can you leave tubes in?
- When to remove?
- How do we determine success?
  - Weight gain
  - Coat quality
  - Increased energy
  - Muscle recovery
  - Client-noted improvements

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Resources

Videos showing how to place and care for tubes

- Nasoesophageal or nasogastric tube in a cat https://youtu.be/-WfuE8djYos
- Esophagostomy tube in a cat https://youtu.be/MiNvX2pF6to
- Caring for a cat with an esophagostomy tube https://youtu.be/UsLcTZ8u8Gk

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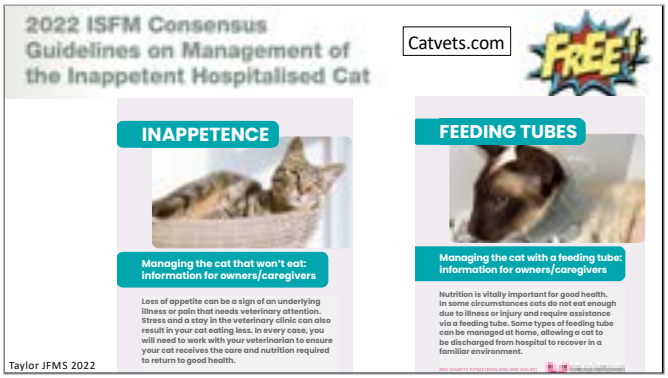

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Summary

- Be proactive
- Alleviate pain
- Calculate nutritional needs
- Try bowl, diet types
- Appetite stimulants
- Feeding tubes!!!
- Correct underlying problems

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Thank you for your interest!

Do you have any questions?

I hope this helps you help more cats!

hypurr@aol.com

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8006

A NEW ERA OF VETERINARY MEDICINE

PRACTICE PEARLS

Brandi Deimling, Hons. BA, OCC, OCGC

Disclaimer: Due to the dynamic nature of policy work and conference timelines, the content of this proceeding and any subsequent information may be subject to change based on new developments at the time of the presentation.

BACKGROUND

In spring 2024, the Ontario government passed legislation to enact the Veterinary Professionals Act. It was a historic moment, representing almost 11 years of advocacy work, and OVMA is proud to have effectively represented our members' needs and perspectives.

Over the past decade association staff have been an effective voice in sharing the concerns and challenges that members have faced in providing care to their patients under an antiquated regulatory framework. Through that time, staff have pushed for legislation that addresses the needs of the profession, and that's fair and balanced, while protecting the health and welfare of the patients and clients members serve. The new legislation addresses the longstanding concerns of members and has created a framework allowing for growth and innovation, ensuring the profession builds upon its excellence in animal care.

While the passing of the new act is wonderful news, the work is just beginning. Over the next year, staff will consult with OVMA's small and large animal issues committees, along with the Board of Directors (all comprised of active veterinarians) and external stakeholders (where necessary), to ensure the new regulations are reflective of the needs of the profession under the College of the Veterinary professionals of Ontario.

OBJECTIVES

- Be empowered to understand the Act
- Efficiencies and accountability
- Better process for the College and members
- Maximizing team-based care – Initial concepts

WHAT'S NEW

Definition of Veterinary Medicine

- "The assessment of the physiological or behavioural status of an animal or group of animals, other than humans, and the diagnosis, treatment, prevention, or control of any condition, disease, disorder or dysfunction".

Concept of Fitness to Practice

- Defined as "fitness to engage in the practice of veterinary medicine".

Title Protection

- Same: Veterinarian, Veterinary Surgeon, Doctor
- New: Registered Veterinary Technician, Registered Veterinary Technologist, Veterinary Technician, Veterinary Technologist
- College will not be issuing "R" for any new licensees under the CVPO
- Exception for Chiropractors to use "Dr" as long as it is in conjunction with chiropractor (DC).
- Veterinary Facility

Order

- Focus on One Health and Workforce Shortages.
- Interprofessional collaboration and consultation.

Risk of Harm Clause

- No person other than a member acting within the scope of the practice of veterinary medicine shall treat an animal or advise an owner or their representative with respect to an animal's health, in circumstances in which it is reasonably foreseeable that serious bodily harm to an animal or person may result from the treatment or advice or from an omission from the treatment or advice.

Scope of Practice Model & Authorized Activities

- Schedule I – 17 authorized activities

Mandatory Reporting

- A member shall file a report with the Registrar in accordance with this section, if in the course of practicing veterinary medicine, the member learns of anything that causes the member to have reasonable grounds to believe that another member's fitness to practice is impaired.
- Self reporting on professional negligence and malpractice inside or outside of Ontario.

Quality Assurance Program

- To be established in regulations and College policy.
- Promote continuing education, competence and quality improvement, and self and peer practice assessments.

Complaints

- Investigations and Resolutions Committee
- Discipline and Fitness to Practice
- Quality Assurance
- Mandatory Mediation (first of its kind in regulated professions)
- Ability to Dismiss
- Publication of length of time for complaints

Maximizing Team Based Care

- From a day-to-day perspective nothing will change for veterinarians.
- Authorized Activities from Accredited Facilities
- Delegation to auxiliaries
- Delegation and co-accountability with RVTs
- Competency, skills, training
- Non-veterinary professionals

Next Steps in the Process

- Regulation Development, Consultations, and Approvals
- College Transition to the College of Veterinary Professionals of Ontario
- Further Guidance for the Profession from all organizations involved (OVMA, CVO and OAVT).

All updates will be provided to OVMA members through NewsHound or on our website: https://www.ovma.org/member-portal/advocacy-outreach

8007

DIALYSIS, PLASMA EXCHANGE, OR TOXIN REMOVAL

PRACTICE PEARLS

Xiu Ting Yiew, DVM, DVSc, DACVECC

Extracorporeal blood purification therapies are advanced adjunctive treatments that provide critical support for renal recovery, control of immune-mediated diseases, and management of life-threatening intoxications.[1] These therapies act as life-support measures, buying time for kidneys to regain function, immunosuppressive treatments to take effect, and the prevention of major organ dysfunction. This is achieved by removing endogenous (e.g. uremic toxins, bilirubin, autoantibodies, inflammatory mediators) and exogenous (e.g. medication overdoses, accidental toxin ingestion) toxins, as well as correcting life-threatening electrolytes, acid-base, and fluid imbalances. Timely integration of these therapies is critical when conventional treatments alone cannot manage clinical signs or severe abnormalities.

Common veterinary applications include dialysis (renal replacement therapy) for renal failure, plasma exchange for immune-mediated diseases, and toxin removal for life-threatening exposures. Modalities such as intermittent hemodialysis (IHD), continuous or prolonged intermittent renal replacement therapy (CRRT/PIRRT), therapeutic plasma exchange (TPE), and hemoperfusion (HP) are employed based on provider capabilities. These therapies share key processes, including extracorporeal blood circulation (a portion of the patient's blood is maintained outside the body), the use of systemic unfractionated heparin or regional citrate anticoagulation to prevent extracorporeal circuit clotting, and the manipulation of plasma constituents to remove harmful substances. For small patients (e.g. puppies, small dogs or cats) who are unable to tolerate extracorporeal circuit volume, dialysis catheter, or where hemodialysis machine is unavailable, peritoneal[2] or pleural[3] dialysis can be viable alternatives.

HEMODIALYSIS

The optimal timing for initiating renal replacement therapy remains uncertain in both human and veterinary medicine.[1] However, best-practice consensus guidelines from the IRIS Working group[4] with extensive extracorporeal expertise provide useful direction:

Indications: [1,4]

1. Persistent (>6 h) anuria or oliguria (<0.3 mL/kg/h) unresponsive to appropriate fluid therapy and medical management
2. Overhydration unresponsive to diuretic therapy, leading to target organ damage
3. Severe electrolyte or acid-base disturbances (e.g. refractory hyperkalemia, metabolic acidosis) unmanageable by medical or surgical intervention
4. Progressive azotemia (serum creatinine >442 umol/L or BUN >35 mmol/L) in euhydrated patients, unresponsive to medical management
5. AKI or ACKD with renal recovery potentials (e.g. leptospirosis, lily toxicity, pyelonephritis)
6. Presence of dialyzable toxins (e.g. ethylene glycol, ethanol, barbiturates, or other low MW substances)

Chronic hemodialysis is rarely performed in veterinary patients but may be considered for those with IRIS Stage 4 CKD when clinical signs cannot be managed by comprehensive medical therapy. However, this is not offered at the speaker's institution due to the limitations of their hemodialysis machine, which is more suitable for AKI and ACKD with renal recovery potentials.

PERITONEAL OR PLEURAL DIALYSIS †

While less efficient than hemodialysis, these options offer life-saving support for specific patient populations.

Indications: [2,3]

1. Similar to hemodialysis, with additional considerations for:
2. Small patients:
    - Inability to hemodynamically tolerate extracorporeal blood volume demands
    - Difficulty obtaining dual-lumen jugular vascular access for hemodialysis
3. Limited blood resources for hemodialysis machine priming
4. Hemodialysis machine unavailability
5. Presence of intra-abdominal pathology† or recent abdominal surgery†

Contraindications: [2]

1. Peritoneal adhesions or fibrosis (ϑ surface area, ϑ efficiency)
2. Pleuroperitoneal leaks (pleural effusion, respiratory compromise)
3. Recent thoracic or abdominal (GI) surgery (risk of dehiscence, infection)
4. DH, PPDH, inguinal or abdominal hernias (intrathoracic effusion, organ herniation)
5. Severe coagulopathy or hypercatabolic states (hypoalbuminemia)

THERAPEUTIC PLASMA EXCHANGE (TPE)

TPE involves the removal and exchange of a patient's plasma, along with substances implicated in disease pathophysiology (e.g. autoantibodies, antigens, circulating immune complexes, activated complement proteins, soluble inflammatory mediators, adhesion molecules, protein- bound toxins), with replacement solutions and donor plasma. It is increasingly used in veterinary medicine as an adjunctive therapy for immune-mediated diseases (e.g. IMHA, ITP, myasthenia gravis, glomerulonephritis, etc.) and toxin removal.[1,5–10]

Immune-Mediated Hemolytic Anemia (IMHA)

TPE offers more immediate control of IMHA compared to standard immunosuppressive therapies, which often take days to achieve full therapeutic effect. Severe intravascular hemolysis can lead to marked hyperbilirubinemia, increasing the risk of acute bilirubin encephalopathy–a life-threatening condition characterized by progressive neurological signs, including refractory seizures and coma. TPE effectively reduces circulating autoantibodies and bilirubin, leading to temporary disease remission and clinical improvement, thereby mitigating the risk of acute bilirubin encephalopathy, transfusion dependency, and potentially mortality.[11,12] Because immunoglobulins can redistribute from tissue stores, 3-5 TPE sessions may be required to achieve sustained disease control. While not a first-line therapy, TPE is a valuable adjunctive treatment for critically ill patients who are transfusion-dependent, refractory to standard therapies, or unlikely to survive until immunosuppressants take effect.[13]

Indications: [1,5,6,11]

1. Severe or refractory disease, especially when protracted recovery is anticipated
    - Avoid referring for TPE as a last-resort treatment for moribund patients
    - Early referrals allow for TPE before declines in stability or treatment tolerance
2. Persistent evidence of ongoing RBC destruction (auto-agglutination, spherocytosis, ghost cells on cytology, pigmenturia)
3. Requirement for 3 or more blood transfusions
4. Severe hyperbilirubinemia with risk of acute bilirubin encephalopathy
    - Bilirubin threshold associated with neurologic decompensation = 608-1070 umol/L (dogs), 299 umol/L (cat)1
5. Before adding a 3rd immunosuppressant
6. Before initiating human IV immunoglobulin (risk of hypersensitivity)

TOXIN REMOVAL

Many toxin ingestions can be effectively managed with early decontamination and supportive care. However, extracorporeal toxin removal (ECTR) should be considered in cases involving life-threatening toxin doses. When initiated promptly, ECTR can prevent major organ dysfunction or death by removing toxins before they fully distribute throughout the body, thereby reducing exposure time. Most intoxications require only 1 session to reduce serum drug concentration below toxic thresholds. However, toxins with a large V_d, slow intercompartmental transfer rates, or high exposure levels may benefit from a second session.[1]

Indications: [1,8]

1. Exposure to a toxin at doses sufficient to cause significant organ dysfunction, serious complications, tissue injury, or death
2. Extracorporeal clearance is likely to exceed the body's endogenous clearance
3. No effective antidote or potentially more effective alternative therapy exists
4. The benefits of ECTR outweigh the risks to patient safety
5. Common veterinary intoxications suitable for ECTR include: NSAIDs** (e.g. naproxen, ibuprofen, carprofen, meloxicam), baclofen, ethylene glycol, chemotherapeutics (e.g. azathioprine, vincristine, vinblastine, 5-FU, methotrexate), anticonvulsants

RISKS & POTENTIAL COMPLICATIONS OF EXTRACORPOREAL THERAPIES [1,4–6,8]

1. Bleeding from uremia (↓ platelet function, GI ulceration), underlying disease (e.g. hemorrhagic leptospirosis, ITP), concurrent coagulation disorders (e.g. DIC), or intra- treatment systemic heparinization
2. Hypotension, hypovolemia
3. Thrombosis (jugular vein, PTE) from underlying disease (e.g. hypercoagulability), extracorporeal circuit clotting
4. Infection (immunosuppression, dialysis catheter-associated)
5. Dialysis disequilibrium syndrome (excessive intracellular fluid shifting, cerebral edema)
6. Transfusion reactions (e.g. pRBC, plasma), including citrate toxicity (hypocalcemia)

HOW CAN YOU HELP?

1. Be familiar with extracorporeal therapies, including indications, patient suitability, potential complications, and cost estimates
2. Assess the client's willingness and ability to proceed with treatment
3. Reach out to providers of extracorporeal therapies to discuss the case specifics**
4. Timely referral is critical to improving outcome!
5. Avoid jugular venipuncture (thrombosis, hematoma), particularly in the right jugular vein
6. Exercise caution with fluids: Maintain euhydration and euvolemia
7. Initiate standard treatments (e.g. decontamination, ILE therapy) while arranging transfer

REFERENCES

References are available from the speaker upon request.

8008

ONTARIO'S AT RISK NATIVE TURTLE POPULATIONS

PRACTICE PEARLS

Sue Carstairs, B.Sc., D.V.M., O.Ont.

Southern Ontario is home to eight species of freshwater semiaquatic turtles, including two globally endangered species (the Spotted Turtle, and Blanding's Turtle). All eight species are considered 'at-risk' federally. Turtles are vital to our remaining wetlands, and wetlands are important to us all, as a source of drinking water, and an efficient flood-mitigator, as well as providing a home for the diverse range of species who live there. Canada holds 25% of the world's wetlands but has lost 70% of its wetlands over the last 100 years.

There are many threats facing turtle populations both in Canada and globally, including habitat loss and fragmentation, illegal harvesting for the pet trade, unsustainable consumption, and road mortality. Turtles are particularly vulnerable to even a small increase in mortality due to their life history; being a long-lived species, with low recruitment into the adult population, slow maturity, and therefore slow population growth rates. Since road mortality is one of the largest threats to Ontario's native turtles, saving turtles injured on roads is one way to help to mitigate declining populations and help to 'buy time' to fix the issues.

The Ontario Turtle Conservation Centre (OTCC) houses the only CVO accredited, dedicated native turtle hospital in Canada, and annually admits over 2,000 injured or ill turtles from across their home range throughout Ontario and beyond. It also collects and incubates approximately 9,000 eggs that are derived from these turtles, which are then incubated and hatched. Admitting so many turtles from a wide geographic range is carried out with the help of 'First Responder' veterinarians across the province. First Responder veterinarians are veterinarians who generously donate their time to help ensure the turtles receive immediate access to emergency treatment, while waiting for transport to our hospital. Approximately 1500 Turtle Taxi Volunteers then help to bring the turtles to us for surgery and ongoing care. Once healed, these turtles are then released back to their home wetland. The hatchlings are also released back to their mother's wetland.

The rehabilitation of turtles can have a population effect and is a viable conservation strategy, as is "head-starting", or the release of turtles hatched in captivity. In addition, rehabilitation centres provide an excellent means to monitor population health. This is therefore an area where veterinarians can have a direct impact on conservation. The Ministry of Natural Resources and Forestry (MNRF) oversee wildlife rehabilitation in Ontario, and their regulations stipulate that any member of the public can hold wildlife for up to 24 hours, with a view to taking them to a veterinarian or wildlife rehabilitator, and veterinarians can carry out treatment of wildlife on an emergency basis. So, any hospital can help wild turtles; even those who don't regularly see exotics in their practice. It does require a rehabilitation license to hold wildlife for rehabilitation, however.

Turtles have a unique ability to withstand and recover from an astonishing amount of trauma; they even have the ability in some cases to regenerate spinal tissue. As such, they are an extremely satisfying species to treat successfully. Historically, many wild turtles presented to veterinary clinics have sadly been unnecessarily euthanized, and their eggs not retrieved. This has been done with good intentions, since most species wouldn't survive the injuries that they endure.

To establish the First Responder program, OTCC has built up a large number of partner veterinarians across the province (46 to date), and helped them feel comfortable in treating injured turtles, if they weren't already familiar, by providing a simple time-efficient method to aid in

initial stabilization. This includes fluid administration, analgesics, and basic wound management. Apart from Controlled substances, all supplies are provided by OTCC, and any training needed is carried out with the participating hospital. The hospital then houses the turtles until they are picked up by our Turtle Taxi Volunteers. This system works best if all staff are motivated, since it does require participation from the reception staff, as well as the veterinary staff. We have received feedback that participation in the OTCC First Responder program is a good morale booster for clinic staff due to the unique ability to participate in conservation, and the novel work they are able to carry out.

The names of first responders are never published, to avoid hospitals having unexpected arrivals. Hospitals are also always called before turtles are directed to their facility and are given the opportunity to decline taking in a turtle, should it not be feasible on that particular day. Our hotline staff averages approximately 10,000 calls during the busiest season (May through September), arranging turtle transport, and answering public queries.

Ontario Turtle Hospital can be reached at 705-741-5000 and inquiries can be sent to info@ontarioturtle.ca

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8009

SURGICAL MANAGEMENT OF BRACHYCEPHALIC SYNDROME

PRACTICE PEARLS

Howard Seim, DVM, DACVS

KEY POINTS

- English bulldogs are significantly over-represented.
- Light general anesthesia is required for accurate evaluation of laryngeal function and defects.
- Limited use of crushing clamps and cautery results in less postoperative swelling.
- Overall prognosis for dogs with brachycephalic syndrome is favorable.

If you would like a video of these surgical procedures go to www.videovet.org or contact the author at videovet@me.com. You may click on the 'Seminar Price' for any surgery video you would like to purchase.

Definition: Brachycephalic syndrome is a combination of upper airway disorders commonly seen in brachycephalic breeds (e.g., English bulldog, Boston terrier, Pugs). Disorders associated with this syndrome include stenotic nares, elongated soft palate, and everted laryngeal saccules. Occasionally patients present with laryngeal collapse. Patients may present with any combination of the above listed disorders.

DIAGNOSIS

Clinical presentation:

Signalment: Brachycephalic breeds are most commonly affected (i.e., English bulldog, French bulldog, Boston terrier, Pug, Pekingese). The age at presentation ranges from less than one year to 11 years. The majority of patients present between 1 and 4 years with English bulldogs presenting at a younger age than other breeds. There is no apparent sex predisposition.

History: Historical findings are generally related to upper airway obstruction and include noisy respiration, heat intolerance, exercise intolerance, cyanosis, and occasionally syncopal attacks. Gagging, retching, and vomiting may also be reported. Historical findings may vary depending upon the number of abnormalities present (i.e., stenotic nares, elongated soft palate, and/or everted laryngeal saccules). Generally, the more abnormalities present the more severe the historical and clinical findings.

Clinical signs: The most frequently reported clinical signs in patients with brachycephalic syndrome include noisy respirations and exercise and/or heat intolerance. Moderate to severely affected patients or patients with multiple defects may present with cyanosis and/or syncope.

Physical examination: Physical examination is generally normal except for patients with stenotic nares. In patients with stenotic nares the wings of the nostril (i.e., dorsolateral nasal cartilage) obstruct airflow resulting in turbulent airflow and resultant noise. Examining the patient after exercise may exacerbate clinical signs (i.e., noise and exercise intolerance) making diagnosis of brachycephalic syndrome more likely. Oral examination of the awake patient is generally unrewarding as the laryngeal apparatus and related abnormalities cannot be seen without light general anesthesia.

Radiography: Diagnosis of brachycephalic syndrome is based on signalment, history, physical examination, and direct visualization of the laryngeal apparatus with the patient under light general anesthesia. Thoracic radiographs are generally recommended to rule out lower airway disorders such as tracheal hypoplasia and pulmonary abnormalities.

Differential diagnosis: Any disorder causing noisy respirations, exercise intolerance, cyanosis, and syncope. Included are laryngeal mass, laryngeal collapse and laryngeal paralysis.

Medical management: Medical management is directed at decreasing airway turbulence and subsequent inflammation and edema. Strict confinement, anti-inflammatory medications (e.g., steroids, NSAIDS), and a cool environment are recommended. Obese patients should be placed on a weight reduction diet plan. As medical management does nothing to change the anatomic deformity of the disorder, it is considered palliative but not curative.

Surgical treatment: The objective of surgical treatment is to provide an adequate airway by relieving any anatomic obstruction.

Preoperative management: Use of anti-inflammatory medication preoperatively is generally recommended. Patients are given intravenous steriods (dexamethasone 0.5 - 1 mg/kg IV) at the time of anesthetic induction.

Anesthesia: Anesthetic management is somewhat dependent upon the severity of clinical signs at presentation and degree of airway abnormality.

Patients with mild signs may be anesthetized with the clinicians' standard anesthetic protocol. Careful evaluation of the laryngeal apparatus is performed prior to intubation and while the patient can still breath on its own (i.e., light general anesthesia). Laryngeal function is carefully evaluated during inspiration and expiration.

Patients with moderate clinical signs may need to be preoxygenated prior to induction. Induction should be performed quickly, the laryngeal anatomy and laryngeal function examined thoroughly, and the patient intubated to establish an open airway.

Patients with severe clinical signs should be preoxygenated 5 - 10 minutes prior to induction. A vagolytic agent (i.e., atropine) should be considered 10 - 15 minutes prior to induction because vagal tone is generally increased and cardio-inhibitory reflexes are enhanced. Induction should be quick, examination of the laryngeal anatomy and function performed, and the patient intubated to establish an open airway.

Laryngeal examination: Once the patient is under a light plain of anesthesia laryngeal function is evaluated. Care is taken to observe for evidence of laryngeal collapse, elongated soft palate, and everted laryngeal saccules.

Surgical anatomy: The soft palate in the dog forms a long and broad movable partition between the oral and nasopharynx. The cranial border is attached to the bony palate; the caudal margin forms the dorsal border of the opening from the mouth into the pharynx. This portion of the palate is in contact with the epiglottis during normal inspiration; during deglutition, the epiglottis moves away from the soft palate to protect the opening of the glottis. At the same time the soft palate moves dorsally to close the nasopharynx and prevent regurgitation of material into the nasal cavity. The dorsal nasopharyngeal surface has a mucous membrane lining continuous with that of the nasal cavity and a slightly convex contour. The mucous membrane of the ventral concave surface is a continuation of the lining of the hard palate and is referred to as the oral surface of the soft palate.

Relevant pathophysiology: Protrusion of an elongated soft palate into the laryngeal inlet during respiration significantly obstructs air passage into the glottis. Stenotic nares, when present, contribute to the severity of the occlusion by increasing the inspiratory effort (and subsequent negative pressure) thus drawing the soft palate deeper into the larynx. Edema and inflammation result from friction against the epiglottis during each respiration. The resultant thickening further lessens airflow. As increased inspiratory effort continues, increased negative pressure in the airway encourages laryngeal saccules to evert.

Positioning: Patients may be positioned in ventral or dorsal recumbancy.

Stenotic nares: The author prefers ventral recumbancy with the head supported on towels so the head position is normal and functional.

Elongated soft palate and everted saccules: Patients can be operated in either ventral or dorsal recumbancy. In dorsal recumbancy, the maxillary canine teeth are taped securely to the operating table. The mandibular canine teeth are taped to an ether stand situated over the patients' head. The mouth is opened wide to enhance visualization. This positioning is critical as oral

cavity exposure is key to adequate visualization and instrumentation.

In ventral recumbancy, the maxillary canine teeth are 'hooked' over the bar of an ether stand. The mandibular canine teeth are then taped to the operating table in such a fashion that the mouth gapes open. The tongue is grasped with tongue forceps and gently pulled from the mouth.

Surgical technique: The surgical technique varies depending upon the defect to be repaired.

Stenotic nares: This technique is illustrated on the Respiratory Surgery I surgery video available via www.videovet.org.

Stenosis is decreased by removing a horizontal wedge of alar cartiladge from the wing of the nostril. The flap created is sutured to remaining tissue of the wing of the nostril using 3-0 or 4-0 Dexon or Vicryl in a simple interrupted suture pattern. Two or three sutures is all that is generally required to complete the nasoplasty.

An alternate technique gaining popularity in Shih Tzu and Boston breeds is to completely excise the alar cartilage. Bleeding is controlled by wedging a gauze sponge in the patient's nostril for 5 minutes by the clock.

Presurgical temporary tracheostomy?: Use of a presurgical tracheostomy facilitates exposure and visualization of the soft palate and laryngeal saccules. However, it is not necessary in the majority of patients. The author considers use of a tracheostomy in patients that present with severe clinical signs (i.e., cyanosis, syncope) and have a combination of defects to repair. Tracheostomy is preferred over exiting the endotracheal tube through a pharyngostomy as the tracheostomy can be used in the postoperative management of the patient if necessary. In our hospital, regardless of the severity of the airway obstruction, the patient is recovered in a critical care environment and instruments necessary to perform an emergency tracheostomy are readily available.

Elongated soft palate: This technique is illustrated on the Respiratory Surgery I surgery video available via www.videovet.org.

When the patient is anesthetized for surgery, light general anesthesia is performed so the surgeon can visualize the relationship of the soft palate with the epiglottis prior to intubation. Using a skin marker a single 'dot' is placed on the location of the elongated soft palate that touches the tip of the epiglottis (see the video for Soft Palate Resection on the Respiratory Surgery I surgery video www.videovet.org). Once the soft palate is marked the patient can be intubated and anesthetized for surgery.

The patient is placed in ventral (the author prefers ventral recumbacy) or dorsal recumbancy with the mouth opened widely (see positioning). A broad malleable retractor can be used to retract the tongue caudally or a tongue clamp can be used to retract the tongue ventrally; either technique greatly facilitates visualization of the soft palate and laryngeal structures. A headlamp also facilitates visualization but is not necessary.

Since postoperative edema and swelling are of major concern following soft palate surgery, it is important to keep surgical trauma to a minimum. Use of clamps and electrocautery may cause excessive surgical inflammation and should be avoided. Use of a laser has been shown to be an atraumatic alternative to excision and suturing.

When suturing, a 3-0 or 4-0 synthetic absorbable braided suture is recommended (Dexon, Polysorb or Vicryl). Dexon, Polysorb or Vicryl is chosen because of its soft supple nature; Maxon, Biosyn or PDS are much to stiff and may cause irritation to the oral cavity postoperatively.

First, a stay suture is placed in the soft palate on each lateral margin of the proposed soft palate excision. A mosquito hemostat is placed on the stay sutures to apply tension to the palate thus facilitating exposure. The mark on the soft palate is used to help determine stay suture location. A third stay suture is placed on the margin of the central portion of the soft palate. This stay suture allows the surgeon to manipulate the palate during resection. The soft palate incision is begun from the left or right margin (stay suture) and one-third of the soft palate is incised using the 'dot' to determine extend of resection. The incised nasal mucosa is then sutured to the incised oral mucosa using a simple continuous suture pattern. Hemorrhage is controlled by suture pressure. No attempt is made to cauterize or clamp bleeding vessels. Once the first 1/3 of the palate excision is sutured the next 1/3 of the palate is cut and sutured. Staging the excision facilitates the surgeon's ability to visualize the oral and nasal mucosal cut surfaces for suturing. When the palate

excision and suturing are complete, the stay sutures are cut and the remaining soft palate replaced and evaluated once again for extent of resection.

Everted laryngeal saccule resection: There is some suggestion that if the stenotic nares and elongated soft palate can be successfully treated (see above), the lateral saccules will return to their normal location in the larynx and no longer cause airway obstruction without the need for surgical resection. The author only removes lateral saccules in patients that present with severe respiratory signs (i.e., severe cyanosis, syncope).

When removing laryngeal saccules, the patient is placed in dorsal recumbancy with the mouth opened widely. Everted laryngeal saccules appear as edematous, translucent tissue 'balls' lying in the ventral aspect of the glottis and obscuring the vocal folds.

If the patient had a tracheostomy tube placed prior to surgery, the saccules are easily visualized and excised as described above. If the patient has an endotracheal tube exiting the laryngeal apparatus, the tube is temporarily removed while the saccules are excised.

Surgical removal is performed using a sharp long-handled laryngeal cup biopsy forceps (or similar long handled biopsy instrument) or a long-handled Allis tissue forceps and #15 BP scalpel blade. If a laryngeal cup biopsy forceps is used the everted saccule is grasped and amputated with the biopsy forceps. Any remaining tags are grasped with a long-handled DeBakey forceps and trimmed with a #15 BP blade or scissors. If an Allis tissue forceps is used the laryngeal saccule is grasped with the Allis forceps and a long-handled scalpel with a #15 BP blade is used to excise the saccule at its base.

SUTURE MATERIAL/SPECIAL INSTRUMENTS:

Malleable retractors or Young tongue retractor (JorVet.com), head lamp, long-handled laryngeal cup biopsy forceps (or similar instrument), long-handled Allis tissue forceps, long-handled scalpel handle, long-handled DeBakey forceps, 3-0 or 4-0 Dexon, Polysorb or Vicryl with a cutting or sharp taper needle.

POSTOPERATIVE CARE AND ASSESSMENT:

Any patient requiring surgery to relieve airway obstruction should be monitored carefully (preferably in a critical care environment) for the first 24 hours postoperatively. The degree of care may vary depending upon the patients presenting signs and surgical manipulations required to correct the airway obstruction. Examples of the authors' degree of postoperative care based on patient presentation and surgery performed are listed below:

Stenotic nares only: These patients are generally held for observation 12 – 24 hours postoperatively and discharged from the hospital the day following surgery. Soft palate resection only: Patients that present with mild clinical signs (i.e., noise, mild exercise or heat intolerance) and are bright and alert 24 hours after surgery can be discharged that day. Patients that present with moderate to severe clinical signs (i.e., severe exercise intolerance, episodes of cyanosis, syncopal attacks) are monitored in a critical care environment until signs resolve. Immediate postoperative gagging and coughing are observed in about 13% of patients. Patients requiring a tracheostomy prior to surgery, or an emergency tracheostomy, remain in a critical care environment until the tracheostomy can be removed.

Combined nares, palate, saccule repair: These patients are treated similarly to patients with soft palate resection and are based on presenting clinical signs. Patients with multiple defects tend to present with moderate to severe clinical signs and may require more intensive care. Immediate postoperative gagging and coughing are observed in about 80% of patients.

Patients that present with mild clinical signs (i.e., noise, mild exercise or heat intolerance) and are bright and alert 24 hours after surgery can be discharged that day. Patients that present with moderate to severe clinical signs (i.e., severe exercise intolerance, episodes of cyanosis, syncopal attacks) are monitored in a critical care environment until signs resolve. Patients requiring a tracheostomy prior to surgery, or an emergency tracheostomy, remain in a critical care environment until the tracheostomy can be removed.

PROGNOSIS:

Prognosis for patients with brachycephalic syndrome is generally dependent upon the defects found at presentation.

Stenotic nares only: About 96% of dogs with stenotic nares will improve postoperatively.

Soft palate resection only: About 85 – 90% of dogs with soft palate resection only will improve postoperatively. Young dogs (i.e., less than 2 years of age) are more likely to improve (90%) than dogs greater than 2 years of age (70%).

Stenotic nares and soft palate resection: Dogs having a combination of stenotic nares repair and soft palate resection are more likely to have a favorable outcome (96%) compared to those that did not (70%).

Soft palate and everted saccule resection: Dogs having this combination of defects repaired will have an 80% chance of significant improvement postoperatively.

8010

WHICH EYES NEED PRESSURES TAKEN?

PRACTICE PEARLS

David Maggs, BVSc (hons), DACVO, MANZCVS

SUMMARY

Although it sounds trite, the answer to the question "Which eyes need their pressure taken?" is "Every eye which may have altered intraocular pressure (IOP)!" Therefore, those eyes which may have conjunctivitis (in which the intraocular pressure (IOP) is always normal), uveitis (in which the IOP is typically low), or glaucoma (in which the IOP is elevated) all need their pressure taken. In other words, tonometry should be performed on any eye demonstrating a sign of conjunctivitis, uveitis or glaucoma, that is, any eye which is reddened, is painful, or has discharge, corneal opacity, vision loss, anisocoria, or altered pupillary light reflexes (PLRs). Having diagnosed uveitis or glaucoma, tonometry is perhaps the most important thing to recheck so as to guide adjustments in therapy.

Assessment of intraocular pressure (tonometry) is essential for differentiation of the two major, vision-threatening conditions in which red-eye is the hallmark feature – uveitis and glaucoma. The availability of easily used and reasonably priced tonometers such as the Tonopen® or TonoVet® make measurement of intraocular pressure (IOP) easier in all species, particularly cats. Unlike the Schiotz tonometer, the Tonopen and TonoVet measure IOP directly and do not require any conversion. They can also be held horizontally and therefore allows measurements to be performed with the patient's head held in a normal, relaxed position. Finally, their small probes/footplates permit easy measurement of IOP in even the smallest feline and pediatric canine eyes.

The TonoPen comes with an excellent instructional video and manual, however the following tips may assist you to get the most from your Tonopen. A drop of topical anesthetic is applied to the cornea. A disposable cover is placed over the Tonopen tip and the pen is turned on with firm, somewhat protracted pressure on the large black button about one third way down the shaft. The equipment should be periodically calibrated according to the manufacturer's directions. Correct patient restraint is essential. The patient should be lightly restrained so as to not artificially raise IOP. In particular, direct pressure on the jugular veins and on the globe itself via the eyelids should be avoided. I prefer to have an assistant (and not the owner!) restrain the patient's head using the angle of the mandible. I then hold the Tonopen in my dominant hand, and gently part the patient's eyelids using my non-dominant hand but such that pressure is applied to the underlying orbital rim; not globe. I then rest the hand holding the Tonopen onto the hand holding the eyelids or onto the patient's head itself and gently touch the central cornea with the Tonopen tip. Minor movements away from the cornea and very gentle "blotting" of the cornea with the tip will enhance the reliability and reproducibility of the readings while reducing the number of readings necessary. Particular attention should be paid to the "approach angle" of the Tonopen tip to the cornea. The tip's flat surface should be exactly parallel to the corneal surface. This is best achieved by viewing the interface between the cornea and the tip from the side. The approach angle of the Tonopen itself should be exactly perpendicular to the corneal surface. However, note that due to corneal curvature, this means the approach angle must be changed dramatically if any area other than the central cornea is used.

Each time the cornea is appropriately "blotted" with the probe, an electronic tone will advise the operator that a reading has been obtained. When a suitable number of readings has been obtained, a tone of a different pitch will sound and no further readings can be obtained without restarting the Tonopen using the large black button again. The number of readings required to achieve an average varies depending on how disparate the readings are from each other and from the normal physiologic range. A small digital screen at the end distant from the

tip displays the IOP in mmHg and provides an estimate of the "reliability" (coefficient of variance) of the result. This appears as a small bar above one of 4 percentage readings. This bar should be above the 5% mark or tonometry should be repeated on that eye. Across large populations, normal canine and feline IOP is reported as approximately 10-25 mmHg. However, significant variation is noted between individuals, technique, and time of day. Comparison of IOP between right and left eyes is therefore critical to interpretation of results. A good rule of thumb is that IOP should not vary between eyes of the same patient by more than 20%.

The obvious application for tonometry is the diagnosis of glaucoma (where IOP is generally elevated). However, tonometry is also used to diagnose uveitis; in which IOP is lowered due to loss of function of the inflamed ciliary body. Perhaps the most important role for tonometry is the monitoring of progress of these diseases and the adjustment of medications based on these data.

9001

SURGICAL MANAGEMENT OF GDV

SURGERY

Howard Seim, DVM, DACVS

If you would like a copy of this surgical procedure on DVD go to www.videovet.org.

KEY POINTS

- Survival is generally determined by early and appropriate presurgical management
- Patients referred for surgery should be decompressed prior to referral with continued decompression provided during transport
- Incisional gastropexy results in a fast, easy, permanent adhesion
- Ventricular tachycardia is a common postoperative complication
- Gastric necrosis signals an unfavourable prognosis

Introduction: Patients with GDV are considered critical care cases; every minute of presurgical treatment is vital to a successful outcome. Survival is generally determined by early and appropriate presurgical management and urgent surgery as soon as the patient is stabilized. Efficient presurgical treatment usually involves a minimum of two people. Gastric decompression and shock therapy should be done simultaneously. If this is not possible; decompression should be performed first. It is stated that gastric decompression is the single most important factor in reversing cardiovascular deficits in patients with GDV.

Decompression: Generally, orogastric intubation can successfully be performed in 80 - 90% of GDV patients. If orogastric intubation is unsuccessful decompression via right flank needle puncture is indicated. It is also suggested that right flank needle puncture is recommended as a first attempt at decompression in severely depressed metabolically deranged patients.

Orogastric Intubation Technique: The stomach tube is measured to the last rib and marked with a piece of tape. A stiff GDV, foal or mare stomach tube with a smooth bevelled tip works best (having several diameter and stiffness tubes is ideal). Apply generous lubrication to the tube. Place a functional mouth speculum; generally a roll of 2" tape secured in the mouth with tape encircling the muzzle. As the stomach tube is passed, you will often meet resistance at the lower esophageal sphincter. Pass the tube firmly in a twisting manner to encourage the tube to pass through the lower esophageal sphincter.

If unsuccessful, place the patient in various positions and attempt to pass the tube (i.e., elevate animal at 45 degree angle with rear feet on floor and front feet on the table, right lateral recumbancy, and left lateral recumbancy). This movement may encourage the stomach to rotate enough to allow the tube to pass into the stomach. Be careful not to position the patient in dorsal recumbancy as this will increase abdominal visceral pressure on the caudal vena cava and may exacerbate signs of shock.

If still unsuccessful, try different diameter tubes; try a smaller diameter, more flexible tube and proceed as described above.

If still unsuccessful, attempt to remove some of the air in the stomach by placing an l8 gauge needle at the point of distention in the right flank region. Ping the area to make sure the spleen is not under the proposed trocarisation site. After trocar decompression, attempt to pass the stomach tube as described above.

If still unsuccessful, sedate the dog with a narcotic (e.g., Oxymorphone) and try to pass the tube again. Mild sedation is recommended if the patient strongly resists physical restraint.

Success in passing a stomach tube depends on the skill of the operator and available assistants.

If you are successful at passing a stomach tube and plan to refer the patient to a referral surgical center for gastropexy, transport the patient with the tube remaining in the stomach (i.e., taped to the mouth) or bring the tube out through a pharyngostomy incision or place a nasogastric tube.

If a stomach tube was successfully passed, stomach contents should be evaluated for color and presence or absence of necrotic looking gastric mucosa. This may give an impression of gastric viability.

Fluids: Shock dosage of polyionic isotonic fluid is carefully administered to expand the vascular compartment. Patients are frequently monitored during fluid administration to help determine ultimate fluid rate and amount. One or two indwelling cephalic catheters are generally placed.

Referral: If you are successful at passing a stomach tube and plan to refer the patient to a referral surgical center for gastric derotation and gastropexy, transport the patient with the tube remaining in the stomach (i.e., taped to the mouth) or bring the tube out through a pharyngostomy as described below.

Pharyangostomy tube placement:

a. Orally palpate the fossa lateral to the hyoid apparatus until a lateral bulge is seen C:\Users\SBeatty\AppData\Local\Microsoft\Windows\INetCache\Content.Outlook\V367Q759\Figure 01.pdf

b. Make a small skin incision over the bulge and press a curved forceps (substitute for finger) through the soft tissues and skin incision.

c. Pull the stomach tube through the incision with curved forceps; then pass the tube over the arytenoid cartilages, down the esophagus, and into the stomach (measure to the 13th rib).

Disadvantages include: heavy sedation or general anesthesia is necessary for placement of the tube.

Rarely a temporary gastrostomy may need to be performed. C:\Users\SBeatty\AppData\Local\Microsoft\Windows\INetCache\Content.Outlook\V367Q759\Figure 02.pdf. The patient is placed in left lateral recumbancy with the right flank area clipped and surgically prepared. Heavy sedation and local infiltration of lidocaine or light general anesthesia is performed. A 4 - 5 cm incision is made in the skin over the point of greatest gastric distention (generally 1 - 2 cm caudal to the 13th rib and 2 - 3 cm distal to the transverse processes of the lumbar vertebrae). A grid technique is used to gain entrance into the peritoneal cavity. Due to severe gastric distention the stomach wall is pressed against the abdominal wall and thus easily identified through the flank incision. The stomach wall is sutured to the skin using a simple continuous pattern with 3-0 Maxon. This is done prior to incising into the stomach lumen. A #11 BP scalpel blade is used to puncture into the lumen of the stomach. Gas and stomach contents are expelled under pressure so stand back! The gastric mucosa is evaluated for viability. Disadvantages of gastrostomy include: the stomach is sutured in its rotated position and more time is required when definitive surgical treatment is performed due to the necessity of closing the gastrostomy.

Successful stomach tube placement:

Once the stomach tube has been passed into the stomach or gastrostomy performed, the stomach is lavaged with warm water. If a stomach tube was successfully passed, the stomach contents should be evaluated for color and presence or absence of necrotic gastric mucosa. This may give an impression of gastric viability.

Surgical Treatment :

A specific 'Surgical Plan' should be in mind before entering the operating room theatre. This will improve the efficiency of surgery and thus decrease overall surgery time. The 'authors' surgical plan is as follows:

Stand on the right side of the patient.

Provide generous abdominal exposure via xyphoid to pubis midline laparotomy.

Remove of all of the falciform ligament to the level of the xyphoid.

Place a 10'' Balfour self-retaining abdominal retractor (metal frame toward the head) with full retraction.

Confirm that the omentum is draped over the exposed surface of the stomach (pathagnomonic for GDV).

Attempt derotation by:

Standing on the patients' right side, first reach your right hand across the abdomen and place it between the left body wall and dilated stomach.

Slide your right hand along the sublumbar body wall and grasp the deep (dorsal) aspect of the stomach at the level of the spine.

Next, place the open palm of your left hand on the exposed surface of the right side of the dilated stomach.

Using both hands simultaneously, pull the deep part of the stomach with your right hand to begin derotation whilst you push the right surface of the stomach down toward the patients sublumbar body wall with your left hand. This maneuver will be successful in the majority of cases.

See this maneuver performed on the Emergency Surgery I, Gastrointestinal Surgery I, and Soft Tissue Surgery II DVD's available at www.videovet.org.

Once the stomach is derotated, evaluate the stomach for evidence of questionable viability (particularly the greater curvature and fundus) and for evidence of gastric motility.

Next, exteriorize the spleen from the abdominal cavity. Evaluate color, texture, blood flow (splenomegaly is often present and is NOT an indication for splenectomy). Splenectomy is rarely performed but may be necessary if splenic vessels are thrombosed (veins feel like threads or rubber bands).

If the stomach is full of air or fluid it should be emptied prior to attempting derotation.

If the stomach is full of food and several attempts to derotate (see author's technique above) are unsuccessful, perform a gastrotomy and manually remove the food from the stomach lumen. Suture the gastrotomy and attempt derotation again.

COMMENCE YOUR GASTROPEXY PROCEDURE.

Incisional gastropexy: This technique is based on a 3-4cm long seromuscular antral incision sutured to a similar length incision in the transversus abdominus muscle. This is the authors' technique of choice for permanent gastropexy.

With the Balfour retractors still in place visually locate the ideal position for the antral wall incision. It should be located equidistant between the pylorus and gastric incisure and equidistant between the greater and lesser curvature of the stomach. A 4cm longitudinal sero-muscular incision is made in this antral location. An easy way to safely make the sero-muscular incision is to grasp the full thickness antral wall with your thumb and finger at the site of the proposed incision, gently retract the wall of the stomach until you feel the mucosa and submucosa 'slip' out of your thumb and finger. The tissue remaining between your thumb and finger is the sero-muscular layer of the antral wall. Using a straight or curved Metzenbaum scissors cut the tissue remaining in your thumb and finger resulting in a perfect depth of the sero-muscular incision. Extend the incision to a 4cm length and gently undermine the edges to allow generous suture bites in the stomach wall during gastropexy.

Once the antral incision is completed remove the Balfour retractors. When selecting the location on the transversus abdominus muscle for the gastropexy, it is important to first visualize the location of diaphragmatic muscle fibers as they radiate into the abdominal cavity and attach near the costal arch. It is important that the gastropexy site be at least 2cm caudal to the diaphragm muscle insertion. After identifying the attachment of the diaphragm, the bleeding surface of the antral incision is brought to the right body wall. With the stomach in a normal position, the bleeding antral surface is touched to the peritoneal wall approximately 3-4 cm deep to the abdominal wall incision and 2cm caudal to the insertion of the diaphragm. A blood mark is created on the peritoneum at this proposed location. This will be the site for the permanent gastropexy. The peritoneum and transverses abdominus muscle are then incised creating a mirror image defect of the antral incision. The incisional defect in the stomach is then sutured to the incisional defect in the abdominal wall. The defects are sutured in two layers using a simple continuous pattern with 2-0 or 3-0 monofilament or multifilament synthetic absorbable suture.

Belt Loop Gastropexy: This technique is based on the construction of a sero-muscular antral flap attached around a segment of transversus abdominus muscle. A

horseshoe shaped incision is made in the serosal layer of the antral portion of the stomach with its base at the greater curvature. The sero-muscular portion of the stomach is identified by grasping full thickness antral wall between the thumb and index finger and "slipping" the mucosal and submucosal layers away so only the sero-muscular portion of the wall remains between thumb and finger. The sero-muscular layer is incised with scissors and the horseshoe shaped sero-muscular antral flap is dissected and elevated of the submucosal layer. The stomach is replaced in the abdominal cavity in normal position and the sero-muscular flap lined up with the transversus abdominus muscle. Once this optimal location is discovered, two longitudinal incisions (along the fibers of the transversus m.) are made in the transversus abdominus m. The segment of muscle between the incisions is undermined. The sero-muscular flap from the stomach (i.e., belt) is passed through the transversus abdominus m. (i.e., loop) and sutured to itself to complete the "Belt-Loop" gastropexy. 2-0 or 3-0 monofilament absorbable synthetic suture in a simple interrupted or continuous pattern is used to secure the flap in place. Advantages of belt loop gastropexy include: it is relatively easy to perform alone and in the middle of the night, it can be performed quickly, and it is an effective means of permanent gastropexy.

POSTOPERATIVE MANAGEMENT

In most cases 3 to 4 days of intensive monitoring is necessary for the successful management of GDV patients. Postoperative considerations are listed below:

a. Shock is a postoperative possibility and the patient should be monitored and treated accordingly.

b. Patients are generally held off food and water for 24 hours following surgery. During this time maintenance fluids should be supplied using polyionic isotonic crystalloid fluid. Vomiting may occur following surgery; the NPO period should be extended accordingly. Gastritis and gastric motility disorder may be seen in post op GDV patients.

c. After 24 hours of no vomiting, oral alimentation should begin gradually with

d. sequence of ice cubes, water, and finally canned dog food. This should occur over a 2-3 day period.

d. Antibiotics should be continued for 7 - 10 days.

e. Routine surgical complications such as infection, dehiscence, seroma, etc. should be watched for and treated accordingly.

f. EKG monitoring: the most common severe postoperative complication is cardiac arrhythmia. Approximately 75% of GDV patients will develop arrhythmia's in the immediate postoperative period. Arrhythmia's can be present at the initial time of presentation but most often occur within 24 - 72 hours after surgery. Ventricular premature contractions, progressing to ventricular tachycardia is most common. Etiology is unknown but shock, hypoxia, acid base alterations, endotoxins, myocardial depressant factor (MDF), reperfusion injury, release of free radicals, and hypokalemia have been identified. Occurrence of a total body potassium deficit has been proposed. Etiology of the hypokalemia includes anorexia, vomiting, tremendous outpouring of potassium rich fluids into a dilated stomach, and use of potassium poor fluids in treatment of shock. For this reason, adding 20-30 mEq of potassium chloride per liter of maintenance fluids during and after surgery are recommended.

g. Gastric motility: occasionally GDV patients develop postoperatove gastric motility abnormalities. Patients with gastric hypomotility or gastric stasis noted at the time of surgery should be treated with a motility modifier (i.e., metaclopramide, erythromycin, etc).

9002

THE 15 MINUTE GASTROPEXY

SURGERY

Howard Seim, DVM, DACVS

Information for 15 Minute Gastropexy can be found included in the Surgical Management of GDV proceedings.

9003

SPLENECTOMY

SURGERY

Howard Seim, DVM, DACVS

INTRODUCTION

Splenectomy can be a life-saving procedure and is often necessary on an emergency basis. Unfortunately, most dogs that present with a spontaneous hemoabdomen associated with a splenic bleed have neoplasia as the underlying etiology, although benign lesions such as hematomas may also be seen. Stable dogs with non-ruptured splenic masses are also candidates for splenectomy. Spontaneous hemoabdomen is a challenging condition that requires rapid diagnosis with timely therapeutic intervention to maximize the chance of a successful outcome. Unfortunately, malignant neoplasia is the most common etiology and despite a successful short-term outcome, a guarded long-term prognosis is common. The peritoneal cavity can be considered a large potential space in which the majority of a dog's blood volume can reside. Consequently, with rupture of a highly vascular intra-abdominal organ, vascular collapse and end-organ ischemia can result rapidly. The major objectives of the veterinarian who is treating a patient with spontaneous hemoabdomen include rapid and effective resuscitation, timed surgical intervention, rapid identification of the point of hemorrhage and efficient elimination of the source of hemorrhage.

INDICATIONS

Splenectomy is indicated for removal of splenic neoplasm, rupture, torsion, infarct, abscess and hypersplenism.

PATIENT POSITIONING

The patient is placed in dorsal recumbency for routine celiotomy.

RECOMMENDED INSTRUMENTS

A Balfour self-retaining abdominal retractor is essential to maintain adequate exposure allowing complete exploration of the abdominal cavity as well as visualization of the splenic blood supply. When large amounts of blood or fluid are present in the abdominal cavity suction, using a Poole suction tube, is helpful. It is best to have a variety of sizes of hemostats available. The author recommends a minimum of 6 medium to large hemostatic forceps (Crile, Kelly or Carmalt) and 4 – 5 small hemostatic forceps (mosquito).

Ligation of individual blood vessels or clusters of vessels is performed using 2-0 or 3-0 synthetic absorbable suture material. Common sutures include Biosyn, Monocryl, Dexon, Vicryl, Polysorb, PDS or Maxon. A secure friction knot such as a Strangle knot, Double Half Hitch or Modified Miller's knot is recommended for secure vascular ligations.

SURGICAL TECHNIQUE

A ventral midline incision from xyphoid to pubis is made to allow adequate exposure of all abdomen organs. The falciform ligament is removed from its attachment to the body wall and xyphoid and a large (10'') Balfour self-retaining retractor is positioned (with the frame of the Balfour toward the cranial aspect of the incision) to provide exposure of the abdominal cavity.

The spleen is located in the cranial left quadrant of the abdominal cavity just caudal to the greater curvature and fundus of the stomach. The spleen is identified, and gently elevated through the abdominal incision. If the surgeon is dealing with a bleeding spleen (e.g., hemangiosarcoma) the exteriorized spleen is placed across the body wall to help place pressure (tether) on the splenic blood vessels. In addition, a dry laparotomy pad can be placed directly on the point of hemorrhage and gentle pressure applied. At this point a rapid and complete abdominal exploratory is performed to rule-out obvious metastasis.

Prior to splenectomy several structures should be identified. The greater curvature of the stomach,

dorsal and ventral layers of the greater omentum, the gastrosplenic ligament and the left limb of the pancreas. These structures are best visualized by entering the epiploic foramen. To do this elevate the greater omentum from the abdominal cavity. The omentum consists of two 'leaves'. Pull the two leaves apart and break into the omental foramen. Work your way down to the splenic vasculature and left limb of the pancreas. Trace the splenic artery and vein as they course from the dorsal layer of the greater omentum into the gastrosplenic ligament. Identify the left gastroepiploic artery and vein, the many splenic arterial and venous branches into the hilus of the spleen, the short gastric vessels and the vessels continuing into the greater omentum.

The spleen receives its blood supply from 3 major sources. Three to four short gastric vessels supply the cranial aspect of the spleen. The central portion of the spleen is supplied by the major splenic artery and vein and the caudal pole of the spleen by 4-5 small omental tributaries.

Once the splenic vasculature has been identified the spleen can safely be removed using a technique requiring only 3 to 4 cluster ligations. Visualization of these vessels is accomplished by first elevating the spleen from the abdominal cavity. When attempting to exteriorize the spleen it is noted that its cranial pole is tethered to the greater curvature of the stomach by the 3 to 4 short gastric vessels. These vessels are identified and cluster ligated with two encircling ligatures. The vessels are transected between ligatures thus releasing the tethering effect. The spleen can now be further mobilized from the abdominal cavity allowing easy exposure of all remaining vessels.

Next the major splenic artery and vein is located and ligated prior to its bifurcation. Care should be taken to visualize the left limb of the pancreas and make certain it is a safe distance from the proposed ligation site. The splenic artery and vein are generally double ligated and depending upon size the artery can be transfixed. Finally, the remaining vessels supplying the caudal pole of the spleen are cluster ligated using one or two ligatures.

During the procedure, several points should be remembered:

1. when ligating the splenic artery and vein, identify the location of the pancreas and do not occlude its blood supply
2. double ligate all major vessels
3. carefully inspect all ligated vessels for evidence of hemorrhage

CLOSURE

The Balfour retractor is removed and the abdominal incision is closed in a routine fashion.

POSTOPERATIVE CONSIDERATIONS

Postoperative care involves monitoring the patient for blood loss that may be encountered should a ligature slip from the ligated vessels.

9004

SURGICAL MANAGEMENT OF CANINE CALCULI

SURGERY

Howard Seim, DVM, DACVS

If you would like a copy of the video of these surgical procedures go to www.videovet.org.

KEY POINTS

- Retropulsion of urethral calculi into the urinary bladder simplifies management of urethral calculi
- Aggressive lavage of the urethra and bladder should be performed during cystotomy
- Permanent urethrostomy is an acceptable method of managing chronic stone formers

Definition: Cystic and urethral calculi have various compositions (i.e., oxalate, struvite, urate, uric acid, cystine, silicate) and may be present in the urinary bladder or lodged in the urethra, respectively. They may be multiple or single, may cause partial or complete obstruction (i.e., urethral), and may require surgical manipulation for removal.

DIAGNOSIS

Clinical presentation:

Signalment: There is no age predisposition. Dalmations are more likely to present with uric acid calculi and commonly present with calculi lodged in the urethra. Schnauzers are more likely to present with struvite calculi and Daschunds are more likely to present with cystine stones.

History: Patients generally present with a history of urinary obstruction and/or signs of urinary tract infection. Common complaints include difficulty urinating, straining to urinate, hematuria, dripping blood tinged urine from the prepuce, and/or a distended abdomen. Patients that present several days after complete obstruction may have a distended and painful abdomen and a history of anuria. These patients may be so compromised that they present in shock.

Clinical signs: The most frequently reported clinical signs in patients with cystic and urethral calculi include unproductive straining to urinate, blood tinged urine dripping from the prepuce, hematuria, and/or polakiuria. Severity of clinical signs may vary with the degree of urethral obstruction and duration of obstruction prior to presentation. Patients with complete obstruction for several days may show signs of post-renal azotemia (i.e., severe depression, recumbant, shocky).

Physical examination: Observation in the examination room may reveal multiple unsuccessful attempts to urinate. Abdominal palpation may reveal a full urinary bladder; occasionally, calculi within the bladder may be palpable.

Patients with severe clinical signs (i.e., presented several days after complete obstruction) may show azotemia, shock, and/or severe depression. Abdominal palpation generally reveals a large, turgid urinary bladder and may result in discomfort to the patient.

Laboratory findings: Results of a complete blood count and serum chemistry profile are generally normal in patients presenting acutely; urinalysis may show evidence of urinary tract infection and and/or crystalluria.

Patients presenting after several days of complete obstruction may have significant changes in their biochemical profile including increased BUN, increased creatine, metabolic acidosis, and severe electrolyte abnormalities. Urine is generally grossly hemorrhagic and urinalysis may show signs of urinary tract infection and crystaluria.

Radiography: Survey radiographs may show presence of radiodense calculi in the urethra and/or urinary bladder as well as a distended urinary bladder. Occasionally, radiolucent calculi occur and can only be visualized using retrograde contrast cystourethrography. The most common location of urethral calculi in male dogs is immediately caudal to the os penis. Careful evaluation of the kidneys and ureters should be done to rule out renal and ureteral calculi.

Ultrasound: Ultrasonographic examination of the bladder, ureters, and kidneys may be helpful in diagnosis of cystic, ureteral, or renal calculi.

Differential diagnosis: Any disorder causing urinary obstruction, including urethral neoplasia, granulomatous urethritis, urethral stricture, and urethral trauma. Definitive diagnosis is based on clinical signs, inability to pass a catheter, and evidence of calculi on survey or contrast radiographs.

Medical management: Immediate care: In animals with complete obstruction of a duration long enough to cause azotemia, temporary urinary diversion is provided by either passing a small urinary catheter (e.g., 5 French) alongside the calculus, performing a prepubic cystostomy (see technique described below), or frequent cystocentesis (i.e, tid to qid). Azotemia is treated with crystalloid IV therapy prior to calculus removal.

Retrograde hydropulsion: Go to www.videovet.org for a detailed video of this technique.

This technique should result in a 90-95% success rate of retropulsing even the most difficult urethral calculi into the urinary bladder!

Technique

1. Select the largest diameter sterile high density polypropylene urinary catheter that will fit past your patients os penis (generally 6, 8, or 10 French diameter)
2. If the selected catheter turns out to be a 6 French diameter then mix 30cc of Sterile KY Jelly with 70cc of sterile physiologic saline solution.
3. If the selected catheter turns out to be an 8 or 10 French diameter then mix 40cc of Sterile KY Jelly with 60cc of sterile physiologic saline solution.
4. Thoroughly mix the sterile saline and KY Jelly in a 35 or 60 cc syringe and attach the syringe to the urinary catheter.
5. Anesthetize the animal, extrude the penis and pass the lubricated urinary catheter in the urethra up to and against the calculus. Place a dry gauze sponge around the extruded tip of the penis and occlude the penis around the catheter by squeezing it with thumb and finger.
6. Using a back and forth action on the catheter, simultaneously inject the saline/lubricant mix under extreme pressure. Be certain the catheter tip hits the calculus like a battering ram to help dislodge it and encourage the saline-lubricant mix to surround the calculus and coat the urethral wall. During injection the calculi and urethra are lubricated by the saline/lubricant mix while the viscosity of the mixture (i.e., KY jelly and saline) encourages the calculus to dislodge and become retropulsed into the urinary bladder.

This technique is successful regardless of how many stones are in the urethra and no matter where the calculi are lodged.

If the above technique fails, place a finger in the rectum, palpate the urethra and occlude its lumen (this dialates the urethra); repeat the above maneuvers and when maximum pressure is exerted on the urethra by the saline/lubricant mix (i.e., the urethral is maximally dialated), suddenly release digital urethral occlusion allowing lodged calculi to flush into the urinary bladder.

Surgical treatment: The objective of surgical treatment is to remove all retropulsed calculi from the urinary bladder and any remaining urethral calculi that were unable to be retropulsed. Bladder calculi are removed via cystotomy, urethral calculi are removed via urethrotomy, and patients that are frequent stone formers may benefit form a permanent urethrostomy to allow continual passage of small urethral calculi.

Preoperative management: Patients that present acutely can be anesthetized immediately and retropulsion

attempted (see above described technique). If urinary tract infection is suspected, preoperative treatment with antibiotics may be instituted.

Patients that present after several days of complete obstruction should be treated medically until the azotemia resolves, blood gas abnormalities resolve, and electrolytes return to normal. The patients electrocardiogram should be monitered if hyperkalemia is present preoperatively. Medical treatment may consist of intravenous fluids, systemic antibiotics, continuous ECG monitoring, and bladder decompression. Bladder decompression may be accomplished via passing a small gauge urinary catheter (e.g., 5 French) past the calculus, multiple cystocentesis (i.e., tid or qid), or placement of a antepubic cystostomy tube (described in detail below).

Anesthesia: Routine general anesthesia is performed in patients that present acutely without signs of azotemia.

Azotemic, shocky patients with moderate to severe biochemical abnormalities should be treated as described above until these abnormalities return to normal.

Surgical anatomy: The male canine penile urethra consists of urethral mucosa (i.e., urothelium) surrounded by corpus cavernosum urethra, which is in turn surrounded by tunica albuginea. Because of the fluid filled corpus cavernosum urethra (blood) and the tough fibrous connective tissue tunica albuginea, the urethra can withstand tremendous pressure (e.g., as with aggressive retropulsion) without the fear of urethral rupture.

The urinary bladder consists of the following layers; serosa, muscular, submucosa and mucosa. The bladder is lined with transitional epithelium.

Positioning: Patients are positioned in dorsal recumbancy for retropulsion, urethrotomy, urethrostomy, cystostomy tube placement and cystotomy.

Surgical technique: The surgical techniques vary depending upon the procedure chosen, and are described in detail below:

Retropulsion: The technique for retropulsion of urethral calculi is described above in medical management.

Percutaneous cystostomy tube placement: Occasionally, it may be necessary to perform a percutaneous antepubic cystostomy to decompress the urinary bladder whilst treating a severely azotemic patient until they become a better anesthetic and surgical risk.

The patient is sedated and placed in dorsal recumbancy. A 3-4cm incision is centered between the umbilicus and pubis. Subcutaneous tissues are disected to expose the ventral midline (i.e., linea alba). A 2-3cm incision is made in the linea alba and the bladder wall located. A 12–14 French Foley catheter is advanced through a skin incision 2-3 cm lateral to the abdominal incision, tunneled in the subcutaneous tissue and brought into the abdominal cavity at a location just lateral to the midline abdominal incision. A pursestring suture is placed in the bladder wall at the proposed site of Foley catheter placement with 3-0 monofilament absorbable suture. A 1cm incision is made into the bladder lumen and the Foley catheter advanced. The pursestring suture is carefully tightened to create a water-tight seal, but not to tight as to create bladder wall necrosis. The bladder wall is pexied to the abdominal wall at the point of entry of the Foley catheter with 3-0 monofilament absorbable suture in a simple interrupted pattern. The abdominal wall is closed in a routine fashion.

The cystostomy catheter is held in place with a Chinese finger trap friction suture technique using #1 monofilament nonabsorbable suture and attached to a closed collection system to avoid urinary tract infection. The cystostomy tube remains in place until the patient is ready for definitive surgical treatment.

Urethrotomy: Go to www.videovet.org for a detailed video of this technique.

The urethral calculus to be removed is located by evaluation of radiographs, palpation of the os penis and its relationship to the calculus, and/or passing a catheter in the urethra until it contacts the stone, removing the catheter and using it as a measure to locate the calculus.

A 2–3 cm skin incision is made directly over the calculus. Subcutaneous tissues are dissected until the retractor penis muscle is exposed. The cream colored retractor penis muscle (smooth muscle) is dissected off the corpus cavernosum penis (the corpus cavernosum penis has a bluish tint from venous blood) and retracted laterally. A sharpe #15 BP scalpel blade is used to incise the urethra directly over the calculus being careful to incise the urethra directly on its midline to help decrease cavernous sinus

bleeding. No attempt is made to control cavernous sinus hemorrhage with cautery or hemostats as this creates excessive urethral trauma and is generally unsuccessful at controling hemorrhage. Rather, hemorrhage is controlled via digital pressure and suction until suturing can commence. The calculus is grasped with forceps and removed from the urethra.

The urethral incision can be closed using 5-0 multifilament or monofilament absorbable suture in a simple interrupted or continuous pattern. Subcutaneous tissues are closed with 3-0 monofilament absorbable suture in a simple continuous pattern and skin with 3-0 or 4-0 nonabsorbable monofilament suture. This method is preferred by the author over healing by second intention as postoperative hemorrhage is significantly reduced.

Alternatively, the urethral incision can be left open to heal by second intention; if this method is chosen moderate to severe hemorrhage can be expected for several days postoperatively.

Both urethrotomy techniques (i.e., sutureless or sutured) result in perdictable urethral healing without evidence of urethral stenosis or stricture.

Scrotal urethrostomy: Go to www.videovet.org for a detailed video of this technique.

Urethrostomy is generally performed in patients that are recurrent stone formers. It provides a permanent opening caudal to the os penis that is large enough to accommodate passage of most urethral calculi. This technique is often performed in Dalmations for treatment of recurrent uric acid calculi.

Scrotal urethrostomy is the location of choice for urethrostomy in dogs. It is a convienent location for surgical manipulation, this area of the urethra generally bleeds the least, the urethral diameter will accommodate passage of most urethral calculi, and there is less urine scald postoperatively. Other locations for urethrostomy include prescrotal and perineal.

Prior to surgery a urethral catheter (the largest size that will fit past the os penis) is passed, if possible. After a routine castration and scrotal ablation have been performed, the subcutaneous tissues are dissected to expose the retractor penis muscle. The retractor penis muscle is smooth muscle and appears light grey to cream colored. The retractor penis muscle is dissected from its attachment to the corpus cavernosum urethra. The blood filled cavernous tissue gives the urethra a bluish color. The urethral catheter is palpated and used as a firm surface to cut against when incising the urethra. Every attempt is made to incise the urethra exactly on the midline to help decrease hemorrhage. A 3–4 cm incision is made in the urethra. The caudal aspect of the urethral incision is positioned directly over the ishial arch. As this is the new point of urine flow it is most efficent to have urine exit before it makes a sharp turn ventrally.

No attempt is made to control cavernous tissue hemorrhage with cautery or hemostatic forceps; only pressure, suction, and suture pressure should be used.

After incision of the urethra, the glistening urethral mucosa is identified, 4-0 or 5-0 nonabsorbable monofilament suture with a swaged on cutting or taper-cut needle is recommended by the author to suture urethral mucosa to skin. The first urethrostomy suture is placed at the midpoint of either side of the urethral incision to include urethral mucosa, tunica albuginea, and skin (suture split thickness of skin). The suture is tied leaving the end without the needle 3-4 cm long to act as a stay suture. The second suture is placed directly across from the first suture and tied as described for the first. The urinary catheter can now be removed. After the first two sutures are placed, the needle end of one suture is used to begin suturing the cranial portion of the urethrostomy using a simple continuous suture pattern.

When the opposite suture is encountered, the stay suture is used to tie off the first continuous suture line. The opposite suture is then used to suture the caudal portion of the urethrostomy in a similar fashion tying the final suture to the remaining stay suture.

Fine ophthalmic instruments make tissue handling and suturing easier. Use of a magnifying loupe (about 2x) and head lamp light source enhances visualization of the urethral mucosa and facilitates accurate suturing. It is critical that the surgeon recognize glistening urethral mucosa and suture it to skin. This will decrease (or eliminate) the chance of urethral stricture. It has been shown that a continuous suture pattern incorporating the urethral mucosa and tunica albuginea (i.e., squeezes the cavernous tissue) results in less postoperative hemorrhage.

Cystotomy: Go to www.videovet.org for a detailed video of this technique.

After successful retropulsion of urethral calculi into the bladder, the catheter used to retropulse calculi is passed into the urethra and bladder and left in place. A portion of the catheter can be left exiting the penis. Leaving a catheter indwelled in the urethra ensures that remaining cystic calculi will not roll back into the urethra during patient transfer to the surgery suite and during patient prep.

Just prior to aseptic preparation of the abdomen a soft, 10-12 French red rubber catheter or feeding tube is placed into the prepuce and a prepucal lavage is performed with 180cc of a 1:50 dilution of saline and 1% betadine solution. This aseptically prepares the penis and prepuce so they can remain in the surgical field throughout the cystotomy procedure. In female patients the vulva and vaginal vault are similarily asepticlly prepared.

A paraperpucial incision is made from just caudal to the umbilicus to pubis. The prepuce is retracted and a midline celiotomy is performed. The bladder is exteriorized and examined. Stay sutures of 3-0 suture are placed in the apex and neck of the bladder. A scalpel blade is used to penetrate the ventral aspect of the bladder and enter the lumen. The ventral cystotomy incision is extended with metzenbaum scissors. The bladder should be opened from apex to neck to allow proper visualization of bladder mucosa and calculi. Stay sutures are placed on each side of the incision at its midpoint to facilitate visualization of the bladder interior. Large hemostats are placed on the stay sutures to help retract the bladder margins to maintain visualization of the bladder interior. A cystotomy spoon is used to scoop the bladder neck for calculi. This is performed several times. When no more calculi can be removed with the spoon, digital palpation of the bladder neck is performed to identify presence of further calculi. If further calculi are palpated further attempts are made to retrieve the calculi. Once no more calculi can be spooned or palpated the previously plced indwelling urethral catheter is removed.

Next, the largest urinary catheter or feeding tube that can be passed through the os penis is passed in the penile urethra to the level of the os penis (i.e., retrograde). A dry sponge is used to grasp the extruded penis to create a water tight seal around the catheter. A 60cc syringe filled with sterile saline is injected through the catheter under moderate pressure. The stay sutures on the bladder incision are retracted to enable visualization of the bladder lumen during lavage. Suction or intermittent spooning is performed during lavage in an attempt to identify and remove any remaining stones. After several lavages and negative results in obtaining stones, the catheter is placed from the bladder to the bladder neck and pelvic urethra (i.e., normograde). Lavage is once again performed in an attempt to identify and remove any remaining stones. After several lavages and negative results the catheter is advanced until it can be seen coming out of the penile urethra. The catheter is run back and forth in the urethra several times to ensure that there are no remaining calculi (i.e., gritty feeling while passing the catheter).

Finally, a piece of bladder mucosa is excised from the cut edge of the cystotomy incision for culture and susceptability testing. The interior of the bladder is examined for urachael diverticulm, masses, etc. and biopsied as necessary. The bladder wall is closed with 3-0 or 4-0 absorbable monofilament suture material using a swaged on taper or taper-cut needle in a simple continuous or simple interrupted appositional suture pattern. Only one layer closure is necessary. Abdominal closure is routine.

Suture material/special instruments:

Urinary catheters of various sizes, Foley catheter, head lamp light source, 2X loupes, ophthalmic instruments, 4-0 and 5-0 monofilament absorbable suture material.

POSTOPERATIVE CARE AND ASSESSMENT:

Postoperative care varies depending upon procedure performed:

Percutaneous cystostomy tube: It is important to keep the percutaneous cystostomy tube attached to a closed collection device. The tube can be connected to a sterile collection bag via a sterile intravenous catheter connection set. An elizabethan collar may be necessary in some patients to prevent iatrogenic removal of the cystostomy catheter. Careful management is important to control catheter related urinary tract infection.

Sutureless Urethrotomy: If urethrotomy without suturing is performed, patients must be monitored for blood loss from the urethrostomy site. Blood loss can be severe enough to lower the PCV by 2 – 3%. An Elizabethan

collar may be necessary in some patients to prevent self-mutilation. Patients should be kept quiet and away from other animals (especially bitches in heat!). Tranqulization is occasionally necessary to control hyperactive or overly excitable patients. Clients should be warned that drops of blood may be present from the urethrotomy site as long as 2 weeks postoperatively.

Sutured Urethrotomy: If a sutured urethrotomy is performed, patients will exhibit very little blood loss. However, an Elizabethan collar should be considered, especially in patients that may be prone to self-mutilation. Patients should be kept quiet and away from other animals (especially bitches in heat!). Tranqulization is occasionally necessary to control hyperactive or overly excitable patients.

Scrotal Urethrostomy: The most common postoperative complication of scrotal urethrostomy is bleeding from the urethrostomy site. Utilization of a simple continuous suture pattern incorproating the urethral mucosa and tunica albuginea (i.e., squeezing the cavernous tissue and creating a air-tight/water-tight seal) has significantly decreased the incidence of postoperative hemorrhage in the authors opinion. An Elizabethan collar should be considered, especially in patients that may be prone to self-mutilation. Patients should be kept quiet and away from other animals (especially bitches in heat!). Over excitement immediately postoperatively can result in frank hemorrhage or subcutaneous hemorrhage. Tranqulization is occasionally necessary to control hyperactive or overly excitable patients.

Cystotomy: An indwelling urethral catheter is not recommended after an uncomplicated cystotomy for removal of cystic calculi. An Elizabethan collar should be considered, especially in patients that may be prone to self-mutilation. Patients should be kept quiet and away from other animals.

Prognosis: The prognosis for surgical management of urethral and cystic calculi is dependant upon preoperative management of azotemic patients prior to anesthesia, success of retropulsion of urethral stones into the urinary bladder, care in removing all stones via cystotomy, and care of ensuring urethral mucosa to skin apposition during urethrostomy.

Patients that have successful retropulsion of urethral calculi and do not require urethotomy or urethrostomy have a excellent prognosis. If careful attention is paid during cystotomy to ensure that no calculi are left behind (see discussion on cystotomy technique), the prognosis for cure is excellent. Long term prognosis is dependant on evaluaiton of calculus composition, dietary management, management of urinary tract infection, and attention to urine pH.

Patients that require sutured or sutureless urethrotomy have a favorable prognosis if all of the remaining calculi are removed from the urinary bladder via cystotomy to ensure that no calculi are left behind (see discussion on cystotomy technique). Attention must be paid to careful lavage during cystotomy to ensure removal of all cystic calculi.

Patients that have an elective urethrostomy have a favorable prognosis if attention is paid to proper surgical technique (i.e., urethral mucosa is sutured to skin). Occasionally, chronic stone forming patients will form a calculus that is to large to pass through the urethrostomy stoma.

9005

INTESTINAL ANASTOMOSIS

SURGERY

Howard Seim, DVM, DACVS

If you would like a copy of the video of these surgical procedures go to www.videovet.org.

KEY POINTS

- Pay attention to basic surgical principles
- Submucosa is the layer of strength
- Use synthetic absorbable suture materials
- Appositional techniques are best
- Intestinal sutures should engage at least 3 - 4 mm of submucosa
- Intestinal sutures should be no further apart than 2 - 3 mm
- Always handle bowel wall using atraumatic technique
- Examine the integrity of your anastomsis visually
- 50 - 60% of the 'small intestine' of dogs and cats can be resected

GENERAL PRINCIPLES OF SMALL INTESTINAL SURGERY

1. Incorporation of the collagen laden submucosal layer in the surgical closure.
2. Minimize trauma and contamination.
3. Maintain good blood supply to the surgical site.
4. Avoid tension across the suture line as this may increase the possibility of leak and/or breakdown.
5. Pay attention to your established criteria when suturing intestinal defects.

OPERATIVE CONSIDERATIONS

1. Proper "packing off" of the surgical field using moistened laparotomy pads should be performed around the exteriorized bowell to prevent accidental abdominal contamination from intestinal contents.
2. Keep abdominal contents warm and moist throughout surgery with a warm, balanced electrolyte solution.
3. Handling abdominal viscera should be kept to a minimum. Gentle manipulation of intestine with moistened gloves or stay sutures is helpful in preventing unnecessary tissue trauma. DeBakey forceps are the most atraumatic forceps for handling abdominal visceral organs.
4. The collagen laden, tough submucosa is the layer of strength in the small intestine; this layer must be incorporated into any small intestinal closure.
5. It may be difficult to visualize the submucosal layer due to mucosal eversion. Visualization of submucosa may be enhanced if everted mucosa is trimmed away.
6. Intestinal contents should be "milked" away from the anastomosis site. Intestinal clamps (e.g., Doyen intestinal clampS, Alice tissue forceps with a rubber feeding tube interposed, hair clips, or Penrose drains) may be used to prevent intestinal contents from contaminating the surgical site whilst manipulating intestine during anastomosis.
7. The anastomosis should be irrigated prior to its return to the abdominal cavity and instruments and gloves changed prior to abdominal closure.

8. Abdominal lavage with 2-3 liters of body temperature, sterile, physiologic saline solution should be accomplished prior to closure. The objectives of repeated abdominal lavage include dilution of bacteria and endotoxin and mechanical removal of fibrin and necrotic debris. The fluid of choice is body temperature, sterile, physiologic saline solution with no additives (i.e. betadine solution, chlorhexidine, antibiotics, etc). Lavage solution is poured into the abdominal cavity using a sterile stainless steel bowl, the abdominal viscera gently aggitated, and fluid and debris suctioned out with a suction device and a Poole suction tip. Injecting antimicrobials or other products into the abdominal cavity is not recommended.

SUTURE MATERIAL

Absorbable suture

Catgut. Catgut is NOT recommended for any visceral organ surgery. Its unpredictable absorption and rapid loss of tensile strength in such situations may result in an unacceptably high number of anastomotic leaks and / or breakdowns. Use of catgut suture in gastrointestinal surgery is not recommended.

Dexon, Polysorb, and Vicryl. Synthetic absorbable braided suture (i.e., polyglactin, poly-glycolic acid) have become very popular. The braided nature however does result in increased tissue drag and difficult knotting ability.

Biosyn and Monocryl. These sutures have similar properties to Dexon, Polysorb and Vicryl however they are monofilament. They were developed to overcome the problem of tissue drag and knot slipping found in the braided synthetic absorbables. Their predictable hydrolytic absorption is unaffected by their immediate environment (i.e., infection, contam-ination, hypoproteinemia). They retain high tensile strength for a long period of time (2 3 weeks) and have very good handling characteristics. These suture materials are ideal for use in gastrointestinal surgery. These sutures are the authors choice for gastrointestinal surgery.

PDS and Maxon. PDS and Maxon, are synthetic absorbable monofilament suture materials with similar properties to that of Dexon and Vicryl. They have been shown to retain approximately 70% of their tensile strength at 3 4 weeks, and are absorbed by hydrolysis (unaffected by infection, contamination, hypoproteinemia). These suture materials are ideal for use in gastrointestinal surgery. Possible disadvantages include stiffness, a tendency to kink and prolonged absorbtion time.

Nonabsorbable suture

Nylon, Polypropylene. Monofilament, nonabsorbables are excellent suture materials for use in contaminated or infected surgical sites. They have a high tensile strength, are relatively inert in tissue, noncapillary, and do not act as a nidus for infection. These materials pass through tissue with essentially no tissue drag and have excellent knot tying security at sizes 3 0 to 5 0.

Silk, Mersilene, Bronamid, Vetafil. Multifilament nonabsorbable sutures should NEVER be used in gastrointestinal surgery. They may harbor infection for years and may result in suture related abdominal abscesses or draining tracts.

SUTURE SIZE

For the majority of small intestinal surgical procedures in dogs, 3 0 or 4 0 size suture material is adequate; in cats, 4-0 is recommended. The tensile strength of this size suture is greater than the tensile strength of the tissues that are being sutured (i.e., intestinal wall). Larger size suture may contribute to anastomotic failure by increased trauma to tissues and its effect on the blood supply of tissue margins.

NEEDLES

Swaged-on “atraumatic” reversed cutting, narrow taper point, or fine taper cut needles can all be used for gastrointestinal surgery. The author prefers a narrow taper point needle. Needle diameter should approach the diameter of the suture.

SUTURE PLACEMENT

When suturing intestine, sutures should be placed 3 4 mm from the cut edge of the intestinal serosa and no more than 2 3 mm apart. It is important to recognize everted mucosa and be sure the 3 - 4 mm bite in the intestinal wall is not just in mucosa but engages all layers of the intestinal wall. Measure your intestinal wall bite from the cut edge of the serosa.

SUTURE PATTERNS

There is considerable controversy regarding specific suture pattern for use in small intestinal surgery. Everting, inverting, and appositional suture patterns have been used experimentally and clinically for suturing enterotomies and anastomoses. Appositional patterns are recommended as they cause little lumen compromise postoperatively.

Everting: Everting patterns (i.e., horizontal mattress) have been shown to encourage adhesions and result in lumen stenosis. This technique is NOT recommended. The everting technique is not to be confused with the mild eversion of mucosa that occurs in the appositional techniques described below.

Inverting: In small animals adequate lumen diameter is an important consideration with any technique. Inverting patterns result in substantial lumen compromise of the small intestine and are NOT recommended in dogs and cats.

Apposition: Anatomic apposition of individual layers of the bowel wall (i.e., mucosa, submucosa, muscularis, and serosa) result in primary intestinal healing. This technique is superior to inverting or everting techniques because apposition of intestinal margins eliminates lumen compromise. This is the authors preferred technique for suturing all hollow viscus organs in the abdominal cavity. Suture patterns of choice include:

1. Simple interrupted apposing. This technique involves suturing all layers of the intestinal wall and tying the knots on top of the serosa to approximate cut edges. The sutures should be tied tight enough to effect a watertight seal, yet not so tight as to blanch the tissue and cause ischemia of intestinal margins. This technique is simple, fast, reliable, and does not result in lumen compromise.

2. Simple continuous apposing. This technique is similar to the simple interrupted appositional technique however, a continuous suture pattern is used rather than an interrupted pattern. Advantages include faster anastomosis, equal suture tension over the entire anastomosis, airtight-watertight seal, and mucosal eversion is minimized. This is the authors preferred suture pattern for suturing all hollow viscus organs in the abdominal cavity.

Intestinal Anastomosis: Intestinal anastomosis is indicated for resection of nonre-ducible intussusception, necrotic bowel wall secondary to complete intestinal obstruction, intestinal volvulus, stricture secondary to trauma, linear foreign body with multiple perforations, and intestinal neoplasia (e.g., leiomyoma, leiomyosarcoma, adenocarcinoma).

After a complete abdominal exploration, the affected length of bowel is delivered from the peritoneal cavity and isolated with the use of moistened laparotomy pads and crib towels. If possible, the intestinal anastomosis should be performed on a water resistant surface (e.g., plastic drape, crib towel) to prevent 'strike' through contamination.

Once the level of resection has been determined, the appropriate mesenteric vessels are identified and ligated, and the portion of intestine to be resected is isolated by clamping the bowel at a 60° angle away from the mesenteric border. This angle ensures adequate blood supply to the antimesenteric border.

Everted mucosa: Occasionally when the segment of intestine to be removed is amputated mucosa 'everts' from the cut edge of the intestinal wall making it difficult to visualize the cut edge of the serosa. If this occurs it is 'highly' recommended to excise the everted mucosa to enable the surgeon to easily visualize the cut edge of the intestinal serosa. It is vital that the surgeon engage at least 3 – 4 mm of intestinal wall with each suture to guarantee adequate bites in the collagen laden submucosa.

Bowel lumen diameters: In cases where the oral end of the bowel is dilated and the aboral end is normal size, several options exist to create intestinal lumens of equal diameter:

1. Increase the angle of resection on the smaller diameter segment of bowel (i.e., aboral segment). This will increase the orifice size by 5 10 mm depending upon bowel diameter (e.g., dog vs cat).

2. In larger lumen size discrepancies the antimesenteric border of the smaller diameter stoma can be incised longitudinally to enlarge the lumen diameter.

3. An end to side anastomosis can be performed by closing the larger diameter stoma of the intestinal resection with a single layer continuous apposing

suture pattern then anastomosing the smaller diameter segment of bowel to an appropriate size enterotomy made in the antimesenteric border of the larger diameter segment of bowel.

4. The larger diameter segment of bowel can be made smaller in diameter by suturing its cut edge until its lumen is equal in size to the smaller diameter intestine (this technique is often used for subtotal colectomy in cats).

INTESTINAL ANASTOMOSIS TECHNIQUE:

See the Practical Techniques on GI Surgery I DVD for a detailed video description of this technique www.videovet.org.

When suturing an anastomosis, atraumatic handling of bowel wall and perfect anatomic apposition of incised margins is important. It is recommended to begin suturing at the mesenteric border as this allows adequate visualization of mesenteric vessels and helps prevent encircling these vessels when placing the first few sutures. Any of the appositional suture patterns previously described (i.e., simple continuous or interrupted) will result in a high success rate, both in the short-term (i.e., leakage, breakdown) and long term (i.e., stricture, stenosis).

The following tips may prove helpful when performing an intestinal anastomosis (see the anastomosis video clip at www.videovet.org for detailed description of the surgery tips below:

1. First, place a stay suture to hold the mesenteric border of each segment of bowel in apposition. Tie this suture, leave the ends long, and place a hemostat on the suture end without the needle.

2. Place a second stay suture in the antimesenteric borders of each segment to be sutured to bring the ends of the intestinal segments into apposition. Place a hemostat on the ends of this suture.

3. Place gentle traction on the mesenteric and antimesenteric stay sutures to bring the two intestinal segments into apposition. Make certain the lumen diameters of each bowel segment are identical.

4. Using the needled segment of suture from the mesenteric stay suture, begin a simple continuous appositional anastomosis being careful to get a 3 - 4 mm bite in the submucosa and placing each suture no more than 2 - 3 mm apart (2 mm apart in cats). When the anastomosis is complete, tie the suture to the mesenteric stay suture.

5. If a simple interrupted apposing suture pattern is used, be careful to get a 3 - 4 mm bite in the submucosa and place each suture no more than 2 - 3 mm apart.

6. Evaluate the integrity of the anastomosis. The author's preference for evaluating the integrity of the anastomotic closure is to visually examine each suture to be certain that suture placement has met your strict criteria (i.e., sutures are no more than 2 - 3 mm apart and have a 3 - 4 mm bite in the submucosa.

POSTOPERATIVE CARE

Intravenous fluids to maintain hydration and ensure renal function are continued postoperatively, until the patient begins to eat and drink. Intravenous fluids should then be tapered over a 24 to 48 hour period.

Feeding: Early return to enteral feeding is best for the overall health of the intestine. Feeding the postoperative gastrointestinal surgical patient is generrally based on the following criteria:

a) preoperative condition of the patient

b) the condition of the bowel at the time of surgery

c) surgical procedure performed (i.e., enterotomy, anastomosis, pylorectomy)

d) presence or absence of peritonitis

e) postoperative condition of the patient.

The earlier patients can be returned to oral alimenation the better.

COMPLICATIONS

The most common postoperative complication of small intestinal surgery is leakage; leak is either associated with breakdown of the anastomosis or improper surgical technique (i.e., improper suture placement, inappropriate suture material, knot failure, sutures to far apart, inappropriate bite in the collagen laden submucosal layer, suturing nonviable bowel).

A presumptive diagnosis may be accomplished by the following:

1. Body temperature (may be up if acute or down if moribund).
2. Abdominal palpation: periodic, gentle abdominal palpation for pain (gas or fluid?).
3. General attitude (depression anorexia).
4. Incision: examination of the patients incision for drainage (look at cytology if drainage is present)
5. CBC: leukocytosis followed by leukopenia (sepsis), or a degenerative left shift may imply breakdown.
6. Glucose: low glucose generally implies sepsis (this occurs early in sepsis and may be used as a screening test).
7. Abdominal radiographs: generally not helpful, they are difficult to critically assess due to the presence of postoperative air and lavage fluid. It can take 1 - 3 weeks for peritoneal air to diffuse from the abdominal cavity after routine abdominal surgery. Time variation is dependant upon the amount of air remaining in the abdominal cavity postoperatively (i.e., large deep chested animal vs a small obese animal).
8. Abdominal tap (paracentesis): a four quadrant abdominal tap is accomplished by aspirating fluid using a 5cc syringe and 20 gauge needle or placing a plastic IV catheter into the peritoneal cavity and allowing fluid to drip onto a slide. This may be the most sensitive diagnostic test for determining the presence or absence of intestinal leak.
9. Peritoneal lavage (if paracentesis is not productive): infuse 10-20cc/kg of sterile physiologic saline solution into the abdominal cavity, then gently palpate the abdomen and repeat the four quadrant paracentesis. This technique increases the sensitivity of paracentesis to 90%.

Once fluid has been obtained, a smear should be stained and evaluated microscopically. Depending upon the cell types seen, a determination of the presence of leakage can be made.

Below are examples of expected cytology in patients with and without leak.

1. Healthy PMNs with few degenerate PMNs and a moderate number of red blood cells: This cytology may be expected in any postoperative abdominal procedure (e.g., OHE, abdominal exploratory, cystotomy). Your index of suspicion for anastomotic breakdown should be low. However, if clinical signs continue to deteriorate, repeat paracentesis (2 - 3 times daily, if necessary) to determine the "trend" of the abdominal fluid cytology is recommended.
2. Healthy polymorphonuclear leukocytes with bacteria located intra or extracellularly, degenerate PMNs with intracellular bacteria, free bacteria, or food particles imply breakdown. Exploratory laparotomy is indicated.

In a recent morbidity/mortality study of patients undergoing intestinal surgery it was found that animals requiring a second abdominal surgery to treat intestinal disorders were less likely to survive than patients requiring only one laparotomy. Also, the longer it took to determine whether or not intestinal leakage had occured the less likely the patient would survive reoperation. The take home message is: pay attention to detail during the first surgery and if a leak occurs, diagnose it and treat it as soon as possible.

Prognosis The overall prognosis for uncomplicated GI surgery is excellent. The surgeon must pay attention to detail when suturing any hollow viscus organ with liquid contents.

9006

CANINE URETHRAL SURGERY

SURGERY

Howard Seim, DVM, DACVS

KEY POINTS

- Patients with urethral calculi present with stranguria
- Retropulsion of urethral calculi into the urinary bladder simplifies management of urethral calculi
- Aggressive lavage of the urethra should be performed during cystotomy
- Permanent urethrostomy is an acceptable method of managing chronic stone formers

Definition: Cystic and urethral calculi have various compositions (i.e., oxalate, struvite, urate, uric acid, cystine, silicate) and may be present in the urinary bladder or lodged in the urethra, respectively. They may be multiple or single, may cause partial or complete obstruction (i.e., urethral), and may require surgical manipulation for removal.

DIAGNOSIS

Clinical presentation:

Signalment: There is no age predisposition. Dalmations are more likely to present with uric acid calculi and commonly present with calculi lodged in the urethra. Schnauzers are more likely to present with struvite calculi and Daschunds are more likely to present with cystine stones.

History: Patients generally present with a history of urinary obstruction and/or signs of urinary tract infection. Common complaints include difficulty urinating, straining to urinate, hematuria, dripping blood tinged urine from the prepuce, and/or a distended abdomen. Patients that present several days after complete obstruction may have a distended and painful abdomen and a history of anuria. These patients may be so compromised that they present in shock.

Clinical signs: The most frequently reported clinical signs in patients with urethral calculi include unproductive straining to urinate, blood tinged urine dripping from the prepuce, hematuria, and/or polakiuria. Severity of clinical signs may vary with the degree of urethral obstruction and duration of obstruction prior to presentation. Patients with complete obstruction for several days may show signs of post-renal azotemia (i.e., severe depression, recumbant, shocky).

Physical examination: Observation in the examination room may reveal multiple unsuccessful attempts to urinate. Abdominal palpation may reveal a distended urinary bladder.

Patients with severe clinical signs (i. e., presented several days after complete obstruction) may show azotemia, shock, and/or severe depression. Abdominal palpation generally reveals a large, turgid urinary bladder and may result in discomfort to the patient.

Laboratory findings: Results of a complete blood count and serum chemistry profile are generally normal in patients presenting acutely; urinalysis may show evidence of urinary tract infection and and/or crystalluria.

Patients presenting after several days of complete obstruction may have significant changes in their biochemical profile including increased BUN, increased creatine, metabolic acidosis, and severe electrolyte abnormalities. Urine is generally grossly hemorrhagic and urinalysis may show signs of urinary tract infection and crystaluria.

Radiography: Survey radiographs show presence of radiodense calculi in the urethra and/or urinary bladder as well as a distended urinary bladder. Occasionally, radiolucent calculi occur and can only be visualized using retrograde contrast cystourethrography. The

most common location of urethral calculi in male dogs is immediately caudal to the os penis. Careful evaluation of the kidneys and ureters should be done to rule out renal and ureteral calculi.

Ultrasound: Ultrasonographic examination of the bladder, ureters, and kidneys may be helpful in diagnosis of cystic, ureteral, or renal calculi.

Differential diagnosis: Any disorder causing urinary obstruction, including urethral neoplasia, granulomatous urethritis, urethral stricture, and urethral trauma. Definitive diagnosis is based on clinical signs, inability to pass a catheter, and evidence of calculi on survey or contrast radiographs.

Medical management: Immediate care: In animals with complete obstruction of a duration long enough to cause azotemia, temporary urinary diversion is provided by either passing a small urinary catheter (e.g., 5 French) alongside the calculus, performing a prepubic cystostomy (see technique described below), or frequent cystocentesis (i.e, tid to qid). Azotemia is treated with crystalloid IV therapy prior to calculus removal.

Retrograde hydropulsion: See the DVD for a detailed video of this technique.

This technique should result in a 90-95% success rate of retropulsing urethral calculi into the urinary bladder!

TECHNIQUE

1. Select the largest diameter sterile high density polypropylene urinary catheter that will fit past your patients os penis (generally 6, 8, or 10 French diameter)
2. If the selected catheter turns out to be a 6 French diameter then mix 30cc of Sterile KY Jelly with 70cc of sterile physiologic saline solution.
3. If the selected catheter turns out to be an 8 or 10 French diameter then mix 40cc of Sterile KY Jelly with 60cc of sterile physiologic saline solution.
4. Thoroughly mix the sterile saline and KY Jelly in a 35 or 60 cc syringe and attach the syringe to the urinary catheter.
5. Anesthetize the animal, extrude the penis and pass the lubricated urinary catheter in the urethra up to and against the calculus. Place a dry gauze sponge around the extruded tip of the penis and occlude the penis around the catheter by squeezing it with thumb and finger.
6. Using a back and forth action on the catheter, simultaneously inject the saline/lubricant mix under extreme pressure. Be certain the catheter tip hits the calculus like a battering ram to help dislodge it and encourage the saline-lubricant mix to surround the calculus and coat the urethral wall. During injection the calculi and urethra are lubricated by the saline/lubricant mix while the viscosity of the mixture (i.e., KY jelly and saline) encourages the calculus to dislodge and become retropulsed into the urinary bladder.

This technique is successful regardless of how many stones are in the urethra and no matter where the calculi are lodged.

If the above technique fails, place a finger in the rectum, palpate the urethra and occlude its lumen (this dialates the urethra); repeat the above maneuvers and when maximum pressure is exerted on the urethra by the saline/lubricant mix (i.e., the urethral is maximally dialated), suddenly release digital urethral occlusion allowing lodged calculi to flush into the urinary bladder.

Surgical treatment: The objective of surgical treatment is to remove all retropulsed calculi from the urinary bladder and any remaining urethral calculi that were unable to be retropulsed. Bladder calculi are removed via cystotomy, urethral calculi are removed via urethrotomy, and patients that are frequent stone formers may benefit form a permanent urethrostomy to allow continual passage of small urethral calculi.

Preoperative management: Patients that present acutely can be anesthetized immediately and retropulsion attempted (see above described technique). If urinary tract infection is suspected, preoperative treatment with antibiotics may be instituted.

Patients that present after several days of complete obstruction should be treated medically until the azotemia resolves, blood gas abnormalities resolve, and electrolytes return to normal. The patients electrocardiogram should

be monitered if hyperkalemia is present preoperatively. Medical treatment may consist of intravenous fluids, systemic antibiotics, continuous ECG monitoring, and bladder decompression. Bladder decompression may be accomplished via passing a small gauge urinary catheter (e.g., 5 French) past the calculus, multiple cystocentesis (i.e., tid or qid), or placement of a antepubic cystostomy tube (described in detail below).

Surgical anatomy: The male canine penile urethra consists of urethral mucosa (i.e., urothelium) surrounded by corpus cavernosum urethra, which is in turn surrounded by tunica albuginea. Because of the fluid filled corpus cavernosum urethra (blood) and the tough fibrous connective tissue tunica albuginea, the urethra can withstand tremendous pressure (e.g., as with aggressive retropulsion) without the fear of urethral rupture.

Positioning: Patients are positioned in dorsal recumbancy for retropulsion, urethrotomy, urethrostomy, cystostomy tube placement and cystotomy.

Surgical technique: The surgical techniques vary depending upon the procedure chosen, and are described in detail below:

Retropulsion: The technique for retropulsion of urethral calculi is described above in medical management.

Percutaneous cystostomy tube placement: Occasionally, it may be necessary to perform a percutaneous antepubic cystostomy to decompress the urinary bladder whilst treating a severely azotemic patient until they become a better anesthetic and surgical risk.

The patient is sedated and placed in dorsal recumbancy. A 3-4cm incision is centered between the umbilicus and pubis. Subcutaneous tissues are disected to expose the ventral midline (i.e., linea alba). A 2-3cm incision is made in the linea alba and the bladder wall located. A 12–14 French Foley catheter is advanced through a skin incision 2-3 cm lateral to the abdominal incision, tunneled in the subcutaneous tissue and brought into the abdominal cavity at a location just lateral to the midline abdominal incision. A pursestring suture is placed in the bladder wall at the proposed site of Foley catheter placement with 3-0 monofilament absorbable suture. A 1cm incision is made into the bladder lumen and the Foley catheter advanced. The pursestring suture is carefully tightened to create a water-tight seal, but not to tight as to create bladder wall necrosis. The bladder wall is pexied to the abdominal wall at the point of entry of the Foley catheter with 3-0 monofilament absorbable suture in a simple interrupted pattern. The abdominal wall is closed in a routine fashion.

The cystostomy catheter is held in place with a Chinese finger trap friction suture technique using #1 monofilament nonabsorbable suture and attached to a closed collection system to avoid urinary tract infection. The cystostomy tube remains in place until the patient is ready for definitive surgical treatment.

Urethrotomy: See DVD for detailed video of this technique.

The urethral calculus to be removed is located by evaluation of radiographs, palpation of the os penis and its relationship to the calculus, and/or passing a catheter in the urethra until it contacts the stone, removing the catheter and using it as a measure to locate the calculus.

A 2–3 cm skin incision is made directly over the calculus. Subcutaneous tissues are dissected until the retractor penis muscle is exposed. The cream colored retractor penis muscle (smooth muscle) is dissected off the corpus cavernosum penis (the corpus cavernosum penis has a bluish tint from venous blood) and retracted laterally. A sharpe #15 BP scalpel blade is used to incise the urethra directly over the calculus being careful to incise the urethra directly on its midline to help decrease cavernous sinus bleeding. C:\Users\SBeatty\AppData\Local\Microsoft\Windows\INetCache\Content.Outlook\V367Q759\Figure 06.pdfNo attempt is made to control cavernous sinus hemorrhage with cautery or hemostats as this creates excessive urethral trauma and is generally unsuccessful at controling hemorrhage. Rather, hemorrhage is controlled via digital pressure and suction until suturing can commence. The calculus is grasped with forceps and removed from the urethra.

The urethral incision can be left open to heal by second intention; if this method is chosen moderate to severe hemorrhage can be expected for several days postoperatively.

Alternately, the urethral incision can be closed using 5-0 multifilament or monofilament absorbable suture in a simple interrupted or continuous pattern. Subcutaneous tissues are closed with 3-0 monofilament absorbable

suture in a simple continuous pattern and skin with 3-0 or 4-0 nonabsorbable monofilament suture. If this method is preferred by the author over healing by second intention as postoperative hemorrhage is significantly reduced.

Both urethrotomy techniques (i.e., sutureless or sutured) result in perdictable urethral healing without evidence of urethral stenosis or stricture.

Penile Urethrotomy: See DVD for detailed video of this technique.

The urethral calculus to be removed is located by evaluation of radiographs, palpation of the os penis and its relationship to the calculus, and/or passing a catheter in the urethra until it contacts the stone, removing the catheter and using it as a measure to locate the calculus.

If the calculus is lodged within the os penis, the penis is extruded and a penrose drain is secured around its base to act as a tournaquet as well as maintaining the penis in an extruded position. Once the calculus is located a 2 – 3 cm incision is made through the penile mucosa directly over the calculus. Maintaining a midline incision through the corpus cavernosum penis helps decrease postoperative hemorrhage and encourages the urethral incision to be directly over the calculus. Once the calculus is identified a straight or curved mosquito hemostat is used to help wedge it from its lodged position in the os penis. If the calculus fractures care is taken to flush out all remaining fragments. Primary penile closure is recommended to help provide immediate postoperative hemostasis. 5-0 or 6-0 monofilament or mulifilament synthetic absorbable suture with a cutting needle (biosyn, monocryl, dexon, vicryl, polysorb). The wound is closed in three layers: urethral mucosa, cavernous tissue and penile mucosa. When suturing the penile mucosa, care is taken to make certain the suture material and knots are buried so there is no contact of suture with prepucial mucosa. The tourniquet is removed and penis replaced in the prepuce. No attempt is made to maintain a urethral catheter post operatively. Blood may drip from the prepuce as many as seven days after surgery.

Scrotal Urethrostomy: See DVD for detailed video of this technique.

Urethrostomy is generally performed in patients that are recurrent stone formers. It provides a permanent opening caudal to the os penis that is large enough to accommodate passage of most urethral calculi. This technique is often performed in Dalmations for treatment of recurrent uric acid calculi.

Scrotal urethrostomy is the location of choice for urethrostomy in dogs. It is a convienent location for surgical manipulation, this area of the urethra generally bleeds the least, the urethral diameter will accommodate passage of most urethral calculi, and there is less urine scald postoperatively. Other locations for urethrostomy include prescrotal and perineal.

Prior to surgery a urethral catheter (the largest size that will fit past the os penis) is passed, if possible. After a routine castration and scrotal ablation have been performed, the subcutaneous tissues are dissected to expose the retractor penis muscle. The retractor penis muscle is smooth muscle and appears light grey to cream colored. The retractor penis muscle is dissected from its attachment to the corpus cavernosum urethra. The blood filled cavernous tissue gives the urethra a bluish color. The urethral catheter is palpated and used as a firm surface to cut against when incising the urethra. Every attempt is made to incise the urethra exactly on the midline to help decrease hemorrhage. A 3–4 cm incision is made in the urethra. The caudal aspect of the urethral incision is positioned directly over the ishial arch. As this is the new point of urine flow it is most efficent to have urine exit before it makes a sharp turn ventrally. No attempt is made to control cavernous tissue hemorrhage with cautery or hemostatic forceps; only pressure, suction, and suture pressure should be used.

After incision of the urethra, the glistening urethral mucosa is identified, 4-0 or 5-0 nonabsorbable monofilament suture with a swaged on cutting or taper-cut needle is recommended by the author to suture urethral mucosa to skin. The first urethrostomy suture is placed at the midpoint of either side of the urethral incision to include urethral mucosa, tunica albuginea, and skin (suture split thickness of skin). The suture is tied leaving the end without the needle 3-4 cm long to act as a stay suture. The second suture is placed directly across from the first suture and tied as described for the first. The urinary catheter can now be removed. After the first two sutures are placed, the needle end of one suture is used to begin suturing the cranial portion of the

urethrostomy using a simple continuous suture pattern. When the opposite suture is encountered, the stay suture is used to tie off the first continuous suture line. The opposite suture is then used to suture the caudal portion of the urethrostomy in a similar fashion tying the final suture to the remaining stay suture. Fine ophthalmic instruments make tissue handling and suturing easier. Use of a magnifying loupe (about 2x) and head lamp light source enhances visualization of the urethral mucosa and facilitates accurate suturing. It is critical that the surgeon recognize glistening urethral mucosa and suture it to skin. This will decrease (or eliminate) the chance of urethral stricture. It has been shown that a continuous suture pattern incorporating the urethral mucosa and tunica albuginea (i.e., squeezes the cavernous tissue) results in less postoperative hemorrhage.

SUTURE MATERIAL/SPECIAL INSTRUMENTS:

Urinary catheters of various sizes, Foley catheter, head lamp light source, 2X loupes, ophthalmic instruments, 4-0 and 5-0 monofilament absorbable suture material.

POSTOPERATIVE CARE AND ASSESSMENT:

Postoperative care varies depending upon procedure performed:

Percutaneous cystostomy tube: It is important to keep the percutaneous cystostomy tube attached to a closed collection device. The tube can be connected to a sterile collection bag via a sterile intravenous catheter connection set. An elizabethan collar may be necessary in some patients to prevent iatrogenic removal of the cystostomy catheter. Careful management is important to control catheter related urinary tract infection.

Sutureless Urethrotomy: If urethrotomy without suturing is performed, patients must be monitored for blood loss from the urethrostomy site. Blood loss can be severe enough to lower the PCV by 2 – 3%. An Elizabethan collar may be necessary in some patients to prevent self-mutilation. Patients should be kept quiet and away from other animals (especially bitches in heat!). Tranqulization is occasionally necessary to control hyperactive or overly excitable patients. Clients should be warned that drops of blood may be present from the urethrotomy site as long as 2 weeks postoperatively.

Sutured Urethrotomy: If a sutured urethrotomy is performed, patients will exhibit very little blood loss. However, an Elizabethan collar should be considered, especially in patients that may be prone to self-mutilation. Patients should be kept quiet and away from other animals (especially bitches in heat!). Tranqulization is occasionally necessary to control hyperactive or overly excitable patients.

Scrotal Urethrostomy: The most common postoperative complication of scrotal urethrostomy is bleeding from the urethrostomy site. Utilization of a simple continuous suture pattern incorproating the urethral mucosa and tunica albuginea (i.e., squeezing the cavernous tissue and creating a air-tight/water-tight seal) has significantly decreased the incidence of postoperative hemorrhage in the authors opinion. An Elizabethan collar should be considered, especially in patients that may be prone to self-mutilation. Patients should be kept quiet and away from other animals (especially bitches in heat!). Over excitement immediately postoperatively can result in frank hemorrhage or subcutaneous hemorrhage.

Prognosis: The prognosis for surgical management of urethral is dependant upon preoperative management of azotemic patients prior to anesthesia, success of retropulsion of urethral stones into the urinary bladder, care in removing all stones via cystotomy, and care of ensuring urethral mucosa to skin apposition during urethrostomy.

Patients that require sutured or sutureless urethrotomy have a favorable prognosis if all of the remaining calculi are removed from the urinary bladder via cystotomy to ensure that no calculi are left behind.

Patients that have an elective urethrostomy have a favorable prognosis if attention is paid to proper surgical technique (i.e., urethral mucosa is sutured to skin). Occasionally, chronic stone forming patients will form a calculus that is to large to pass through

9007

ANAL SACCULECTOMY: A NOVEL APPROACH

SURGERY

Howard Seim, DVM, DACVS

KEY POINTS

- knowledge of anorectal anatomy and neuroanatomy is important to protect vital structures
- remove all anal sac epithelium during anal sacculectomy
- use a Mila Anal Sac catheter or 6F Foley 3cc bulb catheter to facilitate anal sacculectomy

If you would like a video of this surgical procedure on DVD go to www.videovet.org or contact videovet@me.com. You may click on the 'Seminar Price' for any DVD you would like to purchase.

Introduction: Disorders involving the anus and rectum occur frequently in small animal practice. In order to appropriately diagnose and treat these disorders, knowledge of the regional anatomy, physiology, common clinical signs they produce, and proper physical examination techniques are necessary.

Anatomy: The location and function of the following anatomic structures should be reviewed prior to surgical management of diseases of the anus and rectum: internal and external anal sphincter muscle, anal sac and duct and caudal rectal nerve.

THE ANAL SPHINCTER MUSCLE

(From the introduction of a report on hemorrhoidectomy written by WC Bornemeier and published in Am J of Proc, Feb, 1960.):

"The prime objective of a hemorrhoidectomy is to remove the offending varicosity with as little damage as possible to the patient. Of all the structures in the area, one stands out as the king. You can damage, deform, ruin, remove, abuse, amputate, maim, or mutilate every structure in and around the anus except one. That structure is the sphincter ani. There is not a muscle or structure in the body that has a more keenly developed sense of alertness and ability to accommodate itself to varying situations. It is like the goalie in hockey...always alert."

"They say man has succeeded where the animals fail because of the clever use of his hands yet, when compared to the hands, the sphincter ani is far superior. If you place into your cupped hands a mixture of fluid, solid, and gas and then, through an opening at the bottom, try to let only the gas escape, you will fail. Yet the sphincter ani can do it. The sphincter apparently can differentiate between solid, fluid, and gas. It apparently can tell whether its owner is alone or with someone, whether standing up or sitting down, whether its owner has his pants on or off. No other muscle in the body is such a protector of the dignity of man, yet so ready to come to his relief. A muscle like this is worth protecting."

Clinical Signs: Common clinical signs associated anal sacculitis include: anal licking, matting of anal hair, anal discharge and scooting. Patients that present with any of the above clinical signs should have a thorough physical examination with emphasis on the anorectal region, including a digital rectal examination.

Physical Examination: A complete physical examination should be performed in all patients with clinical signs specific for anorectal disease in order to rule-out systemic disorders that manifest themselves with anorectal abnormalities (i.e., pemphigus).

Specific examination of the anorectal region should include close visual examination of the perineum, circumanal area, and base of the tail, as well as careful digital rectal palpation. In many instances this may be all that is necessary to obtain a definitive diagnosis.

Sphincter muscle atonia or areflexia: This form of incontinence occurs when the peripheral nervous

supply to the external anal sphincter muscle or the muscle itself has been partially or totally severed. The external anal sphincter muscle is made up of striated muscle fibers and is partially responsible for the voluntary control of defecation.

Isolated injury of the caudal rectal nerve to the external anal sphincter is uncommon but may occur from iatrogenic causes. Injury can occur during overzealous anal sacculectomy. The caudal rectal nerve originates from the pudendal nerve which lies on the internal obturator muscle deep in the pelvic canal. Injury during anal sacculectomy is therefore caused during the final 'deep' dissection of the anal sac rather than during the initial dissection. Thus deep dissection is when the surgeon must use exceptional care during dissection of the anal sac.

Anal Sacculitis: Anal sac impaction and abscessation is the most common anorectal disorder diagnosed by the small animal practitioner. Diagnosis is confirmed by clinical signs, visual and digital rectal examination. Relief of impaction by digitally expressing the anal sacs is easily performed during rectal examination. If an anal sac abscess is present, infusion of an antibiotic preparation may be sufficient to eliminate the infection. Systemic antimicrobial treatment may be required in resistant cases. If the anal sac abscess becomes a chronic recurrent problem, surgical excision of both anal sacs is the treatment of choice. Surgery should be delayed however until the immediate infection or abscess has been controlled medically as described above.

Surgical Techniques for Anal Sac Removal: There are a variety of techniques currently used to successfully remove anal sacs. The best approach would be one that allows constant palpation of the extent of the anal sac and also allow retraction of the anal sac during dissection. One such technique is described below:

MILA ANAL SAC CATHETER TECHNIQUE

(the authors' preferred technique) or 6 French Foley Catheter with a 3cc bulb Technique:

A novel approach for safely and completely removing anal sacs relies on the use of a 6 French balloon catheter with a 3cc bulb (MILA or Foley). The balloon catheter is placed into the anal sac through the anal sac orifice and its cuff inflated. Once introduced into the sac, the catheter bulb is inflated with 2-3 cc of air or saline. The bulb distends the anal sac making identification and palpation of the gland simple. The protruding catheter allows the surgeon, or the surgeon's assistant, to place gentle traction on the gland during dissection. A 360-degree skin incision is made around anal sac duct and the protruding catheter. Care is taken to leave at least 2mm of skin from the anal sac duct and the incision. Metzenbaum scissors (curved) are then used to dissect to the plane of tissue between the anal sac wall and external anal sphincter. Identification of the anal sac wall is made by identifying its grayish color in comparison to the deep red color of external anal sphincter muscle fibers that will be carefully dissected off of the anal sac wall. As the dissection progresses constant traction is placed on the Foley catheter to accentuate to sac. When performing the deep dissection of the sac wall care is taken to make certain the dissection does not go deep to the sac wall. This is the location of the caudal rectal nerve fibers. Dissection is continued until the sac is completely dissected free and removed from its surrounding tissue.

Closure consists of suturing together any cut fibers of the external anal sphincter muscle with 3-0 Maxon and the skin closed with 4-0 Biosyn using an intradermal technique. This is the authors preferred technique for anal sacculectomy.

This technique is illustrated on the Anal Sacculectomy video located in the GI Surgery I DVD. Check it out at www.videovet.org.

An alternate technique includes using a pair of Metzenbaum scissors to cut into the anal sac through the duct. The sac is opened to expose the glistening greyish colored interior lining. Hemostats are used to grasp the full thickness of the anal sac wall, being careful to avoid the external anal sphincter muscle fibers. A number 15 BP scalpel blade is used to carefully scrape the gland from the underlying external anal sphincter muscle. The external anal sphincter m., subcutaneous tissue and skin are closed with a synthetic absorbable suture material in a simple interrupted pattern.

An alternate method is to incise laterally over the anal sac, dissect through the subcutaneous tissue, locate the sac and excise it toward the duct.

Regardless of the procedure used, if the entire anal sac is removed and the caudal rectal nerve avoided the prognosis is excellent.

10001

FIP PART I: AN UPDATE ON DIAGNOSTIC TOOLS

FOCUS ON INFECTION PROGRAM

Samantha Evans, DVM, PhD, DACVP

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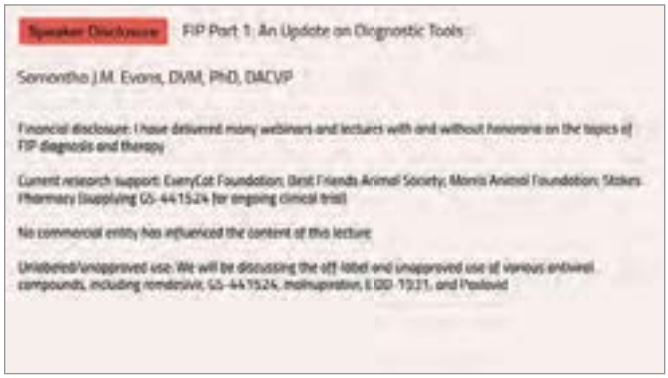

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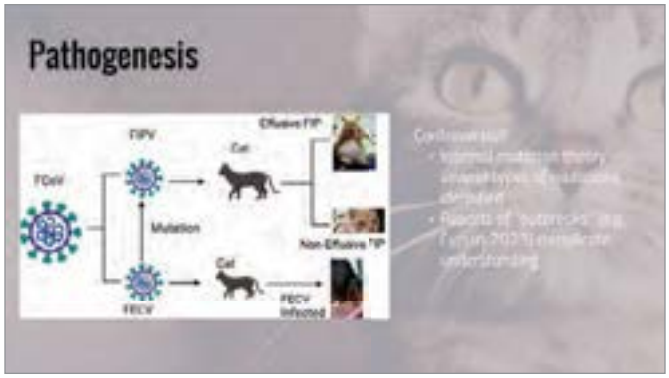

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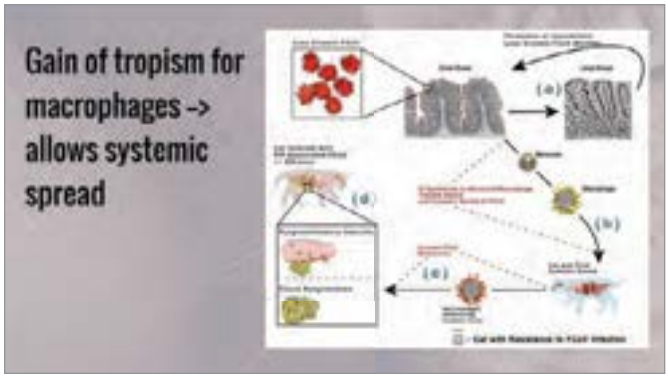

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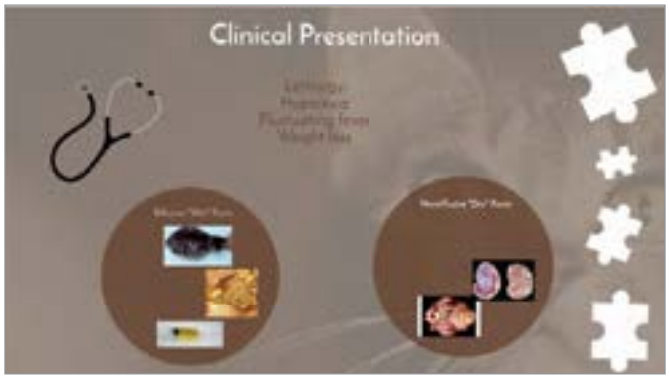

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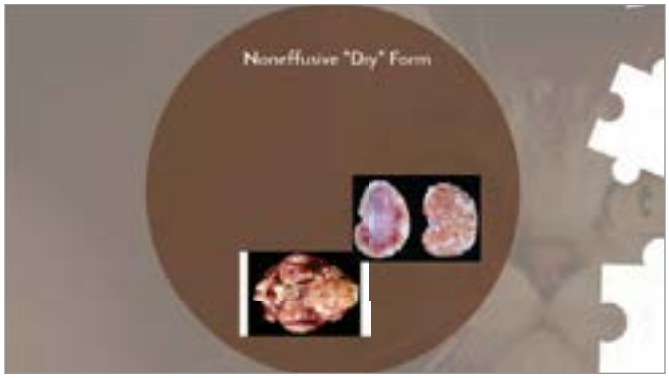

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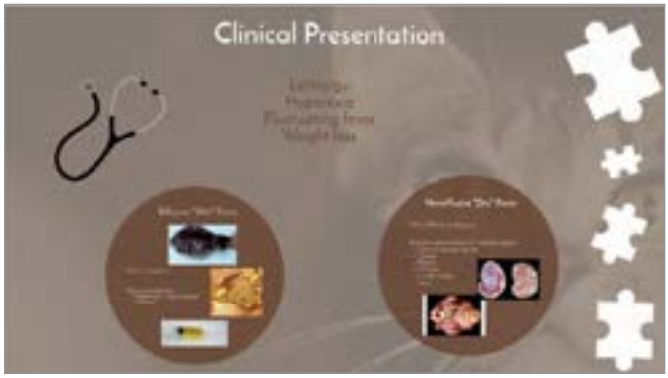

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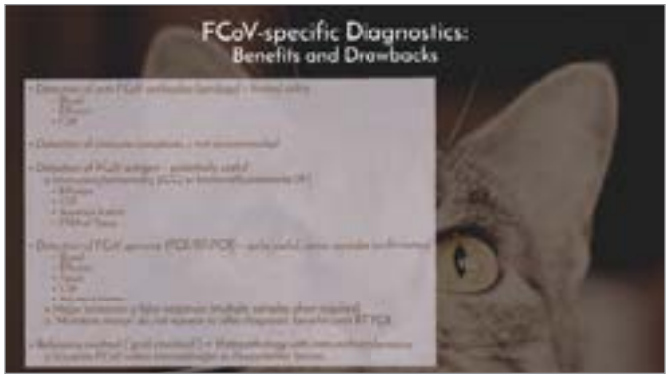

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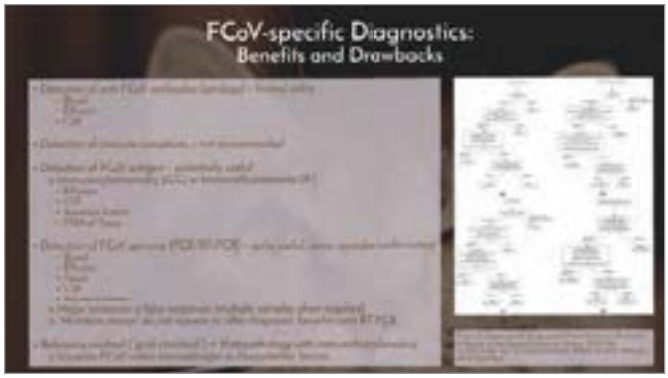

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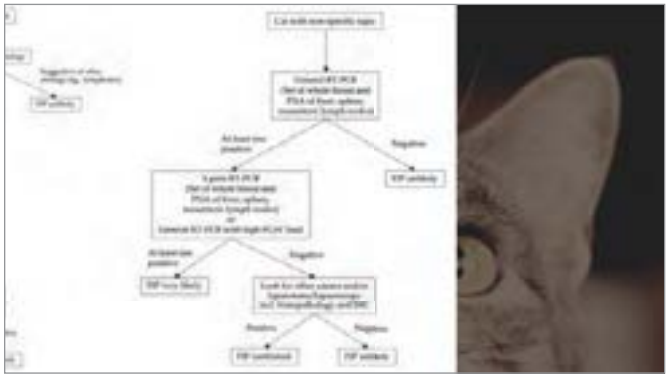

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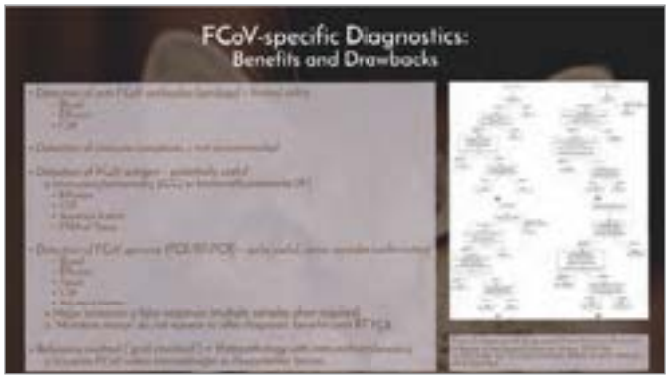

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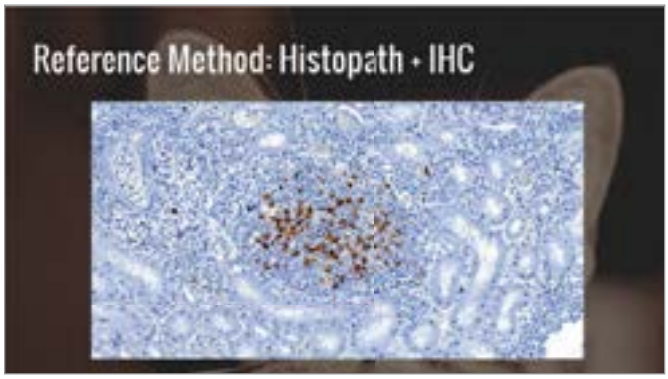

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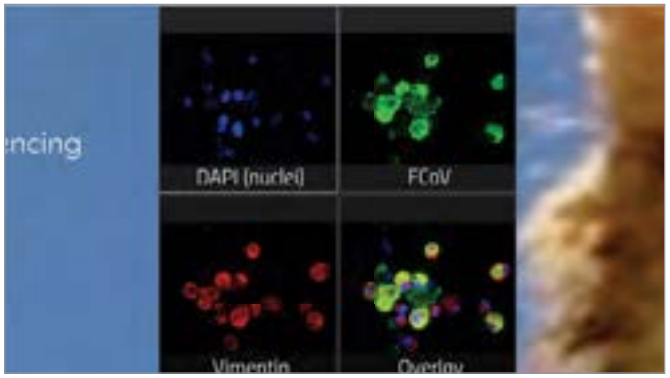

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FIP PART I I: CURRENT USE OF ANTIVIRAL THERAPY

FOCUS ON INFECTION PROGRAM

Samantha Evans, DVM, PhD, DACVP

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Cause of FIP

Coronavirus called feline infectious peritonitis virus (FIPV)

Parent virus = Feline enteric coronavirus (FECV):
- Nearly ubiquitous, mostly commensal
- Colonic inhabitant
- Most cats FECV will never get FIP

Three serotypes:
- Type I - uniquely feline, more common
- Type II - recombinant between feline and closely related canine coronavirus
- FCoV23 - recently described recombinant that emerged in Cyprus in 2023

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Pathogenesis

Controversial! (Unclear)

Internal mutation theory - several types of mutations identified

Gain of tropism for macrophages -> spread systemically

Risk factors:
- Young age
- Genetics (ex: family groups)
- Generalized predisposition in purebred cats is unclear
- Stress/environment
- Crowding/multi-cat household
- Male sex

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A Brief Timeline of FIP Therapy

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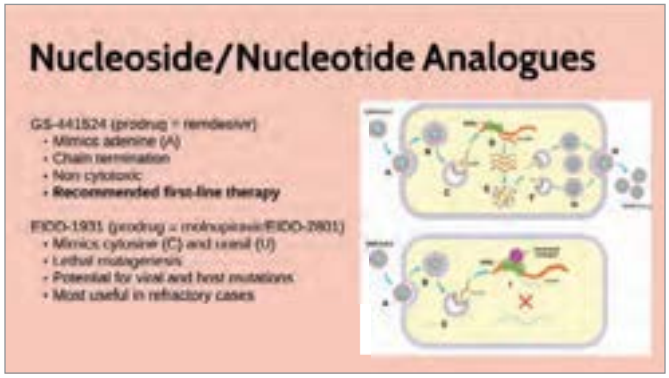

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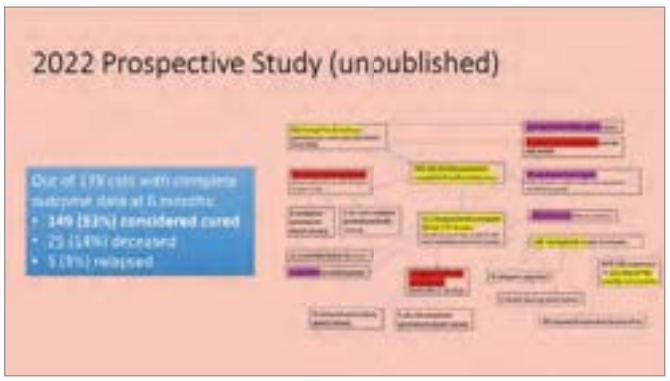

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Labeling on unlicensed oral products

- Often assume ~50% bioavailability and labeled with the amount of the drug which is expected to be absorbed
- Often labeled on a per-kg of cat basis, with an unknown target dosage

Oral dosages CAN NOT be directly translated from black market products to compounded products!

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Efficacy of compounded GS-441524

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OK, but is it really legal in the USA?

- Compounding of GS-441524 is not legal, but expressly "allowable" by the FDA under GFI #256's enforcement discretion
- GS-441524 is under review as a substance approved for office stock, and can be ordered and kept as office stock in the meantime
- GS-441524 remains under patent until 2029, but there have been no efforts to stop compounding pharmacies from producing it in other countries

It is legal for a DVM in the USA to prescribe GS-441524 to a cat with suspected FIP through a licensed compounding pharmacy

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Protease (3CLpro) Inhibitors

GC376
- Developed at KSU
- Currently in FDA-approval pipeline (Anivive)

Nirmatrelvir (contained within Paxlovid)
- Human product; can be used off-label
- Comes with ritonavir (cytochrome P450 inhibitor) to boost PK

Probably not as effective for monotherapy
- Can be useful adjunctive drugs in refractory cases or to decrease treatment duration

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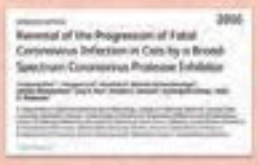
Efficacy of GC376 (protease inhibitor)



Survival in 7 out of 20 client-owned cats with naturally acquired FIP

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Efficacy of Nirmatrelvir (protease inhibitor)

Has been assessed in vitro and as preliminary data (not yet published) in vivo

1 box contains:
- 20 x 150mg Nirmatrelvir tablets (protease inhibitor)
- 10 x 100mg Ritonavir tablets (PK booster)

Preliminary dosage: ¼-½ nirmatrelvir & 1/8-¼ Ritonavir BID PO (20 days treatment per box)

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FIP Antiviral Therapy

Oral therapy with nucleoside/nucleotide analogs is the **standard of care** for FIP

Drugs and Mechanisms

Dosage Regimens

Expected Response

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Oral GS-441524 Administration

- 50mg Quad-scored, tuna flavored tablets
- Or tuna flavored oral suspension 50mg/mL
- Give fasted (withhold food for 1-2 hours), though Ok to give with a tablespoon of wet food or a creamy treat to wash it down. Then a full meal 30mins later.
- Always round dose up to the nearest ¼ tablet as cats grow into the dose.

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FIP Phenotype	Oral GS-441524*
Effusive or non-effusive form, with no CNS/ocular involvement	7.5 mg/kg q12† (or 15 mg/kg q24 if difficult to pill)
Ocular involvement	7.5-10 mg/kg q12† (or 15-20 mg/kg q24 if difficult to pill)
CNS involvement	10 mg/kg q12† (or 20 mg/kg q24 if difficult to pill)

*Give fasted, follow with water bolus or 1 tablespoon of wet food, followed by a full meal 30 minutes later.
† Divided dose is now preferred when using oral GS-441524, however, the total dose may be administered q24 if the cat will not tolerate twice daily pilling.

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Treatment Duration

Current recommendation is **12 weeks (84 days)**

Recent evidence suggests that 6 weeks may be sufficient for some cases

As little as 4 weeks has been documented with combination GS-441524 and GC376

36

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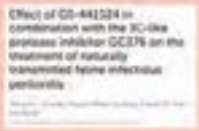

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Minimum Recommended Monitoring

- Body weight measurement **every 2 weeks**
- Monthly PE, CBC, and biochemistry panel
- Focus on clinical response in the first 2-4 weeks of treatment
- If a dosage increase is required, recheck 1-2 weeks after the change to ensure favorable response
- If treatment extension is required, recheck every 2 weeks until remission achieved.

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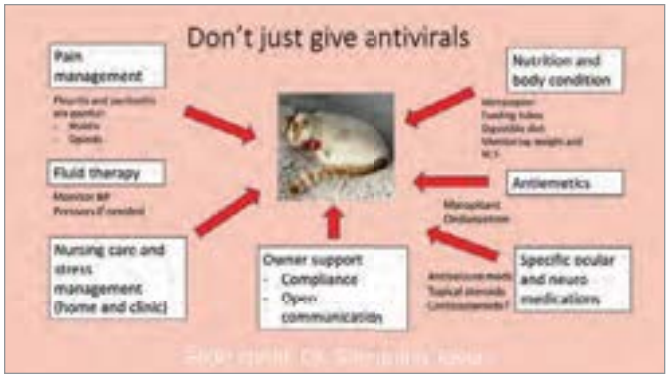

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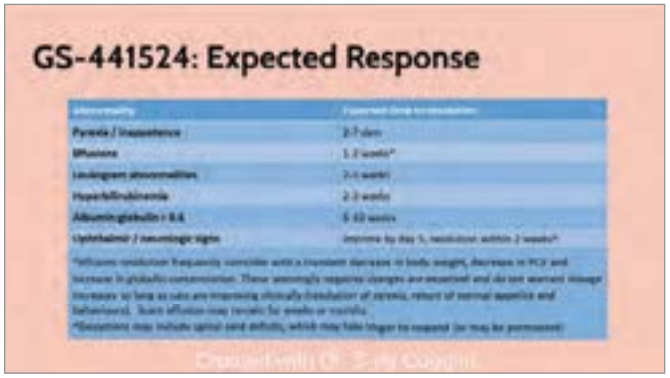

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GS-441524: Adverse Events

Rare!

Mild GI upset (diarrhea)

Most common reported side effects were from injectable (black market) formulations
- Pain and injection-site reactions

Some studies report ALT elevations, but not severe enough to require therapy

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Molnupiravir/EIDD-1931: Adverse Events

Reported more commonly

Appear to be dose dependent
- Folded ears
- Broken whiskers and
- Diarrhea
- Hives, itching, redness, and flaky skin
- Nausea
- Vomiting
- Severe Leukopenia

Reports in human medicine of molnupiravir-associated viral mutations leading to transmission clusters

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Molnupiravir-driven mutations

Analysis of SARS-CoV-2 genomes suggest molnupiravir-associated mutational signatures now exist globally following the introduction of molupiravir for COVID-19 treatment

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Efficacy of molnupiravir

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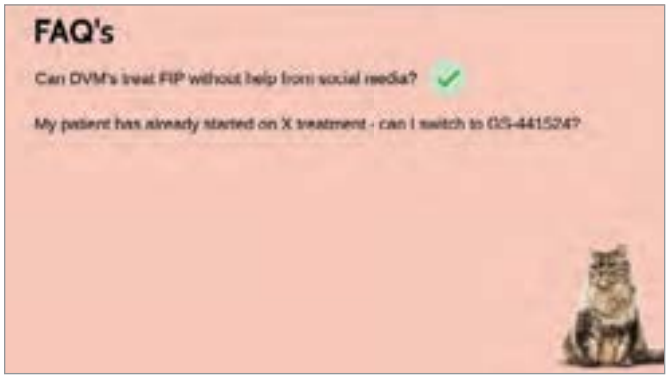

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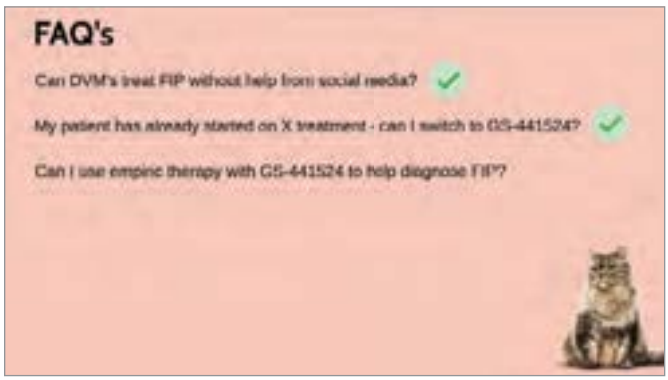

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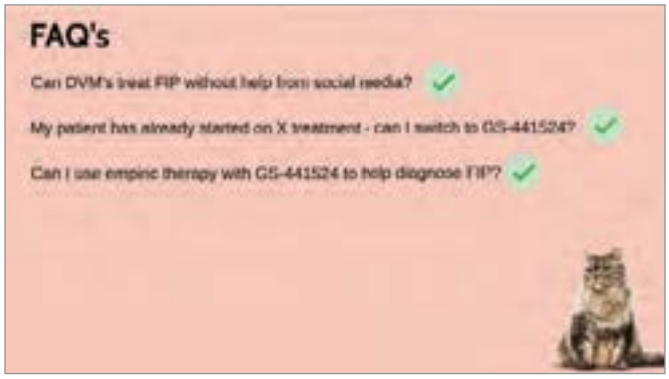

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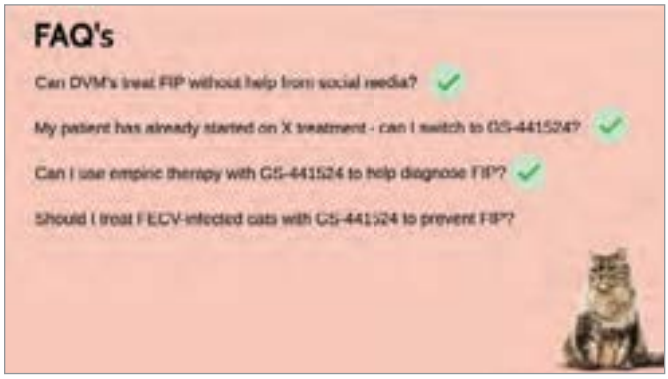

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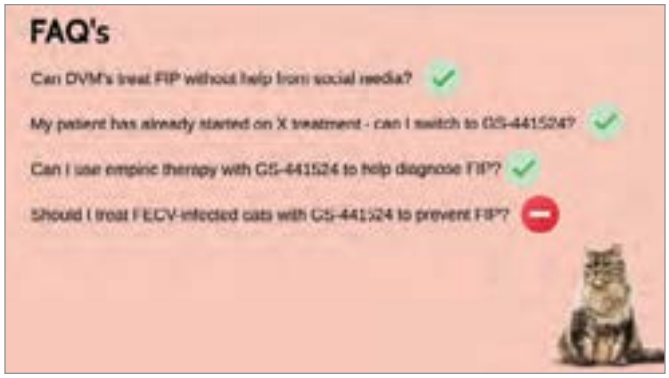

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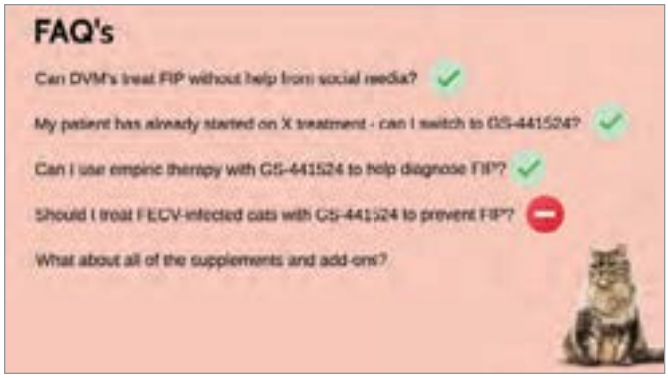

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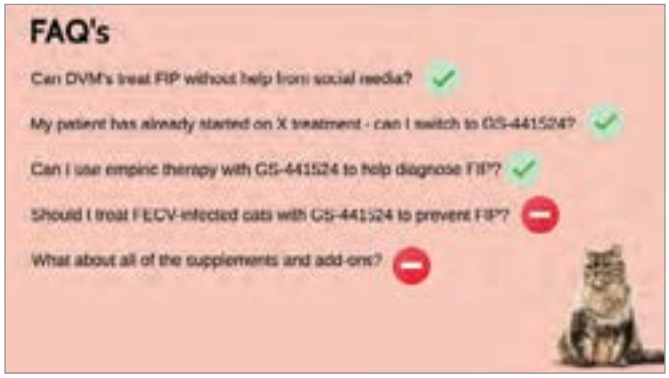

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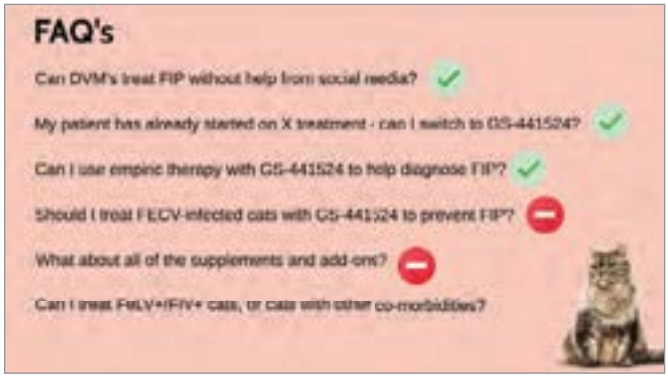

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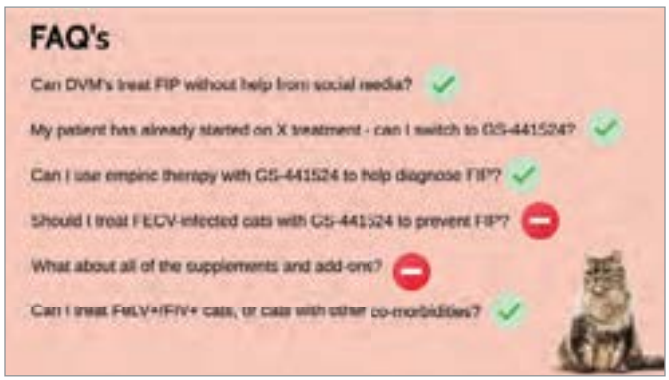

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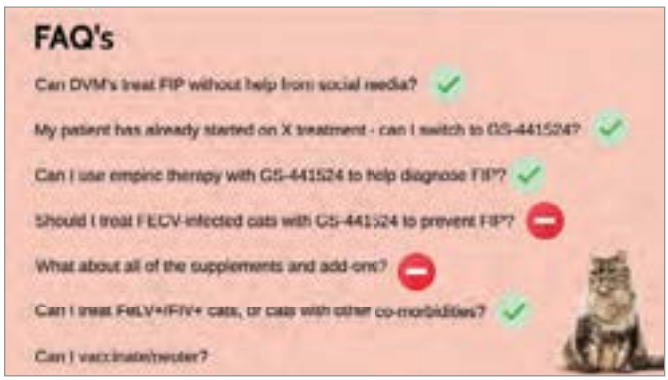

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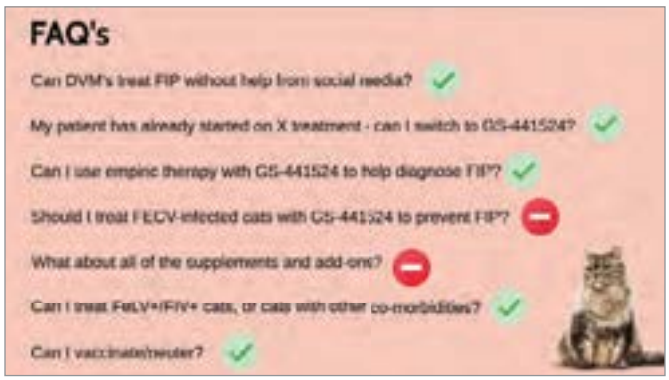

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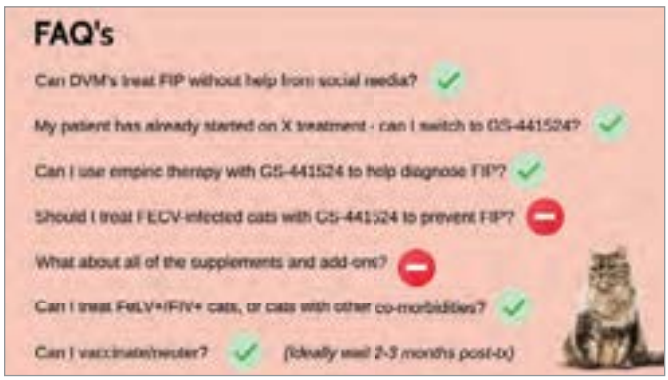

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10003

HOW TO DIAGNOSE FELINE RETROVIRAL DISEASE IN 2025

FOCUS ON INFECTION PROGRAM

Samantha Evans, DVM, PhD, DACVP

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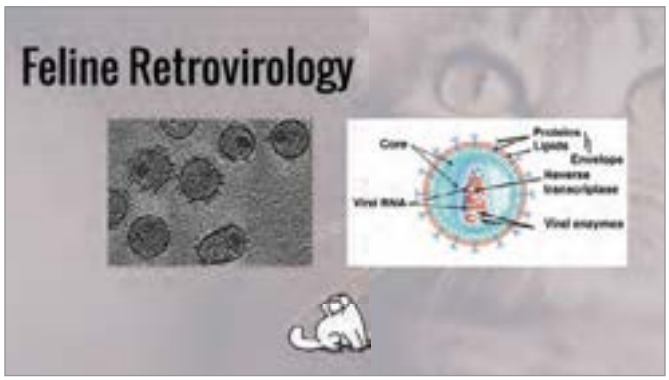

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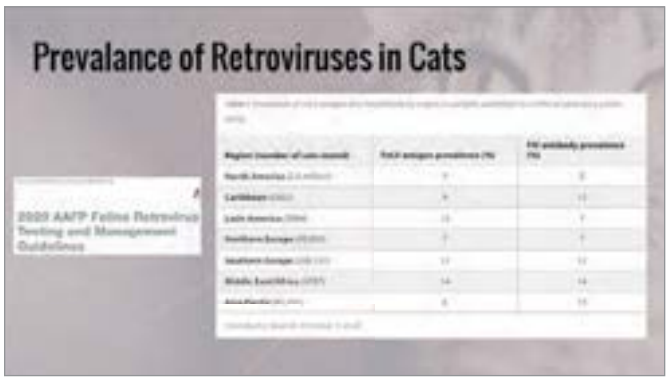

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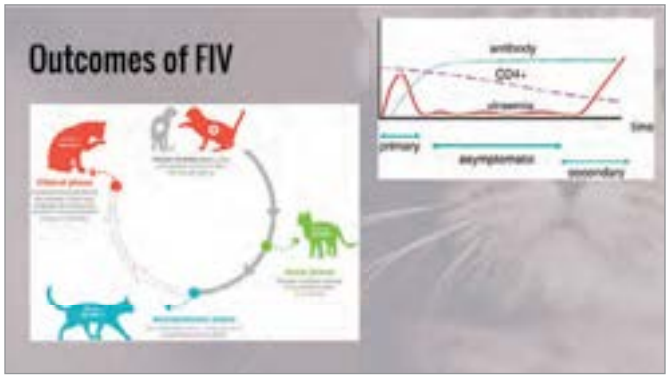

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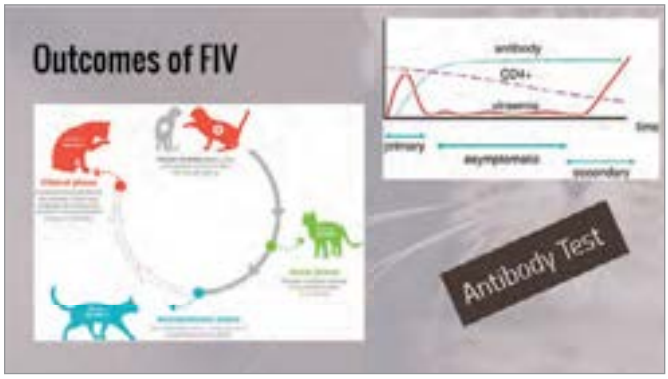

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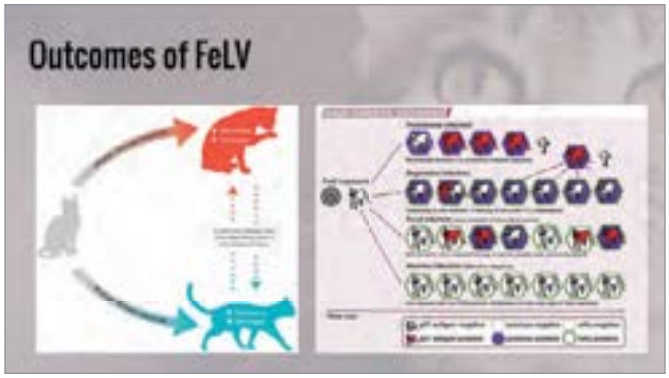

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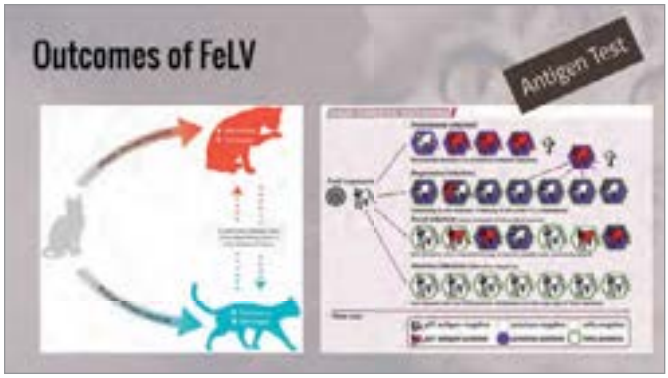

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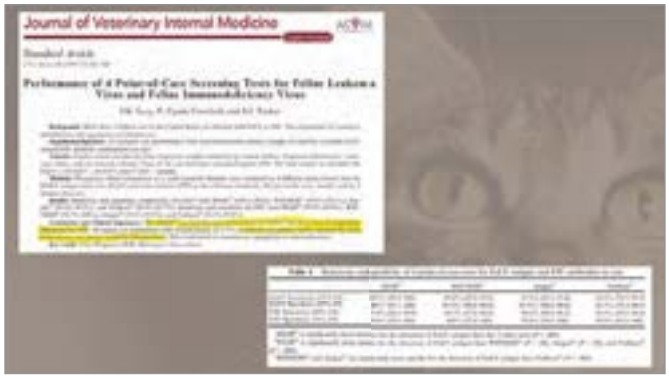

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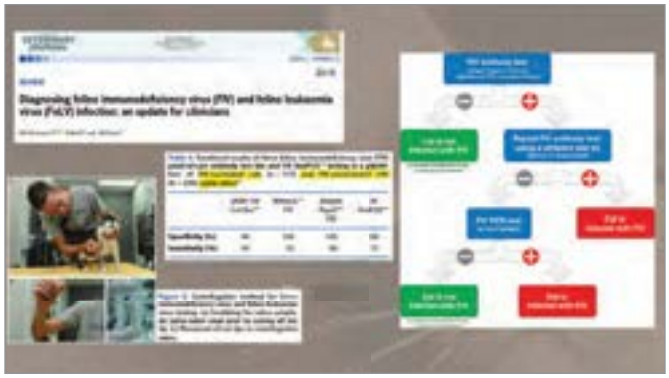

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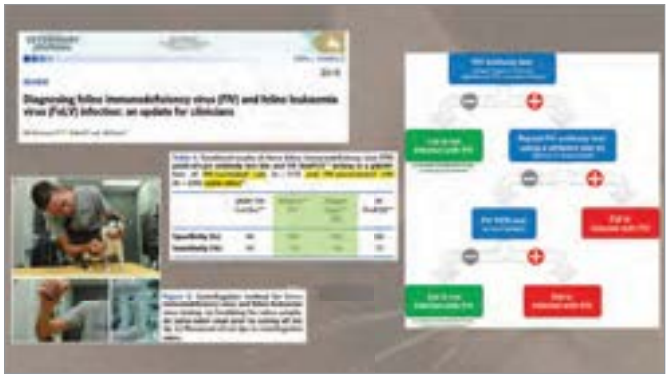

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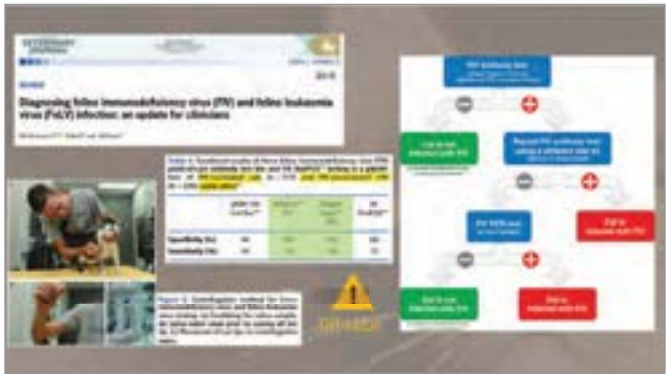

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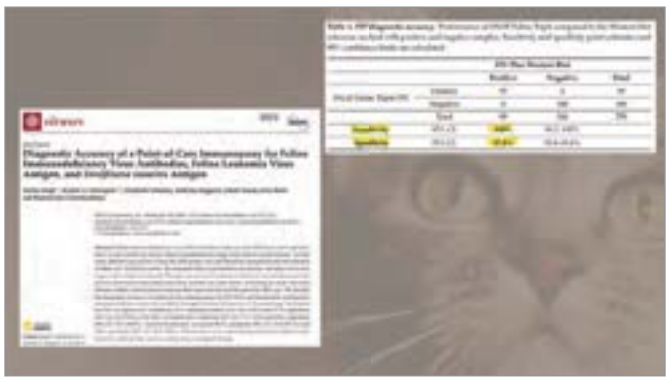

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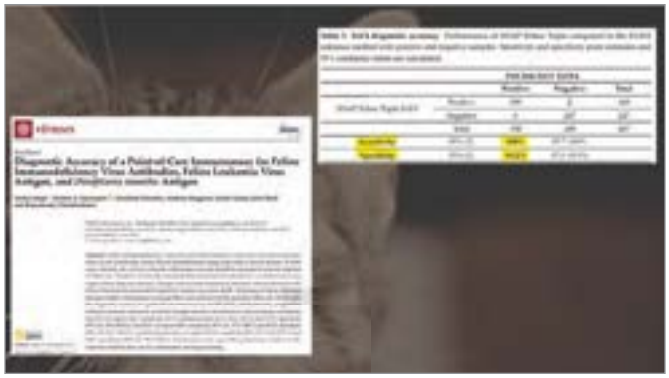

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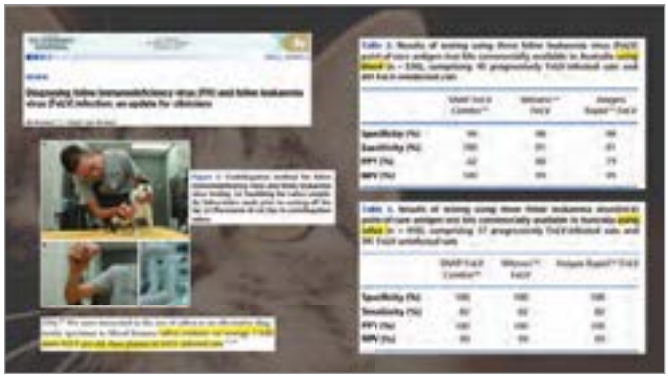

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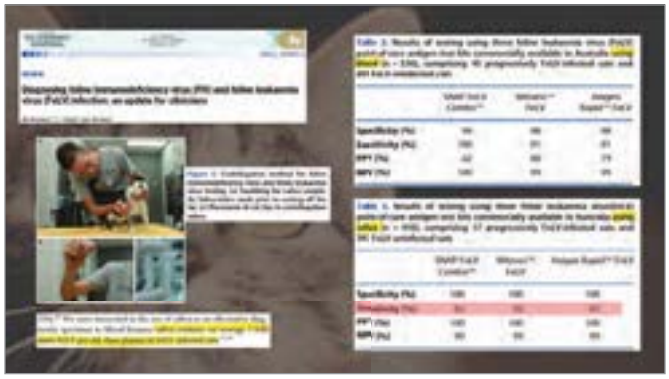

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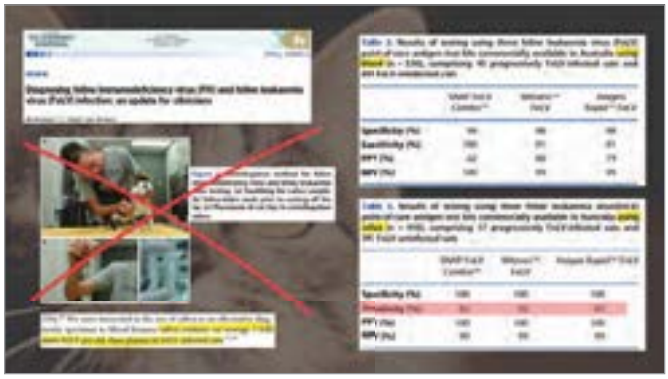

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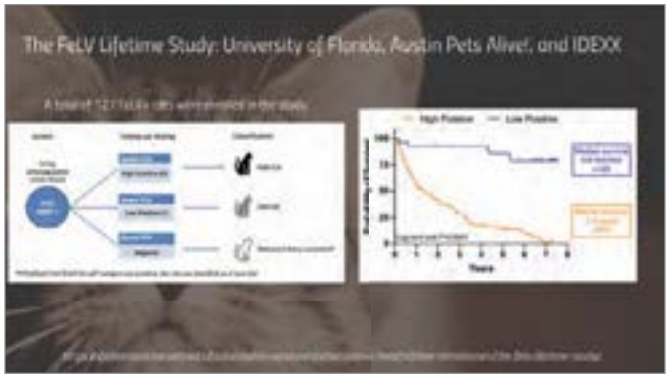

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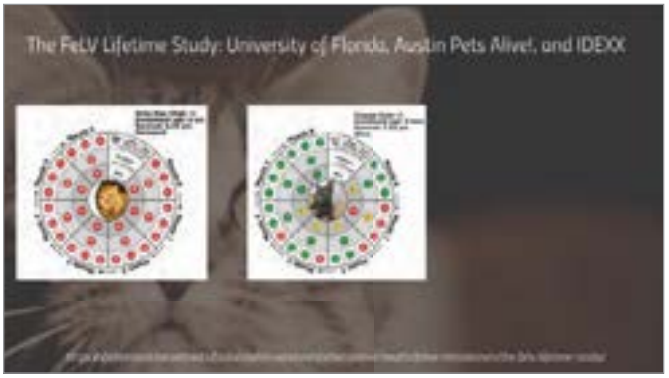

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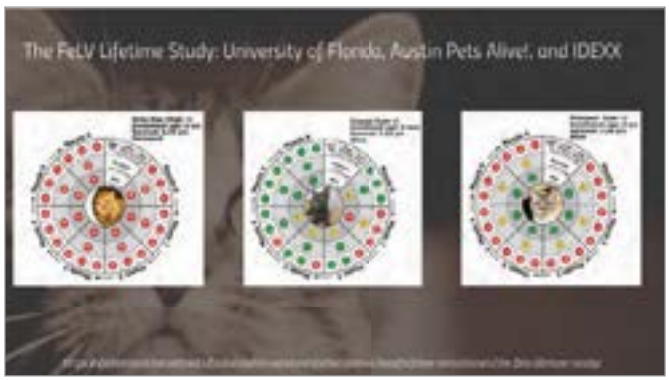

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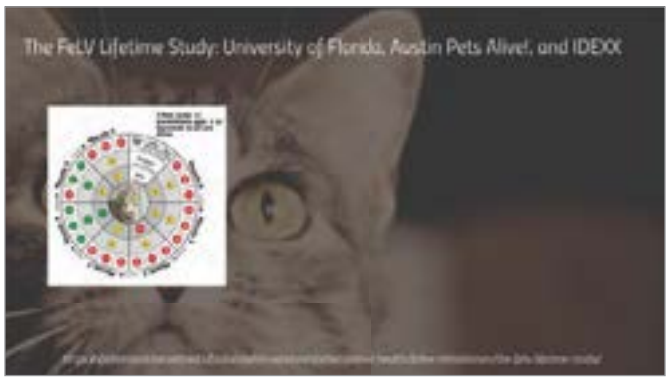

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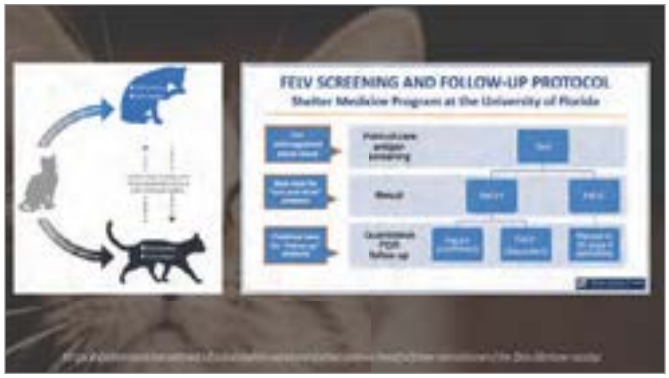

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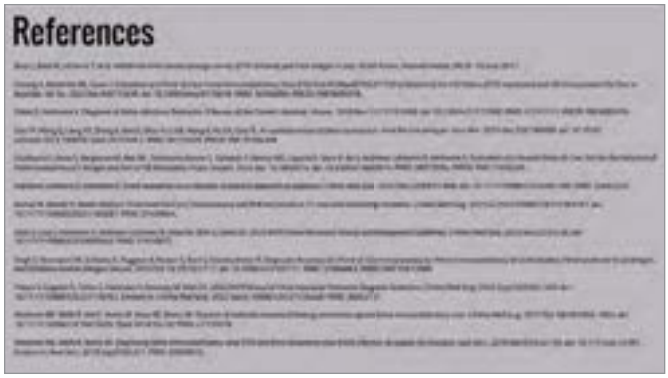

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10004

IDENTIFYING INFECTIOUS AGENTS ON ASPIRATE CYTOLOGY

FOCUS ON INFECTION PROGRAM

Samantha Evans, DVM, PhD, DACVP

INDICATIONS

Aspirate cytology is a very common and extremely useful screening tests for a wide variety of lesions, sample types, and pathologic processes in veterinary patients.

Samples may be taken from almost any organ system in the body, and cytologic evaluation can be a very sensitive and specific test for many types of neoplasia and inflammatory disease. Regarding infectious disease diagnostics, cytology can be both a cost-effective and minimally invasive screening tool for detection of pathogens.

Cytology has the advantage over many other types of infectious disease testing that it is not organism-specific and a variety of organisms may be detected microscopically. In- house cytology is also considerably less expensive and typically much faster than microbial culture, PCR, or immunoassays. On the other hand, microscopic evaluation often lacks sensitivity compared with other techniques, and obviously cannot detect most viral pathogens (apart from the few viruses which cause visible inclusion bodies). Microscopic evaluation of infectious agents may also lack specificity that can be afforded by other techniques and cannot distinguish between morphologically identical organisms such as the wide array of pathogens that appear as bacterial rods.

PROCEDURE

Proper technique in cytologic sampling and submission has huge implications for the clinical utility of cytology but is largely the beyond the scope of this lecture. Multiple slides should be collected from each site of interest to maximize the ability of a cytopathologist to identify infectious agents. A well-made sample with adequate cellularity, minimal blood contamination, minimal artifacts, and well-preserved cellular morphology will increase the likelihood of accurate infectious organism identification. Finally, the sample should be well-stained by following the manufacturer's recommendations, including refreshing the stain at regular intervals. Contaminated stain can be a source of interpretation errors, particularly with bacterial and some fungal agents. Standard in-clinic aqueous based Romanowski ("dip-quick") stains are adequate for most purposes related to infectious disease, and even slightly preferred for some pathogens (ex: canine distemper inclusions). Microscopic evaluation should begin with a low-power review for cellularity and general composition, followed by high power evaluation of cellular morphology. In addition to looking for the infectious agents themselves, the major "clues" of an infectious process are the associated inflammation (including neutrophil degeneration and macrophage activation, if present) and sometimes the lack of a neoplastic population that could be causing a similar lesion.

Extra time and scrutiny on individual cell types may be necessary for rare pathogens (ex: scrutinizing degenerate neutrophils for the presence of rare intracellular bacteria) in cases of strong clinical suspicion. Evaluation of cytologic smears on the 100x objective (1000x total magnification) under oil immersion is essential to increase sensitivity of detecting infectious agents.

DETECTION OF BACTERIA

Example images of some of the most commonly observed bacterial morphologies within cytologic samples of veterinary patients will be described in the presentation.

These include:

- Small and large rod-shaped bacteria
- Cocci bacteria
- Filamentous rods
- Negative-staining rods (Mycobacteria species)
- Spore-forming bacteria
- Spirochetes
- Rickettsial morulae
- Simonsiella-like rods

DETECTION OF FUNGI

Example images of some of the most commonly observed fungal morphologies within cytologic samples of veterinary patients will be described in the presentation.

These include:

- **Yeasts**
  - Histoplasma species
  - Blastomyces species
  - Coccidioides species
  - Cryptococcus species
  - Malasezzia species
  - Candida species
  - Sporothrix schenckii
- **Hyphae**
  - Aspergillus
  - Penicillium
  - Oomycoses (Pythiosis and Lagenidiosis)
  - Phaeohyphomycoses and hyalohyphomycoses
- **Other**
  - Dermatophytes
  - Pneumocystis
  - Rhinosporidium

DETECTION OF PROTOZOA AND ALGAE

Example images of some of the most commonly observed protozoal and algal morphologies within aspirate cytology samples of veterinary patients will be described in the presentation. Myriad organisms can be observed via fecal cytology; these are beyond the scope of this lecture.

The discussed include:

- **Protozoa**
  - Leishmania species
  - Toxoplasma and Neospora species
  - Cytauxzoon
- **Algae**
  - Prototheca species

DETECTION OF VIRUSES

Viral inclusions are only rarely detected by cytology. Example images of some of viral inclusions observed in cytologic samples from veterinary patients will be described in the presentation.

These include:

- Distemper inclusions
- Rabbit myxomatosis
- (Papilloma virus – characteristic cytomorphology)

INFECTIOUS AGENT MIMICS

Finally, we will discuss and provide examples of common artifacts that can mimic infectious agents on cytology. These are important traps to avoid falling into when evaluating a sample for infectious agents.

These include:

- Fibers and debris
- Stain precipitate
- Water artifact
- Superimposed cells

FOLLOW-UP TESTING

At the end of the session, we will discuss appropriate follow-up testing for infectious agents when they are observed or suspected to be present on a cytology review. Often, but not always, confirmatory testing is recommended to verify or further classify morphologic findings (if clinically indicated). Observed agents also need to be distinguished between primary, secondary/opportunistic, and normal flora.

Nevertheless, cytology review can often narrow the differential list and guide appropriate follow-up testing.

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10005

IDENTIFYING INFECTIOUS AGENTS ON A BLOOD SMEAR

FOCUS ON INFECTION PROGRAM

Samantha Evans, DVM, PhD, DACVP

INDICATIONS

Ideally blood smear review would be performed on every patient for which bloodwork is drawn, but it is most important in cases with any abnormal findings on complete blood count (CBC) or suspected analytical errors by automated hematology instruments.

Common examples include evaluation for platelet clumping, RBC morphology changes, left-shifting or toxic change, and suspicion of neoplasia. Regarding infectious disease diagnostics, blood smear review can be a highly useful, cost-effective, and minimally invasive screening tool for detection of blood-borne pathogens. Blood smear review has the advantage over other types of infectious disease testing on blood that it is not organism-specific and a variety of organisms may be detected microscopically. In- house blood film review is also considerably less expensive than PCR or even most basic point of care immunoassays for hematologic pathogens, and is typically much faster than PCR testing. On the other hand, microscopic evaluation often lacks sensitivity compared with immunoassays or PCR, and obviously cannot detect most viral pathogens (apart from the few viruses which cause visible inclusion bodies).

Microscopic evaluation of infectious agents may also lack specificity that can be afforded by immunoassays or PCR and cannot distinguish between morphologically identical organisms such as rickettsial morulae (ie, Anaplasmosis versus Ehrlichiosis).

PROCEDURE

EDTA-anticoagulated whole blood is preferred for microscopic evaluation as it preserves cellular morphology better than other anticoagulants. A well-made blood smear with adequate feathered edge and body ("counting area") is also important to maximize the utility of blood smear review. Finally, the sample should be well-stained by following the manufacturer's recommendations, including refreshing the stain at regular intervals. Contaminated stain can be a source of interpretation errors, particularly with bacterial and some fungal agents. Standard in-clinic aqueous based Romanowski ("dip-quick") stains are adequate for most purposes, and even slightly preferred for some pathogens (ex: canine distemper inclusions). Microscopic evaluation should begin with a low-power review of the edges of the smear, including the back (near the drop), sides, and feathered edge, which is where platelet clumps and larger hemoparasites such as microfilaria and large trypanosomes are frequently observed.

Then red blood cell (RBC) and white blood cell (WBC) estimates can be performed at moderate power, followed by high power examination of RBC, WBC, platelets, and extracellular hemoparasites. In addition to looking for the infectious agents themselves, "clues" of an infectious process include anemia (particularly regenerative anemia for RBC parasites) and inflammatory leukogram changes (leukocytosis with neutrophilia, monocytosis, eosinophilia, left shift, and/or toxic change). Extra time and scrutiny on individual cell types may be necessary for rare or more subtle pathogens (ex: Anaplasma platys) in cases of strong clinical suspicion.

INFECTIOUS AGENTS OF ERYTHROCYTES

Example images of some of the most commonly observed infectious agents of erythrocytes will be described in the presentation.

These include:

- **Protozoal**
  - Babesia species
  - Theileria species
  - Cytauxzoon species
  - Plasmodium species
  - Hemoproteus species
- **Viral**
  - Distempter inclusions
  - Reptarenavirus (boid inclusion body disease)
- **Bacterial**
  - Hemotropic mycoplasmas
  - Anaplasma species
  - Bartonella species

INFECTIOUS AGENTS OF LEUKOCYTES

Example images of some of the most commonly observed infectious agents of leukocytes will be described in the presentation.

These include:

- **Protozoal**
  - Hepatozoon species
  - Leucocytozoon species
- **Viral**
  - Distempter inclusions
- **Bacterial**
  - Anaplasma species
  - Ehrlichia species
- **Fungi**
  - Histoplasma species

INFECTIOUS AGENTS OF PLATELETS

The only common infectious agent of platelets that can be identified on a blood smear is that of Anaplasma platys. Example images will be described in the presentation.

EXTRACELLULAR INFECTIOUS AGENTS

Example images of some of the most commonly observed extracellular infectious agents in blood will be described in the presentation.

These include:

- **Nematodes**
  - Dirofilaria species
  - Dipetalonema species
  - Setaria species
- **Protozoal**
  - Trypanosoma species
- **Bacterial**
  - Borrelia species

INFECTIOUS AGENT MIMICS

Finally, we will discuss and provide examples of common artifacts that can mimic infectious agents on a blood smear. These are important traps to avoid falling into when evaluating a blood smear for infectious agents.

These include:

- Fibers and debris
- Stain precipitate
- Water artifact
- Superimposed cells

FOLLOW-UP TESTING

At the end of the session, we will discuss appropriate follow-up testing for infectious agents when they are observed or suspected to be present on a blood smear review. Often, but not always, confirmatory testing is recommended to verify or further classify morphologic findings (if clinically indicated). Nevertheless, blood smear review can often narrow the differential list and guide appropriate follow-up testing.

REFERENCES

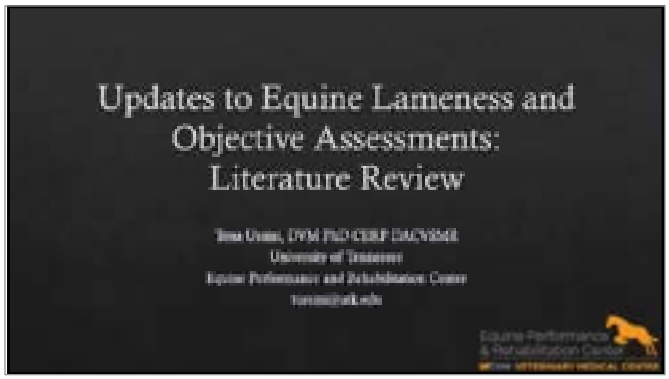
1. *Schalm's Veterinary Hematology. Marjory B. Brooks, Kendal E. Harr, Davis M. Seelig, K. Jane Wardrop, Douglas J. Weiss. First published:4 March 2022. Print ISBN:9781119500506. DOI:10.1002/9781119500537.*
2. *Veterinary Hematology: A Diagnostic Guide and Color Atlas. John W. Harvey. Saunders, Elsevier. ISBN 978-1-4377-0173-9. DOI https://doi.org/10.1016/C2009-0-39565-3.*
3. *eClinPath.com, Cornell University, https://eclinpath.com/hematology/infectious- agents/, November 2024.*

11001

UPDATE TO EQUINE LAMENESS & OBJECTIVE ASSESSMENTS – LITERATURE REVIEW

SPORTS MEDICINE AND REHABILITATION

Tena Ursini, DVM, PhD, DACVSMR, CERP



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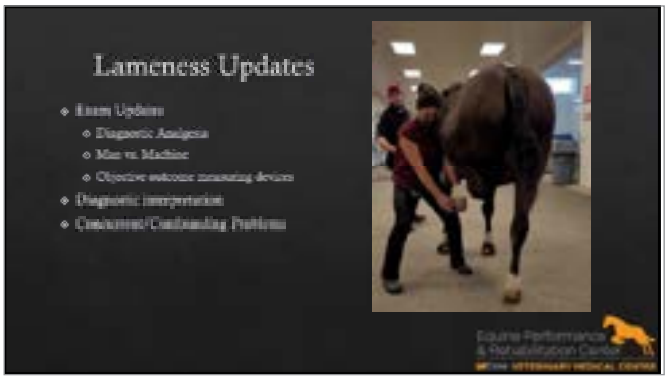

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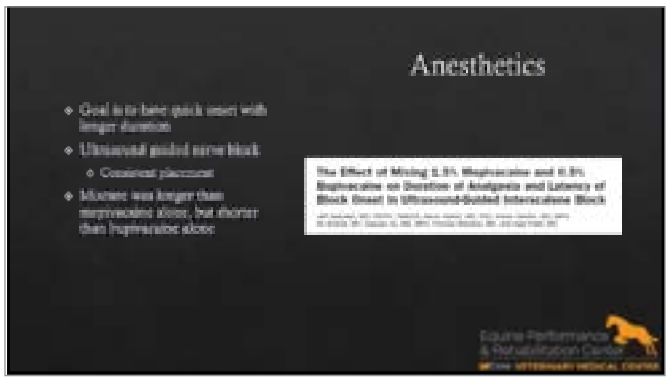

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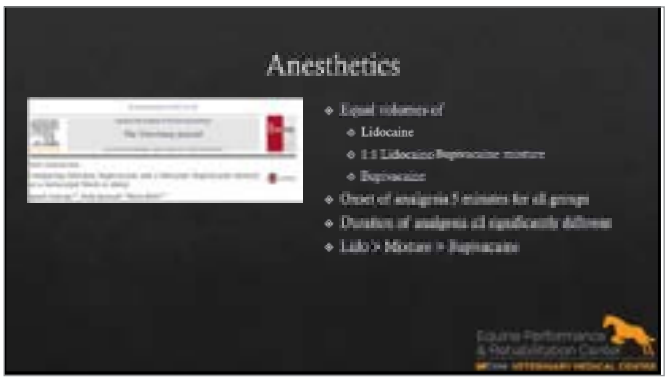

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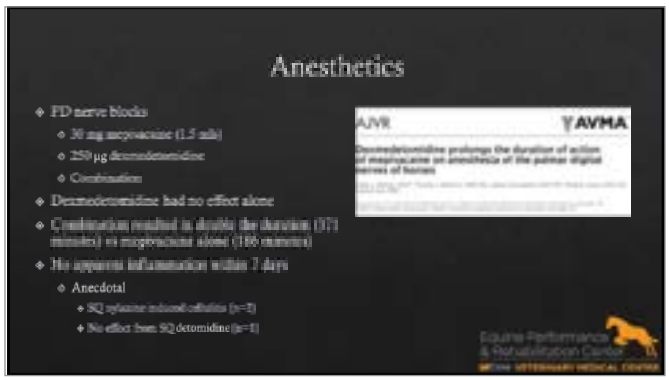

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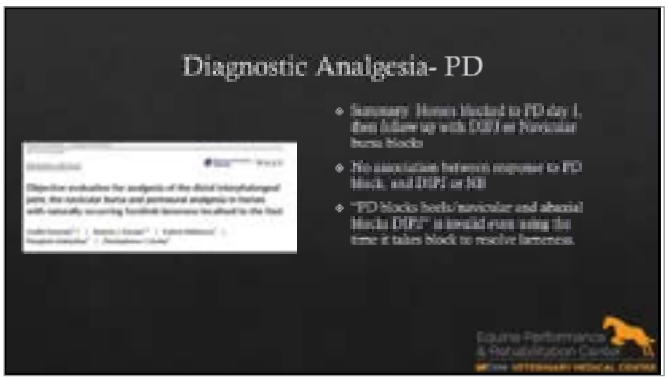

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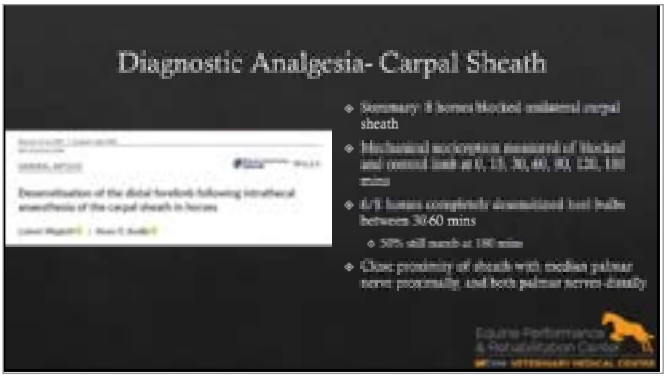

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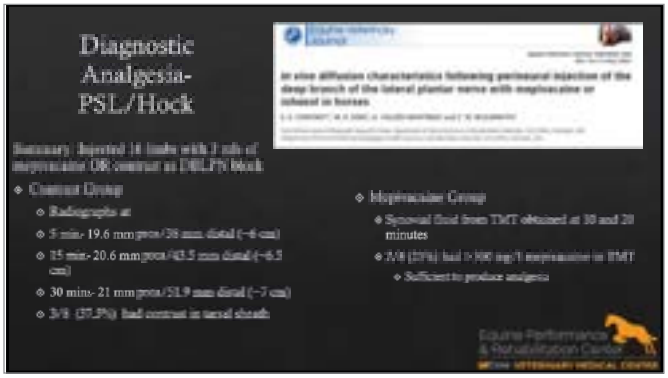

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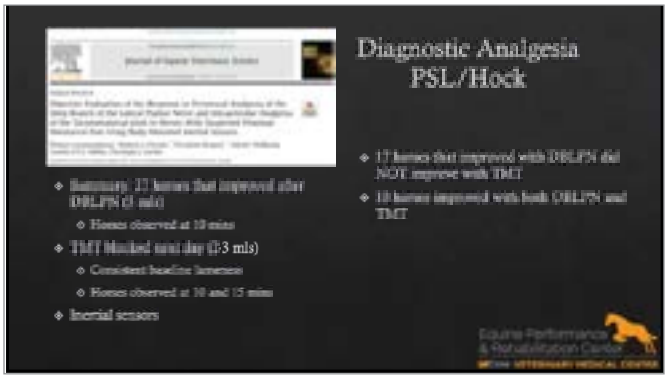

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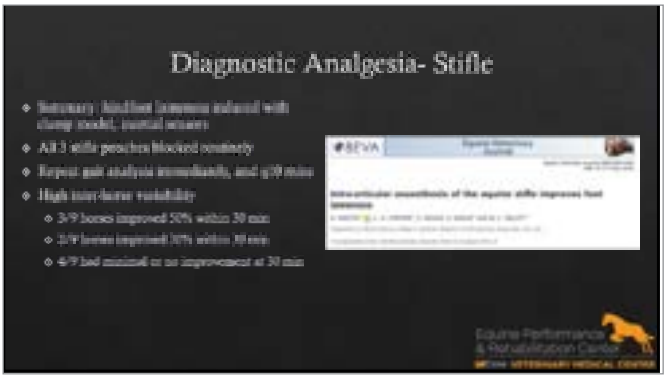

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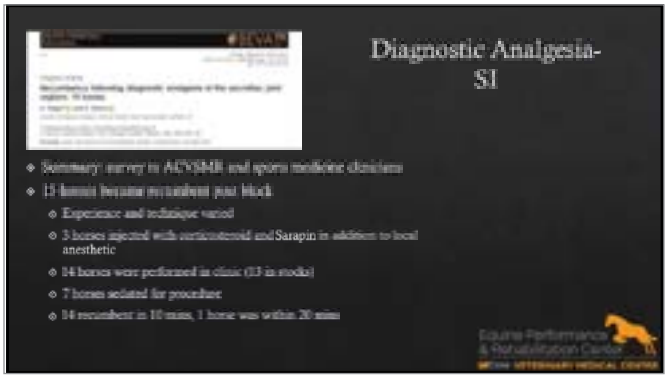

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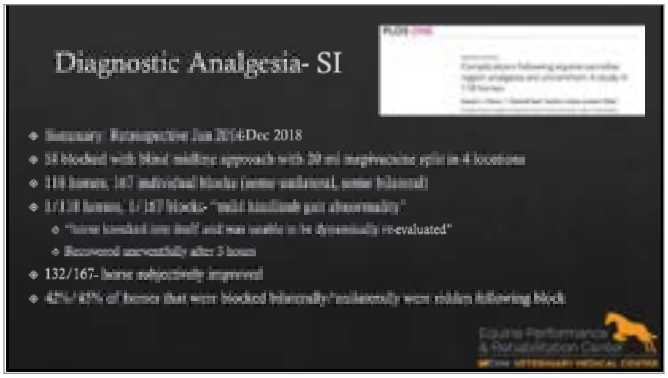

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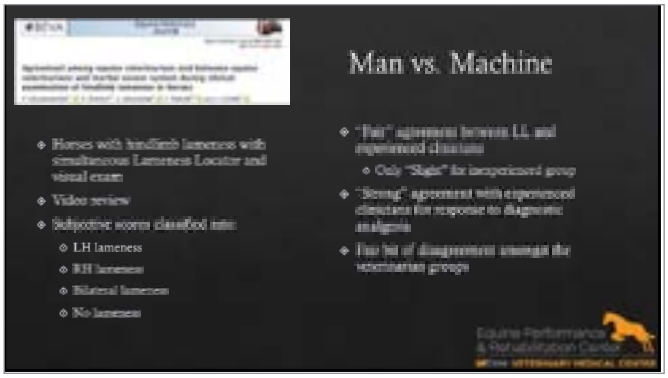

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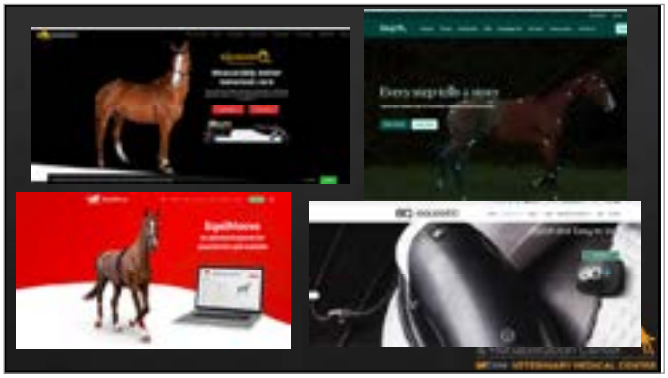

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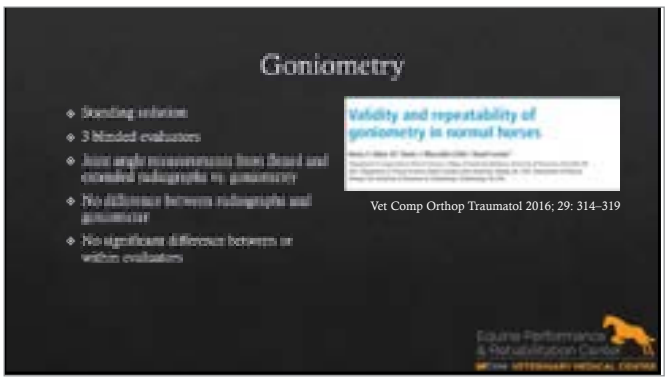

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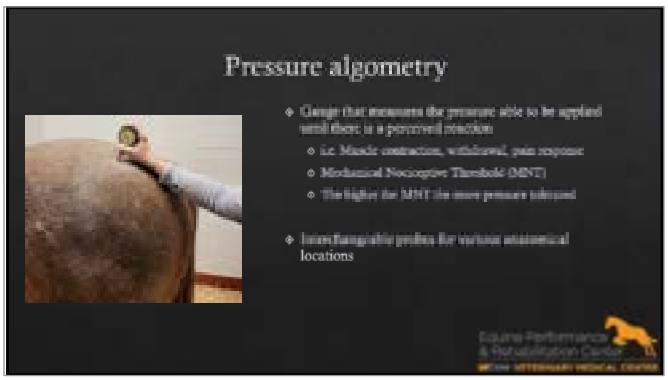

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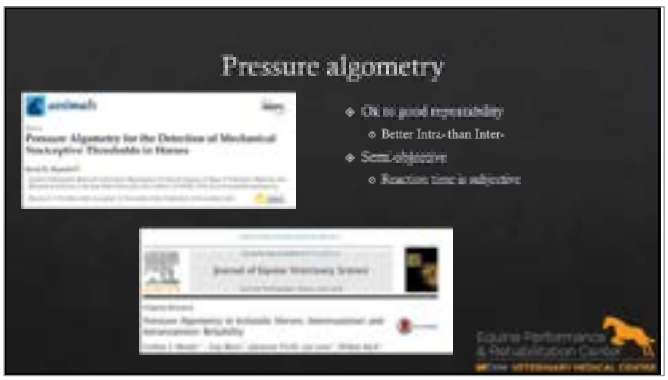

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Confused?

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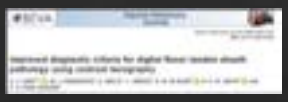
Diagnostics

- High and low field MRI performed day of (<12 hours) or day after (<36 hours) lameness exam
- Blocks:
  - PD
  - Abaxial
  - DIPJ
  - MCP
  - Navicular Bursa
- No artifacts on any sequence that could confound interpretation

24

Diagnostics



- 206 Horses that blocked to tendon sheath
- Contrast radiography- sensitivity and specificity for
  - DDFT tears
  - Manica flexoria (MF) tears
  - Palmar/plantar annular ligament (PAL) constriction
- High specificity and sensitivity for MF tears
- Lower specificity and sensitivity for PAL constriction
- Ponies and Cobs= MF tears
- TB, warmbloods and drafts= DDFT tears
- MF and PAL overrepresented in landrobe
- DDFT overrepresented in Standardbs

25

Diagnostics

- Horses with radiographic and MRI of proximal MT3
- Rads assessed for sclerosis and lucencies
- MRI assessed for endosteal sclerosis and bone marrow lesions, plantar cortical proliferation and enthesiopathy and proximal suspensory desmopathy
- Rads- 54% limbs had enthesis changes
- MRI- 46% limbs had enthesis changes
- No association between presence of radiographic changes and PSD on MRI
- Significant association and positive correlation between presence of radiographic changes and MT3 cortical proliferation or enthesiopathy on MRI

26

Lameness Causes

- 30 horses with DDFT pathology on Ultrasound of pastern
- 75% of all cases had distal enthesiopathy within foot/insertion
- 97% of cases with core lesions in pastern had distal changes within foot

27

Lameness Causes

- Average grade 2.21/5
- 54% forelimb 46% hindlimb
- Forelimb
  - 40% of all FL in distal limb (PD and Abaxial)
- Hindlimb
  - Hock/prox MT in 36% of all cases
  - Stifle 9% all cases
- All around horses most commonly presented with single hindlimb
- Barrel horses most commonly single forelimb

28

Concurrent Dysfunction

- Horses with hindlimb lameness had inertial sensors on
  - withers, T13, T18, L3, sacrum, and left and right tuber coxae
- Measured:
  - Hiphike difference, maximum difference, minimum difference,
  - ROM in flexion-extension (sagittal plane), axial rotation, lateral bending, dorsoventral, lateral/lateral motion and vertical movement symmetry

29

Concurrent Dysfunction- Topline

- After horse blocked sound
  - MinDiff decreased significantly at tuber sacrale, L3 and T18
  - Increased ROM in flexion-extension at T13
  - Increased axial rotation at T13, T18, L3
  - Increased lateral/lateral ROM at L3
- *** Hindlimb lameness causes immediate altered motion of the axial spine***

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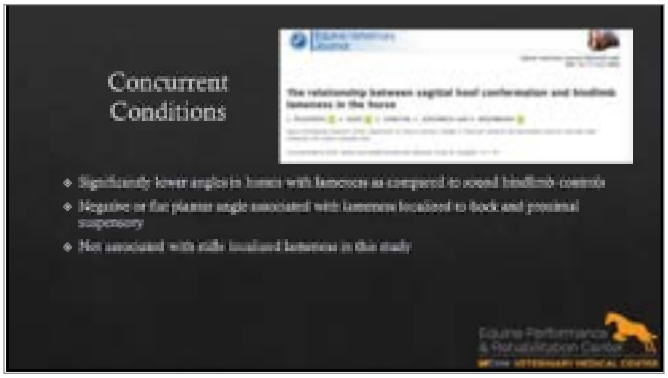

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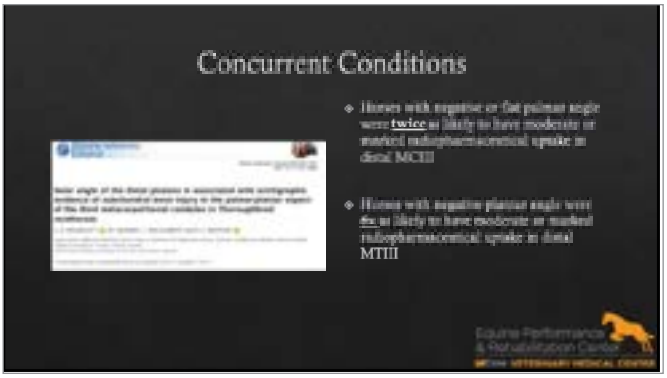

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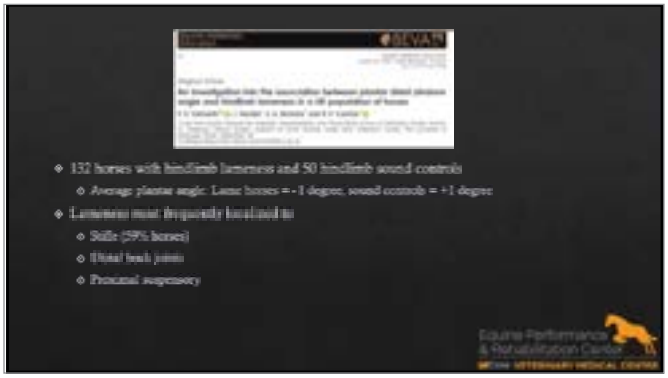

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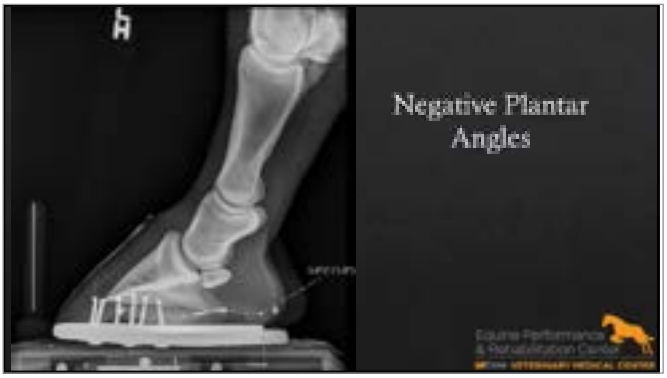

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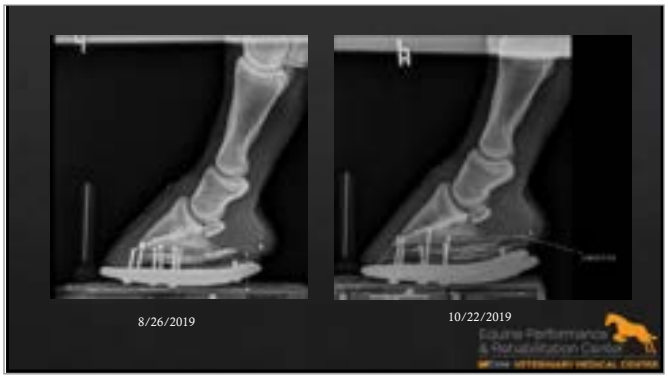

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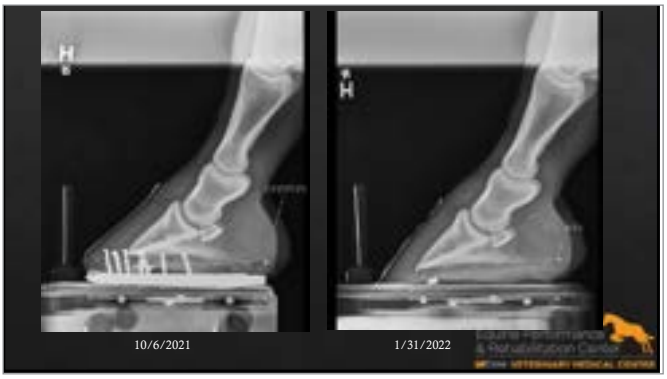

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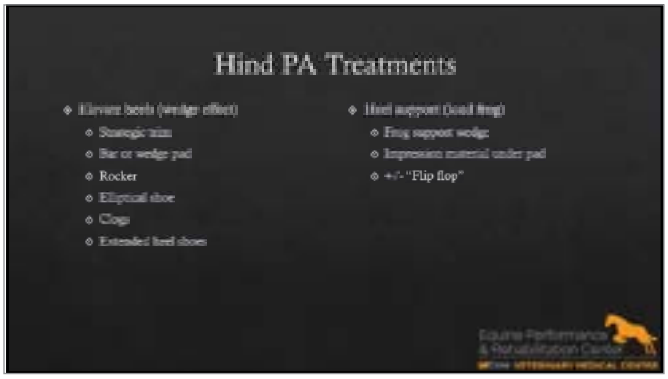

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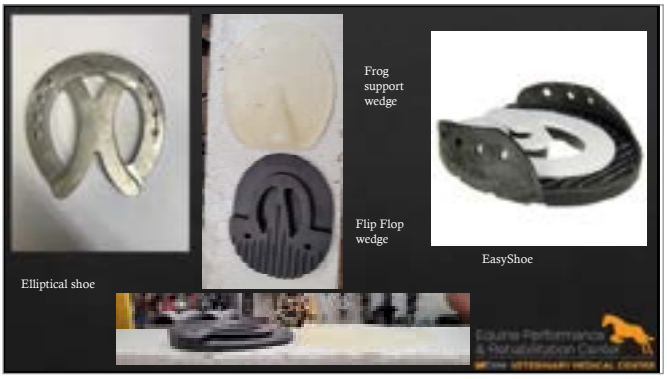

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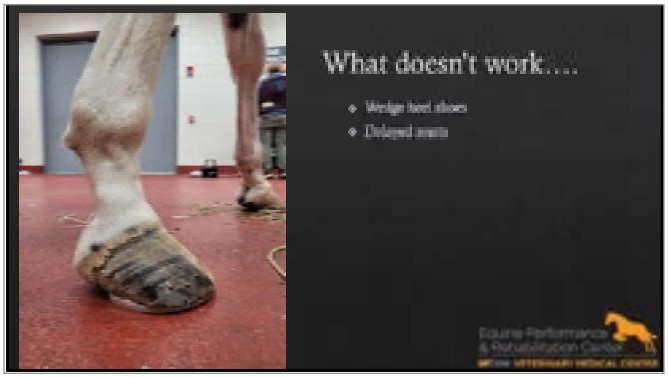
What doesn't work….
Equine Performance & Rehabilitation Centre

39


Denoix Hind Suspensory Shoe
Equine Performance & Rehabilitation Centre

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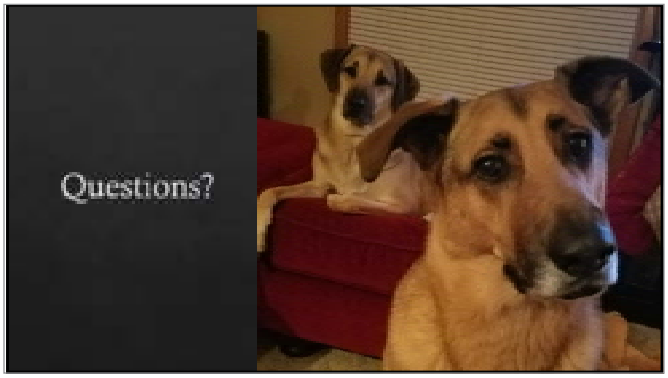
Questions?

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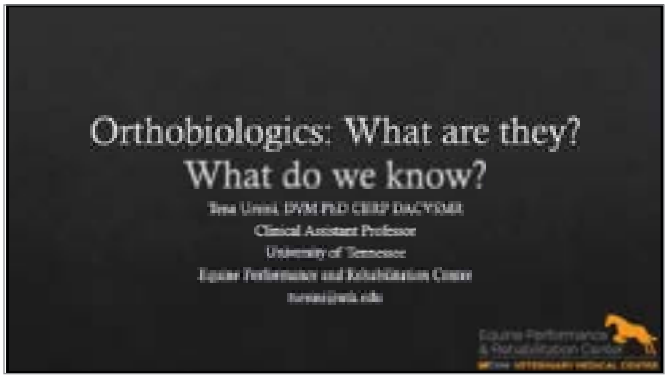
11002

ORTHOBIOLOGICS – WHAT ARE THEY, WHAT DO WE KNOW

SPORTS MEDICINE AND REHABILITATION

Tena Ursini, DVM, PhD, DACVSMR, CERP



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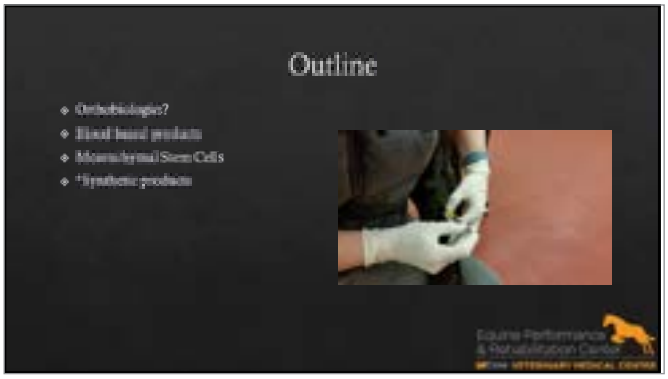

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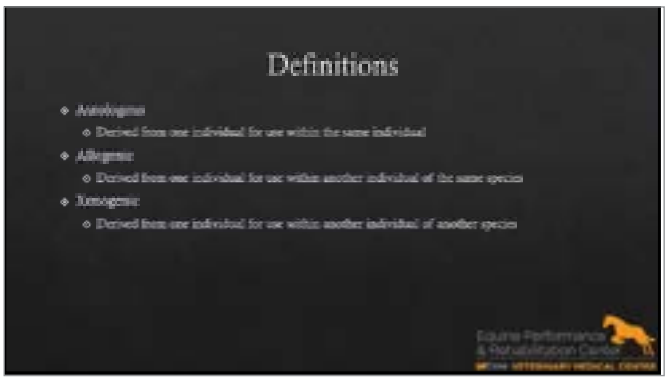

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ACS/IRAP

- IRAP- Interleukin-1 receptor antagonist protein
  - Main pro-inflammatory cytokines
    - IL-1 and TNF-α
    - MANY others!
- Blood collected into incubation device with glass beads (24 hours)
  - Promotes activation of macrophages
    - Secrete anti-inflammatory cytokines
    - IL-1ra and MANY OTHERS

7

ACS/IRAP

- Typically used in synovial structures
- Synovitis
- Repeat injections q 10-14 days
- Alternative to IA corticosteroids
  - FEI
  - Metabolic status
  - Juvenile OA

https://www.arthrexvetsystems.com/vet-orthobiologics/irap-ii
https://www.dechra-us.com/our-products/us/equine/horse/prescription/orthokine-vet-irap-60

8

Autologous conditioned serum. The comparative cytokine profiles of two commercial methods (IRAP and IRAP II) using equine blood

- Multitude of cytokines measured
  - Anti-inflammatory
    - IL-1 Receptor Antagonist
    - IL-10
  - Growth Factors
    - IGF
    - TGF-β
  - Pro-inflammatory
    - IL-1β
    - TNF-α
- All cytokines were significantly higher in both ACS products after 24 hours when compared to the 1 hour control
- Compared to the 24 hour control, both ACS products showed significant increases in IL-1ra, and IGF
- No significant changes in any other cytokine at 1 hour 24 hrs

9

Effects of serum and autologous conditioned serum on equine articular chondrocytes treated with interleukin-1()

- In vitro evaluation of ACS product on chondrocyte pellets produced from normal horses
- Pellets were treated with IL-1β (model for OA) and ACS or unprocessed serum
- Measured
  - GAG synthesis
  - GAG total concentration
  - IGF-1 concentration
  - mRNA content for
    - COX-2, MMP-3, and Aggrecan
- ACS significantly increased IL-1ra, but no anabolic effect on proteoglycan metabolism was seen
- Chondroprotective!
- Role of ACS in diseased cartilage!

10

Evaluation of Autologous Conditioned Serum Using an Experimental Model of Equine Osteoarthritis

AAEP Proceedings, 2005

- Induced osteochondral fragment of middle carpal joint
- 6 mls of ACS or saline injected 14, 21, 28 and 35 days post-op
- Exercised starting 14 days post-op
- Synovial fluid, serum, and lameness evaluated every 2 weeks
- ACS treated horse had significantly reduced
  - Lameness scores
  - Synovial membrane hyperplasia
- ACS treated horses tended to have improved cartilage on gross necropsy and immunohistochemistry evaluation
  - Not significantly
- No change in the IL-1ra levels of the synovial fluid measured overtime

11

PRP

- Platelet rich plasma
- Stall side bio-immediate injection
- Can process manually- over many aliquots
- Concentrates platelets
  - Growth factors released from α-granules
    - IGF1, TGF-β
- Ideally, concentrations of 1-4x systemic blood
- Intra- or extra-(synovial tuff tissue issues)
- Controversy!- Leukocyte count

12

Effects of Platelet-Rich Plasma on the Quality of Repair of Mechanically Induced Core Lesions in Equine Superficial Digital Flexor Tendons: A Placebo-Controlled Experimental Study

- Experimental induced core lesion in both forelimb SDFTs 7-8 cm in length using arthroscopic burr, just proximal to the flexor tendon sheath
- 7 days post-op, single 3 ml injections of PRP injected with ultrasound guidance into one core lesion, 3 mls of saline injected into the other forelimb
- 16 weeks post operatively
  - PRP treated tendons
    - Higher collagen, GAG, cellularity
    - Better organization of repair
    - Higher strength at failure
    - Increased metabolic activity
- PRP seems to advance maturation of repair tissue over non treated tendons

13

Extracorporeal Shockwave Therapy Increases Growth Factor Release from Equine Platelet-Rich Plasma In Vitro

- Shockwave increased:
  - TGF-β concentration 33-46% compared to negative control (without a freeze thaw cycle)
  - PDGF (platelet derived growth factor) concentration 396-219% compared to negative control
- PDGF and TGF-β concentrations highest in positive control (3 rest-thaw cycle)

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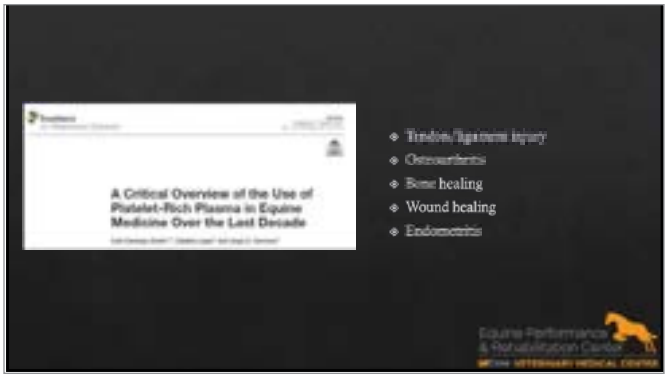

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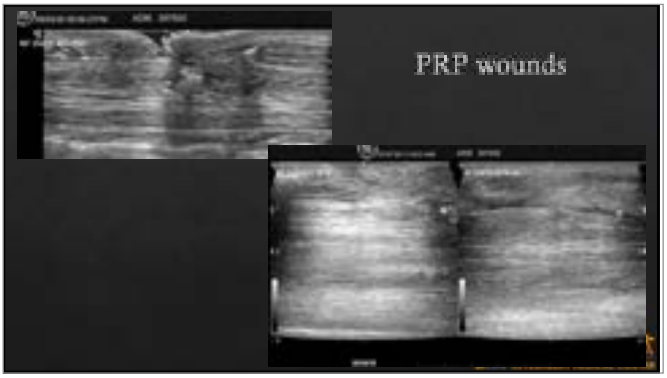

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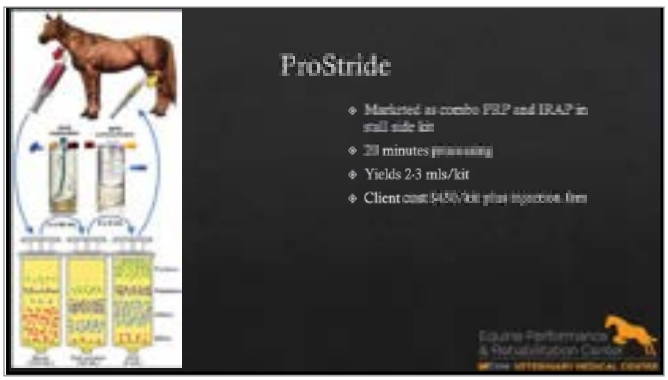

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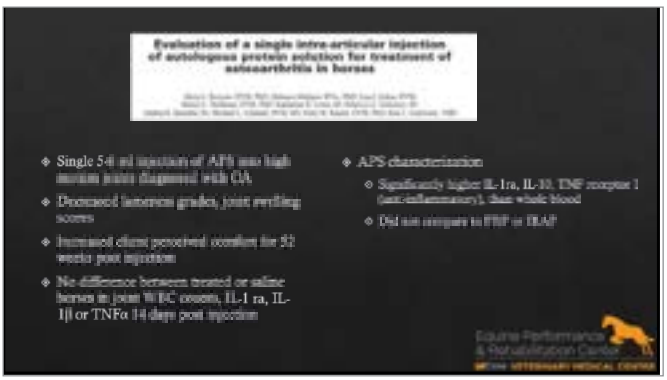

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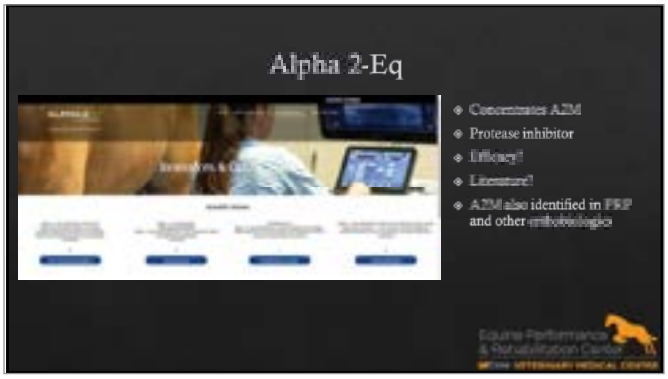

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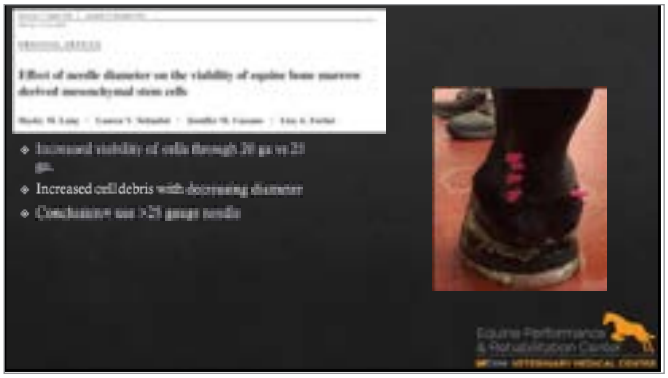

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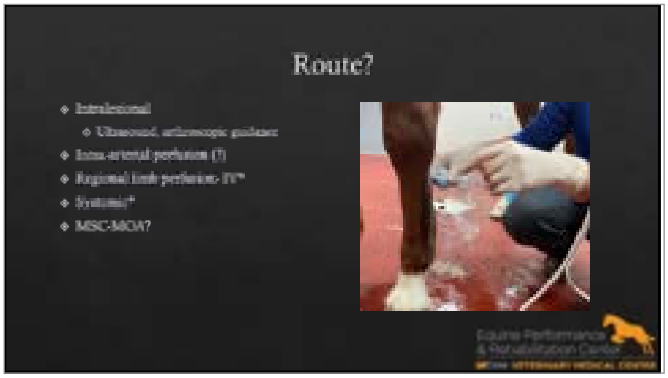

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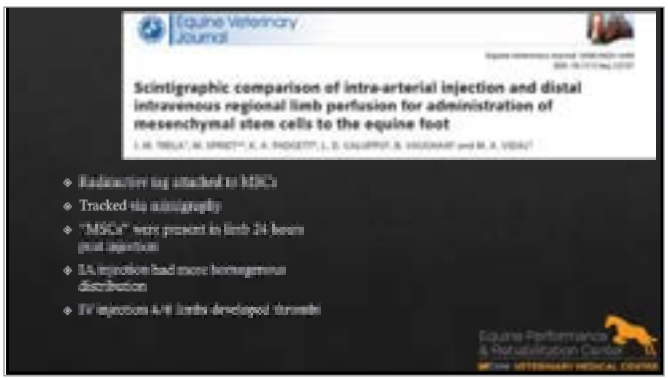

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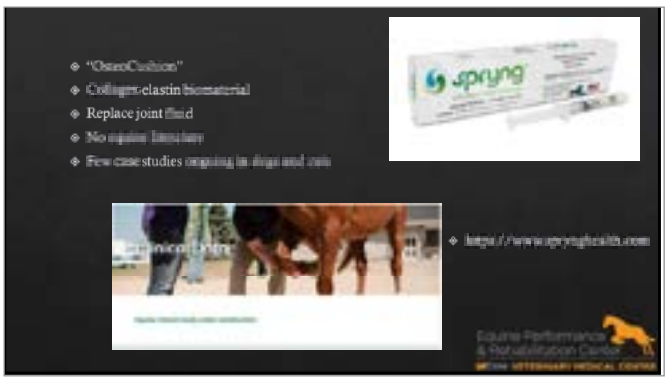

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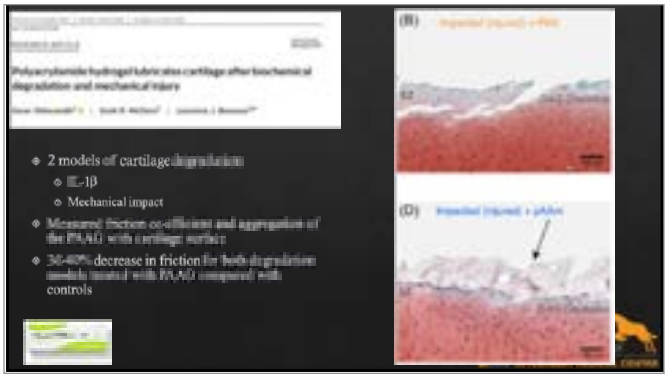

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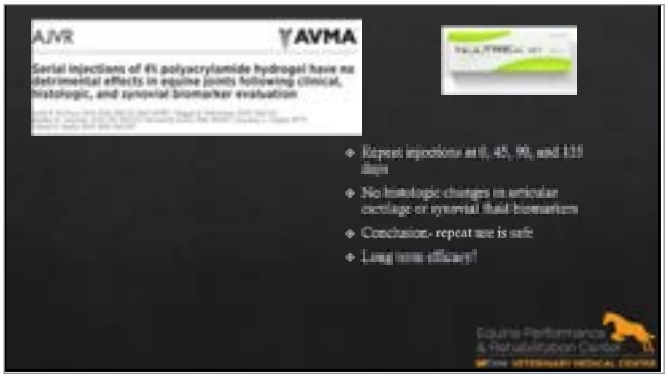

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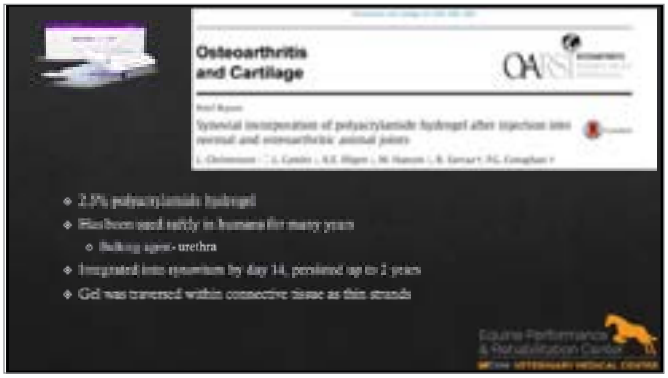

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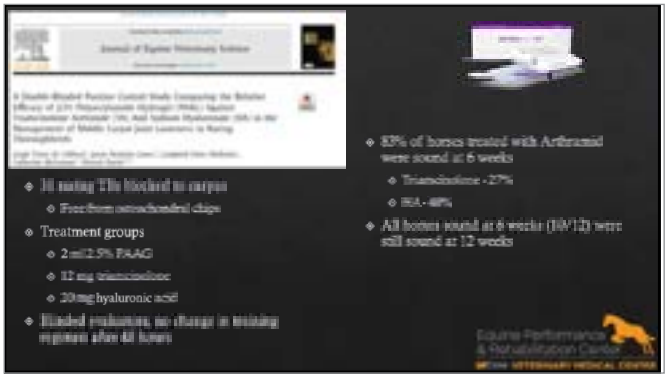

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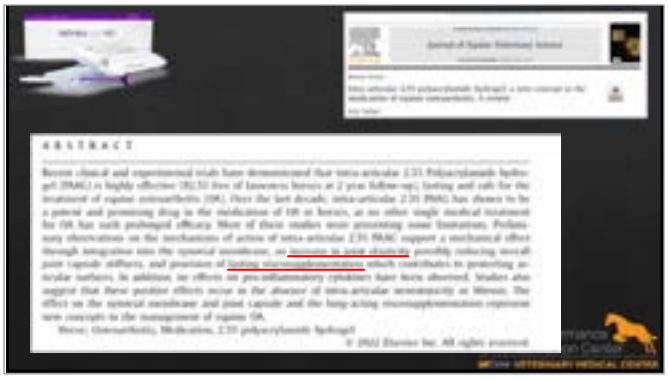

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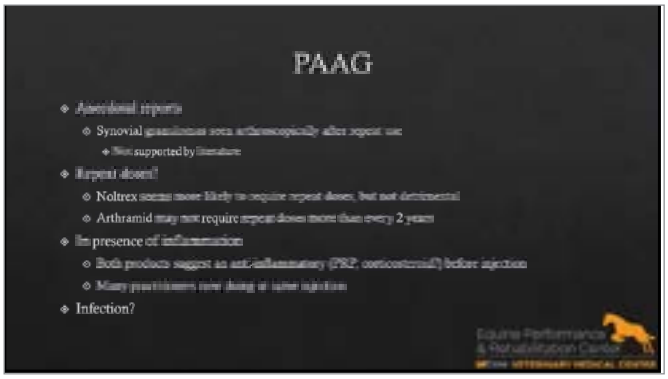

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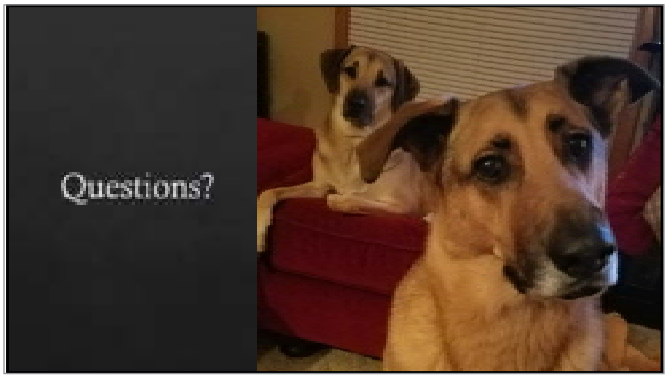

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11003

TOPLINE DYSFUNCTION – THE NEW DEFINITION OF BACK PAIN

SPORTS MEDICINE AND REHABILITATION

Tena Ursini, DVM, PhD, DACVSMR, CERP

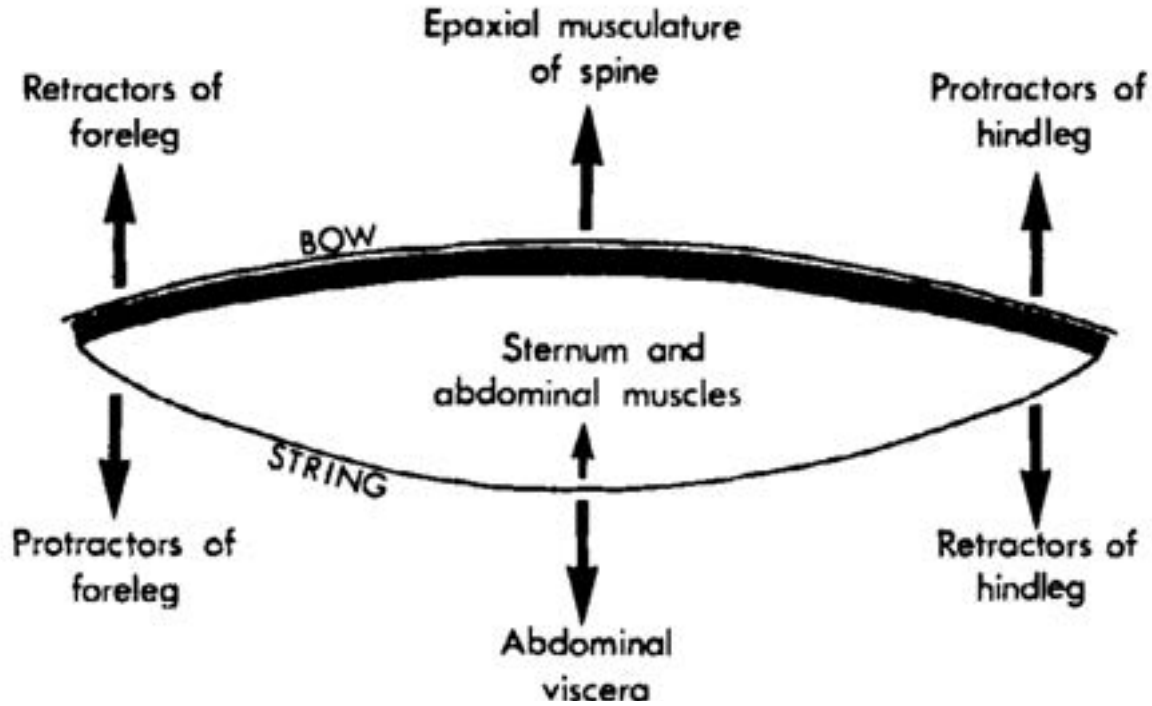
WHAT IS BACK PAIN?

Back pain has become a catch all term in equine practice that could refer to anything from "my horse flinches when I brush him" to "he tries to murder me when I set a saddle pad on his back." Back pain can be caused by a multitude of underlying conditions such as: overriding dorsal spinous processes, vertebral facet disease, and topline dysfunction to name a few. However, I could argue that often times it's the topline dysfunction (further defined below) that becomes the most clinical. Back pain is becoming an incredibly popular diagnosis and is typically being treated without the most directed therapies in many cases.

BIOMECHANICS OVERVIEW

The bow and string model is the most widely accepted descriptor of forces applied to the equine skeleton.

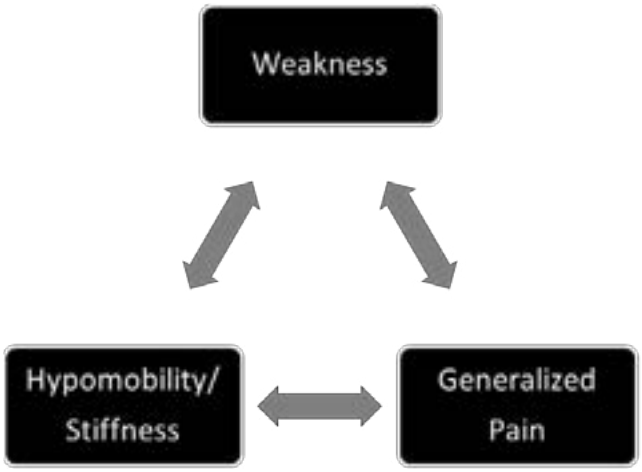
However, this figure [1] does not take into account external conditions. The magnitude of these forces could change widely with the added weight of a rider or certain athletic activities such as jumping. Therefore, the topline of ridden horses must be strong, flexible and able adapt and respond to dynamic changes in stability on a stride to stride basis.



TOPLINE DYSFUNCTION

Topline dysfunction is a term this author has coined in order to more accurately describe back problems in horses and provide a more complete diagnosis. "Back pain" just isn't descriptive enough to be able to describe the three main components this author appreciates when horses present for poor performance and lameness.

Topline dysfunction has three main components: weakness, hypomobility, and generalized pain.



Regardless of the initial stimulus to one of these components, the author notices the other two components of the disease process coincide quickly. Then, the horse is trapped in a vicious cycle. For example, once they are weak, they're generally in pain, which leads to compensatory mechanisms contributing to stiffness and hypomobility. Without proper motion, the muscles continue to not fire properly, leading to more weakness. The same is true no matter where in the cycle the horse starts, whether it be pain from an improperly fitting saddle, stiffness from lack of stretching, or weakness from confirmation or age. Therefore, the practitioner MUST ensure that all three components are being addressed with equal focus in the rehabilitation plan. Just treating pain, for example, does nothing for the other two components, and the horse will not be able to perform.

INTRINSIC FACTORS

There are many intrinsic factors that are associated with and may predispose horses to topline dysfunction.

1. Plantar angles- In my opinion, the most important intrinsic factor is hind foot angle. Flat or negative plantar angels of the third phalanx are highly associated in horses with lameness or pain originating from the hock/proximal metatarsal, stifle, sacroiliac, and back.

2. Hindlimb lameness- this could be listed in order of importance as 1B as usually horses with negative angles do have hind limb lameness as well.

3. Age- whether a factor of the accumulated miles and "wear and tear" on the equine athlete, or a function of decreasing muscle mass, older horses tend to be over-represented. However, the young horses (~2-3 year olds) are also seen, usually from being asked to carry a load on an unbalanced/growing frame or without the adequate core strength.

4. Conformation- the long thoracolumbar, short hindquarter, straight hind leg conformation seems to be predisposed. This is likely due to the inherent core weakness these horses present naturally.

5. Breed- likely more related to confirmation, but the breeds more likely to be long backed with less hind quarter strength are seen frequently

EXTRINSIC FACTORS

Extrinsic factors are also of major concern.

1. Discipline- probably the most common extrinsic contributing factor is the profession or discipline of the horse, however this is seen across ALL disciplines. Western performance horses are routinely worked in a saddle that allows little to no flexibility for lateral bending. Additionally, many disciplines ask horses for an upright head and neck carriage (i.e jumpers, dressage, saddle seat, performance arabs) which if done incorrectly contributes to a lordotic thoracolumbar posture

2. Rider Ability- as eluded to above, the overall ability of the rider to be able to have the horse engage the core and hindquarters is paramount. Youth and adult amateur horses are over-represented, likely for two reasons. Youth riders require more training repetitions and lessons to develop confidence and some degree

of competence, especially in over fences disciplines. Amateur riders are also less likely to be able to engage the horse to use its body appropriately. Thus, lots of miles of improper motion.

3. Saddle fit- in this scenario the saddle itself causes the pain, thus the horse is less willing or unable to engage the core and topline, leading to weakness and the vicious cycle begins.

4. Rider/horse mismatch- this could be on several levels. The overall athletic ability, talent, and goals of both the horse and the rider should match. The horse also has to physically be able to carry the rider in question. The "rule" is for the rider and tack to consist of no more than 15% of the horses body weight. However, there is emerging belief carrying a load even 10% of their body weight changes the biomechanics of the body. Muscle mass should also be taken into account, as the author has had several clients present with the belief that if they just feed the horse more (thus increasing body condition and fat content) that the 15% rule will be more attainable, making it acceptable for them to ride a horse too small for them.

COMORBIDITIES

Even though they are stated above, Id like to especially highlight certain conditions that predispose horses to topline dysfunction: Flat or negative plantar angles, and lameness both need to be assessed and address concurrently with topline dysfunction, or the horse will continue to move improperly and will never build the strength and mobility required.

Cervical Muscle Pain		Cervical Stiffness to Baited Lateral Bending	
0	No pain, mild withdrawal with firm pressure	0	No palpable restriction, fluid motion
1	Mild, mod withdrawal with firm pressure	1	Mild stiffness, mild reduced range of motion
2	Mod, Mod withdrawal with light pressure	2	Mod, moderate reduced ROM, adequate hold
3	Severe, Mod withdrawal with light pressure	3	Mod, moderate reduced ROM, inability to hold
4	Complete avoidance of contact	4	Severe, marked reduced ROM
Thoracolumbar Expaxial Muscle Pain		**Thoracolumbar Expaxial Muscle Hypertonicity**	
0	No pain, mild withdrawal with firm pressure	0	No palpable hypertonicity
1	Mild, mod withdrawal with firm pressure	1	Mild hypertonicity with firm pressure
2	Mod, Mod withdrawal with light pressure	2	Moderate hypertonicity with firm pressure
3	Severe, Mod withdrawal with light pressure	3	Moderate hypertonicity with light pressure
4	Complete avoidance of contact	4	Severe hypertonicity
Thoracolumbar Expaxial Muscle Atrophy		**Thoracolumbar Stiffness to Lateral Bending**	
0	No atrophy of Longissimus Dorsi, Spinalis, or Iliocostalis	0	No stiffness to lateral bending
1	Mild (circle): Long. Dorsi - Spinalis - Iliocostalis	1	Mild stiffness, improves with mobilizations
2	Moderate (circle): Long. Dorsi - Spinalis - Iliocostalis	2	Moderate stiffness, does not improve with mobilizations
3	Severe (circle): Long. Dorsi - Spinalis - Iliocostalis	3	Severe stiffness, elicits splinting and counter bend

Axial Traction/Tail Pull		Sternal Lift		Pelvic Flexion/Butt Tuck	
0	Accepts pressure, attempts to walk forward	0	Full ROM, holds position	0	Full ROM, holds position
1	Accepts pressure, no attempt to walk forward	1	Dec ROM, short hold	1	Dec ROM, short hold
2	Does not accept pressure, slight avoidance	2	Dec ROM, no hold, spasm	2	Dec ROM, no hold, spasm
3	Does not accept pressure, mod avoidance (backs)	3	No response, avoidance	3	No response, avoidance

ASSESSMENT

All three components need to be assessed and described in order to assess response to treatment and dictate the treatment plan going forward. The author has developed this grading scale for simplicity, but it can easily be modified to fit any practitioner's needs.

1. Weakness assessment- muscle fluidity and evidence of spasm during active motion (baited stretch), overall topography/atrophy, and the horse's ability to hold each posture. Achieving the position is not enough. You will find many horses can "throw" their body around to snatch a treat, but real strength through motion is associated with being able to hold the posture

2. Mobility assessment- ability of horse to achieve each position during baited stretch, taking into account the horse's confirmation (short necked horses are going to have a harder time in general, but that doesn't mean we don't compare them to the gold standard). I also do direct perturbations of the thoracolumbar spine with lateral manipulations to assess stiffness of each segment.

3. Pain assessment- avoidance behavior during any portion of the exam should be taken as a sign of pain. This includes during direct palpation and during mobilizations. This should be specifically noted during sternal lifts, tail pulls, butt tucks, and lateral mobilizations.

WHAT IS "GENERALIZED PAIN?"

In the authors opinion, it is important to distinguish between generalized muscle and body pain and focal acute pain. Focal pain palpated in specific locations (specifically associated with a dorsal spinous process or interspinous ligament) is very different than the pain referred to here, associated with topline dysfunction. The elephant in the room with this whole discussion is the currently common diagnosis of overriding or impinging dorsal spinous processes. Despite it commonly being diagnosed and treated with extensive surgery at times, the diagnostics available to definitively rule in or out bone pain associated with the dorsal spinous processes is limited. Some base it simply on the clinical signs of "back pain" and radiographic evidence of "narrow spaces." Without going too far off topic, its been very well established that radiographic evidence of bony change has a very poor correlation to clinical signs. Horses with severe pain can have minimal signs, and horses with no signs of back pain can have horrendous radiographs. A recent review of jumpers showed that the greater number and severity of lesions was directly correlated with competing at the highest levels, and being successful. Said another way, the horses that won at the highest levels, over the highest jumps had the worst lesions. Additionally, even if a horse is suspicious for overriding dorsal spinous processes, one can almost guarantee there is concurrent topline dysfunction. A conservative approach such as that outlined below will resolve a vast number of horses without surgery. Surgical procedures now are related to destabilizing the spine and likely denervating the multifidus muscle (one thought to be a major contributor to intersegmental stabilization). Therefore, other portions of the spine could be predisposed to injury after surgery. And again, the vast majority of horses are likely to improve with continued and progressive therapeutic exercise.

TREATMENT

Since the condition itself consists of three separate problems, treatment should also be directed to each of those. While every horse will have one or more predominant components, the practitioner MUST address all three to ensure that the horse improves and is able to return to athletic use and does not decompensate. Treatment MUST start with addressing the underlying components outlined above first (i.e lameness, plantar hoof angles, etc). The following treatments for the topline can be started concurrently, but failure to address the underlying problems will result in failure to respond to treatment.

Hypomobility

1. Baited carrot stretches, especially those directed at getting the horse to reach as caudally as possible to each side (hocks/hind fetlocks).

2. Radiofrequency therapy- modality shown to provide heat at depth

3. Routine chiropractic adjustments

4. Leg yields and lateral motion (NOT formal half-pass)

Weakness

1. Underwater treadmill- especially deep levels only achieved with the in-ground units.
2. Ground poles- shown to increase muscle activation of both the longissimus dorsi and multifidus muscles at the trot
3. Equicore- the equicore device will increase activation of the rectus abdominis, but decrease the longissimus dorsi and multifidus, so I use this device a MAX of 2 days per week
4. Transitions- both between gaits (i.e canter-walk, trot-walk) but also within a gait (extended trot to collected) with the focus on balance and muscle control
5. Long-lining- this is a great alternative to encourage rounding of the back without having a rider add weight to the back.

Pain

Acute

1. Therapeutic Exercise
2. Electroacupuncture- especially if there is a soft tissue/ muscle spasm component
3. Chinese Herbs- (Jing Tang Body sore is the one I prefer in the first 3 months of treatment
4. +/- NSAIDs
5. +/- muscle relaxers (i.e methocarbamol)

Chronic

1. Therapeutic Exercise
2. Chiropractic manipulations
3. Chinese Herbs- (Jing Tang Body Sore)
4. Radiofrequency
5. Shockwave
6. +/- NSAIDs
7. +/- muscle relaxers (i.e methocarbamol)

RE-EVALUATION

The overall therapeutic plan should be reassessed regularly. During intensive inpatient plans, patients are formally evaluated once weekly, for us to continue to tailor and customize the plan to continue progression. At-home plans are inherently less intensive (no owner can recreate the 4-6 daily exercise sessions our facility can provide, no matter how diligent or committed they are), and thus should be re-evaluated every 6-8 weeks. At-home plans also have limited or no access to underwater treadmill, which I believe to be a major contributor to success. In general, I tell owners that what I can do with a 3 month intensive in-patient plan, will take them a minimum of 6 months to achieve at home, assuming they are diligent about exercise 5-6 days per week.

RIDDEN WORK

Depending on the severity of topline dysfunction, I may suggest that the horse not receive ridden work during the beginning of the therapeutic exercise program. The vast majority of my clients are amateurs who do not have the overall ability to make a horse round its back and drive from the hind end. Therefore, every step they take under saddle with an inverted posture, takes 5-10 steps of proper motion to combat the detrimental effects to weakness, hypo-mobility, and pain. Horses with moderate to severe changes on exam, that continue to be ridden, are the ones that fail to achieve full athletic potential. IN my experience its best to build the horse back up first, then allow the rider to just try to maintain the strength, and mobility while enjoying their horse in a pain-free state.

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2. *The content provided here has been previously presented by the author at:*

- *Veterinary Medical Expo, Orlando, FL January, 2024*
- *American College of Veterinary Sports Medicine and Rehabilitation Symposium, Naples, FL April, 2024*

11004

NEUROLOGIC REHABILITATION – RETRAINING AND REBUILDING

SPORTS MEDICINE AND REHABILITATION

Tena Ursini, DVM, PhD, DACVSMR, CERP

REHABILITATION CONSIDERATIONS OF THE NEUROLOGIC EXAM

A complete neurologic exam should always be performed. However, there are specific considerations to take note of during the neurologic exam that pertain specifically to rehabilitation and implementing a successful rehabilitation and reconditioning plan.

Definitions

- Posture: the "resting position"
- Proprioception: awareness of body position in space
- Ataxia: lack of muscle coordination
- Gait Deficits: deviation from a normal gait pattern
- Paresis: Muscle weakness

Cranial Nerves/Mentation

In reference to rehabilitation of the centrally affected neurologic horse, cranial nerves can be assessed by asking the following questions:

1. Is the horse visual?
2. Can the horse prehend and swallow enough calories to maintain body condition with exercises prescribed?
3. Is there normal facial expression? (Facial nerve paralysis can also benefit from a specific rehabilitation and treatment program, however, that is beyond the scope of this manuscript.)
4. Is the horse mentally appropriate?

Standing exam

Observation of the horse at rest, can give the practitioner a significant amount of information. Attitude, mentation and general living assessments can be made at distance with the horse loose in a box stall. Pay particular attention to how the horse interacts with the environment of the stall without restraint. Does the horse bump into things? What is its overall temperament (excited, aggressive, sullen, etc)? Getting a feel for how the horse is tolerating the neurologic condition will give you a lot of insight on which exercises or treatment modalities may be considered. For example, a horse that tends to overreact, panic, and not respect his boundaries may need a period of retraining before it is safe to introduce that horse to the underwater treadmill.

After observing the horse loose in a box stall, perform a complete musculoskeletal palpation exam, paying special attention to mobility, muscle atrophy, compensatory muscle or soft tissue pain or spasm, secondary joint injury, and hoof balance. These will all need addressed concurrently with the primary neurologic condition.

Moving exam

Given the subjective nature of the entire neurologic exam (covered elsewhere), practitioners should assess each neurologic parameter (posture, proprioception, ataxia, paresis, and gait deficits) during each manipulative test to get a firm idea of the horse's baseline condition. For example, proprioception may be worse on a firm surface as compared to soft impressionable footing. Additionally, gait deficits or lameness may be more apparent on one surface or condition than another. These could be subtle differences that may help track progress going forward.

The horse should be walked and trotted on multiple surfaces to assess for concurrent lameness in addition to neurologic deficits. The horse should also be assessed while performing basic tasks such as navigating an obstacle. This could be stepping on and off a short curb in the driveway, or waking a serpentine over a series of ground poles placed end to end lengthwise, or stepping on and off a small pedestal.

All these tests (with the exception of trotting) should also be performed while trying to limit the horse's ability to compensate visually. The author prefers to just elevate the head to limit visibility of the ground. However, horses with cervical pain may resent this motion. Another option is to blindfold the horse with a towel or other small cloth. I've seen too many neurologic horses panic at this stage, and it can become very dangerous in a neurologic animal. I will reserve blindfolding only for the mildest of cases in horses that are reasonably well tempered.

Manipulations

Some horses that have fairly subtle clinical signs, require further manipulations to get a true baseline for each deficit. These include walking up and down a hill (with and without the head elevated), placing the feet base narrow and base wide to assess their response, the "clunk" test where a front foot is picked up and unexpectedly dropped, and tail pulls to both sides. While these are explained in further detail elsewhere, a few important notes. The clinician must take into account the level of training and overall willingness of the horse to "get along." Horses with halter or showmanship training are trained to leave their feet wherever they are placed and not move. Additionally, some horses just tend to go along with the flow, especially in placement tests and tail pulls. The subjective nature of all of these tests makes them difficult to judge at times. The "clunk" test is one that the author finds especially useful. The front foot is lifted and held, such as to assess the sole. Then the foot is abruptly dropped without giving the horse warning. Typically, on the first attempt, the horses foot will "clunk" into the ground. As we say, "the first one is free" because the horse didn't really know you would do that. However, on attempts 2+ the horse should learn what you are about to do and catch the limb, before clunking the foot into the ground, in order to place the foot appropriately.

DIFFERENTIALS AMENIABLE TO REHABILITATION

Differentials to rule OUT

- Acute spinal or skull fracture
- Acute infection (Equine protozoal myeloencephalitis, Rabies, Equine Herpes Virus-1, West Nile Virus, Tetanus, other Encephalitidies)

Differentials to rule IN

- Subacute/ recovering infection (EPM, EHV-1, WNV, Encephalitidies)
- Stabilized fracture/ post intervertebral fusion
- Cervical Stenotic Myelopathy
- Other resolved neurologic disease or trauma
- "Sidewinder" syndrome

SYSTEMIC CONSIDERATIONS

Treat primary differential

Whatever the primary cause of neurologic disease, it should be appropriately treated either medically, surgically, or the combination as clinically appropriate

Anabolic metabolism

Weakness (paresis) and atrophy being a common component to neurologic disease, either as a primary cause or secondary to the gait changes, it is important that an overall anabolic state is encouraged. This is not to say every horse requires high levels of calories. However, practitioners should ensure the horse is receiving a balanced diet with proper levels of protein, macro-, and micro-nutrients and energy.

Protein supplements such as Purina Super Sport provide branch chain amino acids to promote muscle building.

Anti-inflammatories

Almost every horse with neurologic disease benefits from some level of anti-inflammatory treatment. Those with acute musculoskeletal pain could receive typical NSAIDs (phenylbutazone, flunixin, firocoxib). Vitamin E is of benefit as a neuro-protective anti-oxidant. Additionally, Omega-3 fatty acids, specifically those with therapeutic levels of decosahexaenoic acid (DHA) could be added to the diet.

REHABILITATION GOALS

The main goals of every neurologic rehabilitation plan should be to improve the neurologic parameters (posture, proprioception, ataxia, paresis, and gait deficits) each horse is particularly weak in. A customized approach based on the needs of each horse is always going to be more successful than a "cook book recipe" approach to every case.

THERAPEUTIC EXERCISES

Specific exercises and an overall therapeutic exercise plan is based on two main factors: fatigue and safety. Neurologic horses in general will fatigue much faster than normal horses, thus it is important to constantly assess their overall status and strength before pushing to the next level. This can change on a day-to-day basis and thus the plan needs to be malleable and adaptable to the horse on each individual day. However, progress doesn't happen by being complacent. Athletes of all species need to be pushed out of their current level of comfort to continue to progress and improve.

The same rules of a general exercise plan apply:

- Increase duration OR intensity every 3-5 days, not both at the same time. If you increase complexity and difficulty, you will have to decrease the time.
- Periods of rest and recovery are important. Maximum of 5 days a week of intense forced exercise or 6 days a week of moderate exercise.

Specific exercises that benefit neurologic horses

1. Underwater treadmill
    a. Indications: This modality is the most effective I have seen for building core and hind limb strength. Especially when horses are strong enough to tolerate the high resistance of added turbulence in the water (blowers or jets). I find the in-ground units to be safest and most effective, but the above ground units could also be used.
    b. Contraindications: Horses with moderate to severe hind end weakness may have difficulty navigating the ramps entering and exiting the in-ground treadmill. The above ground machines are unable to reach the same water depth as the in-ground units. They also require a step up into the treatment area. Horses with moderate to severe proprioceptive deficits may have difficulty safely getting into the above-ground treadmill.
    c. Important considerations: while UWTM is the best at whole body strengthening, aquatic therapy is known to decrease proprioception overtime. Thus, UWTM should never be a stand alone treatment for neurologic horses.
2. Ground poles/obstacles
    a. Indications: asking a horse to navigate through obstacles such as ground poles, on and off a short pedestal or around cones help challenge the horse to build "eye-hoof" coordination and thus increases proprioception. Additionally, ground poles set at regular walk or trot intervals will encourage gait patterning for gait deficits. Lastly, ground poles have been shown to significantly increase muscle activity of the epaxial muscles [1,2]
    b. Contraindications: None.
    c. Considerations: The complexity of the obstacle course should be tailored to each individual horse. The simplest version of the exercise is poles on the ground at regular predictable intervals. This can be intensified by making them random intervals to challenge proprioception, as well as elevating one or both ends to develop 3-D proprioception. The most advanced version would be the "pick up sticks" pile with poles in a haphazard type pile the horse has to navigate through.
3. Core exercises
    a. Indications: core strengthening is vital to all neurologic horses to help promote whole body conditioning and balance.
    b. Contraindications: none. Every horse can benefit from dynamic mobilization exercises and core strength
    c. Considerations: general exercises include-

carrot stretches (especially reaching to hind fetlocks laterally and between front fetlocks ventrally), butt tucks, sternal lifts, ground poles (see above), lateral work/leg yields, transitions, backing, general "rolled over/ long and low" posture

4. Hind end exercises
    a. Indications: traditionally, neurologic horses tend to be more severely affected in the hind limbs, especially hind limb weakness
    b. Contraindications: some exercises may be too advanced based on baseline strength. Specific exercises should be tailored to every patient.
    c. Considerations: general exercises include- transitions (especially collected to extended), hill work (walking up, serpentine down), UWTM (see above).

MEASURABLE OUTCOMES

One of the most important aspects of managing a horse within a rehabilitation program is to continue to assess measurable outcomes to determine efficacy and needs for modification of any program. These should be assessed formally at least once every 4-6 weeks, and informally on a continuous basis.

Fatigue: fatigue is the major player in how much a neurologic horse can exercise on any given day. Neurologic horses definitely fatigue faster than normal horses, and thus, exercises may need to be tailored on a day by day/hour by hour basis. This would mostly be up to the owner or horseman performing the exercises. Horses should be challenged on a daily basis to encourage growth and conditioning, but we don't want to push them to overt fatigue as safety will become a major player.

Lameness: it is not uncommon for neurologic horses to have concurrent lameness. Whether this is due to overloading of certain structures due to the nature of neurologic disease, or concurrent arthritic or developmental conditions. Lameness should be monitored to ensure it doesn't worsen with the exercise program and require further diagnostics or treatment.

Neurologic exam: the complete neurologic exam should be repeated every 4-6 weeks to ensure the rehabilitation exercises are of the appropriate complexity to continue to drive improvement. Special attention should be paid to each of the neurologic parameters (posture, proprioception, ataxia, paresis, and gait deficits) to ensure a balanced improvement in all categories. Realize proprioception will be one of the first parameters lost and one of the last to recover. But that shouldn't mean we don't expect slow progress throughout the program.

LIFELONG COMMITMENT

The last, but potentially most important aspect, is to remind owners that this is a life-long commitment. We are not "healing" the neurologic system. We are retraining spinal tracts to take over carrying signals they previously haven't. This is not a life long adaptation. Without continued exercise and training to maintain the progress, horses will detrain and return back to their original neurologic baseline overtime, or get worse.

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3. *The content provided here has been previously presented by the author at:*

- *Veterinary Medical Expo, Orlando, FL January,2024*

11005

SEVERE FLEXOR TENDON INJURY - REHABILITATION TO SAVE A CAREER

SPORTS MEDICINE AND REHABILITATION

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INTRODUCTION

Superficial (SDFT) and deep digital flexor tendons (DDFT) are commonly injured in horses of all breeds and disciplines. There are two main mechanisms of tendon injury: 1. An abnormal load applied to a normal tendon; 2. A normal physiologic load applied to a compromised tendon. In the first case, a horse with no previous injury takes a "bad step" and the excessive load exceeds the material strength of the tendon and causes acute rupture. This mechanism is thought to be fairly rare. In the second case, low grade microdamage accumulates due to cyclic trauma that goes undiagnosed until the tendon becomes compromised and eventually is no longer able to withstand normal loads, leading to larger areas of fiber rupture.

Racehorses and western performance horses are over-represented in cases of midbody SDFT core lesions, and DDFT tendinopathies are frequently found as a contributor in horses with caudal heel pain. For this presentation, we are going to focus on the severe SDFT and DDFT lesions encountered in the metacarpal region.

TYPES OF LESIONS

The two main types of lesions encountered in the metacarpal region are core lesions secondary to the acute on chronic cyclic injury described in the introduction, or transverse lacerations secondary to trauma that encompass partial or complete transection of the tendon(s).

PRESENTATION AND DIAGNOSTICS

Horses with these severe injuries are typically lame at the walk and may have significant fetlock drop during weight bearing. There is usually significant swelling associated with the damaged tendon, and there may be a large amount of regional edema if the owner has not bandaged the limb before presentation. Delicate palpation of the area of swelling with the limb non weight bearing, can help determine which structure(s) are involved, however ultrasound should be employed for a definitive diagnosis. Diagnostic nerve blocks are rarely required and may be contraindicated in cases with a laceration. The author finds that partial lacerations usually lead to extensive parasagittal splits proximal and distal to the laceration in the remaining tendon area, which could become completely ruptured with excessive weight bearing after regional analgesia.

Horses presenting with a laceration should have all potential synovial structures explored and treated as clinically dictated, the details of which are beyond the scope of this material. Even without synovial involvement, it is typically helpful to surgically explore, debride, and lavage the laceration. Attempted suture or anchors of the lacerated tendon ends could be performed, however, should not be assumed in every case. Compression necrosis, implant infection, and foreign body reaction are all complications the author has seen clinically. Additionally, no matter with material or technique is used, the damaged tendon may still be unable to withstand physiologic loads, especially during recovery of general anesthesia, even with external coaptation. However, there is benefit of keeping the lacerated ends of the tendon in closer proximity to encourage granulation tissue to fill the gap.

Acute Injury Period (Inflammatory stage day 0-7)

For acutely presented injuries and lacerations immediately post-operative, the rehabilitation focus should be on limiting further damage and reducing inflammation.

Cryotherapy, systemic anti-inflammatories, therapeutic laser, compressive bandaging and external coaptation depending on the specific case scenario may be employed at this time. I do not typically apply external "sweats" or liniment as they could contribute to dermatitis and blistering, which could compromise wound healing or prevent regenerative injections through compromised skin. If an osmotic bandage is required for severe edema, the author prefers silver sulfadiazine topically. However, dry compression bandaging and aggressive cryotherapy twice daily is usually sufficient to resolve subcutaneous edema and swelling. Horses in this stage are typically stall rested and may receive short hand walks on firm footing several times daily as the injury allows.

Post-acute injury period (Repair stage day 7-30)

During this stage the rehabilitation focus should be on promoting fiber regeneration and repair. If possible, NSAIDs should be discontinued in this stage as long-term use could suppress fibroblast proliferation, however this has not been proven in vivo. Some horses may still require anti-inflammatories to continue to reduce swelling and keep the horse comfortable enough to combat support limb laminitis. Multimodal pain control should be used as clinically necessary.

In this stage, I rely heavily on regenerative medicine and utilize both platelet rich plasma (PRP) and mesenchymal stem cells (MSC's) during this stage. Healing lacerations anecdotally benefit significantly from several PRP injections into the granulation tissue at the laceration site. I have injected PRP into these every 5-7 days for up to 6 injections to provide growth factors and start to stimulate scaffold regeneration. I also believe PRP injections help to seal off the exposed tendon fibers sooner so as to prevent adhesion formation. PRP injections are often followed with 1-2 injections of MSCs as dictated by the clinical picture. Stem cell dose is still very much anecdotal. Typical core lesions 3-5 cm in length encompassing 30-50% of the cross sectional area are usually treated with 20 million MSCs in 1.5 ml of diluent. When extrapolated, this could correlate to an incredibly large dose of MSCs for severely damaged tendons. I have injected 120 million MSCs within one structure, however, I have also had good clinical results by using a lower overall dose of MSCs but adding more diluent to provide greater lesion coverage. Most severe lesions require 2 stem cell injections, but I have rarely had to do more than that unless there is a reinjury later in the horse's career.

Therapeutic laser could be continued during this stage as well as bandaging and supportive shoeing to the contralateral limb (i.e heart-bar with pour in) should be employed as needed. Exercises at this stage should be focused on maintaining core strength and mobility with "stall gymnastics" consisting of carrot stretches, sternal lifts, and butt tucks. Gentle weight shifting (wither or tail pulls) to the affected side or picking up the contralateral limb are good ways to promote early loading. During this stage, horses should be becoming more comfortable and able to hand walk 3-5 times daily for very short distances on firm footing. I prefer five 5-minute sessions daily, to one 25-minute session to prevent fatigue and overall continue low level stimulation for early loading.

Maturation stage (Remodeling- Day 30-180)

The maturation stage is arguably the hardest to manage from a rehabilitation standpoint. Progressive loading must be performed; however, it can be difficult to determine how aggressive to be. Superficial digital flexor tendons almost always have a period of contracture and shortening during this stage, regardless of the mechanism of injury. This can happen seemingly overnight. If fetlock hyperflexion or carpal flexion during standing is seen, aggressive physical rehabilitation must be started immediately to restore functional length. Pain levels should be carefully assessed to determine if pain shielding is promoting decreased weight bearing. Acetaminophen, gabapentin, pregabalin, or low dose NSAIDs have been useful to promote weight bearing in this stage. However, the most important aspect to battle contracture is therapeutic exercise. Underwater treadmill, walking in softer surfaces (i.e arena footing), stretching, and massage can all promote tendon lengthening. Additionally, rehabilitation modalities such as therapeutic ultrasound, transcutaneous electrical capacitive and resistive (TECAR) therapy, or other thermotherapy (heat) can also promote elasticity before exercise.

THERAPEUTIC SHOEING

Throughout the entire healing process, careful attention should be given to shoeing and trimming. We believe that wedges will increase the load of the SDFT and decrease the

load of the DDFT, so depending on which structure(s) are involved wedges may be indicated or contraindicated early on. If there is significant fetlock drop, fish-tail shoes can be helpful at capturing the vertical force and supporting the heel. Occasionally I have had to build foam or padding into a block between the heel of the shoe and the fetlock to prevent excessive drop, but careful attention is required to monitor for wounds or pressure sores on the palmar/plantar aspect of the fetlock and at the heel bulbs. Later in the healing process, especially if contracture occurs, a wedged or rocker shoe may need to be applied to promote loading of the SDFT.

OUTCOME MEASURES

Main outcome measures to assess in order to tailor the program should be repeated ultrasound exams (every 1-4 weeks), lameness evaluation weekly (first at a walk, but don't be too scared to have them trot a step or two), as well as limb circumference/swelling, or pain on palpation.

REFERENCES

References available upon request.

12001

THE ABNORMAL RADIOGRAPHIC FINDINGS IN YEARLING REPOSITORY IMAGES AND THEIR SIGNIFICANCE FOR RACING

AILMENTS OF THE YOUNG HORSE

Nathalie Cote, DMV, DVSc, ACVS

TAKE HOME MESSAGE:

Pre-sale radiographs can be very helpful in making the right choice when purchasing a yearling; however, one should avoid overinterpreting the radiographic findings.

INTRODUCTION:

Radiographs have become a standard practice in the pre-sale examination of Thoroughbred (TB) yearlings and are also commonly conducted before or after the sale of Standardbred (STB). The veterinary pre-sale repository radiographs can significantly impact both the seller and potential buyers. However, interpreting these radiographs can be challenging, as overinterpretation may lead to a canceled sale. It is essential to comprehend the implications of any abnormal findings and their potential impact on a horse's racing abilities. Additionally, it is important to recognize which lesions can be surgically corrected with a good prognosis and which ones do not offer a favorable outcome.

The risks associated with orthopaedic lesions observed in thoroughbred yearling repository radiographs on future racing performance have been the focus of numerous studies.[1-8] For example, moderate to extreme palmar supracondylar lysis of the third metacarpal bone (MC3), intercarpal joint disease, enthesophytes on proximal sesamoid bones, and proximal dorsal fragmentation of the first phalanx (P1) were less likely to start racing at 2–3 years of age than those without these abnormalities.[1] Another study on 2,401 yearlings suggested that a bone defect greater than 10 mm in length at the sagittal ridge of a metatarsus or sesamoid remodeling in forelimbs could affect future racing performance.[9] It has been also reported that yearlings with enlarged vascular canals in their sesamoids could start fewer races and earn less prize money than horses with normal vascular canals.[10] However, a report on 348 yearlings presented no significant association between radiographic abnormalities and future performance.[7] Miyakoshi concluded although radiographic abnormalities at yearling sales can be associated with failure to start racing at 2–3 years of age, these radiographically detected abnormalities might not necessarily cause that failure.[4]

The purpose of this presentation is to review the most common abnormalities observed in repository radiographs, discuss some grading systems, and explore their significance and implications for a horse's racing career, as well as to correlate some of these lesions with surgical findings.

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The presentation aims to review common abnormalities in repository radiographs, discuss various grading systems, explore their significance and implications for a horse's racing career, and correlate these lesions with surgical findings.

Revue of topic/information:

Veterinarians should anticipate finding radiographic changes in Thoroughbreds and Standardbreds examined before or after yearling sales. The most common disorders encountered are listed below by anatomical region. Their incidences and impact on racing will be reviewed.

FETLOCK:

Proximal Sesamoid bone:

Sesamoiditis:

Radiological findings in the proximal sesamoid bones (sesamoids) are a persistent source of controversy at Thoroughbred sales, primarily due to inconsistent classification and conflicting assessments of their potential clinical significance. A recent classification system introduced by Peat standardizes the radiological findings related to the appearance of sesamoid vascular channels, changes in the abaxial contour, and the presence of sesamoid fragments apical and abaxial. [11] Grade 3 vascular channels, the presence of abaxial new bone, and apical or abaxial fragments in the forelimb sesamoids have been associated with a decreased likelihood of racing. [11] Furthermore, the existence of concurrent findings in the proximal sesamoid bones and adjacent suspensory ligament branches in yearling and two-year-old Thoroughbred sales horses has been well established.[12]

Radiological appearance of the proximal sesamoid bone	Definition
Grade 0 vascular channels and no abaxial contour change	The sesamoid contains any number of vascular channels that are <2 mm wide for at least two-thirds of their visible length. The sesamoid also has no abaxial new bone or abaxial margin concavity.
Grade 1 vascular channels	One vascular channel that is ≥2 mm wide for more than one third of its visible length. Any number of vascular channels <2 mm wide.
Grade 2 vascular channels	Two vascular channels that are ≥2 mm wide for more than one third of their visible length[a]. Any number of vascular channels <2 mm wide.
Grade 3 vascular channels	Three or more vascular channels that are ≥2 mm wide for more than one third of their visible length[b]. Any number of vascular channels <2 mm wide.

TABLE 1. Grading system for the radiological appearance of vascular channels and other abaxial border changes in the proximal sesamoid bones. (Peat 2024)

Radiological appearance of the proximal sesamoid bone	Definition
Abaxial new bone only	New bone formation on the abaxial margin of the sesamoid, in the absence of any enlarged vascular channels.
Abaxial margin concavity only	A distinct radiolucent concave defect on the abaxial margin of the sesamoid, in the absence of any enlarged vascular channels.

- *a Grade 2 can include one very enlarged vascular channel with a width equivalent to the combined width of two enlarged vascular channels, that is, ≥ 4 mm total width.*
- *b Grade 3 can include one or more very enlarged vascular channels with a total width equivalent to the combined width of three enlarged vascular channels, that is, ≥ 6 mm total enlarged channel width.*

Other Sesamoid abnormalities:

Observations included elongation of the proximal sesamoid bone, fractures of the proximal sesamoid bones classified into apical, abaxial, basal, midbody, and comminuted types, as well as the presence of enthesophytes and osteophytes on the proximal sesamoid bones.[2,9]

FETLOCK FRAGMENTATION:

The reported prevalence of OCD in young Standardbreds in fetlocks ranges 14–29%. In a review performed on Thoroughbred yearlings, hind fetlocks had higher incidence of first phalanx fragmentation then the fore fetlock.[2] Lucencies, fragments or loose bodies were detected at the dorsal aspect of the distal third metacarpus in 2.8% and, at the same location on the third metatarsus, 3.2%.[2]

Most common disorders found on fetlock's radiographs includes:

1. Proximal dorsal P1 fragment
2. Proximal palmar/plantar P1 (articular and non-articular)
3. Subchondral cyst distal metacarpal/tarsal 3 or proximal P1
4. Change on the dorsal aspect distal metacarpal/tarsal 3 (notch, lucency, fragment/loose body
5. Change on the distal sagittal ridge of metacarpal/tarsal 3 (flat, lucency, fragment)
6. Change distal palmar/plantar metacarpal/tarsal 3 (flattening, lucency)
7. Palmar/plantar supracondylar lysis metacarpal/tarsal 3.

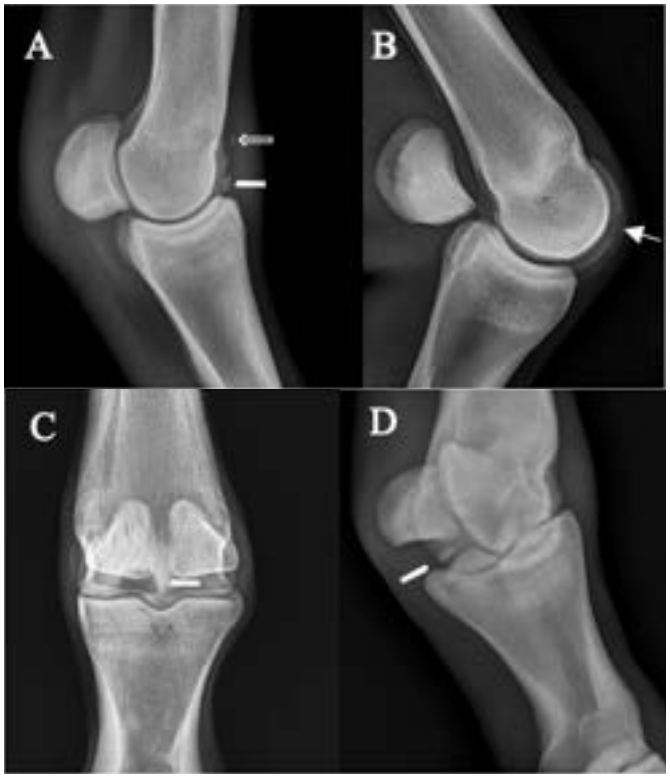

Fig 1: Proximal dorsal P1 fragment (white arrow), fragment on the dorsal aspect distal metacarpal (open white arrow) (A), Lucency in the mid sagittal ridge (MSR) (white arrow) (B), subchondral cyst (white arrow) (C), fragmentation of the proximal palmar aspect P1 (white arrow) (D).

PASTERN:

Most common radiographic abnormalities of the pastern reported are:

1. Osteoarthritis characterized by osseous changes at the articular margins of the joint with osteophytes and enthesophytes.
2. Remodelling of the dorsal aspect of the middle phalanx (irregular bone formation).
3. Osseous fragments located in the proximal interphalangeal joint (PIPJ).
4. Well-demarcated lucency (cyst) in the proximal aspect of the middle phalanx (P2) or the distal aspect of the proximal phalanx (P1).

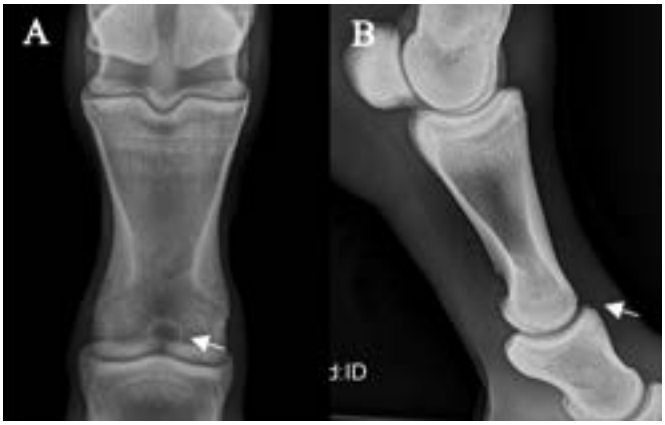

Fig 2: Subchondral lucency (Cyst) distal P1(A), Osseous fragments located in the proximal interphalangeal joint (PIPJ) (white arrow) (B).

CARPI

Criteria which had been considered in the radiographic evaluation of the carpi includes:

1. The presence of osseous cyst-like lesions (OCLLs). These lesions are characterized as focal, radiolucent areas located particularly in the ulnar carpal bone.
2. Osseus fragment originating from the joint margins of the third, intermediate, radial, ulnar, or fourth carpal bones.
3. Remodeling of the carpal bones and distal radius showed irregular bone formation (osteophytes), enthesophytes as well as sclerosis or lysis of the bone contours.
4. Cyst lesion in the distal radius or any carpal bones
5. Fractures (Complete and incomplete) of the accessory carpal bone
6. Miscellaneous: Osseus fragment off the proximal aspect of the MC2 or MC4,

The presence of circular lucencies seen in the palmar was the most common abnormal finding in one study. [2]

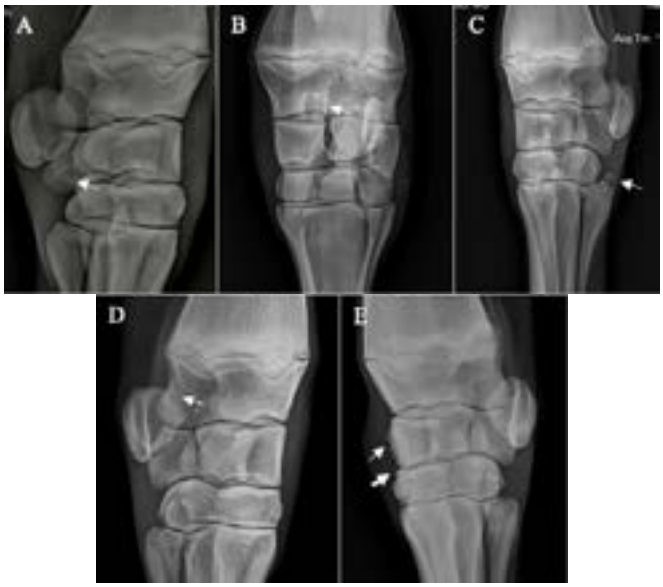

Fig 3: Osseous cyst-like lesions (OCLLs) in ulnar carpal bone (A), Subchondral cyst in distal radius (B), Osseus fragment off metacarpal 4 (C), Osseus fragment off the accessory carpal bone (D), Enthesophytes on radial carpal bone (thin arrow) and Osseus fragment from RCB (thick arrow) (E).

TARSUS:

The reported prevalence of tarsal osteochondritis dissecans (OCD) in clinically normal young Standardbreds ranges from 10 to 26%. 13,14 In a survey of 582 Thoroughbred sale yearlings, lesions of OCD were found in the distal intermediate ridge of the tibia or the distal lateral trochlear ridge of the talus in 19 out of 710 (3%) tarsi.15

Lesions reported:

1. OCD lesions: Distal intermediate ridge (DIRT), Medial malleolus (MM) (lucencies or fragmentation), lateral trochlea of the talus (LTT) and medial trochlea of the talus (MTT) (flattening, lucencies or fragmentation). These lesions can appear as a concavity (+/- surgery) or with fragmentation.

2. Osseous opacity at the distal medial trochlear ridge (attached or detached).

3. The presence of osteophytes or enthesophytes at the distal intertarsal or tarsometatarsal joint margins.

4. Subchondral lucency in the distal intertarsal or tarsometatarsal joint margins.

5. Wedging, convexity or collapse of the tarsal bones (ankylosis, narrowing, and compression). Wedging involves either the central and third tarsal bones tarsal bones extending beyond the dorsal border of the third metatarsal bone, and convexity refers to the dorsal border curving plantar.

6. Miscellaneous: Fragmentation of the dorsal aspect of the distal talus (FDDT), [16] slab fracture central or third tarsal bone bi-articular or incomplete and others.

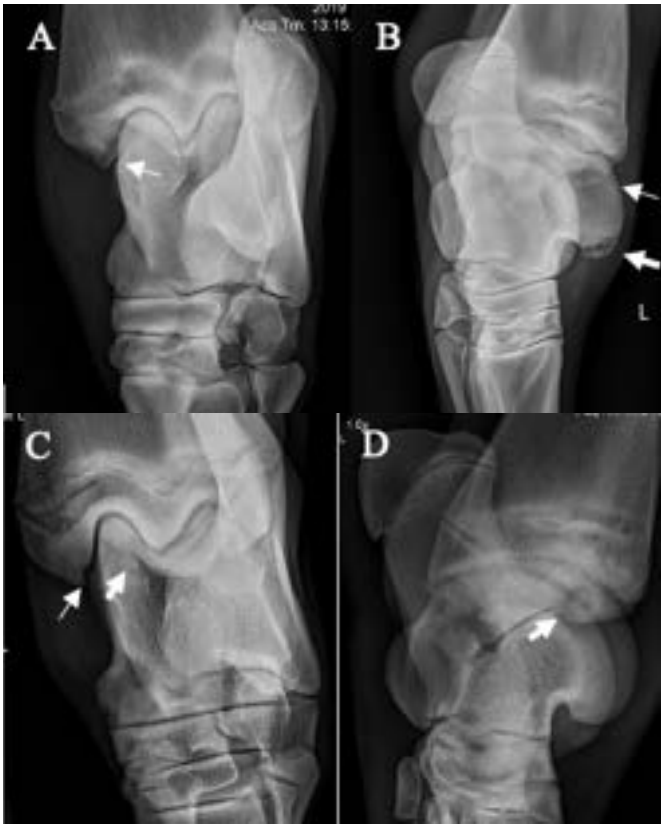

Fig 4: Osteochondrosis dissecans (OCD) of medial malleolus (MM) (A), OCD of distal intermediate ridge (DIRT) (thin arrow) and lateral trochlea of the talus (thick arrow) (B), OCD of the medial malleolus (thin arrow) and abaxial edge of DIRT (thick arrow) (C) OCD of abaxial edge of DIRT on DPMLO (D).

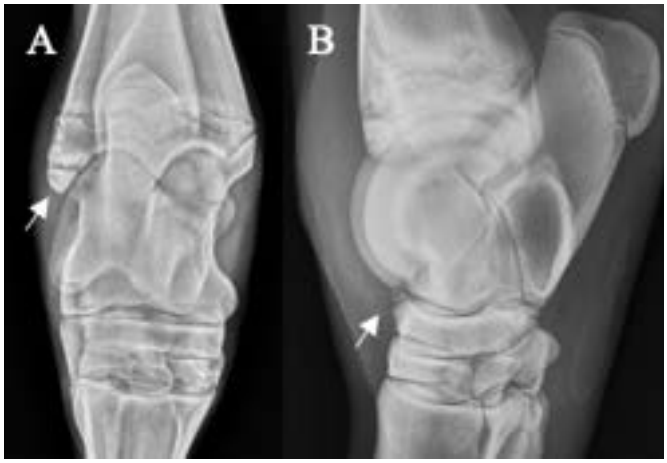

Fig 5: Fracture of the lateral malleolus (A), Fragmentation of the dorsal aspect of the distal talus (FDDT) (B)

STIFLE:

Developmental osteochondrosis disorders in the stifle joint are well described and classified. The incidence at these lesions can vary. The lateral trochlear ridge (LTR) is the most common site for OC in the equine stifle.[17] Studies assessing the prevalence of osteochondritis dissecans (OCD) in Thoroughbred yearlings from central Kentucky have reported a prevalence of 5.1% for LTR OCD and 10.9% for stifle OCD. [6] Subchondral lucencies (SCLs) observed in the distal aspect of the medial femoral condyle (MFC) of young Thoroughbred horses can be controversial on presale radiographs, as there is limited scientific evidence concerning their risk of progression and their impact on future racing performance. [18] One study indicated that neither the length nor the depth of these lesions affected racing performance.[19]

Lesions found in the stifle and their classification:

1. Lateral and medial trochlear ridges of the femur: Flattening, subchondral lucent defects with or without fragmentation (OCD)

2. Lucency or fragmentation in the trochlear groove

3. Lucency of the patella and fragmentation of the distal patella

4. Presence of cysts in the medial, lateral condyle of the femur or tibial plateau (SCL)

5. Miscellaneous: Calcinosis circumscripta, enthesophyte, osteophyte

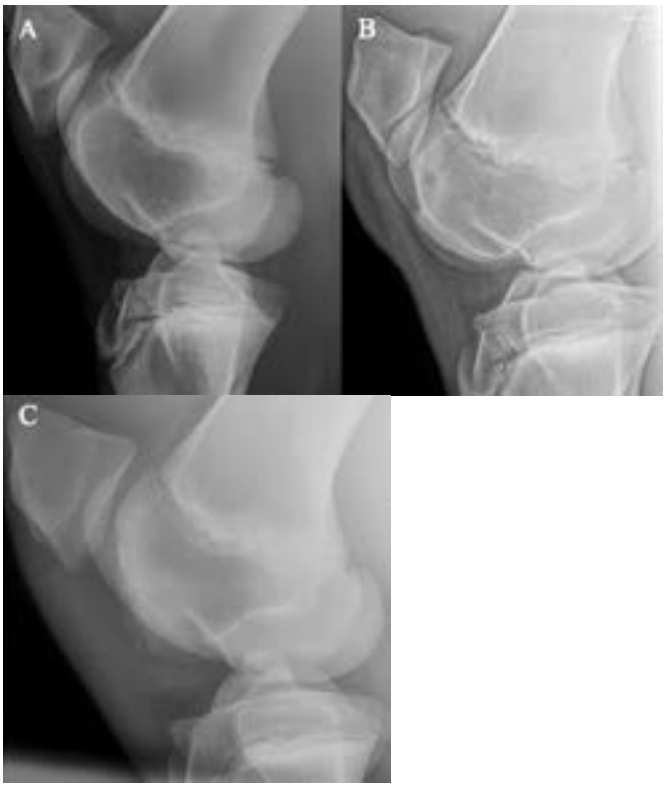

Fig 6: Lateral and medial trochlear ridges of the femur OCD grading system based on length. Grade 1: <20 mm (A), Grade 2: 20–40 mm (B), Grade 3: >40 mm

Recent grading system for subchondral cyst of the lateral of medial femoral condyle used to evaluate the outcome of yearling and 2-year-old Thoroughbred:

Grade 0 = no subchondral lucency. Flattening of the distal articular contour of the condyle is allowed.

Grade 1 = SCL is a mild, shallow, crescent-shaped lucency with a proximodistal depth ≤3 mm and with an axial-abaxial width greater than its depth.

Grade 2 = SCL is a moderate, dome-shaped lucency that extends through the subchondral bone, with a depth approaching that of its width.

Grade 3 = SCL is a large, spherical or ovoid cystic lesion that communicates with the central articular surface of the condyle.

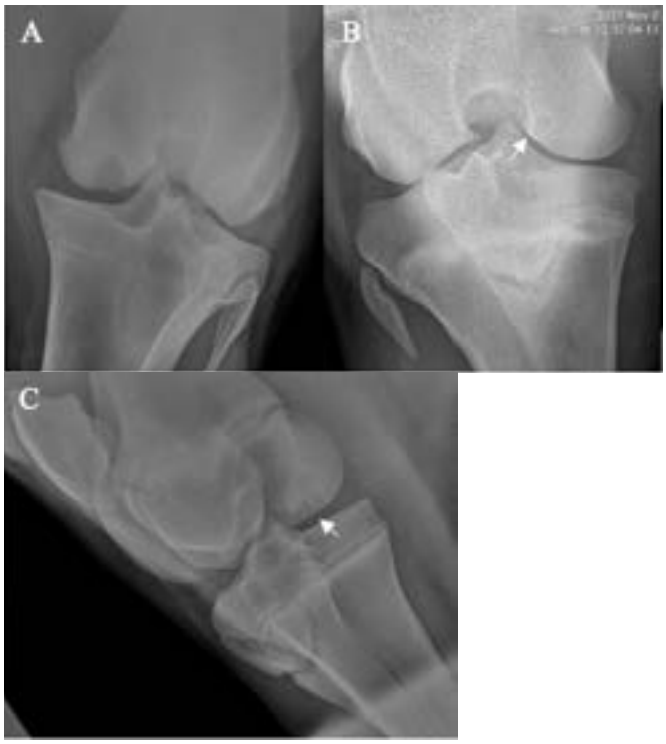

Fig 7: Grade 3 subchondral lysis (SCL) in the medial condyle of the femur (A), Atypical medial SCL (white arrow) (B and C)

CONCLUSION:

Further research is needed to explore the impact of all radiographic lesions observed during repository radiographic examinations on racing abilities. It is encouraging that recent studies have shown that osteochondritis dissecans (OCD) of the lateral trochlear ridge of the femur and subchondral lucencies of the medial femoral condyle have a better prognosis than previously thought.[18,19] Additionally, there is an improved understanding of how sesamoid lesions are associated with the prevalence, progression, and effects on racing performance.[11] These research can help us provide appropriate guidance to our clients.

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12002

THE OUTCOME OF OSTEOMYELITIS LESION WITH JOINT INVOLVEMENT IN OLDER FOALS – CASE SERIES

AILMENTS OF THE YOUNG HORSE

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TAKE HOME MESSAGE

Haematogenous septic arthritis, physitis, and osteomyelitis (SAPO) in older foals (those over 1 month old) do not necessarily have a poor prognosis.

INTRODUCTION

Haematogenous septic arthritis, physitis, and osteomyelitis (SAPO) is an uncommon condition in foals that can lead to severe complications or even be life-threatening. Research has indicated that the short-term survival rate of foals with SAPO ranges from 42% to 84%.[1-4]

SAPO can affect foals up to 7 months old. A retrospective study revealed that among 108 foals diagnosed with osteomyelitis, 19 (17.6%) were more than 21 days old at presentation. [3] Foals younger than 30 days and those with multiple bones or joints affected had a much lower likelihood of being discharged from the hospital.[3] Recent research has also suggested that SAPO may contribute to osteochondrosis, potentially increasing the risk for joint diseases in older ages.[5]

The clinical signs observed in SAPO cases can range from mild to severe. Common symptoms include joint swelling, lameness, or gait abnormalities, often accompanied by some degree of systemic disease. SAPO is classified into four types: S-type, E-type, P-type, and T-type. These classifications are based on the origin of the infection, which may involve the synovial membrane, epiphysis, physis, or the metaphysis of cuboidal bones in the tarsus or carpus.[6,7] Diagnostic modalities typically includes radiographic imaging and the analysis of synovial fluid. However, radiography may fail to identify E- and P-type infections in the early stages of the disease, as a loss of 30-50% of bone mass is required for visibility.[8] Repeating radiographic series over time may be necessary. When available, CT or high-field MRI should be considered as they represent the gold standard for diagnosing challenging SAPO cases.[8]

Transphyseal vessels trap bacteria from the bloodstream in the synovial membrane and epiphysis, leading to more S-type and E-type infections in foals up to 7 to 10 days old. After these vessels close, P-type infections become more common in older foals.[6] The main bacteria isolated from SAPO cases include E. coli, Actinobacillus species, Klebsiella species, Staphylococcus species, Streptococcus species, and Rhodococcus equi.[2,9,10,11] In one study, 51.9% of the isolate were gram-positive (Streptococcus spp, Staphylococcus spp and Listeria spp.), while gram-negative organisms accounted for 48.1%, with Escherichia coli and Salmonella spp being the most common pathogens.[3] Studies suggest that the type of bacterial infection varies with age. Gram-negative infections are more common in newborns, while gram-positive infections usually occur later.[2,11] However, not all studies agree on this connection, so more research is needed to fully understand these patterns. Osteomyelitis and septic arthritis are included among the extrapulmonary disorders (EPD) associated with Rhodococcus equi infection in older foals. In one retrospective study of EPD in foals, 5 out of 150 cases had osteomyelitis, and 15 presented with septic synovitis. Rhodococcus equi has been the culprit in a few cases seen in this case series.[12]

Treatment for septic arthritis and osteomyelitis generally includes joint lavage (via arthroscopy, needles, or arthrotomy), bone debridement, regional perfusion, intralesional antimicrobial therapy, intraarticular injections, and systemic antibiotics. Stem cell therapies, which have shown antimicrobial properties in vivo, are also being investigated for their potential benefits.[13,14]

There is a consensus that foals with septic synovial structures and osteomyelitis often have a poor prognosis. Due to the high cost of treatment coupled with low success rates, euthanasia may sometimes be considered in the field. The aim of this review is to examine several cases observed in Ontario to assess how treatments, bacterial types, and lesions influenced outcomes.

REVUE OF TOPIC

Case report:

A 1-month-old Thoroughbred colt was presented to the OVC for evaluation and treatment of a 3-day-old history of a right hind lameness, fever, and diarrhea. The colt was bright alert, and responsive on admission. His vital parameters were elevated – HR 96 bpm, RR 40 bpm, T 38.7 °C. Auscultation of the lungs revealed harsher lung sounds in all fields. The colt presented odorous diarrhea and moderate right hind limb lameness at the walk (Grade 4/5 AAEP scale) with moderate effusion within the lateral femorotibial joint and marked swelling of the medial aspect of the stifle region. Radiographs of the right stifle taken by the referring veterinarian failed to reveal anything significant. (Fig 1) Ultrasound findings revealed signs of a moderate pneumonia bilaterally and thickened synovium and fibrin with moderately increased effusion within the right femorotibial joint. (Fig 2)

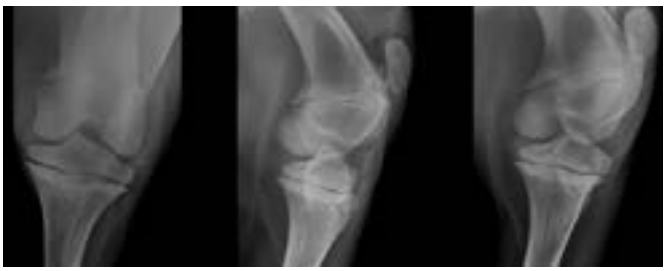

Fig 1: Radiographs of the right stifle at presentation.

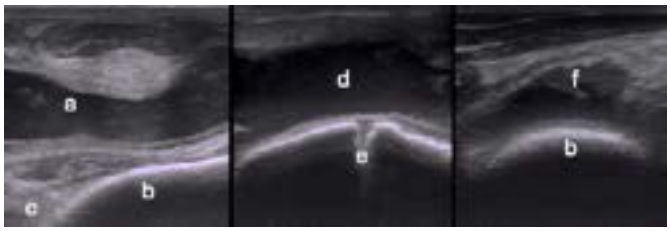

Fig 2: Ultrasound of the lateral aspect of the lateral femorotibial joint demonstrating effusion and fibrin. Joint fluid with increase cellularity (a), lateral femoral condyle (b), femorotibial joint space (c), joint effusion (d), tibial physis (e), Fibrin (f).

A cytology analysis of the synovial fluid revealed an elevated white blood cell (WBC) count of 181,800 cells/µL, with over 96% of these being neutrophils. Additionally, total protein levels were measured at 52 g/L. Cultures from the joint identified Streptococcus criceti, and the equine neonatal diarrhea panel tested positive for Rhodococcus equi. To treat the septic joint, the following procedures were implemented: systemic antibiotics (penicillin, gentamicin, and chloramphenicol), anti-inflammatory therapy with flunixin meglumine, arthroscopic lavage and debridement, and needle joint lavage on Days 3, 5, 7, and 11. Intra-articular antibiotic infiltration with amikacin was performed on Days 1, 2, 3, 5, 7, 10, 11, 13, and 15. Radiographs taken four days after the initial set of images revealed a circular lytic lesion on the lateral aspect of the tibial epiphysis. This lesion progressed during the foal's hospitalization. (Fig 4) Curettage was not attempted due to the risk of collapsing the articular surface. Regular joint fluid analysis of the lateral femorotibial and femoropatellar joint showed a steady decline in the WBC count and protein level. The foal was discharged 16 days after presentation and is presently lameness free. Improvement of the lesion was noted on radiographs taken 2 weeks after leaving the hospital. (Fig 5)

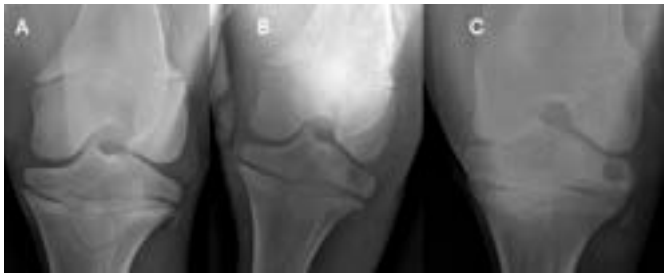

Fig 4: Radiographs of the right stifle. Progression of the lytic lesion in the epiphysis of the tibia on day 1 (A), day 4 (B) and day 12 (C).

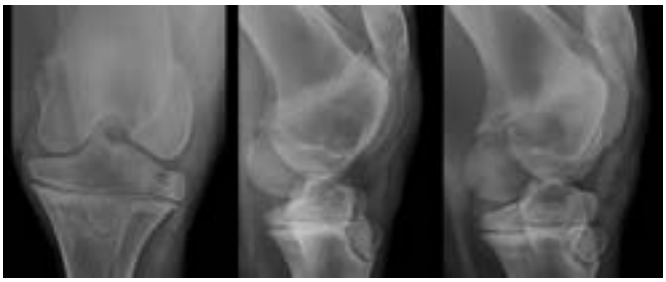

Fig 5: Radiographs of the right stifle 2 weeks after release from the hospital.

CLINICAL COMMENTARY:

In recent years, a series of cases similar to the one currently described have been treated at the OVC and King Animal Hospital, as well as in private practices for milder cases. We reviewed their varying presentations, treatments, and outcomes. Clinical observations included swollen synovial structures, focal leg edema, acute or subacute lameness, and changes in gait or movement. In some cases, involving the proximal aspects of the legs, joint distension and swelling were not immediately obvious, making diagnosis more challenging. Additionally, some foals presented with concurrent septic conditions, such as pneumonia or diarrhea. Treatment of these cases included systemic antibiotics, joint lavage (via arthroscopy, needles, or arthrotomy), bone debridement, regional perfusion, intralesional antimicrobial therapy and intraarticular injections. The outcome of these foals was more successful than previously thought.

CONCLUSION

The treatment of haematogenous septic arthritis, physitis, and osteomyelitis (SAPO) in older foal may be successful.

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12003

REVIEW OF VARIOUS PATHOLOGY OF THE EQUINE UPPER RESPIRATORY TRACT & THEIR SIGNIFICANCE AND PROGNOSIS

AILMENTS OF THE YOUNG HORSE

Nathalie Cote, DMV, DVSc, ACVS

TAKE HOME MESSAGE

The anatomy of the equine upper respiratory tract is complex, and issues in any of its components can lead to secondary problems. A precise evaluation of the issue enhances the likelihood of a successful outcome.

INTRODUCTION:

Laryngeal and pharyngeal dysfunction in horses falls under the category of upper respiratory tract (URT) disorders. These disorders can lead to dynamic upper airway obstruction (DUAO), which significantly affects a horse's performance. [1, 2] The function of the upper airway is often assessed through endoscopy, both in horses with disabilities and in healthy ones as part of pre-purchase or post-purchase examinations. [3-6] Moreover, dynamic endoscopic examinations offer valuable insights into conditions that may not be visible when the horse is at rest. [7] Identifying any abnormalities in the URT and understanding the pathophysiology of these disorders is crucial for effectively guiding and advising clients. This presentation aims to review common pathologies of the upper respiratory tract in horses, focusing on their etiology, consequences, prognosis, and treatment options.

REVIEW OF TOPIC/ INFORMATION:

The pharynx and larynx rely on muscle activity to maintain patency and are often prone to dynamic airway collapse. This can lead to impaired respiratory function in the lower airways and hinder gas exchange, ultimately resulting in decreased athletic performance, particularly in racehorses that are working at high speeds. [8,9] Dynamic upper airway obstruction (DUAO) is thought to occur when soft tissue structures cannot withstand inspiratory pressure. This condition may arise due to factors such as neuromuscular dysfunction, fatigue, immaturity, or anatomical changes in the structures responsible for controlling the nasopharynx. [10] Types of dynamic upper airway obstruction observed in horses include, but are not limited to, dorsal displacement of the soft palate (DDSP), medial deviation of the aryepiglottic folds (MDAF), nasopharyngeal collapse (NPC), dynamic laryngeal collapse (DLC), epiglottic entrapment (EE), and epiglottic retroversion (ER).

DYNAMIC UPPER AIRWAY OBSTRUCTION (DUAO) INVOLVING THE LARYNX:

The larynx is assessed for: 1) arytenoid asymmetry (laryngeal function), 2) epiglottic positioning and structure and subepiglottic disorders, 3) vocal fold collapse (VFC) and/or medial deviation of the arytenoid folds (MDAF) and 4) arytenoid chondritis and dysplasia.

1. Laryngeal function or arytenoid asymmetry

The laryngeal function is characterized by the ability of obtaining and maintaining a complete abduction of both arytenoids. [11] Lack of symmetry, delayed, incomplete or absent abduction of the arytenoid cartilage at rest or exercise is highly suggestive of a recurrent laryngeal neuropathy (RLN) which results in an impaired laryngeal function. RLN is characterized by a progressive neurogenic atrophy of the laryngeal musculature resulting in loss of the arytenoid function. [12] The cricoarytenoideus dorsalis muscle's atrophy is responsible for the loss of arytenoid abduction and clinical signs observed. [13]

GRADE	DESCRIPTION	SUB-GRADE
I	All arytenoid cartilage movements are synchronous and symmetrical and full arytenoid cartilage abduction can be achieved and maintained.	
II	Arytenoid cartilage movements are asynchronous and/or larynx at times but full arytenoid cartilage abduction can be achieved and maintained.	II.1. Transient synchrony, flutter or delayed movements are seen II.2. There is asymmetry of the rima glottidis much of the time due to reduced mobility of the affected arytenoid and vocal fold but there are occasions, typically after swallowing or nasal occlusion when full symmetrical abduction is achieved and maintained
III	Arytenoid cartilage movements are asynchronous and/or asymmetric. Full arytenoid cartilage abduction cannot be achieved and maintained.	III.1. There is asymmetry of the rima glottidis much of the time due to reduced mobility of the arytenoid and vocal fold but there are occasions, typically after swallowing or nasal occlusion when full symmetrical abduction is achieved but not maintained. III.2. Obvious arytenoid abductor deficit and arytenoid asymmetry. Full abduction is never achieved. III.3. Marked but not total arytenoid abductor deficit and asymmetry with little arytenoid movement. Full abduction is never achieved
IV	Complete immobility of the arytenoid cartilage and vocal fold.	

Table 1: Havemeyer grading system for endoscopic evaluation of the laryngeal function in standing unsedated horses (Dixon 2003)

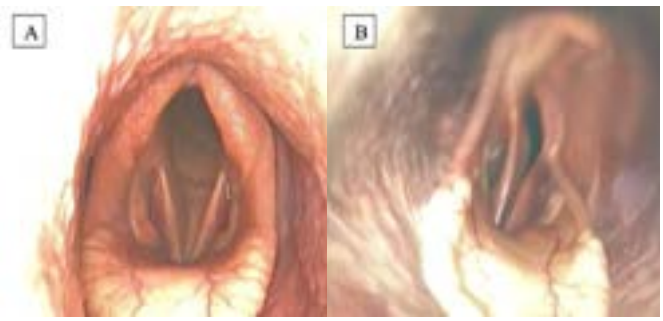

Fig 1: Endoscopy at rest (A) and at exercise (B) of recurrent laryngeal neuropathy

2. Epiglottis anomalies

It is essential to assess the anatomical position, size, shape, and structure of the epiglottis during endoscopy. Abnormal epiglottic positioning includes conditions such as epiglottic retroversion, [15] epiglottic deviation, and entrapment of the epiglottis beneath the subepiglottic mucosa. [16] The latter is often associated with ulceration of the apex of the epiglottis or ulceration along the border of the soft palate. [17] A grading system has been developed to categorize the epiglottic structure using a 4-point scale, as detailed in Table 2. [18] A thorough examination of the ventral aspect of the epiglottis while the horse is swallowing is crucial for detecting subepiglottic cysts and any abnormal ulceration or adhesions. [19]

GRADE	DESCRIPTION
I	Slightly flaccid epiglottis and good length and texture but was slightly thinner than normal without serrated edges.
II	Mild flaccidity, adequate length, thinner than normal, curled edges, and no dorsal vasculature.
III	Moderately flaccid, very thin, and bent easily.
IV	Severely flaccid, extremely thin, was markedly short, and bent easily

Table 2: Epiglottic structure grading system (Pierce & Embertson, 2001)

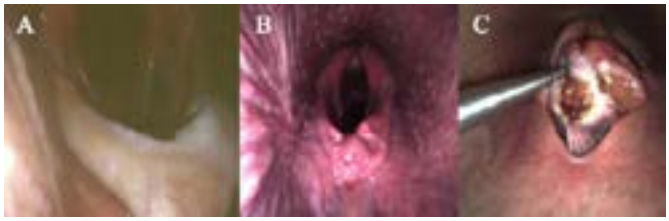

Fig 2: Endoscopy of an epiglottic entrapment (A), epiglottic malformation (B), and a sub-epiglottic ulceration (C).

DYNAMIC LARYNGEAL COLLAPSE

These conditions are primarily observed in racehorses and can be caused by several factors, including collapse of the arytenoid cartilage, [11, 20, 21] vocal fold collapse, [11,21] axial deviations of the aryepiglottic folds, [21,22] and collapse of the epiglottic margin. [21] Recently, ventrorostral displacement of the dorsal laryngeal mucosa was recently described as another type of dynamic laryngeal collapse.[23]

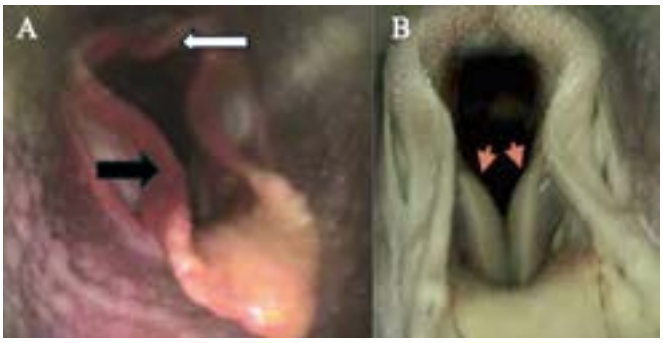

Fig 3: Axial deviation of the aryepiglottic fold (black arrow) and collapse of apex of corniculate process of arytenoid (white arrow) (A) and vocal fold (pink arrow) (B) at exercise.

3. Arytenoid cartilage abnormalities

Endoscopically, chondropathies are characterized by an abnormal shape of the arytenoid cartilage, resulting in impaired abduction. [24] Dysplasia was defined as a mechanical arytenoid dysfunction caused by bilateral incomplete arytenoid abduction[21] or corniculate processes incongruity.

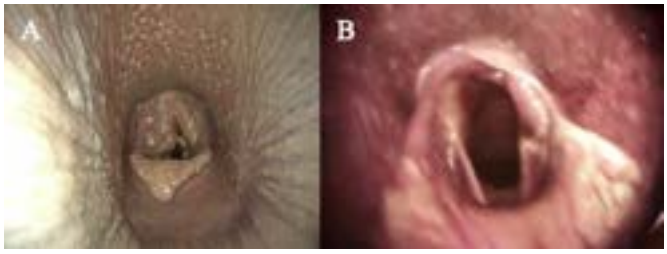

Fig 4: Chondritis of the right arytenoid (A), Collapse of the palato pharyngeal arch (B)

DYNAMIC UPPER AIRWAY OBSTRUCTION (DUAO) INVOLVING THE PHARYNX:

Common disorders find at the level of the pharynx includes palatal instability, dorsal displacement of the soft palate, pharyngeal collapse and pharyngeal lymphoid hyperplasia, and mucopurulent secretions at the rima glottis.

1. Palatal dysfunction

Palatal dysfunction included palatal instability (PI) and dorsal displacement of the soft palate (DDSP). Palatal instability is described as a dorsoventral billowing

movement of the caudal soft palate. 11 This condition may evolve to a DDSP. [25, 26] Dorsal displacement of the soft palate is categorized based on the durability of the occurrence, intermittent (iDDSP) or persistent (pDDSP), and based on the condition of occurrence, at rest and/or at exercise. [5, 27, 28] The center of the free border of the soft palate should also be assessed for ulceration, which may be indicative of repetitive DDSP in racehorses. [20,2]

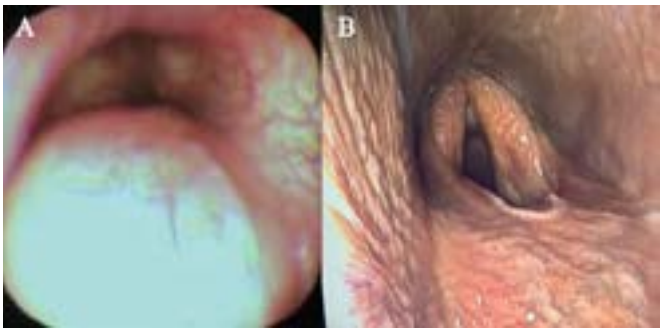

Fig 5: Billowing of the soft palate (A) and Dorsal displacement of the soft palate(B)

2. Pharyngeal Collapse

Pharyngeal collapse is characterized by the axial displacement of at least one wall of the pharynx. [21] This condition is diagnosed using dynamic endoscopy and has been observed in both Thoroughbred (TB) and Standardbred (SB) racehorses [29, 5 21, 26]

Fig 6: Collapse of the palatopharyngeal arch

3. Pharyngeal Hyperplasia

Pharyngeal lymphoid hyperplasia (PLH) is a chronic pharyngitis involving the lymphoid system. The condition is graded using a 4-point scale based on the size, aspect, and distribution of the lymphoid follicles in the pharynx. PLH has been wildly investigated in young racehorses and is a parameter often included in the endoscopic examination criteria. [5]

GRADE	DESCRIPTION
I	Few small white lymphoid follicles scattered over the dorsal pharyngeal wall. These appear to be inactive and shrunken.
II	Many small white lymphoid follicles lying close together on the dorsal and lateral pharyngeal walls. Laterally they extend to or slightly below the pharyngeal openings to the guttural pouches. Scattered among the white follicles, may be a few pink, edematous, active appearing follicles.
III	Pink and white lymphoid follicles which lie very close together, covering the entire dorsal and lateral pharyngeal walls. They extend below the openings to the guttural pouches and often involve the dorsal surface of the soft palate. These follicles essentially involve all the visible surface of the pharyngeal mucous membrane.
IV	Follicles appear large and edematous and cover an extensive area as in Grade III. They may extend into the guttural pouches and are frequently accompanied by numerous polyps arising from the mucosa of the pharyngeal diverticulum or the dorsal and lateral pharyngeal walls.

Table 5: Grading system of the pharyngeal lymphoid hyperplasia in horses (Raker & Boles, 1978)

CONCLUSION:

There is still much that remains unknown about upper respiratory disorders in horses. Treating these conditions can often be frustrating, and the results may not always be satisfactory. More research is needed to fully understand the interactions among the various structures involved and to clarify the pathophysiology of these disorders so that we can work towards preventing them.

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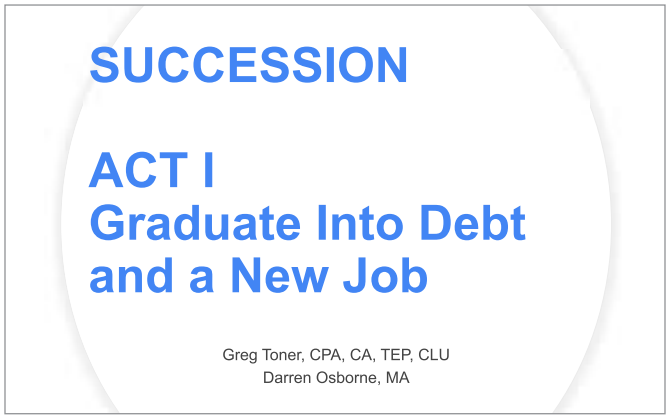
13001

GRADUATE INTO DEBT AND A NEW JOB

SUCCESION

Greg Toner, CPA, CA, TEP, CLU
Darren Osborne, MA



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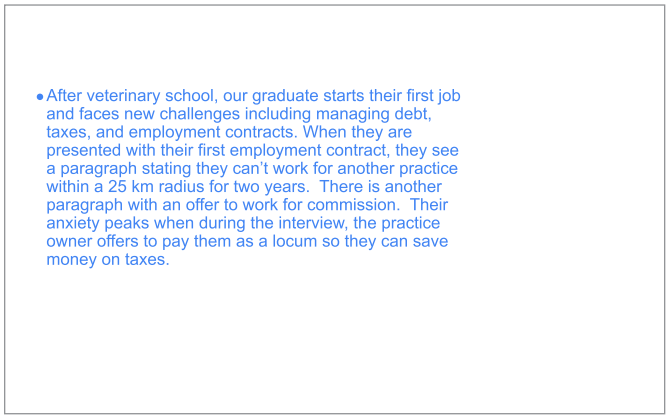

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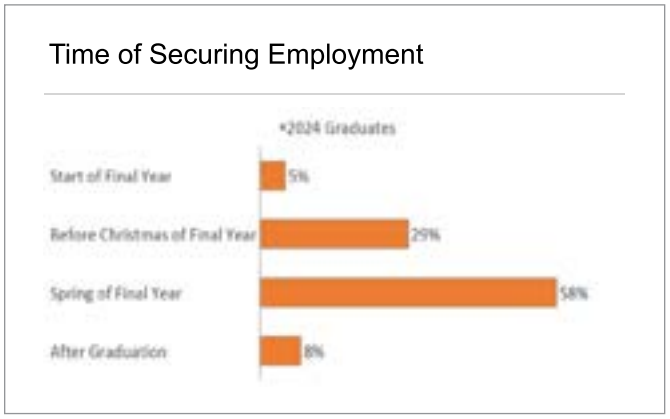

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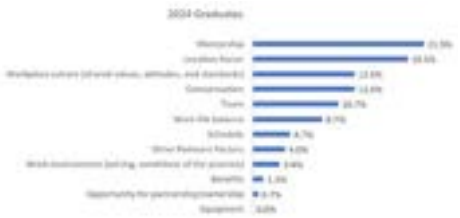
Factors in Determining Job Selection



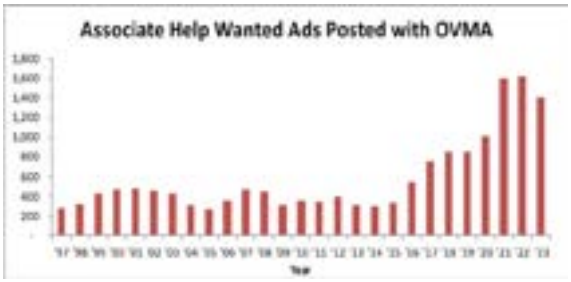
5

What is Driving Salaries for New Graduates?

Median Annual Compensation			
	2023	2024	Change
New Graduate	$110,000	$115,000	4.5%
All Associates	$127,000	$130,000	2%

6

What is Driving Salaries for New Graduates?



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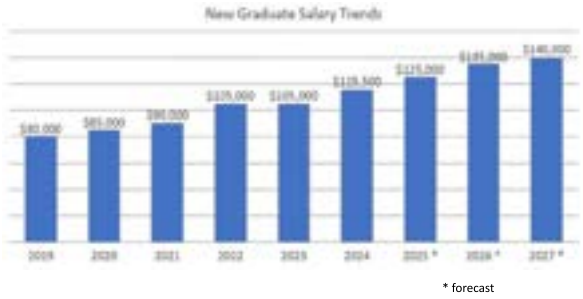
New Graduate Salary Forecast



8

Proportion Graduating with Student Debt

9

Balance of Debt

Balance of Debt at Graduation	Mean Debt	Median Debt	Number of Respondents
2024 Graduates – Domestic Students	$58,226	$50,000	19

Current Balance of Debt	Mean Debt	Median Debt	Number of Respondents
2024 Graduates – Domestic Students	$55,255	$45,000	19

10

Monthly Expenditures

Expense Category	2024 Graduates
Rent/Mortgage	$1,168
Student Loan	$1,067
Car Loan/Lease Payment	$264
Car Insurance	$188
Other Personal Loans	$581
Disability Insurance	$67
All Other Insurance	$30
Transportation	$129
Utilities	$85
Oil/Gas	$107
Home Maintenance	$31
Food	$360
Household Items	$14
Pets	$238
Cable/TV Costs	$9
Internet	$34
Phone/Cell Phone	$43
Entertainment	$48
Other	$64
Total Monthly	$4,527

11

Ability to Pay Debt

- Monthly Income $6,200
- Monthly Expenses $4,500 including $1,000 student debt payment
- Surplus $1,700
- Student Debt $55,000
- Term 6 years
- Increase Debt Payment $2,000 $1,000 more
- Pay student debt off 3 years vs 6 years

12

Just Sign Here

Contract Basics

13

Topics

Employee vs. Independent Contractor
What Makes a Good Agreement?
Remuneration – What Should You Earn?
Benefits
Employee Standards Act and Veterinarians
The “Breach” Clause
Non-Competition/Solicitation Agreements
Importance of Legal Advice

14

Employee Vs. Independent Contractor

They seem the same but they are different

15

Employee Vs. Independent Contractor

- Independent Contractor Benefits To Practice Owner
  - Does not have to pay payroll taxes
    - (EI, CPP, EHT)
  - Does not pay for benefits
    - Health, Dental, CE
  - Does not have to pay severance
    - 1 month for every year
  - Does not have to keep job open when on maternity/parental/leave

16

Employee Vs. Independent Contractor

- Independent Contractor benefits to Associate
  - Don't have to pay EI Premiums
    - $1,077
  - May write off more expenses as tax deductions
    - $7,500
  - If you don't contribute to EI, you cannot receive EI benefits (e.g. maternity/parental leave)

17

Self Employed EI Option

- Self-employed can be eligible for the following benefits:
  - Maternity/Parental
- Self-employed are not eligible for unemployment benefits
  - To "opt in", must pay EI premiums for at least one year before taking a leave
  - Once you've accessed EI, you must continue paying premiums (up to $1,002 a year) for as long as you're self-employed

18

Employee Vs. Independent Contractor

Important to "pass the test" (CRA, WSIB)

- Control of the Schedule
- Ownership of Tools
- Risk of Loss
- Integral to the Organization

If you only work for the same practice(s), CRA will likely view you as an employee

Professional Independence

19

What is a Good Agreement?

- Protects interests of both parties
- Deals with issues likely to be contentious
- Fair to both parties
- Prevents problems from arising down the road by clearly setting out expectations
- **Written contracts only become important when things go wrong**
  - *The best contracts are never read*

20

It's Not Just About the Money

OVMA Career Satisfaction Survey

- **What makes a career satisfying?**
  - **Financial Stability**
  - **Good Relationship with Colleagues**
  - **Access to Quality CE**
  - Positive Relationship with Clients
  - **Good Work/Life Balance**
  - Excellent Medical Skills

21

Negotiating "No Regrets" Contract

Negotiate Appropriate Salary & Benefits	Negotiate collegiality
• *OVMA Survey of Compensation and Benefits*	• All the "other stuff" • Hours of Work • On Call Rotation • Vacation Duration and Timing • Holidays/Time Off • Non-Competition/ Solicitation Clauses

22

How You Get Paid Makes a Difference

Method of Compensation	Median Annual Compensation ($)	Median Hours Worked	Calculated Hourly Wage ($)	Number of Responses
Hourly	140,000	1,610	86.96	159
Annual Salary	126,000	1,645	76.60	195
Annual Base Salary + Profit Sharing (eg. Paid a bonus based on total practice revenue)	157,500	1,656	83.03	20
Annual Base Salary + Emergency Fees	120,000	1,880	63.83	33
% Of Gross Billings (Revenue) without Guaranteed Salary	189,500	1,652	114.71	6
% of Gross Billings + Guaranteed Annual Salary (ProSal)	139,000	1,656	83.94	99

23

Commission Targets

- Target Income $120,000
- Pay Percentage 23%
- Gross Required $521,739
- Days/week 5
- Holidays 3 weeks
- Days Year 235
- Gross / Day $2,220
- ART $200

- Clients per day 11

24

Negotiating Benefits

CE Fees	87%	$2,000
CE Time off	79%	4.0 days ($2,500)
Dental / Health	73%	$3,000
OVMA Dues	70%	$500
CVO Dues	89%	$1,200
Malpractice Insurance	83%	$525
Uniform	66%	$200
TOTAL COST		**$10,000**

25

How to know what to ask for?

Associate Compensation & Benefits Report

- Salary
- Production Compensation
- Benefits
- Vacation
- Broken down by area, type of practice, full or part-time, years in practice, etc.

26

Negotiating Your Compensation

Method of Compensation	Component	25th Percentile	Median Value	75th Percentile	Number of Responses
Hourly	Hourly Rate	$75.00	$95.00	$115.00	159
Annual Base Salary + Profit Sharing	Salary Portion	$109,500	$130,000	$151,350	18
	% of Profit Share	3.5%	15%	22%	7
	$ of Profit Share	$9,000	$10,000	$16,000	15
% Of Gross Billings (Revenue)	% of Gross Billings	22.0%	23.5%	25.0%	4
Guaranteed Annual Salary + % of Gross Billings (ProSal)	Guaranteed Salary	$100,000	$116,000	$135,000	64
	% of Gross Billings	20%	22%	23%	93

27

Hours Worked

- In 1991, veterinarians worked an average of 2,130 hours a year (43 hours a week) and rarely took vacation time
- Today, veterinarians work an average of 1,655 hours a year (36 hours a week) with 3 weeks holidays

28

Hours Worked Decreases With Experience

	Total Years in Practice	Median Annual Compensation ($)	Median Hours Worked	Calculated Hourly Wage ($)	Number of Responses
Full-time	<1 Year	115,000	1,840	62.50	28
	1-2 Years	120,000	1,692	70.92	59
	3-5 Years	131,000	1,692	77.42	101
	6-10 Years	135,000	1,692	79.79	129
	11-15 Years	144,000	1,584	90.91	73
	>15 Years	130,000	1,459	89.10	158

29

Vacation Ramps up After First Year

	Years at Current Practice	Median Weeks of Vacation	Number of Responses
Full-time	<1 Year	3.0	88
	1-2 Years	4.0	126
	3-5 Years	4.0	117
	6-10 Years	4.3	56
	11-15 Years	4.0	37
	>15 Years	4.0	37
Part-time	1-2 Years	3.0	9
	3-5 Years	3.0	9
	6-10 Years	4.0	6
	>15 Years	3.5	14

30

Veterinarians and the *Employment Standards Act*

Act sets out requirements governing:

- Hours of work
- Overtime
- Vacation Time/Pay
- Statutory Holidays
- Pregnancy/Parental Leave
- Termination of Employment

31

Veterinarians and the *Employment Standards Act*

Provision:	Veterinarians Covered
Termination	Yes
Pregnancy/Parental leave	Yes
Hours of work	No
Vacation time/pay	No
Statutory Holidays	No
Overtime	No

32

What Should a Contract Specify?

- How many hours/week will you be working?
- How will hours over and above that amount be handled?
- How many weeks of vacation are you entitled to?
- Vacation coverage?
- On call?
- **How are statutory holidays handled?**

33

Time Off

- Sick time
  - 5 days
- Vacation
  - 3 weeks
- Other
  - 20% of new grads have extra pay or time off for longer work weeks, in lieu time*
- Paid Emergency Leave (PEL)
  - 10 days (2 paid)

34

Non-*Employment Standards Act* Matters You May Want to Address

- Large vs. small animal practice
- Work schedule (evenings and weekends vs. weekdays)
- Vacation coverage
- On Call Requirements

35

On-Call Compensation

Type of Practice	Percentage of Respondents Required for On-Call	Median Number of On-Call Weekends per Month	Median Number of On-Call Nights per Week	Percentage of Respondents Compensated for On-Call	Median Percentage of After-Hours Fees Paid	Median Amount per After-Hours Call
Companion Animals	8%	1.0	1.0	67%	50%	$130.00
Mixed	93%	1.0	1.0	70%	50%	$200.00
Food Animals	93%	1.0	1.0	69%	n/a	n/a
Equine	100%	1.0	1.0	77%	75%	$200.00
Emergency	33%	1.0	1.0	33%	n/a	n/a

36

Non-Competition Agreement

- 53% of associate veterinarians have a non-competition agreement
- 56% have non-solicitation agreements

37

Non-Competition Agreement

- **What is it ?**
  - Typically states that you cannot work within a specified distance of the practice or municipality for a specified period of time
  - E.g. You cannot work with 25 kilometers of the municipal boundary for 3 years
- **Is it legal?**
  - October 2021 employers were prohibited from entering into agreements with employees that include a non-compete
- **Exclusions**
  - Executives (e.g. Chief Operating / Chief Executive)
  - Practice sellers are not included

38

Alternative to Non-Competition Agreements

- Non-solicitation agreement
- Should you leave your current practice, you will not solicit any of the practice's clients for a specified period (e.g. 12 - 18 months)

39

Non-Solicitation Agreement

- Protects owner from losing clients
- Easier to enforce than a non-competition covenant
- Doesn't deny you the ability to earn a living in the local community

40

Importance of Legal Advice

Signing a contract that is unfavourable to you could:

- Force you to stay at a practice where you are unhappy
- Deprive you of your ability to earn a living as a veterinarian in your community
- Cost you thousands of dollars in lost income or future legal fees

41

Questions?

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42

13002

STARTUP OR BUY AN EXISTING PRACTICE

SUCCESION

Greg Toner, CPA, CA, TEP, CLU
Darren Osborne, MA

ACT II
Startup or Buy an Existing Practice

Greg Toner, CPA, CA, TEP, CLU
Darren Osborne, MA

1

- After ten years of working as an associate, our veterinarian wants to own their own practice and questions whether to buy an existing practice or opens a practice from scratch. They go to Greg and Darren with a list of practices for sale and weigh the pros and cons of purchasing an existing practice or opening one up from scratch. In this session Greg Toner and Darren Osborne will walk you through two different practice valuations. They will also show how to forecast the first three years of the startup practice demonstrating what the banks are looking for to provide financing and what a new practice owner can expect in terms of clients, revenue, expenses and income. Find out what can go wrong and how the wrong management decision can affect the bottom line.

2

Buying vs Starting

- Buying a practice
  - If **priced right**, you can pay yourself as a DVM from start, pay all the bills and have enough to pay the bank
  - Pay off in **five** years
- Obstacles
  - No practice available
  - Practice available is "over valued"
    - Will take 10 years to pay off practice loan
- Which scenario will make you better off in one year, three years, five years, and twenty years?

3

How a Veterinary Practice is Valued

- EBITDA
  - Earnings
  - Before
  - Interest
  - Taxes
  - Depreciation
  - Amortization
- Pay yourself Fair Market Value, take off loan interest, income tax and depreciation

4

Accounts Not Included in EBITDA

- Drugs and Supplies
- Laboratory
- Non-DVM Wages
- Associate Wages
- Specialists
- Rent
- Office Supplies
- Bank Charges – loan portion
- Repair & Maintenance
- Utilities
- Advertising
- Legal and Accounting
- Professional Dues
- Insurance
- Continuing Education
- Equipment Rental
- Bad Debt
- Grooming Expenses
- Depreciation
- Automotive

5

Practice Value Estimate

- EBITDA
  - Total Revenue $1,000,000
  - Less Non-DVM Expenses 500,000
  - Less Associate Wages 100,000
  - Less "Fair" Owner Wages 200,000
    - (80 hr or 25% of rev)
- **$200,000**
- Capitalization Rate – 4
- Practice Value Estimate
- $800,000

6

Capitalization Rate or Multiple

- The expected return on EBITDA.
  - Higher risk – lower Multiple
  - Lower risk – higher Multiple
- Bank will only lend a veterinarian a maximum of 5 x EBIDTA
- Why do corporate consolidators pay more?

7

Sample practice valuations

8

- Our veterinarian was not able to find a practice that suited their needs so they choose a startup.

9

Startup Checklist

- Location
  - Bylaws
- Rent or Buy
  - Size
  - Market
  - Interest Rates
- Staffing
  - Staff Mix
  - Wages

10

Rent or Buy

- Purchase Property and Building
  - Veterinarians occupying more than 50% of their space qualify for a commercial mortgage
    - at prime
    - 100% financing
    - 20 years
  - Mortgage payments may be cheaper than rent
  - Practice pays for a personal asset – infinite return on investment

11

Mortgage vs Leasehold Improvement Loan

- Mortgage pays for the outside
- Term Loan for leasehold improvements pays for the inside

12

Rent or Buy

- Cost to Borrow Land and Building
- $1,000,000 $6,400 per month
- $800,000 $5,100 per month
- Is rent cheaper than mortgage?
- Surrender personal savings to contribute to mortgage

13

Rent vs Buy and Long Game

- $2,000,000 building
  - Purchase for $76,800 annual mortgage (25 years at prime)
  - Rent for $42,000
- Assuming land and building appreciate 3% per year
- Invest difference ($35,000) at 7% return for 25 years

14

… 25 Years later

- Purchased land and building for $2 million
  - Value $4 million
- Rented and invested the difference at 7%
  - Value of asset $2.84 million

15

Rental Considerations

- Rent – less than 6% of gross
  - High visibility
  - Parking for clients and staff
  - Proximity to other veterinary hospitals
  - Terms of lease
    - Demolition clause?
    - Renewal terms?
    - Transferability?
  - Cost including TMI or common fees
    - Taxes Maintenance and Insurance

16

Building Considerations

- Smaller footprint
  - Less expensive rent
  - Less expensive leaseholds
  - $300 per sqft
  - $420,000 for 1200 sqft or $1.2 million for 3600 sqtf
  - $4600 or $13,800 per month
- "You should take two units now before they are sold, or you might have nowhere to grow."
  - Cost $100,000 more pet year
  - 112 more clients needed in the first year to break even

17

Best Case Startup

- One "unit" (1,200 square feet) or less
- Three exam rooms
- Leaseholds under $400,000
  - Don't fall for the – "its only $20,000 more for …
- Buy used equipment
  - No moving parts allowed
- Least expensive option assuming you are going to reno or replace
  - Ikea cabinets
  - Formica countertops

18

19

What if…

- Rent is too high
- Construction costs are too high
- Construction took longer than expected
- Equipment costs are too high
- Legal costs for zoning were too high

- Work locum shifts into the mix
- One emerge locum shift per weekend
  - offset $72,000 in cost overuns

20

21

Additional Startup Costs

- Legal $10,000
- Accounting $10,000
- Architect $25,000
- Permit Application $5.000
- Tender $5,000

- Separate financing

22

How Many Loans Do You Need

- Mortgage to buy land and building
- Term Loan for leasehold construction $400,000
- Term Loan for equipment $150,000

- Line of Credit $100,000
  - Startup costs
  - Cost overruns in first year

23

Approaching the Bank

- Business plan
  - OVMA 3 year projections
  - Bank template
- You Do It
  - Makes you really think about why you want it
- Use their template
  - Acts as a checklist
  - Keeps it brief
  - Some poor sole has to read this
- Don't use AI

24

Staffing a Startup

- The stress of not having enough staff and hiring more is better than having too many staff and "laying off" staff because you are not growing fast enough.

- 40 clients per month
- 10 clients per week
- 2 clients per day

- Start with a skeleton staff
  - 1 RVT
  - 0.5 reception

25

26

Pro Tips

- Equipment providers have equipment checklists
  - Buy used equipment when you can – nothing that moves
- New Practice Startup Packages
  - Equipment manufacturers
  - Service providers
  - Food and Pharma companies
- Consultants have startup checklists

27

Professional Chain of Events

- OVMA
  - Work out a plan
- Bank
  - Work out financing
- Mentor – classmate who has been down the road

- Accountant
  - Set up your "books"
- Lawyer
  - Set up your leases, contracts, corporation(s)

28

Questions?

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29

13003

EXPANSION AND SELLING THE PRACTICE

SUCCESION

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Darren Osborne, MA

ACT III

Expansion and Selling the Practice

Greg Toner, CPA, CA, TEP, CLU

Darren Osborne, MA

1

The new practice exceeds expectations, and our practice owner starts looking toward expansion. They struggle with the decision of when to bring on a new associate and whether to offer partnership. They have the practice valued and explore the possibility of buying their own building and relocating the practice. The valuation for the practice comes in higher than expected and the bank makes an offer to finance their new building with no money down. Success abounds and our practice owner looks to sell the practice. The practice has grown to three veterinarians, and they are surprised that the value is not three times higher than the first valuation. They consider selling to corporate, selling privately and after a lot of soul searching, decide to sell the practice to the employees to take advantage of the new Employee Ownership Trust. Greg Toner and Darren Osborne will go through the short history of practice values in Ontario, how corporations have affected value and forecast how much a practice in Ontario will be worth in the coming years. They will also show the tax considerations regarding selling a practice and whether the new Employee Ownership Trust is for you.

2

Expanding - Space

1. Square footage - 4,000 Feet!
2. Exam Rooms - 3 more - total of 5
3. Dental Suite
4. Surgery Suite
5. New Revenues
   a. Dentistry
   b. Complex surgeries
   c. RVT appointments

3

Expansion - Lonely at the Top

1. It's all on your shoulders
2. Live and die by your decisions
3. Staffing issues
4. Practice challenges
5. Financial challenges

4

Expansion - Classmate

1. Best friend at OVC
2. Moving home
3. Experience working in a ER practice
4. Never managed a team or practice

5

Partnership - Pros

1. Shared Responsibilities - Workload and Distribution
2. Financial Support - Shared Costs
3. Collaboration and Idea Generation - Diverse Perspectives
4. Risk Mitigation - Shared Risks and Support
5. Work-Life Balance - Coverage and Spread Stress
6. Enhanced Client Service - Broader Expertise & Availability
7. Support - Emotional and Professional

6

Partnership - Cons

1. Disagreements - Decisions & Vision
2. (Perceived) Uneven Workload - Time & Skills
3. Shared Financial Risks - Debt and Profit Allocation
4. Loss of Autonomy - Compromises
5. Relationship Strain - Friendships
6. Exit Challenges - Timing & $$$
7. Risk Tolerance
8. Admin & Legal Complexity - Agreements & Spending

7

8

Purchase of a Building - Banking

1. The bank will finance 100% of the purchase of the right building
   a. And the renovation
2. "The Right Building"
   a. You can afford it
   b. You can afford it
   c. You can afford it
   d. You can afford it
   e. It's close to the old practice
   f. It's big enough
   g. You don't have to renovate too much
3. Debt
   a. 20 year amortization
   b. Prime less 0.25%

9

Purchase of Building - Structure

1. New Company
2. Ownership?
   a. TOSI
   b. SCI
3. Rent
   a. @ Market
   b. Should be enough to cover mortgage payments
4. Expenses - Clinic Paid
   a. Property Taxes
   b. Maintenance
5. Renovations - Clinic Paid

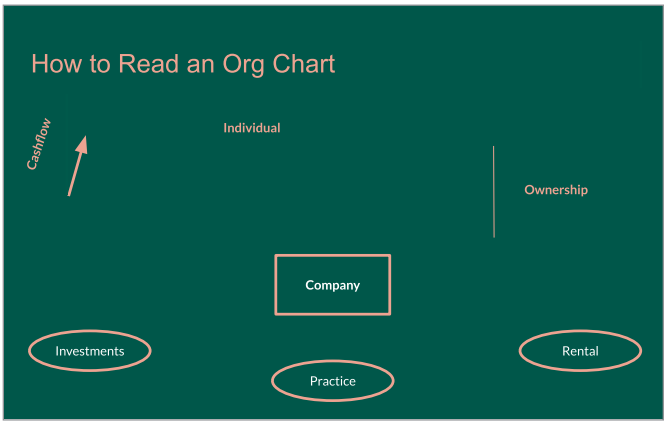
10

Partnership Dynamics

1. Who does what?
2. Bookkeeping is tougher
3. Expense management is tougher
4. Compliance is harder.
5. Mismatched cash flow needs for debt
6. Management
   a. Role definition
   b. Compensation

11

How to Read an Org Chart



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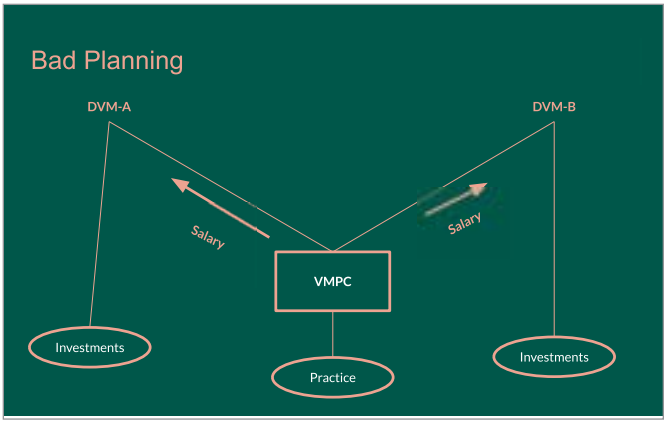
Don’t tie yourself to your partner’s lifestyle decisions & defer as much as possible.

13

Bad Planning



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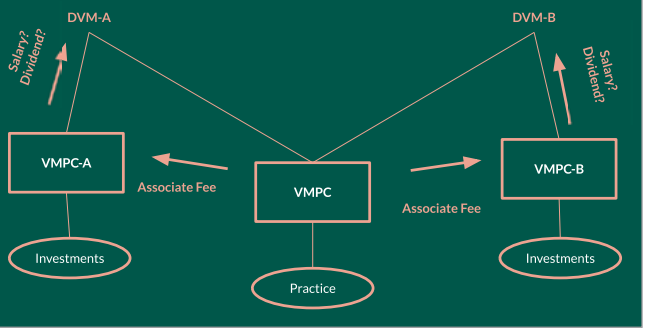
Independent Compensation Structure



15

A Brief History of Practice Values

- 90’s
  - US Corporate consolidator moves up to Canada
  - Opens 6 veterinary hospitals attached to pet stores
  - Regulators fought to keep them out
  - Separate entrance / steering / etc
- The Consolidator eventually gave up and sold the “practice” to existing associate DVM
- Newly Private / Previously Corporate practices flourished

16

Take Two – Corporate Veterinary Medicine In Canada

- 2010 (ish) US Corporate Consolidator starts purchasing existing practices
  - Previous model involved building new practices
- Canadian Corporate Consolidator emerges to compete directly with US Corporation
- Started offering more than 5 times profit
  - "They are not getting their money from the bank."

17

Multiples Start Going Up

- "EBITDA" and "multiple" become part of veterinary vocabulary
- Corporate consolidators
  - Start paying 7 times EBITDA
  - Start recruiting for veterinarians at the vet school
  - Start getting really big

18

Multiples Keep Going Up

- Interest Rates are Low
  - Cost of borrowing is low
  - Lack of alternative investments makes "*the vet space*" more attractive
- Would you rather…
  - Invest in bonds paying 1%
  - Invest in a veterinary hospital yielding 5%
- Competing for a limited number of practices drive the multiple up to 20+

19

And Then…

Interest soared

Help Wanted Advertisements for associate veterinarians soared

Pandemic Demand started to level off

IPOs never took off

Multiple went from 20+ to 10

20

What is Your Practice Worth Today

21

Buyers - Associate

1. Working in the practice for 8 years
2. Staff love them
3. Clients love them
4. Knows the systems
5. Friendly relationship

22

Buyers - New Grad

1. Graduating in May
2. Worked as RVT and Practice Manager at another clinic
3. Limited surgical experience

23

NCT3Cor

1. Large consolidator
2. Owns 200 clinics
3. Owns clinics nearby
4. Mixed reviews from other vendors

24

EOT

1. A trust established for the benefit of the employees
2. It purchases the shares of the clinic from the vendor
3. Distributes profits from the practice to ALL employees
4. MANY restrictions

25

What Does the Practice Look Like?

1. Suburban Practice in Stand Alone Building
2. 4,000 sqft / 5 Exam rooms
3. 2.5 million in Revenue
4. 3.5 FTE DVM Practice - 6 DVMs with 20 Employees
5. Owners and Associates work 1200 hours each
6. 65% Service vs 35% Product
7. EBITDA $320,000

26

What Does the Practice Look Like - Normalized EBITA

1. Suburban Practice in Stand Alone Building
2. 4,000 sqft / 5 Exam rooms
3. 2.5 million in Revenue
4. 3.5 FTE DVM Practice - 6 DVMs with 20 Employees
5. Owners and Associates work 1200 hours each
6. 65% Service vs 35% Product
7. EBITDA $320,000
8. Normalized EBITDA $400,000

27

28

The Offers

1. Associate offers 5x EBITDA - $2.0 Million
   a. Owners paid salary with two year commitment
2. New Grad - $2.0 Million with mentoring for two years
   a. Owners paid Pro-Sal?
3. NC3Cor offers $3.0 Million with 2 year earn out, 10% JV, 3 year commit
   a. Owners paid Pro-Sal / must stay for two years
   b. Owners retain 10% each to be sold at “fair market value” at a later date
4. EOT - $2.0 Million
   a. No owner commitment
   b. Banking challenges

29

Big Comparison Slide

1. Offers
2. Post Transaction
   a. Hours
   b. Salary
   c. Work Obligations
   d. Management Obligations
3. Taxes
4. After Tax
5. Compensation
6. Total Proceeds and Comp

30

Questions?

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13004

RETIRING WITH MONEY

SUCCESION

Greg Toner, CPA, CA, TEP, CLU
Darren Osborne, MA

ACT IV

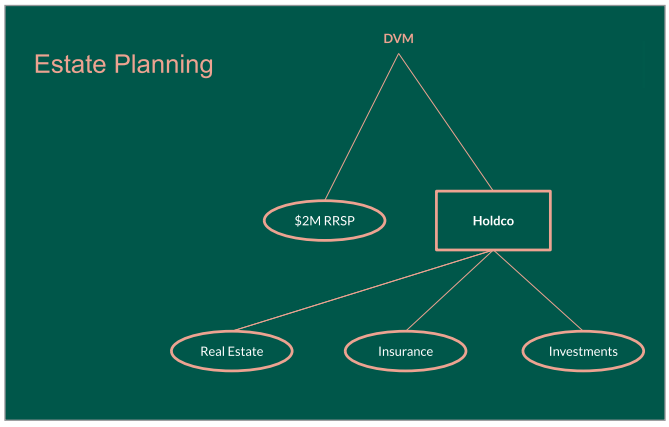
Retiring with Money

Greg Toner, CPA, CA, TEP, CLU
Darren Osborne, MA

1

After starting a practice, expanding into partnership, buying a building and selling a practice, the tax structure for our retiring veterinarian looks like a crime drama murder board. In this session, Greg Toner will walk through different scenarios for our retiring veterinarian so they can **minimize their tax burden, maximize their annual income in retirement and leave money to their successors**.

2



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Be Ready

You need to be ready to sell.

Now.

Because you never know when it will happen.

4

Sale Planning - Isolate the Practice (and Building)

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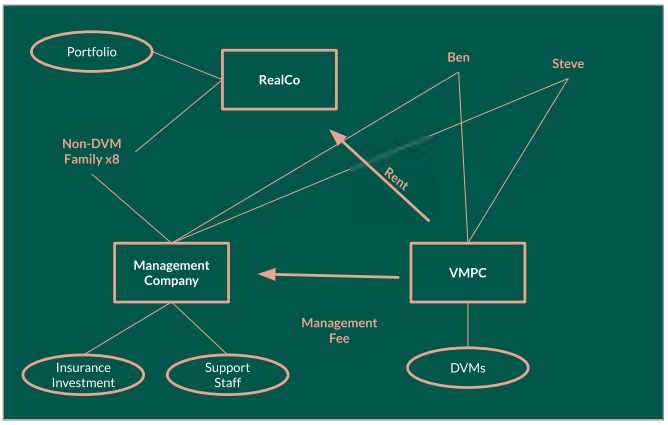

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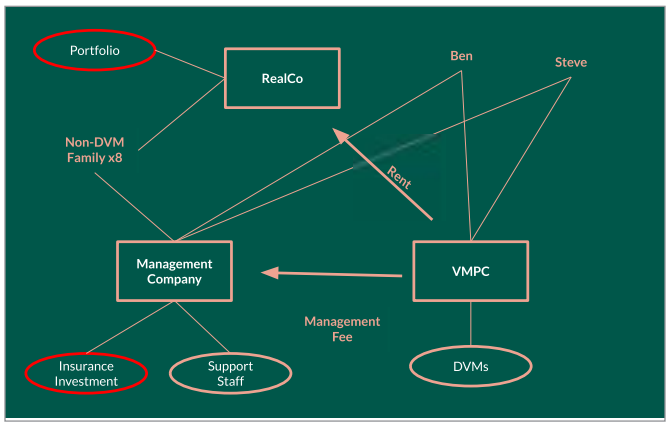

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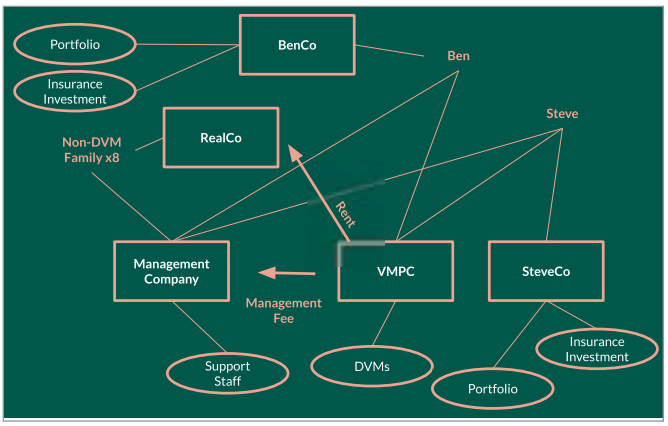

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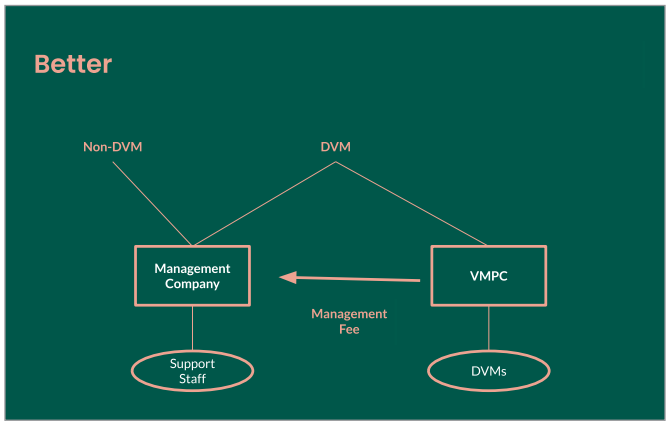

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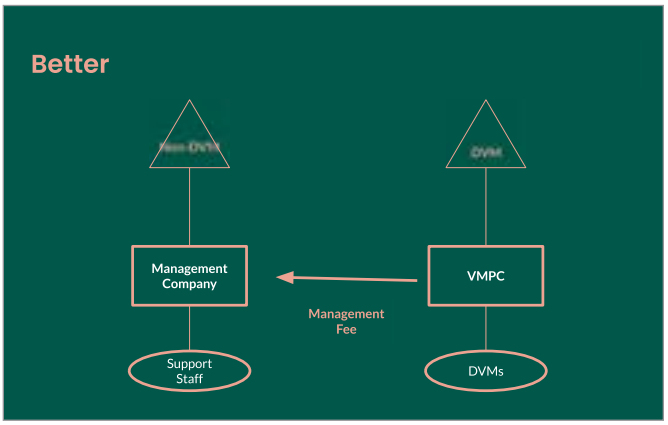

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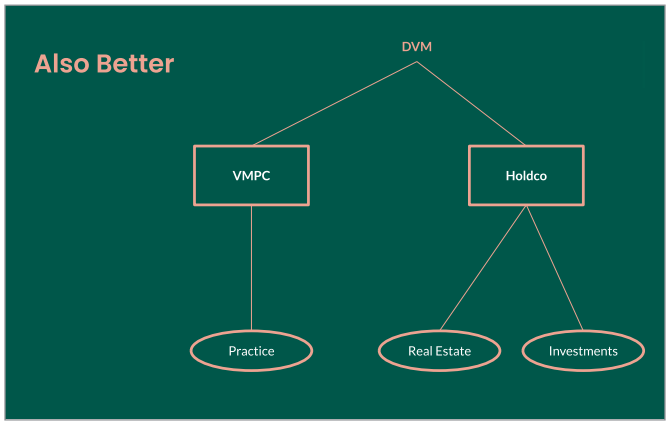

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Questions?

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14001

TALKING WITH THE TEAM: I'D RATHER PLAY WITH POOP

STAFF MANAGEMENT

Andrea Crabtree, BS, CVPM, PHR, SPHR, PHRca, CCFP, FFCP

OVERVIEW OF THE ISSUE

Imagine a Veterinary Practice with clear, concise, and consistent communication.... Although communication is key, it doesn't always work seamlessly in a Veterinary Practice. Learn the importance of communication styles through how YOU communicate and want to be communicated to and how that may not work in sticky situations or with impenetrable and complicated employees. So then what? Recognizing how others hear and want to be heard will go a lot farther when dishing out heard-to-hear feedback. Understanding ourselves goes a long way to understanding others.

OBJECTIVES OF THE PRESENTATION

Our learning objectives are:

- A How-To Guide to help leadership communicate with impenetrable and complicated employees.
- Learn the importance of communication styles.
- Create an environment that encourages and fosters open, honest, transparent communication.

SUMMARY

The most dreadful part of the day is knowing a tough conversation with a team member is on the horizon, past due, and needed, yet there never seems to be a good time, emergencies happen, there are never enough team members to cover, and honestly, it just never goes well.

Imagine an environment that encourages and fosters open, honest, transparent communication. What would that look like? Why not work on creating that ideal situation, anytime, any day, with anyone? Let's start with the obstacles, since there are usually too many to count, let's address the BIG ones. Space is limited, sometimes non-existent. Timing is never right between emergencies and coverage, let's face it, it's not going to happen. These conversations are seldom received well, almost always uncomfortable, and left unresolved.

Privacy

Be creative! If there is no place on site to hold a confidential conversation, have it off-site. Sometimes, being in public will help control an otherwise unruly team member and cause them to pause any unprofessional behavior. This can also help ensure keeping the conversation confidential without any other employees overhearing. If meeting off-site, scheduling the meeting before a shift, before or after a lunch break, or at the end of a shift also allows for carved quiet, uninterrupted time. At a minimum, this can be filtered through a call or text from the practice.

If on-site is an option, be sure to keep voices lowered to avoid others from overhearing. Hanging a sign on the door that signals Do Not Disturb also will help with disruptions.

During the meeting, be sure to have examples, be prepared, be vulnerable, and be authentic. Body language is key, keep that in check. Listening is a skill that takes practice and must be developed over time. Some things that can help include taking notes and asking open-ended questions that don't lead to the answer to the questions. If there are any action items, be sure to follow up and circle back in the appropriate time frame.

Communication Styles

Who are you? What stories and experiences makeup who you are? There are a vast majority of personalities with

an endless mix of each in every practice. Discovering who YOU are will help you understand how to best communicate with others. There are many personality assessments, including Myers Briggs, NueroColor, DISC profile, High 5, and so many more. Try one out for free at https://www.personalityperfect.com/.

Discovering that people not only communicate but listen differently can help us deliver important information that is received and absorbed. Often, people are just not aware of how their behavior affects others or defines the culture or environment they are a part of. This is the perfect time to ask the why. Why did the behavior occur? What were the contributing factors?

Was there a specific trigger? Dig deep, ask why over and over in a variety of ways, peeling the layers of the onion back until the root of it all is uncovered.

Just Do It

Be prepared for pushback, denial, and excuses and for them to transfer blame. Sometimes, being a broken record or circling back to the conversation is necessary to stay on track. Have specific examples to explain and show how the behavior can affect others. Have a few, one or two, ways that outline a more productive, team-player approach with a different outcome.

This takes practice. Practice takes time. This is an intentional and deliberate choice. It is uncomfortable and is a stretching exercise that leaves us tired and sore, like a hard day at the gym. But that muscle will develop and become toned over time.

Physiological responses are the body's automatic reactions to a stimulus. Stress is a stimulus. These types of conversations can be stressful, causing a stimulus and, therefore, physiological responses such as a pit in the stomach, sweaty palms or armpits, flush or redness in certain areas like the face and neck, crying, and sometimes just the need to find the nearest trashcan to puke, UGH! These are all normal, unfortunately. However, recognizing and preparing for these can help. Pep talks in the mirror or from a friend may seem silly but are very helpful. Write down what to say and memorize the bullet points. Awkward silence is a trusty friend. Take a few (or several) deep breaths, review your thoughts, check your notes, and start again.

Celebrate the wins! This can be done even with the smallest of baby steps after the conversation is over, regardless of whether it went poorly, you made it! A good pat on the back is due. Take time to recover, review what was said, look for areas of improvement, and recall what went right. Be prepared to do it all over again. Mistakes are an opportunity to learn and improve.

14002

HOW TO IMPROVE YOUR PRACTICE CULTURE IN THE "NEW NORMAL" CHAOS

STAFF MANAGEMENT

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OVERVIEW OF THE ISSUE

Learn to cultivate and develop a healthy practice culture in the "New Normal" post-COVID-19 veterinary practice. Assess the damage of the chaotic, busy practice life and what damage control can and needs to be done in the aftermath. Look at creative ways to work towards a healthy and efficient team-focused work environment and boost employee engagement and motivation through creative policy and protocol changes. Define appropriate and professional communication guidelines and take time for team accolades on their terms.

OBJECTIVES OF THE PRESENTATION

Our learning objectives are:

- Discuss creative ways that work towards a healthy and efficient team-focused working environment.
- Boost employee engagement and employee motivation through effective policy and protocol changes.
- Define guidelines for appropriate and professional communication.
- Incorporate team accolades on their terms during key engagement sessions.

SUMMARY

What is Culture?

The classic definition of formal company culture is:

The widely held set of beliefs that are established by the key leaders in a company, who then define the proper behaviors expected throughout the multiple layers of the company and for the individual employees.

Consider the following:

- High efficiency can mean increased mistakes.
- Lack of accountability can lead to a sacred cow.
- Value-centric can translate to a high volume where shortcuts are OK.
- Client-centric can result in abuse where the clients are always right mentality.
- Patient-centric can make the client wait time higher.
- Owner-centric can be a lack of work-life integration, a nose-to-the-grind feeling.

However, consider a good culture, where "good" can be measured in several ways. An engaged team has lower absenteeism, higher retention, and increased efficiency, all resulting in a bottom- line net profit increase.

Who defines culture?

A company's culture can be defined in many ways and perceived through many lenses, whether that be the formal leadership team, the organizational structure of the company, the key leaders within the practice, the individual teams (or departments), or the employees themselves.

Here are some things to take into account when describing current culture. What does your Organizational Chart look like? Do you have a "sacred cow" affecting culture? Does the Leadership team walk the walk & talk the talk? Who does the team walk on eggshells around?

When does it affect the team?

Culture affects the entire team and drives engagement, fulfillment, & PROFITABILITY!

Employee performance and productivity, employee absenteeism, and tardiness are a part of the culture. Trust includes theft of services, products, and drugs, employee injuries and safety, employee turnover, client service and satisfaction, and patient care are all a part of the culture and are affected daily.

Tradition

Traditionally, practices were and still are either patient-centric or client-centric. Which one does your practice mostly identify as? Then COVID-19 happened, and we made more changes daily than we would traditionally make in a month or even annually. Changes were inevitable and quick, and yet we made it through. Some of us might still be making changes regularly due to COVID. Who is still offering curb-side services?

Consider this an employee-centric practice. What does this look like to you?

Healthy: What is the breakroom filled with? What about psychological safety, Employee Assistance Programs, and mental health breaks?

Efficiency: What about using radios, messaging software, making systems repeatable, and using technology to reduce clunky systems?

Team-focused: Can we hold team meetings and team-building events, encourage time off, and rethink compensation models?

Employee engagement: Increase commitment to the practice's Mission, Vision, and Values.

Employee motivation: Drive employees towards a common goal, increase productivity, and have greater practice profitability.

Effective protocol changes: Review how each department is/will be affected by policy change, bring in key practice leaders to design and implement changes, communicate changes team-wide, and be willing to modify as needed.

Appropriate and professional communication:

- Listen with the intent to understand and NOT with the intent to respond.
- Ask more questions and be inquisitive.
- Outline what is acceptable and what is not!
- Feedback, LOTS of it!
- Incorporate EQ, not just IQ.
- Institute accountability.

Team accolades: Provided regularly, when appropriate, find out HOW it is best received, acknowledge milestones, and celebrate successes (even the small ones).

Engagement sessions: Have one-on-ones, team meetings, morning rounds, and collaboration that create platforms for two-way conversations.

Career mapping: Ask where THEY want to go. Create environments for continual learning, pay for school, training, training, & more training. Consider leveling positions that allow team members to level up with knowledge, skill, and ability development.

Culture exists, period. Define yours, Imagine the possibilities, and think BIG! Create a plan and map it out. Implement ideas and make it the "New Normal," Create change – we know we can.

Be Employee Centric

14003

WORK ON YOUR PRACTICE NOT JUST IN YOUR PRACTICE

STAFF MANAGEMENT

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OVERVIEW OF THE ISSUE

We often get caught up in working IN our practices - answering the phone, handling a disgruntled client, covering during lunch breaks since we are short-staffed, drawing blood on a difficult patient, helping restrain a large breed wiggle worm on the x-ray table, placing an order for the out-of-stock inventory items, and the list goes on and on and on… it's never-ending because it's literally never-ending!

We have projects that work ON the practice - developing a new training manual, updating the Employee Manual, strategic planning for the new year, rolling out a new Employee Assistance Program with an emphasis on Mental Health Awareness Program to the team, and SO much more that we just don't and can't do.

So how can we work ON the practice?

OBJECTIVES OF THE PRESENTATION

Our learning objectives are:

- Discuss WHY it is important to work ON practice improvements.
- Identify obstacles that get in our way and some solutions to address the obstacles.
- We take steps to build the ideal practice, including designing, excavating, foundation, framing, building, decorating, and furnishing.
- Define guidelines for appropriate delegation.
- Incorporate core values that allow the entire team to live IN the practice.

SUMMARY

What is the difference between working ON your practice vs. IN your practice?

Do you want your practice to stay at status quo or GROW? If you want to grow, then how will this happen? What results do you want to see? What is the end goal of practice development? Taking time to outline how you want your practice to grow is vital to end success. So how do we do this?

Building the Ideal Practice

There are several steps involved when building the ideal practice. The first is the design phase; this is where dreams become reality, and the sky is the limit! This looks like creating Practice Values, Core Ethic Statements, or Behavioral Norms. What does the perfect practice look like to you? How does the team interact, treat clients, and behave at work?

Then, once the dream is described and the design is defined, the physical process starts and begins to excavate the dirt! Where is the dirt in your current practice? Are their employees that cause drama, a lack of protocols or processes in place, or poor training for the team either for new hires or on a continual ongoing basis?

Once we have cleaned up, we lay the foundation by developing the Mission and Vision. Where is the practice heading? What Vision does the practice owner have, and does the team know it? Is the team on board to help carry out the Mission to achieve the Vision?

Build the team by developing their knowledge, skills, and ability, or KSA. This means keeping the team engaged in their job, building them through professional development and job enrichment. How does the practice measure team engagement? Is there any career mapping or professional development plans?

We put some finishing touches by decorating and furnishing through constant coaching and feedback. How often does leadership meet with employees to coach them by delivering feedback in a manner that cares personally while challenging them directly?

However, NONE of this happens if we don't have the time for the process. So, what's stopping you? Well, let's see, the list is endless, literally endless, considering there will always be another patient, another upset client, another employee riff to handle, just to start, and all before noon.

The obstacles, distractions, and fires that burn us to the ground keep us from working ON the practice. The only way to dig out the bottomless pile is to delegate and offload tasks and duties to clear a path for more constructive projects. But who and how can we delegate properly, especially when it is faster to just do it?

Delegation goes both ways and is not a one-way street. To properly delegate, start with something small, then be sure to outline expectations, provide instructions, and set expectations for the desired outcome. Be sure to follow up and offer support and feedback on how the project is coming along before the deadline. Last is reviewing the final drafted project. Match up the expected outcome with the final draft, and decide if there needs to be more edits, changes, etc., or if the work meets expectations. Sometimes deadlines need to be renegotiated, sometimes more support or training is needed, and sometimes the outcome is better than expected! Keep in mind, the how isn't always important.

In summary, it takes time, dedication to the practice, commitment to the team, and, most of all, to yourself. Remember your WHY. There will always be an excuse. However, it is never going to be a better time to grab a hard hat, put on the steel-toe boots, and work ON your practice!

14004

DEVELOPING YOUR LEADERSHIP TEAM

STAFF MANAGEMENT

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OVERVIEW OF THE ISSUE

When employees are engaged, developed, and supported, we see increased performance and decreased turnover and absenteeism. The benefits of a well-managed practice are palpable, and it starts with the development of the leaders on the team.

OBJECTIVES OF THE PRESENTATION

Our learning objectives are:

- Identify willing and capable team members to coach, teach, and train to develop the practice.
- Understand and learn proper delegation techniques and training strategies to equip newly promoted leads, supervisors, and managers, setting them up for success in new roles.
- Broaden the knowledge, skills, and ability of the team and deepen the Organizational Chart, allowing for delegation of duties to the team and organically creating employee engagement.
- Discover ways to better leverage staff through leadership development, causing a shift in tasks and assignments.
- Provide support and training, identify platforms of communication, and create an environment of professional development.

SUMMARY

Sometimes, it may feel like the to-do list adds tasks faster than they can be crossed off. Sometimes, it feels like there just aren't enough hours in the day to get it all done. Suppose you could just clone yourself to get twice as much accomplished. It seems like it takes a Village! Or does it take a Leadership Team?

You can't be everywhere, every day. And if there is ever a chance to go on a real vacation, the practice MUST be independent of your physical presence. A fully functional, complete, well- managed practice can function independently of the practice owner and practice manager. But for that to happen, a leadership team must happen!

If we can think outside of the box for a moment, identify and leverage staff more appropriately, and leverage surrounding support to help train up and develop the team, it just might be possible to develop and leverage a leadership team to get results!

We start with WHO. Who is on the leadership team? This is different in every practice as there are a variety of roles within a veterinary practice. Consider a Supervisor, Lead, Assistant

Manager, Lobby Concierge, Director of Human Resources, Payroll Specialist, OSHA & Safety Officer, Front Office Manager, Inventory Manager, Marketing Manager, Director of Business Services, Practice Manager, Medical Director, Practice Administrator, and Operations Manager and many more. But titles are just titles until the role has clarity and is defined to fit the team member IN the role and to those that interact with that role. For this, we look to the Organizational Chart.

There are many layers to the Organizational Chart. Developing the Org Chart is a great first- start exercise to lay the groundwork for a leadership team. However, the Organizational Chart legend is almost more important than the Org Chart itself. The legend gives the entire team clarity for all roles. The Org Chart and legend let the team know WHO to go to about WHAT. For example, an employee may want to make changes to their personal

information, maybe they moved or changed bank accounts for their direct deposit. Whom do they ask about updating this information? The legend on the Org Chart will guide them. This also allows for one employee to redirect another employee when they bring something to the wrong person. For example, in the same situation, the employee brings this change request to the practice owner, now can be redirected with a simple, "That is not under my direction however, you can discuss it with the Payroll Specialist who takes care of all personal data changes."

Leaders are not always managers, and managers are not always leaders. Vocabulary.com defines a leader as, "a leader is the one in the charge, the person who convinces other people to follow. A great leader inspires confidence in other people and moves them to action." https://www.vocabulary.com/dictionary/leader

Susan Ward defines a leader and leadership as, "Leadership is the art of motivating a group of people to act toward achieving a common goal." A leader is someone "directing workers and colleagues with a strategy to meet the company needs." https://www.thebalancesmb.com/leadership-definition-2948275

When we think of a leader, some character traits may include honesty, integrity, accountability, and so on. These are typically NOT skills we usually interview for or evaluate during a working interview. These skills or aptitude to develop these skills can certainly be what we are searching for and identifying in current employees when considering leadership roles. We can start to leverage employees by identifying potential leaders. Think outside the box. WHO are leaders in your practice? How can we develop leaders? What are the tasks, duties, and projects we are delegating to leaders?

Who is asking for more duties and responsibilities on your team? Are they ready for more? Start with assigning small tasks and duties and build on that. Consider asking them to create the agenda for the team meeting next month (identify key areas of the practice that should be discussed) or empower them to develop a protocol for a problem area they brought attention to (critical thinking and problem-solving skills). Leadership skill assessments help identify natural leaders, go-getters, and overachievers. Assessments will identify personality types that provide insight and help create a balanced Leadership Team.

What support is offered? Once someone is identified for potential leadership, what kind of training is provided? Often, we set our teams up for failure by promoting without outlining expectations or giving the right tools, and then both parties are frustrated things are not going well. So, how do we train leaders or a leadership team? Continued education, internal meetings, Job Descriptions, mentors, discussing and outlining expectations, and providing resources are all excellent tools to use in this process. Leverage surrounding support for training through programs offered by allied industry partners such as Paterson Veterinary University.

Support and follow-up are vital to success. Communicate and review progress along the way, adjust goals and deadlines, offer feedback on performance, and discuss any obstacles in the way. One thing that often prevents and gets in the way is that darn to-do list. As tasks are added and projects are assigned, triage and delegation become important. A great exercise to use is asking, "Do I have to do this? Or can someone else?" Are tasks being delegated appropriately? Delegation can free up time for new projects. Setting SMART goals will bring clarity to tasks and projects by defining and setting deadlines, making sure the project is specific and realistic, and defining the end goal. Just be sure to follow up, coach, and evaluate along the way, or the result might not look the way it was originally anticipated. Be sure to evaluate the outcome once a project or task is completed. For example, is the new website buildout what we originally expected to see? Reward the success and discuss the disconnect where the project fell short. This evaluation process is key to future success.

When the leadership team comes together and is leveraged to manage the practice, they must function as a team or a unit. The book by Patrick Lencioni, The 5 Dysfunctions of a Team, provides steps for this process. These steps include building trust, which requires vulnerability, healthy conflict implies candid debate, commitment follows healthy conflict, and taking accountability, which requires commitment, all to deliver measurable results.

Do you have trust within the leadership team? Is there psychological safety when bringing up topics without feeling shame or blame? Is there confidence in the confidentiality of conversations? What are the intentions of others? Is there vulnerability and credibility?

Do you have healthy conflict with your leadership team? Are there productive ideas and healthy conflict versus drama and politics? Are issues discussed and resolved instead of having heated debates or a dictatorship with a 'do what I say' standard?

Is there a commitment from your leadership team? Are the leaders in the practice on board with the mission and vision? Is there clarity? Is there buy-in from everyone?

Is there accountability in your leadership team? Do some get away with what others can't? Are there assignments with no results? Policies without regard to who follows them? Is there follow-through?

What are the results? Using tools like SMART goals with accountability and follow-through will lead to results, an endpoint. Evaluate each along the way, each process. Learn from failures. Not every project or every task will be a success. Learn to pivot instead of wallowing in disappointment. Celebrate the wins, even the small ones.

It takes a village to manage a practice. There are several steps in the process. Begin by selecting leaders, defining roles and expectations, applying training, creating a leadership team, creating a safe space for trust and conflict, outline goals for commitment and accountability.

Results = WIN!

14005

REHIRE VS. RETENTION

STAFF MANAGEMENT

Andrea Crabtree, BS, CVPM, PHR, SPHR, PHRca, CCFP, FFCP

OVERVIEW OF THE ISSUE

It is no secret how hard it is to recruit, hire, and train employees these days. What if we didn't have to rehire? What if we can find a way to make them stay? In this presentation, retention is our key focus, along with being able to retain top talent and decrease turnover. We will look at four major areas that are the pillars of employee retention, including culture and engagement, comprehensive training and development, compensation and financial planning, and total team utilization. Our goal is to keep the team intact!

OBJECTIVES OF THE PRESENTATION

Retention is our key focus!

- We will identify areas that allow for job enlargement and job enrichment.
- We will dive into ways to enhance employee development and increase employee engagement.
- Identify skillset and how to train to deepen that skillset.
- Ways to maximize total team utilization.
- Discover creative compensation methods to pay a living wage.

WHO

Let's start with evaluating WHO we have on our team now. Can we assess knowledge, skills, and abilities, or KSAs? Let's level the field, literally. Define 2 to 4 levels in each department, the KSAs for each level, and outline expectations of each level and the pay scales for each level.

This may include adding extra assignments for individual roles, such as inventory or the employee schedule.

This may also include a lead or supervisor level in a department. Leveling should include the skill, knowledge, and experience needed and required, along with the pay range for that level. This helps team members know what they need to learn to level up, increase their wages, and be more valuable team members.

UTILIZATION

Once we understand each team member's skill set, how are we maximizing that skill and each person? Team members feel more valuable when they are contributing to the overall success of the business, and this can be done by one blood draw, follow-up call, appointment booking, or Rx refill at a time. So long as they have the skill, allow them to use it. The most expensive doctor is the one doing a technician's job! And the most expensive technician is the one doing a kennel's job! That is not to say we all can't work to support each other. Anyone in the practice should be able to pick up poop in the lobby and not step over it.

Job Enlargement and Job Enrichment are two areas that help the Practice Manager successfully delegate tasks to other team members at the same time of developing the knowledge, skills, and abilities or KSAs of the team members.

Job Enlargement allows for a team member to add more tasks to their job description but may not need more skills for those tasks. For example, team members who have a good grasp on their position and still have downtime, potentially shopping on Amazon or gossiping with another team member, can have more tasks. We can give them watering the plants in the practice.

There is no major disaster if this isn't done right, on time, or at all, we can always buy another plant. No harm, no foul. But it is an easy task that can keep idle hands busy.

Job Enrichment is adding additional tasks that require skill development whereby the team member learns something new. For example, a well-trained, seasoned CSR can learn how to manage the A/R report every month, taking this off the manager's To-Do List. Training and oversight may still be required however, minimal time in comparison to the entire project.

ENGAGEMENT

Do we want a team of low performers with inconsistent or poor behavior? Do we want a team of efficient problem solvers who are engaged? Can we coach up? If so, how? If not, what now?

Assess the behavioral norms and the desired culture. Then, look internally and see if we are meeting or missing the mark. Assess the expectations. Have we clearly outlined, and does the team understand and is meeting those expectations? All of these components make up employee engagement. Engagement compromises and is built on several key components, some of which include training and development, career mapping, coaching and feedback, and psychological safety.

If we have low performers, that may be because they do not know what is expected of them. Set a meeting and review expectations. What tools, training, and resources will they need to be successful? What obstacles might they face, and what can we do to circumvent, work around, and break through these obstacles? Provide direct feedback and speak to the areas of focus the employee needs to be working on to meet expectations. Include the time frame, and be sure to revisit and follow up. If they meet the expectations outlined, then the coaching and feedback worked! If not, the next steps might be discipline, including write-up, Performance Improvement Plans (PIPs), or termination.

Psychological safety is a robust concept and can be broken down into a simple definition of the belief that you won't be punished or humiliated for speaking up with ideas, questions, concerns, or mistakes (Wikipedia https://en.wikipedia.org/wiki/Psychological_safety).

Research is abundant in this area, specifically in the workplace. Practice Managers can assess the current psychological safety in the workplace and work towards creating a safe place for employees to voice their opinions and offer creative ideas and feedback within guidelines and parameters to cultivate a better culture. This, in turn, can provide a sense of belonging for employees and, therefore, deepen employee engagement.

Career mapping, training, and development are also key factors in employee engagement that were previously mentioned.

FINANCE & COMPENSATION

As the cost of living increases, as well as the cost of doing business, employee wages must also rise. Paying a livable wage meets basic necessities in people's lives but does not always make their jobs a long-term chosen profession. Licensed technicians have an industry shelf-life

of 5 to 7 years, and CSRs have a high turnover rate. How can we keep them? By meeting basic needs, starting with a career wage.

Back to utilization, are we using employees to the maximum of their skill set as the law allows? Investing in training the team, developing skills, and then using those skills can add to the Average Client Transaction or ACT, create ways the employees themselves can generate revenue, and allow the DVMs to stop working on non-revenue tasks below their skill level instead of working on revenue-generating tasks that only a DVM can perform, i.e., Prognosis, diagnosis, surgery, and prescribing.

We can also look internally and evaluate areas in which we may have room for improvement, such as missed charges, practice inefficiencies, use for IT or AI, decreased expenses such as expired drugs or theft, wasting materials, etc. Often, we can find "missed" revenue right in front of us!

SUMMARY INCLUDING 5 KEY "TAKE HOME" POINTS

1. Assessing the current KSAs identifies the areas that can be developed through training and development.

2. Creating department levels outlines duties, skills, and compensation for employees to work toward leveling up for professional growth.
3. Developing career maps for employee development.
4. Total team utilization of employees to the maximum of their individual skill set engages the employee in the work they perform.
5. Compensating employees fairly and providing a livable wage to ensure they can make their chosen profession one they can stay in!

SUMMARY

Our goal is to be able to retain top talent and decrease turnover, keeping the team intact! Look internally at these four major areas of employee retention, including utilization, total compensation, growth and development, and overall employee buy-in. Where is there room for improvement? What can the practice do to move the needle in one or all four areas that might keep top talent in your practice from leaving? Retention is the key focus!

15001

BUILDING EMOTIONAL CONNECTIONS

CLIENT EXPERIENCE

Alison Lambert, BVSc CMRS

There is often something of a fundamental mismatch between the things we communicate as scientists and clinicians, and the things clients hear as anxious owners. We can quote facts, prognoses and case studies, use complicated terminology and drug names and forget when summing up our findings that most clients don't know as much detail about feline anatomy as we do. This can create barriers between us, which prevent the vital emotional bonds being formed between vet and owner. Instead, we need to:

- Consider feelings as well as facts
- Find common ground (a mutual wish to provide the pet with the very best care)
- Grasp the opportunity to care

Remembering why people have pets, and appreciating the role they play in the family dynamic, helps us build genuine and strong connections with our clients and reframe 'difficult' clients simply as worried or stressed owners.

Five types of pet owners

I believe there are five different categories into which all pet owners fit. And whilst their motivations and behaviours may differ, they all have one crucial thing in common – they care for the pet. I've categorised them according to how they see their pet, as a:

1. Baby
2. Friend
3. Child
4. Manifestation of their expertise
5. Lodger (the pet may have come into their life by chance – feeding a stray, caring for a relative's pet etc.)

In all cases, when you speak with the client, they are responding not as someone choosing a service based on cost, convenience or preference, but rather as a parent, friend or guardian. Someone with a vested emotional interest in the wellbeing of the pet. This means never referring to 'the animal', 'it' or 'your cat'. The language you use and the tone of voice you adopt should reflect the status of the relationship between pet and client. Only then can you build emotional connections, demonstrate that you care and come to see 'difficult' clients as people acting in the best interests of a loved one. Emotions may be heightened at times of stress – when pets are injured or unwell – but placing the pet's needs front and centre will show the client that you truly care, and they will trust you to do the right thing.

There are some practical communication tools you can employ to help build these emotional connections with your clients. These include:

Minimizing jargon

As members of the veterinary team, we're in the business of helping our clients provide the best care for the pets in their lives. If we're expecting them to follow our treatment plans at home, or keep up with medication regimes as prescribed, then it's vital that they understand what we're asking them to do. Owners want the best for their pets, so if they don't do as we ask then it's most likely to be because they didn't really know what it was we were asking. To avoid this, in the consult ask yourself two things:

- What do you want the client to hear?
- What do you need the owner to do?

Intentionally design the conversation around these objectives, so you can be confident that the client will

understand and remember what you have told them when they get home. Think about the words and phrases you choose and strip out any jargon (unless you're talking to a fellow vet or medic). Think how it feels to be the pet owner on the receiving end of an overly detailed post-surgical debrief, like the one who I recently witnessed looking befuddled, after being asked by an orthopaedic veterinary surgeon to watch for distal limb oedema, when pointing at the dog's leg and saying "look out for any swelling here" instead, would have been instantly understood.

SIMPLIFY THE MESSAGE.

Clarify next steps.

Research consistently proves that the words we use make a massive difference in the outcome of the conversation. A study in the human medicine field by Heritage et al (2007)* found that changing just one word in a conversation delivered a statistically significant uplift in the number of patients reporting all their concerns:

In the scenario, "Before we deal with that, are there any other things you would like us to address?" 50% of patients reported additional concerns. When the words were changed to "Before we deal with that, are there some other issues you would like us to address?" a massive 90% of patients reported additional concerns. Once again, an opportunity to apply best practice on conversations in the consult room, with fewer words, better said.

* "Reducing Patients' Unmet Concerns in Primary Care: The Difference One Word Can Make" Heritage et al

I hear you, I feel for you

Social psychologist Dr Amy Cuddy has written about the disconnect between medical professionals and patients. The former generally value competence above empathy, whilst for the latter group the opposite is true. Clients would much rather feel that you truly understand how worried they are about Max's leg injury than hear you tell them in detail exactly how you are going to fix it. When our clients trust us, when they know that we will always recommend what's best for their pet, then those pets will always benefit from the very best care because the owner is fully bought-in to what is being done and why. Dr Cuddy explores the delicate balance between trustworthiness and strength in her 2012 TED talk, 'Your body language may shape who you are', named by The Guardian as 'One of 20 online talks that could change your life'. It's well worth a watch.

Communicating authentically

Throughout every experience with your clients, it's vital that the team create memorable moments so that clients trust them and feel bonded to your practice. Only in this way will they become advocates and recommend you to family and friends. This is crucial, because Onswitch research consistently shows that the biggest single reason quoted by pet owners for choosing a practice is word of mouth.

Everybody in the team, whether on the front desk or in a nurse or vet-led consult, must show the owner that they get it. They understand what it is to have a pet in the family, they respect the client's views as the person who knows the pet best. Treatment and care pathways are tailored to the individual needs of each animal. It's about being authentic and genuine in every conversation.

Communicating effectively

The communication process is made up of four key components:

- Encoding
- Medium of transmission
- Decoding
- Feedback

If we want clients to hear what we're saying and act on it, it's vital that we consider each of these things. But we must also consider two other factors in the process – the sender and the receiver. The person who you are communicating to in not always in the same metaphorical place as you, they may be angry, stressed, worried about cost, distracted by some other crisis unfolding in their life etc. Without an understanding of the context, and without an emotional connection, communication can never be truly effective.

15002

WITHOUT TRUST THERE'S NOTHING

CLIENT EXPERIENCE

Alison Lambert, BVSc CMRS

In this session we'll look at the fundamental elements of trust: credibility, reliability and intimacy. Personal interactions can't be outsourced, and they are essential for establishing trust. We'll explore the roles of 'social proof', human instinct and reciprocity in shaping the client experience, driving recommendations and boosting reputations.

THE IMPORTANCE OF TRUST

Trust is a key component of any strong relationship, and that between clinician and owner is no exception. The Trust Equation was first introduced in David Maister's book 'The Trusted Advisor', published in 2000. He postulates that as trust is the foundation of all relationships (including ones based on commercial principles), without it the relationship is poor.

Of course, when any business has a weak relationship with its clients, those clients drift away, spend less and do not recommend to their friends. Trust is key, and it is made up of three factors, the sum of which is reduced by the extent to which the professional is deemed to be acting in self-interest rather than protecting the needs of the client and their pet:

$$\text{Trustworthiness} = \frac{\text{Credibility} + \text{Reliability} + \text{Intimacy}}{\text{Self-orientation}}$$

Our profession does not generally need to work on credibility, and hopefully your service is reliable. Where there is often more work to do is in the areas of improving intimacy with clients (building genuine empathy and developing emotional connections) and in dialling down the self-orientation. It's not about us, it's about the pet and the client.

Communicating openly and honestly with owners helps build trust - presenting a range of costed options and discussing each with the owner so they are involved in decision making, letting the owner know where treatment is not required and providing prescriptions so clients can source long-term medications more cheaply online without judgement are all practical ways in which in which vets and practices can become more trusted.

The principles of reciprocity

Fundamentally, the world works better when we all get on. When we are thoughtful, considerate and kind. When we behave towards others in a way that we hope they reciprocate. Growing up we were told "being nice costs nothing", and whilst it's certainly true that it comes at no expense to ourselves, I would argue that it brings valuable benefits both in life and in business.

In veterinary practice, practical examples of what reciprocity looks and feels might include:

- Working closely with clients, listening to their concerns and history, and tailoring an action plan according to the needs of both pet and owner
- Using active listening – making eye contact, nodding in agreement, not standing with your back to the owner whilst you type up notes as they speak
- Ensure that all interactions are grounded in emotions as well as facts (acknowledging and accommodating when clients are upset, angry, scared etc.)
- Being honest about costs, options, waiting times etc.
- Taking care not to lose the personal touch when using technology (apps, chatbots and messaging services might be quick, but they can feel very impersonal).

This isn't just some idea that you begrudgingly pay lip service to; being human and caring as a service provider makes clients like you, trust you and tell others about you. And strong levels of repeat business and recommendation are always going to be good for business.

Building trust

At Onswitch, we know that the best patient outcomes only happen when the practice team work closely with the people who care for them. It takes time and empathy to build relationships founded on trust and respect. If you think about the time our clients spend with their pets, relative to the very small number of veterinary minutes they experience in any given year, the ratio is hugely in their favour. They know their pets inside out, after a ten or fifteen minute consult with the vet, it's they who do the caring – checking wounds, administering medication, following treatment plans and observing progress. They are just as much experts as we are in the care of their individual, beloved four-legged family member.

Onswitch carries out a lot of research with pet owners, and one thing that always comes up is the importance of the vet / client / patient relationship. Many owners are evangelical about their practices, citing multiple examples of how the team greet their pets by name, understand their personalities and work with clients as equal and trusted partners. They tell us time and again how they found their practice through recommendation – often from family and friends, but also through breed forums, social media, google reviews and other animal services (catteries, tack shops, groomers etc.) Great customer care is about making positive memories – we remember how people made us feel, and we tell others about it.

The concept of social proof

Humans are pack animals, we like to have others around us and we often mirror and mimic the opinions and actions of others in order to fit in. Social proof is a psychological phenomenon where people copy others so that they behave 'normally' or in a way that's deemed societally acceptable. It was first named by Robert Cialdini in his 1984 book 'Influence: Science and Practice.

In the veterinary context we can use this concept to build connections with our clients. When we make recommendations by framing them as "most of our clients book an annual health check for their pets", we're helping our clients feel like part of the gang. Obviously this is counter-productive if you're recommending products or services that are clearly not delivering the best care for the pet, or are felt to be driven by profit. But when the client trusts you and you have established a strong emotional connection, social proof helps strengthen that trust further.

15003

MOMENTS OF TRUTH

CLIENT EXPERIENCE

Alison Lambert, BVSc CMRS

In order to understand what moments of truth are, and where they occur, we first need to consider the customer journey. Several touchpoints on the customer journey happen before an owner even speaks to you. This means that it's crucial that your web and socials amplify your ethos, beginning emotional connection with pet owners right at the start. When they are doing their research about which practice to choose, potential clients want to see your team and feel that you 'get it', they don't need photos of equipment and reams of sterile copy.

UNDERSTANDING THE CUSTOMER JOURNEY TO AND THROUGH YOUR PRACTICE

The concept of the customer journey is not new, describing the many touch points experienced between client and practice. This journey begins long before the owner has set foot inside the building, with searches carried out online for reviews and information, recommendations sought from friends, family and professionals (groomers, catteries, pet shops etc.) and impressions formed through local advertising and open days, not to mention practice branding and appearance.

There are four stages to the customer journey, and the experience your team provide at each one will be crucial in determining whether the owner moves on to the next step:

- Find you
- Contact you (call and contact conversion)
- Visit you (consult conversion)
- Talk about you (recommendation)

MOMENTS OF TRUTH

Marketers used to talk about the first moment of truth in the lifecycle of a product or brand, the point at which consumers become aware of it. Historically for vet practices this would have been as a result of walking past the building on the high street, or hearing recommendation from a friend. However, now that we all have access to limitless online resources, potential clients find out about a practice often without even looking for it. Social media, blogs, online forums, webcasts, websites and the like fill our subconscious with ideas. This has led to the idea of the Zero Moment of Truth, becoming aware of a service, product or business in the virtual, online space rather than in a physical one.

There are many touchpoints on the customer journey, and new ones are being created all the time. Technology is certainly a useful tool at every touchpoint, but it is purely an enabler for connection – emotional bonds are only formed when the tone and messages shared are authentic and consistent. All along the customer journey, clients are looking for genuine interest and care to be shown towards their beloved pet, and for a rapport to develop when both owner and practice share the same values and aspirations as to what great customer care feels like.

A potential client who calls to ask the price of your booster vaccinations should therefore not just be told the cost, but engaged in a friendly conversation that demonstrates warmth and interest – asking the pet's name and age, sharing an insight or anecdote about the joys / pains of pets and explaining that a full heath check is part of the booster appointment. You're not selling a product, you're providing a whole experience. People care about people, they choose businesses where they feel the team 'get them', they choose a practice where the team clearly get what it is to love a pet.

THE FIRST CALL

When pet owners have done their research, they may well pick up the phone to speak to the practice. At this key touchpoint it's vital that they feel valued, respected and

understood. Onswitch's 5 Steps is a great way to structure the way you handle these inbound calls effectively and with warmth, helping pets and their owners access the best care from your team.

1. Use your name and give a great greeting
2. Use the pet's name and get the picture
3. Answer the price question at the end (demonstrate Love, Value, Price in that order)
4. Provide practice information (social media, website, health plan)
5. Always offer an appointment

Fewer words, better said at this crucial client touch point will help the customer care team deliver a consistently excellent customer experience for clients. Great customer care on the telephone can be summarised as follows:

- Listen
- Empathise
- Personalise – use the pet's name
- Sort a plan
- Recommend next steps

By the time clients get to the Second Moment of Truth – experiencing your clinical care in the consult room – owners should already be primed to your unique practice ethos, having begun to bond with the practice through a consistently warm and engaging approach. Stepping into the consult room, they have begun to trust the team. Your job as vets and nurses is to further cement this engagement through a great consult experience.

DEVELOPING CONTEXT-SPECIFIC CARE

Consults are part of your everyday routine in practice, you see many patients and clients every day. If this seems obvious, I mention it only because it's far from normal for your clients. They perhaps come to see you a couple of times a year, for ten minutes. They've had to plan each visit into their busy lives and the consult makes up a tiny amount of the total time they spend with their pet. Your client experiences the consult in a totally different way to you, because their context is so very different. To you it's a job, to them it's a worry, a logistical challenge or a relief.

It's important to remember that when a client comes to see you, their emotional state will be affected by the reason for their visit. If their pet is injured or unwell, the consultation will not have been planned in advance and the owner will not know what the outcome is going to be. Heightened emotions can make people act more irrationally, and so this is something to be aware of when structuring your conversations. Conversely, if the appointment has been planned for routine care (vaccines, de-sexing etc.) then the client will be in a very different emotional state and practical context.

All the research carried out by Onswitch over many years tells us that clients want to see and feel that their vet understands their pet and can deliver care that is personalised and appropriate. They want a clear plan as to what you've found and what you're going to do. They want clear recommendations and care that is specific to the needs and personality of their much-loved family member.

BUILDING A COLLABORATIVE CARE AGREEMENT

Good patient outcomes are only achieved when we work closely with the people who live with them. People who know and understand their animals far better than we ever can. Developing a strong patient / client / vet relationship is therefore always the goal, and this is only achieved when we build trust and develop a connection. Following a proven consult structure allows for a repeatable process to be put in place in the consult room, so that the short time you are with the client and their pet really counts.

7 STEPS TO A GREAT CONSULT

Onswitch train and promote use of the 7 Steps - a simple and clear set of guidelines to help vets and nurses deliver superior consults, developed from the Calgary Cambridge model used in medical schools across the world. The model is based on the principle of building an open and trusting client / clinician relationship through a standard process - asking for information and listening to the client, collecting evidence through a physical examination, explanation of findings, recommendation and planning of next steps, followed by a decisive close:

1. Prepare yourself (make sure the room is clean and tidy, and the items you're going to need are close at hand - read the notes and remind yourself what the patient is in for)
2. Create a rapport (introduce yourself, make eye contact, listen actively, engage in conversation and use the pet's name throughout)
3. Ask open questions (what, when, why, how - questions that require elaboration rather than a yes / no answer)
4. Carry out an obvious patient examination. The biggest cause for complaints in practice is that clients did not understand what was going to happen in the consult room, or did not see it happening. Narrating what you're looking at, and why, is very helpful here
5. Make clear recommendations (don't use phrases such as "I think..." "Perhaps we could..." and "let's wait and see...")
6. Check understanding and signpost next steps (studies show that compliance is good when owner and vet agree on the priority for the patient, but conversely it is poor when the vet does not address the owner's initial concern)
7. Book the next appointment / contact

* "Skills for communicating with patients". Third edition, 2013. Jonathan Silverman, Suzanne Kurtz, Juliet Draper

15004

TECHNOLOGY IS TRANSACTIONAL BUT HUMANS NEED RELATIONAL

CLIENT EXPERIENCE

Alison Lambert, BVSc CMRS

Between us, the Onswitch team talk to lots of veterinary professionals, working with business owners, vets, nurses and customer care teams around the world to help them deliver a truly customer-centred service. And whilst there are always things to be done within their businesses - improvements to processes, training for people, development of longer-term strategies etc. – increasingly we find ourselves talking about the stuff that happens outside the practice. The bit before you.

You can bet that every single pet owner who calls your practice, sends a message or launches a live chat with your team has done a lot of research and a lot of thinking before reaching out to you. There are countless sources of advice, information and support available to anyone looking for help:

- Friends and family recommendations
- Groomer / farrier / cattery advice
- Video vets (often free with pet insurance policies)
- Breeder forums
- Facebook pages
- Social media influencers
- Online reviews
- Blogs

Of course, this informed reality doesn't just relate to veterinary care. Think about the last holiday you booked, the last time one of your household appliances broke down, the driving lessons / dance class / Scout group you needed to find. Chances are you used at least one of the places above to find solutions. It's just what we do now, it's human nature to try and find answers ourselves before enlisting the help of others. Which means that whilst there are undoubtedly many more 'bits' before you than there used to be, many practices haven't yet understood what this means for them

DIRECTING THE CUSTOMER JOURNEY

Long before pet owners call you, if they are not already clients they're finding out about you. Asking around, checking your socials, looking at reviews, googling 'vets near me'. That's why it's so important that the picture of your practice that they create from all this research reflects the warmth and professionalism of your team:

- Your website should be engaging and unique, featuring photos of the team as well as your practice story and mission
- Social media pages are refreshed regularly with lots of stories and photos of the people and animals you care for
- Online reviews are always acknowledged and responded to

This is where owners are forming impressions of their options, deciding who to call, who to trust with the care of their beloved family member. Because you aren't the only vet they could choose, and they aren't just going to choose any old practice.

CONNECTING PATIENTS TO GOOD OUTCOMES

Given all the work that's gone into researching their options and deciding who to contact, once you have a potential client on the phone or in your inbox there's only one thing to do – offer them an appointment. Welcome them into your care, reassure them that they have done the right thing by calling you, and that your team will get

the best outcome for their pet. Make it easy, show that you understand. You may well answer 50 of these calls today, but for the person on the phone with you right now, this is the only call they'll make to a practice today, and right now it matters more to them than anything else. Make them feel heard, acknowledge their unique needs and reassure them that you can help.

Please don't tell them to go to your website to register first.

Don't tell them that you haven't got any appointments.

Don't ask them to email in because there isn't anyone who can help right now.

It may well be that you don't have many appointments available, especially if you're struggling to recruit and retain a full team. If that's the case, it's vital that you review your practice protocols and look at where you can work smarter:

- Booking nurse-led appointments where appropriate
- Improving admission procedures to reduce double-handling of data and time
- Ensuring that everything that can be checked and measured before the patient gets into the consult room is done, so that vets can use their clinical minutes doing clinical things

Do everything you can to see the animal that the caller so desperately wants to bring to you.

PRICE IS A FACT. VALUE IS A FEELING

In the average practice it's hard to get away from the sense that cost is a 'big thing'. There are laminated price lists at the reception desk, notices in capital letters telling clients there are no credit facilities, printed estimates run to several sheets with every single canula and bandage itemised with product codes (with and without taxes). It all feels very transactional. It's easy to see how clients may feel that vets are only interested in the money – after all, that's mostly what they talk about. Facts, percentages, fees – as scientists, these can be what clinicians choose to focus on. They are objective and tangible.

But owners need emotions more than facts. They may be stressed, worried and sad about their beloved family pet and they want the best for them. Sure, they understand that it will cost, and they know that you are an expert, but they need to feel valued, respected, cared for. How you communicate is more important that what you're saying, especially in highly emotive situations. This is especially important to get right when using technology. Apps, chatbots, AI, virtual vets and messaging services have all greatly increased convenience both for our clients and the team, but this can come at a price. Humans need connection, we buy from and do business with people we like. So:

- Make sure the standard templates you programme into chatbots to acknowledge the contact reflect the tone of your other practice communications
- Ask for and use client's and pet's names when messaging
- Use your own name and sign off with a cheery goodbye when appropriate
- Manage expectations – if it's going to be a while till they hear back, then say so
- Keep jargon out of the conversation
- Type how you talk

FINDING COMMON GROUND

If vets are about facts and owners are about feelings; when the practice sets a price but the client needs value, the common ground both sides share is the patient. Great care happens when practice and client collaborate, when vets and nurses talk about outcomes before cost. We need to tell stories, build narratives about the benefits of the care we provide. It's not just removing a cracked tooth, it's helping Mavis enjoy her food again and maintain a healthy weight.

As always, this narrative needs to run through every client touchpoint, so it starts before you've even met the patient. Web copy and social media feeds the put care front and centre. Show that you understand, you get it and you're open and honest. That you'll always do what's right for the animal. A great way to do this is with fixed pricing for everyday items and common procedures. Clients know that a hip replacement costs $xx, a spay for a large Labrador costs $xx. No scary range of prices, where there can be hundreds of pounds between best and worst case. No estimates running to several pages. Just one fee – you do this often enough to know what these

things cost. You can come up with a price that covers your costs across the range of patients you see. Several referral hospitals and orthopaedic centres already offer this, not to mention an increasing number of small animal practices. Clients prefer the knowable, especially at a time when so much else might be uncertain. When you have a relationship based on trust, they understand that your price is fair, they can see the great care the whole team provide that gives them value.

"PRICE IS WHAT YOU PAY, VALUE IS WHAT YOU GET"

Warren Buffett's much-quoted mantra is so relevant to every veterinary practice. Make your pricing simple and honest, show care and compassion at every stage of the client journey and you can't help but prove the value that your team provide.

15005

LISTEN, EMPATHIZE, PERSONALIZE

CLIENT EXPERIENCE

Alison Lambert, BVSc CMRS

It's time to consider the principles of effective consult communication – helping you find common ground with the client so the patient receives the best care. The words you use and the way they are delivered affect what the client hears. Trust is only earned when genuine connections are forged.

LISTEN, EMPATHISE, PERSONALISE, SORT A PLAN, RECOMMEND NEXT STEPS

This is a simple, but highly effective, tool that everyone in the team can use to help build trust with the client. It starts when you listen to what the client tells you they are concerned about (which might be different to what's in the notes or what you have spotted as the pet comes in). Put away preconceptions and judgement and listen to what the owner says. We need to create space for them to say how they feel and really be heard.

LISTENING AND HEARING ARE NOT THE SAME.

The customer experience is shaped by a wide range of senses and emotions. Practising active listening and using the KLARDOC approach in client conversations will both help both parties get the most out of the situation (especially if it is any way fraught):

Keep calm

Listen: Put assumptions aside and really hear what the client is telling you

Acknowledge: Repeat what you have just heard, for clarity

Refine and Define: Summarise the key facts, aside from the emotion

Overcome: Present possible solutions and alternatives

Close: Thank the client and detail the timings of any further action

Almost always, a 'difficult' conversation stems from the fact that your client is frustrated, confused, worried or distracted. Understanding their state of mind and looking at the situation from their perspective will usually help inform a solution, and it always helps to use calming, reassuring language:

- Don't use technical language or practice jargon
- Keep your body language open and professional
- Speak at the same pace as them
- Repeat key words and phrases back to them
- Use open questions to ascertain what the problem is
- Ask politely for clarification of the issue, don't assume

DEMONSTRATING EMPATHY

Empathy is a crucial component of trust – the client should feel that you truly understand their concerns, and feel how they feel. It's about demonstrating that you understand. "I can see that you're worried about Kitty's dry skin, I'm going to carry out a thorough examination so that we can find out what's causing her to scratch so much, and then I can recommend the right treatment."

Finally, personalise your recommended care pathway, "Because Kitty is a hunter, I recommend that we give her a monthly wormer" for example.

As vets, our aim is always to optimise patient outcomes. We do this best when we work collaboratively with the people

who live with them – their owners, our clients. This means showing that we care, building trusting relationships and making it easy for them to find information and advice from everyone in the team.

Every day, with every client, at every touchpoint on the customer journey.

BUILDING A COLLABORATIVE CARE AGREEMENT

Good patient outcomes are only achieved when we work closely with the people who live with them. People who know and understand their animals far better than we ever can. Developing a strong patient / client / vet relationship is therefore always the goal, and this is only achieved when we build trust and develop a connection.

Following a proven consult structure such as the 7 Steps allows for a repeatable process to be put in place in the consult room, so that the short time you are with the client and their animal really counts.

1. Prepare yourself (make sure the room is clean and tidy, and the items you're going to need are close at hand - owners hate it when you leave the room mid-consult so read the notes and remind yourself what the patient is in for)
2. Create a rapport (introduce yourself, make eye contact, listen actively, engage in conversation and use the pet's name throughout)
3. Ask open questions (what, why, how - questions that require elaboration rather than a yes / no answer)
4. Carry out an obvious pet examination
5. Make clear recommendations (don't use phrases such as "I think..." "Perhaps we could..." and "let's wait and see...")
6. Check understanding and signpost next steps (studies show that compliance is good when owner and vet agree on the priority for the patient, but conversely it is poor when the vet does not address the owner's initial concern)
7. Book the next appointment / contact

Increasingly in the UK, the NHS and animal healthcare sectors are prioritising the provision of context-specific care – understanding the needs of each pet and the circumstances and expectations of their owners. It's not about a gold-standard veterinary service, it's more important to do the right thing for every individual pet. A pragmatic approach to what's 'best', agreed in collaboration with clients, will create strong connections with pet owners, build the practice's reputation and boost recommendations from appreciative clients.

TOP TEN TIPS FOR EFFECTIVE COMMUNICATION

Finally, here are my top tips to remember when speaking with clients. Get into the habit of using these techniques in your consults and you'll find that patient outcomes improve (owners understand what you need them to do at home) as will client engagement levels.

1. Fewer words, better said
2. Listen more than you speak
3. Empathise with the client
4. Personalise your approach
5. Avoid jargon
6. Active listening - open body language and eye contact
7. Make clear recommendations
8. Use the patient's and client's names
9. Focus – don't type up notes as you speak
10. Control your emotions – stay calm

Effective communication builds strong connections.

16001

THE CLIENT EXPERIENCE, UNPLUGGED

PLENARY SESSIONS

Alison Lambert, BVSc CMRS

It's the human interactions that determine how consumers feel about any service they receive. Empathy, understanding and authenticity are essential for an excellent customer experience. Your clients value actual intelligence, not artificial – emotional connection trumps apps and algorithms every time. This session will explore the theory and evidence supporting an unplugged client experience.

We'll start by going right back to basics, considering why people have pets, what are the different roles they play in your clients' lives? Because in order to fully understand what makes the best client experience, we need to fully understand who our clients are. What they want, how they think and how they behave. What makes them tick. Looking at the world that our clients live in, and considering how many of things we take for granted (as customers and clients of other businesses ourselves) can be delivered by the average veterinary practice. And there are not as many as there should be!

Some things are particular to pet owners, but a significant number of the factors that influence customer expectations and behaviours are common across all areas of life. Think about how you find a new product or service – you might look at TripAdvisor and google reviews, check social media comments and feedback, ask your friends and family or Insta community. Then when you've found it, you will probably download an app to help you manage appointments, use Apple Pay to settle up, sync appointments with your phone calendar, check messages for reminders, subscribe to a monthly delivery etc. All of these things you do without thinking, but how many of them does your practice offer to the pet owners who choose you?

Our clients are busy people who live in a different world than the one that exists in the context of the traditional veterinary practice. Today's clients' needs and expectations are very different to those from the 'glory days' when some of you first set out into practice, and this is not a bad thing. We simply need to make sure that the customer experience we provide is in line with what they rightly expect from every service business.

BUILDING STRONG AND ENDURING CONNECTIONS

We can only truly deliver good patient outcomes when we work with the people that live with them. This means clinicians and customer care team members developing trusting relationships based on a mutual desire to provide the best care for the pet. Without a human connection trust cannot exist. All the process efficiencies and AI technologies in the world cannot replace a personal bond. We must bring our clients closer to us, form genuine connections and demonstrate empathy when they are worried, stressed or angry.

This has always been important, but since Covid it's become more important than ever. During the pandemic we pushed our clients away to a safe distance, quite literally. We held consultations in car parks and over the phone, body language cues were hidden behind masks and social distancing. And whilst that was almost five years ago now, many of the outsourced communication channels and technologies that we quickly adopted remain with us. Mostly that's a good thing – it's great that clients can now book an appointment online when they suddenly remember an overdue booster vaccination, sitting on the sofa late at night.

But. We seem to have lost sight of the fact that our clients are human beings who love their pet deeply. They don't want to know how much their pet weighs when they book a health check, they want to be reassured that their beloved family member is healthy and happy. They want the nurse to remember their pet's name and make a fuss of them.

They want eye contact, genuine interest and a recognition that their pet has unique and specific needs. In short, they need to see some empathy and emotion. They want their service providers to unplug literally and metaphorically from their phones and computers and be fully present.

The starting point for every single interaction, every day, that occurs between clients and everyone at your practice is the love the client has for their pet. Fail to acknowledge and honour that, and you fail to deliver a great customer experience.

A DIFFERENT APPROACH FOR DIFFERENT TIMES

In case you're thinking this is all a bit 'new age', a 'nice to have' rather than a 'need to do', let's also consider how the veterinary business context has changed. Covid shifted many things in our society, and the veterinary profession is no different. Some relevant facts to consider when it comes to delivering a great, unplugged customer experience in our 'new normal' are:

Covid has levelled the playing field

Everyone was hit hard by coronavirus, but not every practice was badly affected. Many practices weathered the pandemic well, especially independents. The common factor for success was that they all had effective and rapid decision-making processes in place.

Clients value consistency

Many practices are struggling to recruit and retain vets, often running on locums and blocking out days with no appointments as there is no vet on site. This is both detrimental to the business and irritating to clients, who want to see familiar faces and develop bonds of trust with their vets and nurses.

Veterinary practices are essential, even with many products and services now online

The vast majority of small animal owners want care that's local (within a ten-minute drive) and convenient (evening and weekend opening, remote consulting, online options for making appointments and payments etc.) Being able to self-service as much as possible online, out of hours is expected – clients want to book appointments, check medical histories, send messages and register with a practice in their own time online.

The cost of living crisis has changed the rules about affordability

With mortgage rates high and household budgets squeezed, even middle class pets owners are finding they have fewer funds available to pay for unexpected veterinary care. Funding expensive referral fees is likely to be much more challenging for the 'squeezed middle'. First opinion practices that can carry out a majority of tests and procedures in-house will thus do well.

The peak age for pet ownership is 30 – 50

It's important to have sufficient potential patients, so your target audience is clients aged 30-50. Pet ownership is most likely in owner-occupied houses (rented accommodation and flats are often not permitted to have pets). Some cultural and religious beliefs also make pet ownership less likely. All of these considerations must be applied when looking at potential business success of veterinary practices.

17001

INFECTIOUS DISEASE ROUNDUP 2025

PLENARY SESSIONS

Maureen E.C. Anderson, DVM, DVSc, PhD, Dip. ACVIM
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STAY ALERT: HIGHLY PATHOGENIC AVIAN INFLUENZA A(H5N1) IN COWS, CATS, MICE

Between March and October 2024, highly pathogenic avian Influenza A(H5N1) was detected in almost 400 US dairy herds in 14 US states. As of October 2024, it has yet to be detected in dairy cattle or dairy products in Canada. Infected cattle all seem to be infected with the same strain of the currently circulating H5N1 virus 2.3.4.4b (genotype B3.13), which indicates that a single cross-over event occurred (probably in late 2023, likely from wild birds), and this strain subsequently spread amongst dairy cows and dairy farms, most likely via milking equipment and animal movement. However, spread of this strain from cattle to commercial poultry has been noted. Cats and rodents on affected US farms appear to be at increased risk of infection with H5N1 , likely at least in part to the large amount of virus shed in milk of infected cows and therefore increased exposure on these farms. Infections in dairy herds and increased surveillance testing in humans exposed to infected cattle and poultry resulted in detection of 36 cases of human H5N1 infection in the US in the same time period in 2024. Fortunately clinical signs were mild in all these cases, and no human-to-human transmission was suspected, but scientists remain on alert for genetic mutations in the virus that may allow it to transmit more easily between mammals, and potentially spillover more easily into people with subsequent human-to human transmission.

No H5N1 outbreaks were detected on poultry premises in Ontario from February to October 2024, but cases were detected in commercial poultry in BC and Saskatchewan in October 2024. The virus continues to circulate in wild birds in North America, and vigilance remains critical, particularly during spring and fall migration seasons. The current H5N1 virus has been detected in over 30 mammalian species. To date, H5N1 has only been detected in one dog that passed away several days after being found scavenging a dead goose that also tested positive for the virus, in March 2023.

Three feral cats with severe respiratory / neurological signs in Peel Region and Lambton County (Ontario) also tested positive for H5N1 virus back in 2023. A recent series of H5N1 cases in cats from Colorado included two "indoor" cats with no clear exposure to known sources of the virus, which highlights the ongoing gaps in our current knowledge of how this virus may be transmitted and spread. The companion animal and wildlife OAHN teams, along with CWHC, continue to collaborate on a joint pilot project on H5N1 influenza in primarily outdoor / feral cats. Visit the project webpage for details on case eligibility, sample collection and submission.

Veterinarians are encouraged to keep H5N1 infection in mind for any cat presenting with severe acute neurological or respiratory signs or sudden death, especially (but not exclusively) if they have potential contact with other infected animals in the area (e.g. birds, cattle, rodents). PCR testing for Influenza A is available through veterinary diagnostic labs in Ontario. Any influenza strain in a companion animal is immediately notifiable and will be reported by Ontario laboratories to both OMAFA and the Ontario Ministry of Health. Although the risk of members of the general public contracting this strain of H5N1 influenza is still considered to be very low, owners are encouraged to take appropriate precautions to protect their pets and themselves by avoiding direct and indirect contact with sick or dead wildlife, especially migratory birds. There are also precautions to take around livestock.

FELINE INFECTIOUS PERITONITIS (FIP) DRUGS UPDATE

In February, Health Canada approved access to remdesivir and compounded GS-441524 for the treatment of feline infectious peritonitis (FIP) through the emergency drug release (EDR) process. Efforts continue to improve access to these and other FIP treatments in Canada. Compounded GS-441524 can also be accessed by US veterinarians as of June 2024.

There is also now published evidence to support the use of a shorter treatment course (42 days vs 84 days) in cases of wet FIP that show a favourable initial response. Read the synopsis of the results, or the full study (Zuzzi-Krebitz et al. 2024). Check out the OAHN podcast: FIP treatments (and where to find them), with special guest Dr. Kelly St. Denis, for tips on using and accessing these drugs, as well as the Worms & Germs Pod episode on this topic for more information.

MPOX IN ANIMALS: GOOD NEWS, BAD NEWS

In May 2022, a global outbreak of mpox (formerly known as monkeypox) began, which ultimately resulted in over 87 000 human cases in 110 countries. Prior to this, this zoonotic disease occurred primarily in central and west Africa. African rodents are the most likely reservoir of the virus, but information on susceptibility of other animal species is very limited.

Good news: A US study that tested 24 dogs, 9 cats and 1 rabbit from households with at least one human mpox case in 2022-2023 found no evidence of infection in any of the pets, though some of the pets were contaminated with virus from the infected humans.

A new outbreak of mpox caused by the clade I MPX virus began spreading in 2024, which differs from the clade IIb MPX virus that caused the 2022 mpox outbreak. Bad news: As we cannot be certain that the two clades will behave the same way in other species, despite encouraging results from the study described above, the same precautions as before are recommended for avoiding animal contact, and for managing pets that may have been exposed to infected humans.

- Treat pets like a human member of the household by avoiding close contact as much as possible, covering (human) skin lesions and practicing good hand hygiene when handling the pet or its environment. Keep exposed pets indoors as much as possible.
- Preventing exposure of rodents, especially wild rodents, is particularly important, in order to avoid introducing the virus to the wildlife population where it could create a reservoir.

ECHINOCOCCUS MULTILOCULARIS IN ONTARIO

In the last decade, it has become clear that *Echinococcus multilocularis* (EM) is now endemic in wildlife in southwestern Ontario. In 2024, alveolar echinococcosis (AE) was detected in a chipmunk in Durham region (as well as in 2022 and 2023) and in a non-human primate. Fortunately infection in Ontario dogs remains rare (or at least rarely diagnosed), but with the increased availability of PCR testing more cases are likely to be detected. In 2024, fecal PCR- positive dogs were reported in Simcoe county, Middlesex and Niagara. All dogs were promptly treated with praziquantel and subsequently tested negative. No cases of AE in people were reported in Ontario from January 2023 to October 2024. Due to the insidious and severe nature of AE, it is crucial that we keep this disease on everyone's radar.

A recently published review of over 2 million fecal PCR tests from Antech Diagnostics (March 2022 to July 2024) found 26 dogs in the US and Canada that were positive for EM (Evason et al. 2024). It was notable that fecal floatation was only positive in 8/17 infected dogs that were also tested by this method. The reported geographic distribution was based on the location of the submitting clinic, not necessarily where the dog lived or was infected, but 25/26 dogs were reported not to have travelled outside their home country/state/province in the prior 6 months.

Remember that infected people develop AE, but they can only be infected by ingesting eggs from canid feces. Dogs are typically infected by ingesting tissues from infected small mammals and develop intestinal infections, *but occasionally can also develop AE from ingesting large numbers of eggs!* Check out the OAHN EM infographic or the 2022 OMAFA veterinary update on EM risk in Ontario.

Human rabies case, 2024

The first domestically-acquired case of human rabies in Ontario since 1967 was detected in a person who had direct contact with a bat in August 2024. The individual was hospitalized but nonetheless passed away in September 2024, once again demonstrating the extremely serious nature of this disease and the importance of knowing the risks associated with and seeking medical attention following any potential contact with bats (or any other potentially rabid mammal).

Raccoon-, fox- and bat-variant rabies in Ontario, 2024

As of October 2024, raccoon-variant rabies has not been detected in Ontario outside the St. Catharines region since 2022. No cases were detected in the spring or summer of 2024. The Ministry of Natural Resources continues oral rabies vaccine baiting for wildlife within 50 km of active cases (i.e. detected in the last 2 years) as well as preventative baiting along key parts of the Ontario-US border. Fox-variant rabies has not been detected in Ontario since 2018, which represents the longest period it has gone undetected since variant typing began in the 1980s. A publication detailing the efforts to achieve this momentous milestone is currently under review.

The highest annual number of rabid bats in Ontario since 2003 was recorded in 2024 (68 as of October, which represents approximately 16% of all bats submitted). While this was certainly a peak in the annual numbers, it is not unprecedented, and there is no reason to suspect an increase in the prevalence of rabies in the general bat population. Rabies is considered endemic in all bat populations in North America at a low level (~1%), which is why contact with bats is always a risk for rabies exposure. While it's important to beware of the risk of bats as rabies reservoirs, it is equally important to be kind to these amazing and ecologically critical creatures! Check out OAHN's N2K: Bats in Ontario resource for more information.

Rabies information for Ontario veterinarians is available on the Ontario.ca website, including risk assessment and post-exposure management guidelines. You can view or download a pdf of the OMAFA rabies risk assessment flowchart on the OAHN website. For additional resources and flowcharts, log in and visit the OAHN rabies resource page for veterinarians. Rabies information for the general public is available at Ontario.ca/rabies, including the MNR's interactive rabies case map.

Veterinarians should contact OMAFA for assistance with rabies risk assessments, sample submission or post-exposure management, as needed. Veterinarians can submit a request for assistance online using the rabies response request form. Requests submitted within business hours will receive a response the same day, typically within 1-2 hours. Requests can also be submitted outside of business hours and will receive a response the next business day; interim triage guidance is provided on the webpage. If you require assistance with completing the online form due to limited internet access or due to any other accessibility issue, please contact the OMAFA Agricultural Information Contact Centre at 1-877-424-1300 (option 1) during business hours (weekdays 8:30 AM - 4:30 PM). Animal owners who contact OMAFA directly concerning potential rabies exposures will be advised to contact their local veterinarian.

For more resources, see OMAFA's recent veterinary advisory on rabies in Ontario.

Chlamydia caviae in young guinea pigs

In early summer 2024, many companion animal veterinarians reported young guinea pigs from commercial retail outlets in Ontario with bilateral conjunctivitis, sometimes with respiratory signs. Given the likely common source, it is likely these guinea pigs had chlamydial conjunctivitis due to *Chlamydia caviae*.

An important differential for this condition in guinea pigs is infection with *C. psittaci*, the cause of psittacosis (also known as parrot fever) in people, which is often carried by psittacine birds. *Chlamydia psittaci* is notifiable to OMAFA, and notifiable to public health when it is found in birds. Guinea pigs are generally not capable of infecting humans with *C. psittaci; C. caviae* is a rare human zoonosis from exposure to infected guinea pigs. For more information, check out the OAHN *C. caviae* factsheet.

Viral encephalitides: EEE and WN

Eastern equine encephalitis (EEE) is an ever-present risk in certain geographical areas of Ontario. As for many

diseases, viral encephalitides like EEE and West Nile (WN) tend to cycle over the years. In 2024 there were at least 24 confirmed cases and numerous additional suspect cases of EEE in horses throughout Ontario, but especially in the eastern part of the province. Previous notable outbreaks of EEE include the same region in 2023, Parry Sound area in 2021 and Niagara region in 2018. There were also at least 11 confirmed cases of WN in horses, and over 100 cases in wild birds (compared to 61 in 2023). Wild birds and horses are both important sentinels for disease risk in people, and risk can also be affected by climate change and warmer, wetter weather. Rabies is an important differential diagnosis in any horse with signs of acute encephalitis. More information on the occurrence of EEE, WN and other notifiable diseases in horses can be found on the interactive Ontario Equine Immediately Notifiable Disease Dashboard (2018-2024).

RABBIT HEMORRHAGIC DISEASE VIRUS 2

No additional cases of rabbit hemorrhagic disease virus 2 (RHDV2) have been reported in Ontario since the end of June 2022, and a bivalent (RHDV1 & RHDV2) vaccine is available in Canada through CEVA Animal Health and veterinary distributors. However, cases have been detected in Quebec in 2023 and 2024, so vigilance is still needed! The latest case was a 7-year- old indoor rabbit that died 3 days after becoming ill. There were no other rabbits on the property, but this virus can be easily transmitted by fomites and survives in feed for months. Diagnostic testing is available in Ontario.

ADDITIONAL INFECTIOUS DISEASE RESOURCES & LINKS OF INTEREST

- UPDATED OAHN anti-parasitics for dogs and cats (Canada 2024): Available in both simplified (open access) and veterinary (OAHN login required) editions
- Raw meat-based diets infosheet developed collaboratively with OAHN and Worms & Germs Blog, to help talk to clients about the risks of raw
- OAHN drug-resistant hookworms in dogs factsheet: how to differentiate resistance vs larval leak, and how to manage cases (OAHN login required)
- OAHN antimicrobials for dogs and cats (Canada 2024): quick reference table for categorization of on label and off label drugs to help improve antimicrobial stewardship
- OAHN infographic for veterinarians on ticks and Lyme disease in Ontario with the latest risk area map for *Ixodes spp.* ticks from PHO. It also has quick tips on monitoring, screening, and when not to treat dogs. There is also an OAHN tick checklist for pet owners.
- OAHN "Need-2-Know: Rabies in Pets" whiteboard video: Narrated by Pogo the rescue dog, this 4.5 minute video helps explain to pet owners some important basics of how rabies infection works and the importance of vaccination, as well as why vaccination or antibody titres can't necessarily eliminate the risk of rabies in dogs from high-risk areas (either in Canada or outside Canada).
- Summary surveillance graphics about all the individuals and groups that contribute to disease surveillance in companion animals, horses and cattle in Ontario
- OAHN online disease reporting portal (for non-notifiable diseases) https://www.oahn.ca/companion-animal-disease-surveillance-submission-form/